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HUDSON RIVER PCBs REASSESSMENT RI/FS
RESPONSIVENESS SUMMARY FOR
VOLUME 2F - HUMAN HEALTH RISK ASSESSMENT

MARCH 2000

For

U.S. Environmental Protection Agency
Region 2
and
U.S. Army Corps of Engineers
Kansas City District

Book 1 of 1
Upper Hudson Risk Assessment

TAMS Consultants, Inc.
Gradient Corporation

304475

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 2
290 BROADWAY
NEW YORK, NY 10007-1866

March 24, 2000

To All Interested Parties:

The U.S. Environmental Protection Agency (USEPA) is pleased to release the Responsiveness Summary for the Human Health Risk Assessment for the Upper Hudson River (HHRA), which is part of Phase 2 of the Reassessment Remedial Investigation/Feasibility Study for the Hudson River PCBs Superfund site. For complete coverage, the HHRA and this Responsiveness Summary should be used together.

In the Responsiveness Summary, USEPA has responded to all significant comments received during the public comment period on the HHRA. In addition, the Responsiveness Summary contains revised calculations of cancer risks and non-cancer health hazards based on the modified future concentrations of PCBs in sediment, water and fish presented in USEPA's January 2000 Revised Baseline Modeling Report. The Responsiveness Summary also contains a comparison of the revised calculations to those reported in the HHRA. Importantly, the overall conclusions of the HHRA regarding the cancer risks and non-cancer hazards due to PCBs in the Upper Hudson River remain unchanged.

The HHRA is being peer reviewed by a panel of independent experts. The peer reviewers will discuss their comments on the HHRA at a meeting that will be held on May 30 and 31, 2000 at the Holiday Inn in Saratoga Springs, New York. The Ecological Risk Assessment will be peer reviewed by a separate panel on June 1 and 2, 2000 at the same location. Observers are welcome and there will be limited time for observer comment.

If you need additional information regarding the Responsiveness Summary for the HHRA, please contact Ann Rychlenski, the Community Relations Coordinator for this site, at (212) 637-3672.

Sincerely yours,

Richard L. Caspe, Director
Emergency and Remedial Response Division

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RESPONSIVENESS SUMMARY FOR
VOLUME 2F – HUMAN HEALTH RISK ASSESSEMENT

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ACRONYMS

ATSDR	Agency for Toxic Substances and Disease Registry
CDI	Chronic Daily Intake
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CSF	Carcinogenic Slope Factor
GE	General Electric
HI	Hazard Index
HHRA	Human Health Risk Assessment
HHRASOW	Human Helath Risk Assessment Scope of Work
HROC	Hudson River PCBs Oversight Committee
HQ	Hazard Quotient
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NPL	National Priorities List
NYSDEC	New York State Department of Environmental Conservation
NYSDOH	New York State Department of Health
PCB	Polychlorinated Biphenyl
RfD	References Dose
RI	Remedial Investigation
RI/FS	Remedial Investigation/Feasibility Study
ROD	Record of Decision
RM	River Mile
RI/FS	Remedial Investigation/Feasibility Study
SARA	Superfund Amendments and Reauthorization Act of 1986
SOW	Scope of Work
TAGM	Technical and Administrative Guidance Memorandum
TCDD	2,3,7,8-Tetrachlorodibenzo-p-dioxin
TEF	Toxicity Equivalency Factor
TSCA	Toxic Substances Control Act
UCL	Upper Confidence Limit
USEPA	United States Environmental Protection Agency

I. INTRODUCTION AND COMMENT DIRECTORY

1. Introduction

The United States Environmental Protection Agency (USEPA) has prepared this Responsiveness Summary for Volume 2F: Human Health Risk Assessment Report (HHRA) for the Upper Hudson River, Hudson River PCBs Reassessment Remedial Investigation/Feasibility Study (RI/FS), dated August 1999. It addresses comments received during the public comment period on this Report.

For the Reassessment RI/FS, USEPA has established a Community Interaction Program (CIP) to elicit feedback from the public through regular meetings and discussion and to facilitate review of and comment upon work plans and reports prepared during all phases of the Reassessment RI/FS.

The HHRA is incorporated by reference and is not reproduced herein. No revised copy of the HHRA will be published as such. The comment responses and revisions noted herein are considered to amend the Report. For complete coverage, the Report and this Responsiveness Summary must be used together.

The first part of this Responsiveness Summary is entitled "Introduction and Comment Directory." It describes the HHRA review and commenting process, explains the organization and format of comments and responses, and contains a comment directory.

The second part, entitled "Responses to Comments on the Upper Hudson River Human Health Risk Assessment," contains USEPA's responses to all significant comments. Responses are grouped according to the section number of the HHRA to which they refer. For example, responses to comments on Section 2.1 of the HHRA are found in Section 2.1 of the Responsiveness Summary. Additional information about how to locate responses to comments is contained in the Comment Directory.

The fourth part, entitled "Comments on the Upper Hudson River Human Health Risk Assessment," contains copies of the comments submitted to the USEPA on the HHRA. Not all references provided by the commentors are provided in this document. The comments are identified by commentor and comment number, as further explained in the Comment Directory.

2. Commenting Process

This section documents and explains the commenting process and the organization of comments and responses in this document. To find a response to a particular comment, the reader may skip this section and go to the tab labeled "Comment Directory."

2.1 Distribution of HHRA

The HHRA, issued in August 1999, was distributed to federal and state agencies and officials, participants in the CIP, and General Electric Company, as shown in Table 1. Distribution was made to approximately 100 agencies, groups, and individuals. Copies of the HHRA were also made available for public review in 17 information repositories, as shown in Table 2 and on the USEPA Region 2 internet webpage, entitled "Hudson River PCBs Superfund Site Reassessment," at www.epa.gov/hudson.

2.2 Review Period and Public Availability Meetings

USEPA held a formal comment period on the HHRA from August 4, 1999 to September 7, 1999, although USEPA has welcomed comments on the Reassessment throughout the study. USEPA held two Joint Liaison Group meetings to present the HHRA, one on August 4, 1999 in Albany, NY, and one on August 5, 1999 in Poughkeepsie, NY, that were both open to the public to answer questions from the public regarding the HHRA. Subsequently, USEPA sponsored two availability sessions to answer questions on August 18, 1999 from 2:30 to 4:30 p.m. and from 6:30 to 8:30 p.m. These meetings were conducted in accordance with USEPA's "Community Relations in Superfund: Handbook, Interim Version" (1998a). Minutes of the Joint Liaison Group meeting are available for public review at the Information Repositories listed in Table 2.

As stated in USEPA's letter transmitting the HHRA, all citizens were encouraged to participate in the Reassessment process and to join one of the Liaison Groups formed as part of the CIP.

2.3 Receipt of Comments

Comments on the HHRA were received in letters sent to USEPA. All written comments received on the HHRA are addressed in this Responsiveness Summary.

Comment were received from eight commentors. Total comments numbered approximately 100.

2.4 Distribution of the Responsiveness Summary

This Responsiveness Summary, will be distributed to the Liaison Group Chairs and Co-Chairs and interested public officials. This Responsiveness Summary will be placed in the 16 Information Repositories and is part of the Administrative Record.

TABLE 1
DISTRIBUTION OF HHRA

HUDSON RIVER PCBs OVERSIGHT COMMITTEE MEMBERS

- USEPA ERRD Deputy Division Director (Chair)
- USEPA Project Managers
- USEPA Community Relations Coordinator, Chair of the Steering Committee
- NYSDEC Division of Hazardous Waste Management representative
- NYSDEC Division of Construction Management representative
- National Oceanic and Atmospheric Administration (NOAA) representative
- Agency for Toxic Substances and Disease Registry (ATSDR) representative
- US Army Corps of Engineers representative
- New York State Thruway Authority (Department of Canals) representative
- USDOI (US Fish and Wildlife Service) representative
- New York State Department of Health representative
- GE representative
- Liaison Group Chairpeople
- Scientific and Technical Committee representative

SCIENTIFIC AND TECHNICAL COMMITTEE MEMBERS

The members of the Science and Technical Committee (STC) are scientists and technical researchers who provide technical input by evaluating the scientific data collected on the Reassessment RI/FS, identifying additional sources of information and on-going research relevant to the Reassessment RI/FS, and commenting on USEPA documents. Members of the STC are familiar with the site, PCBs, modeling, toxicology, and other relevant disciplines.

- Dr. Daniel Abramowicz
- Dr. Donald Aulenbach
- Dr. James Bonner, Texas A&M University
- Dr. Richard Bopp, Rensselaer Polytechnic Institute
- Dr. Brian Bush
- Dr. Lenore Clesceri, Rensselaer Polytechnic Institute
- Mr. Kenneth Darmer
- Mr. John Davis, New York State Dept. of Law
- Dr. Robert Dexter, EVS Consultants, Inc.
- Dr. Kevin Farley, Manhattan College
- Dr. Jay Field, National Oceanic and Atmospheric Administration
- Dr. Ken Pearsall, U.S. Geological Survey
- Dr. John Herbich, Texas A&M University
- Dr. Behrus Jahan-Parwar, SUNY - Albany
- Dr. Nancy Kim, New York State Dept. of Health
- Dr. William Nicholson, Mt. Sinai Medical Center
- Dr. George Putman, SUNY - Albany
- Dr. G-Yull Rhee, New York State Dept. of Health
- Dr. Francis Reilly, The Reilly Group
- Ms. Anne Secord, U.S. Fish and Wildlife Service
- Dr. Ronald Sloan, New York State Dept. of Environmental Conservation

TABLE 1
DISTRIBUTION OF HHRA (cont.)

STEERING COMMITTEE MEMBERS

- USEPA Community Relations Coordinator (Chair)
- Governmental Liaison Group Chair and two Co-chairs
- Citizen Liaison Group Chair and two Co-chairs
- Agricultural Liaison Group Chair and two Co-chairs
- Environmental Liaison Group Chair and two Co-chairs
- USEPA Project Managers
- NYSDEC Technical representative
- NYSDEC Community Affairs representative

FEDERAL AND STATE REPRESENTATIVES

Copies of the HHRA were sent to relevant federal and state representatives who have been involved with this project. These include, in part, the following:

- The Hon. Daniel P. Moynihan
- The Hon. Charles Schumer
- The Hon. John Sweeney
- The Hon. Nita Lowey
- The Hon. Maurice Hinchey
- The Hon. Ronald B. Stafford
- The Hon. Michael McNulty
- The Hon. Sue Kelly
- The Hon. Benjamin Gilman
- The Hon. Richard Brodsky
- The Hon. Bobby D=Andrea

16 INFORMATION REPOSITORIES (*see* Table 2).

TABLE 2
INFORMATION REPOSITORIES

Adriance Memorial Library
93 Market Street
Poughkeepsie, NY 12601

Catskill Public Library
1 Franklin Street
Catskill, NY 12414

^ Cornell Cooperative Extension
Sea Grant Office
74 John Street
Kingston, NY 12401

Crandall Library
City Park
Glens Falls, NY 12801

County Clerk's Office
Washington County Office Building
Upper Broadway
Fort Edward, NY 12828

* ^ Marist College Library
Marist College
290 North Road
Poughkeepsie, NY 12601

* New York State Library
CEC Empire State Plaza
Albany, NY 12230

New York State Department
of Environmental Conservation
Division of Hazardous Waste Remediation
50 Wolf Road, Room 212
Albany, NY 12233

* ^ R. G. Folsom Library
Rensselaer Polytechnic Institute
Troy, NY 12180-3590

Saratoga County EMC
50 West High Street
Ballston Spa, NY 12020

* Saratoga Springs Public Library
49 Henry Street
Saratoga Springs, NY 12866

* ^ SUNY at Albany Library
1400 Washington Avenue
Albany, NY 12222

* ^ Sojourner Truth Library
SUNY at New Paltz
New Paltz, NY 12561

Troy Public Library
100 Second Street
Troy, NY 12180

United States Environmental Protection Agency
290 Broadway
New York, NY 10007

White Plains Public Library
100 Martine Avenue
White Plains, NY 12601

* ***Repositories with Database Report CD-ROM (as of 10/98)***

^ ***Repositories without Project Documents Binder (as of 10/98)***

3. Organization of HHRA Comments and Responsiveness Summary

3.1 Identification of Comments

Each submission commenting on the HHRA was assigned the letter "H" for HHRA and one the following letter codes:

F - Federal agencies and officials;
S - State agencies and officials;
L - Local agencies and officials;
P - Public Interest Groups and Individuals; and,
G - General Electric Company.

The letter codes were assigned for the convenience of readers and to assist in the organization of this document. Priority or special treatment was neither intended nor given in the responses to comments.

Once a letter code was assigned, each submission was then assigned a number, in the order that it was received and processed, such as HP-1. Each different comment within a submission was assigned a separate sub-number. Thus, if a federal agency submitted three different comments, they are designated HF-1.1, HF-1.2, HF-1.3. Comment letters have been reprinted following the fourth tab of this document.

The alphanumeric code associated with each reprinted written submission is marked at the top right corner of the first page of the comment letter. The sub-numbers designating individual comments are marked in the margin. Comment submissions are reprinted in numerical order by letter code in the following order: F, S, L, P, and G.

3.2 Location of Responses to Comments

The Comment Directory, following this text, contains a complete listing of all commentors and comments.

- The first column lists the names of commentors. Comments are grouped in the following order: HF (Federal), HS (State), HL (Local), HP (Public Interest Groups and Individuals) or HG (General Electric).
- The second column identifies the alphanumeric comment code, *e.g.*, HF-1.1, assigned to each comment.
- The third column identifies the location of the response by the HHRA section number. For example, comments raised on Section 3.2 of the HHRA can be found in the corresponding Section 3.2 of the Responses section, following the third tab of this document.
- The fourth, fifth, and sixth columns list key words that describe the subject matter of each comment. Readers will find these key words helpful as a means to identify subjects of interest and related comments.

Responses are grouped and consolidated by section number of the HHRA in order that all responses to related comments appear together for the convenience of the reader interested in responses to related or similar comments.

4. Comment Directory

This section contains a diagram illustrating how to find responses to comments. As stated in the Introduction, this document does not reproduce the HHRA. Readers are urged to utilize this Responsiveness Summary in conjunction with the HHRA.

4.1 Guide To Comment Directory

Step 1	Step 2	Step 3
Find the commentor or the key words of interest in the Comment Directory.	Obtain the alphanumeric comment codes and the corresponding HHRA Section.	Find the responses following the Responses tab. Use the Table of Contents to locate the page of the Responsiveness Summary for the HHRA Section.
Key to Comment Codes:		
Comment codes are in this format HX-a.b H=HHRA X=Commentor Group (F=Federal, S=State, L=Local, P= Public Interest Groups and Individuals, G=General Electric Company) a=Numbered letter containing comments b=Numbered comment		

Example:

COMMENT RESPONSE ASSIGNMENT FOR THE HHRA

AGENCY/ NAME	COMMENT CODE	REPORT SECTION	KEY WORDS		
			1	2	3
NOAA /Rosman	**HF-1.7**	2.1.3	Milk	Dairy farms	

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Comment Directory

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4.2 Comment Directory for the Upper Hudson HHRA

AGENCY/NAME	COMMENT CODE	REPORT SECTION	KEYWORDS 1	2	3
NOAA/Rosman	HF-1.1	1.2	Risk assessment	Lower Hudson	
NOAA/Rosman	HF-1.2	2.3	Baseline modeling	Uncertainty	Incorporation
NOAA/Rosman	HF-1.3	2.3	Other PCB sources	Upstream boundary condition	High flow events
NOAA/Rosman	HF-1.4	4.3	TEQs	Data quality	Dioxin-like PCBs
NOAA/Rosman	HF-1.5	2.1.3	Floodplain contamination	Homegrown crops	Local produce and meat
NOAA/Rosman	HF-1.6	1.2	Fish advisories	Hudson River	NYSDOH
NOAA/Rosman	HF-1.7	2.1.3	Milk	Dairy farms	
NOAA/Rosman	HF-1.8	2.1.3	Plant uptake	Floodplain soils	Fruits and vegetables
NOAA/Rosman	HF-1.9	2.1.3	Snapping turtles	PCB concentrations	Advisory
NOAA/Rosman	HF-1.10	2.3.3	Water concentrations	Nearshore areas	Mid-channel areas
NOAA/Rosman	HF-1.11	2.3.4	Air	PCB sources	
NOAA/Rosman	HF-1.12	3.2.4	Start date	1999	Exposure assessment
NOAA/Rosman	HF-1.13	4.3	TEQs	Data quality	Dioxin-like PCBs
NOAA/Rosman	HF-1.14	2.3	High flow events	Remobilization	
NOAA/Rosman	HF-1.15	2.3.1	Baseline modeling	2069	Extrapolation
NOAA/Rosman	HF-1.16	2.3.4	Volatilization	Sediments	
NOAA/Rosman	HF-1.17	4.3	TEQs	Data quality	Dioxin-like PCBs
NYSDEC/Ports	HS-1.1	2.1.3	Exposure Pathways	Homegrown crops	Local produce/meat
NYSDEC/Ports	HS-1.2	General	Rogers Island	Risk assessment	Comparison
NYSDEC/Ports	HS-1.3	3.2.4	Lifetime exposure duration	Point estimates	High-end
NYSDEC/Ports	HS-1.4	3.2.4	Past exposures	Risk assessment	
NYSDEC/Ports	HS-1.5	3.2.1	Sub-populations	Fish ingestion distribution	Monte Carlo analysis
NYSDEC/Ports	HS-1.6	5.2.2	Monte Carlo analysis	Sensitivity analysis	Nature of distributions
NYSDEC/Ports	HS-1.7	5.1.2	NCP	Acceptable risk range	Risk Management
NYSDEC/Ports	HS-1.8	2.3.1	Species fractions	Brown Bullhead	Perch
NYSDEC/Ports	HS-1.9	2.1.2	Children	High-end	Point estimates
NYSDEC/Ports	HS-1.10	5	FDA tolerance level	Fish concentrations	Comparison
NYSDEC/Ports	HS-1.11	3.2.4	Lifetime exposure duration	Residence duration	Non-angling fish consumers
NYSDEC/Ports	HS-1.12	3.3.1	Assumptions	Monte Carlo analysis	Sensitivity analysis
NYSDEC/Ports	HS-1.13	2.3.1	Baseline modeling	2069	Extrapolation
NYSDEC/Ports	HS-1.14	4.2	Toxicity values	Selection	Cancer slope factors
NYSDEC/Ports	HS-1.15	4.1	Reference concentration	Air exposures	Route-to-route extrapolation
NYSDEC/Ports	HS-1.16	5.3.2	Uncertainty	Animal-human extrapolation	Non-cancer effects
NYSDEC/Ports	HS-1.17	4.1	Summary information	Toxicity values	Non-cancer effects
NYSDEC/Ports	HS-1.18	Appendix C	Toxicity profile	Out of date	New information
SCEMC/Hodgson	HL-1.1	3.2.1	Fish ingestion rate	Consumption ban	Conservatism
SCEMC/Hodgson	HL-1.2	Appendix C	Kimbrough study	Critique	Epidemiology studies
SCEMC/Hodgson	HL-1.3	2.3	95% Confidence Limit	PCB concentration	
SCEMC/Hodgson	HL-1.4	2.3	Baseline modeling	Uncertainty	Incorporation
SCEMC/Hodgson	HL-1.5	2.3.1	Baseline modeling	2069	Extrapolation
SCEMC/Hodgson	HL-1.6	2.3.1	Fishing locations	Limited	NYSDOH 1996 study
SCEMC/Hodgson	HL-1.7	2.4	Ingestion	Body burden	PCBs
SCEMC/Hodgson	HL-1.8	3.2.1	Connelly survey	Upper Hudson	Incomplete
SCEMC/Hodgson	HL-1.9	3.2.3	Cooking loss	Pan drippings	High-end
SCEMC/Hodgson	HL-1.10	3.2.1	Diary study	12-month recall	Comparison
SCEMC/Hodgson	HL-1.11	3.2.1	1993 Connelly Surveys	Great Lakes	
SCEMC/Hodgson	HL-1.12	3.2.1	Hudson angler surveys	Inconsistencies	Combined results

SCEMC/Hodgson	HL-1.13	3.2.1	Mid Hudson survey	Fish ingestion rate	Awareness of advisories
SCEMC/Hodgson	HL-1.14	3.2.1	Consumption ban	Kimbrough study	Conservatism
SCEMC/Hodgson	HL-1.15	3.2.1	Hudson angler surveys	Fish ingestion rate	Upper Hudson
SCEMC/Hodgson	HL-1.16	3.2.1	Children	Fish ingestion rate	High-end
SCEMC/Hodgson	HL-1.17	3.2.4	Fishing population	Preferences for leisure time	Steady state
SCEMC/Hodgson	HL-1.18	3.2.4	Exposure duration	Tables 3-6 and 3-7	
SCEMC/Hodgson	HL-1.19	3.2.4	Limited survey size	Validity of risk estimates	Conservatisms
SCEMC/Hodgson	HL-1.20	3.2.4	Upper Hudson counties	Steady state	Exposure duration
SCEMC/Hodgson	HL-1.21	3.2.4	Exposure duration	Limited survey data	Lower upper bound
SCEMC/Hodgson	HL-1.22	3.2.4	Meaning unclear	Fishing duration	Residence duration
SCEMC/Hodgson	HL-1.23	4.1	Uncertainty factors	Multiplied	PCBs
SCEMC/Hodgson	HL-1.24	4.1	Uncertainty	Toxicity values	Monte Carlo analysis
SCEMC/Hodgson	HL-1.25	4.2	Upper bound CSF	Monte Carlo analysis	Conservatism
SCEMC/Hodgson	HL-1.26	5.3.1	Start date	1999	Conservatism
SCEMC/Hodgson	HL-1.27	5.3.1	Exposure duration	Uncertainty	Underestimates
SCEMC/Hodgson	HL-1.28	5.3.3	Maine angler study	Fish ingestion rate	Cumulative effects
SCEMC/Hodgson	HL-1.29	4	ATSDR	Toxicological profile	Draft
SCEMC/Hodgson	HL-1.30	Appendix C	Kimbrough study	Epidemiology studies	Deficiencies
SCEMC/Hodgson	HL-1.31	Appendix C	Kimbrough study	Critique	Deficiencies
SCEMC/Hodgson	HL-1.32	Appendix C	Cancer slope factor	Upper bound	Conservatism
SCEMC/Hodgson	HL-1.33	Appendix C	Brunner	Norback and Weltman	Discussion
SCEMC/Hodgson	HL-1.34	Appendix C	Patandin and Lanting	Dioxins	Breast cancer
SCEMC/Hodgson	HL-1.35	4.1	Uncertainty factor	Multiplication	Conservatism
SCEMC/Hodgson	HL-1.36	5.1.2	PCBs in drinking water	Acceptable risk level	10^{4}
SCEMC/Hodgson	HL-1.37	General	Compounded conservatism	Realistic estimates	Risk assessment
Aulenbach	HP-1.1	General	Qualifying words	Inconclusive	Misleading
Aulenbach	HP-1.2	General	Grammer	Singular subject	Singular verb
Aulenbach	HP-1.3	3.2.1	Area above Hudson Falls	Size of angler population	Fishing ban
Aulenbach	HP-1.4	5	Comparative risks	Lifetime	
Aulenbach	HP-1.5	2.3.1	Sentence	Confusing	
Aulenbach	HP-1.6	2.4	Cumulative exposure	Body burden	Ingestion
Aulenbach	HP-1.7	3.2.1	Fish caught elsewhere	Nearby lakes	Saratoga Lake
Aulenbach	HP-1.8	3.2.3	Cooking loss	Midpoint	Bias
Aulenbach	HP-1.9	2.4.3	Dermal absorption	Fraction	Rate
Aulenbach	HP-1.10	3.1	2-D analysis	Monte Carlo analysis	Insufficient information
Aulenbach	HP-1.11	3.2.1	Shared fish	Household members	Bias
Aulenbach	HP-1.12	3.2.1	Non-fish-consumers	Inconsistency	
Aulenbach	HP-1.13	3.2.1	Connelly survey	1,000 meals/year	Cancer
Aulenbach	HP-1.14	3.2.4	Exposure duration	Start date	Age started fishing
Aulenbach	HP-1.15	4	Toxicity assessment	Kimbrough study	Critique
Aulenbach	HP-1.16	5.1.2	Population risk	Cancer cases	Insignificant
Aulenbach	HP-1.17	5	Conclusions	No public health hazard	Fish consumption
Bush	HP-2.1	4.1	Neurological effects	2,2-dichlorobiphenyl	Analytical methods
Bush	HP-2.2	2.3.4	Air	PCB concentration	Analytical methods
Bush	HP-2.3	General	Support	Risk Assessment	
Scenic Hudson	HP-3.1	2.1.2	Non-angling fish consumers	Women	Children
Scenic Hudson	HP-3.2	1.2	Risk assessment	Lower Hudson	
Scenic Hudson	HP-3.3	5.1	Multiple pathways	Fish ingestion	Total risk
Scenic Hudson	HP-3.4	2.3.4	Inhalation	Risk assessment	Food chain
Scenic Hudson	HP-3.5	Appendix C	Toxicity profile	Out of date	New information
Scenic Hudson	HP-3.6	Appendix C	Rothman	Moysich	Kimbrough study
Scenic Hudson	HP-3.7	Appendix C	Developmental toxicity	Patandin and Lanting	Unpublished
Scenic Hudson	HP-3.8	4.3	Dioxin-like PCBs	Non-cancer risks	Toxicity values

Scenic Hudson	HP-3.9	4.4	Endocrine disruptors	NRC report	Precautionary approach
CLH/Pittignano	HP-4.1	General	Single Chemical	Screening analysis	
CLH/Pittignano	HP-4.2	2.3	Historical sources	Ongoing sources	Fingerprinting analysis
CLH/Pittignano	HP-4.3	General	Probabilistic analysis	Determinisitic analysis	Emphasis
CLH/Pittignano	HP-4.4	4.3	Uncertainty	Dioxin CSF	Re-interpretations
CLH/Pittignano	HP-4.5	3.2.1	Fish consumption	Site specific data	Fish advisories
CLH/Pittignano	HP-4.6	3.2.1	Fish consumption rate	Connelly survey	Limitations
CLH/Pittignano	HP-4.7	2.1.3	Water pathways	Elimination	Drinking water standards
CLH/Pittignano	HP-4.8	2.3	Ongoing sources	Atmospheric deposition	Storm sewer outfall
CLH/Pittignano	HP-4.9	Appendix C	Epidemiology studies	Kimbrough study	Weight of evidence
CLH/Pittignano	HP-4.10	2.2	Upper bound estimate	Fish ingestion rate	Exposure parameters
CLH/Pittignano	HP-4.11	5	Risk characterization	Perspective	Regulatory actions
GE	HG-1.1	5	Present conditions	No unacceptable risk	Risk communication
GE	HG-1.2	General	Implausible assumptions	Fish ingestion rates	Credibility
GE	HG-1.3	1.2	Risk assessment	Lower Hudson	
GE	HG-1.4	Appendix C	Kimbrough study	Critique	No evidence of human effects
GE	HG-1.5	5	Present conditions	No unacceptable risk	Risk communication
GE	HG-1.6	Appendix C	Kimbrough study	Critique	No evidence of human effects
GE	HG-1.7	General	Implausible assumptions	Fish ingestion rates	Credibility
GE	HG-1.8	General	Flaws	Basis	Decision-making
GE	HG-1.9	5	Present conditions	No unacceptable risk	Risk communication
GE	HG-1.10	General	Implausible assumptions	Fish ingestion rates	Credibility
GE	HG-1.11	4	Epidemiology studies	Weight of evidence	No evidence of human effects
GE	HG-1.12	2.3	Baseline modeling	Preliminary	Flaws
GE	HG-1.13	3.2.1	Fish ingestion rates	Connelly survey	Plausibility
GE	HG-1.14	General	Angler population	Mobility rates	Cooking losses
GE	HG-1.15	4	Kimbrough study	Critique	
GE	HG-1.16	3	Monte Carlo analysis	Annual events	Single events
GE	HG-1.17	3.2.4	Non-cancer risks	Exposure duration	
GE	HG-1.18	3	Probabilistic analysis	Description	Inadequate
GE	HG-1.19	3	Monte Carlo analysis	USEPA guidance	Deficiencies
GE	HG-1.20	3.1	Separation	Uncertainty	Variability
GE	HG-1.21	3.3.1	Sources of uncertainty	Relative importance	Arbitrary
GE	HG-1.22	5	Future risks	Limited	Natural recovery
GE	HG-1.23	4	Epidemiology studies	Weight of evidence	No evidence of human effects
GE	HG-1.24	Appendix C	Kimbrough study	Critique	
GE	HG-1.25	4.1	Monte Carlo analysis	RfD values	Uncertainty
GE	HG-1.26	4.1	Monte Carlo analysis	Toxicity values	Uncertainty
GE	HG-1.27	4.3	Dioxin-like PCBs	Risk assessment	Flaws
GE	HG-1.28	3.2.1	Connelly survey	Fish ingestion rates	Limitations
GE	HG-1.29	3.2.1	Fish ingestion rates	Year-to-year variability	
GE	HG-1.30	2.1.2	Angler population	Infrequent anglers	Inconsistencies
GE	HG-1.31	3.2.4	Exposure duration	Equation	Error
GE	HG-1.32	3.2.3	Cooking loss	Cooking methods	Probability distribution
GE	HG-1.33	2.3.1	Species preferences	Connelly survey	Species groupings
GE	HG-1.34	2.3	Baseline modeling	Preliminary	Flaws
GE	HG-1.35	2.3.1	PCB concentrations	Baseline modeling	Inconsistencies
GE	HG-1.36	3	Monte Carlo analysis	USEPA guidance	Deficiencies
GE	HG-1.37	3	Monte Carlo analysis	Annual events	Single events
GE	HG-1.38	3	Probabilistic analysis	Description	Inadequate
GE	HG-1.39	3	Fish ingestion rates	Cooking methods	Fish species
GE	HG-1.40	3.2.4	Non-cancer risks	Exposure duration	
GE	HG-1.41	3.1	Separation	Uncertainty	Variability
GE	HG-1.42	3.1	Separation	Uncertainty	Variability

GE	HG-1.43	3.3.1	Sources of uncertainty	Relative importance	Arbitrary
GE	HG-1.44	3.1	Separation	Uncertainty	Variability
GE	HG-1.45	5	Present conditions	No unacceptable risk	Risk communication
GE	HG-1.46	Appendix C	Kimbrough study	Critique	
GE	HG-1.47	4	Epidemiology studies	Weight of evidence	No evidence of human effects
GE	HG-1.48	2.3	Baseline modeling	Preliminary	Flaws
GE	HG-1.49	General	Implausible assumptions	Fish ingestion rates	Credibility
GE	HG-1.50	General	Angler population	Mobility rates	Cooking losses
GE	HG-1.51	General	Revisions	Reissue report	
GE	HG-1.52	4	Epidemiology studies	Weight of evidence	No evidence of human effects
GE	HG-1.53	3.2.1	Fish ingestion rates	Connelly survey	Plausibility
GE	HG-1.54	3	Monte Carlo analysis	USEPA guidance	Deficiencies

II. RESPONSES TO COMMENTS ON THE HUMAN HEALTH RISK ASSESSMENT FOR THE UPPER HUDSON RIVER (HHRA)

Responses to General Comments

Response to HL-1.37, HG-1.2, HG-1.7, HG-1.8, HG-1.10, HG-1.14 HG-1.49, HG-1.50, and HG-1.51

Consistent with the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) (USEPA 1990a) and USEPA policy and guidance (USEPA, 1989, 1990b, 1992a, 1992b, 1995, 1996a, and 1997a), the exposure parameters used in the HHRA are appropriately protective of human health and do not reflect a worst-case exposure scenario. Specifically, USEPA evaluated both high-end (Reasonable Maximum Exposure or RME) and central tendency exposure (CTE or average) cancer risks and non-cancer hazards in the HHRA. As stated in the HHRA (*e.g.*, pp. ES-2 and 2), the RME is the maximum exposure that is reasonably expected to occur in the Upper Hudson River under baseline conditions (*e.g.*, no institutional controls or remediation, such as the fish consumption advisories currently in place). The RME is reasonable because it is a product of factors, such as concentrations (*e.g.*, fish, sediment, and surface water) and exposure frequency and duration, that are an appropriate mix of values that reflect averages and high-end distributions (USEPA, 1989, 1990b).

The fish ingestion rates and exposure durations for the Upper Hudson River were derived from the 1991 New York Angler study (Connelly *et al.*, 1992) and population mobility data from the U.S. Census Bureau for the five counties surrounding the Upper Hudson River (see, p. ES-2). The fraction from source was assumed to be 1 (*i.e.*, 100%) (see, p. 22), which is reasonable given the 40-mile extent of the Upper Hudson River and the variety of fish species it can support. The concentrations of PCBs in fish beginning in 1999 were based on observed fish data from 1993 NYSDEC/NOAA and modeled fish concentrations forecast in the Baseline Modeling Report (USEPA, 1999d). The toxicity values used were taken from USEPA's Integrated Risk Information System or IRIS, USEPA's consensus database of toxicity values, which considers both toxicological studies in animals and human epidemiological studies in determining appropriate toxicity values for use in risk assessments throughout the Agency (see, Appendix C and Responsiveness Summary for HHRASOW, pp. 25-26).

Exposure and risk estimates were recalculated based on the revised BMR results (USEPA, 2000a). These revised exposure and risk estimates are presented in Section 4 of this report.

Response to HP-1.1

Consistent with USEPA policy and guidance on risk characterization (USEPA, 1992b, 1995), USEPA conveyed the uncertainties associated with the calculated cancer risks and non-cancer hazards presented in the HHRA by using such words as "can," "could," "might," "probably," "perhaps," and "estimate." The use of these words are intended to convey the uncertainties and variability associated with the exposure and risk estimates. The overall conclusions of the HHRA are stated in the Executive Summary and in Chapter 5 of the HHRA.

Response to HP-1.2

The comment concerning grammar is noted.

Response to HP-2.3

USEPA acknowledges this comment in support of the HHRA.

Response to HP-4.1

Screening analyses may be used in Superfund risk assessments to focus an analysis on the chemicals most likely to have the largest contributions to cancer risks and non-cancer hazards, but they are not required in all Superfund risk assessments (USEPA, 1989, Section 5.9). As described in the HHRA (p. 10), a screening of contaminants of concern was not conducted because PCBs were previously identified as the contaminants of concern for the Reassessment RI/FS for the Hudson River PCBs site (USEPA, 1991a). By site definition and consistent with the purposes of reassessing the 1984 Interim No-Action ROD, the site-related contaminants are limited to PCBs.

Response to HP-4.3

USEPA disagrees with the comment that the results of the point estimate calculations are given far greater emphasis in the HHRA than the results of the Monte Carlo analysis. Considerable emphasis is given to the Monte Carlo analysis in the HHRA as a whole (*e.g.*, pp. 33-59, 70-71, and Appendix B) and particularly in the Executive Summary (pp. ES5-ES6). Contrary to the commentor's statement, the Major Findings section of the Executive Summary discusses both the Monte Carlo and Point Estimate Results.

The fact that the point estimate RME result for cancer risk is comparable to the 95th percentile of the base case Monte Carlo analysis does not indicate that USEPA used highly skewed input distributions, as suggested by the comment. Comparison of all of the Monte Carlo analysis results with the RME point estimates for both cancer risk and non-cancer hazards reveals that the RME point estimates for cancer risk and non-cancer hazards fall just above and at, respectively, the 75th percentile and fall as high as the 99th percentile (see, Figures 5-3a and 5-3b). Frequency distributions of the exposure factors are provided in the HHRA (see, Figures 3-3a to 3-3d, 3-4a to 3-4d, and 3-5a and 3-5b). In addition, Table 3-2 shows that the fish ingestion rates used in the HHRA are consistent with ingestion rates obtained from other angler surveys conducted using different survey methods and conducted in other waterbodies.

Response to HS-1.2

In a separate matter, in July 1999 USEPA released a Human Health Risk Assessment for Rogers Island, located in the Town of Fort Edward in the Upper Hudson River. Both the Rogers Island and the Upper Hudson River risk assessments quantify cancer risks and non-cancer hazards to human health using USEPA policy and guidance and the current toxicity values for PCBs (USEPA, 1989, 1992a, 1992b, 1996a, and 1999 a-c). However, the risk assessments quantify cancer risks and non-cancer hazards for different exposure pathways and using site-specific exposure values developed for the two different sites. For example, the Rogers Island risk assessment evaluated both residential and recreational exposure over a relatively small area, whereas the Upper Hudson River risk assessment evaluated recreational exposure only, over a 40-mile stretch of river. In cases where the risk assessments evaluated the same route of exposure (*i.e.*, dermal contact with sediments), the exposure assumptions are different to reflect the difference in activity patterns between residents and recreators based on accessibility of the river, frequency of contact, and age at time of exposure. In addition, at the time of the Rogers Island risk assessment, the USEPA Dermal Workgroup (a group which includes Region and Headquarters EPA staff) recommended a skin adherence factor of 1 mg/cm^2 for adults and children (USEPA, 1998b, based on Duff and Kissel, 1996). Subsequently, the Dermal Workgroup's

recommended skin adherence factor changed to 0.2 mg/cm^2 for children and 0.3 mg/cm^2 for adults, which was used in the Upper Hudson River risk assessment (USEPA, 1999f, based on a review and analysis of a number of recent soil adherence studies).

1. Overview of Risk Assessment

1.1 Introduction

No significant comments were received on this section.

1.2 Site Background

Response to HF-1.1, HP-3.2

USEPA has previously responded to public comment regarding its decision to quantify cancer risks and non-cancer hazards to individuals for the Upper and Mid-Hudson River, but not to individuals in the Lower Hudson River between Poughkeepsie, New York and the Battery in New York City, (USEPA, 1999g, Responsiveness Summary for the Human Health Risk Assessment Scope of Work, p. 14). USEPA's approach to assess cancer risks and non-cancer hazards only in the Upper and Mid-Hudson River is protective of human health (*e.g.*, will not underestimate RME cancer risks and non-cancer hazards) because site-related risks to individuals closer to the sources of PCBs (*i.e.*, in the Upper Hudson River) are expected to be higher than the cancer risks and non-cancer hazards to individuals farther away from the sources (*i.e.*, south of Poughkeepsie), based on the higher concentrations of PCBs found in fish and sediments in the Upper Hudson River compared to those in the Lower Hudson River.

Response to HG-1.3

USEPA addressed this comment regarding the extent of the site in the Responsiveness Summary for the Human Health Risk Assessment Scope of Work (pp. 14-15). USEPA has consistently defined the site to include the Lower Hudson River since at least April 1984, when the Agency issued its Feasibility Study for the site and before the site was listed on the National Priorities List (codified at 40 CFR Part 300, App. B). In its September 25, 1984 Record of Decision (ROD), USEPA defines the site by reference to three figures which, together, depict the site as the entire 200-mile stretch of the River from Hudson Falls to the Battery in New York City, plus the remnant deposits. In addition, during the Reassessment RI/FS, USEPA has consistently defined the site as including the Upper and Lower River (*e.g.*, USEPA, 1990c, 1991).

1.3 General Risk Assessment Process

No significant comments were received on this section.

1.4 Discussion of 1991 Phase 1 Risk Assessment

No significant comments were received on this section.

1.5 Objectives of Phase 2 Risk Assessment

No significant comments were received on this section.

2. Exposure Assessment

2.1 Exposure Pathways

2.1.1 Potential Exposure Media

No significant comments were received on this section.

2.1.2 Potential Receptors

Response to HS-1.9

As presented in the HHRA (p. 69), the RME cancer risk for a child ingesting fish was approximated to be 3 x 10^{-4} (3 additional cancers in 10,000 people exposed), assuming that a child meal portion is approximately 1/3 that of an adult (227 grams for adults, 76 grams for children). This assumed value falls between the mean fish meal sizes reported by the USEPA for children less that five year and children aged six to eleven years old (67 grams and 89 grams, respectively) (USEPA, 1997a).

Further, separate calculations of cancer risks and non-cancer hazards using body weights and ingestion rates that are appropriate for a child (ages 1 to 7 years old) yield similar calculated cancer risks and non-cancer hazards to those for the adult. For example, assuming an average daily fish ingestion rate for children of 10.6 g/day, the high-end PCB concentration in fish (5.1 mg/kg), an exposure frequency of 365 days, an exposure duration of 7 years, and a body weight of 18 kg (USEPA, 1997a), the RME child risk would be about 6 x 10^{-4}, and the RME child hazard index would be about 150.

Response to HP-3.1

USEPA calculated cancer risks and non-cancer hazards by combining site-specific exposure parameters and the Agency-wide consensus values for PCB toxicity in IRIS. Cancer risks and non-cancer hazards for non-anglers consuming Upper Hudson River fish caught by a friend or family member were not evaluated in the HHRA because there is little or no information available to quantify non-angler fish ingestion rates (HHRA, pp. 72 and 74). Despite this lack of information, the cancer risks and non-cancer hazards for non-anglers consuming sport fish are expected to be lower than for anglers based on expected lower fish consumption rates. Women and children anglers were represented in the HHRA to the extent they are represented in the 1991 New York angler survey data (Connelly et al., 1992). The toxicity values in IRIS that were used in the HHRA are protective of sensitive populations, such as women and children (see, USEPA, 1999g, p. 18).

Response to HG-1.30

The population of concern was described in the HHRA (p. 5) as anglers "who may fish" because USEPA evaluated current and future cancer risks and non-cancer hazards from exposure to PCB-contaminated fish under baseline conditions of no remediation or institutional controls, such as the fish consumption advisories currently in place. For purposes of the HHRA, the cancer risks and non-cancer hazards were calculated for Upper Hudson River anglers who consume at least one self-caught fish meal per year (*e.g.*, pp. 37 and 72). Cancer risks and non-cancer hazards were not calculated for anglers consuming less than one fish meal per year, since risks to those anglers would be expected to be lower based on the lower fish consumption rates. The commentor's suggested approach would not be protective of human health, because including anglers who eat less than one fish meal per year into the calculation of fish ingestion rates would inappropriately reduce the mean and high-end ingestion rates, thus reducing the calculated cancer risks and non-cancer hazards.

2.1.3 Potential Exposure Routes

Response to HF-1.5, HF-1.7, HF-1.8, HS-1.1, and HP-3.4

Consistent with the focus of the Reassessment RI/FS, the HHRA calculated cancer risks and non-cancer hazards associated with exposure to PCBs in the sediments, water and fish in the Upper Hudson River. The HHRA does not quantify cancer risks and non-cancer hazards due to uptake of PCBs via floodplain soils. As discussed in the HHRA (p. 8), USEPA qualitatively assessed available data and literature regarding PCB uptake in forage crops and cow's milk, and concluded that risk via ingestion of foods other than Hudson River fish is likely to be minimal. Therefore, the collection of additional PCB data from vegetables, meat, eggs, and milk is not warranted. The information regarding the more than 18,200 samples of cow's milk analyzed for PCBs by the New York State Department of Agriculture and Markets (NYSDA&M) was obtained directly from Dr. Rudnick of NYSDA&M (Rudnick, 1999, personal communication) (see also, USEPA, 1999g, p. 15). The sample results are not contained in any computerized database, and it would be resource-intensive to determine the location and number of dairy farms along the Upper Hudson River that are represented in this state-wide database. However, a representative of the New York State Department of Health confirmed that the samples represent individual farms, not composite samples of milk from more than one farm (Montione, 2000, personal communication). USEPA is aware of two abstracts concerning the Chicago "urban plume" of PCBs (Eisenreich *et al.*, 1996 and Baker *et al.*, 1996). While they are relevant to PCBs in general, due to the different hydrodynamics between lake Michigan and the Upper Hudson River, USEPA believes they are not directly relevant to assessing human health risk to individuals exposed to PCBs in the Upper Hudson River.

Response to HF-1.9

The literature regarding PCBs concentrations in Hudson River snapping turtles by Stone *et al.* (1980) and Olaffson *et al.* (1983) were cited in the HHRA, but the PCB concentrations were not provided because the data may not be representative for purposes of estimating cancer risks and non-cancer hazards to human health due to the small number of turtles analyzed and because turtle consumption rates for individuals are unknown (HHRA, pp. 8-9). In addition, the data are more than 15 years old and concentrations of PCBs in turtles would be expected to have declined since the early 1980s, as observed in other media (*i.e.*, water, sediment, fish). Nevertheless, the data can be summarized as follows: Stone *et al.* (1980) found PCB concentrations from ten snapping turtles, collected from the Hudson River in 1976-1978, to range from 306 to 7,990 mg/kg PCBs (mean 2,991 mg/kg PCBs) in fat tissue, 0.54 to 683

mg/kg PCBs (wet weight) in liver, and 0.19 to 27.62 mg/kg PCBs (wet weight) in muscle. Olafsson *et al.* (1983) reported a PCB concentration in fat tissue of 3,608 mg/kg from one snapping turtle from the Upper Hudson River near Hudson Falls, New York.

The HHRA quantified cancer risks and non-cancer hazards under baseline conditions, which assumes no remediation or institutional controls, such as the turtle and fish consumption advisories currently in place. There is currently a state-wide consumption advisory for women of childbearing age, infants, and children under the age of 15 to avoid eating snapping turtles or soups made with their meat due to PCB contamination (NYSDOH, 1999a, p. 14).

Response to HP-4.7

Dermal contact with river water was quantitatively evaluated in the HHRA and found to present insignificant cancer risk and non-cancer hazard to the RME individual. The use of the federal Maximum Contaminant Level (MCL) for PCBs in drinking water to eliminate dermal exposure as a pathway of concern is not consistent with USEPA human health risk assessment guidance (USEPA, 1989), and therefore would not be appropriate for eliminating dermal contact with Upper Hudson River water as a pathway of concern in the HHRA.

2.2 Quantification of Exposure

Response to HP-4.10

Consistent with the NCP (USEPA, 1990a) and as stated in the HHRA (*e.g.*, pp. ES-2 and 2), the RME represents the maximum exposure that is reasonably expected to occur in the Upper Hudson River. The RME is reasonable because it is a product of factors, such as concentrations and exposure frequency and duration, that are an appropriate mix of values that reflect averages and high-end distributions (USEPA, 1989, 1990b).

In the HHRA, USEPA did not use the 90th percentile or greater for every exposure parameter to characterize the RME exposure scenario, as suggested by the comment. As shown in Tables 2-6, 2-7, 2-8, and 2-12, the exposure point concentration for the RME point estimate for fish ingestion was based on the mean (or average) concentration of PCBs, averaged over the RME exposure duration (*i.e.*, 40 years) and location (40 miles of the Upper Hudson River). The concentration of PCBs in fish used was 2.2 and 5.1 mg/kg for evaluating cancer risks and non-cancer health hazards (HHRA, pp. 23-24 and 68), respectively, not 28.7 mg/kg as indicated in the commentor's Table 1, which is the concentration in sediment (HHRA, p. 28). Mean (or average) adult body weight was used to be consistent with the assumptions used to derive the IRIS toxicity values. Upper-bound values (*i.e.*, the 90th percentile or above) were used for both the fish ingestion rate and the exposure duration to reflect RME exposure, since it is reasonable to assume that an avid angler would have both an upper-bound fish ingestion rate and an upper-bound exposure duration.

2.3 Exposure Point Concentrations

Response to HF-1.2, HL-1.4, HG-1.12, HG-1.34 and HG-1.48

As stated in the HHRA (pp. ES-1, ES-2, 11-13), USEPA used the results from the Baseline Modeling Report (USEPA, 1999d) to develop the exposure point concentrations for PCBs in fish. These exposure points concentrations and the resulted cancer risk and non-cancer health hazards were recalculated based on the revised BMR results (USEPA, 2000a). These revised exposure and risk estimates are presented in Section 4 of this report. The overall conclusions from the August 1999 HHRA (USEPA, 1999) remain unchanged for this revised HHRA.

Waiting until after the peer review for the Baseline Modeling Report to use the model output would have unnecessarily delayed issuance of the risk assessments by about one year. The risk assessments themselves will be peer-reviewed in May 2000. The results of the independent peer review will be evaluated by USEPA and the Agency will respond to significant comments raised during the peer review in a Responsiveness Summary. USEPA's approach accomplishes both the Agency's policy to use sound, credible science in its decision-making and its commitment to release a Proposed Plan identifying its preferred cleanup alternative in December 2000.

Response to HF-1.3, HF-1.14

Based on the findings of the Baseline Modeling Report, a 100-year peak flow event is not expected to have substantial impacts on the recovery rate of the Upper Hudson River because the forecast long-term, summer average concentrations of PCBs in the water column with and without the 100-year peak flow are virtually indistinguishable one year after the event (USEPA, 1999d, p. ES-5). Therefore, a high flow event is not expected to affect the overall conclusions of the HHRA.

Response to HL-1.3

The calculation of a 95% upper confidence limit on the mean (UCLM) is not appropriate for the exposure point concentrations of fish, sediment and water in the Upper Hudson River. When models are used to predict concentrations, the number of data points is determined by the extent of the modeling effort and is not constrained by a finite data set of observed values. Because the concentrations used in the HHRA are based on modeled projections of future concentrations, the model mean (or average) and model 95% UCLM converge to the same value (HHRA, p. 10).

Response to HP-4.2, and HP-4.8

From 1957 through 1975, between 209,000 and 1,300,000 pounds of PCBs were discharged to the Upper Hudson River from two General Electric Company (GE) facilities in Hudson Falls and Fort Edward, New York (HHRA at p. 1, citing USEPA, 1991). USEPA (USEPA, 1997b, 1998b) provided evidence demonstrating that the PCBs in the sediments are consistent with the historical releases of PCBs from GE's facilities (*i.e.*, the PCB congener patterns in the sediments are "fingerprints" of GE's PCBs). For the exposure point concentrations used in the HHRA, USEPA used modeling results for the total PCB concentrations assuming a constant upstream source of 10 ng/L PCBs in river water, rather than no ongoing source as stated in the comment (see HHRA, p. 15). Other potential historical or ongoing sources of PCBs to the river mentioned in the comment, such as atmospheric deposition, spills and leaks

from boats, combined sewer overflows, and non-point sources, are minor compared to the releases from the GE facilities in Hudson Falls and Fort Edward, New York.

2.3.1 PCB Concentration in Fish

Response to HF-1.15, HS-1.13, HL-1.5

In the Baseline Modeling Report (USEPA, 1999d), the models were run to forecast concentrations of PCBs in fish for 20 years from 1998 to 2018 to match the 20 years of hindcast data used to calibrate the fate and transport models. Given the reasonably good fit of the exponential trend/regression line for the fish concentrations (R^2 of 0.94 to 0.99, see Figures 2-1 to 2-9), it is unlikely that the extrapolation of fish concentrations would significantly underestimate or overestimate expected future exposures (HHRA, p. 15). Nonetheless, as part of the fine-tuning of the Baseline Modeling Report (USEPA, 1999d), USEPA has extended the modeled concentrations to 2069 to cover the full 70 years exposure duration period used in the Monte Carlo analysis to quantify cancer risk. These exposure points concentrations and the resulted cancer risk and non-cancer health hazards were recalculated based on the revised BMR results (USEPA, 2000a). These revised exposure and risk estimates are presented in Section 4 of this report. The overall conclusions from the August 1999 HHRA (USEPA, 1999) remain unchanged for this revised HHRA.

The model results used current upstream boundary conditions of 10 ng/L PCBs. The potential for a new, site-specific source of PCBs to the river that is currently unknown is unaccounted for in the modeling and is an unquantifiable source of uncertainty (see HHRA, p. 72).

Response to HS-1.8, HP-1.5, and HG-1.33

The 1991 New York Angler survey (Connelly *et al.*, 1992) reported fish consumption for six species that are potentially caught in the Upper Hudson River for consumption: bass, walleye, bullhead, carp, eel, and perch (HHRA, p. 14 and Table 3-3). In the Baseline Modeling Report (1999d), USEPA forecast concentrations of PCBs in three species: brown bullhead, largemouth bass, and yellow perch. Forecast concentrations were not available for walleye, carp and eel. To estimate concentrations of PCBs in carp and eel, USEPA used the forecast concentrations of PCBs in the brown bullhead, which is similar to carp and eel based on similar bottom-feeding and bottom-dwelling characteristics over their respective life-cycles. These fish constitute Group 1. For the walleye, USEPA used the forecast concentrations of PCBs in the bass, which is similar to bass based on the large size and pisciverous diet of these fish. These fish are Group 2. Group 3 consists only of the perch, for which the forecast concentrations of PCBs in the yellow perch were used (HHRA, p. 14).

USEPA modeled future concentrations of PCBs in white perch, but no measured data from the Upper Hudson were available to validate/calibrate the model (USEPA, 1999d). Although white perch migrate to the lower two lock pools of the Upper Hudson River to spawn, they are typically found in the Lower Hudson River, not the Upper Hudson River for the remainder of the year (USEPA, 1999i, Appendix D). Foe these reasons, information from angler surveys that white perch were caught in the Upper Hudson River maybe due to misidentification of the fish (see, p. 39).

USEPA noted that there is some uncertainty in the exposure point PCB concentrations in fish used in the HHRA (p. 72). This uncertainty is unavoidable because the angler surveys in the Upper Hudson River (Barclay, 1993 and NYSDOH, 1999b, see pp. 39-40) could not be used to quantify fish

consumption by species due to the fish consumption advisories. The adjustments made to the 1991 New York Angler survey (Connelly *et al.*, 1992) data, such as excluding the "other" category, which may include fish species found in the Upper Hudson, and excluding fish species not found in the Upper Hudson, as well as extrapolating the percent of all fish in flowing water bodies to percent of Hudson species (Table 3-3) were necessary so that the fish species percentages for the Upper Hudson totaled 100%. While there is some uncertainty associated with grouping three fish species that were not modeled to estimate their PCB concentrations (*i.e.*, walleye, carp, and eel), these fish represent 9%, 6%, and 2% of the total fish intake, respectively (see Table 3-4). USEPA used modeled fish concentrations for the remaining 83% of the fish intake.

The uncertainty in the exposure point PCB concentrations in fish cannot be quantified based on available information. If concentrations of PCBs in carp and eel are generally higher than were assumed based on similarity to the brown bullhead (all Group 1), then the cancer risks and non-cancer hazards from the intake of these species (carp, 6% and eel, 2%, see Table 3-4) would be higher. However, based on the relatively low intake percentages reported for carp and eel (see Table 3-3), USEPA would not expect the total cancer risks and non-cancer hazards from ingesting fish to be substantially greater than those calculated in the HHRA. Conversely, if concentrations in fish not modeled are lower on average than fish actually consumed, the risk would be lower.

With respect to the statement in the HHRA (p. 14) that bass and walleye reach several feet in length, the National Audubon Society Field Guide to North American Fishes, Whales, and Dolphins (1997) indicates lengths of up to three feet two inches for largemouth bass and three feet five inches for walleye. USEPA agrees that this is an upperbound for the fish length, not an average or typical length.

Response to HL-1.6

Information on the 1996 and 1991-1992 Hudson Angler Surveys (NYSDOH, 1999b; Barclay, 1993) is presented in the HHRA at pp. 39-40.

Response to HG-1.35

The drop in PCB concentrations in largemouth bass from Stillwater in 1999 is observed in the projections in the HHRA. The figures in the HHRA and Baseline Modeling Report simply have different scales, so the drop is not as obvious in Figure 2-5 of the HHRA.

2.3.2 PCB Concentration in Sediment

No significant comments were received on this section.

2.3.3 PCB Concentration in River Water

Response to HF-1.10

The model has 47 river segments along the river in a north-south direction and three segments across the river (HHRA, p. 16). Little detailed information on near-shore *versus* mid-channel water concentrations is available; however, water samples were analyzed from west, center, and east locations at each river mile (RM) between RM 188.5 and 194.1. These samples offer some perspective on the variability of water concentrations across the river. The east and west samples averaged 19.8 ng/L, while

the average of the center samples is 21.1 ng/L, only slightly higher. Thus, the averaging approach for PCB concentrations in river water used in the HHRA is unlikely to have resulted in a significant underestimate of cancer risks and non-cancer hazards to recreators.

2.3.4 PCB Concentration in Air

Response to HF-1.11, HF-1.16

The cancer risks due to inhalation of PCBs in air were evaluated based on historical measurements of PCBs in air, as well as modeled concentrations of PCBs volatilized from river water into air (HHRA, pp. 16-21 and Appendix A). PCB-contaminated sediment and floodplain soil also potentially contribute to PCBs in air. USEPA did not quantify the contribution of PCBs in air from contaminated sediment and floodplain soil because a) the contribution is expected to be minor compared to the concentrations of PCBs in air that were used in the HHRA, which were obtained during periods of high activity (*i.e.*, Remnant Deposit remediation); b) the calculated cancer risks from inhalation of volatilized PCBs were *de minimus* (*i.e.*, insignificant); and c) consistent with the scope of the Reassessment RI/FS, the HHRA addresses the cancer risks and non-cancer hazards from PCBs in Upper Hudson River water and sediments, not floodplain soils. The uncertainty associated with concentrations of PCBs in air from all sources, which could include river sediments periodically exposed to air, is acknowledged in the HHRA (p. 75).

Response to HP-2.2

The mass transfer coefficient used to estimate PCB losses from the water column to the air was based upon tri-and tetra-chlorobiphenyls, as stated in the comment. This coefficient was applied to total PCBs in the water column, such that the lower chlorinated homologues in the water column were incorporated into the estimated exposure point concentration in air used in the cancer risk calculation. Use of a mass transfer coefficient based on tri- and tetra-chlorinated homologues may underpredict the release of lower chlorinated homologues to the air, which would reduce the estimates of ambient airborne PCB concentrations. However, given that the mass transfer was applied to total PCBs in the water column, the underprediction is likely to be considerably less than an order of magnitude, and would not significantly impact the calculated cancer risks presented for this pathway. Specifically, the gas transfer coefficient is estimated to be no more that 10 percent higher for the mono and di fraction relative to the tri+ PCBs fraction. Gas exchange flux for PCBs in the Upper Hudson is examined in Appendix C of USEPA (2000b, in preparation). From this analysis the di to tetra homologue ratio of the gas transfer coefficients is 1.1, as represented by BZ #4 to BZ #52.

2.4 Chemical Intake Algorithms

Response to HL-1.7, HP-1.6

The HHRA calculated cancer risks and non-cancer hazards by estimating a lifetime and average daily dose of PCBs taken into the body, averaged over the appropriate exposure duration (HHRA, p. 9). Metabolism of PCBs in humans, with an emphasis on the mode of action in the liver, is discussed in PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures (USEPA, 1996b). Consistent with USEPA guidance and policy (USEPA 1992a, 1992b, 1996a, 1996b, 1999j), the average daily dose of PCBs to humans is not lowered to account for excretion or metabolism because PCBs,

especially the more toxic forms, are known to bioaccumulate in the human body. Not lowering the average daily dose of PCBs to account for their elimination by the human body of PCBs is appropriately protective of humans. USEPA also notes that, consistent with the recommendations of the external peer review panel (USEPA, 1996b, p. 11), USEPA did not increase the average daily dose in the HHRA to account for existing body burdens of PCBs (see also, USEPA, 1999g, p. 28).

2.4.1 Ingestion of Fish

No significant comments were received on this section.

2.4.2 Ingestion of Sediment

No significant comments were received on this section.

2.4.3 Dermal Contact with Sediment

Response to HP-1.9

By convention, the dermal absorption fraction is unitless (USEPA, 1989, pg. 6-41) because it assumes exposure times that are the same as in the experimental study upon which it is based. The fraction used for PCBs is based on Wester *et al.* (1993), in which the exposure time per event was 24 hours.

2.4.4 Dermal Contact with River Water

No significant comments were received on this section.

2.4.5 Inhalation of PCBs in Air

No significant comments were received on this section.

3. Monte Carlo Analysis (MCA) of Exposure of Fish Ingestion Pathway

Response to HG-1.16, HG-1.18, HG-1.19, HG-1.29, HG-1.36, HG-1.37, HG-1.38, HG-1.39, and HG-1.54

USEPA disagrees with the comment that the HHRA does not comply with USEPA's policy and guidance on Monte Carlo analysis (USEPA, 1997c). In the HHRA, USEPA provided extensive documentation on the methods and results of the Monte Carlo analysis, which is consistent with good scientific practices and is adequate for critical review. On September 2, 1999, USEPA provided supplemental information, such as the computer modeling code, that was requested by General Electric Company on August 31, 1999. While this supplemental information is needed to reproduce the Monte Carlo analysis, it is not required to conduct a meaningful review of the HHRA.

USEPA acknowledges the comment that the Monte Carlo modeling approach outlined in the HHRA is generally sound. However, USEPA disagrees that the Monte Carlo analysis models angler doses as a single block of time varying from one to >30 years. Rather, the doses are annual and summed over the exposure duration (see HHRA, p. 36, Equation 3-1 and Figure 3-1). Furthermore, USEPA disagrees that modeling angler's doses for the exposure durations derived from the 1991 New York Angler survey data (Connelly *et al.*, 1992), as shown by the distribution (*i.e.*, from one year to 70 years), is a limitation. Rather, this is a strength of Monte Carlo analysis, in that the variability of the fish consumption patterns within the population (*i.e.*, some individuals eat fish for a short time and others over a lifetime, and some individuals eat more fish while others eat less) can be quantitatively evaluated. The commentor's approach to model doses as separate, random events, such as the microexposure event analysis described by ChemRisk (1995) and Price *et al.* (1996), is not protective of human health because it would assume that fish ingestion rates are not highly correlatable, which could systematically "average-out" a high-end fish consumer (*i.e.*, a high-end fish consumer for one meal could be a low-end or average fish consumer for the next meal). USEPA considers it more reasonable that a person consuming fish at the high-end for one fish meal would generally consume fish at the high end for subsequent fish meals, and cook fish the same way year after year rather than randomly (see HHRA, p. 74). The approach described in the comment could therefore underestimate the cancer risks and non-cancer hazards to the high-end individual and may not be protective of human health for the high-end or reasonably maximally exposed individual.

In the Monte Carlo analysis, USEPA did not assume fish were harvested from the same locations. USEPA evaluated several scenarios, one of which included catching fish over the entire Upper Hudson River study area (HHRA, p. 59). Moreover, the Monte Carlo analysis did not assume the same mixture of fish species. The fish species consumption fractions were selected randomly from a distribution derived based on the 1991 New York Angler survey (Connelly *et al.*, 1992) responses (HHRA, p. 48). Furthermore, the statement that the Monte Carlo analysis did not evaluate year to year variations in PCB intake via fish ingestion is incorrect. The forecast PCB concentrations (which were used in the Monte Carlo analysis) are not constant from one year to the next. Fish ingestion rates were correlated from year to year, but varied as a function of age of the angler. Both fish species consumption patterns and cooking loss varied randomly from year to year.

In the Monte Carlo simulations, some of the anglers had an exposure duration greater than 40 years because some of the respondents in the 1991 New York Angler survey (Connelly et al, 1992) indicated fishing for extended periods of their lives (*i.e.*, fishing duration) and some people live along the Upper Hudson River for that long, as shown by US Census Bureau data (*i.e.*, residence duration) (HHRA, pp. 49-57). In the Monte Carlo analysis, USEPA modeled anglers with exposure durations throughout the distribution of plausible values, including low, middle and high-ends of the distribution.

Consistent with USEPA guidance (USEPA 1989a), non-cancer health hazards are assessed using chronic doses (7 years or more), which are compared to the appropriate chronic Reference Dose (RfD). Because the forecast concentration of PCBs in fish declines with time, the average concentration (and by extension, average PCB intake in terms of mg/kg-day) in a 7-year exposure period is greater than the average concentration over a longer period of time (HHRA, p. 23).

3.1 Discussion of Variability and Uncertainty

Response to HG-1.20, HG-1.41, HG-1.42, HG-1.44 and HP-1.10 [also some of Attachment C]

The difference between variability and uncertainty is discussed in the HHRA (pp. 33-35). Separating the variability (*i.e.*, differences within a population) and uncertainty (*i.e.*, that which is not known) requires substantially more data, including a probability distribution defining the variability for a particular parameter, and a quantitative measure of the uncertainty for the probability distribution (see, HHRA p. 35). For example, fish ingestion rates were derived from data in the 1991 New York Angler survey (Connelly *et al.*, 1992); however, there are no data within the survey to distinguish between variability (*i.e.*, the true differences in fish consumption rates within the angler population) and uncertainty (*i.e.*, differences attributable to the precision with which anglers report their fishing activity).

Although methods do exist to quantitatively characterize uncertainty [e.g., GE Attachment C, p. 5], the level of data available was not sufficient to support their use in the HHRA. Instead, USEPA performed a sensitivity analysis to examine the effect of a range of values for important exposure variables. As stated on HHRA p. 35, "...an explicit 2-Dimensional (2-D) analysis [to address uncertainty and variability] was not performed due to insufficient information available to define quantitative uncertainty distributions for several important exposure variables. The analysis conducted here includes a 1-D Monte Carlo analysis of the variability of exposure as a function of the variability of individual exposure variables. The second component of the [expanded 1-D Monte Carlo] analysis includes an uncertainty/sensitivity analysis for the important exposure variables."

USEPA's tiered approach for risk assessments at Superfund sites calls for standard point estimate calculations as a basis for comparison (USEPA, 1997c). As data allow, the point estimate calculations may be followed by a 1-D Monte Carlo analysis in Tier 2 and a 2-D Monte Carlo analysis. The available data for the HHRA supported the point estimate calculations and an expanded 1-D Monte Carlo analysis, but not a full 2-D Monte Carlo analysis of Tier 3. The lack of sufficient information to perform a 2-D Monte Carlo analysis that explicitly separates variability and uncertainty does not limit the appropriateness of conducting the point estimate calculations and expanded 1-D Monte Carlo analysis, or the validity of the overall conclusions of the HHRA.

Uncertainty associated with the toxicity factors was qualitatively addressed in the HHRA (pp. 61-66, pp. 76-77, and Appendix C). Consistent with USEPA policy on Monte Carlo analysis (USEPA, 1997c), the HHRA did not quantitatively assess this uncertainty in the Monte Carlo analysis because the Agency has not yet developed and approved a methodology for using Monte Carlo Analysis to eva;uate dose response (HHRA, p. 35).

3.2 Derivation of Exposure Factor Distributions

3.2.1 Fish Ingestion Rate

Response to HS-1.5

Highly exposed and lesser exposed anglers are represented in the distributions of cancer risk and non-cancer hazards generated in the Monte Carlo Analysis using the high-end and low-end of the fish

ingestion rate distribution derived from the 1991 New York Angler survey (Connelly *et al.*, 1992) (see, HHRA p. 45). As shown in Table 3-1 of the HHRA, the 10th percentile of the fish ingestion rate distribution consumed one fish meal per year, the 95th percentile consumed 102 fish meals per year (about two meals per week), the 98th percentile consumed 292 fish meals per year (five to six meals per week), and the 99th percentile consumed 393 fish meals per year (over one meal per day). Review of the limited literature available on subsistence (Wendt, 1989) or highly exposed angler populations supports the assumption that these subpopulations are likely to be adequately represented in the total distribution of fish ingestion rates developed for Upper Hudson River anglers (HHRA, p. 45). USEPA notes that NYSDEC's August 31, 1998 comment letter on the HHRASOW did not include NYSDOH's May 20, 1998 comment regarding highly exposed and lesser exposed subpopulations.

Response to HL-1.1, HL-1.14

Consistent with USEPA policy and guidance (USEPA 1989, 1990a, 1992a, 1992b, 1995), the HHRA is a baseline risk assessment, and thus evaluates current and future cancer risks and non-cancer health hazards to human health based on the assumption of no remediation or institutional controls, such as the fish consumption advisories currently in place (see, for example, HHRA, pp. ES-1, 1, 41 and 73). As stated in the NCP preamble, "[t]he baseline risk assessment is essentially an evaluation of the no-action alternative. Institutional controls, while not actively cleaning up the contamination at the site can control exposure and, therefore, are considered to be limited action alternatives. The effectiveness of the institutional controls in controlling risk may appropriately be considered in evaluating the effectiveness of a particular remedial alternative, but not as part of the baseline risk assessment." (USEPA, 1990b, p. 8711). The findings of a study of mortality among workers at two capacitor plants (Kimbrough *et al.*, 1999) do not obviate USEPA's need to conduct a baseline risk assessment. The comment that the workers at these plants "were likely anglers in the Upper Hudson" is unsupported by Kimbrough *et al.* (1999), which evaluated occupational exposures only.

To protect human health and provide a full characterization of the PCB cancer risks and non-cancer health hazards, both an average (central tendency) exposure estimate and a Reasonably Maximally Exposed (RME) estimate were calculated. The RME is the maximum exposure that is reasonably expected to occur. The RME is not a worst case exposure scenario as suggested by the comment.

In the HHRA (p. 70), cancer risks and non-cancer hazards were calculated in the Monte Carlo analysis using concentrations from specific locations including the Thompson Island Pool (River Mile 189), the Waterford/Federal Dam area (River Miles 157-154), and using the average of Thompson Island Pool, Stillwater (River Mile 168) and Waterford/Federal Dam area (base case). The results for the base case (average of three modeled locations) and for the high-end (Thompson Island Pool) are discussed in the HHRA on p. 78. The cancer risks and non-cancer hazards for the low-end (Waterford/Federal Dam area) are shown in the HHRA (Appendix B), but are not discussed in detail because they are not directly comparable to the point estimate calculations of cancer risks and non-cancer hazards for the central tendency and RME scenarios used by USEPA in its risk management decision-making.

Response to HL-1.8 HP-4.5, HP-4.6

As discussed in the HHRA (p. 41), Hudson-specific fish ingestion information is inappropriate to determine the fish ingestion rate because the objective of the HHRA is to estimate cancer risk and non-cancer hazards under baseline condition (*i.e.*, in the absence of remediation and institutional controls, such as the fish consumption advisory currently in place). As noted on p. 44 of the HHRA, the 1991-1992 (Barclay, 1993) and 1996 Hudson angler surveys (NYSDOH, 1999) showed that 92% of Upper

Hudson River anglers reported never eating their catch, indicating that the fish consumption advisories affect angler behavior.

The 1991 New York Angler survey (Connelly *et al.*, 1992) included completed responses from 1,030 licensed anglers (HHRA, p. 38). Eight of the respondents reported having fished in the Upper Hudson River, and three respondents reported having caught and eaten fish from the Upper Hudson River despite the fishing ban in place at the time. The 1991 New York Angler survey (Connelly *et al.*, 1992) is appropriate for use in determining a fish ingestion rate under baseline conditions, in part, because most of the respondents did not fish in the Hudson River.

Although respondents of the New York Angler survey (Connelly *et al.*, 1992) were not asked about meal size, the half-pound fish meal assumed in the HHRA is reasonable and is not likely to be a significant source of uncertainty. The half-pound meal size is consistent with typical assumptions about meal size made by NYSDOH for its fish consumption advisories in the Hudson River (NYSDOH, 1999a, assumes 30 grams per day *vs.* 31.9 grams per day in HHRA), by other state agencies (Cunningham *et al.*, 1990), and by the Great Lakes Sport Fish Advisory Task Force (GLSFATF, 1993) (HHRA, p. 42). In addition, when Lake Ontario anglers were shown sample fish fillets of various sizes, they reported an average meal size of 232 grams/meal, or approximately one-half pound (HHRA, p. 42, citing Connelly *et al.*, 1996). The issue of recall bias in mail surveys, such as the 1991 New York Angler survey (Connelly *et al.*, 1992) is addressed in the HHRA (pp. 39 and 46). Connelly and Brown (1995) reported that the amount of fish consumed was about 10% higher in the 12-month recall mail questionnaires for Lake Ontario anglers than in the diary responses (HHRA, p. 39). The uncertainty associated with fish ingestion rate is discussed in the HHRA (pp. 73-74); however, given the above, meal size and recall bias are not identified as major sources of uncertainty.

The 1988 New York Angler survey (Connelly *et al.*, 1990) reported that 10,310 anglers fished on the Upper Hudson River. However, the number of anglers estimated to fish the Upper Hudson is not central to the HHRA, which calculated cancer risks and non-cancer hazards to the individual under baseline conditions.

Response to HL-1.10

The 1992 Lake Ontario Diary Study (Connelly and Brown, 1995; Connelly *et al.*, 1996) data were used in the HHRA to provide additional perspective on fish consumption rates using different survey instruments (HHRA, pp. 39-40). Connelly and Brown (1995) reported that the number of days fished was 44-45% higher and the amount of fish consumed was about 10% higher in the 12-month recall mail questionnaires than in the diary responses.

Response to HL-1.11

The Connelly and Knuth (1993) and Connelly et al, (1993) surveys were not discussed in more detail because they focused specifically on angler knowledge and response to Great Lakes health advisories and communication techniques (HHRA, p. 39), which is outside the scope of the HHRA. USEPA determined that it was appropriate to use this information in the HHRA as background information and included it for completeness. In contrast, the 1992 Lake Ontario Diary Study (Connelly and Brown, 1995; Connelly *et al.*, 1996) was discussed, despite the fact that it targeted Lake Ontario anglers, because it allowed comparison of mail (recall) surveys and diary studies.

Response to HL-1.12 and HL-1.15

The objective of the HHRA is to estimate cancer risks and non-cancer hazards under baseline conditions, in the absence of remediation and institutional controls such as the fish consumption advisories currently in place. The indication that few Upper Hudson anglers currently eat their catch (HHRA, pp. 40 and 44) strongly suggests that the current fish consumption advisory affects angler behavior, and therefore that Hudson-specific data are inappropriate for use in the baseline HHRA. Therefore, the data from the 1991-1992 and 1996 Hudson Angler surveys (Barclay, 1993; NYSDOH, 1999b) were not used quantitatively in the HHRA to develop fish ingestion rates. The fact that some anglers reported eating fish despite the fish consumption advisories supports the statement in the HHRA that the fish consumption advisories are not 100% effective (HHRA, pp. 40 and 44-45).

Consistent with USEPA policy and guidance (USEPA, 1989, 1992b, 1996a, 1997c), USEPA calculated baseline point estimate cancer risks and non-cancer hazards to the central tendency and Reasonably Maximally Exposed individual, and calculated low estimate, base case and high estimate risks to an individual using Monte Carlo analysis; these calculations do not consider the size of the population consuming fish. For purposes of calculating risks to an individual, it is immaterial that 92% of anglers reportedly did not eat their catch.

The summary of 1991-1992 and 1996 Hudson Angler surveys (Barclay, 1993; NYSDOH, 1999b) was provided to help characterize the composition of the Hudson River angler population using existing data (HHRA, p. 39). These two surveys were combined by the study authors in the 1999 NYSDOH report (NYSDOH, 1999b). Combining the study results was appropriate because the two studies used almost identical surveys, used the same sampling approaches, and covered the same geographic areas of the Hudson River (i.e., Fort Edward to south of Tappan Zee Bridge). It is difficult to extrapolate the data reported "per month" in these surveys to average annual ingestion rates, because it is unknown whether the ingestion rates should be multiplied by 12 months of the year, by 9 months of spring, summer and fall, by 3 months of late spring/early summer, or by some other time period, due to the seasonal variations in freshwater fishing (HHRA, p. 44).

Response to HL-1.13

The Mid-Hudson Angler survey (Jackson, 1990) focused on the Mid-Hudson between Stuyvesant and Kingston, which has different fish consumption advisories, fish species, and PCB concentrations than the Upper Hudson River, and therefore was not discussed in detail in the HHRA for the Upper Hudson River (see, p. 40). However, in response to the comment, less than half of the anglers kept and ate the fish they caught (the percentage varied depending on the target fish species) and 81% of the anglers were aware of the fish consumption advisories.

Response to HL-1.16

Cancer risks and non-cancer hazards to women and children who are avid anglers were quantitatively assessed in the HHRA to the extent that they are represented in the 1991 New York Angler survey (Connelly *et al.*, 1992), because it is plausible that they would consume just as many fish meals as male anglers. Because children eat less fish per meal than adults, fish meal sizes for children were scaled according to body weight in the Monte Carlo assessment (HHRA, p. 46) and were considered to be 1/3 of an adult portion for the point estimate RME child cancer risks (HHRA, p. 69). The non-cancer health hazard for a child aged 1 to 7 years consuming fish at a rate of 1/3 of the adult portion, for 7 years, with a body weight of 18 Kg, was 150. (see Response on p. 19). Cancer risks and non-cancer hazards to men, women and children who are not anglers but who might receive fish from an angler friend or family

member were not quantitatively evaluated because there is insufficient data to quantify fish consumption rates for non-anglers (HHRA, pp. 72 and 74).

Response to HP-1.3

In the 1988 New York Angler survey (Connelly *et al.*, 1990), the "Upper Hudson" was defined as the area above the Federal Dam at Troy. Thus, the "Upper Hudson" as defined by Connelly *et al.* (1990) may extend north of the Hudson Falls, the northernmost area assessed in the HHRA. Consumption rates for various portions of the river were not specified, so it is not possible to segregate the survey data that pertain to the study area of the HHRA. However, the number of anglers in the Upper Hudson (as defined by Connelly *et al.*, 1990) was mentioned in the HHRA (p. 38) only to convey that fishing is a popular recreational activity; it was not used in the calculation of cancer risks and non-cancer hazards. Consistent with USEPA guidance (1989), the HHRA quantifies cancer risk and non-cancer hazardsto the individual, not to a population.

Response to HP-1.7

The assumption in the HHRA (p. 22) that all sportfish consumed are caught in the Upper Hudson River is protective of human health. The assumption is reasonable given that 56.5% of the respondents in the 1991 New York Angler survey (Connelly *et al.*, 1992) reported that they fished in only one or two locations (35.5% in one location, 21% in two locations).

Response to HP-1.11

USEPA evaluated the 1993 Maine Angler survey results (Ebert *et al.*, 1993) which show, for example, that an angler consuming the fish and not sharing it with household members would have a fish ingestion rate of 27 grams/day at the 95th percentile, while an angler sharing fish equally with other household members would have a fish ingestion rate of 12 grams/day at the 95th percentile (HHRA, p. 40 and Table 3-2). The 1993 Maine Angler survey results (Ebert *et al.*, 1993) for an angler not sharing fish were used in the Monte Carlo analysis (HHRA, pp. 78-79). However, the 1993 Maine Angler survey results for shared fish were not used in calculating cancer risks and non-cancer hazards because there is little quantitative information available on such exposures (HHRA, p. 72) and because the lower fish ingestion rate would not be protective of human health for the RME individual.

Response to HP-1.12

The 1991-1992 and 1996 Hudson River Angler Survey (Barclay, 1993; NYSDOH, 1999b) reported that 92% of the respondents never eat their catch (HHRA, p. 40). The 1991 New York Angler survey (Connelly *et al.*, 1992) reported that 42.7% of respondents never eat their catch (HHRA, p. 42). Because these two surveys were conducted at different times and for different anglers, it is not surprising to find that the reported angler activities vary. USEPA used the information from these surveys to characterize the impact of fish consumption advisories (HHRA, pp. 45-46) on fishing practices.

Response to HP-1.13

In the HHRA, USEPA excluded total fish ingestion rates derived from the 1991 New York Angler survey (Connelly *et al.*, 1992) that were greater than 1,000 fish meals per year, based on their implausibility given that three meals every day for a year would total 1,095 meals (HHRA, p. 42). Specifically, using this criterion USEPA excluded one respondent who reported eating 2,228 meals per year from various lakes and rivers in New York State (the Hudson River was not one of the rivers

indicated by that respondent). It is not possible to identify that respondent for any epidemiological or forensic medical studies because the survey does not identify the names of respondents in order to protect their confidentiality.

Response to HG-1.13, HG-1.28, and HG-1.53

The 1991 New York Angler survey (Connelly *et al.*, 1992) was used to derive the fish ingestion rates for the point estimate calculations of cancer risks and non-cancer hazards. In the HHRA, USEPA compared the central (or average) and high-end fish ingestion rates used in the HHRA to the 1993 Maine Angler survey (Ebert *et al.*, 1993) and the 1992 Lake Ontario diary study (Connelly *et al*, 1996) and other surveys (see, HHRA, p. 44 and Table 3-2). The fish ingestion rates used in the HHRA are within the range of ingestion rates found in these other surveys and the ingestion rates recommended in the USEPA Exposure Factors Handbook (USEPA, 1997a) (HHRA, p. 43). The rationale for using the 1991 New York Angler survey data rather than the 1993 Maine Angler survey data is addressed in the HHRA (p. 42). The 1992 Lake Ontario Diary Study (Connelly *et al.*, 1996) was not used to develop a fish ingestion rate distribution for the point estimate calculations, in part, because the survey results documented that the fish consumption advisories in place at the time of the survey reduced fish consumption by the participants (i.e, 32% indicated that they would eat more fish if there were no health fish consumption advisories) (HHRA, p. 39). Of the available studies of sportfish ingestion, the 1991 New York Angler survey is considered the preferred study to represent Upper Hudson River anglers because, among other reasons, it was conducted in New York State and included a large sample size (HHRA, p. 73).

In the Monte Carlo analysis, USEPA compared the cancer risks and non-cancer hazards using the 1991 New York Angler survey (base case) to the results obtained using the 1993 Maine Angler survey (assuming fish is not shared) and the 1992 Lake Ontario Diary Study (for all sources of fish) (HHRA, p. 70). The results for the 1993 Maine Angler survey, which has a lower fish ingestion rate, are presented in the HHRA (p. 79).

The limitations of the 1991 New York Angler survey (Connelly *et al.*, 1992) relating to survey response rate, weighting non-respondents, recall bias, and meal sizes are recognized as sources of uncertainty; these issues are addressed qualitatively in the HHRA (Section 3.2.1).

3.2.2 PCB Concentration in Fish

No significant comments were received on this section.

3.2.3 Cooking Loss

Response to HL-1.9 and HP-1.8

Studies on the loss of PCBs from fish during food preparation and cooking reported a range from 74% loss to a net gain of 17% (HHRA, pp. 24, 48-49 and Table 3-5). The assumption of no cooking loss is within the high-end but is not the worst case, and therefore is appropriate for use in the point estimate RME calculations in the HHRA. A value of 20% was used in the central tendency point estimate calculation to reflect the range from 10 to 40% found in most studies (HHRA, p. 48). In the Monte Carlo analysis, cooking loss was evaluated in the sensitivity analysis using 0% for the high-end estimate, 20% for the central tendency estimate, and 40% for the low-end estimate (HHRA, p. 49).

Response to HG-1.32

In the HHRA, USEPA summarized laboratory studies of fish preparation and cooking methods conducted to quantify the extent of PCB loss prior to consumption (HHRA, Table 3-5). Many of the fish species used in these studies are not found in the Upper Hudson River. Moreover, the studies with similar fish species were conducted over more than 20 years, and the results may not be comparable due to developments in the study and analytical methodologies. In addition, total losses of PCBs during cooking can be affected by factors other than cooking method, such as length of time the fish is cooked, the temperature during cooking, preparation techniques, the lipid content of the fish, the fish species, the magnitude of the PCB contamination in the raw fish, and the extent to which lipids separate during cooking (HHRA, pp. 48-49). For these reasons, USEPA determined that the available literature was inadequate to develop a site-specific distribution of PCB losses during fish preparation and cooking.

3.2.4 Exposure Duration

Response to HF-1.12, HS-1.4

The start date for the exposure of anglers is 1999 (HHRA pp. 12 and 53; see also, USEPA, 1999g, USEPA, 1999g, pp. 28 and 29). This is appropriate because the HHRA evaluates current and future risk, and 1999 is the year in which the HHRA was completed. Use of a start date before 1999 would not be consistent with USEPA risk assessment guidance (USEPA, 1989 and USEPA, 1996b). Specifically, the expert panel that reviewed the current PCB cancer slope factors did not support adjusting for internal dose to reflect previous PCB exposure and current body burdens because the data were not available to determine the appropriate dosimetric for PCB carcinogenicity based on existing PCB body burdens (USEPA, 1996b, p. 11) (see also, USEPA, 1999g, p. 28). USEPA notes that NYSDEC's August 31, 1998 comment letter on the HHRASOW did not include NYSDOH's May 20, 1998 comment letter.

Response to HS-1.3, HS-1.11

Use of a lifetime exposure duration (*e.g.*, 70 years) in the point estimate calculations of cancer risks and non-cancer hazards is inconsistent with USEPA guidance (USEPA, 1989) and is more representative of a "worst case" exposure scenario than an RME scenario. For the cancer risk calculations, the exposure duration distribution for the Monte Carlo analysis ranged from 10 to 60 years (HHRA, Table 3-5b), covering the possibility of extended exposure. For comparison, the current USEPA default recommendation (*i.e.*, in the absence of site-specific data) for the exposure duration parameter for Superfund risk assessments is 30 years for the RME based on national mobility statistics for the general population (USEPA, 1989; USEPA 1997a, as cited in HHRA, p. 57).

Response to HL-1.17

Fishing appears to be an increasingly popular recreational activity, based on the increase in the estimated number of angler days from [year] to [year] reported by Jackson (1990) (HHRA, p. 7). However, USEPA is not aware of any studies that have evaluated angler age profiles over the long-term (HHRA, p. 52). The uncertainty associated with assuming no change in the age profile of anglers with time (i.e., steady state) is discussed in the HHRA (pp. 52 and 74). Forecasting future concentrations of PCBs in fish was deemed to be a greater source of uncertainty than the assumption of a steady state angler population (HHRA, p. 57).

Response to HL-1.18

Table 3-6: Joint Distribution Over Current Age and Age at Which Individual Started Fishing, was developed using data from the 1991 New York Angler survey (Connelly *et al.*, 1992) and the approach described in Section 3.2.4.1 of the HHRA (pp. 51-53). The first sentence of Section 3.2.4.1 is amended to include a reference to Table 3-6.

Table 3-7: Time Until Individual Stops Fishing, was developed using data from the 1991 New York Angler survey (Connelly *et al.*, 1992) and the approach described in Section 3.2.4.1 of the HHRA (pp. 53-54). The first sentence of Section 3.2.4.2 is amended to include a reference to Table 3-7.

Response to HL-1.19

USEPA disagrees with the comment that the 1991 New York Angler survey (Connelly *et al.*, 1992) population is "too small to get reliable values of fish consumption and exposure duration." The 1991 New York Angler survey (Connelly *et al.*, 1992) is a comprehensive mail survey of licensed anglers in New York State. The survey included 1,030 respondents and 919 non-respondents (not 913, see HHRA, pp. 51 and 52), of whom 100 were surveyed by telephone to account for potential non-response bias (HHRA, p. 38). The adjustments to the 1991 New York Angler survey (Connelly *et al.*, 1992) data to determine time remaining until an individual stops fishing (HHRA, pp. 53-54) are reasonable for this application.

Response to HL-1.20

In the HHRA, USEPA acknowledged that the age distribution of the population is likely not strictly steady state (HHRA, p. 57). The HHRA also stated that an assumption of future age distributions based on historical census data was deemed to be a greater source of uncertainty than the assumption of steady state. An age distribution that differs from steady state would not affect the cancer risks and non-cancer hazards in the HHRA, which are calculated for a reasonably maximally exposed individual and not the population.

Response to HL-1.21 and HL-1.22

The comment makes incorrect assumptions regarding the number of anglers and the use of 70 year exposure duration in the Monte Carlo analysis.

Table 3-6: Joint Distribution Over Current Age and Age at Which Individual Started Fishing, is based on the 1991 New York Angler survey (Connelly *et al.*, 1992), which included 1,030 survey respondents and 919 non-respondents, of whom 100 were surveyed by telephone, not the subset of 226 respondents who indicated consumption of fish from non-flowing water bodies (see, HHRA, p. 51).

The angler population was assumed to be exposed until the individual stops fishing, moves out of the area, or dies (see, HHRA, pp. 22-23 and 50). Therefore, exposure duration was determined to be the minimum of either the fishing duration (how long an individual fishes, regardless of the reason for stopping) or the residence duration (how long an individual lives in the five-county area that borders the Upper Hudson River) (see, HHRA, pp. 22-23). The exposure duration distribution ranges from 10 years to 60 years (see, HHRA, p. 57). The exposure duration for the 50^{th} percentile, which was used as the central tendency value in the point estimate calculations, is 12 years; the exposure duration for the 95^{th} percentile, which was used as the RME value in the point estimate calculations, is 40 years (see, HHRA, pp. 50 and 57 and Figure 3-5b). The 98^{th} percentile exposure duration is 54 years and the 99^{th} percentile

exposure duration is 60 years (see, HHRA, Figure 3-5b). The HHRA did not use a 70-year fishing duration directly in the point estimate calculations of cancer risks and non-cancer hazards.

In the Monte Carlo analysis, USEPA used the exposure duration distribution derived as the minimum of fishing duration and residence duration as the base case. As a sensitivity analysis, residence duration alone was used to examine the effect of this parameter on calculations of non-cancer hazards and cancer risk (see, HHRA, pp. 58-59 and 79).

Response to HP-1.14

The age at which an angler begins fishing is important in the calculations of cancer risk and non-cancer hazards because a younger angler has a lower body weight, and body weight is inversely proportional to cancer risks and non-cancer hazards. In addition, the age at which an angler begins fishing can affect the duration of exposure, in that a younger angler has the potential to consume fish for a longer period of time than an angler beginning to fish at an older age. A longer duration of exposure would increase potential cancer risks and non-cancer hazards. It is also important to consider the age at which an angler begins fishing because children are a sensitive population (*i.e.*, they may be more susceptible to the adverse health effects of PCBs) based on bodyweight and the toxicity of PCBs.

Response to HG-1.17 and HG-1.40

To evaluate non-cancer hazards, the Monte Carlo analysis used a maximum exposure duration of seven years, because seven years is the minimum time period for evaluating chronic exposure using USEPA's Reference Doses (RfDs) (see, HHRA, pp. 23 and 70). Due to the declining concentration of PCBs in fish with time, the average daily dose over the first seven years of exposure is expected to be greater than the average daily dose over a longer period of time, although the total dose will be greater with a longer exposure duration (see, HHRA, pp. 23 and 70; USEPA, 1988). As an example, consider an angler with a total exposure duration of 20 years of eating fish from the Upper Hudson. For this angler, it is possible that the average daily dose during the first seven years could exceed the oral RfD for PCBs, while the average daily dose during the last seven years, or the average over the full twenty years of exposure, would not exceed the oral RfD to the same extent. The approach taken in the HHRA was designed to ensure that non-cancer hazards for such anglers are identified. (Similarly, non-cancer hazards to children from ingestion of soil typically are evaluated separately in Superfund risk assessments, rather than averaging the daily doses over the entire exposure duration).

In the Monte Carlo analysis, exposures for one or two years were not, as suggested in the comment, evaluated as if they occurred over seven years or more. Rather, exposures of one or two years (*i.e.*, subchronic exposure) were compared to the chronic RfD based on the limitations of conducting a Monte Carlo analysis using multiple RfDs. While this approach could overestimate non-cancer hazards, based on the existing subchronic RfD for Aroclor 1254, the overestimate is approximately a factor of 2.5. This approach is conservatively protective of human health. In any event, the approach used is not expected to significantly affect the conclusions of the Monte Carlo analysis, given that less than 10 percent of the individuals modeled in the Monte Carlo analysis had exposure durations as low as one or two years, and many of those individuals would also have had relatively low fish ingestion rates. The chronic RfD for Aroclor 1254 is based on a subchronic study including an uncertainty factor of 3 to adjust the RfD for chronic exposures (USEPA, 1997d).

Response to HG-1.31

USEPA acknowledges that the formula adopted in the conversion from five-year to one-year move probabilities is an approximation and that the formula indicated by the commentor is valid.

3.2.5 Body Weight

No significant comments were received on this section.

3.3 Summary of Simulation Calculations

3.3.1 Input Distributions Base Case and Sensitivity Analysis

Response to HS-1.12, G-1.21 and HG-1.43

For the Monte Carlo analysis of the fish ingestion pathway, the HHRA identified values for both base case and sensitivity/uncertainty analysis for a number of input parameters (*i.e.*, fish ingestion rate, exposure duration, fishing location, and cooking loss), which were selected based on their impact on PCB exposure. A total of 72 different combinations of input parameters were analyzed (see, HHRA, pp. 58-59). Four separate fish ingestion studies were examined in the sensitivity analysis in recognition of the fact that the ingestion rates determined from any one study may bias ingestion estimates high or low -- a discussion of recall bias is presented in the HHRA. The fish ingestion studies examined included studies that the USEPA lists as key studies in the USEPA Exposure Factors Handbook (USEPA, 1997a) and studies identified by General Electric Company in comments on the HHRA Scope of Work (see, Responsiveness Summary for HHRA Scope of Work, pp. 17 to 21). The results of the sensitivity/uncertainty analysis are presented in Chapter 5: Risk Characterization (see, HHRA, pp. 77-79).

Consistent with USEPA policy (USEPA, 1997c), the quantification of uncertainties associated with cancer and non-cancer toxicity values were not included in the Monte Carlo analysis, but were addressed qualitatively in the uncertainty discussion section of the risk characterization (see, HHRA, pp. 35, 64, 76-77, see also, USEPA, 1999g, p. 28). A qualitative discussion of the uncertainty associated with toxicity values were also identified in Chapter 4: Toxicity Assessment (see, HHRA, pp. 61-62 and 65-66) and Appendix C: PCB Toxicological Profile (see, HHRA, pp. C-3 and C-5).

3.3.2 Numerical Stability Analysis

No significant comments were received on this section.

4. Toxicity Assessment

Response to HL-1.29

USEPA is aware of ATSDR's February 1999 draft toxicological profile for PCBs and, along with GE and others, submitted comments on the document to ATSDR. In addition, in September 1999

ATSDR held an external review meeting of experts on PCB toxicology, which included an expert from USEPA. ATSDR is currently revising its toxicological profile for PCBs based on comments and the panel's recommendations. USEPA did not include a discussion of ATSDR's document because it is a draft document subject to revision and also covers much of the same literature reviewed by USEPA in the cancer reassessment and the non-cancer hazard assessments for Aroclors 1016 and 1254. Consistent with USEPA guidance and risk assessment policies, the HHRA uses current Agency consensus values for non-cancer toxicity and carcinogenicity from USEPA's IRIS. It should also be noted that data on the acute toxicity of PCBs (exposures ranging from 1 day to 90 days) would not directly affect the conclusions of the HHRA, which evaluates cancer risks and non-cancer hazards due to chronic exposures (i.e., an exposure period of 7 years or more) to PCBs.

Response to HP-1.15, HG-1.15 and HG-1.24

Consistent with USEPA risk assessment policy and guidance (USEPA, 1996a, 1992), the HHRA uses the current toxicity values in IRIS, the Agency's consensus database of toxicity values. The cancer slope factors in IRIS are from USEPA's 1996 reassessment of PCB carcinogenicity (USEPA, 1996b), which was externally peer-reviewed. USEPA's cancer slope factors in IRIS are based on a number of published studies that evaluate the carcinogenic potential of PCBs in both humans and animals, which were conducted by researchers and scientists around the world, not USEPA. Consistent with USEPA risk assessment policy and guidance (USEPA, 1992), the HHRA also contains a summary of the results of the Kimbrough *et al.* (1999) study and the USEPA's preliminary analysis of the data and its effect on the characterization of the carcinogenicity of PCBs (see, HHRA, pp. C2-C3). A summary of the results of the peer review of the cancer reassessment for PCBs and the IRIS chemical files for Aroclors 1254 and 1016 are available on USEPA's web site at www.epa.gov/iris/subst/0294.htm and www.epa.gov/ncea/pcbs.htm.

Response to HG-1.23, HG-1.47, and HG-1.52

USEPA used a weight-of-evidence approach to evaluate PCBs (USEPA, 1996b, 1999a-c). USEPA's cancer and non-cancer toxicity assessments for PCBs considered both human epidemiology and animal carcinogenicity data, as well as other supporting studies (*e.g.*, mutagenicity tests, metabolism data, etc.). USEPA's evaluations of cancer and non-cancer effects of PCBs were externally peer-reviewed and went through internal Agency consensus review before inclusion in IRIS.

USEPA concluded that many of the human epidemiological studies are suggestive that exposure to PCBs can cause cancer and non-cancer health effects in humans; however, limitations of study design, exposure determinations, cohort size, make them inadequate to use in deriving quantitative toxicity values (see, USEPA, 1996b; USEPA, 1999a-c. See also, USEPA, 1999g, pp. 24-27). Due to similar limitations identified by the USEPA in its review of the Kimbrough *et al.* (1999) study, USEPA expects that the Kimbrough study will not change the Agency's conclusions regarding the weight of evidence of the human PCB data, or the health effects of PCBs in general (See, HHRA, pp. C-3). The ATSDR document referred to in the comment is a draft, and is currently undergoing revisions. USEPA, along with numerous others, has submitted comments to ATSDR regarding the draft document and also participated in the external review panel set-up by ATSDR to address the comments received on the document. (See Response to HL-1.29).

USEPA has not developed a new CSF for PCBs based on the Kimbrough *et al.* (1999) study or any of the other human epidemiological studies precisely because of their inadequacies and limitations as described in the IRIS file. USEPA is currently reassessing the RfDs for PCBs and the overall weight of evidence for PCB health effects, as well as considering the significance of recent human epidemiological

studies of PCBs, although USEPA has not yet determined whether existing PCB RfDs currently in IRIS will be amended following the RfD reassessment. Consistent with risk assessment policy and guidance, USEPA considered relevant new toxicological information prior to using the existing IRIS toxicity values in the HHRA (USEPA, 1999h, HHRA Appendix C, pp. C-1 to C-6).

4.1 Non-cancer Toxicity Values

Response to HS-1.15

Had a route-to-route conversion been applied to the oral RfDs for Aroclor 1254 and 1016 as suggested in the comment (assuming an adult inhalation rate of 20 m^3/day and an adult body weight of 70 kg), the resulting inhalation reference concentrations would be 7.0×10^{-5} and 2.5×10^{-4} mg/m^3, respectively. The central tendency and RME exposure point concentrations used in the HHRA for PCBs in air were 1.0×10^{-6} and 1.7×10^{-5} mg/m^3, and are lower than the reference concentrations for both Aroclors 1016 and 1254 and therefore the Hazard Index is for the central tendency 0.004 (Aroclor 1016) and 0.01 (Aroclor 1254) while for the RME the hazard index is 0.07 (Aroclor 1016) and 0.2 (Aroclor 1254).

Response to HS-1.17

The critical studies, critical effects, and uncertainty factors for the RfDs for Aroclor 1016 and Aroclor 1254 are discussed in the HHRA (see, pp. 62 and C5-C6) and details are provided in the IRIS files for the individual Aroclors (USEPA, 1999a-c).

Response to HL-1.23

USEPA has used all four standard uncertainty factors in deriving RfDs for some chemicals (yielding total uncertainty factors of 1,000 or greater). However, this is not the case for PCBs. The oral RfDs for Aroclor 1016 has a total uncertainty factor of 100 due to intraspecies variability and protection of sensitive individuals (UF=3), interspecies variability (UF=3), database limitations (UF=3), and use of a subchronic study (UF=3). The oral RfD for Aroclor 1254 has a total uncertainty factor of 300 due to intraspecies variability (UF=10), interspecies variability (UF=3), use of a Lowest Observed Adverse Effect Level dose (UF=3), and use of a subchronic study (UF=3) (see, HHRA, p. C-6). Total uncertainty factors of 100 or 300 are not particularly large compared to uncertainty factors associated with RfDs for other chemicals. The use of uncertainty factors in deriving the RfDs for Aroclor 1016 and 1254 is consistent with USEPA policy, guidance, guidelines, and risk assessment practices (USEPA, 1989, 1999a-c and Dourson *et al.*, 1989). The use of uncertainty factors does not suggest that the data upon which the RfD is based are questionable, or that they are insufficient to use in human health risk assessments, but rather provide a means of protecting sensitive subpopulations such as children, elderly, and individuals with existing medical conditions.

Response to HL-1.24, HG-1.25 and HG-1.26

Consistent with USEPA policy (USEPA, 1997c), the uncertainties associated with non-cancer toxicity values were not included in the Monte Carlo analysis as a distribution, but were qualitatively addressed in the uncertainty section of the risk characterization (see, HHRA, pp. 35 and 76-77, see also, USEPA, 1999g, p. 28). Uncertainty associated with non-cancer toxicity values were also identified in Chapter 4: Toxicity Assessment (see, HHRA, pp. 61-62 and 65-66) and Appendix C: PCB Toxicological Profile (see, HHRA, p. C-5).

Response to HL-1.35

Rounding total uncertainty factors is used in developing RfD values, that are designed to be protective of human health including sensitive populations such as children. The RfD for Aroclor 1016 was externally reviewed, (USEPA, 1992). Straight multiplication of the individual uncertainty factors would imply a greater degree of precision concerning the uncertainty than exists. Moreover, the rounding the uncertainty factors from 81 to 100 and from 270 to 300 in deriving the RfD does not change the overall conclusions of the HHRA with respect to non-cancer health hazards.

Response to HP-2.1

Neurobehavioral effects in monkeys (Seegal *et al.*, 1990, 1991) were considered by USEPA in deriving the RfD for Aroclor 1016 (USEPA, 1999a). In addition, the sophisticated analytical chemistry program implemented for the Hudson River PCBs Reassessment required the use of state-of-the-art gas chromatography methodology. A total of 90 PCB congeners were selected as target congeners based on their significance in environmental samples and the availability of calibration standards at the start of the overall Reassessment sampling program. Qualitative and quantitative information for an additional 53 to 58 PCB congeners was obtained from each sample analysis using relative retention time information detailed in the literature, and more recently verified with actual standards (see, USEPA 1997e). Overall, the analytical method focused on 12 "principal" target congeners, one of which was 2,2'-dichlorobiphenyl (see, USEPA, 1997e).

4.2 PCB Cancer Toxicity

Response to HS-1.14

In the HHRA, USEPA selected cancer slope factors based on the environmental medium being evaluated, which is consistent with current USEPA guidance (USEPA, 1996a; USEPA, 1999e). The IRIS file recommends using congener analyses to identify PCB mixtures where congeners with more than 4 chlorines comprise less than one-half percent of the total PCBs (which is not applicable in the Hudson River) or to conduct a supplemental analysis of dioxin TEQs (which was performed in the HHRA, see pp. 69-70, Tables 5-35 to 5-37 and 4-5).

Response to HL-1.25

The central-estimate cancer slope factor of 1.0 was used in the central tendency point estimate calculations (see, HHRA, pp. 64 and C-3). Consistent with USEPA policy (USEPA, 1997c), the uncertainties associated with toxicity values were not quantitatively included in the Monte Carlo analysis, but were qualitatively addressed in the uncertainty section of the risk characterization (see, HHRA, pp. 35, 76-77, see also, USEPA, 1999g, p. 28). Uncertainty associated with cancer toxicity values was also identified in Chapter 4: Toxicity Assessment (see, HHRA, pp. 63-64) and Appendix C: PCB Toxicological Profile (see, HHRA, p. C-3).

4.3 Toxic Equivalency Factors (TEFs) for Dioxin-Like PCBs

Response to HF-1.4, HF-1.13, HF-1.17

The HHRA evaluated all but one of the PCB congeners for which toxic equivalency factors (TEFs) are available (Tables 4-5, 5-35, and 5-36 of the HHRA summarize the congener data and TEFs used). The one exception is the non-ortho PCB congener 81, for which a published TEF was not available until 1998. As noted in the discussion of uncertainties (see, HHRA, p. 76), the toxicity of each individual PCB congener has not been fully characterized, and TEF values have not been developed for all PCB congeners. Nonetheless, the toxicity of these congeners is likely to be reflected in the toxicity values for total PCBs. However, if there were congeners that were not accurately reflected in the PCB congener data, such that the congener distribution in fish were dramatically different from the commercial Aroclors on which the toxicity values are based, then it is possible that risk could have been underestimated.

Data quality issues for PCBs in fish were discussed in the Data Usability Report for PCB Congeners (USEPA, 1997e). Overall, the sophisticated analytical chemistry program implemented for the Hudson River PCB Reassessment RI/FS required the use of state-of-the-art gas chromatography methodology. A total of 90 PCB congeners were selected as target congeners (and 12 "principal" target congeners were chosen) based on their significance in environmental samples and the availability of calibration standards at the start of the Reassessment RI/FS sampling program (USEPA, 1997e). Qualitative and quantitative information for an additional 53 to 58 PCB congeners was obtained from each sample analysis using relative retention time, information detailed in the literature or, more recently, verified with internal standards. Table A-1 in the Data Usability Report for PCB Congeners lists all 148 PCB congeners that were selected as part of the Phase 2 analysis program (USEPA, 1997e).

Response to HP-3.8, HP-4.4, HG-1.27

Consistent with the recommendations in USEPA's 1996 reassessment of PCB carcinogenicity (USEPA, 1996b), USEPA calculated TEQs for PCBs to calculate the cancer risks of ingestion of dioxin-like PCBs (see, HHRA, pp. 69-70). However, the analysis of dioxin-like PCBs was not the focus of the HHRA. The primary purpose of calculating risks of dioxin-like PCBs was to ensure that the relative concentrations of dioxin-like congeners have not been significantly enhanced in environmental mixtures in the Upper Hudson River to change the overall conclusions regarding cancer risk. The USEPA cancer slope factor (CSF) for 2,3,7,8-TCDD is 150,000 $(mg/kg\text{-}day)^{-1}$, based on a study showing liver and respiratory system tumors in rats, as described in USEPA's 1997 Health Effects Assessment Summary Tables (HEAST) (USEPA, 1997d) (see, HHRA, p. 69). Currently, there are no USEPA-recommended toxicity criteria for non-cancer effects of 2,3,7,8-TCDD such as an RfD, and thus a quantitative assessment on non-cancer effects based on dioxin-like toxicity is not feasible at this time. The USEPA acknowledges that there are uncertainties associated with the cancer slope factor for TCDD. The cancer and non-cancer toxicities of 2,3,7,8-TCDD are currently being reevaluated by the USEPA as part of the Dioxin Reassessment.

Nonetheless, the results of the HHRA are not "driven" by the TCDD cancer slope factor. The HHRA found that the RME cancer risk for ingestion of dioxin-like PCBs in fish was approximately equivalent to the RME risk calculated without separate consideration of the dioxin-like congeners (see, HHRA pp.69-70). Because there was no evidence that the concentrations of dioxin-like congeners were

enhanced enough in the Upper Hudson River to change the overall conclusions regarding cancer risk estimates, USEPA did not perform more detailed analyses of dioxin-like PCB risks.

4.4 Endocrine Disruption

Response to HP-3.9

The USEPA Science Policy Council's interim position on endocrine disruption is that "based on the current state of the science, the Agency does not consider endocrine disruption to be an adverse endpoint per se, but rather to be a mode or mechanism of action potentially leading to other outcomes, for example carcinogenic, reproductive, or developmental effects, routinely considered in reaching regulatory decisions" (see, HHRA, p. 66). In other words, although endocrine disruption is an important mechanism of action and the subject of on-going research by federal agencies and other organizations, many of the expected health endpoints are already assessed in standard non-cancer and cancer toxicity testing, and thus are likely to already be accounted for in USEPA toxicity values.

5. Risk Characterization

Response to FS-1.10

The modeled PCB concentrations, by fish species and location, are shown in Figures 2-1 through 2-10. Consistent with USEPA guidance, the HHRA calculates cancer risks and non-cancer hazards using site-specific information rather than comparing the modeled future fish concentrations to the U.S. Food and Drug Administration (FDA) tolerance level of 2 ppm PCB in fish and shellfish (edible portion) shipped in interstate commerce. A discussion of the FDA tolerance level and its limitations is presented in Appendix C (p. C-7).

USEPA notes that the FDA tolerance level is not an Applicable or Relevant and Appropriate Requirement (ARAR) with respect to the Hudson River PCBs site, because the FDA tolerance level is not a standard, requirement, criterion, or limitation promulgated under a Federal environmental law, or a more stringent State environmental or facility siting law.

Response to HP-1.4

The goal of this baseline risk assessment is to evaluate cancer risks and non-cancer hazards from exposure to PCBs in the Upper Hudson River in the absence of remedial action or institutional controls (see, HHRA, pp. ES-1 and 1). Therefore, a discussion of the risks from other sources, as suggested in the comment, is beyond the scope of the HHRA.

Response to HP-1.17

The USEPA disagrees with the comment that a conclusion of the HHRA is that "there is no public health hazard from consuming fish from the Upper Hudson River either now or in the future." For the Reasonably Maximally Exposed individual who eats fish from the Upper Hudson River, the cancer risk is 1,000 times higher than USEPA's goal of protection and ten times the highest risk level generally allowed under federal Superfund law. For the RME individual eating fish, the non-cancer

hazard is more than 100 times USEPA's level of concern while for the central tendency exposure the hazard is 10 times USEPA's level of concern.

Response to HP-4.11

A discussion of Travis *et al.* (1987) would not be appropriate in the HHRA because it concerns risk management in the remedy selection process. Consistent with USEPA guidance (1989), risk management is undertaken after completion of the risk assessment, and therefore is beyond the scope of the HHRA.

Response to HG-1.1, HG-1.5, HG-1.9, HG-1.22 and HG-1.45

USEPA agrees that the HHRA found cancer risks associated with inhalation of air, the cancer risks and non-cancer hazards associated with exposure to river water, and the non-cancer hazards associated with exposure to sediment were below levels of concern. However, the cancer risk to the RME individual from exposure to sediment was found to be 10 times greater than USEPA's goal for protection, which is one additional case of cancer per one million people exposed.

The HHRA did not, as suggested by the commentor, evaluate cancer risks and non-cancer hazards associated with eating fish under the current catch and release program or attempt to quantify compliance with the catch and release program (see, HHRA, p. 80). Rather, the HHRA evaluated cancer risks and non-cancer hazards from exposure to PCBs in the Upper Hudson River, including eating fish, in the absence of remedial action or institutional controls such as the fish consumption advisories (see, HHRA, pp. ES-1 and 1). The cancer risk to the RME individual eating fish is 1,000 times higher than USEPA's goal of protection and ten times the highest risk level generally allowed under federal Superfund law, and the non-cancer hazard is more than 100 times USEPA's level of concern. USEPA disagrees with the comment that the future risks of eating fish from the Upper Hudson are "clearly limited" and that "natural recovery will lead to edible fish in the not too distant future," given that the cancer risk and non-cancer hazards from eating fish would remain above USEPA's generally acceptable levels for the 40-year exposure period evaluated in the HHRA.

USEPA cannot directly respond to the commentor's statement that "a remedy such as dredging will not materially accelerate [the time needed to achieve edible fish under natural recovery]," because USEPA has not yet completed its Feasibility Study, in which USEPA will evaluate remedial alternatives for the PCB-contaminated sediments in the Upper Hudson River. The Feasibility Study will include USEPA's analysis of whether alternatives can appreciably accelerate the time required to achieve acceptable human health and ecological risk levels. Concurrent with the Feasibility Study, USEPA will issue a Proposed Plan that presents USEPA's preferred remedial alternative, and will solicit public comment on the Proposed Plan before selecting a remedy based on the nine criteria set forth in the NCP (USEPA, 1990a). The use of the HHRA by the Agency's risk managers in the remedy selection process is beyond the scope of the HHRA (see, USEPA, 1999g, p. 29).

5.1 Point Estimate Risk Characterization

Response to HP-3.3

USEPA agrees that individuals are likely to be exposed via multiple pathways, for example, anglers who eat fish would also be exposed to river water and sediments while they are fishing.

However, because the cancer risks and non-cancer hazards from exposure to sediment, water, and air are at least two orders of magnitude less than the cancer risks and non-cancer hazards from fish ingestion, the total cancer risks and non-cancer hazards would not be significantly different from the cancer risks and non-cancer hazards due to fish consumption alone. Therefore, USEPA determined that it would be more informative to present the cancer risks and non-cancer hazards due to exposure from each pathway rather than to combine those risks across all pathways.

5.1.1 Non-Cancer Hazard Indices

No significant comments were received on this section.

5.1.2 Cancer Risks

Response to HS-1.7

The statements in the HHRA (pp. ES-4 and 68) regarding the acceptable risk range are drawn from the NCP (USEPA, 1990a) which states, "For known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between 10^{-4} and 10^{-6} using information on the relationship between dose and response (40 CFR § 300.430(e)(2)(i)(A)(2), USEPA, 1990a).

Response to HL-1.36

Maximum contaminant levels (MCLs) for chemicals in drinking water are promulgated as part of the National Primary Drinking Water Regulations, a different federal program than Superfund. Regardless, both risk levels presented in the HHRA for ingestion of PCBs in drinking water at the MCL, namely 10^{-4} (based on the old CSF) and 10^{-5} (based on the new CSF), are within the generally acceptable cancer risk range of 10^{-4} to 10^{-6} established in the NCP for Superfund (see, 40 CFR § 300.430(e)(2)(i)(A)(2), USEPA, 1990a).

Response to HP-1.16

The risk estimate referred to in the comment, 3.2 additional cases of cancer in 100,000 people, is for the central tendency estimate; the high-end estimate of cancer risk, *i.e.*, risk to the Reasonably Maximally Exposed individual, is 1.1 additional cases of cancer in 1,000 people (see, HHRA, p. 69). The RME risk is 1,000 times greater than USEPA's goal for protection and ten times the highest risk level generally allowed under federal Superfund law. USEPA agrees that this is a cancer incidence and not a projected death. As a point of clarification, the doses in the HHRA represent lifetime average daily doses of PCBs over a 70-year lifetime based on a 40-year exposure period. Also, it is impossible to determine the number of anglers who eat fish from the Upper Hudson River despite the fish consumption advisories and the number of additional anglers who might fish in the absence of fish consumption advisories (see, HHRA, pp. 7 and 80)

5.1.3 Dioxin-Like Risks of PCBs

Comments on the dioxin-like risks of PCBs were addressed in Section 4.3.

5.2 Monte Carlo Risk Estimates for Fish Ingestion

Comments on the Monte Carlo approach were addressed in Section 3.

5.2.1 Non-Cancer Hazards

Comments on the Monte Carlo approach were addressed in Section 3.

5.2.2 Cancer Risks

Response to HS-1.6

USEPA acknowledges that a sensitivity analysis of distributional assumptions (normal, lognormal, uniform, etc.) might provide additional insights on the impact of exposure variability. However, the sensitivity analysis that was performed in the Monte Carlo analysis, whereby 72 separate combinations of four key exposure factors were examined, covered an adequately wide range of exposure factor variability. Furthermore, the probability distributions for fish ingestion and exposure duration, two factors with a major impact on exposure, were based on empirical distributions (not fitted distributions) for the base case analysis.

5.3 Discussion of Uncertainties

5.3.1 Exposure Assessment

Response to HL-1.26

Individuals born after 1999 or who move to the Upper Hudson River area after 1999 are not quantitatively evaluated in the HHRA (see, HHRA, p. 72). A start date for the exposure of anglers of 1999 is appropriate because the HHRA evaluates current and future cancer risks and non-cancer hazards, and 1999 is the year in which the HHRA was completed (see, USEPA, 1999g, p. 29). Due to the observed trend of decreasing concentrations of PCBs with time, individuals born or moving to the study area (Upper Hudson) after 1999 would be have less exposure to PCBs than the current angler population, so USEPA's approach is appropriately protective of human health. The HHRA calculates cancer risk and non-cancer hazards to an individual, not a population risk.

Response to HL-1.27

The comment misquotes the HHRA, which states that "the resulting point estimates ... are unlikely to underestimate [emphasis added] actual exposure durations significantly" (see, HHRA, p. 74). This statement is based on the fact that the exposure duration estimates were not based on national default values, but were based on data from New York anglers and considering both fishing durations and residence durations. Furthermore, exposure durations used in the point estimates calculations (central tendency duration of 12 years based on 50^{th} percentile and RME duration of 40 years based on 95^{th} percentile) are reasonable when compared to national mobility statistics (median of 9 years, high end of 30 years).

5.3.2 Toxicity Assessment

Response to HS-1.16

In the HHRA, USEPA used the current toxicity values in IRIS. The HHRA provides an overall discussion on the toxicity of PCBs and identifies some additional information available since USEPA last reassessed cancer toxicity in 1996 and non-cancer toxicity in 1992 and 1994 (USEPA, 1999 a-c and e). In particular, the HHRA noted the two studies (*i.e.*, Arnold *et al.*, 1995 and Rice, 1999) that were mentioned by the commentor (see, HHRA, pp. 76-77 and C-4 to C-6). The USEPA is currently reassessing the non-cancer toxicity values for PCBs on a national level (Federal Register, 1998).

5.3.3 Comparison of Point Estimate RME and Monte Carlo Results

Response to HL-1.28

The sensitivity analysis performed as part of the Monte Carlo assessment was designed to examine such effects, one parameter at a time.

References

Response to HF-1.6

The NYSDOH reference is revised to read: "New York State Department of Health (NYSDOH). 1999. Health Advisories: Chemicals in Sport Fish or Game 1999-2000. Albany, New York."

Appendix C, PCB Toxicological Profile

Response to HS-1.18, HP-3.5

In the HHRA, USEPA used the current toxicity values in IRIS. The HHRA provides an overall discussion on the toxicity of PCBs and identifies some additional information available since USEPA last reassessed cancer toxicity in 1996 and non-cancer toxicity in 1992 and 1994 (USEPA, 1999a-c and e). USEPA is currently reassessing the non-cancer toxicity values for PCBs on a national level. See response to HS-1.16. Although articles on PCB non-cancer toxicity and carcinogenicity have been published recently, it is beyond the scope of the HHRA to present a thorough evaluation of all the available scientific literature on PCBs in view of USEPA's current national reassessment. The comment regarding the Lanting/Patandin studies is acknowledged. The first sentence of the second to last paragraph on p. C4 is revised to read "There are several on-going studies assessing the non-cancer health effects of PCBs in children."

Response to HG-1.4, HG-1.6, HG-1.11, HG-1.15, HG-1.24, HG-1.46 HL-1.2, HL-1.30, HL-1.31, HP-3.6, and HP-4.9

Presumably, comment HL-1.2 refers to the Kimbrough *et al.* (1999) study. The Kimbrough *et al.* (1999) study was specifically mentioned in the HHRA because it focused on workers exposed at two General Electric Company capacitor manufacturing plants in upstate New York, was published as the HHRA was being completed, and generated a great deal of attention and controversy in the scientific and regulatory communities. USEPA did not intend to imply that other studies cited "have no imperfections." Many important epidemiology studies were reviewed by the USEPA in the 1996 reassessment of PCB carcinogenicity (USEPA, 1996b) and are described in the IRIS Weight of Evidence classification (USEPA, 1999e). It is also acknowledged that other PCB epidemiology studies, such as the Rothman (Rothman *et al.*, 1997) and Moysich (Moysich *et al.*, 1998) studies, have since been published. However, it is beyond the scope of the HHRA to present a complete evaluation of all the available scientific literature on PCBs.

In evaluating the toxicity of PCBs, USEPA reviewed the epidemiology literature for PCBs, as well as animal carcinogenicity data (USEPA, 1996b; USEPA, 1999e). Based on this information, USEPA concluded that the available evidence from human studies is inadequate, but suggests that exposure to PCBs can cause cancer and non-cancer health effects. The expert panel convened by USEPA (USEPA, 1996b) did not recommend that the epidemiological studies be used to derive CSFs for PCBs, noting inadequacies in the epidemiological data with regard to limited cohort size, problems in exposure assessments, lack of data on confounding factors, and the fact that occupational exposures may be to different congener mixtures than those found in environmental exposures, as well as other limitations and complications associated with interpreting data from human epidemiological studies (see, USEPA, 1999g).

USEPA conducted a preliminary review of the Kimbrough *et al.* (1999) study (HHRA, pp. C2-C3), but identified a number of limitations that suggest the study may not change USEPA's conclusions regarding the health effects of PCBs (see, HHRA, pp. C2-C3. See also response to HP-1.15, above (Section 4, Toxicity Assessment)). Similar limitations have since been identified by other scientists as well (*e.g.*, Bove *et al.*, 1999; Frumkin and Orris, 1999).

With regard to the extent of workers' exposure in the Kimbrough *et al.* (1999) study, the degree of inhalation and dermal exposure to PCBs for all plant occupants is not well characterized. The individuals not working directly with PCBs would have had significantly lower exposures, and their exposure levels would be virtually impossible to quantify without air monitoring data during the exposure. Although some historical air measurements are available for the GE plants, the measurements are only available for 1975 and 1976 and not for earlier time periods when exposures would have been higher. Similarly, with regard to the length and latency of workers' exposure and the population studied (healthy workers), the USEPA recognizes that most of the available human epidemiological studies of PCB exposure have similar limitations supporting USEPA's classification of this data as inadequate but suggestive (USEPA, 1999e).

The USEPA did not intend to imply in the HHRA that the Kimbrough *et al.* (1999) study is "worse" than the other available studies, or that the other epidemiological studies are "cited with approval by EPA." Rather, the USEPA's cancer classification for PCBs is based on a weight of evidence approach – the animal studies are considered "sufficient," while the human data are deemed "inadequate, but suggestive." The deficiencies in the previous human epidemiology studies for PCBs are precisely the reason why they have not been used as the primary basis for deriving USEPA cancer slope factors. At this point, based on its limitations, USEPA does not believe that the results of the Kimbrough *et al.*

(1999) study will be sufficient to change the Agency's conclusions regarding the weight of evidence of the human PCB data, or the health effects of PCBs in general (see, HHRA, p. C-2). Note, however, that these issues are currently being evaluated and the Agency has yet to issue a determination on this matter.

Complete details of USEPA's review and critique of the numerous human epidemiology studies for PCBs are presented in USEPA's IRIS file for PCBs and the USEPA 1996 PCB cancer reassessment document (USEPA, 1999e; USEPA, 1996b). For example, the expert panel convened by USEPA for the reassessment of the PCB cancer slope factor (USEPA, 1996b) noted inadequacies in the epidemiological data with regard to limited cohort size, problems in exposure assessments, lack of data on confounding factors, and the fact that occupational exposures may be to different congener mixtures than those found in environmental exposures, as well as other limitations and complications associated with interpreting human data (USEPA, 1996b).

Response to HL-1.32

The central-estimate CSF of 1.0 $(mg/kg\text{-}day)^{-1}$ for PCBs is not an upper bound. To clarify, the opening sentence of Section C.2.3 refers to the upper-bound CSF of 2.0 $(mg/kg\text{-}day)^{-1}$ for PCBs (see, HHRA, p. C-3). Note that PCBs are somewhat unusual in that both an upper-bound and a central-estimate CSF is included in USEPA's IRIS file. For most chemicals, only an upper-bound CSF is included in IRIS.

Response to HL-1.33

The discussions of the Brunner *et al.* (1996) and Norback and Weltman (1985) studies, as well as numerous other PCB studies discussed in Appendix C, are based on the reviews presented in the USEPA's IRIS file for PCBs (USEPA, 1999e) and USEPA's 1996 reassessment of PCBs (USEPA, 1996b). The complete references for those studies are listed therein (USEPA, 1996b; 1999e) and are as follows:

Brunner, M.J., T.M. Sullivan, A.W. Singer, et al. 1996. An assessment of the chronic toxicity and oncogenicity of Aroclor-1016, Aroclor-1242, Aroclor-1254, and Aroclor-1260 administered in diet to rats. Study No. SC920192. Chronic toxicity and oncogenicity report. Battelle, Columbus OH.

Norback, D.H. and R.H. Weltman. 1985. Polychlorinated biphenyl induction of hepatocellular carcinoma in the Sprague-Dawley rat. Environ. Health Perspect. 60: 97-105.

Both the Brunner *et al.* (1996) and Norback and Weltman (1985) studies are carcinogenicity studies in rats exposed to PCB mixtures identified as commercial Aroclors. The Norback and Weltman (1985) study mentioned on p. C-3 is discussed in some detail in the HHRA (see, HHRA, p. C-2). The 1996 Brunner study, mentioned on p. C-3, was later published by Mayes *et al.* (1998) and is described in the HHRA on p. C-1 (there is a typographical error in this reference in the HHRA – the year should be 1998 and not 1999). The 1996 Brunner reference is a Battelle study report of the 2 years carcinogenicity study in rats (not published). The Mayes *et al.* (1998) article presents the results of the same study in a peer-reviewed article published in the Journal Toxicological Sciences (vol. 41, pp. 62-76).

Response to HL-1.34 and HP-3.7

The Patandin (1999) and Lanting (1998) studies were mentioned in the HHRA to highlight some of the current work on developmental toxicity of PCBs in children (see, HHRA, p. C-4). The studies are

still being evaluated by the USEPA as part of the ongoing reassessment of PCB non-cancer health effects RfD and the overall weight of evidence for PCB health effects.

Breast cancer is discussed in a paragraph on endocrine disruption. Endocrine disruption may be a mechanism of action for various PCB-related health effects, including both cancer and non-cancer health endpoints. The fact that the relationship between PCB exposure and breast cancer is not consistently observed does not affect the strength of the causal association between PCB exposure and liver cancers identified in numerous animal studies.

The Patandin (1999) and Lanting (1999) studies were mentioned in the HHRA as examples of some of the ongoing work on developmental toxicity of PCBs in children. The HHRA did not intend to imply that they were the only basis for inference of PCB health effects. Incidentally, since issuance of the HHRA, Patandin and Lanting have published this work (*e.g.*, Patandin et al. (1998), Patandin (1999), and Lanting et al. (1998)).

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III. RISK ASSESSMENT REVISIONS

1. Summary

This section of the Responsiveness Summary presents the revised baseline Human Health Risk Assessment results for the Upper Hudson River (HHRA). The revision reflects revised concentrations of PCBs in sediment, water column, and bioaccumulation as presented in the Revised Baseline Modeling Report (RBMR) (USEPA, 2000). This revised HHRA incorporates the modified forecast concentrations of PCBs in fish, sediments, and river water from the RBMR and compares the revised results with the August 1999 HHRA results and conclusions.

The overall conclusions from the August 1999 HHRA (USEPA, 1999) remain unchanged for this revised HHRA. That is the revised HHRA shows that cancer risks and non-cancer health hazards to the reasonably maximally exposed (RME) individual associated with ingestion of PCBs in fish from the Upper Hudson River are above USEPA levels of concern as defined by the WCPC (USEPA, 1990). In addition, the revised HHRA indicates that fish ingestion represents the primary pathway for PCB exposure and for potential adverse non-cancer health effects, and that cancer risks and non-cancer health hazards from other exposure pathways are generally below or within levels of concern.

1.1 Introduction

This revised HHRA summarizes the modifications made to the previous exposure parameter estimates and the results of the revised risk calculations. In addition, all tables and figures contained in the previous HHRA are presented in their entirety in this revised HHRA. Those tables and figures that were modified are labeled "Revised," whereas those with no changes are labeled "Unchanged". To facilitate in the ease of comparing revised results with the August 1999 HHRA results, all table and figure numbering have retained their original designations.

1.2 Revisions to Exposure Parameter Estimates

The only exposure parameter modifications made in the revised HHRA were to the fish, sediment, and river water exposure point concentrations (EPCs) in the point estimate analysis. The revised exposure point concentrations were calculated using the revised forecasts from the revised HUDTOX fate and transport model and the revised FISHRAND bioaccumulation model results. Air EPCs were previously based on modeled and empirical estimates. Because the average PCB concentration in the water column, which serves as the source term to the estimates of EPC concentrations of PCBs in air, did not change significantly (from 24 ng/L to 20 ng/L), the air modeling result would also not change significantly. No change was made in the air EPCs and no revision was made to the exposure point concentrations and cancer risk estimates for the air pathway.

No change has been made to the Monte Carlo analysis that was performed for the fish ingestion pathway in the August 1999 HHRA. The reason for this is that the revised PCB concentration forecasts for fish (the only exposure parameter from the modeling revisions that could affect the Monte Carlo analysis of fish ingestion) have not changed substantially (see, Table 4.1). As described below, the revised forecasts of PCB concentration in fish averaged over the the Upper Hudson exposure area range

from a high of a 2-fold decrease down to 1.2-fold decrease compared with the 1999 results. Because exposure (and risk) is proportional to concentration, the decline in PCB concentration in fish would translate to a corresponding decline in exposure. Yet, even if the Monte Carlo exposure estimates for fish consumption were scaled by as much as 2-fold, this adjustment would not significantly change the overall Monte Carlo results. In view of this fact, it was deemed unnecessary to repeat the Monte Carlo analysis.

1.2.1 Fish

Revised Tri+ PCB annual averages for brown bullhead, largemouth bass, and yellow perch are summarized in the Ecological Responsiveness Summary (USEPA, 2000). In the August 1999 HHRA, modeled results of Tri+ PCB concentrations in fish were available for a forecast period of 20 years. An exponential curve-fit procedure was used in the 1999 HHRA to extend the forecast period to 70 years to reflect the possible exposures evaluated in the Monte Carlo analysis. The revised BMR forecast Tri+ PCB concentrations in fish for a 70 year time-period such that no curve fitting procedure was used for the EPC values in fish in this revised HHRA.

As was the procedure in the August 1999 HHRA, EPCs were calculated for the adult angler by species-weighting and averaging the forecasted fish concentrations over river mile segment and exposure duration. A comparison of the revised fish EPCs is shown in Table 4.2. In general, the forecast PCB concentration in fish declined from the 1999 BMR results. When averaged over the three locations, the RME concentration decline is approximately 2-fold for brown bullhead, with smaller declines, 1.3- to 1.2-fold for largemouth bass and yellow perch, respectively. The species weighted RME (40-year) concentration in fish declines from 2.2 mg/kg in the 1999 HHRA, to 1.4 mg/kg in this revised HHRA, or approximately a 1.6-fold decline. A discussion of the reasons for the decreased forecasts in the August 1999 and revised BMR is provided in the Ecological Responsiveness Summary (USEPA, 2000).

Table 4.1 Comparison of 1999 and Revised PCB Concentrations in Fish
Reasonable Maximum Exposure (RME) Over 40 Years

	Thompson Island Pool		River Mile 168		River Mile 157 & 154		RME Average Over 3 Locations	
Fish	**1999**	**Revised**	**1999**	**Revised**	**1999**	**Revised**	**1999**	**Revised**
Brown Bullhead	4.7	2.3	2.6	1.4	0.9	0.35	2.7	1.3
Largemouth Bass	2.3	3.1	2.0	0.8	1.1	0.34	1.8	1.4
Yellow Perch	2.1	3.0	1.6	0.6	0.9	0.25	1.5	1.3

RME values from Tables 2-6, Table 2-7 and Table 2-8.

1.2.2 Sediment and River Water

The RBMR provides forecasts of Tri+ PCB annual averages in sediment and river water for the Upper Hudson River (USEPA, 2000). As was the case for the previous HHRA, the modeled sediment and river water data assumed a constant upstream boundary condition of 10 ng/L PCBs.

The exposure point concentration values used in the 1999 HHRA are based on Total PCBs. For this Responsiveness Summary, the Tri+ PCB concentrations from the RBMR were converted to total PCBs based on the ratio of Tri+ to Total (TAMS, 2000). PCB concentrations in sediment and river water were forecast in the revised RBMR through the year 2067. The EPCs were calculated by averaging the forecasted results over the corresponding exposure durations for adults, adolescents, and children.

Overall, revised sediment EPCs were two- to four-fold lower than the August 1999 EPCs. Revised river water EPCs were slightly higher than the 1999 BMR results by less than two-fold. Reasons for the projected sediment decrease and water increase are provided in the Ecological Responsiveness Summary for the Ecological Risk Assessment (USEPA, 2000).

1.3 Results

For fish consumption, the RME estimate of the increased risk of an individual developing cancer averaged over a lifetime based is 7×10^{-4}, or seven additional case of cancer in 10,000 exposed people. The central tendency (average) estimate of risk is 2×10^{-5}, or two additional cases of cancer in 100,000 exposed people. For known or suspected carcinogens, acceptable exposure levels for Superfund are generally concentration levels that represent an incremental upper bound lifetime cancer risk to an RME individual of between 10^{-4} and 10^{-6} (USEPA, 1990). The central tendency cancer risks are provided to more fully describe the health effects associated with average exposure (USEPA, 1995a).

Estimated cancer risks relating to PCB exposure in sediment and water while swimming or wading, or from inhalation of volatilized PCBs in air by residents living near the river, are lower than those for fish ingestion, falling generally at the low end, or below, the range of 10^{-4} to 10^{-6}. A summary of the point estimate cancer risk calculations is presented in Table 4.2.

Table 4.2 Revised Point Estimate Cancer Risk Summary

Pathway	Central Tendency Risk	RME Risk
Ingestion of Fish	2×10^{-5} (2 in 100,000)	7×10^{-4} (7 in 10,000)
Exposure to Sediment*	2×10^{-7} (2 in 10,000,000)	2×10^{-6} (2 in 1,000,000)
Exposure to Water*	2×10^{-8} (2 in 100,000,000)	2×10^{-7} (2 in 10,000,000)
Inhalation of Air*	2×10^{-8} (2 in 100,000,000)	1×10^{-6} (1 in 1,000,000)

**Total risk for child (aged 1-6), adolescent (aged 7-18), and adult (over 18).*

The evaluation of non-cancer health hazards involved comparing the average daily exposure levels (dose) to determine whether the estimated exposures exceed the Reference Dose (RfD). The ratio of the site-specific calculated dose to the RfD for each exposure pathway is summed to calculate the Hazard Index (HI) for the exposed individual. An HI of one (1) is the reference level established by USEPA above which concerns about non-cancer health effects must be evaluated.

Ingestion of fish resulted in the highest Hazard Indices, with an HI of 6 for the central tendency point estimate and an HI of 65 for the RME point estimate. The total HIs for exposure to sediment, water, and air are all below one. Non-cancer hazards due to inhalation of PCBs were not calculated

because IRIS does not contain a toxicity value for inhalation of PCBs. A summary of the point estimate non-cancer hazards is presented below.

Table 4.3 Revised Point Estimate Non-Cancer Hazard Summary		
Pathway	**Central Tendency Non-Cancer Hazard Index**	**RME Non-Cancer Hazard Index**
Ingestion of Fish	6	65
Exposure to Sediment*	0.03	0.04
Exposure to Water*	0.01	0.02
Inhalation of Air	Not Calculated	Not Calculated

**Values for child and adolescent, which are higher than adult for these pathways.*

1.4 Comparison/Discussion

The revised RME cancer risks for fish ingestion, the pathway with the highest risks, declined approximately 30%, to 7×10^{-4} compared to 1×10^{-3} in the 1999 HHRA. This decline in the risk assessment results does not alter the overall conclusions from the previous HHRA. These revised results indicate that cancer risks and non-cancer health hazards to the reasonably maximally exposed (RME) individual associated with ingestion of PCBs in fish from the Upper Hudson River are above USEPA levels of concern. In addition, these revised results indicate that fish ingestion represents the primary pathway for PCB exposure and for potential adverse health effects, whereas cancer risks and non-cancer hazards from other exposure pathways are at or below USEPA levels of concern.

References

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Limno-Tech, Inc., Menzie Cura & Associates, Inc. and Tetra-Tech, Inc. USEPA, Region II, New York, New York.

TABLE 2-1 (Unchanged)

SELECTION OF EXPOSURE PATHWAYS – Phase 2 Risk Assessment

UPPER HUDSON RIVER

Scenario Timeframe	Source Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/Future	Fish	Fish	Upper Hudson Fish	Angler	Adult Child **	Ingestion	On-Site	Quant	PCBs have been widely detected in fish
	Sediment	Sediment	Banks of Upper Hudson	Recreator	Adult	Ingestion	On-Site	Quant	Recreators may ingest or otherwise come in contact with contaminated river sediment while engaging in activities along the river.
						Dermal	On-Site	Quant	
					Adolescent	Ingestion	On-Site	Quant	
						Dermal	On-Site	Quant	
					Child	Ingestion	On-Site	Quant	
						Dermal	On-Site	Quant	
	River Water	Drinking Water	Upper Hudson River	Resident	Adult	Ingestion	On-Site	Quant	Considered in Phase 1 Risk Assessment and determined to have de minimis risk. Concentrations below the MCL does not pose a risk during occasional exposure, such as during swimming. Not evaluated further in this HHRA
					Adolescent	Ingestion	On-Site	Quant	
					Child	Ingestion	On-Site	Quant	
		River Water	Upper Hudson River (wading/swimming)	Recreator	Adult	Dermal	On-Site	Quant	Recreators may come in contact with contaminated river water while wading or swimming
					Adolescent	Dermal	On-Site	Quant	
					Child	Dermal	On-Site	Quant	
		Outdoor Air	Upper Hudson River (River and near vicinity)	Recreator	Adult	Inhalation	On-Site	Quant	Recreators may inhale volatilized PCBs while engaging in river-related activities
					Adolescent	Inhalation	On-Site	Quant	
					Child	Inhalation	On-Site	Quant	
				Resident	Adult	Inhalation	On-Site	Quant	Nearby residents may inhale volatilized PCBs outside of their home
					Adolescent	Inhalation	On-Site	Quant	
					Child	Inhalation	On-Site	Quant	
	Home-grown Crops	Vegetables	Upper Hudson vicinity	Resident	Adult	Ingestion	On-Site	Qual	Limited data; studies show low PCB uptake in forage crops
					Adolescent	Ingestion	On-Site	Qual	
					Child	Ingestion	On-Site	Qual	
	Beef	Beef	Upper Hudson vicinity	Resident	Adult	Ingestion	On-Site	Qual	Limited data; studies show non-detect PCB levels in cow's milk in NY
					Adolescent	Ingestion	On-Site	Qual	
					Child	Ingestion	On-Site	Qual	
	Dairy Products	Milk, eggs	Upper Hudson vicinity	Resident	Adult	Ingestion	On-Site	Qual	Limited data; studies show non-detect PCB levels in cow's milk in NY
					Adolescent	Ingestion	On-Site	Qual	
					Child	Ingestion	On-Site	Qual	

** Child angler considered in Monte Carlo analysis

TABLE 2-2 (Revised)
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
UPPER HUDSON RIVER - Fish

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Upper Hudson Fish

CAS Number	Chemical	Minimum Concentration (1)	Minimum Qualifier	Maximum Concentration (1)	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value	Screening Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection (2)
1336-36-3	PCBs (3)	0.09	N/A	8.0	N/A	mg/kg wet weight	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Yes	FD, TX, ASL

(1) Minimum/maximum modeled concentration between 1999-2067 (USEPA, 2000).

(2) Rationale Codes: Selection Reason: Infrequent Detection but Associated Historically (HIST)
Frequent Detection (FD)
Toxicity Information Available (TX)
Above Screening Levels (ASL)
Deletion Reason: Infrequent Detection (IFD)
Background Levels (BKG)
No Toxicity Information (NTX)
Essential Nutrient (NUT)
Below Screening Level (BSL)

(3) Occurrence and distribution of PCBs in fish were modeled, not measured (USEPA, 2000)

Definitions: N/A = Not Applicable
SQL = Sample Quantitation Limit
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
MCL = Federal Maximum Contaminant Level
SMCL = Secondary Maximum Contaminant Level
J = Estimated Value
C = Carcinogenic
N = Non-Carcinogenic

TABLE 2-3 (Revised)
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
UPPER HUDSON RIVER - Sediment

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson

CAS Number	Chemical	Minimum Concentration (1)	Minimum Qualifier	Maximum Concentration (1)	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value	Screening Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection (2)
1336-36-3	PCBs (3)	0.14	N/A	21.1	N/A	mg/kg	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Yes	FD, TX, ASL

(1) Minimum/maximum modeled concentration between 1999-2067 (USEPA, 2000).

(2) Rationale Codes: Selection Reason:
- Infrequent Detection but Associated Historically (HIST)
- Frequent Detection (FD)
- Toxicity Information Available (TX)
- Above Screening Levels (ASL)

Deletion Reason:
- Infrequent Detection (IFD)
- Background Levels (BKG)
- No Toxicity Information (NTX)
- Essential Nutrient (NUT)
- Below Screening Level (BSL)

(3) Occurrence and distribution of PCBs in sediment were modeled, not measured (USEPA, 2000).

Definitions:
- N/A = Not Applicable
- SQL = Sample Quantitation Limit
- COPC = Chemical of Potential Concern
- ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
- MCL = Federal Maximum Contaminant Level
- SMCL = Secondary Maximum Contaminant Level
- J = Estimated Value
- C = Carcinogenic
- N = Non-Carcinogenic

TABLE 2-4 (Revised)
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
UPPER HUDSON RIVER - River Water

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River

CAS Number	Chemical	Minimum Concentration (1)	Minimum Qualifier	Maximum Concentration (1)	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	Background Value	Screening Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for Contaminant Deletion or Selection (2)
1336-36-3	PCBs (3)	5.70E-06	N/A	8.80E-05	N/A	mg/L	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Yes	FD, TX, ASL

(1) Minimum/maximum modeled concentration between 1999-2067 (USEPA, 2000).

(2) Rationale Codes Selection Reason: Infrequent Detection but Associated Historically (HIST)
Frequent Detection (FD)
Toxicity Information Available (TX)
Above Screening Levels (ASL)
Deletion Reason: Infrequent Detection (IFD)
Background Levels (BKG)
No Toxicity Information (NTX)
Essential Nutrient (NUT)
Below Screening Level (BSL)

(3) Occurrence and distribution of PCBs in river water were modeled, not measured (USEPA, 2000).

Definitions: N/A = Not Applicable
SQL = Sample Quantitation Limit
COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
MCL = Federal Maximum Contaminant Level
SMCL = Secondary Maximum Contaminant Level
J = Estimated Value
C = Carcinogenic
N = Non-Carcinogenic

TABLE 2-5 (Revised)
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
UPPER HUDSON RIVER - Outdoor Air

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Water Vapor

CAS Number	Chemical	(1) Minimum Concentration	Minimum Qualifier	(1) Maximum Concentration	Maximum Qualifier	Units	Location of Maximum Concentration	Detection Frequency	Range of Detection Limits	Concentration Used for Screening	(2) Background Value	(3) Screening Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	(4) Rationale for Contaminant Deletion or Selection
1336-36-3	PCBs (5)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	Yes	FD, TX, ASL

(1) Minimum/maximum concentration

(2) N/A - Refer to supporting information for background discussion
Background values derived from statistical analysis. Follow Regional guidance and provide supporting information.

(3) Provide reference for screening toxicity value.

(4) Rationale Codes: Selection Reason:
- Infrequent Detection but Associated Historically (HIST)
- Frequent Detection (FD)
- Toxicity Information Available (TX)
- Above Screening Levels (ASL)

Deletion Reason:
- Infrequent Detection (IFD)
- Background Levels (BKG)
- No Toxicity Information (NTX)
- Essential Nutrient (NUT)
- Below Screening Level (BSL)

(5) Occurrence and distribution of PCBs in outdoor air is based on modeled river water concentrations, not measured (USEPA, 2000)

Definitions:
- N/A = Not Applicable
- SQL = Sample Quantitation Limit
- COPC = Chemical of Potential Concern
- ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
- MCL = Federal Maximum Contaminant Level
- SMCL = Secondary Maximum Contaminant Level
- J = Estimated Value
- C = Carcinogenic
- N = Non-Carcinogenic

TABLE 2-6 (Revised)
MEDIUM-SPECIFIC MODELED EXPOSURE POINT CONCENTRATION SUMMARY
UPPER HUDSON RIVER FISH - Thompson Island Pool

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Upper Hudson Fish - Thompson Island Pool

Chemical of Potential Concern	Units	Arithmetic Mean*	95% UCL of Normal Data	Maximum Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
PCBs												
In Brown Bullhead	mg/kg wet weight	1.7	**	7.2	N/A	mg/kg wet weight	2.3	Mean-N	Averaged over RME ED	4.6	Mean-N	Averaged over CT ED
In Largemouth Bass	mg/kg wet weight	2.5	**	8.0	N/A	mg/kg wet weight	3.1	Mean-N	Averaged over RME ED	5.0	Mean-N	Averaged over CT ED
In Yellow Perch	mg/kg wet weight	2.4	**	7.0	N/A	mg/kg wet weight	3.0	Mean-N	Averaged over RME ED	4.7	Mean-N	Averaged over CT ED
Species-weighted (1)	mg/kg wet weight	2.2	**	7.5	N/A	mg/kg wet weight	2.7	Mean-N	Averaged over RME ED	4.8	Mean-N	Averaged over CT ED
Species-weighted for chronic exposure (2)	mg/kg wet weight	2.2	**	7.5	N/A	mg/kg wet weight	5.5	Mean-N	Averaged over RME ED	4.8	Mean-N	Averaged over CT ED

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N)

* Mean is for 1999-2067 modeling time period. See text for discussion.

** 95% UCLM not calculated (see text)

ED = Exposure Duration

CT = Central Tendency

(1) PCB concentrations for each species were weighted based on species-group intake percentages (Connelly et al., 1992) and averaged over the central tendency exposure duration (12 years) to calculate the CT EPC, and over the RME exposure duration (40 years) to calculate the RME EPC for cancer risks.

(2) PCB concentrations for each species were weighted based on species-group intake percentages (Connelly et al., 1992) and averaged over the central tendency exposure duration (12 years) to calculate the CT EPC, and over the RME exposure duration (7 years) to calculate the RME EPC for non-cancer hazards.

TABLE 2 7 (Revised)
MEDIUM-SPECIFIC MODELED EXPOSURE POINT CONCENTRATION SUMMARY
UPPER HUDSON RIVER FISH - River Mile 168

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Upper Hudson Fish - River Mile 168

Chemical of Potential Concern	Units	Arithmetic Mean*	95% UCL of Normal Data	Maximum Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
PCBs												
in Brown Bullhead	mg/kg wet weight	1.1	**	4.2	N/A	mg/kg wet weight	1.4	Mean-N	Averaged over RME ED	2.4	Mean-N	Averaged over CT ED
in Largemouth Bass	mg/kg wet weight	0.63	**	2.4	N/A	mg/kg wet weight	0.80	Mean-N	Averaged over RME ED	1.4	Mean-N	Averaged over CT ED
in Yellow Perch	mg/kg wet weight	0.49	**	1.9	N/A	mg/kg wet weight	0.61	Mean-N	Averaged over RME ED	1.1	Mean-N	Averaged over CT ED
Species-weighted (1)	mg/kg wet weight	0.82	**	3.2	N/A	mg/kg wet weight	1.0	Mean-N	Averaged over RME ED	1.8	Mean-N	Averaged over CT ED
Species-weighted for chronic exposure (2)	mg/kg wet weight	0.82	**	3.2	N/A	mg/kg wet weight	2.2	Mean-N	Averaged over RME ED	1.8	Mean-N	Averaged over CT ED

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N)

* Mean is for 1999-2067 modeling time period. See text for discussion.

** 95% UCLM not calculated (see text).

ED = Exposure Duration

CT = Central Tendency

(1) PCB concentrations for each species were weighted based on species-group intake percentages (Connelly et al., 1992) and averaged over the central tendency exposure duration (12 years) to calculate the CT EPC, and over the RME exposure duration (40 years) to calculate the RME EPC for cancer risks.

(2) PCB concentrations for each species were weighted based on species-group intake percentages (Connelly et al., 1992) and averaged over the central tendency exposure duration (12 years) to calculate the CT EPC, and over the RME exposure duration (7 years) to calculate the RME EPC for non-cancer hazards.

TABLE 2-8 (Revised)
MEDIUM-SPECIFIC MODELED EXPOSURE POINT CONCENTRATION SUMMARY
UPPER HUDSON RIVER FISH - River Mile 154

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Upper Hudson Fish - River Mile 154

Chemical of Potential Concern	Units	Arithmetic Mean*	95% UCL of Normal Data	Maximum Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
PCBs												
in Brown Bullhead	mg/kg wet weight	0.27	**	1.1	N/A	mg/kg wet weight	0.35	Mean-N	Averaged over RME ED	0.68	Mean-N	Averaged over CT ED
in Largemouth Bass	mg/kg wet weight	0.26	**	1.1	N/A	mg/kg wet weight	0.34	Mean-N	Averaged over RME ED	0.65	Mean-N	Averaged over CT ED
in Yellow Perch	mg/kg wet weight	0.19	**	0.81	N/A	mg/kg wet weight	0.25	Mean-N	Averaged over RME ED	0.48	Mean-N	Averaged over CT ED
Species weighted (1)	mg/kg wet weight	0.26	**	1.1	N/A	mg/kg wet weight	0.34	Mean-N	Averaged over RME ED	0.65	Mean-N	Averaged over CT ED
Species weighted for chronic exposure (2)	mg/kg wet	0.26	**	1.1	N/A	mg/kg wet weight	0.79	Mean-N	Averaged over RME ED	0.65	Mean-N	Averaged over CT ED

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N).

* Mean is for 1999-2067 modeling time period. See text for discussion.

** 95% UCLM not calculated (see text).

ED = Exposure Duration

CT = Central Tendency

(1) PCB concentrations for each species were weighted based on species-group intake percentages (Connelly et al., 1992) and averaged over the central tendency exposure duration (12 years) to calculate the CT EPC, and over the RME exposure duration (40 years) to calculate the RME EPC for cancer risks.

(2) PCB concentrations for each species were weighted based on species-group intake percentages (Connelly et al., 1992) and averaged over the central tendency exposure duration (12 years) to calculate the CT EPC, and over the RME exposure duration (7 years) to calculate the RME EPC for non-cancer hazards.

TABLE 2-9 (Revised)
MEDIUM-SPECIFIC MODELED EXPOSURE POINT CONCENTRATION SUMMARY
UPPER HUDSON RIVER SEDIMENT

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson

Chemical of Potential Concern	Units	Arithmetic Mean (1)	95% UCL of Normal Data	Maximum Concentration (1)	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
PCBs	mg/kg	1.91	**	21	N/A	mg/kg						
Adult							3.8	Mean-N	Averaged over RME ED	7.0	Mean-N	Averaged over CT ED
Adolescent							5.4	Mean-N	Averaged over RME ED	7.7	Mean-N	Averaged over CT ED
Child							6.7	Mean-N	Averaged over RME ED	7.7	Mean-N	Averaged over CT ED

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N).

** Not applicable because sediment data was modeled, not measured (see text).

(1) Mean/maximum of modeled concentration 1999-2067 (USEPA, 2000).

TABLE 2-10 (Revised)
MEDIUM-SPECIFIC MODELED EXPOSURE POINT CONCENTRATION SUMMARY
UPPER HUDSON RIVER WATER

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River

Chemical of Potential Concern	Units	Arithmetic Mean (1)	95% UCL of Normal Data	Maximum Concentration (1)	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
PCBs	mg/L	2.0E-05	**	8.8E-05	N/A	mg/L						
Adult							2.9E-05	Mean-N	Averaged over RME ED	4.3E-05	Mean-N	Averaged over CT ED
Adolescent							3.6E-05	Mean-N	Averaged over RME ED	4.6E-05	Mean-N	Averaged over CT ED
Child							4.3E-05	Mean-N	Averaged over RME ED	4.6E-05	Mean-N	Averaged over CT ED

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N)

** Not applicable because river water data was modeled, not measured (see text).

(1) Mean/maximum of modeled concentration 1999-2067 (USEPA, 2000)

TABLE 2-11 (Unchanged)
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
UPPER HUDSON RIVER AIR

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
PCBs	mg/m³	--	--	--	N/A	mg/m³	1.7E-05	Used high-end empirical transfer coefficient estimate	High-end estimate	1.0E-06	Used midpoint between modeled concentration and empirical transfer coefficient estimate	Central estimate

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N).

-- Not applicable because outdoor air concentrations based on modeled river water concentrations (refer to Table A-2) and water to air transfer coefficient.

TABLE 2-12 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Upper Hudson Fish
Receptor Population: Angler
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	C_{fish}-C	PCB Concentration in Fish (Cancer)**	mg/kg wet weight	1.4	See Tables 2-6 through 2-8	2.4	See Tables 2-6 through 2-8	Average Daily Intake (mg/kg-day) =
	C_{fish}-NC	PCB Concentration in Fish (Non-cancer)**	mg/kg wet weight	2.8	See Tables 2-6 through 2-8	2.4	See Tables 2-6 through 2-8	$C_{fish} \times IR_{fish} \times (1 - Loss) \times FS \times EF \times ED \times CF \times 1/BW \times 1/AT$
	IR_{fish}	Ingestion Rate of Fish	grams/day	31.9	90th percentile value, based on 1991 NY Angler survey.	4.0	50th percentile value, based on 1991 NY Angler survey.	
	Loss	Cooking Loss	g/g	0	Assumes 100% PCBs remains in fish.	0.2	Assumes 20% PCBs in fish is lost through cooking.	
	FS	Fraction from Source	unitless	1	Assumes 100% fish ingested is from Upper Hudson.	1	Assumes 100% fish ingested is from Upper Hudson.	
	EF	Exposure Frequency	days/year	365	Fish ingestion rate already averaged over one year.	365	Fish ingestion rate already averaged over one year.	
	ED	Exposure Duration (Cancer)	years	40	95th percentile value, based on 1991 NY Angler and 1990 US Census data.	12	50th percentile value, based on 1991 NY Angler and 1990 US Census data.	
	ED	Exposure Duration (Noncancer)	years	7	see text	12	50th percentile value, based on 1991 NY Angler and 1990 US Census data.	
	CF	Conversion Factor	kg/g	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	70	Mean adult body weight, males and females (USEPA, 1989b).	70	Mean adult body weight, males and females (USEPA, 1989b).	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b).	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b).	
	AT-NC	Averaging Time (Noncancer)	days	2,555	ED (years) x 365 days/year.	4,380	ED (years) x 365 days/year.	

** Species-weighted PCB concentration averaged over river location

TABLE 2-13 (Revised)
VALUES USED FOR DAILY INTAKE CALCULATIONS
UPPER HUDSON RIVER SEDIMENT - Adult Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	$C_{sediment}$	Chemical Concentration in Sediment	mg/kg	3.8	See Table 2-9	7.0	See Table 2-9	Average Daily Intake (mg/kg-day) =
	$IR_{sediment}$	Ingestion Rate of Sediment	mg/day	50	Mean adult soil ingestion rate (USEPA, 1997f).	50	Mean adult soil ingestion rate (USEPA, 1997f)	$C_{sediment}$ x $IR_{sediment}$ x FS x EF x ED x CF x 1/BW x 1/AT
	FS	Fraction from Source	unitless	1	Assumes 100% sediment exposure is from Upper Hudson.	1	Assumes 100% sediment exposure is from Upper Hudson	
	EF	Exposure Frequency	days/year	13	1 day/week, 3 months/yr	7	Approximately 50% of RME	
	ED	Exposure Duration	years	23	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	5	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	kg/mg	1.00E-06		1.00E-06	--	
	BW	Body Weight	kg	70	Mean adult body weight, males and females (USEPA, 1989b).	70	Mean adult body weight, males and females (USEPA, 1989b)	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	8,395	ED (years) x 365 days/year	1,825	ED (years) x 365 days/year	
Dermal	$C_{sediment}$	Chemical Concentration in Sediment	mg/kg	3.8	See Table 2-9	7.0	See Table 2-9	Average Daily Intake (mg/kg-day) =
	DA	Dermal Absorption	unitless	0.14	Based on absorption of PCBs from soil in monkeys (Wester, 1993).	0.14	Based on absorption of PCBs from soil in monkeys (Wester, 1993).	$C_{sediment}$ x DA x AF x SA x EF x ED x CF x 1/BW x 1/AT
	AF	Adherance Factor	mg/cm²	0.3	50% value for adult (reed gatherer) - hands, lower legs, forearms, and face (USEPA, 1999f).	0.3	50% value for adult (reed gatherer) - hands, lower legs, forearms, and face (USEPA, 1999f)	
	SA	Surface Area	cm²/event	6,073	Ave male/female 50th percentile - hands, lower legs, forearms, feet, and face (USEPA, 1997f).	6,073	Ave male/female 50th percentile - hands, lower legs, forearms, feet, and face (USEPA, 1997f)	
	EF	Exposure Frequency	event/year	13	1 day/week, 3 months/yr	7	Approx. 50% of RME	
	ED	Exposure Duration	years	23	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	5	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	kg/mg	1.00E-06	--	1.00E-06	--	
	BW	Body Weight	kg	70	Mean adult body weight, males and females (USEPA, 1989b).	70	Mean adult body weight, males and females (USEPA, 1989b)	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	8,395	ED (years) x 365 days/year	1,825	ED (years) x 365 days/year	

TABLE 2-14 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER SEDIMENT - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	$C_{sediment}$	Chemical Concentration in Sediment	mg/kg	5.4	See Table 2-9	7.7	See Table 2-9	Average Daily Intake (mg/kg-day) =
	$IR_{sediment}$	Ingestion Rate of Sediment	mg/day	50	Mean soil ingestion rate (USEPA, 1997f)	50	Mean soil ingestion rate (USEPA, 1997f).	$C_{sediment}$ x $IR_{sediment}$ x FS x EF x ED x CF x 1/BW x 1/AT
	FS	Fraction from Source	unitless	1	Assumes 100% sediment exposure is from Upper Hudson	1	Assumes 100% sediment exposure is from Upper Hudson.	
	EF	Exposure Frequency	days/year	39	3 days/week, 3 months/yr	20	Approximately 50% of RME	
	ED	Exposure Duration	years	12	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	kg/mg	1.00E-06	-	1.00E-06	-	
	BW	Body Weight	kg	43	Mean adolescent body weight, males and females (USEPA, 1989b)	43	Mean adolescent body weight, males and females (USEPA, 1989b)	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	4,380	ED (years) x 365 days/year	1,095	ED (years) x 365 days/year	
Dermal	$C_{sediment}$	Chemical Concentration in Sediment	mg/kg	5.4	See Table 2-9	7.7	See Table 2-9	Average Daily Intake (mg/kg-day) =
	DA	Dermal Absorption	unitless	0.14	Based on absorption of PCBs from soil in monkeys (Wester, 1993)	0.14	Based on absorption of PCBs from soil in monkeys (Wester, 1993)	$C_{sediment}$ x DA x AF x SA x EF x ED x CF x 1/BW x 1/AT
	AF	Adherance Factor	mg/cm^2	0.25	Midpoint of adult and child AF: Hands, lower legs, forearms, and face (USEPA, 1999f)	0.25	Midpoint of adult and child AF: Hands, lower legs, forearms, and face (USEPA, 1999f).	
	SA	Surface Area	cm^2/event	4,263	Ave male/female 50th percentile age 12: hands, lower legs, forearms, feet, and face (USEPA, 1997f)	4,263	Ave male/female 50th percentile age 12: hands, lower legs, forearms, feet, and face (USEPA, 1997f).	
	EF	Exposure Frequency	event/year	39	3 days/week, 3 months/yr	20	Approximately 50% of RME	
	ED	Exposure Duration	years	12	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	kg/mg	1.00E-06	-	1.00E-06	-	
	BW	Body Weight	kg	43	Mean adolescent body weight, males and females (USEPA, 1989b)	43	Mean adolescent body weight, males and females (USEPA, 1989b)	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	4,380	ED (years) x 365 days/year	1,095	ED (years) x 365 days/year	

TABLE 2.15 (Revised)
VALUES USED FOR DAILY INTAKE CALCULATIONS
UPPER HUDSON RIVER SEDIMENT - Child Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Ingestion	$C_{sediment}$	Chemical Concentration in Sediment	mg/kg	6.7	See Table 2-9	7.7	See Table 2-9	Average Daily Intake (mg/kg-day) =
	$IR_{sediment}$	Ingestion Rate of Sediment	mg/day	100	Mean child soil ingestion rate (USEPA, 1997f).	100	Mean child soil ingestion rate (USEPA, 1997f)	$C_{sediment}$ x $IR_{sediment}$ x FS x EF x ED x CF x 1/BW x 1/AT
	FS	Fraction from Source	unitless	1	Assumes 100% sediment exposure is from Upper Hudson.	1	Assumes 100% sediment exposure is from Upper Hudson.	
	EF	Exposure Frequency	days/year	13	1 day/week, 3 months/yr	7	Approx. 50% of RME	
	ED	Exposure Duration	years	6	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	kg/mg	1.00E-06	--	1.00E-06	--	
	BW	Body Weight	kg	15	Mean child body weight, males and females (USEPA, 1989b).	15	Mean child body weight, males and females (USEPA, 1989b)	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70 year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	2,190	ED (years) x 365 days/year	1,095	ED (years) x 365 days/year	
Dermal	$C_{sediment}$	Chemical Concentration in Sediment	mg/kg	6.7	See Table 2-9	7.7	See Table 2-9	Average Daily Intake (mg/kg-day) =
	DA	Dermal Absorption	unitless	0.14	Based on absorption of PCBs from soil in monkeys (Wester, 1993)	0.14	Based on absorption of PCBs from soil in monkeys (Wester, 1993)	$C_{sediment}$ x DA x AF x SA x EF x ED x CF x 1/BW x 1/AT
	AF	Adherance Factor	mg/cm²	0.2	50% value for children (moist soil) - hands, lower legs, forearms, and face (USEPA, 1999f).	0.2	50% value for children (moist soil) - hands, lower legs, forearms, and face (USEPA, 1999f).	
	SA	Surface Area	cm²/event	2,792	50th percentile ave for male/female child age 6 hands, lower legs, forearms, feet, and face (USEPA, 1997f).	2,792	50th percentile ave for male/female child age 6 hands, lower legs, forearms, feet, and face (USEPA, 1997f).	
	EF	Exposure Frequency	event/year	13	1 day/week, 3 months/yr	7	Approx. 50% of RME	
	ED	Exposure Duration	years	6	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	kg/mg	1.00E-06	--	1.00E-06	--	
	BW	Body Weight	kg	15	Mean child body weight, males and females (USEPA, 1989b).	15	Mean child body weight, males and females (USEPA, 1989b)	
	AT-C	Averaging Time (Cancer)	days	25,550	70 year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	2,190	ED (years) x 365 days/year	1,095	ED (years) x 365 days/year	

TABLE 2-16 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Dermal	C_{water}	Chemical Concentration in River Water	mg/L	2.9E-05	See Table 2-10	4.3E-05	See Table 2-10	Average Daily Intake (mg/kg-day) =
	Kp	Dermal Permeability Constant (for PCBs)	cm/hour	0.48	Hexachlorobiphenyl (USEPA, 1999f)	0.48	Hexachlorobiphenyl (USEPA, 1999f)	C_{water} x Kp x SA x DE x EF x ED x CF x 1/BW x 1/AT
	SA	Surface Area	cm^2	18,150	Full body contact (USEPA, 1997f)	18,150	Full body contact (USEPA, 1997f)	
	DE	Dermal Exposure Time	hours/day	2.6	National average for swimming (USEPA, 1989b).	2.6	National average for swimming (USEPA, 1989b).	
	EF	Exposure Frequency	days/year	13	1 day/week, 3 months/yr	7	Approx. 50% of RME	
	ED	Exposure Duration	years	23	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	5	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	L/cm^3	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	70	Mean adult body weight, males and females (USEPA, 1989b).	70	Mean adult body weight, males and females (USEPA, 1989b).	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b).	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	8,395	ED (years) x 365 days/year.	1,825	ED (years) x 365 days/year.	

TABLE 2-17 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Dermal	C_{water}	Chemical Concentration in River Water	mg/L	3.6E-05	See Table 2-10	4.6E-05	See Table 2-10	Average Daily Intake (mg/kg-day) =
	Kp	Dermal Permeability Constant (for PCBs)	cm/hour	0.48	Hexachlorobiphenyl (USEPA, 1999f)	0.48	Hexachlorobiphenyl (USEPA, 1999f)	C_{water} x Kp x SA x DE x EF x ED x CF x 1/BW x 1/AT
	SA	Surface Area	cm^2	13,100	Full body contact (USEPA, 1997f)	13,100	Full body contact (USEPA, 1997f)	
	DE	Dermal Exposure Time	hours/day	2.6	National average for swimming (USEPA, 1989b).	2.6	National average for swimming (USEPA, 1989b).	
	EF	Exposure Frequency	days/year	39	3 days/week, 3 months/yr	20	Approx. 50% of RME	
	ED	Exposure Duration	years	12	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	L/cm^3	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	43	Mean adolescent body weight, males and females (USEPA, 1989b).	43	Mean adolescent body weight, males and females (USEPA, 1989b)	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b).	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	4,380	ED (years) x 365 days/year	1,095	ED (years) x 365 days/year.	

TABLE 2-18 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER WATER - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Dermal	C_{water}	Chemical Concentration in River Water	mg/L	4.3E-05	See Table 2-10	4.6E-05	See Table 2-10	Average Daily Intake (mg/kg-day) =
	Kp	Dermal Permeability Constant (for PCBs)	cm/hour	0.48	Hexachlorobiphenyl (USEPA, 1999f)	0.48	Hexachlorobiphenyl (USEPA, 1999f)	C_{water} x Kp x SA x DE x EF x ED x CF x 1/BW x 1/AT
	SA	Surface Area	cm^2	6,880	Full body contact (USEPA, 1997f)	6,880	Full body contact (USEPA, 1997f)	
	DE	Dermal Exposure Time	hours/day	2.6	National average for swimming (USEPA, 1989b).	2.6	National average for swimming (USEPA, 1989b).	
	EF	Exposure Frequency	days/year	13	1 day/week, 3 months/yr	7	Approx. 50% of RME	
	ED	Exposure Duration	years	6	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	L/cm^3	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	15	Mean child body weight, males and females (USEPA, 1989b).	15	Mean child body weight, males and females (USEPA, 1989b)	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	2,190	ED (years) x 365 days/year	1,095	ED (years) x 365 days/year	

TABLE 2-19 (Unchanged)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER AIR - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Inhalation	C_{air}	Chemical Concentration in Air	µg/m³	1.7E-02	See Table 2-11	1.0E-03	See Table 2-11	Average Daily Intake (mg/kg-day) =
	IR_{air}	Inhalation Rate of Air	m³/hour	1.6	Mean inhalation rate for adults during short-term, moderate activities (USEPA, 1997f).	1.6	Mean inhalation rate for adults during short-term, moderate activities (USEPA, 1997f).	$C_{air} \times IR_{air} \times DE \times EF \times ED \times CF \times 1/BW \times 1/AT$
	DE	Duration of Event	hours/day	4	Site-specific assumption	4	Site-specific assumption	
	EF	Exposure Frequency	days/year	13	1 day/week, 3 months/yr	7	Approx. 50% of RME	
	ED	Exposure Duration	years	23	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	5	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	mg/µg	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	70	Mean adult body weight, males and females (USEPA, 1989b).	70	Mean adult body weight, males and females (USEPA, 1989b).	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b).	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	8,395	ED (years) x 365 days/year	1,825	ED (years) x 365 days/year	

TABLE 2-20 (Unchanged)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER AIR - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Inhalation	C_{av}	Chemical Concentration in Air	µg/m³	1.7E-02	See Table 2-11	1.0E-03	See Table 2-11	Average Daily Intake (mg/kg-day) =
	IR_{av}	Inhalation Rate of Air	m³/hour	1.6	Mean inhalation rate for adults during short-term, moderate activities (USEPA, 1997f)	1.6	Mean inhalation rate for adults during short-term, moderate activities (USEPA, 1997f)	$C_{av} \times IR_{av} \times DE \times EF \times ED \times CF \times 1/BW \times 1/AT$
	DE	Duration of Event	hours/day	4	Site-specific assumption	4	Site-specific assumption	
	EF	Exposure Frequency	days/year	39	3 days/week, 3 months/yr	20	Approx. 50% of RME	
	ED	Exposure Duration	years	12	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	mg/µg	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	43	Mean adolescent body weight, males and females (USEPA, 1989b).	43	Mean adolescent body weight, males and females (USEPA, 1989b).	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	4,380	ED (years) x 365 days/year.	1,095	ED (years) x 365 days/year.	

TABLE 2-21 (Unchanged)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER AIR - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Inhalation	C_{air}	Chemical Concentration in Air	µg/m³	1.7E-02	See Table 2-11	1.0E-03	See Table 2-11	Average Daily Intake (mg/kg-day) =
	IR_{air}	Inhalation Rate of Air	m³/hour	1.2	Mean inhalation rate for children during short-term, moderate activities (USEPA, 1997f)	1.2	Mean inhalation rate for children during short-term, moderate activities (USEPA, 1997f)	$C_{air} \times IR_{air} \times DE \times EF \times ED \times CF \times 1/BW \times 1/AT$
	DE	Duration of Event	hours/day	4	Site-specific assumption	4	Site-specific assumption	
	EF	Exposure Frequency	days/year	13	1 day/week, 3 months/yr	7	Approx. 50% of RME	
	ED	Exposure Duration	years	6	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	mg/µg	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	15	Mean child body weight, males and females (USEPA, 1989b).	15	Mean child body weight, males and females (USEPA, 1989b).	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b).	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	2,190	ED (years) x 365 days/year.	1,095	ED (years) x 365 days/year	

TABLE 2-22 (Unchanged)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER AIR - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Inhalation	C_{air}	Chemical Concentration in Air	µg/m³	1.7E-02	See Table 2-11	1.0E-03	See Table 2-11	Average Daily Intake (mg/kg-day) =
	IR_{air}	Inhalation Rate of Air	m³/day	20	RME inhalation rate (USEPA, 1991b).	20	RME inhalation rate (USEPA, 1991b).	$C_{air} \times IR_{air} \times EF \times ED \times CF \times 1/BW \times 1/AT$
	EF	Exposure Frequency	days/year	350	USEPA (1991b)	350	USEPA (1991b)	
	ED	Exposure Duration	years	23	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	5	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	mg/µg	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	70	Mean adult body weight, males and females (USEPA, 1989b)	70	Mean adult body weight, males and females (USEPA, 1989b).	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b).	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b).	
	AT-NC	Averaging Time (Noncancer)	days	8,395	ED (years) x 365 days/year.	1,825	ED (years) x 365 days/year	

TABLE 2-23 (Unchanged)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER AIR - Adolescent Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Adolescent

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Inhalation	C_{air}	Chemical Concentration in Air	µg/m³	1.7E-02	See Table 2-11	1.0E-03	See Table 2-11	Average Daily Intake (mg/kg-day) =
	IR_{air}	Inhalation Rate of Air	m³/day	13.5	Mean long-term inhalation rate for adolescents, aged 12-14 (USEPA, 1997f).	13.5	Mean long-term inhalation rate for adolescents, aged 12-14 (USEPA, 1997f).	$C_{air} \times IR_{air} \times EF \times ED \times CF \times 1/BW \times 1/AT$
	EF	Exposure Frequency	days/year	350	USEPA (1991b)	350	USEPA (1991b)	
	ED	Exposure Duration	years	12	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	mg/µg	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	43	Mean adolescent body weight, males and females (USEPA, 1989b)	43	Mean adolescent body weight, males and females (USEPA, 1989b).	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b).	
	AT-NC	Averaging Time (Noncancer)	days	4,380	ED (years) x 365 days/year	1,095	ED (years) x 365 days/year	

TABLE 2-24 (Unchanged)

VALUES USED FOR DAILY INTAKE CALCULATIONS

UPPER HUDSON RIVER AIR - Child Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/ Reference	CT Value	CT Rationale/ Reference	Intake Equation/ Model Name
Inhalation	C_{air}	Chemical Concentration in Air	µg/m³	1.7E-02	See Table 2-11	1.0E-03	See Table 2-11	Average Daily Intake (mg/kg-day) =
	IR_{air}	Inhalation Rate of Air	m³/day	8.3	Mean long-term inhalation *rate for children aged* 3-5 years (USEPA, 1997f).	8.3	Mean long-term inhalation *rate for children aged* 3-5 years (USEPA, 1997f).	$C_{air} \times IR_{air} \times EF \times ED \times CF \times 1/BW \times 1/AT$
	EF	Exposure Frequency	days/year	350	USEPA (1991b)	350	USEPA (1991b)	
	ED	Exposure Duration	years	6	derived from 95th percentile of residence duration in 5 Upper Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Upper Hudson Counties (see text)	
	CF	Conversion Factor	mg/µg	1.00E-03	--	1.00E-03	--	
	BW	Body Weight	kg	15	Mean child body weight, males and females (USEPA, 1989b).	15	Mean child body weight, males and females (USEPA, 1989b).	
	AT-C	Averaging Time (Cancer)	days	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	25,550	70-year lifetime exposure x 365 d/yr (USEPA, 1989b)	
	AT-NC	Averaging Time (Noncancer)	days	2,190	ED (years) x 365 days/year	1,095	ED (years) x 365 days/year	

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Table 3-1 (Unchanged)
Summary of Fish Ingestion Rates
1991 New York Angler Survey [a]

Percentiles	Ingestion Rate (meals/yr)	Ingestion Rate (g/day)
10	1	0.62
20	2	1.2
30	3	1.9
40	5	3.1
50	6.4	4.0
60	10	6.2
70	15	9.3
80	28	17.4
90	51	31.9
95	102	63.4
98	292	182
99	393	244
Arith. Mean	28	17.3

Notes:

[a] *Distribution percentiles from the 1991 New York Angler Survey (Connelly et al., 1992)*

Table 3-2 (Unchanged)
Fish Ingestion Rate Summary for Several Surveys

Study	Average Daily Fish Consumption (g/day)	
	Central Estimate[a]	High End Estimate[b]
1991 New York angler survey (Connelly et al., 1992)		
All flowing waterbodies	4.0	31.9
EPA Exposure Factors Handbook (USEPA, 1997f)		
Recreational freshwater anglers	8	25
1993 Maine Angler Survey (Ebert et al., 1993)		
All flowing waterbodies		
Assuming fish shared with household	0.99	12
Assuming only angler consumes fish	2.5	27
1992 Lake Ontario Diary Study (Connelly et al., 1996)		
Sport-caught fish	2.2	17.9
Fish – all sources	14.1	42.3
1989 Michigan Survey (West et al., 1989 as cited in USEPA, 1997f)		
Recreational fish intake	10.9	38.7

Notes:

[a] *Central estimate represents mean intake for value from the EPA Exposure Factors Handbook (1997f), and 50th percentile values from all other studies listed.*

[b] *High end estimate is 90th percentile for 1991 New York Angler survey and 95th percentile for all others.*

Table 3-3 (Unchanged)
Summary of 1991 New York Angler Survey
Fish Consumption by Species Reported

Water Body Type/ Species Group	Number Reporting Eating Fish	Total Caught	Total Eaten	Average Number Eaten [b]	Standard Deviation [a]	Maximum Number Eaten	Percent of Hudson Species	Percent of All Fish
Flowing								
Bass	68	1,842	584	8.6	19.2	145	38%	14%
Walleye	36	333	134	3.7	4.2	20	9%	3%
Bullhead	23	1,092	558	24.3	61.9	300	36%	14%
Carp	2	[b]	90	45.0	42.4	75	6%	2%
Eel	4	38	38	9.5	10.6	25	2%	0.9%
Perch	17	833	139	8.2	12.5	51	9%	3%
Subtotal		4,138	1,543				100%	38%
Salmon	35	559	193	5.5	5.3	25		5%
Trout	130	3,099	1,230	9.5	15.7	133		30%
Catfish	11	158	113	10.3	15.5	50		3%
Other	45	2,871	1,025	22.8	50.1	200		25%
Total All Fish		10,825	4,104					100%
Not Flowing								
Bass	154	3,370	1,032	6.7	12.0	100	29%	14%
Walleye	112	2,292	1,054	9.4	14.2	75	30%	14%
Bullhead	53	1,200	634	12.0	21.5	100	18%	8%
Carp	4	7	29	7.3	6.7	14	0.8%	0.4%
Eel	2	2	3	1.5	0.7	2	0.1%	0.04%
Perch	51	2,289	816	16.0	32.4	200	23%	11%
Subtotal		9,160	3,568				100%	47%
Salmon	55	538	480	8.7	15.2	80		6%
Trout	152	2,428	1,400	9.2	18.3	150		18%
Catfish	10	46	46	4.6	6.9	20		0.6%
Other	94	5,976	2,125	22.6	58.1	403		28%
Total All Fish		18,148	7,619					100%
Not Reported								
Bass	128	4,006	1,110	8.7	17.0	100	42%	17%
Walleye	34	389	206	6.1	8.8	40	8%	3%
Bullhead	55	2,374	1,099	20.0	43.2	225	41%	16%
Carp	5	16	11	2.2	1.6	5	0.4%	0.2%
Eel	5	9	13	2.6	2.5	7	0.5%	0.2%
Perch	24	338	222	9.3	21.7	100	8%	3%
Subtotal		7,132	2,661				100%	40%
Salmon	14	139	120	8.6	7.3	20		2%
Trout	148	2,836	1,319	8.9	16.8	157		20%
Catfish	4	40	17	4.3	2.8	7		0.3%
Other	104	7,731	2,559	24.6	72.2	630		38%
Total All Fish		17,878	6,676					100%

Notes

[a] *Mean and Standard Deviation are over number of anglers reporting they ate particular species.*

[b] *Number caught not reported*

Modeled PCB concentration estimates are available for species in ***Bold***

Source: Connelly et al (1992)

Table 3-4 (Unchanged)
Species-Group Intake Percentages
Using 1991 New York Angler Survey Data

Group 1		Group 2		Group 3	
Brown bullhead	36%	Bass	38%	Perch	9%
Carp	6%	Walleye	9%		
Eel	2%				
Species Group Totals	44%		47%		9%

Table 3-5 (Unchanged)
Summary of PCB Losses from Fish due to Cooking

Study	Type of Fish	Location	Preparation Method	Cooking Method	Percent PCB Loss from Fish
Armbruster *et al*., 1987	Striped Bass	Long Island Sound, NY	trimmed, skin-off	Baked 31-40 minutes	21
				Broiled 15-20 minutes	11
				Pan-fried, about 10 minutes	15
				Microwaved, 5-10 minutes	19
				Poached, 5-10 minutes	12
				Boiled, 10-20 minutes	(+4%)
Armbruster *et al*., 1989	Bluefish	Long Island Sound	trimmed, skin-off	various	8
Moya *et al*., 1998	Winter Flounder		filleted and sectioned	Deep fried - 1 minute	48
				Pan fried - 1 min/side	(+15%)
				Broiled - 2 minutes	(+17%)
Puffer and Gossett, 1983	White Croaker	Orange County, CA	trimmed, skin-off	Pan Fried	28
		Santa Monica, CA	trimmed, skin-off		65
Salama *et al*., 1998	Bluefish	Massachusetts	filleted	Smoked	65
				Microwaved	60
				Charbroiled (skin on)	47
				Charbroiled (skin off)	37
				Pan-fried	27
				Baked	39
Schecter *et al*., 1998	Catfish	New York	filleted	Broiled - approx 30 minutes	47
Skea *et al*., 1979	Smallmouth Bass	Lake Ontario	trimmed	Deep-fried for 3-4 minutes	74
			untrimmed	Baked	16
	Brown Trout		untrimmed	Smoked	27
			trimmed	Broiled for 15 minutes	0
Smith *et al*., 1973	Chinook Salmon	Lake Michigan	cleaned steaks	Baked or Poached	2-8
			cleaned steaks	Baked-in-Bag	11-16
Zabik *et al*., 1979	Lake trout		trimmed, skin-off	Broiled	53
			trimmed, skin-off	Baked	34
			trimmed, skin-off	Microwaved	26
			trimmed, skin-off	Baked	50
			trimmed, skin-on	Baked	40

Table 3-5 (Unchanged) (cont.)
Summary of PCB Losses from Fish due to Cooking

Study	Type of Fish	Location	Preparation Method	Cooking Method	Percent PCB Loss from Fish
Zabik *et al.*, 1995a	Chinook Salmon	Lakes Huron/Michigan	trimmed, skin-on	Baked	37
		Lakes Huron/Michigan	trimmed, skin- off	Baked	37
		Lakes Huron/Michigan	trimmed, skin-on	Charbroiled	45
		Lakes Huron/Michigan	trimmed, skin- off	Charbroiled	48
	Carp	Lakes Erie and Huron	trimmed, skin-on	Pan-fried	31
		Lakes Erie and Huron	trimmed, skin- off	Pan-fried	32
		Lakes Erie and Huron	trimmed, skin-on	Deep-fried	32
		Lakes Erie and Huron	trimmed, skin-off	Deep-fried	26
		Lake Erie	trimmed, skin-on or off	Deep fried or Pan fried	22
		Lake Huron	trimmed, skin-on or off	Deep fried or Pan fried	44
Zabik *et al.*, 1995b	Walleye	Lakes Erie, Huron and Michigan	filleted - skin on	Baked	19
			filleted - skin on	Charbroiled	25
		Lake Erie	filleted - skin on	Baked or Charbroiled	17
		Lake Huron	filleted - skin on	Baked or Charbroiled	24
		Lake Michigan	filleted - skin on	Baked or Charbroiled	25
	White Bass	Lake Erie	filleted - skin on	Pan fried	18
		Lake Huron	filleted - skin on	Pan fried	44
Zabik *et al.*, 1996	Lake Trout (lean)	Lakes Huron, Michigan and Ontario	filleted - skin off	Baked	13
			filleted - skin off	Charbroiled	11
		Lake Michigan	filleted - skin off	Baked	10
			filleted - skin off	Charbroiled	7
			filleted - skin off	Saltboiled	10
			filleted - skin on	Smoked	41
	Fat Trout (Siscowets)	Lake Superior	filleted - skin off	Baked	18
			filleted - skin off	Charbroiled	32
			filleted - skin off	Saltboiled	19
		Lake Huron	filleted - skin on	Smoked	37

Note: PCB losses for Armbuster (1987) and Zabik et al. (1995a, b, and 1996) were calculated from values in the studies for mass of PCB in fish before and after cooking.

Table 3-6 (Unchanged)
Joint Distribution Over Current Age and Age at Which Individual Started Fishing

Age		Fraction of Individuals Among	
Started Fishing	Now	All Anglers Currently Living in the Upper Hudson Region	Individuals in the Upper Hudson Region Who Started Fishing Recently
10	10	16.8%	72.3%
	20	16.8%	
	30	16.8%	
	40	16.8%	
	50	8.6%	
	60	5.5%	
	70	0.9%	
	80	0.2%	
20	20	2.6%	11.2%
	30	2.6%	
	40	2.5%	
	50	0.8%	
	60	0.7%	
	70	0.3%	
	80	0.1%	
30	30	1.9%	8.3%
	40	1.9%	
	50	0.6%	
	60	0.2%	
	70	0.1%	
	80	0.0%	
40	40	1.3%	5.5%
	50	0.6%	
	60	0.3%	
	70	0.1%	
	80	0.0%	
50	50	0.4%	1.8%
	60	0.4%	
	70	0.0%	
	80	0.0%	
60	60	0.2%	0.7%
	70	0.1%	
	80	0.0%	
70	70	0.0%	0.1%
	80	0.0%	
80	80	0.0%	0.1%

Source: 1991 New York Angler Survey, (Connelly, et al., 1992).

Table 3-7 (Unchanged)
Time Until Individual Stops Fishing

Age		Probability that Individual Will Stop Fishing in Exactly This Many Years						
Started Fishing	Now	10	20	30	40	50	60	70
10	10	0%	0%	0%	48%	19%	27%	6%
	20	0%	0%	48%	19%	27%	6%	
	30	0%	48%	19%	27%	6%		
	40	48%	19%	27%	6%			
	50	36%	53%	11%				
	60	83%	17%					
	70	100%						
20	20	0%	4%	64%	4%	17%	10%	
	30	4%	64%	4%	17%	10%		
	40	67%	5%	18%	10%			
	50	14%	55%	31%				
	60	64%	36%					
	70	100%						
30	30	0%	69%	19%	9%	3%		
	40	69%	19%	9%	3%			
	50	62%	29%	10%				
	60	75%	25%					
	70	100%						
40	40	53%	20%	22%	4%			
	50	43%	48%	10%				
	60	83%	17%					
	70	100%						
50	50	0%	93%	7%				
	60	93%	7%					
	70	100%						
60	60	67%	33%					
	70	100%						
70	70	100%						

Source: 1991 New York Angler Survey, (Connelly, et al., 1992).

Table 3-8 (Unchanged)
County-to-County In-Migration Data for Albany County, NY

	No Move	Move In										Total from Outside Region[a]
		Total	From Abroad	Domestic								
				Total	Outside Region[a]	Inside Region[a]						
						Total	From					
Age Group							Albany	Rensselaer	Saratoga	Warren	Washington	
5 to 9	8,638	9,002	228	8,774	2,111	6,663	5,795	536	262	18	52	2,339
10 to 14	10,128	6,482	226	6,256	1,604	4,652	4,253	304	86	0	9	1,830
15 to 19	11,284	9,642	236	9,406	4,958	4,448	3,713	428	177	61	69	5,194
20 to 24	8,012	19,788	428	19,360	11,187	8,173	6,188	995	705	165	120	11,615
25 to 29	5,515	18,568	640	17,928	6,825	11,103	9,111	1366	526	83	17	7,465
30 to 34	8,196	17,658	558	17,100	5,388	11,712	10,256	840	558	23	35	5,946
35 to 44	24,243	20,419	407	20,012	5,818	14,194	12,533	980	592	53	36	6,225
45 to 54	20,091	7,999	277	7,722	2,185	5,537	4,866	458	208	5	0	2,462
55 to 64	20,764	4,837	97	4,740	1,225	3,515	3,099	222	170	24	0	1,322
65 to 74	19,380	4,189	78	4,111	982	3,129	2,867	179	74	0	9	1,060
75 to 84	10,929	2,914	22	2,892	644	2,248	1,984	190	49	0	25	666
85+	3,670	1,746	0	1,746	355	1,391	1,227	117	41	0	6	355

Notes

a. The Upper Hudson Region consists of Albany, Rensselaer, Saratoga, Warren, and Washington Counties.

Source: 1990 U.S. Census.

Table 3-9 (Unchanged)
County-to-County In-Migration Data for Rensselaer County, NY

	No Move	Move In										Total from Outside Region[a]
		Total	From Abroad	Domestic								
				Total	Outside Region[a]	Inside Region[a]						
						Total	From					
Age Group							Albany	Rensselaer	Saratoga	Warren	Washington	
5 to 9	5,577	4,769	80	4,689	965	3,724	656	2,902	131	0	35	1,045
10 to 14	6,155	3,608	73	3,535	686	2,849	438	2,283	101	0	27	759
15 to 19	6,820	5,126	213	4,913	2,301	2,612	368	2,084	128	14	18	2,514
20 to 24	4,911	8,940	436	8,504	3,670	4,834	776	3,777	215	21	45	4,106
25 to 29	3,763	8,867	435	8,432	2,144	6,288	1211	4,713	295	18	51	2,579
30 to 34	5,236	7,976	221	7,755	1,935	5,820	1419	4,076	273	37	15	2,156
35 to 44	14,632	9,049	130	8,919	1,994	6,925	1503	5,030	297	20	75	2,124
45 to 54	10,930	3,214	40	3,174	599	2,575	495	1,951	85	13	31	639
55 to 64	11,355	2,125	46	2,079	482	1,597	264	1,303	24	0	6	528
65 to 74	10,010	1,712	5	1,707	320	1,387	216	1,101	62	0	8	325
75 to 84	5,613	1,146	7	1,139	154	985	205	730	41	6	3	161
85+	1,522	520	0	520	99	421	75	328	12	0	6	99

Notes:

a *The Upper Hudson Region consists of Albany, Rensselaer, Saratoga, Warren, and Washington Counties.*

Source: 1990 U.S. Census.

Table 3-10 (Unchanged)
County-to-County In-Migration Data for Saratoga County, NY

Age Group	No Move	Move In										Total from Outside Region[a]
		Total	From Abroad	Domestic								
				Total	Outside Region[a]	Inside Region[a]						
						Total	From Albany	From Rensselaer	From Saratoga	From Warren	From Washington	
5 to 9	3,149	5,752	80	5,672	675	4,997	474	293	3,885	198	147	755
10 to 14	2,652	3,728	73	3,655	611	3,044	287	140	2,403	119	95	684
15 to 19	2,155	6,006	213	5,793	2,305	3,488	185	171	2,964	113	55	2,518
20 to 24	3,303	9,955	436	9,519	3,685	5,834	443	229	4,792	229	141	4,121
25 to 29	4,791	12,284	435	11,849	1,203	10,646	1230	580	8,130	413	293	1,638
30 to 34	4,614	10,539	221	10,318	1,372	8,946	1375	419	6,639	342	171	1,593
35 to 44	6,540	11,469	130	11,339	1,478	9,861	1179	622	7,450	381	229	1,608
45 to 54	2,804	4,089	40	4,049	484	3,565	426	111	2,826	112	90	524
55 to 64	1,558	2,452	46	2,406	228	2,178	347	53	1,630	75	73	274
65 to 74	978	1,868	5	1,863	228	1,635	187	35	1,257	103	53	233
75 to 84	577	997	7	990	235	755	52	34	581	50	38	242
85+	248	506	0	506	100	406	57	6	314	14	15	100

Notes:

a *The Upper Hudson Region consists of Albany, Rensselaer, Saratoga, Warren, and Washington Counties.*

Source: 1990 U.S. Census.

Table 3-11 (Unchanged)
County-to-County In-Migration Data for Warren County, NY

	No Move	Move In										Total from Outside Region[a]
		Total	From Abroad	Domestic								
				Total	Outside Region[a]	Inside Region[a]						
						Total	From					
Age Group							Albany	Rensselaer	Saratoga	Warren	Washington	
5 to 9	1,760	2,429	44	2,385	680	1,705	35	0	184	1,333	153	724
10 to 14	2,109	1,879	32	1,847	482	1,365	19	33	180	1,020	113	514
15 to 19	2,646	1,765	32	1,733	671	1,062	6	20	136	828	72	703
20 to 24	1,550	2,538	57	2,481	611	1,870	13	2	155	1,479	221	668
25 to 29	1,187	3,392	30	3,362	1,136	2,226	97	19	223	1,637	250	1,166
30 to 34	1,635	3,247	47	3,200	967	2,233	113	0	190	1,757	173	1,014
35 to 44	4,833	4,111	83	4,028	1,215	2,813	42	48	326	2,153	244	1,298
45 to 54	4,521	1,700	31	1,669	571	1,098	13	14	93	878	100	602
55 to 64	4,078	1,263	10	1,253	527	726	45	8	71	507	95	537
65 to 74	3,709	1,128	17	1,111	429	682	3	12	81	540	46	446
75 to 84	2,149	540	0	540	144	396	7	0	57	313	19	144
85+	677	348	0	348	75	273	0	0	39	208	26	75

Notes:

a *The Upper Hudson Region consists of Albany, Rensselaer, Saratoga, Warren, and Washington Counties.*

Source: 1990 U.S. Census.

Table 3-12 (Unchanged)
County-to-County In-Migration Data for Washington County, NY

	No Move	Move In										Total from Outside Region[a]
		Total	From Abroad	Domestic								
				Total	Outside Region[a]	Inside Region[a]						
						Total	From					
Age Group							Albany	Rensselaer	Saratoga	Warren	Washington	
5 to 9	2,438	1,878	3	1,875	483	1,392	14	48	148	193	989	486
10 to 14	2,544	1,541	0	1,541	442	1,099	8	34	92	162	803	442
15 to 19	2,756	1,483	30	1,453	372	1,081	0	26	83	99	873	402
20 to 24	1,731	2,638	12	2,626	824	1,802	6	58	148	187	1403	836
25 to 29	1,464	3,595	32	3,563	1,336	2,227	96	70	133	324	1604	1,368
30 to 34	2,093	3,159	68	3,091	1,161	1,930	75	77	267	265	1246	1,229
35 to 44	5,534	3,233	6	3,227	1,118	2,109	45	80	227	355	1402	1,124
45 to 54	4,350	1,538	2	1,536	432	1,104	21	49	132	134	768	434
55 to 64	4,313	953	2	951	285	666	3	25	74	116	448	287
65 to 74	3,824	749	0	749	254	495	2	25	40	47	381	254
75 to 84	1,822	492	2	490	112	378	0	6	47	54	271	114
85+	656	228	0	228	90	138	0	0	26	26	86	90

Notes:

a. The Upper Hudson Region consists of Albany, Rensselaer, Saratoga, Warren, and Washington Counties.

Source: 1990 U.S. Census.

Table 3-13 (Unchanged)
County-to-County In-Migration Data for The Upper Hudson Region[a]

Age Group	No Move	Move In										Total from Outside Region[a]
		Total	From Abroad	Domestic								
				Total	Outside Region[a]	Inside Region[a]						
						Total	From					
							Albany	Rensselaer	Saratoga	Warren	Washington	
5 to 9	21,562	23,830	435	23,395	4,914	18,481	6,974	3,779	4,610	1,742	1,376	5,349
10 to 14	23,588	17,238	404	16,834	3,825	13,009	5,005	2,794	2,862	1,301	1,047	4,229
15 to 19	25,661	24,022	724	23,298	10,607	12,691	4,272	2,729	3,488	1,115	1,087	11,331
20 to 24	19,507	43,859	1,369	42,490	19,977	22,513	7,426	5,061	6,015	2,081	1,930	21,346
25 to 29	16,720	46,706	1,572	45,134	12,644	32,490	11,745	6,748	9,307	2,475	2,215	14,216
30 to 34	21,774	42,579	1,115	41,464	10,823	30,641	13,238	5,412	7,927	2,424	1,640	11,938
35 to 44	55,782	48,281	756	47,525	11,623	35,902	15,302	6,760	8,892	2,962	1,986	12,379
45 to 54	42,696	18,540	390	18,150	4,271	13,879	5,821	2,583	3,344	1,142	989	4,661
55 to 64	42,068	11,630	201	11,429	2,747	8,682	3,758	1,611	1,969	722	622	2,948
65 to 74	37,901	9,646	105	9,541	2,213	7,328	3,275	1,352	1,514	690	497	2,318
75 to 84	21,090	6,089	38	6,051	1,289	4,762	2,248	960	775	423	356	1,327
85+	6,773	3,348	0	3,348	719	2,629	1,359	451	432	248	139	719

Notes:

a. The Upper Hudson Region consists of Albany, Rensselaer, Saratoga, Warren, and Washington Counties.

Source: 1990 U.S. Census.

Table 3-14 (Unchanged)
Computation of 1-Year Move Probabilities for the Upper Hudson Region

Age Group (k)		$In_{1985\text{-}90,k}$ [a]	$Start_{1985\text{-}90,k}$ [b]	$Start_{1985\text{-}90,k+1}$ [c]	$Out_{1985\text{-}90,k}$ [d]	Probability of Moving in a 5-year Period [e]	$p_{k,1}$ [f]
5 to 9	(1)	5,349	21,562	23,588	3,323	12.3%	2.5%
10 to 14	(2)	4,229	23,588	25,661	2,156	7.8%	1.6%
15 to 19	(3)	11,331	25,661	19,507	17,485	47.3%	9.5%
20 to 24	(4)	21,346	19,507	16,720	24,133	59.1%	11.8%
25 to 29	(5)	14,216	16,720	21,774	9,162	29.6%	5.9%
30 to 34	(6)	11,938	21,774	27,891 [g]	5,821	17.3%	3.5%
35 to 44	(7)	12,379	55,782	42,696	25,465	37.4%	7.5%
45 to 54	(8)	4,661	42,696	42,068	5,289	11.2%	2.2%
55 to 64	(9)	2,948	42,068	37,901	7,115	15.8%	3.2%
65 to 74	(10)	2,318	37,901	21,090	19,129	47.6%	9.5%
75 to 84	(11)	1,327	21,090	6,773	15,644	69.8%	14.0%
85+	(12)	719	6,773	NA [h]	7,492		100% [i]

Notes:

a *Taken from the column labeled, "Total from Outside Region" in Table 3-13.*

b *Taken from the column labeled, "No Move" in Table 3-13.*

c *Set equal to the value of $Start_{1985\text{-}90,k}$ in the preceding row.*

d $Out_{1985\text{-}90,k} = (Start_{1985\text{-}90,k} - Start_{1985\text{-}90,k+1}) + In_{1985\text{-}90,k}$

e *Set equal to* $\dfrac{Out_{1985-90,k}}{Start_{1985-90,k} + In_{1985-90,k}}$.

f *Set equal to 1/5 × the probability of moving in a 5-year period.*

g *The value in this cell is 1/2 the value listed for $Start_{1985\text{-}90,7}$ to make $Start_{1985\text{-}90,6}$ and $Start_{1985\text{-}90,7}$ comparable. The adjustment addresses the fact that Age Group 7 represents 10 years (ages 35 to 44), whereas Age Group 6 represents 5 years (ages 30 to 34).*

h *Since Age Group 12 (ages 85+) is the last age group, there is no value for $Start_{1985\text{-}90,13}$.*

i *Assumes no exposure after age 85. This assumption has no effect on the estimated risk since it is assumed that individuals stop fishing by age 80.*

Table 3-15 (Unchanged)
Annual Probability That Individual Will Leave Region[a]

Current Age	Annual Probability of Leaving Upper Hudson Region
10-14	1.6%
15-19	9.5%
20-24	11.8%
25-29	5.9%
30-34	3.5%
35-44	7.5%
45-54	2.2%
55-64	3.2%
65-74	9.5%
75-84	14.0%
85+	100%

Notes:

a. From $P_{k,l}$ in Table 3-14.

Table 3-16 (Unchanged)
Age-Specific Body Weight Distributions

Age (Years)	Gender	Body Weight (kg)			
		Arithmetic Mean[a]	Arithmetic Std Deviation[a]	Geometric Mean	Geometric Standard Deviation
1	both	11.8	1.4	11.72	1.13
2	both	13.6	1.6	13.51	1.12
3	both	15.7	1.7	15.61	1.11
4	both	17.8	2.3	17.65	1.14
5	both	20.1	2.8	19.91	1.15
6	both	23.1	3.5	22.84	1.16
7	both	25.1	3.8	24.82	1.16
8	both	28.4	5.2	27.94	1.20
9	both	31.3	5.0	30.91	1.17
10	both	37.0	7.5	36.26	1.22
11	both	41.3	10.5	40.03	1.28
12	both	44.9	10.0	43.83	1.25
13	both	49.5	10.5	48.42	1.23
14	both	56.6	10.3	55.69	1.20
15	both	60.5	9.7	59.74	1.17
16	both	67.7	11.6	66.73	1.19
17	both	67.0	11.5	66.03	1.19
>18	both	71.0	15.9	69.28	1.25
>18	male	78.7	13.5	77.57	1.19
>18	female	65.4	15.3	63.68	1.26

Notes:

a. Source: Finley et al. (1994), Table 2.

TABLE 4-1 (Unchanged)

NON-CANCER TOXICITY DATA -- ORAL/DERMAL

UPPER HUDSON RIVER

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD Value	Oral RfD Units	Oral to Dermal Adjustment Factor	Adjusted Dermal RfD	Units	Primary Target Organ	Combined Uncertainty/Modifying Factors	Sources of RfD: Target Organ	Dates of RfD: Target Organ (1) (MM/DD/YY)
Aroclor 1254	Chronic	2.00E-05 (2)	mg/kg-d	--	--	--	LOAEL	300	IRIS	6/1/97
Aroclor 1016		7.00E-05 (3)	mg/kg-d	--	--	--	NOAEL	100	IRIS	6/1/97

N/A = Not Applicable

(1) IRIS value from most recent updated PCB file.

(2) Oral RfD for Aroclor 1254; there is no RfD available for total PCBs. PCBs in fish are considered to be most like Aroclor 1254.

(3) Oral RfD for Aroclor 1016; there is no RfD available for total PCBs. PCBs in sediment and water samples are considered to be most like Aroclor 1016.

TABLE 4-2 (Unchanged)

NON-CANCER TOXICITY DATA -- INHALATION

UPPER HUDSON RIVER

Chemical of Potential Concern	Chronic/ Subchronic	Value Inhalation RfC	Units	Adjusted Inhalation RfD	Units	Primary Target Organ	Combined Uncertainty/Modifyin Factors	Sources of RfC:RfD: Target Organ	Dates (1) (MM/DD/YY)
PCBs	N/A	N/A	N/A	N/A	N/A	N/A	N/A	IRIS	6/1/97

N/A = Not Applicable

(1) Most recent updated PCB file in IRIS and HEAST (1997) were reviewed.

TABLE 4-3 (Unchanged)

CANCER TOXICITY DATA -- ORAL/DERMAL

UPPER HUDSON RIVER

Chemical of Potential Concern	Oral Cancer Slope Factor	Oral to Dermal Adjustment Factor	Adjusted Dermal Cancer Slope Factor	Units	Weight of Evidence/ Cancer Guideline Description	Source Target Organ	Date (1) (MM/DD/YY)
PCBs	1 (2)	--	--	$(mg/kg\text{-}d)^{-1}$	B2	IRIS	6/1/97
	2 (3)	--	--	$(mg/kg\text{-}d)^{-1}$	B2	IRIS	6/1/97
	0.3 (4)	--	--	$(mg/kg\text{-}d)^{-1}$	B2	IRIS	6/1/97
	0.4 (5)	--	--	$(mg/kg\text{-}d)^{-1}$	B2	IRIS	6/1/97

IRIS = Integrated Risk Information System

HEAST= Health Effects Assessment Summary Tables

EPA Group:

A - Human carcinogen

B1 - Probable human carcinogen - indicates that limited human data are available

B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans

C - Possible human carcinogen

D - Not classifiable as a human carcinogen

E - Evidence of noncarcinogenicity

Weight of Evidence:

Known/Likely

Cannot be Determined

Not Likely

(1) IRIS value from most recent updated PCB file.

(2) Central estimate slope factor for exposures to PCBs via ingestion of fish, ingestion of sediments, and dermal contact (if dermal absorption fraction is applied) with sediments.

(3) Upper-bound slope factor for exposures to PCBs via ingestion of fish, ingestion of sediments, and dermal contact (if dermal absorption fraction is applied) with sediments.

(4) Central estimate slope factor for exposures to PCBs via dermal contact (if no absorption factor is applied) with water soluble congeners in river water and inhalation of evaporated congeners in air.

(5) Upper-bound slope factor for exposures to PCBs via dermal contact (if no absorption factor is applied) with water soluble congeners in river water and inhalation of evaporated congeners in air.

TABLE 4-4 (Unchanged)

CANCER TOXICITY DATA -- INHALATION

UPPER HUDSON RIVER

Chemical of Potential Concern	Unit Risk	Units	Adjustment	Inhalation Cancer Slope Factor	Units	Weight of Evidence/ Cancer Guideline Description	Source	Date (1) (MM/DD/YY)
PCBs	N/A	N/A	--	0.3 (2)	$(mg/kg-d)^{-1}$	B2	IRIS	6/1/97
	N/A	N/A	--	0.4 (3)	$(mg/kg-d)^{-1}$	B2	IRIS	6/1/97

IRIS = Integrated Risk Information System

HEAST= Health Effects Assessment Summary Tables

Weight of Evidence

Known/Likely

Cannot be Determined

Not Likely

EPA Group:

A - Human carcinogen

B1 - Probable human carcinogen - indicates that limited human data are available

B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans

C - Possible human carcinogen

D - Not classifiable as a human carcinogen

E - Evidence of noncarcinogenicity

(1) IRIS value from most recent updated PCB file.

(2) Central estimate slope factor for exposures to PCBs via dermal contact (if no absorption factor is applied) with river water and inhalation of air.

(3) Upper-bound slope factor for exposures to PCBs via dermal contact (if no absorption factor is applied) with river water and inhalation of air.

Table 4-5 (Unchanged)
Toxic Equivalency Factors (TEFs) for Dioxin-Like PCBs

IUPAC Number	Structure	1994 WHO/IPCS TEFs (Ahlborg *et al.*, 1994)	1998 WHO/IPCS TEFs (Van den Berg *et al.*, 1998)
Non-ortho PCBs			
77	3,3',4,4'-TCB	0.0005	0.0001
81	3,4,4',5-TCB	Not evaluated	0.0001
126	3,3',4,4',5-PeCB	0.1	0.1
169	3,3',4,4',5,5'-HxCB	0.01	0.01
Mono-ortho PCBs			
105	2,3,3',4,4'-PeCB	0.0001	0.0001
114	2,3,4,4',5-PeCB	0.0005	0.0005
118	2,3',4,4',5-PeCB	0.0001	0.0001
123	2',3,4,4',5-PeCB	0.0001	0.0001
156	2,3,3',4,4',5-HxCB	0.0005	0.0005
157	2,3,3',4,4',5'-HxCB	0.0005	0.0005
167	2,3',4,4',5,5'-HxCB	0.00001	0.00001
189	2,3,3',4,4',5,5'-HpCB	0.0001	0.0001
Diortho PCBs			
170	2,2',3,3',4,4',5-HpCB	0.0001	Withdrawn
180	2,2',3,4,4',5,5'-HpCB	0.00001	Withdrawn

TABLE 5-1-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Upper Hudson Fish
Receptor Population: Angler
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	PCBs	2.8	mg/kg wt weight	2.8	mg/kg wt weight	M	1.3E-03	mg/kg-day	2.0E-05	mg/kg-day	N/A	N/A	65
												Total Hazard Index Across All Exposure Routes/Pathways	65

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-1-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Upper Hudson Fish
Receptor Population: Angler
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	PCBs	2.4	mg/kg wt weight	2.4	mg/kg wt weight	M	1.1E-04	mg/kg-day	2.0E-05	mg/kg-day	N/A	N/A	6
												Total Hazard Index Across All Exposure Routes/Pathways	6

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-2-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER SEDIMENT- Adult Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	PCBs	3.8	mg/kg	3.8	mg/kg	M	9.6E-08	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.001
Dermal	PCBs	3.8	mg/kg	3.8	mg/kg	M	4.9E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.007
												Total Hazard Index Across All Exposure Routes/Pathways	0.008

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-2-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER SEDIMENT- Adult Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	PCBs	7.0	mg/kg	7.0	mg/kg	M	9.6E-08	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.001
Dermal	PCBs	7.0	mg/kg	7.0	mg/kg	M	4.9E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.007
										Total Hazard Index Across All Exposure Routes/Pathways			0.008

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-3-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER SEDIMENT- Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	PCBs	5.4	mg/kg	5.4	mg/kg	M	6.7E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.010
Dermal	PCBs	5.4	mg/kg	5.4	mg/kg	M	2.0E-06	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.028
										Total Hazard Index Across All Exposure Routes/Pathways			0.04

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-3-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER SEDIMENT- Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	PCBs	7.7	mg/kg	7.7	mg/kg	M	4.9E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.01
Dermal	PCBs	7.7	mg/kg	7.7	mg/kg	M	1.5E-06	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.02
												Total Hazard Index Across All Exposure Routes/Pathways	0.03

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation

TABLE 5-4-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER SEDIMENT - Child Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	PCBs	6.7	mg/kg	6.7	mg/kg	M	1.6E-06	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.02
Dermal	PCBs	6.7	mg/kg	6.7	mg/kg	M	1.2E-06	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.02
												Total Hazard Index Across All Exposure Routes/Pathways	0.04

(1) Specify Medium Specific (M) or Route-Specific (R) EPC selected for hazard calculation

TABLE 5-4-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER SEDIMENT - Child Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Ingestion	PCBs	7.7	mg/kg	7.7	mg/kg	M	9.9E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.014
Dermal	PCBs	7.7	mg/kg	7.7	mg/kg	M	7.7E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.011
												Total Hazard Index Across All Exposure Routes/Pathways	0.03

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation

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TABLE 5-5-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Dermal	PCBs	2.93E-05	mg/L	2.93E-05	mg/L	M	3.4E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0048
											Total Hazard Index Across All Exposure Routes/Pathways		0.0048

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-5-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Dermal	PCBs	4.32E-05	mg/L	4.32E-05	mg/L	M	2.7E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0038
												Total Hazard Index Across All Exposure Routes/Pathways	0.0038

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-6-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Dermal	PCBs	3.65E-05	mg/L	3.65E-05	mg/L	M	1.5E-06	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0212
												Total Hazard Index Across All Exposure Routes/Pathways	0.0212

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-6-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Dermal	PCBs	4.62E-05	mg/L	4.62E-05	mg/L	M	9.6E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0137
												Total Hazard Index Across All Exposure Routes/Pathways	0.0137

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-7-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER WATER - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Dermal	PCBs	4.26E-05	mg/L	4.26E-05	mg/L	M	8.7E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0124
											Total Hazard Index Across All Exposure Routes/Pathways		0.0124

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation

TABLE 5-7-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER WATER - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Dermal	PCBs	4.62E-05	mg/L	4.62E-05	mg/L	M	5.1E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0 0072
												Total Hazard Index Across All Exposure Routes/Pathways	0 0072

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation

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TABLE 5-8-RME (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m³	R	5.5E-08	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
												Total Hazard Index Across All Exposure Routes/Pathways	N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-8-CT (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	1.8E-09	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
												Total Hazard Index Across All Exposure Routes/Pathways	N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-9-RME (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m^3	R	2.7E-07	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
											Total Hazard Index Across All Exposure Routes/Pathways		N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-9-CT (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	8.2E-09	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
											Total Hazard Index Across All Exposure Routes/Pathways		N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-10-RME (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	4 20E-05	mg/L	1.70E-05	mg/m³	R	1.9E-07	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
											Total Hazard Index Across All Exposure Routes/Pathways		N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-10-CT (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	6.1E-09	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
												Total Hazard Index Across All Exposure Routes/Pathways	N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-11-RME (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River – Volatilized PCBs
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m³	R	4.7E-06	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
												Total Hazard Index Across All Exposure Routes/Pathways	N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-11-CT (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	2.7E-07	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
												Total Hazard Index Across All Exposure Routes/Pathways	N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-12-RME (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Adolescent Resident

Scenario Timeframe Current/Future
Medium River Water
Exposure Medium Outdoor Air
Exposure Point Upper Hudson River -- Volatilized PCBs
Receptor Population Resident
Receptor Age Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m^3	R	5.1E-06	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
												Total Hazard Index Across All Exposure Routes/Pathways	N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-12-CT (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Adolescent Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	3.0E-07	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
												Total Hazard Index Across All Exposure Routes/Pathways	N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-13-RME (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Child Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m³	R	9.0E-06	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
											Total Hazard Index Across All Exposure Routes/Pathways		N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation.

TABLE 5-13-CT (Unchanged)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Child Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Hazard Calculation (1)	Intake (Non-Cancer)	Intake (Non-Cancer) Units	Reference Dose	Reference Dose Units	Reference Concentration	Reference Concentration Units	Hazard Quotient
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m^3	R	5.3E-07	mg/kg-day	N/A	mg/kg-day	N/A	N/A	N/A
											Total Hazard Index Across All Exposure Routes/Pathways		N/A

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for hazard calculation

TABLE 5-14-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Upper Hudson Fish
Receptor Population: Angler
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	PCBs	1.4	mg/kg wt weight	1.4	mg/kg wt weight	M	3.6E-04	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	7.1E-04
										Total Risk Across All Exposure Routes/Pathways	7.1E-04

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-14-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Upper Hudson Fish
Receptor Population: Angler
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	PCBs	2.4	mg/kg wt weight	2.4	mg/kg wt weight	M	1.9E-05	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	1.9E-05
										Total Risk Across All Exposure Routes/Pathways	1.9E-05

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

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TABLE 5-15-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER SEDIMENT- Adult Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	PCBs	3.8	mg/kg	3.8	mg/kg	M	3.2E-08	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	6.3E-08
Dermal	PCBs	3.8	mg/kg	3.8	mg/kg	M	1.6E-07	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	3.2E-07
										Total Risk Across All Exposure Routes/Pathways	3.9E-07

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-15-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER SEDIMENT- Adult Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	PCBs	7.0	mg/kg	7.0	mg/kg	M	6.9E-09	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	6.9E-09
Dermal	PCBs	7.0	mg/kg	7.0	mg/kg	M	3.5E-08	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	3.5E-08
										Total Risk Across All Exposure Routes/Pathways	4.2E-08

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-16-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER SEDIMENT- Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	PCBs	5.4	mg/kg	5.4	mg/kg	M	1.1E-07	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	2.3E-07
Dermal	PCBs	5.4	mg/kg	5.4	mg/kg	M	3.4E-07	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	6.8E-07
										Total Risk Across All Exposure Routes/Pathways	9.1E-07

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-16-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER SEDIMENT- Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	PCBs	7.7	mg/kg	7.7	mg/kg	M	2.1E-08	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	2.1E-08
Dermal	PCBs	7.7	mg/kg	7.7	mg/kg	M	6.3E-08	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	6.3E-08
										Total Risk Across All Exposure Routes/Pathways	8.4E-08

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-17-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER SEDIMENT - Child Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	PCBs	6.7	mg/kg	6.7	mg/kg	M	1.4E-07	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	2.7E-07
Dermal	PCBs	6.7	mg/kg	6.7	mg/kg	M	1.1E-07	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	2.1E-07
										Total Risk Across All Exposure Routes/Pathways	4.9E-07

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-17-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER SEDIMENT - Child Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Upper Hudson
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Ingestion	PCBs	7.7	mg/kg	7.7	mg/kg	M	4.2E-08	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	4.2E-08
Dermal	PCBs	7.7	mg/kg	7.7	mg/kg	M	3.3E-08	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	3.3E-08
										Total Risk Across All Exposure Routes/Pathways	7.5E-08

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-18-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Dermal	PCBs	2.93E-05	mg/L	2.93E-05	mg/L	M	1.1E-07	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	4.4E-08
										Total Risk Across All Exposure Routes/Pathways	4.4E-08

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-18-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Dermal	PCBs	4.32E-05	mg/L	4.32E-05	mg/L	M	1.9E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	5.7E-09
										Total Risk Across All Exposure Routes/Pathways	5.7E-09

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-19-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Dermal	PCBs	3.65E-05	mg/L	3.65E-05	mg/L	M	2.5E-07	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	1.0E-07
										Total Risk Across All Exposure Routes/Pathways	1.0E-07

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-19-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Dermal	PCBs	4.62E-05	mg/L	4.62E-05	mg/L	M	4.1E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	1.2E-08
										Total Risk Across All Exposure Routes/Pathways	1.2E-08

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-20-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER WATER - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Dermal	PCBs	4.26E-05	mg/L	4.26E-05	mg/L	M	7.4E-08	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	3.0E-08
										Total Risk Across All Exposure Routes/Pathways	3.0E-08

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-20-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER WATER - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Upper Hudson River
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Dermal	PCBs	4.62E-05	mg/L	4.62E-05	mg/L	M	2.2E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	6 5E-09
										Total Risk Across All Exposure Routes/Pathways	6.5E-09

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-21-RME (Unchanged)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m³	R	1.8E-08	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	7.28E-09
										Total Risk Across All Exposure Routes/Pathways	7.28E-09

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-21-CT (Unchanged)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m^3	R	1.3E-10	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	3.76E-11
										Total Risk Across All Exposure Routes/Pathways	3.76E-11

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

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TABLE 5-22-RME (Unchanged)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m^3	R	4.6E-08	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	1.85E-08
										Total Risk Across All Exposure Routes/Pathways	1.85E-08

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-22-CT (Unchanged)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River – Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	3.5E-10	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	1.05E-10
										Total Risk Across All Exposure Routes/Pathways	1.05E-10

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-23-RME (Unchanged)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m^3	R	1.7E-08	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	6.64E-09
										Total Risk Across All Exposure Routes/Pathways	6.64E-09

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-23-CT (Unchanged)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Recreator
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	2.6E-10	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	7.89E-11
										Total Risk Across All Exposure Routes/Pathways	7.89E-11

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

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TABLE 5-24-RME (Unchanged)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m³	R	1.5E-06	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	6.12E-07
										Total Risk Across All Exposure Routes/Pathways	6.12E-07

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-24-CT (Unchanged)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	2.0E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	5.87E-09
										Total Risk Across All Exposure Routes/Pathways	5.87E-09

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-25-RME (Unchanged)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Adolescent Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m^3	R	8.8E-07	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	3.51E-07
										Total Risk Across All Exposure Routes/Pathways	3.51E-07

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation

TABLE 5-25-CT (Unchanged)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Adolescent Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Adolescent

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	1.3E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	3.87E-09
										Total Risk Across All Exposure Routes/Pathways	3.87E-09

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-26-RME (Unchanged)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
UPPER HUDSON RIVER AIR - Child Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	4.20E-05	mg/L	1.70E-05	mg/m^3	R	7.7E-07	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	3.09E-07
										Total Risk Across All Exposure Routes/Pathways	3.09E-07

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-26-CT (Unchanged)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
UPPER HUDSON RIVER AIR - Child Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: Outdoor Air
Exposure Point: Upper Hudson River -- Volatilized PCBs
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Chemical of Potential Concern	Medium EPC Value	Medium EPC Units	Route EPC Value	Route EPC Units	EPC Selected for Risk Calculation (1)	Intake (Cancer)	Intake (Cancer) Units	Cancer Slope Factor	Cancer Slope Factor Units	Cancer Risk
Inhalation	PCBs	2.40E-05	mg/L	1.00E-06	mg/m³	R	2.3E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	6.82E-09
										Total Risk Across All Exposure Routes/Pathways	6.82E-09

(1) Specify Medium-Specific (M) or Route-Specific (R) EPC selected for risk calculation.

TABLE 5-27-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

UPPER HUDSON RIVER - Adult Angler

Scenario Timeframe: Current/Future
Receptor Population: Angler
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Fish	Fish	Upper Hudson Fish	PCBs	7.1E-04	--	--	7.1E-04	PCBs	LOAEL	65	--	--	65
						Total Risk Across Fish:	7.1E-04					Total Hazard Index Across All Media and All Exposure Routes	65
						Total Risk Across All Media and All Exposure Routes	7.1E-04					Total LOAEL HI =	65

TABLE 5-27-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

UPPER HUDSON RIVER - Adult Angler

Scenario Timeframe: Current/Future
Receptor Population: Angler
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Fish	Fish	Upper Hudson Fish	PCBs	1.9E-05	--	--	1.9E-05	PCBs	LOAEL	6	--	--	6
						Total Risk Across Fish	1.9E-05					Total Hazard Index Across All Media and All Exposure Routes	6
						Total Risk Across All Media and All Exposure Routes	1.9E-05					Total LOAEL HI =	6

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TABLE 5-28-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

UPPER HUDSON RIVER - Adult Recreator

Scenario Timeframe: Current/Future
Receptor Population: Recreator
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Sediment	Banks of Upper Hudson	PCBs	6.3E-08	--	3.2E-07	3.9E-07	PCBs	NOAEL	0.001	--	0.007	0.008
River Water	River Water	Upper Hudson River	PCBs	--	--	4.4E-08	4.4E-08	PCBs	NOAEL	--	--	0.0048	0.0048
River Water	Outdoor Air	Upper Hudson River - Volatilized PCBs	PCBs	--	7.3E-09	--	7.3E-09	PCBs	NOAEL	--	N/A	--	N/A

Total Risk Across Sediment	3.9E-07
Total Risk Across River Water	5.2E-08
Total Risk Across All Media and All Exposure Routes	4.4E-07

Total Hazard Index Across All Media and All Exposure Routes	0.01
Total NOAEL HI =	0.01

TABLE 5-28-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

UPPER HUDSON RIVER - Adult Recreator

Scenario Timeframe: Current/Future
Receptor Population: Recreator
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Sediment	Banks of Upper Hudson	PCBs	6.9E-09	--	3.5E-08	4.2E-08	PCBs	NOAEL	0.001	--	0.007	0.008
River Water	River Water	Upper Hudson River	PCBs	--	--	5.7E-09	5.7E-09	PCBs	NOAEL	--	--	0.0038	0.0038
River Water	Outdoor Air	Upper Hudson River - Volatilized PCBs	PCBs	--	3.8E-11	--	3.8E-11	PCBs	NOAEL	--	N/A	--	N/A

Total Risk Across Sediment	4.2E-08
Total Risk Across River Water	5.8E-09
Total Risk Across All Media and All Exposure Routes	4.8E-08

Total Hazard Index Across All Media and All Exposure Routes	0.01
Total NOAEL HI =	0.01

TABLE 5-29-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

UPPER HUDSON RIVER - Adolescent Recreator

Scenario Timeframe: Current/Future
Receptor Population: Recreator
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Sediment	Banks of Upper Hudson	PCBs	2.3E-07	--	6.8E-07	9.1E-07	PCBs	NOAEL	0.01	--	0.03	0.04
River Water	River Water	Upper Hudson River	PCBs	--	--	1.0E-07	1.0E-07	PCBs	NOAEL	--	--	0.021	0.021
River Water	Outdoor Air	Upper Hudson River - Volatilized PCBs	PCBs	--	1.9E-08	--	1.9E-08	PCBs	NOAEL	--	N/A	--	N/A
						Total Risk Across Sediment	9.1E-07					Total Hazard Index Across All Media and All Exposure Routes	0.06
						Total Risk Across River Water	1.2E-07						
						Total Risk Across All Media and All Exposure Routes	1.0E-06					Total NOAEL HI =	0.06

TABLE 5-29-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

UPPER HUDSON RIVER - Adolescent Recreator

Scenario Timeframe: Current/Future
Receptor Population: Recreator
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Sediment	Banks of Upper Hudson	PCBs	2.1E-08	--	6.3E-08	8.4E-08	PCBs	NOAEL	0.01	--	0.02	0.03
River Water	River Water	Upper Hudson River	PCBs	--	--	1.2E-08	1.2E-08	PCBs	NOAEL	--	--	0.014	0.014
River Water	Outdoor Air	Upper Hudson River - Volatilized PCBs	PCBs	--	1.0E-10	--	1.0E-10	PCBs	NOAEL	--	N/A	--	N/A

Total Risk Across Sediment	8.4E-08
Total Risk Across River Water	1.2E-08
Total Risk Across All Media and All Exposure Routes	9.6E-08

Total Hazard Index Across All Media and All Exposure Routes	0.04
Total NOAEL HI =	0.04

TABLE 5-30-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

UPPER HUDSON RIVER - Child Recreator

Scenario Timeframe: Current/Future
Receptor Population: Recreator
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Sediment	Banks of Upper Hudson	PCBs	2.7E-07	--	2.1E-07	4.9E-07	PCBs	NOAEL	0.02	--	0.02	0.04
River Water	River Water	Upper Hudson River	PCBs	--	--	3.0E-08	3.0E-08	PCBs	NOAEL	--	--	0.012	0.012
River Water	Outdoor Air	Upper Hudson River - Volatilized PCBs	PCBs	--	6.6E-09	--	6.6E-09	PCBs	NOAEL	--	N/A	--	N/A

Total Risk Across Sediment	4.9E-07	Total Hazard Index Across All Media and All Exposure Routes	0.05
Total Risk Across River Water	3.6E-08		
Total Risk Across All Media and All Exposure Routes	5.2E-07	Total NOAEL HI =	0.05

TABLE 5-30-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

UPPER HUDSON RIVER - Child Recreator

Scenario Timeframe: Current/Future
Receptor Population: Recreator
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
Sediment	Sediment	Banks of Upper Hudson	PCBs	4.2E-08	--	3.3E-08	7.5E-08	PCBs	NOAEL	0.01	--	0.01	0.03
River Water	River Water	Upper Hudson River	PCBs	--	--	6.5E-09	6.5E-09	PCBs	NOAEL	--	--	0.0072	0.0072
River Water	Outdoor Air	Upper Hudson River - Volatilized PCBs	PCBs	--	7.9E-11	--	7.9E-11	PCBs	NOAEL	--	N/A	--	N/A

Total Risk Across Sediment	7.5E-08	Total Hazard Index Across All Media and All Exposure Routes	0.03
Total Risk Across River Water	6.6E-09		
Total Risk Across All Media and All Exposure Routes	8.2E-08	Total NOAEL HI =	0.03

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TABLE 5-31-RME (Unchanged)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

UPPER HUDSON RIVER - Adult Resident

Scenario Timeframe: Current/Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
River Water	Outdoor Air	Upper Hudson River -- Volatilized PCBs	PCBs	--	6 1E-07	--	6 1E-07	PCBs	LOAEL	--	N/A	--	N/A
						Total Risk Across Air	6.1E-07					Total Hazard Index Across All Media and All Exposure Routes	N/A
						Total Risk Across All Media and All Exposure Routes	6.1E-07					Total LOAEL HI =	N/A

TABLE 5-31-CT (Unchanged)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

UPPER HUDSON RIVER - Adult Resident

Scenario Timeframe: Current/Future
Receptor Population: Resident
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
River Water	Outdoor Air	Upper Hudson River -- Volatilized PCBs	PCBs	--	5.9E-09	--	5.9E-09	PCBs	LOAEL	--	N/A	--	N/A
						Total Risk Across Air	5.9E-09					Total Hazard Index Across All Media and All Exposure Routes	N/A
						Total Risk Across All Media and All Exposure Routes	5.9E-09					Total LOAEL HI =	N/A

304655

TABLE 5-32-RME (Unchanged)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

UPPER HUDSON RIVER - Adolescent Resident

Scenario Timeframe: Current/Future
Receptor Population: Resident
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
River Water	Outdoor Air	Upper Hudson River -- Volatilized PCBs	PCBs	--	3.5E-07	--	3.5E-07	PCBs	LOAEL	--	N/A	--	N/A
						Total Risk Across Air	3.5E-07					Total Hazard Index Across All Media and All Exposure Routes	N/A
						Total Risk Across All Media and All Exposure Routes	3.5E-07					Total LOAEL HI =	N/A

TABLE 5-32-CT (Unchanged)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

UPPER HUDSON RIVER - Adolescent Resident

Scenario Timeframe: Current/Future
Receptor Population: Resident
Receptor Age: Adolescent

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
River Water	Outdoor Air	Upper Hudson River -- Volatilized PCBs	PCBs	--	3.9E-09	--	3.9E-09	PCBs	LOAEL	--	N/A	--	N/A

Total Risk Across Air: 3.9E-09

Total Risk Across All Media and All Exposure Routes: 3.9E-09

Total Hazard Index Across All Media and All Exposure Routes: N/A

Total LOAEL HI = N/A

304657

TABLE 5-33-RME (Unchanged)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

UPPER HUDSON RIVER - Child Resident

Scenario Timeframe	*Current/Future*
Receptor Population	Resident
Receptor Age	Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
River Water	Outdoor Air	Upper Hudson River -- Volatilized PCBs	PCBs	--	3.1E-07	--	3.1E-07	PCBs	LOAEL	--	N/A	--	N/A
						Total Risk Across Air	3.1E-07					Total Hazard Index Across All Media and All Exposure Routes	N/A
						Total Risk Across All Media and All Exposure Routes	3.1E-07					Total LOAEL HI =	N/A

TABLE 5-33-CT (Unchanged)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

UPPER HUDSON RIVER - Child Resident

Scenario Timeframe: Current/Future
Receptor Population: Resident
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Inhalation	Dermal	Exposure Routes Total		Primary Target Organ	Ingestion	Inhalation	Dermal	Exposure Routes Total
River Water	Outdoor Air	Upper Hudson River -- Volatilized PCBs	PCBs	--	6.8E-09	--	6.8E-09	PCBs	LOAEL	--	N/A	--	N/A

Total Risk Across Air: 6.8E-09

Total Risk Across All Media and All Exposure Routes: 6.8E-09

Total Hazard Index Across All Media and All Exposure Routes: N/A

Total LOAEL HI = N/A

304659

Table 5-34 (Unchanged)
Total (Tri+) PCB Concentrations - Phase 2 Fish Data - Upper Hudson

Fish Sample	Species	River Mile	Total (Tri+) PCB Concentration (ug/kg wet weight)
EC-F09-0001	SPOT	159	1,770
EC-F09-0002	SPOT	159	1,823
EC-F09-0003	SPOT	159	1,380
EC-F08-0001	LMB	169.5	2,719
EC-F08-0002	LMB	169.5	4,788
EC-F08-0003	LMB	169.5	3,554
EC-F08-0001	PKSD	169.5	5,900
EC-F08-0002	PKSD	169.5	9,765
EC-F08-0003	PKSD	169.5	12,550
EC-F08-0004	PKSD	169.5	10,292
EC-F08-0005	PKSD	169.5	11,173
EC-F08-0001	SPOT	169.5	1,899
EC-F08-0002	SPOT	169.5	1,828
EC-F08-0003	SPOT	169.5	1,442
EC-F08-0001	YP	169.5	10,710
EC-F08-0002	YP	169.5	9,926
EC-F08-0003	YP	169.5	15,208
EC-F08-0004	YP	169.5	21,207
EC-F08-0005	YP	169.5	20,421
EC-F04-0001	LMB	189.5	15,522
EC-F04-0002	LMB	189.5	23,287
EC-F04-0003	LMB	189.5	14,070
EC-F04-0001	PKSD	189.5	40,174
EC-F04-0002	PKSD	189.5	41,422
EC-F04-0003	PKSD	189.5	33,657
EC-F04-0004	PKSD	189.5	56,776
EC-F04-0005	PKSD	189.5	48,177
EC-F04-0001	SPOT	189.5	20,957
EC-F04-0002	SPOT	189.5	11,514
EC-F04-0003	SPOT	189.5	8,799
EC-F04-0001	YP	189.5	35,884
EC-F04-0002	YP	189.5	23,588
EC-F04-0003	YP	189.5	16,057
EC-F04-0004	YP	189.5	19,213
EC-F04-0005	YP	189.5	13,590
EC-F03-0001	PKSD	191.5	14,045
EC-F03-0002	PKSD	191.5	11,090
EC-F03-0003	PKSD	191.5	7,528
EC-F03-0004	PKSD	191.5	12,543
EC-F03-0005	PKSD	191.5	12,178
EC-F03-0006	PKSD	191.5	13,696
EC-F03-0001	SPOT	191.5	4,394
EC-F03-0002	SPOT	191.5	3,167
EC-F03-0003	SPOT	191.5	3,215
EC-F03-0001	YP	191.5	8,797
EC-F03-0002	YP	191.5	26,629
EC-F03-0003	YP	191.5	17,816
EC-F03-0004	YP	191.5	31,776
EC-F03-0005	YP	191.5	28,577
EC-F02-0001	LMB	194.1	17,355
EC-F02-0002	LMB	194.1	7,174
EC-F02-0003	LMB	194.1	6,332
EC-F02-0001	PKSD	194.1	28,859
EC-F02-0002	PKSD	194.1	26,488
EC-F02-0001	SPOT	194.1	23,711
EC-F02-0002	SPOT	194.1	16,420
EC-F02-0003	SPOT	194.1	15,279
EC-F02-0001	YP	194.1	40,163
EC-F02-0002	YP	194.1	48,526
EC-F02-0003	YP	194.1	45,172
EC-F02-0004	YP	194.1	31,330
EC-F02-0005	YP	194.1	47,196
EC-F20-0001	BB	196.9	8,000

Table 5-35 (Unchanged)
Fraction of Dioxin-Like PCB Congeners in Upper Hudson Fish

			Ratio of Congener Concentration to Total (Tri+) PCB Concentration													
Fish Sample	Species	River Mile	77	105	114	118	123	126	156	157	167	169	189	170	180	Total
EC-F09-0001	SPOT	159	3.4E-03	1.7E-02	2.0E-03	3.7E-02	0.0E+00	0.0E+00	2.4E-03	1.1E-03	1.8E-03	0.0E+00	2.2E-04	3.4E-03	8.0E-03	7.6E-02
EC-F09-0002	SPOT	159	3.4E-03	1.7E-02	2.0E-03	3.7E-02	0.0E+00	0.0E+00	2.8E-03	6.1E-04	1.8E-03	0.0E+00	2.2E-04	3.4E-03	8.3E-03	7.6E-02
EC-F09-0003	SPOT	159	3.1E-03	1.8E-02	2.0E-03	3.8E-02	0.0E+00	9.8E-05	2.8E-03	2.7E-04	1.5E-03	0.0E+00	8.4E-05	3.1E-03	8.7E-03	7.7E-02
EC-F08-0001	LMB	169.5	3.4E-03	2.1E-02	2.2E-03	4.0E-02	0.0E+00	2.3E-04	2.8E-03	8.1E-04	1.0E-03	0.0E+00	1.5E-04	3.3E-03	9.0E-03	8.4E-02
EC-F08-0002	LMB	169.5	2.8E-03	1.8E-02	2.1E-03	4.2E-02	0.0E+00	2.2E-04	3.0E-03	5.0E-04	2.0E-03	0.0E+00	1.6E-04	3.7E-03	9.4E-03	8.3E-02
EC-F08-0003	LMB	169.5	2.8E-03	1.6E-02	2.0E-03	3.7E-02	0.0E+00	2.7E-04	2.9E-03	5.9E-04	1.8E-03	0.0E+00	1.4E-04	3.5E-03	9.3E-03	7.7E-02
EC-F08-0001	PKSD	169.5	3.3E-03	1.2E-02	1.3E-03	2.6E-02	0.0E+00	2.3E-04	1.9E-03	3.1E-04	8.6E-04	0.0E+00	0.0E+00	1.5E-03	3.1E-03	5.1E-02
EC-F08-0002	PKSD	169.5	3.3E-03	1.1E-02	1.1E-03	2.4E-02	0.0E+00	0.0E+00	1.5E-03	1.1E-04	6.2E-04	0.0E+00	0.0E+00	1.1E-03	2.4E-03	4.5E-02
EC-F08-0003	PKSD	169.5	3.6E-03	1.3E-02	1.6E-03	2.7E-02	0.0E+00	7.7E-05	1.4E-03	1.3E-04	7.8E-04	0.0E+00	4.8E-05	1.2E-03	2.8E-03	5.1E-02
EC-F08-0004	PKSD	169.5	3.0E-03	1.3E-02	7.7E-04	3.1E-02	9.2E-04	9.2E-04	1.8E-03	2.4E-04	9.9E-04	0.0E+00	0.0E+00	1.5E-03	4.0E-03	5.8E-02
EC-F08-0005	PKSD	169.5	3.0E-03	1.4E-02	9.9E-04	3.0E-02	8.4E-04	0.0E+00	1.7E-03	2.7E-04	8.1E-04	0.0E+00	0.0E+00	1.5E-03	3.5E-03	5.7E-02
EC-F08-0001	SPOT	169.5	2.5E-03	1.7E-02	1.9E-03	3.7E-02	0.0E+00	0.0E+00	2.6E-03	3.0E-04	1.7E-03	0.0E+00	0.0E+00	2.6E-03	7.5E-03	7.4E-02
EC-F08-0002	SPOT	169.5	2.9E-03	1.6E-02	1.8E-03	3.5E-02	0.0E+00	0.0E+00	2.5E-03	2.8E-04	1.7E-03	0.0E+00	0.0E+00	2.6E-03	7.1E-03	7.0E-02
EC-F08-0003	SPOT	169.5	2.8E-03	1.6E-02	1.9E-03	3.6E-02	0.0E+00	0.0E+00	2.6E-03	2.6E-04	1.8E-03	0.0E+00	0.0E+00	2.9E-03	8.2E-03	7.3E-02
EC-F08-0001	YP	169.5	2.9E-03	1.7E-02	3.6E-03	3.5E-02	1.2E-03	0.0E+00	2.5E-03	3.4E-04	1.4E-03	0.0E+00	1.0E-04	2.3E-03	5.9E-03	7.2E-02
EC-F08-0002	YP	169.5	2.8E-03	1.7E-02	2.0E-03	3.6E-02	0.0E+00	0.0E+00	2.4E-03	1.6E-04	1.2E-03	0.0E+00	0.0E+00	2.1E-03	5.1E-03	6.8E-02
EC-F08-0003	YP	169.5	3.1E-03	1.6E-02	1.8E-03	3.2E-02	0.0E+00	7.7E-05	2.3E-03	3.7E-04	1.0E-03	0.0E+00	0.0E+00	1.8E-03	4.2E-03	6.2E-02
EC-F08-0004	YP	169.5	3.0E-03	1.2E-02	3.1E-03	2.3E-02	9.9E-04	0.0E+00	1.9E-03	2.7E-04	9.0E-04	0.0E+00	7.7E-05	1.6E-03	3.8E-03	5.0E-02
EC-F08-0005	YP	169.5	3.2E-03	1.3E-02	1.8E-03	2.7E-02	0.0E+00	8.8E-05	1.7E-03	2.1E-04	8.6E-04	1.2E-05	6.7E-05	1.5E-03	3.9E-03	5.4E-02
EC-F04-0001	LMB	189.5	5.8E-03	1.7E-02	2.0E-03	3.0E-02	1.2E-04	1.4E-04	1.8E-03	4.8E-04	1.0E-03	0.0E+00	8.1E-05	1.8E-03	4.6E-03	6.6E-02
EC-F04-0002	LMB	189.5	7.3E-03	2.3E-02	3.7E-03	4.3E-02	7.6E-04	1.9E-04	3.2E-03	7.9E-04	1.8E-03	0.0E+00	1.3E-04	3.1E-03	7.0E-03	9.4E-02
EC-F04-0003	LMB	189.5	6.7E-03	2.4E-02	3.5E-03	4.5E-02	5.9E-04	1.7E-04	3.2E-03	7.8E-04	1.8E-03	0.0E+00	1.3E-04	3.2E-03	7.3E-03	9.6E-02
EC-F04-0001	PKSD	189.5	5.3E-03	1.2E-02	1.4E-03	2.4E-02	0.0E+00	7.9E-05	1.3E-03	2.5E-04	6.1E-04	0.0E+00	5.7E-05	1.0E-03	2.2E-03	4.9E-02
EC-F04-0002	PKSD	189.5	4.4E-03	1.2E-02	1.5E-03	2.6E-02	0.0E+00	8.9E-05	1.5E-03	1.4E-04	7.8E-04	0.0E+00	6.7E-05	1.3E-03	2.6E-03	5.0E-02
EC-F04-0003	PKSD	189.5	5.3E-03	1.2E-02	1.4E-03	2.5E-02	0.0E+00	1.1E-04	1.3E-03	1.9E-04	6.1E-04	0.0E+00	5.2E-05	9.6E-04	2.2E-03	4.9E-02
EC-F04-0004	PKSD	189.5	6.0E-03	1.4E-02	1.6E-03	2.6E-02	1.1E-04	8.7E-05	1.3E-03	9.6E-05	6.6E-04	0.0E+00	4.1E-05	9.0E-04	2.0E-03	5.2E-02
EC-F04-0005	PKSD	189.5	6.4E-03	1.5E-02	1.6E-03	2.7E-02	2.2E-04	1.0E-04	1.1E-03	3.1E-04	6.5E-04	0.0E+00	3.9E-05	8.8E-04	2.0E-03	5.5E-02
EC-F04-0001	SPOT	189.5	8.0E-03	2.3E-02	2.9E-03	4.1E-02	3.5E-04	0.0E+00	2.4E-03	5.1E-04	1.4E-03	0.0E+00	9.7E-05	2.1E-03	4.5E-03	8.5E-02
EC-F04-0002	SPOT	189.5	7.0E-03	2.4E-02	2.5E-03	4.5E-02	0.0E+00	0.0E+00	2.4E-03	3.0E-04	1.5E-03	0.0E+00	1.7E-04	2.2E-03	4.9E-03	9.0E-02
EC-F04-0003	SPOT	189.5	7.1E-03	2.4E-02	2.5E-03	4.0E-02	0.0E+00	0.0E+00	2.6E-03	3.6E-04	1.4E-03	0.0E+00	2.0E-04	2.5E-03	5.4E-03	8.6E-02
EC-F04-0001	YP	189.5	3.6E-03	1.4E-02	2.8E-03	2.8E-02	1.0E-03	3.3E-05	2.0E-03	3.7E-04	9.6E-04	0.0E+00	6.4E-05	1.7E-03	3.6E-03	5.8E-02
EC-F04-0002	YP	189.5	2.0E-03	8.2E-03	1.1E-03	1.9E-02	0.0E+00	0.0E+00	1.2E-03	7.9E-05	7.5E-04	0.0E+00	5.8E-05	1.1E-03	2.4E-03	3.6E-02
EC-F04-0003	YP	189.5	4.1E-03	1.7E-02	3.4E-03	3.4E-02	1.1E-03	0.0E+00	2.2E-03	4.0E-04	1.2E-03	0.0E+00	8.3E-05	1.9E-03	4.9E-03	7.0E-02
EC-F04-0004	YP	189.5	5.5E-03	1.6E-02	2.6E-03	3.1E-02	0.0E+00	1.1E-04	2.1E-03	5.0E-04	1.0E-03	0.0E+00	7.7E-05	1.9E-03	4.1E-03	6.5E-02
EC-F04-0005	YP	189.5	4.4E-03	1.9E-02	2.2E-03	3.8E-02	0.0E+00	0.0E+00	2.4E-03	2.9E-04	1.4E-03	0.0E+00	1.5E-04	2.0E-03	4.6E-03	7.5E-02
EC-F03-0001	PKSD	191.5	5.9E-03	1.7E-02	2.5E-03	3.8E-02	0.0E+00	1.2E-04	2.2E-03	4.2E-04	1.0E-03	0.0E+00	7.2E-05	1.5E-03	3.6E-03	7.2E-02
EC-F03-0002	PKSD	191.5	5.6E-03	1.7E-02	2.6E-03	3.6E-02	0.0E+00	1.3E-04	2.5E-03	1.7E-04	1.1E-03	0.0E+00	8.6E-05	1.8E-03	4.1E-03	7.2E-02
EC-F03-0003	PKSD	191.5	4.8E-03	1.8E-02	1.4E-03	3.7E-02	0.0E+00	2.1E-04	2.6E-03	3.9E-04	1.2E-03	0.0E+00	2.4E-04	1.7E-03	3.7E-03	7.1E-02
EC-F03-0004	PKSD	191.5	5.1E-03	1.7E-02	2.7E-03	4.0E-02	0.0E+00	1.2E-04	2.3E-03	3.5E-04	1.1E-03	0.0E+00	7.4E-05	1.7E-03	3.8E-03	7.5E-02
EC-F03-0005	PKSD	191.5	3.7E-03	1.5E-02	1.7E-03	3.0E-02	0.0E+00	1.8E-04	1.6E-03	1.3E-04	8.4E-04	0.0E+00	5.7E-05	1.2E-03	2.7E-03	5.7E-02
EC-F03-0006	PKSD	191.5	4.2E-03	1.6E-02	1.4E-03	3.5E-02	0.0E+00	1.8E-04	2.2E-03	3.6E-04	1.1E-03	0.0E+00	1.6E-04	1.6E-03	3.3E-03	6.6E-02

Table 5-35 (Unchanged)
Fraction of Dioxin-Like PCB Congeners in Upper Hudson Fish

			Ratio of Congener Concentration to Total (Tri+) PCB Concentration													
Fish Sample	**Species**	**River Mile**	**77**	**105**	**114**	**118**	**123**	**126**	**156**	**157**	**167**	**169**	**189**	**170**	**180**	**Total**
EC-F03-0001	SPOT	191 5	4 5E-03	2 3E-02	2.5E-03	4 6E-02	0.0E+00	1.3E-04	3.1E-03	1.3E-04	1.8E-03	0.0E+00	0.0E+00	2.5E-03	5 8E-03	8 9E-02
EC-F03-0002	SPOT	191 5	3.9E-03	2.5E-02	2.9E-03	4.9E-02	0.0E+00	1.3E-04	3.5E-03	1.8E-04	2.2E-03	0.0E+00	7.8E-05	3.0E-03	7.2E-03	9.8E-02
EC-F03-0003	SPOT	191 5	3 5E-03	2.2E-02	2.6E-03	4.5E-02	1.1E-03	1.3E-04	2.7E-03	1.1E-03	1.8E-03	0.0E+00	2.1E-04	3 0E-03	6 9E-03	9 0E-02
EC-F03-0001	YP	191 5	1 8E-03	9.5E-03	1 1E-03	2.8E-02	0 0E+00	0 0E+00	2.7E-03	5 5E-04	1 8E-03	0.0E+00	2.8E-04	5.2E-03	1.5E-02	6 6E-02
EC-F03-0002	YP	191 5	5.8E-03	2.1E-02	3.3E-03	3.9E-02	3.1E-04	1.4E-04	3.0E-03	2.8E-04	1.3E-03	0.0E+00	8.7E-05	2.2E-03	4.8E-03	8.2E-02
EC-F03-0003	YP	191 5	4 6E-03	2 2E-02	3 1E-03	4 0E-02	2.6E-04	1 2E-04	2 8E-03	2 8E-04	1 2E-03	0.0E+00	7.6E-05	2 0E-03	4.5E-03	8.1E-02
EC-F03-0004	YP	191 5	5 1E-03	2 0E-02	2 6E-03	3.7E-02	2 1E-04	1 3E-04	3.4E-05	0.0E+00	9 3E-04	0.0E+00	5.8E-05	1 5E-03	3 1E-03	7 0E-02
EC-F03-0005	YP	191 5	5 0E-03	2 2E-02	3 2E-03	4 0E-02	2 9E-04	1 3E-04	2 8E-03	3 5E-04	1 3E-03	0.0E+00	7 9E-05	2.1E-03	4.6E-03	8 2E-02
EC-F02-0001	LMB	194 1	4 9E-03	2.1E-02	3.2E-03	4.4E-02	2 6E-04	1 1E-04	3.2E-03	5.3E-04	1.8E-03	0.0E+00	1.3E-04	3.0E-03	6.6E-03	8 9E-02
EC-F02-0002	LMB	194 1	5 3E-03	1 6E-02	1 3E-03	3 1E-02	8 6E-05	0.0E+00	2 0E-03	3 4E-04	1 2E-03	0.0E+00	1.0E-04	2.0E-03	4 8E-03	6 4E-02
EC-F02-0003	LMB	194 1	4.6E-03	1.4E-02	1.6E-03	2 9E-02	0 0E+00	0.0E+00	1.8E-03	5.0E-04	1 2E-03	0.0E+00	1.2E-04	2.1E-03	5 3E-03	6.1E-02
EC-F02-0001	PKSD	194 1	9 7E-03	1 4E-02	3.4E-03	2.7E-02	4.0E-04	6 3E-04	2.7E-03	3 8E-04	1.4E-03	0.0E+00	9 3E-05	2.0E-03	4 6E-03	6.7E-02
EC-F02-0002	PKSD	194 1	5 4E-03	1.5E-02	2.1E-03	3.1E-02	2.5E-04	8.2E-05	1.6E-03	4 0E-04	7 8E-04	0.0E+00	6.5E-05	1.3E-03	2 9E-03	6.0E-02
EC-F02-0001	SPOT	194 1	6 2E-03	2 0E-02	2.4E-03	4.1E-02	3.9E-04	0.0E+00	2.0E-03	3.5E-04	1.3E-03	0.0E+00	8 6E-05	2.0E-03	4.4E-03	8.0E-02
EC-F02-0002	SPOT	194 1	4.8E-03	2.1E-02	2.5E-03	4.3E-02	6.5E-05	0.0E+00	2.1E-03	1.2E-04	1.3E-03	0.0E+00	8.9E-05	1.9E-03	4 4E-03	8 1E-02
EC-F02-0003	SPOT	194 1	5.5E-03	1 9E-02	2.5E-03	3.9E-02	1.9E-04	0.0E+00	2.0E-03	2 4E-04	1.2E-03	0.0E+00	7.8E-05	1.9E-03	4.3E-03	7 5E-02
EC-F02-0001	YP	194 1	4 7E-03	1.5E-02	2.5E-03	2.9E-02	1 2E-03	0.0E+00	2.0E-03	3.9E-04	9.4E-04	0.0E+00	5.2E-05	1 3E-03	3.0E-03	6.1E-02
EC-F02-0002	YP	194 1	5 2E-03	1 8E-02	2 2E-03	3 5E-02	3.0E-04	0 0E+00	1.6E-03	2 4E-04	8 8E-04	0 0E+00	5.7E-05	1.3E-03	3.0E-03	6 8E-02
EC-F02-0003	YP	194 1	1 1E-03	2 0E-02	4 4E-04	3.7E-02	5 3E-05	0 0E+00	3.3E-04	2.9E-05	1.7E-04	0.0E+00	1.2E-05	2 5E-04	5.6E-04	6 0E-02
EC-F02-0004	YP	194 1	5 2E-03	1 8E-02	2 7E-03	3 2E-02	1.1E-03	0 0E+00	1 8E-03	3 3E-04	9.0E-04	0.0E+00	5 6E-05	1 3E-03	3 2E-03	6.6E-02
EC-F02-0005	YP	194 1	5 5E-03	1.7E-02	2.4E-03	3 2E-02	1.3E-04	0.0E+00	1 8E-03	2.5E-04	9.0E-04	0.0E+00	6.0E-05	1.3E-03	2 9E-03	6 5E-02
EC-F20-0001	BB	196 9	2 5E-03	2 3E-02	2 6E-03	5.1E-02	2 5E-04	1 4E-04	3 5E-03	4 0E-04	2.4E-03	0 0E+00	7 5E-05	3 0E-03	7.0E-03	9 6E-02
		Average	4.5E-03	1 7E-02	2.2E-03	3.5E-02	2.4E-04	9.7E-05	2.2E-03	3.5E-04	1.2E-03	1.8E-07	8.6E-05	2.0E-03	4.9E-03	7 0E-02
		Std Dev	1 6E-03	3.9E-03	7 4E-04	6.9E-03	3.8E-04	1.5E-04	7.0E-04	2 2E-04	4.5E-04	1.5E-06	6.5E-05	8 6E-04	2 4E-03	1.8E-02

Table 5-36 (Revised)
Dioxin TEQs for Dioxin-Like PCB Congeners

Congener	Structure	Average Congener / Total PCB Ratio	Congener Concentration High End Estimate (2.2 mg/kg total PCBs)	1998 WHO/ IPCS TEFs (Van den Berg *et al.*, 1998)	Dioxin TEQ High End Estimate
Non-ortho PCBs					
77	3,3',4,4'-TCB	0.0045	6.30E-03	0.0001	6.30E-07
81	3,4,4',5-TCB	na	na	0.0001	na
126	3,3',4,4',5-PeCB	0.000097	1.36E-04	0.1	1.36E-05
169	3,3',4,4',5,5'-HxCB	0.00000018	2.52E-07	0.01	2.52E-09
Mono-ortho PCBs					
105	2,3,3',4,4'-PeCB	0.017	2.38E-02	0.0001	2.38E-06
114	2,3,4,4',5-PeCB	0.0022	3.08E-03	0.0005	1.54E-06
118	2,3',4,4',5-PeCB	0.035	4.90E-02	0.0001	4.90E-06
123	2',3,4,4',5-PeCB	0.00024	3.36E-04	0.0001	3.36E-08
156	2,3,3',4,4',5-HxCB	0.0022	3.08E-03	0.0005	1.54E-06
157	2,3,3',4,4',5'-HxCB	0.00035	4.90E-04	0.0005	2.45E-07
167	2,3',4,4',5,5'-HxCB	0.0012	1.68E-03	0.00001	1.68E-08
189	2,3,3',4,4',5,5'-HpCB	0.000086	1.20E-04	0.0001	1.20E-08
	Sum of Dioxin-Like PCB Congeners (mg/kg)		0.09		2.5E-05
	Sum of Non-Dioxin-Like PCB Congeners (mg/kg)		1.3		--

Table 5-37 (Revised)
Risk Estimates for Dioxin and Non-dioxin-like PCBs
Angler Ingestion of Fish

Chemical Name	C_{fish} (mg/kg wet weight)	IR_{fish} (g/d)	FS	EF (d/yr)	ED (yrs)	Conversion Factor (kg/g)	BW (kg)	AT_{Cancer} (d)	Lifetime Avg. Daily Intake (Cancer) (mg/kg-d)	Oral Slope Factor $(mg/kg\text{-}d)^{-1}$	Cancer Risk
High-End*											
Dioxin TEQ	2.5E-05	31.9	1	365	40	1.0E-03	70	25,550	6.5E-09	150,000	9.7E-04
Non-dioxin-like PCBs	1.3	31.9	1	365	40	1.0E-03	70	25,550	3.4E-04	2	6.8E-04

Notes:

Average Daily Intake Equation: $Risk = \frac{(Cfish \times IRfish \times FS \times EF \times ED \times Conversion\ Factor)}{(BW \times AT)} \times Slope\ Factor$

For dioxin, only a plausible upper bound slope factor is available; therefore, a central-tendency estimate was not calculated.

Table 5-38 (Revised)
Comparison of Point Estimate and Monte Carlo Non-cancer Hazard Index Estimates for Fish Ingestion

	Point Estimate HI	Monte Carlo Estimate HI	Monte Carlo Scenario
Central Estimate	6	11.4	Base - 50th percentile
		1.8	Low - 50th percentile
		51.5	High - 50th percentile
High-End Estimate	65	137	Base - 95th percentile
(RME)		18.6	Low - 95th percentile
		366	High - 95th percentile

Note that the Monte Carlo Estimates did not change in this Revised HHRA.

Table 5-39 (Revised)
Comparison of Point Estimate and Monte Carlo Cancer Risk Estimates for Fish Ingestion

	Point Estimate	Monte Carlo Estimate	Monte Carlo Scenario
Central Estimate	1.9×10^{-5}	6.4×10^{-5}	Base - 50th percentile
		9.7×10^{-6}	Low - 50th percentile
		4.1×10^{-4}	High - 50th percentile
High-End Estimate	7.1×10^{-4}	8.7×10^{-4}	Base - 95th percentile
(RME)		1.1×10^{-4}	Low - 95th percentile
		3.1×10^{-3}	High - 95th percentile

Note that the Monte Carlo Estimates did not change in this Revised HHRA.

Figure 2-1 (Revised)
PCB Concentration in Fish
Brown Bullhead - Thompson Island Pool

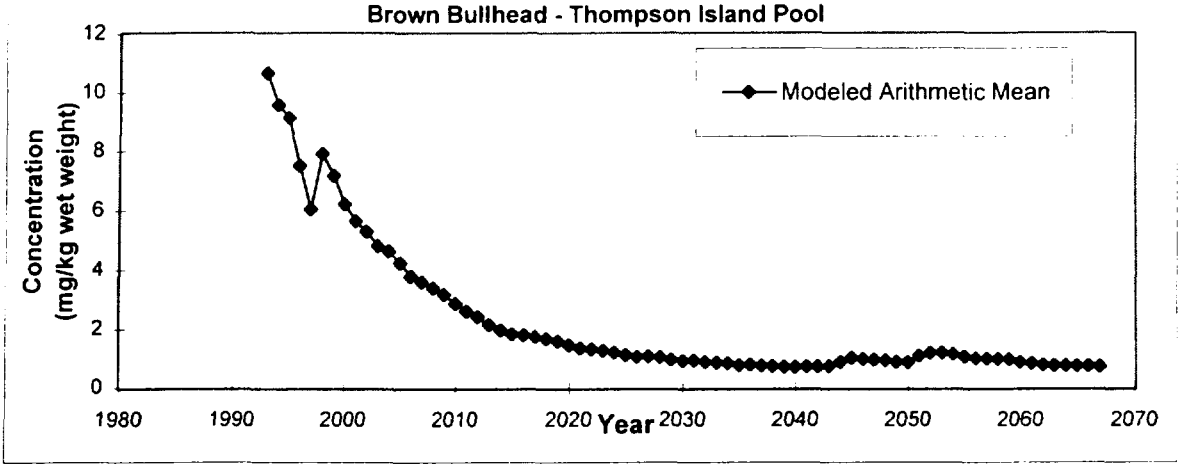


Figure 2-2 (Revised)
PCB Concentration in Fish
Brown Bullhead - River Mile 168

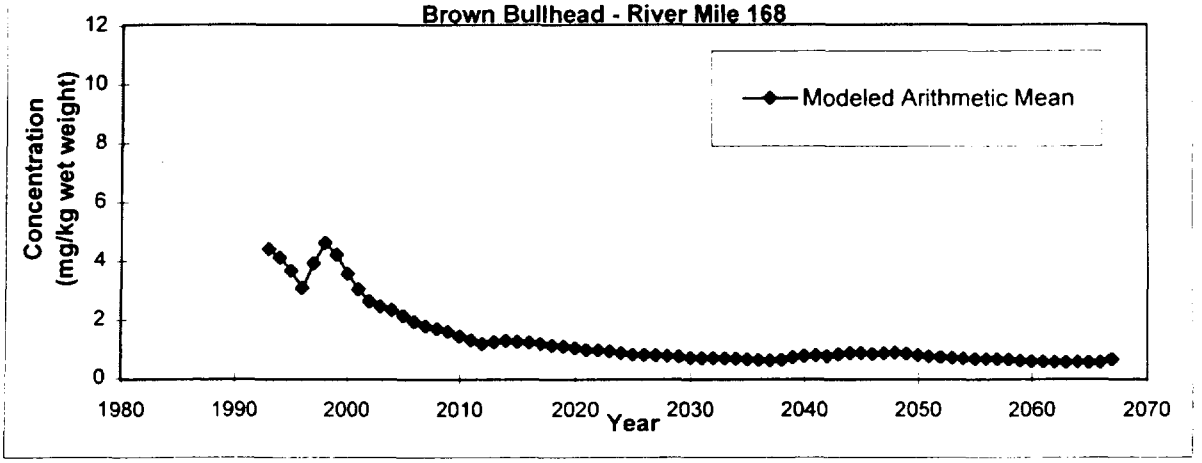
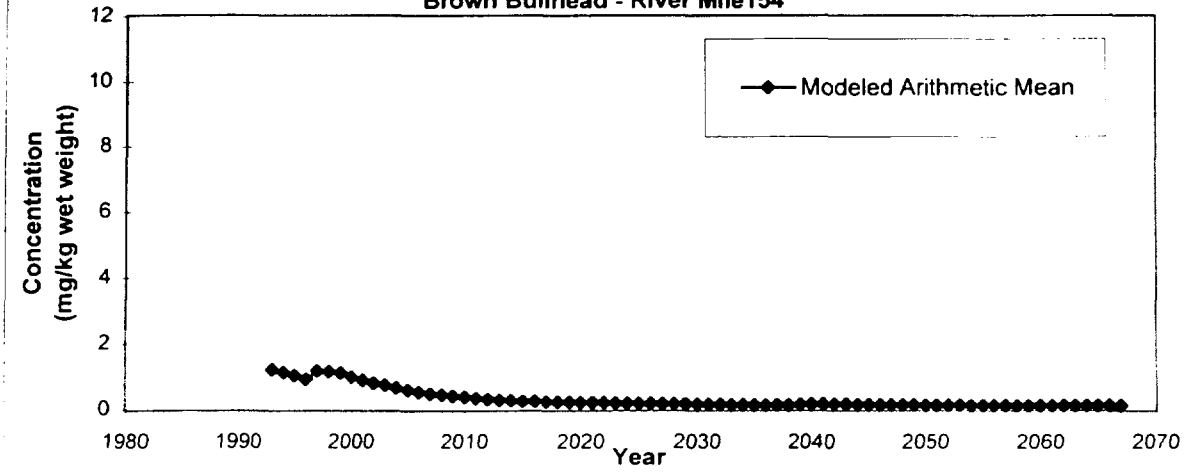


Figure 2-3 (Revised)
PCB Concentration in Fish
Brown Bullhead - River Mile154



Note: Modeled arithmetic mean from FISHRAND model in Revised BMR (USEPA, 2000).

Figure 2-4 (Revised)
PCB Concentration in Fish
Largemouth Bass - Thompson Island Pool

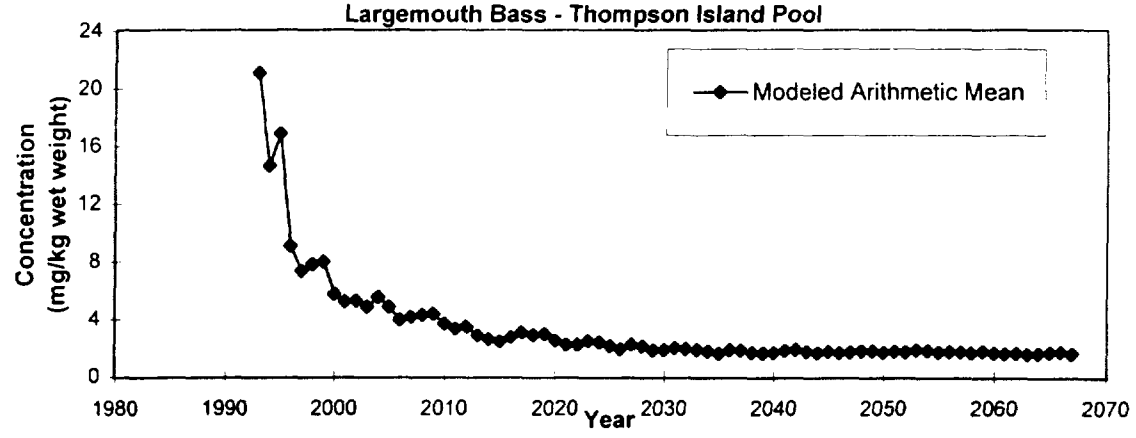


Figure 2-5 (Revised)
PCB Concentration in Fish
Largemouth Bass - River Mile 168

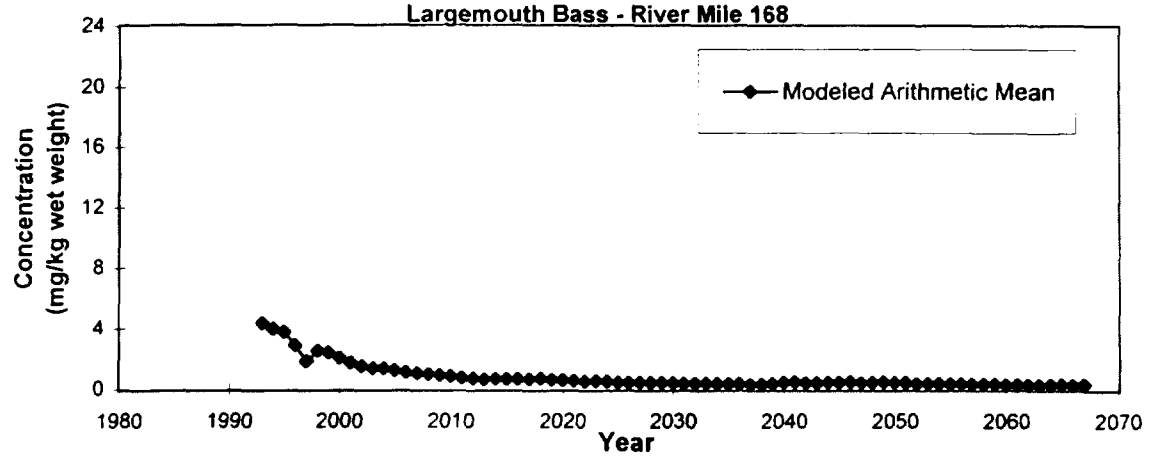
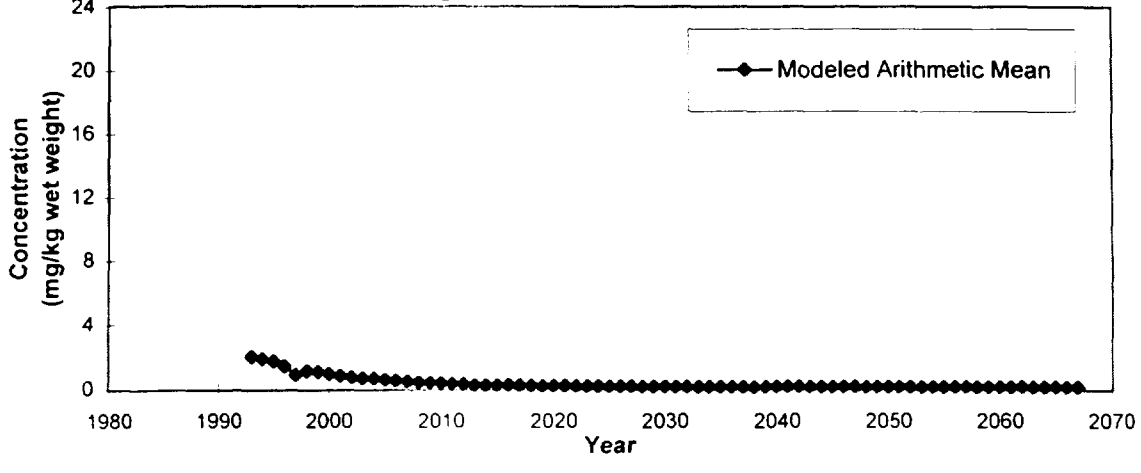


Figure 2-6 (Revised)
PCB Concentration in Fish
Largemouth Bass - River Mile154



Note Modeled arithmetic mean from FISHRAND model in Revised BMR (USEPA, 2000).

Figure 2-7 (Revised)
PCB Concentration in Fish
Yellow Perch - Thompson Island Pool

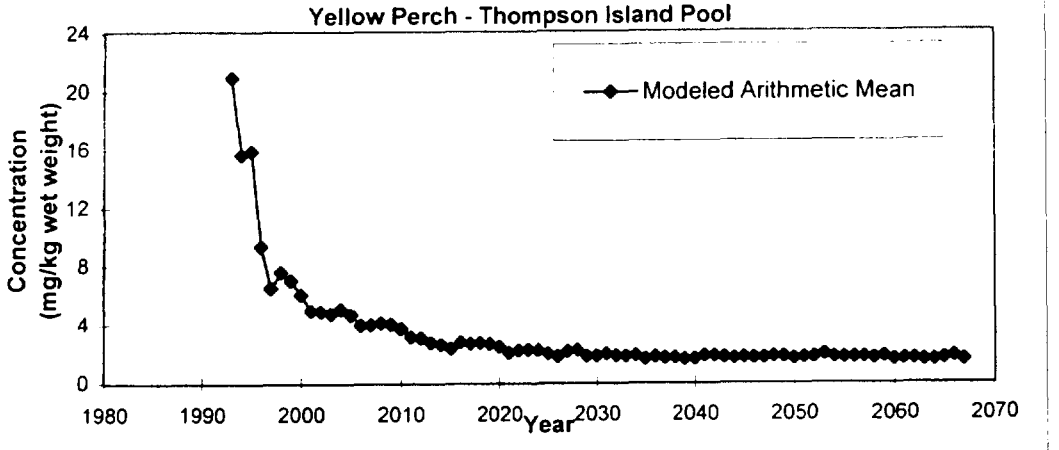


Figure 2-8 (Revised)
PCB Concentration in Fish
Yellow Perch - River Mile 168

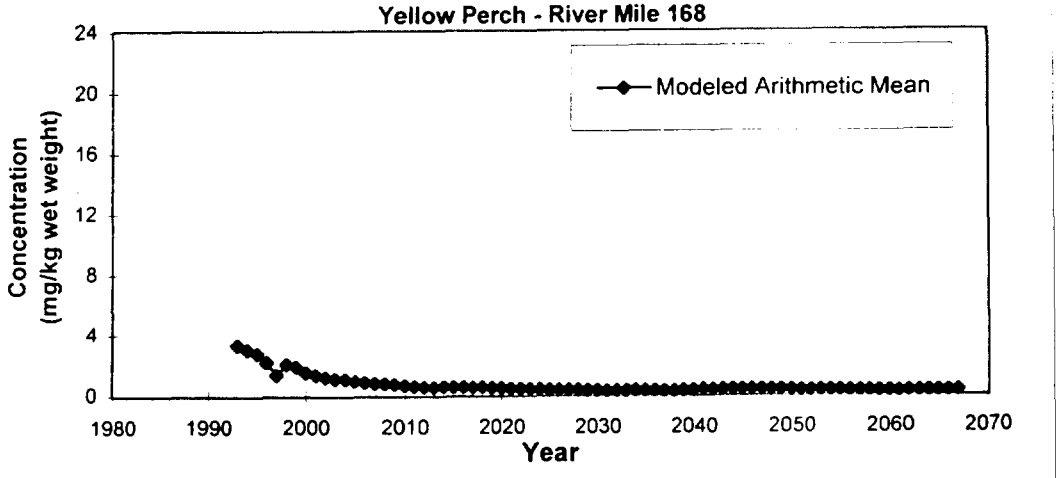
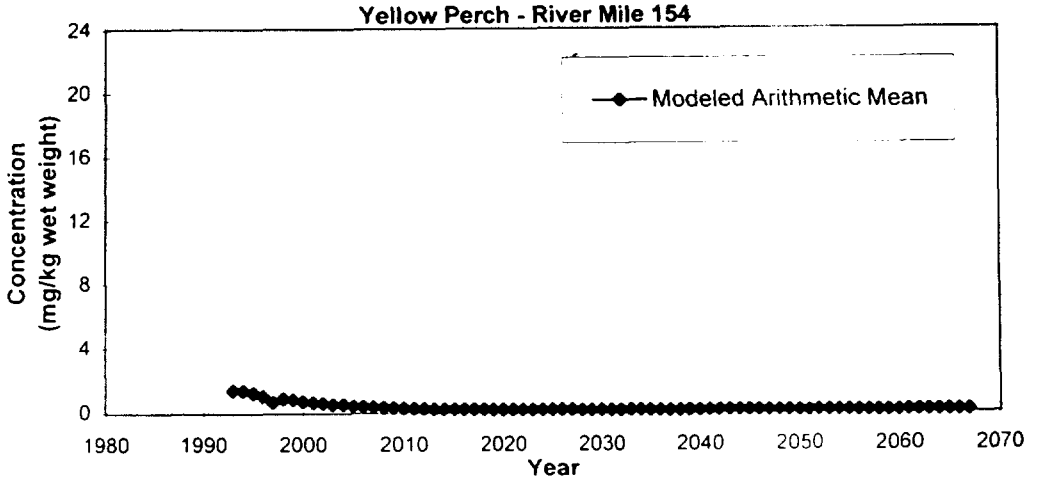


Figure 2-9 (Revised)
PCB Concentration in Fish
Yellow Perch - River Mile 154



Note Modeled arithmetic mean from FISHRAND model in Revised BMR (USEPA 2000)

Figure 2-10 (Revised)
PCB Concentration by Species, 1999-2067
(averaged over location)

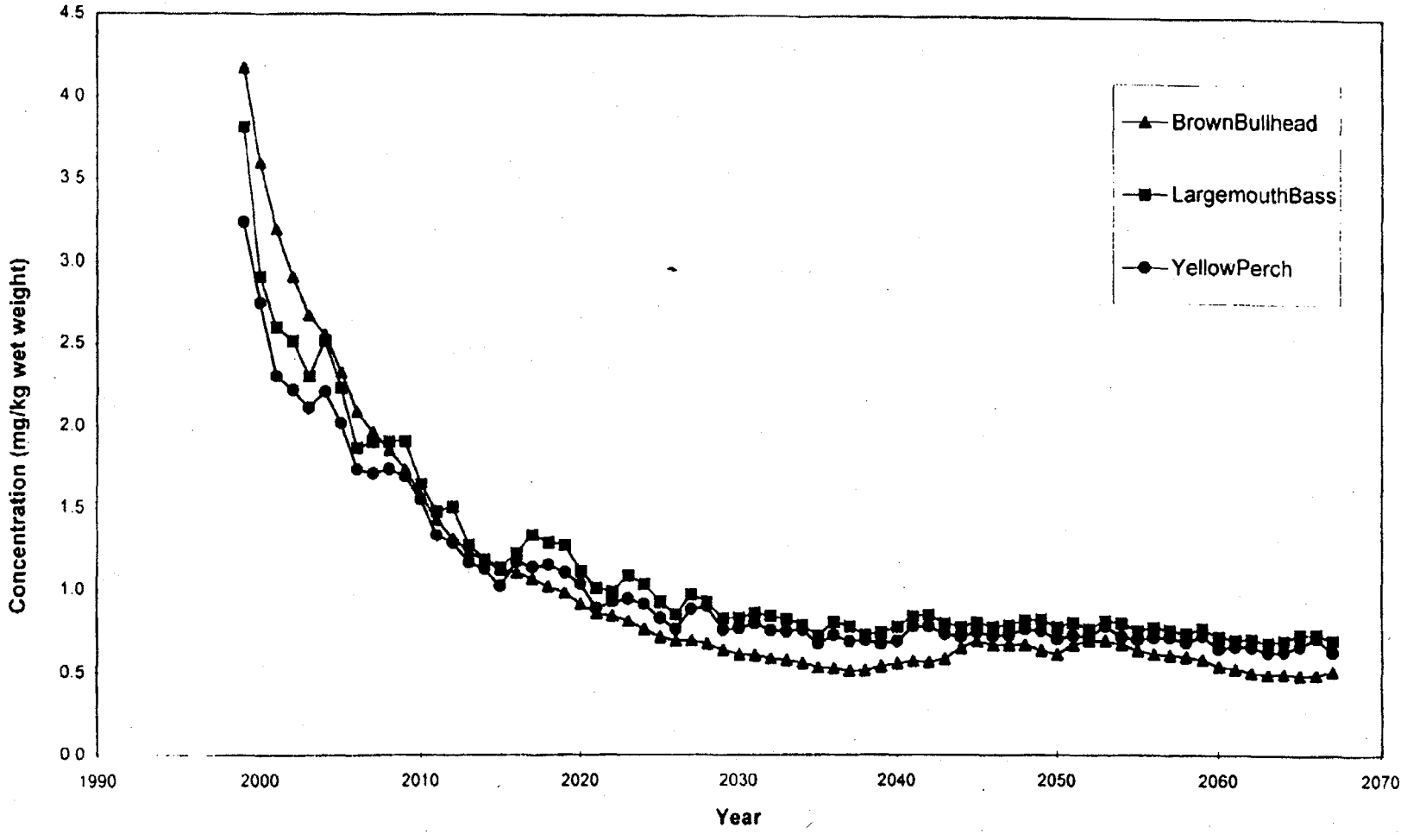
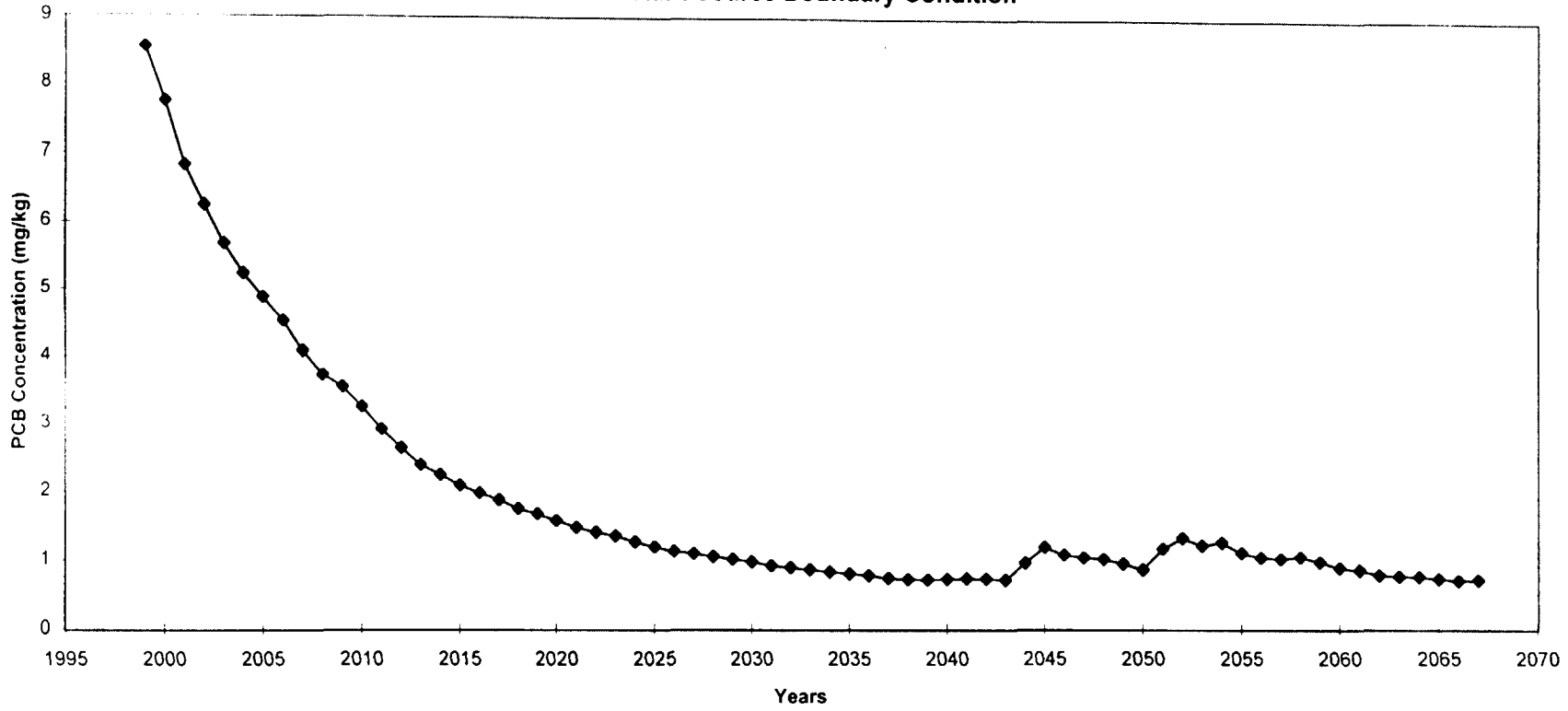


Figure 2-11 (Revised)
Segment Averaged Total PCB Concentration in Sediment (1999 - 2067)
Weighted Cohesive and Non-Cohesive Results*
Constant Source Boundary Condition



* *Sediment data are a weighted average of cohesive (75%) and noncohesive (25%) sediments (Revised BMR, 2000).*

Figure 2-12 (Revised)
Modeled Water Column Total PCB Concentration (1999 - 2067)
Constant Source Boundary Condition

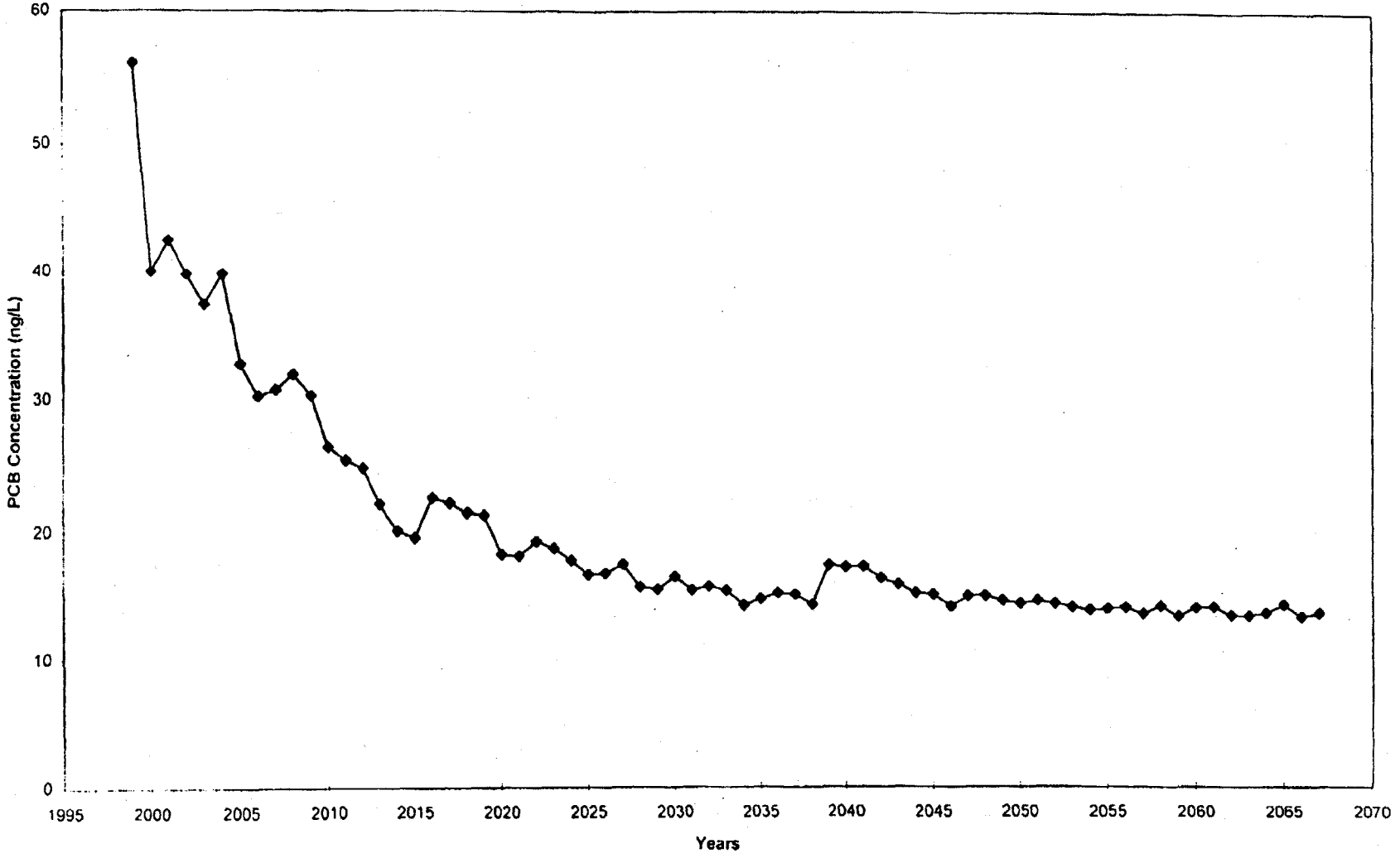


Figure 3-1 (Unchanged)
Diagram of Monte Carlo Simulation Process

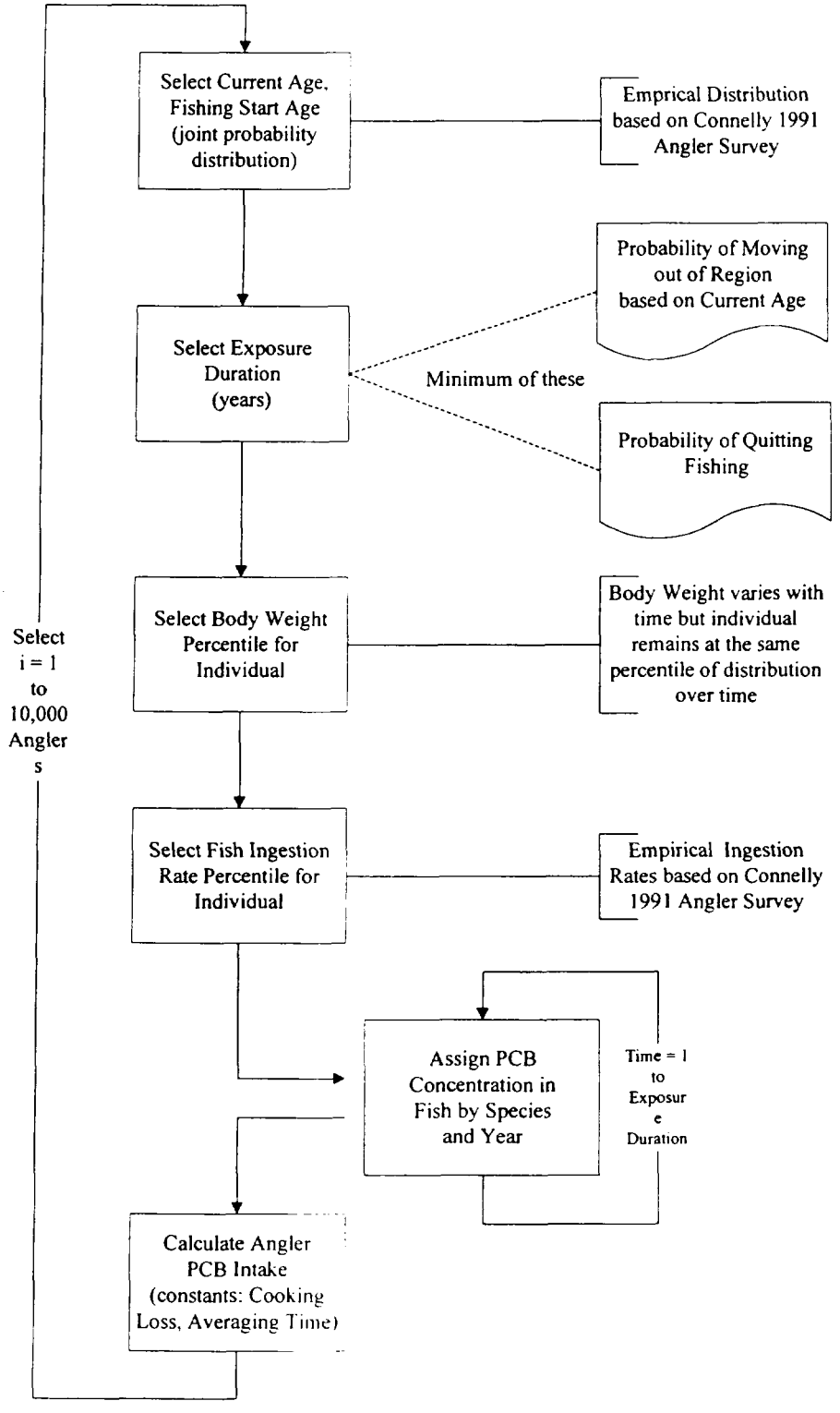


Figure 3-2a (Unchanged)
Lognormal Probability Plot - Respondents (N=226)

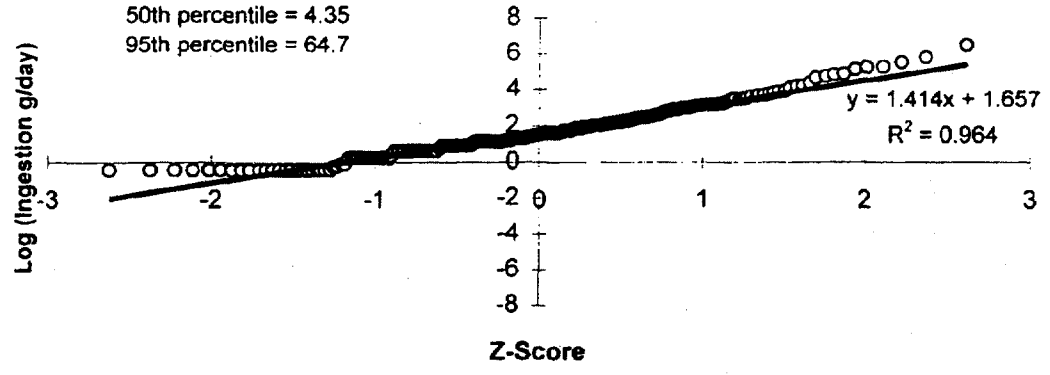


Figure 3-2b (Unchanged)
Lognormal Probability Plot - Non-Respondents (N=55)

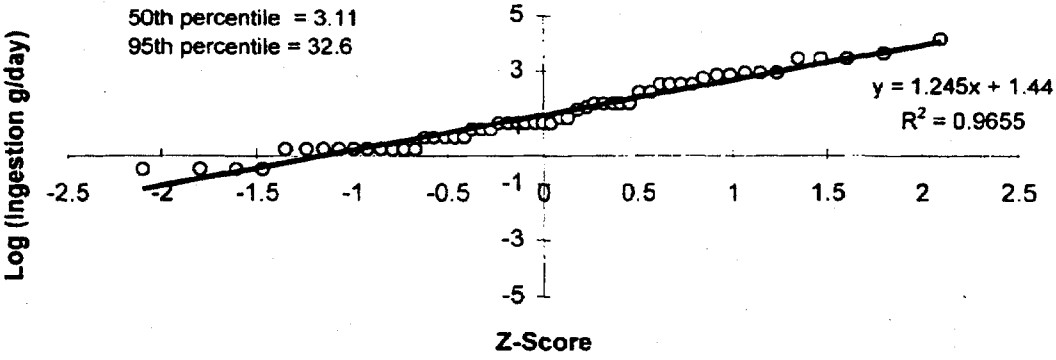
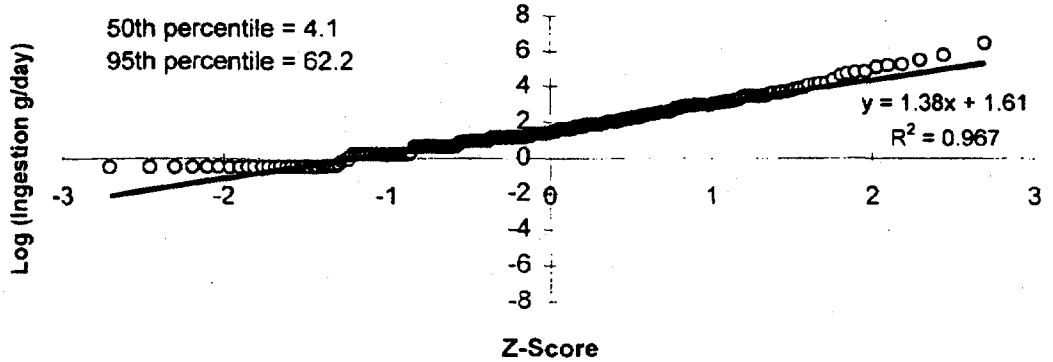


Figure 3-2c (Unchanged)
Lognormal Probability Plot
Combined Respondent + Non-Respondent (N=281)



Source: 1991 NY Angler Survey (Connelly et al., 1992).

Figure 3-3a (Unchanged)
Frequency Histogram of Self-Caught Fish Ingestion - New York (Connelly et al., 1992)

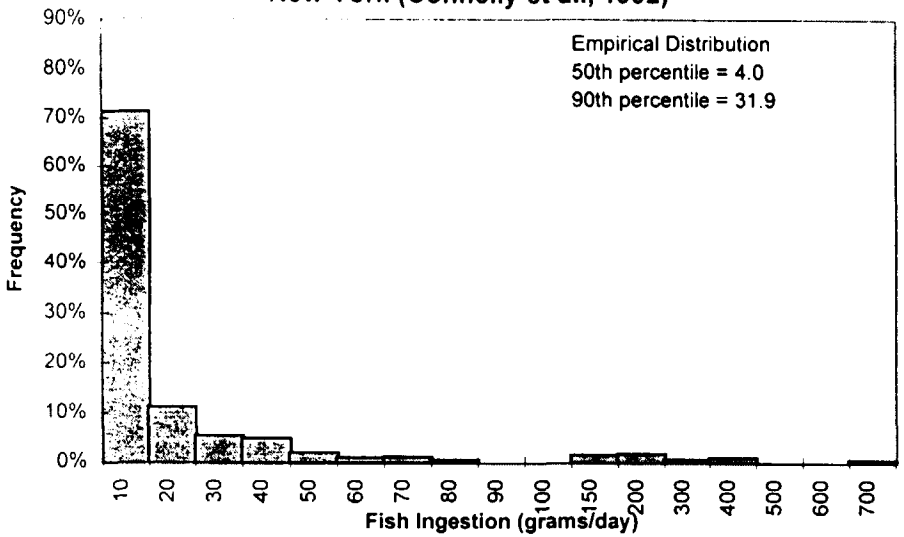


Figure 3-3b (Unchanged)
Frequency Histogram of Recreational Fish Ingestion - Lake Ontario (Connelly et al., 1996)

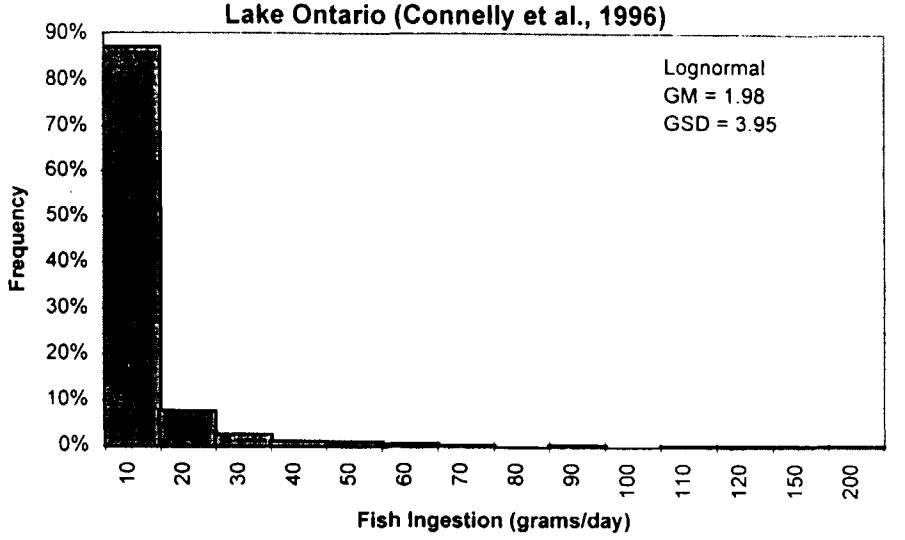


Figure 3-3c (Unchanged)
Frequency Histogram of Recreational Fish Ingestion - Michigan (West et al., 1989)

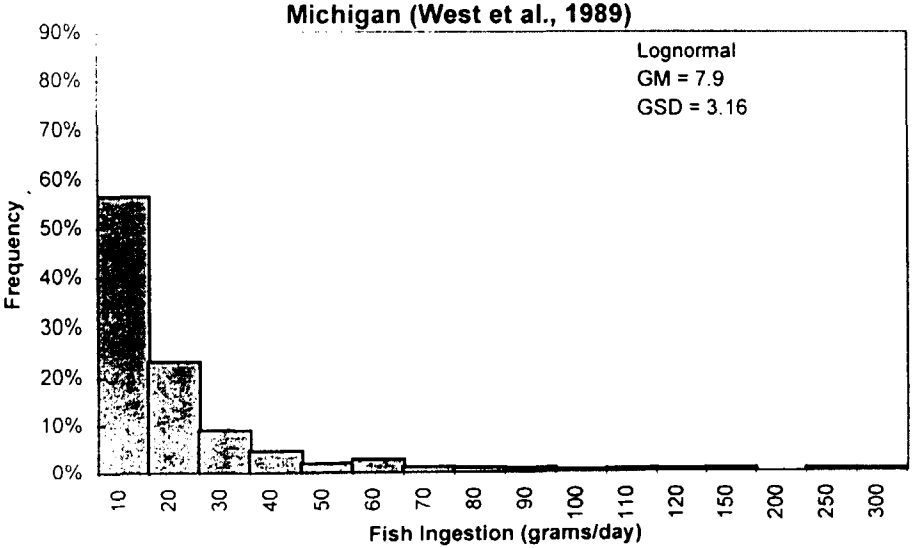


Figure 3-3d (Unchanged)
Frequency Histogram of Self-Caught Fish Ingestion - Maine (Elbert et al., 1993)

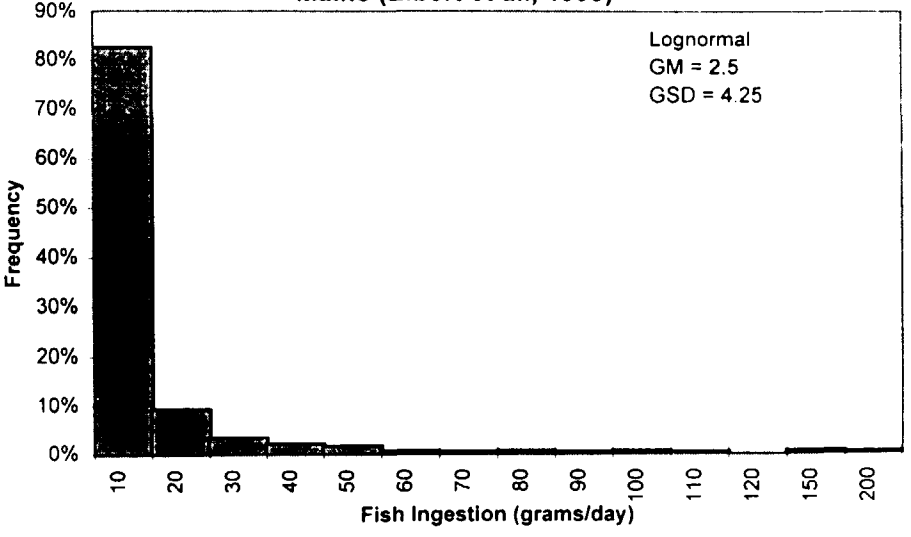


Figure 3-4a (Unchanged)
Fishing Cessation -- Number of Years Until Angler Will Cease Fishing (Derived)

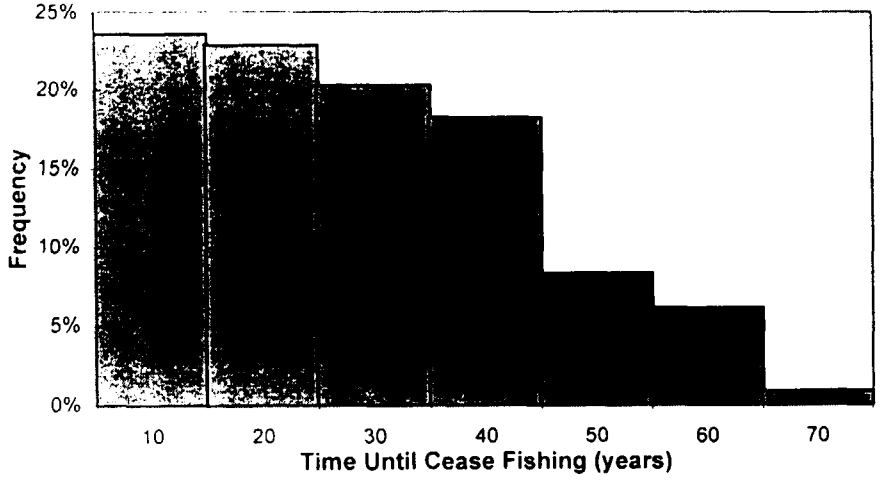


Figure 3-4b (Unchanged)
Age at Which Angler Respondent Reported Began Fishing

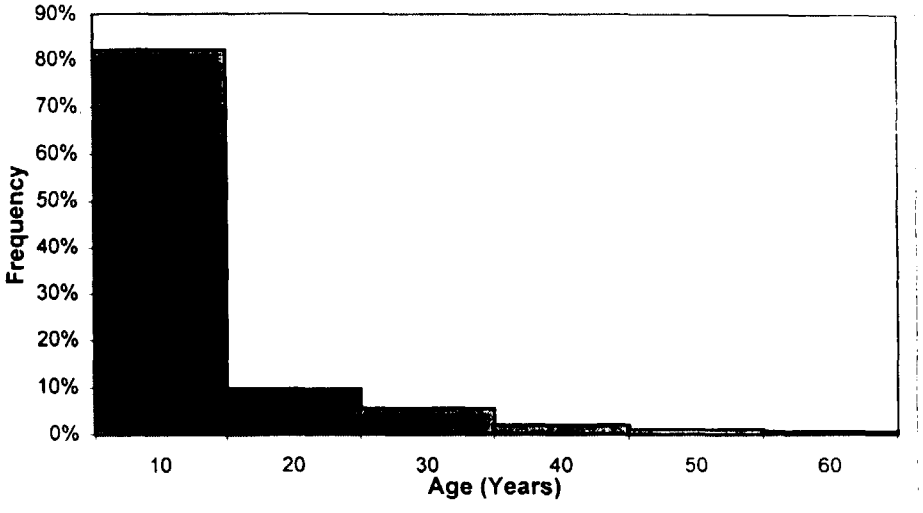


Figure 3-4c (Unchanged)
Current Age of Anglers When Responded to Survey

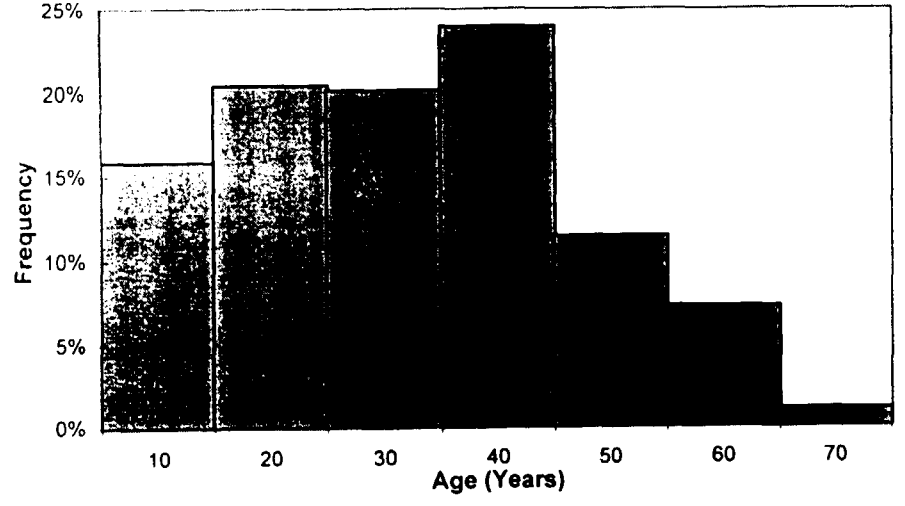
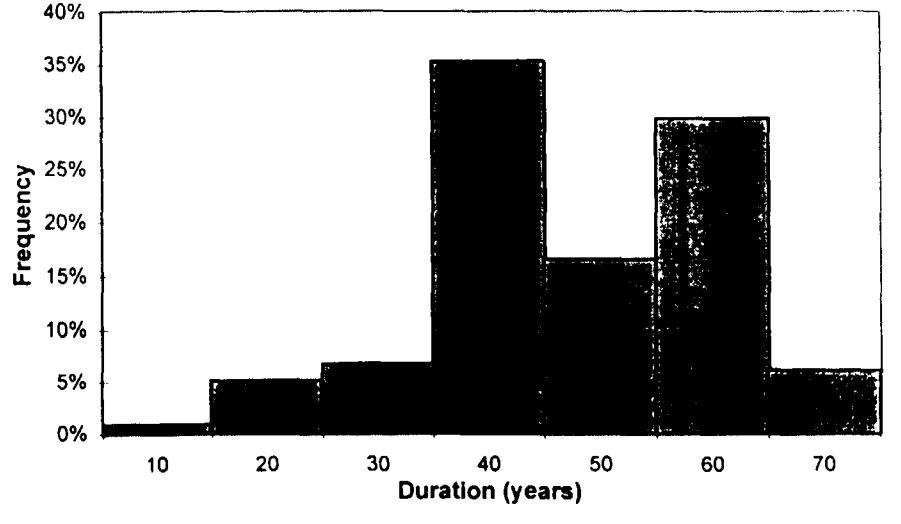
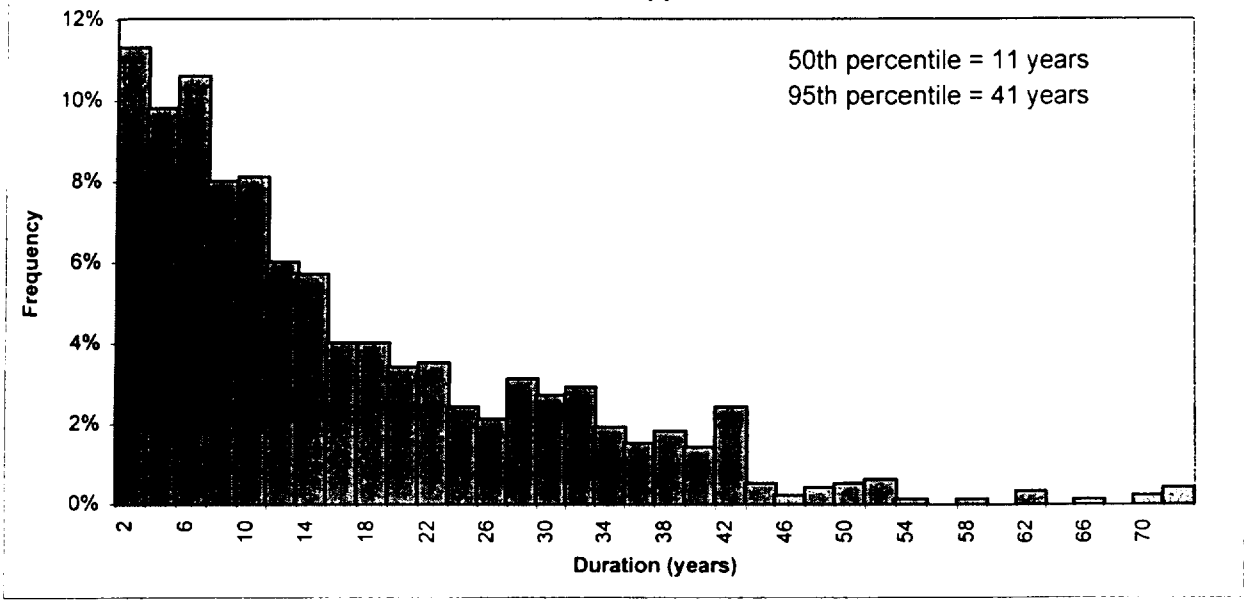


Figure 3-4d (Unchanged)
Total Fishing Duration All Ages (Derived)



Source: Distributions based on 1991 NY Angler Survey (Connelly et. al., 1992).

Figure 3-5a (Unchanged)
Residence Duration in 5 Upper Hudson Counties



Source: Derived using In-Migration data from 1990 Census (see text).

Figure 3-5b (Unchanged)
Overall Exposure Duration
(Combination of Residence Duration and Fishing Duration)

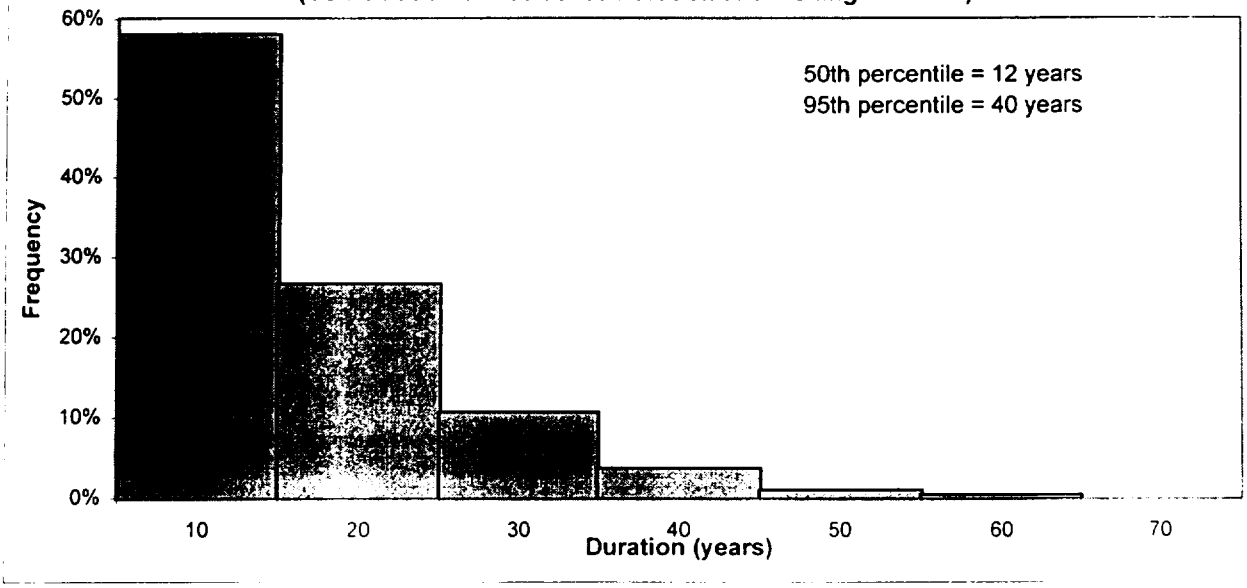


Figure 5-1a (Revised)
Monte Carlo Estimate Non-Cancer Hazards
Base Case Scenario

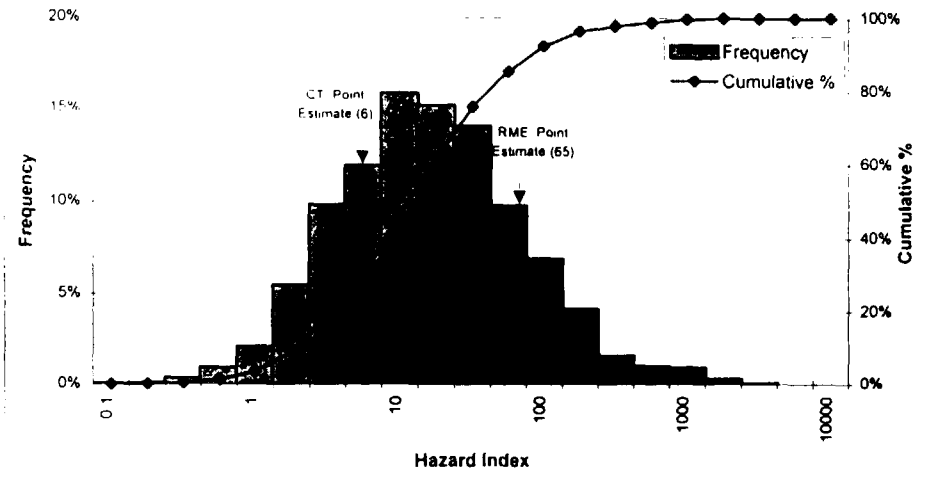


Figure 5-1b (Revised)
Monte Carlo Estimate Non-Cancer Hazards
High End Exposure Duration

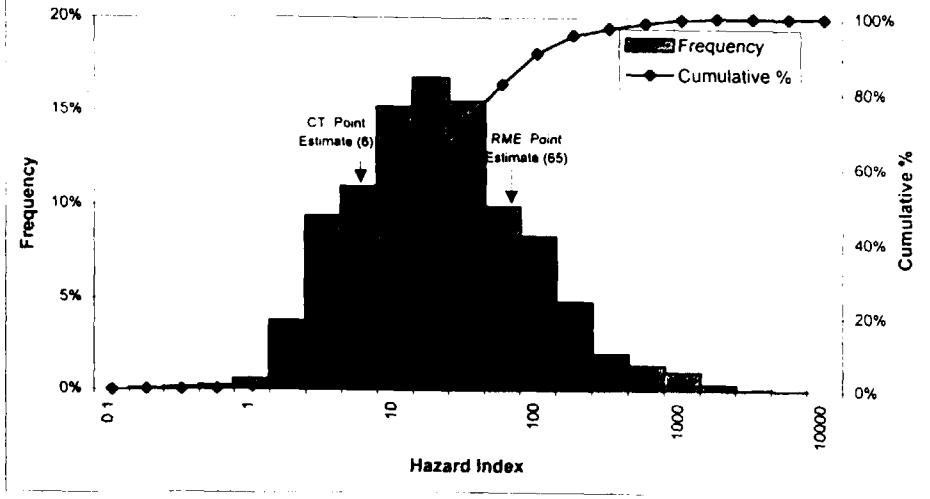


Figure 5-1c (Revised)
Monte Carlo Estimate Non-Cancer Hazards
Maine Fish Ingestion

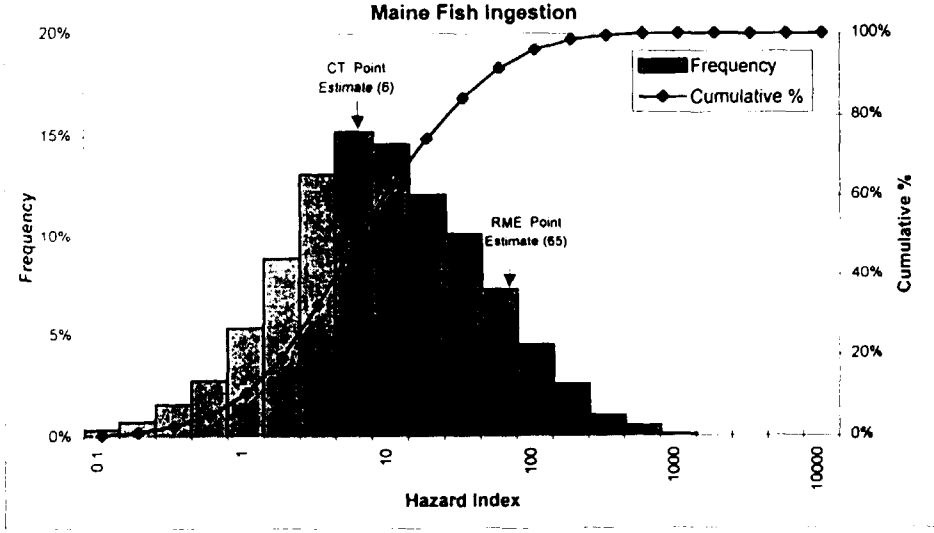


Figure 5-1d (Revised)
Monte Carlo Estimate Non-Cancer Hazards
High End PCB Concentration (Thompson Is. Pool)

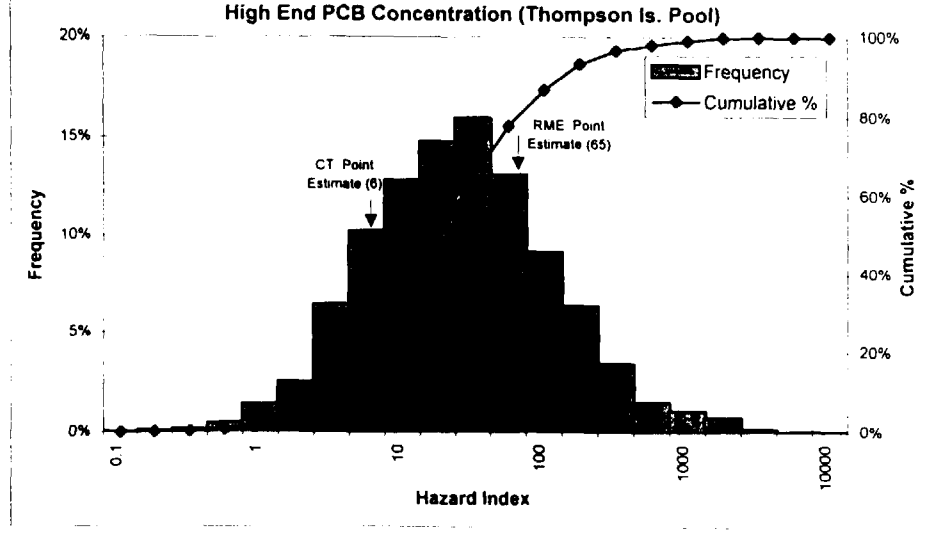


Figure 5-2a (Revised)
Monte Carlo Estimates of Cancer Risk
Base Case Scenario

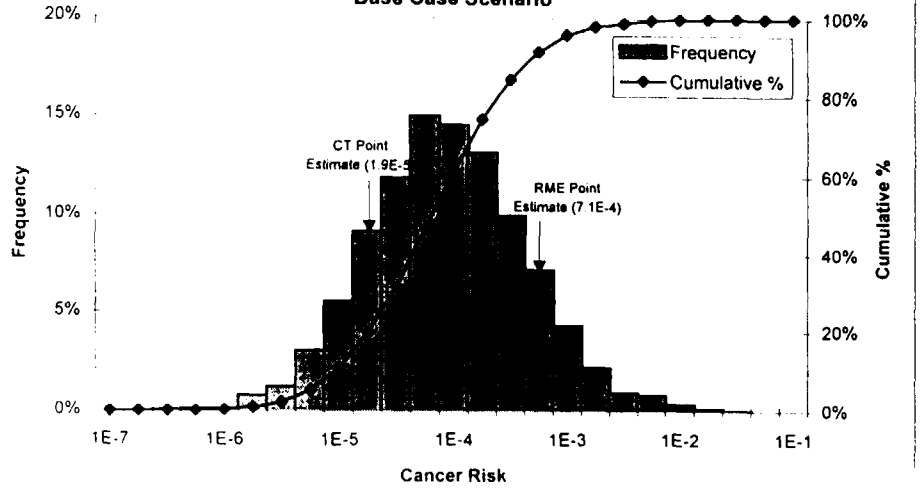


Figure 5-2b (Revised)
Monte Carlo Estimate of Cancer Risk
High-End Exposure Duration

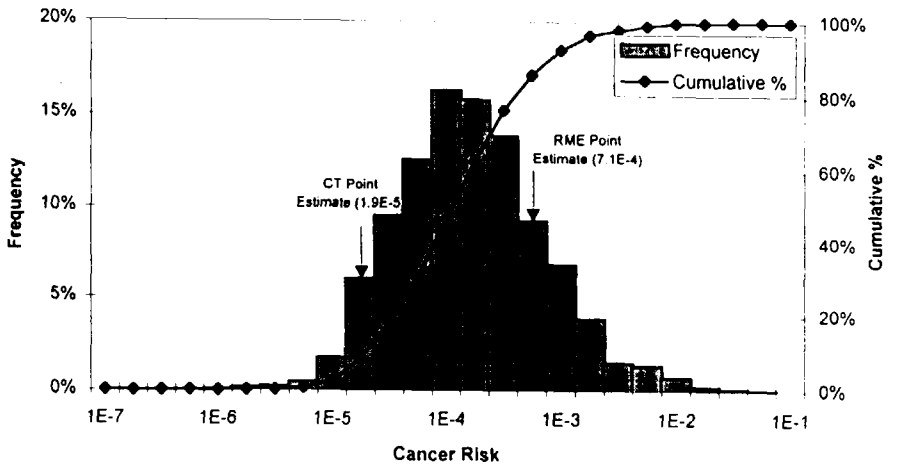


Figure 5-2c (Revised)
Monte Carlo Estimate of Cancer Risk
Maine Fish Ingestion Rate

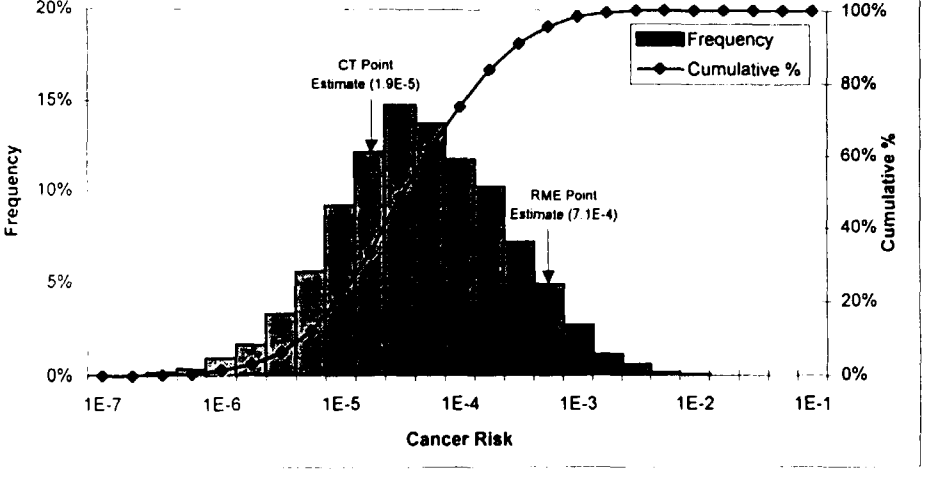


Figure 5-2d (Revised)
Monte Carlo Estimate of Cancer Risk
High End PCB Concentration (Thompson Is. Pool)

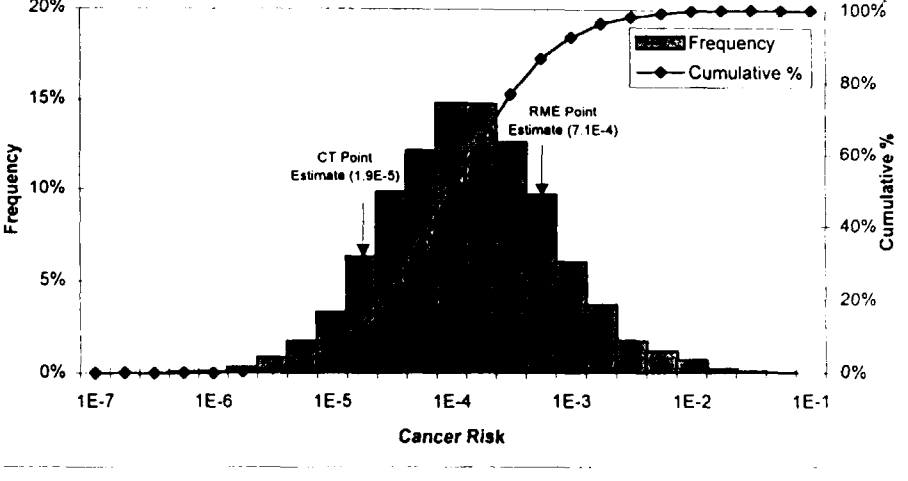


Figure 5-3a (Revised)
Monte Carlo Non-Cancer Hazard Index Summary
All Scenarios

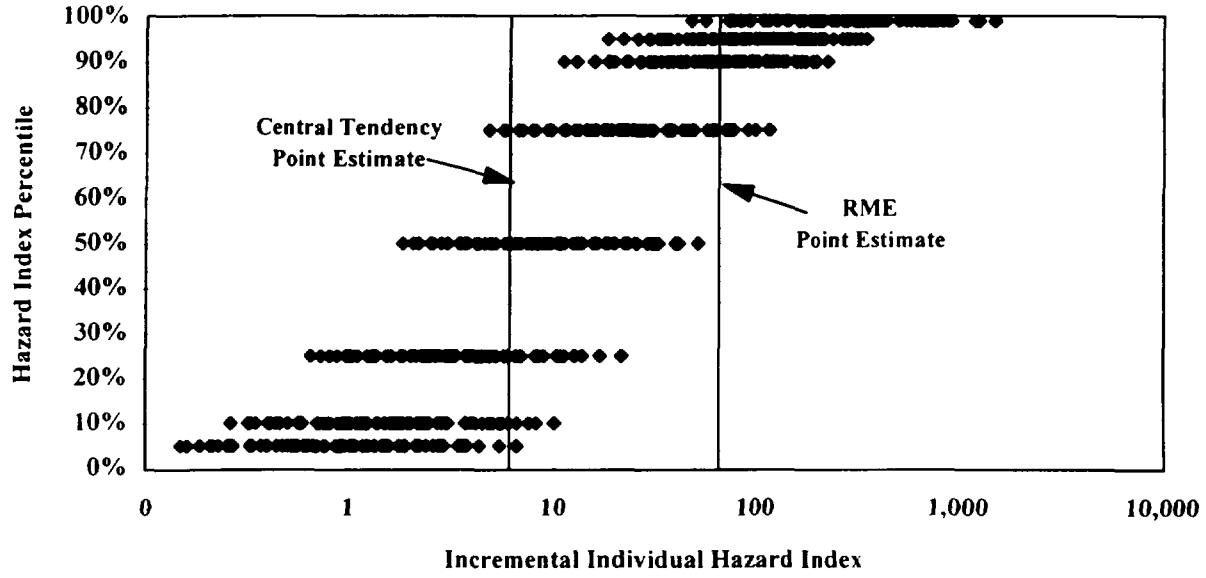
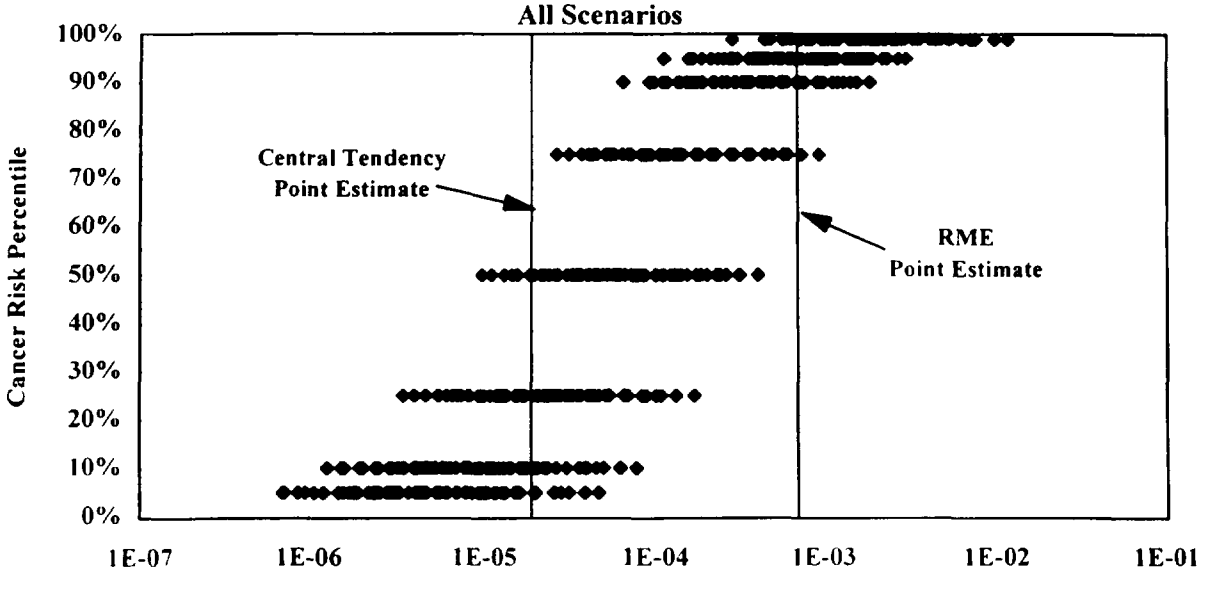


Figure 5-3b (Revised)
Monte Carlo Cancer Risk Summary
All Scenarios



Comments

304680

Federal

U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
National Ocean Service
Office of Response and Restoration
Coastal Protection and Restoration Division
290 Broadway, Rm 1831
New York, New York 10007

HF-1

September 7, 1999

Alison Hess
U.S. EPA
Emergency and Remedial Response Division
Sediment Projects/Caribbean Team
290 Broadway
New York, NY 10007

Dear Alison:

Thank you for the opportunity to review the August 1999 Phase 2 Report - Review Copy, Further Site Characterization and Analysis, Volume 2F- Human Health Risk Assessment, Hudson River PCBs Reassessment RI/FS. The following comments are submitted by the National Oceanic and Atmospheric Administration (NOAA).

Summary

The baseline Hudson River Human Health Risk Assessment (HHRA) assessed exposures and risks for the Upper Hudson study area starting in 1999. The objectives were to update the Phase I HHRA findings and to provide central tendency (50th percentile) and high end (>90th to 99th percentiles) estimates of risk. The HHRA examined potential cancer and non-cancer risks using dose-response relationships for carcinogenicity and systemic toxicity from ingestion of fish, incidental ingestion of sediment, dermal contact with sediment and river water, and inhalation of volatilized PCBs in air. Receptors include angler; adult, adolescent and child recreator; and resident. Species-weighted PCB fish concentration distributions (brown bullhead, largemouth bass and yellow perch), area-weighted sediment and area-weighted water concentration were derived from the Baseline Modeling effort.

Ingestion of fish resulted in the highest cancer risk (3.2×10^{-5} central tendency, 1.1×10^{-3} high end) with the high end or reasonably maximally exposed (RME) 10-1000 times greater than the acceptable risk defined by NCP (10^{-4} to 10^{-6}). Exposure from sediment, air or water were did not result in a significant cancer risk. Ingestion of fish also resulted in the highest noncancer risk where both the central tendency (Hazard Index (HI)=10) and RME (HI=116) point estimates exceeded acceptable levels. Monte Carlo analyses were also performed for the fish ingestion pathway to assess uncertainty and variability. The incremental cancer risk was between 5×10^{-6} (5th percentile) and 9×10^{-4} (95th percentile) for the base case.

Lifetime cancer risks for exposure to sediment or water, or inhalation of air ranged from 10^{-7} the 10^{-8} for central tendency risk and 10^{-5} the 10^{-7} for RME risk. For non-cancer effects, the HQ associated with exposure to sediment and water was significantly less than one.

General Comments

The August 1999 baseline HHRA for the Upper Hudson River covers approximately 40 miles of the Upper Hudson River (from Hudson Falls to the Federal Dam in Troy). A separate HHRA will be performed to evaluate risks to the Mid-Hudson (between the Federal Dam to Poughkeepsie) once the Thomann-Farley modeling effort is completed. Neither of these tasks addresses NOAA's main criticism of the SOW (letter dated 8/28/98) - EPA's failure to evaluate risk to humans from the Lower Hudson River between Poughkeepsie and the Battery. Since the Phase I HHRA concluded that ingestion of fish in the Lower Hudson River would produce similar risks to those determined for the Upper Hudson River, it is unclear why the scope of the proposed activities does not include either a quantitative or qualitative assessment for the Poughkeepsie to Battery stretch of the Hudson River, especially since EPA defines the Superfund Site as extending as far south as the Battery in New York City. Because of the risk identified in the Phase I report and because there is an advisory due to PCBs throughout the Site, a Lower Hudson risk assessment should also be performed. HF-1.1

NOAA submitted extensive comments (dated 7/1/99) on the fate and transport and bioaccumulation components of the baseline modeling effort. These comments should be reviewed and their implications to the HHRA should be considered. There are a number of aspects of the Hudson River system that the fate and transport and bioaccumulation models are not addressing. The absence of these factors may result in significant underestimation of resuspension of sediments and/or PCB loading to the river. This represents major uncertainty in the exposure assessment for the risk assessment, since the future sediment, water and fish tissue PCB concentrations forecasted by these models are used to predict future risk. The implications of the uncertainty resulting from the model inputs to risk assessment should be addressed within the HHRA since the modeled sediment and water concentrations drive the fish exposure concentrations that are used to derive risk to the public. Moreover, future work is planned on the fate and transport and bioaccumulation models. It would be useful to indicate how the data from these supplemental analyses will be incorporated into the models and how they might affect the predictions in the HHRA. HF-1.2

In addition, sediment and water calculations utilized total PCBs assuming a constant upstream boundary condition. The assumption that the upstream boundary condition remains constant ignores the potential impact of high flow events, such as the one experienced in January 1999, on remnant deposits and other high concentration PCB areas in the vicinity of the GE facilities. HF-1.3

The modeled fish data utilized in the assessment need to be evaluated for sufficiency and quality.

The HHRA fails (1) to consider that the calculation of TEQs does not include all the mono- and non-ortho congeners, (2) to acknowledge or discuss data quality issues for fish PCBs (some congeners not reported above detection limits, some not analyzed, some not reliably quantified), (3) to address the underestimation of risk by conducting such comparisons and (3) to evaluate these issues in the uncertainty section. HF-1.4

The Phase I assessment did not quantitatively evaluate risk from floodplain PCB contamination via consumption of home grown crops, dairy products, eggs and meats since data was not available. The Phase 2 assessment should have included data collection from farms along the Hudson River to ensure that risk from floodplains is characterized. HF-1.5

Specific Comments

Page 2: The most recent Hudson River fish advisories can be found in NYSDOH (1999). HF-1.6

Page 8, Section 2.1.3, Para 3: Perspective would be provided if the following were identified with respect to the NYS Department of Agriculture and Markets analysis of milk for PCBs: the total number of dairy farms along the Hudson River, the number of dairy farms along the river sampled for PCBs, and an indication whether analyses were from individual farms or from cooperatives representing a composite sample of many farms. A reference should be provided for this information. HF-1.7

Plant uptake of PCBs has been demonstrated from both sediment and water in the aquatic environment (Lovett Doust et al. 1994). Likewise, floodplain soils could act as a source of PCBs to vegetables or fruit grown at farms and orchards adjacent to the Hudson River. Risks from ingestion of vegetables and fruits grown on potentially contaminated soils has not been adequately addressed. Dr. Buckely investigated forage crops to estimate exposure to farm animals; no evidence is provided about uptake of PCBs in vegetables or fruit grown at farms and orchards adjacent to the Hudson River. HF-1.8

Page 8 Last Para: Documented concentration of PCBs in snapping turtles should be listed. The "avoid eating" advisory of snapping turtle or soups made with their meat due to PCBs for women of childbearing age, infants and children under the age of 15 (NYSDOH 1998, pg. 14) should have been noted. HF-1.9

Page 16, Section 2.3.3: Water PCB concentrations are averaged across 47 river segments and then averaged over time. This treatment of water data ignores how the public may be exposed to this medium, primarily through wading, swimming and fishing. Nearshore areas are more accessible to the public than mid channel areas. They also represent important habitat for fish. Hence, they are more likely to present a more relevant exposure pathway to human receptors. Water column concentrations have been shown to differ in nearshore and midchannel areas with higher concentrations in the nearshore and backwater areas. HF-1.10

Page 17 Para 1: PCB-contaminated sediment, floodplain soils and river water potentially contribute to PCBs in the air. HF-1.11

Page 50: The exposure assessment assumes a start date of 1999. The assumption that no exposure occurred prior to that date, could underestimate risk. HF-1.12

Page 69: TEQs were calculated from the Upper Hudson River Phase II fish data. TEQs were calculated from a select group of congeners. The standard calculation multiplies the concentration of a given mono- and non-ortho chlorinated congener by the toxic equivalency factor (TEF) and then all of these values are summed. The congeners in this summation are BZ #'s 77, 81, 105, 114, 118, 123, 126, 156, 157, 167, 169, and 189. The text should specify what congener data was available for calculating TEQs for each species. Moreover, the central and high end total dioxin TEQ derived from the wet weight data are underestimates since an incomplete list of non-ortho and mono-ortho congeners were measured and available for the calculation. This is important in assessing risk and should be clearly explained. HF-1.13

Page 74 Para 6: There is an assumption that sediment and water concentrations will decrease with time but this ignores the potential of a high flow event resulting in the remobilization and release of PCBs from remnant deposits and other high PCB concentrations in the Upper Hudson, thereby modifying sediment, water and fish concentrations. Increases in sediment or water concentrations could have a cascading effect on PCBs in the trophic foodweb and on exposure to anglers and recreators. In addition, uncertainty would decrease if fish concentrations were modeled out to the year 2069 instead of extrapolating out beyond the year 2018. HF-1.14 HF-1.15

Page 75: The uncertainty section on air does not discuss volatilization from sediments exposed during the draw down of water by the hydroelectric facilities on the Upper Hudson. HF-1.

Page 76: The uncertainty section on TEF for dioxin-like PCBs does not address data quality issues and lack of specific mono- and ortho-congener fish data used to derive TEQs. HF-1.17

Thank you for your continual efforts in keeping NOAA apprised of the progress at this site. Please contact me at (212) 637-3259 or Jay Field at 206-526-6404 should you have any questions or would like further assistance.

Sincerely,

Lisa Rosman
NOAA Coastal Resource Coordinator

cc: Mindy Pensak, DESA/HWSB
Marian Olsen, ERRD/PSB
Gina Ferreira, ERRD/PSB
Robert Hargrove, DEPP/SPMM
Charles Merckel, USFWS
William Ports, NYSDEC
Ron Sloan, NYSDEC
Anton P. Giedt, NOAA

State

New York State Department of Environmental Conservation
Division of Environmental Remediation
Bureau of Central Remedial Action, Room 228
50 Wolf Road, Albany, New York 12233-7010
Phone: (518) 457-5637 FAX: (518) 457-7925

John P. Cahill
Commissioner

HS-1

SEP 7 1999

Post-it® Fax Note 7671	Date 9/10/99	# of pages ▶ 31
To Alison Hess	From Bill Ports	
Co./Dept. EPA	Co. NYSDEC	
Phone # 212-637-3959	Phone # 518-457-5637	
Fax # 212-637-4439	Fax #	

Ms. Alison Hess
Project Manager
US Environmental Protection Agency
290 Broadway, 19th Floor
New York, New York 10007-1866

Dear Ms. Hess:

Enclosed are comments on the August 1999 Phase 2 Report - Further Site Characterization and Analysis, Volume 2E - Human Health Risk Assessment, Hudson River PCBs Reassessment RI/FS. The comments were prepared by the New York State Department of Health.

If you have any questions regarding the comments, please contact me at (518) 457-5637.

Sincerely,

William T. Ports, P.E.
Project Manager
Bureau of Central Remedial Action
Division of Environmental Remediation

cc: John Davis, NYSDOL
Robert Montione, NYSDOH
Jay Fields, NOAA
Lisa Rossman, NOAA
Anne Secord, USF&W

STATE OF NEW YORK
DEPARTMENT OF HEALTH

Flanigan Square, 547 River Street, Troy, New York 12180-2216

Antonia C. Novello, M.D., M.P.H.
Commissioner

Dennis P. Whalen
Executive Deputy Commissioner

September 7, 1999

Mr. William Ports
Bureau of Environmental Remediation
New York State Department of Environmental Conservation
50 Wolf Road
Albany, NY 12233

Re: Human Health Risk Assessment
Hudson River PCBs
Saratoga County
Site #546031

Dear Mr. Ports:

We have reviewed the United States Environmental Protection Agency's (US EPA) August 1999 "Phase 2 Report – Review Copy, Further Site Characterization and Analysis, Volume 2 F - Human Health Risk Assessment, Hudson River PCBs Reassessment RI/FS." The assessment provides the results of the analysis detailed in the EPA's Scope of Work, which we reviewed in 1998. We agree with the overall conclusion of the assessment that the highest estimated human health risk due to PCBs in the Hudson River is from fish ingestion and that other routes of exposure are of less risk. However, as described below, we have a number of comments on the assessment.

GENERAL COMMENTS

1. The assessment does not include a quantitative evaluation of many possible residential exposure pathways. These pathways include soil and sediment ingestion, dermal contact with sediments and river water, incidental ingestion of river water, homegrown vegetable ingestion and the ingestion of beef and dairy products produced at current or future farms along the floodplain. While the environmental data needed to evaluate these pathways may be limited at this time, to the extent feasible, a quantitative evaluation of all relevant young child and adult residential exposure pathways is needed to characterize the possible risks to residents. HS-1.1

2. NYS DOH staff have completed a preliminary comparison of elements of the assessments prepared by US EPA's consultants for the Hudson River and Rogers Island sites. This comparison identifies numerous differences in the approaches used in the two risk assessments (e.g., different receptors/pathways evaluated, differences in certain exposure parameter values, differences in the toxicological parameters). US EPA should use similar approaches in the HS-1.2

Hudson River and Rogers Island risk assessments unless there are valid technical reasons for not doing so.

3. In a May 20, 1998 letter from Robert Montione to William Ports of the NYS Department of Environmental Conservation, New York State Department of Health (NYS DOH) staff provided comments on the US EPA Scope of Work for this assessment. A copy of that letter is attached for your reference. A number of our comments on the Scope of Work were not addressed in the assessment. Some of the comments not addressed include the following:

- The point estimates of high-end risk should assume lifetime Hudson River fish consumption (comment 3). HS-1.3
- The risk assessment should address the effects of past exposures on current and future exposures and risks (comment 4). HS-1.4
- Statements to the effect that "sub-populations of highly exposed and lesser exposed anglers will be represented in the distributions of risk generated in the Monte Carlo analysis" need to be specifically supported (comment 6). HS-1.5
- The discussion of the Monte Carlo results should include a sensitivity analysis that addresses how using different assumptions about range, frequency or nature of the distribution (e.g., normal, lognormal, uniform) affects the outcome (comment 9). HS-1.6

Addressing these issues would provide valuable information to risk managers.

EXECUTIVE SUMMARY

Executive Summary (page ES-4), and Section 1.4 (page 3) and Section 5.1.2 (page 68)

Statements about the NCP acceptable risk range for carcinogens are misleading to the reader and should either be deleted from the risk assessment document or revised to reflect the NCP and EPA risk management policy. Cancer risks of 1.0 E-6 or less are usually considered insignificant and not a public health concern. Cancer risks greater than 1.0 E-4, on the other hand, typically will trigger actions to lower exposures. When cancer risk estimates are between 1.0 E-6 and 1.0 E-4, a risk management decision must be made on a case-by-case basis whether or not to pursue risk reduction measures. The NCP and EPA state (e.g., US EPA, 1991, Risk Assessment Guidance for Superfund: Volume I – Human Health Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals), Office of Emergency and Remedial Response, p. 18) the preference for managing risks at the more protective end of the risk range, other things being equal. Preferably, statements about acceptable risk should be deleted from the risk assessment document. If, on the other hand, US EPA determines that such a discussion should be included, then the contractor must provide an accurate and balanced discussion of the risk management process to avoid the perception that as long as the risks fall in the 1.0 E-6 to 1.0 E-4 range, they are *a priori* deemed acceptable. HS-1.7

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CHAPTER 2 - EXPOSURE ASSESSMENT

1. The *PCB Concentration Weighted by Species-Consumption Fractions* section on pages 13 and 14 describes how the assessment classified six species of fish consumed by Hudson River anglers into three groups. There are several deficiencies in this section: HS-1.8

- Group 1 inappropriately uses brown bullhead PCB data to represent PCB levels in American eels and carp. Because brown bullhead generally have lower PCB levels than American eels or carp, exposures from eating Group 1 fish are underestimated. For example, in 1992 collections at Stillwater, average PCB levels were 9.4 ppm in brown bullhead, versus 33.7 ppm in American eel and 38.6 ppm in carp.

- The composition of Group 2 is reasonable, but the third sentence in the second complete paragraph on page 14 states that bass and walleye reach "several feet in length". The term "several" implies that these fish achieve three or more feet in length. Since bass or walleye rarely reach three feet in length this statement is misleading and should be removed.

- No rationale is provided for Group 3 (yellow perch).

- This assessment did not consider white perch consumption because "they're not commonly found in the Upper Hudson River" (last paragraph on page 12). This is inappropriate because white perch was the most frequently caught species (19.7% of all fish caught) in the 1991-1992 Clearwater survey in these waters (between Hudson Falls and Troy).

2. The assessment addresses child fish consumption in the Monte Carlo assessment, but not in the deterministic assessment. PCB exposures and risks from fish consumption should be assessed for at least the high-end child fish consumer. Although most angler surveys do not provide direct measures, fish consumption rates for children can be estimated by applying child/adult fish consumption rate data from other sources to findings from the angler studies of interest. For example, data on meal sizes from Pao et al. (1975, page 264-265) indicate that the average fish meal size for a 1-2 year old child is 68 grams and the average fish meal for a 19-34 year-old male is 191 grams; thus, the child/adult meal ratio is 68/191 = 0.36. If you assume the child eats Hudson River fish whenever the parent does, the child fish consumption rate could be assumed to be equal to the adult consumption rate multiplied by 0.36. HS-1.9

3. In order to expedite the Feasibility Study, the risk characterization section (Section 5) should include a comparison of the modeled fish concentration over time for the different sections of the Upper Hudson to the FDA tolerance level of 2 ppm, which is an Applicable Relevant and Appropriate Requirement (ARAR). HS-1.10

4. The assessment assumes that the high-end fish consumer eats Hudson River fish for 40 years, based on census data regarding local residence duration and survey data on how long an individual fishes. There are two flaws in this approach: HS-1.11

- If the conditional probability of moving out of the area is lower for individuals who have already lived in the area for a long period of time, it is possible that US EPA will have

underestimated the fraction of the population whose residence times are very long.

- The assessment assumes that only anglers consume Hudson River fish, so that individuals are only exposed during the part of their lives when they are fishing. This assumption is faulty because angling is often a family tradition where the catch is shared by the extended family, and it is likely that Hudson River fish are included in family meals. Thus, individuals may eat Hudson River fish for their entire lives even if they themselves do not fish or they fish for just a portion of their life.

Based on the likelihood that some avid anglers/fish consumers will reside near and eat Hudson River fish for their lifetimes, we believe the point estimates of high-end risk should assume lifetime consumption of Hudson River fish.

CHAPTER 3 – MONTE CARLO EXPOSURE ANALYSIS of FISH INGESTION PATHWAY

US EPA used the Monte Carlo analysis of the fish ingestion pathway as a means for evaluating its deterministic assessment of the pathway. The comments below are of limited importance given this use of the Monte Carlo analysis. However, if the scope of the Monte Carlo analysis is expanded for any reason, these comments are of greater importance; in addition, we may have additional comments.

1. The Monte Carlo assessment of the fish consumption pathway relies on a number of assumptions that are not supported. For example, the probability that someone moves out of the region is assumed to be a function of age and the assessment assumes that body weights in the population are perfectly correlated over time. Although the Monte Carlo assessment was used to evaluate the CT and RME point estimates of risk, the assumptions used in the Monte Carlo exercise were not evaluated in a sensitivity analysis; therefore, the significance of their potential impact on the outcome of the assessment is unknown. A sensitivity analysis should be done on all the important parameters in this pathway and the significance of the outcome should be discussed in the assessment. HS-1.12

2. The fish concentrations used in the assessment were taken from the 1999 Baseline Modeling Reports. While the modeling exercise only predicted fish concentrations through the year 2018, the risk assessors extrapolated the modeled results to the year 2069 using an exponential trend/regression line. The assessment should discuss why the fish concentrations were extrapolated to 2069 while the point estimates for concentrations in river water and sediment were based on only the modeled concentrations to the year 2018. The assessment should discuss that the fish concentrations are predicted based on current conditions and that there is no guarantee that future events (similar to 1991 plant site releases) will not occur to change the accuracy of these predictions. HS-1.13

CHAPTER 4 - TOXICITY ASSESSMENT

The assessment maintains an artificial dichotomy between the toxicity values for the cancer and non-cancer effects of PCBs. Three examples of this dichotomy are shown below.

- The toxicity values used to evaluate the cancer and non-cancer human health risks of the same exposure (sediment ingestion, dermal contact with sediment, dermal contact with water) are not based on the same Aroclor(s). The dichotomy is not supportable and should be reconciled.

	Aroclor on Which the Toxicity Value is Based	
Exposure Route	**Cancer Slope Factor**	**Reference Dose**
water ingestion	not evaluated	not evaluated
fish ingestion	1254/1260	1254
sediment ingestion	1254/1260	1016
dermal contact with sediment	1254/1260	1016
dermal contact with water	1242	1016
inhalation	1242 (oral study)	not evaluated

- On page 63, it is explained that the RfD for Aroclor 1016 (and not Aroclor 1254) was used to evaluate the non-cancer risks from PCBs in sediments because the congener profile in the sediments more closely resembles Aroclor 1016 than Aroclor 1254. It also is explained that the RfD for Aroclor 1254 (and not Aroclor 1016) was used to evaluate the non-cancer risks from PCBs in fish because the congener profile in fish more closely resembles Aroclor 1254 than Aroclor 1016. We agree with these choices and the scientific reasoning supporting the selections. HS-1.14

  We suggest that the same scientific reasoning for selecting RfDs should be applied to the selection of cancer slope factors (CSFs) to evaluate the cancer risks of exposure to sediments and air. We recommend that the cancer risk assessment for these media follow the advice given in the IRIS datafile for PCBs in Section II.B.4. Discussion of Confidence (Carcinogenicity, oral exposures): "When available, congener information is an important tool to define a potency estimate that was based on exposure pathway." The consideration of dioxin-like PCBs in the assessment of the cancer risks from fish exposures is consistent with this advice. If the CSFs used to assess sediment and air exposures do not change, then the uncertainty associated with using CSFs for Aroclor mixtures that may not adequately match the environmental mixtures found in sediments and air should be discussed in the section on Risk Characterization.

- On page 63, it is stated that the non-cancer risks of inhaling PCBs were not assessed because there are no Reference Concentrations for either Total PCBs or any Aroclor mixture. This situation should not prevent the assessment of non-cancer risks from air exposures. Data from ingestion studies are used to evaluate risks from other routes of exposures in three cases. (1) Oral CSFs for ingestion exposures are used to calculate unit risks values for evaluating inhalation exposures. (2) Oral CSFs for ingestion exposures are used to evaluate HS-1.15

the cancer risks of dermal exposures. (3) An oral RfD is used to evaluate the non-cancer risks of dermal exposures. We recommend two additions: (1) the evaluation of air exposures using RfDs and (2) a discussion of the uncertainties inherent when oral studies are used to assess the cancer and non-cancer risks of dermal and inhalation exposures.

CHAPTER 5 - RISK CHARACTERIZATION

1. The discussion (pages 76-77) does not fully characterize the uncertainties in the toxicity assessment. Three major areas could be more fully discussed.

- The discussion does not fully characterize the uncertainty that arises when estimated human PCB exposures are compared to the non-cancer results of animal studies published after the completion of the IRIS RfDs.

The study by Arnold et al. (1995) on reproductive effects seen in rhesus monkeys should be more fully discussed. Arnold et al. (1995) reported that statistical analysis of the conception rates showed that they were significantly lower in those females ingesting 20, 40, or 80 ug Aroclor 1254/kg/day (P-values of 0.007, 0.043, and 0.003, respectively), and approached significance ($P < 0.059$) in those females ingesting 5 ug Aroclor 1254/kg/day. Moreover, the study also showed that infants of monkeys ingesting 5 ug Aroclor 1254/kg/day showed clinical signs of toxicity during nursing. These effects included inflammation and/or enlargement of tarsal glands, nail bed prominence, elevated nails, nails folding on themselves, and gum recession. These findings, especially the potential effects on reproductive success, should be discussed before concluding that the IRIS RfD for Aroclor 1254 is considered to be "health protective" (page 76). The RfD was derived using, among other factors, a reduced uncertainty factor of 3 because the changes observed in the adult monkeys were not considered to be of marked severity. The new data suggest that the margin of protection afforded by the IRIS RfD may be less. HS-1.16

The average daily dose for an high-end angler is 2.3 ug/kg/day. The LOEL used to derive the Aroclor RfD is 5 ug/kg/day. Thus, the angler's dose is close to the LOEL. The perception of risk at this dose differs with the nature of the end-points observed at the LOEL. Concern increases with the severity of the observed effects. The draft discussion implies that the only effects seen at the LOEL were mild dermal and immunological effects in the adults. It does not fully address the potential that more severe effects (failure to conceive, developmental toxicity) may also occur at the same LOEL.

Recent studies on rhesus monkeys show long-term behavioral effects in young animals dosed with 7.5 ug/kg/day of Aroclor 1254 from birth to 20 weeks of age (Rice, 1999a). This dose was chosen because it represented a breast milk dose considered "safe" by Health Canada. Moreover, it lead to blood and fat levels in the monkeys that were within the range of levels seen in the human population. The doses ingested by child anglers, who may consume PCB contaminated fish, should be compared to this LOEL to obtain information on potential risks of neurobehavioral effects. As stated elsewhere, an evaluation of the non-cancer risks of fish consumption by children could be included in the assessment.

- There is a large body of information on the potential reproductive and developmental effects of consuming sport-fish containing PCBs and other contaminants (see attached bibliography). Estimated fish consumption rates and PCB intakes from Hudson River fish could be compared to fish consumption rates and expected PCB intakes (when available) associated with effects in cohort studies in New York State, Michigan, Wisconsin, Sweden, and Quebec. Such an analysis could provide valuable human data to support/contradict the statement (page 76) that the IRIS RfD is considered to be "health protective."

- As stated earlier, the uncertainty associated with using CSFs for Aroclor mixtures that may not adequately match environmental mixtures found in sediments and air should be discussed.

2. The third paragraph of section 5.3.2 (page 76) could be revised to present clearly the summary information (critical studies, critical effects, and uncertainty factors) for the Aroclors 1016 and 1254. HS-1.17

APPENDIX C - TOXICITY PROFILE

The profile is not an up-to-date review of PCB toxicity because it limits itself largely to material contained in the IRIS datafiles for PCBs, Aroclor 1016, and Aroclor 1254. Since the IRIS files were completed, new information has been published, and important studies on the oncogenic, reproductive, and developmental toxicity of PCBs could be incorporated into the text. This is not a request to make the section longer, but to re-focus the section on important studies that are critical to understanding the potential public health risks of environmental exposures. Several suggestions follow: HS-1-18

1. The section on the carcinogenic potential in humans could include a discussion of the potential links between PCBs and specific cancer types (i.e., melanoma, non-Hodgkin's lymphoma, and breast cancer) (see attached bibliography).

2. The discussion on PCBs and breast cancer in the Summary of Non-Cancer Effects in Humans (page C-4) should be placed in the section on the carcinogenic potential in humans.

3. The discussion on potential effects associated with background exposure to PCBs, including PCBs in fish, could be more fully developed. This is a major area of uncertainty. The summary statements on studies Lanting/Patandin (Dutch studies) should be compared with animal studies and other human studies. The discussion could include the findings of cohort studies in New York State, Michigan (infant and adult studies), Sweden, and Quebec on the possible development, reproductive, and neurotoxic effects associated with the consumption of fish containing PCBs and other contaminants (see attached bibliography).

4. The studies by Lanting/Patandin assessed the non-cancer effects of background exposures to PCBs. A recent publication indicates that only a small percentage of a child's daily exposure is from fish (Patandin et al., 1999a). Thus, they are not, as indicted on page C-4, studies of children consuming PCBs in fish.

5. The discussion of non-cancer effects does not include all of the recent studies on reproductive and developmental effects seen in low-dosed animals. Several studies published after the IRIS RfDs for Aroclors 1016 and 1254 were derived could be identified and briefly discussed (see attached bibliography). These include studies (e.g., Arnold et al., 1995; Rice, 1999a) on the reproductive, developmental, and neurobehavioral effects of low-level Aroclor 1254 exposures in rhesus monkeys.

If you have any questions please call me at (518) 402-7870.

Sincerely,

Robert J. Montione, Public Health Specialist III
Bureau of Environmental Exposure Investigation

Attachments

cc: Mr. Tramontano
Dr. Kim
Dr. Carlson/ Dr. Wilson
Dr. Horn/Dr. Grey
Mr. Fear GFDO
Mr. Daigle DEC
Mr. Steenberge DEC Reg. 5
Mr. Ulrich ATSDR

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Local

SARATOGA COUNTY

ENVIRONMENTAL MANAGEMENT COUNCIL

PETER BALET
CHAIRMAN

GEORGE HODGSON
DIRECTOR

HL-1

September 2, 1999

Alison A. Hess, C.P.G.
USEPA Region 2
290 Broadway, 19th Floor
New York, New York 10007-1866

Attn: Hudson River HHRA
ERA Comments

Dear Ms. Hess:

Enclosed you will find the Saratoga County Environmental Management Council's (SCEMC's) comments prepared by member David Adams on the Hudson River PCB's Reassessment Phase 2 Human Health Risk and Ecological Risk Assessment Reports.

The Council, although sensitive to the need to provide conservative estimates when assessing human health and ecological risks related to the Hudson River PCB Reassessment, finds both the HHR and ER Assessments to reflect an unrealistic degree of "scientific" over-conservatism often based upon inaccuracies and what we believe to be fallacious scientific assumptions.

Sincerely,

George Hodgson, Jr.
Director

Enc.
cc: Doug Tomchuk, USEPA, Region 2
SCEMC Members
Darryl Decker, Chr., Government Liaison Committee, CIP
The Honorable John Sweeney

50 WEST HIGH STREET BALLSTON SPA, N.Y. 12020 (518) 884-4778

SARATOGA COUNTY

ENVIRONMENTAL MANAGEMENT COUNCIL

PETER BALET
CHAIRMAN

GEORGE HODGSON
DIRECTOR

COMMENTS ON THE PHASE 2 HUMAN HEALTH RISK ASSESSMENT
VOLUME 2F, BOOK 1; AUGUST 1999
HUDSON RIVER PCBs REASSESSMENT RI/FS

Prepared by: David D. Adams, Member, Saratoga County EMC and Government Liaison Committee
August 30, 1999

1. **Executive Summary, Ingestion of Fish, P.ES-2 and Section 2-1.2, P.7:** The fish ingestion rates are based on people ignoring the NYS ban on eating fish from the Upper Hudson River. In view of all the other conservatisms in this assessment this is an overly conservative approach, especially for the RME person, as will be discussed in later comments on fish ingestion rates. As a minimum, EPA should include risk factors using a best-estimate of the degree the NYS consumption ban is honored in order to give a better perspective of the risks to human health. HL-1.1

   The health risks should be calculated for fish PCB concentrations for each separate reach of the river rather than averaging the fish PCB concentrations over the entire Upper Hudson River. This would give a better perspective of the risks along the river considering the significant reduction in fish PCB concentrations with decreasing river miles.

2. **Executive Summary, Toxicity Assessment, P. ES-4:** It is unfair that EPA chooses only to comment on possible problems/limitations on the study sponsored by GE. The implication is that the other studies cited have no imperfections. Later comments will more fully address this subject. HL-1.2

3. **Section 2.3, P. 10:** A 95% confidence limit on the mean PCB concentration should be calculated. Certainly there is information on the possible variation of the input parameters to the PCB concentration model so model calculations can be made to generate information from which to determine confidence limits. HL-1.3

4. **Section 2.3.1, P. 11 and Section 5.3.1, P. 72:** Both of these sections discuss the models used to calculate the fish PCB concentrations and reference the Baseline Modeling Report issued in 1999. At meetings in 1999, EPA (or its contractors) acknowledged deficiencies and/or errors in both the PCB Fate and Transport model and the FISHRAND model. Neither of these sections indicate whether the Human Health risk assessment in this report is based on the model as presented in the published 1999 Baseline Modeling Report or on some unpublished correction to that report. If the former is true, publication of this report is premature and the risks presented should be withdrawn until the corrected results are available. If the latter is true, then it is unfair to ask for review of a report that is based on information not available to the reviewer and again, the risks presented should be withdrawn until the correct modeling information is presented. It is also noted that there are unresolved differences between the EPA and GE models and that the EPA model has not been peer reviewed. Both of these factors could change the calculated risks in this report making the presentation of the risks at this time premature. HL-1.4

50 WEST HIGH STREET BALLSTON SPA, N.Y. 12020 (518) 884-4778

5. **Section 2.3.1, P. 12:** It is not understood why the model was not run out to 70 years rather than extrapolating the 20 year curve. Running the model out to 70 years should give better values than extrapolation and should be done (unless EPA has no faith in their model's ability to forecast for 70 years). HL-1.5

6. **Section 2.3.1, P. 13:** The information from the NYSDOH 1996 study should be presented even if it is "limited." The use of "limited" information in other areas has not bothered EPA (for example, see P. 48 where the use of only 226 respondents is used to assess the type of fish consumed and without any knowledge of even how many of the 226 fished the Upper Hudson River). HL-1.6

7. **Section 2.4.1, P. 22:** The formula for PCB exposure assumes 100% of the PCBs ingested are retained for the duration of the exposure. EPA should provide justification for this assumption which seems overly conservative. HL-1.7

8. **Section 2.4.1, P. 24 and Section 3.2.2, P. 48:** The information presented on the 1,000 New York anglers (Connally, et al, 1992) is incomplete. EPA should provide information on how many of the 1,000 anglers fished in the Upper Hudson River and of this number, how many reported eating fish from the Upper Hudson River. Of those eating fish from the Hudson River, what were the percentile values for fish consumed? Also, EPA should provide an assessment of the statistical validity of the small sample size available for the Upper Hudson River versus the estimated 10,000 plus anglers estimated to fish this area (see Section 2.1.2, P-7) HL-1.8

9. **Section 2.4.1, P. 25:** There is no basis for assuming that people who eat more fish (the RME person) eat the pan drippings. Unless EPA can provide data to substantiate this assumption, the 20% cooking loss should apply to the RME estimates. HL-1.9

10. **Section 3.2.1.1, P. 39:** The discussion of the "1992 Lake Ontario Diary Study" indicates that 12 month recall responses of fish consumption were higher than those from diary data but fails to give any information on the amount of the overestimate. EPA should provide this information as it represents another conservatism in the risk estimates. HL-1.10

11. **Section 3.2.1.1, P. 39:** While EPA provides a discussion of the "1992 Lake Ontario Diary Survey" EPA fails to provide any discussion of the "additional Connally Surveys" of 1993. The reason for this omission is not given. It can not be that the 1993 surveys focused on Great Lakes anglers because the 1992 Diary Study also focused on Great Lakes anglers. EPA should either discuss the 1993 surveys or explain why the 1993 surveys were ignored. HL-1.11

12. **Section 3.2.1.1, PP. 39 & 40 and Section 3.2.1.4, P. 5:** Presenting only the combined results of the 1996 and 1991-92 surveys is unacceptable. EPA is requested to provide the separate survey results so that a better assessment of the significance of the survey results can be made. What was the rationale for including the percentages at the bottom of P. 39? It is not clear what relationship any of the categories specified have to the risk assessment. The 1996 survey results (NYSDOH, 1999) at the top of P. 40 are significant in that they indicate the assumption that fish from the Upper Hudson River are eaten despite the NYS ban is incorrect. Using the 1996 survey results 92% of the surveyed anglers don't eat the fish, would cause the calculated risks to decrease by a factor of 10. The 1996 survey results also contradict the conclusions EPA made at the bottom of P. 45 from the analysis of the data not given, that the NYS consumption ban would have no effect. The 1996 survey data reinforce comment No. 1 and are another example of the over-conservatism that EPA has used in this risk assessment. Also, EPA is requested to indicate whether the 1991 survey (Connally, et. al.) showed any difference in the 95th percentile number of meals eaten between areas with and areas without fish advisories. This information is significant to assessing the RME risk. HL-1.12

13. **Section 3.2.1.1, P. 40:** The discussion of the 1990 Mid-Hudson Survey (Jackson, 1990) says the survey included the percentage of anglers that keep and eat fish and are aware of fish advisories. The EPA discussion of the 1990 survey, however, focuses on matters irrelevant to the risk assessment such as what fish the anglers were after, sex and age of the anglers, differences between shore and boat anglers, and fishing tournaments. The EPA discussion ignores the factor significant to the risk assessment, i.e. the percentage of anglers that keep and eat fish and the awareness of fish advisories. EPA is requested to provide this information. HL-1.13

14. **Section 3.2.1.2, P. 41:** The stated objective of evaluating exposures to PCBs in fish in the absence of Hudson River-specific health advisories is overly conservative and should be abandoned in light of the 1996 NYSDOH survey. As a minimum, the recommendation of Comment 1 should be honored by EPA. An additional argument for this course is the Kimbrough, et al (1999) study of GE workers at the Ft Edward and Hudson Falls plants. Workers at these plants were likely anglers in the Upper Hudson yet the Kimbrough study showed no significant increase in causes over that of the general US population. HL-1.14

15. **Section 3.2.1.3, P.44:** The discussion at the end of this section of the 1996 and 1991-92 surveys is unconvincing in its disregard of these surveys showing significant impact of the fish advisories on the consumption of fish. Despite difficulties (and just what are these difficulties?) in extrapolating the 1996 and 1991-92 values to annual average ingestion rates, the fact remains that the 1996 and 1991-92 surveys provide direct data on the Upper Hudson River and are more recent than EPA's preferred 1991 Connally, et al survey. How many of the anglers in the 1991 survey were from the Upper Hudson River and how much of the year did the respondents say they fished? HL-1.15

16. **Section 3.2.1.4, P. 46:** The contention that children (and perhaps women also) eat as many fish meals as men is suspect, especially given the merchant food preferences of younger children. This could be especially true for the RME estimate. Consideration of children eating less fish should be factored in to the RME estimates. HL-1.16

17. **Section 3.2.4.1, P. 52:** The assumptions stated here that the fishing population is in steady state and the corollary that 1991 survey data represents 1999 and 70 years into the future is speculative and highly questionable. Preferences for spending leisure time do not remain consistent, especially in today's climate of rapidly changing technology. Witness the rise in time spent on the web and computer games which could especially impact the younger age groups. Another example is the leveling off in the number of downhill skiers indicating changing demographics for this sport. Is there any survey results from years before the 1991 Connally, et al survey that could shed light on this subject? The current EPA position represents an unsubstantiated assumption and another facet of the conservatism in EPA's risk assessment. HL-1.17

18. **Section 3.2.4.1, P. 53:** Where do Tables 3-6 and 3-7 fit in? I could find no reference to them in the text. Also, aren't Tables 3-6 and 3-7 constructed using procedures given in this section and not directly from the 1991 survey as implied in the footnote to these tables? HL-1.18

19. **Section 3.2.4.2, P. 54:** The uncertainty discussions on this page highlight a significant problem with this risk assessment. The survey population is too small to get reliable values of fish consumption and duration of exposure. This raises questions about the validity of the risks calculated in this report, especially when all the conservatisms in the calculations are considered. HL-1.19

20. **Section 3.2.4.3, P. 56:** What evidence is there that the populations in the Upper Hudson counties are in steady state? It seems doubtful they are. Couldn't census data be used to evaluate this assumption? It also seems unlikely that the assumption of the same number of individuals moving each year in a 5-year period is true. What are the effects on the risk assessment if these assumptions are incorrect? HL-1.20

21. **Section 3.2.4.3, P. 57:** Table 3-6 shows that of 226 anglers in the 1991 survey, less than 1 angler (or 0-1 angler) fished for 70 years. This seems like very meager data on which to base using 70 year fishing duration in the Monte Carlo analysis. Consideration should be given to using a lower upper bound in the Monte Carlo analysis. Perhaps 60 years should be used as discussed in the last paragraph of this section. HL-1.21

22. **Section 3.3.1, P. 59:** The meaning of "Minimum of Fishing Duration and Residence Duration" for the Base Case calculation is not clear. Please provide additional explanation of this parameter. HL-1.22

23. **Section 4.1, P. 62:** Can the four standard uncertainty factors be multiplied together giving a total factor of safety of 10,000 in some cases? If so, do any of the data used in this assessment have safety factors of 1000 or 10,000? Even the uncertainty factor of 100 for Aroclor 1016 and 300 for Aroclor 1254 are so large as to raise questions about whether any conclusions about risk can be drawn from the base data. HL-1.23

24. **Section 4.1, P. 63:** Given the apparent uncertainty in the RfD as evidenced by the large factors of safety cited on P. 62, it is recommended that the Monte Carlo analysis include uncertainty and variability in the toxicity values. HL-1.24

25. **Section 4.2, P. 64 and Section 5.2.2, P. 71:** In view of all the other conservatisms present in the risk analysis, it is an unreasonable additional conservatism to use the upper bound CSF of 20 in the Monte Carlo analysis. The CSF of 1.0 should be used as in the central point estimate. HL-1.25

26. **Section 5.3.1, P. 72:** The fact that the risk assessment does not quantitatively evaluate those born after 1999 or those who move to the area after 1999 raises questions as to the validity of the risk assessment, as these populations would have less exposure to PCBs. This makes it even more important to be very careful and not overly conservative in calculating the PCB exposure, the duration of exposure, and the total population at risk. HL-1.26

27. **Section 5.3.1, P. 74:** EPA's statement that the point estimates of angler exposure duration are likely to be underestimates is not substantiated. EPA should either acknowledge that the point estimate could also be overestimates or provide the basis for the statement on this page. HL-1.27

28. **Section 5.3.3, P. 79:** The statement that the 50% decrease in the risks using the Maine angler study does not change the results significantly is true in itself but is misleading in that it ignores the potential cumulative effects of this change plus others that remove unnecessary conservatism. The cumulative effects of such changes could be very significant. HL-1.28

29. **Section C.2.2, P. C-2 and Section C.3.1, P. C-4:** It is my understanding that the Agency for Toxic Substances and Disease Registry (ATSDR), part of the US Department of Health and Human Services, is charged by Congress with specifying toxicities for various substances including PCBs. EPA references this agency on P. 61 of the risk assessment where EPA refers to a 1997 review of the toxicity profiles by ATSDR. I have seen excerpts from an updated draft of ATSDR s toxicological profile of PCBs dated February 1999. This updated draft concludes that no studies have shown death in humans due to PCBs by any exposure route and that acute lethality data do not suggest PCBs would be acutely toxic in humans. Excerpts go on to cite ATSDR conclusions that the weight of evidence does not show PCB's cause cancer or have other toxic health effects. How is EPA going to factor this latest information from ATSDR into the risk assessment? HL-1.29

30. **Section C.2.2., P. C-2:** Kimbrough, et al (1999) state that the studies cited on this page by EPA in suppor of the carcinogenic potential of PCBs in humans are deficient in that the standardized mortality ratios (SMRs) reported in these studies were not correlated with factors which would suggest a PCB dose-response relationship, namely the SMRs were not correlated with higher and/or longer exposures to PCBs or longer latency periods. Why does EPA not include a discussion of these deficiencies and their significance in this section? (Note: some discussion of deficiencies in the Sinks Study are presented). These deficiencies are the same reasons EPA has cited for rejecting studies of TRVs. HL-1.30

31. **Section C.2.2., P. C-3:** EPA cites what it believes are several deficiencies of the Kimbrough study on this page. EPA statement - More than 75% of workers in the study never worked with PCBs. Comment – the Kimbrough study still included about 1750 people who did work with PCB's, almost the same as the 2100 people in the Bertazzi study used by EPA. HL-1.31

    - EPA statement: Less than 25% of workers who were exposed to PCBs at the General Electric facilities were, employed in their jobs for less than 1 year. (Note: I believe this statement quotes incorrectly EPAs position, but the point is that EPA criticizes the "short exposure"). Comments: The median exposure time for those heavily exposed in the Kimbrough study is 1.2 years for hourly male workers (1,268 workers) and 1.6 years for females hourly workers and even longer for the smaller number of heavily exposed salaried workers. Also, the Kimbrough study only included employees who worked at least 90 days at the GE facilities. In contrast, the Bertazzi study used by EPA included people who had only worked at least one week.

      EPA cites other deficiencies of the Kimbrough study such as the actual PCB exposure level is not confirmed, the age of the workers was young at the end of the study period, and vulnerable populations were not evaluated. However, EPA provides no evidence or discussion that these same deficiencies are not present in the studies being used by EPA. It does not seem that EPA has given a fair evaluation of the Kimbrough study in relation to the studies cited by EPA in defense of the potential for PCBs to cause cancer.

32. **Section C.2.3, P. 6-3:** Is the CSF "central estimate" an upper bound as implied by the opening sentence of this section? If no, the upper bound of the central estimate is "an upper bound of an upper bound" making the upper bound very conservative. HL-1.32

33. **Section C. 2.3, P. C-3:** Why aren't the Brunner and Norback and Weltman stidies included in the list of references? Some discussion of these studies should also be included. How do these studies relate to those cited on page C-2? HL-1.33

34. **Section C. 3.1, P. C-4:** Since the Patandin and Lanting studies relate both to exposure of PCBs and dioxins, it would appear that conclusions can not be drawn from these studies regarding the effects of PCBs. EPA should provide a discussion as to why these studies can be used to predict PCB effects. Also, why is the discussion of breast cancer here and not in cancer section (C. 2.2)? The lack of a cause and effect relationship for breast cancer suggests there may also be a lack of such a relationship for other cancers. HL-1.34

35. **Section C. 3.2.1, P. 6-6 and Section C. 3.2.2, P. C-6:** The uncertainty factor of 100 in C. 3.2.1 is greater than the straight multiplication of the factors (81) as is the uncertainty factor of 300 in C. 3.2.2 (270 by direct multiplication). There are further examples of extra conservatism in the risk analysis. HL-1.35

36. **Section C. 4.2, P. C-7:** EPA's acceptance of a risk of 10^{-4} for ingestion of PCBs in drinking water would seem to define this as the acceptable risk for this report. Does EPA agree? HL-1.36

37. **General:** Many instances have been mentioned throughout these comments where EPA has compounded conservatism on top of conservatism in the risk assessments. EPA should consider removing some of the conservatisms to achieve a more realistic estimate of the health risks, including the upper bound risks. HL-1.37

DONALD B. AULENBACH, PHD

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HP-1

To comply with the standard 911 numbering system, effective October 1, 1999 our house our house number will be:
28 Valencia Lane

17 August 1999

Alison A. Hess, C.P.G.
USEPA Region 2
290 Broadway - 19th Floor
New York, NY 10007-1866
Attn: Upper Hudson River HHRA Comments

Dear Alison Hess:

I have reviewed the PHASE 2 REPORT - REVIEW COPY, FURTHER SITE CHARACTERIZATION AND ANALYSIS, VOLUME 2F - HUMAN HEALTH RISK ASSESSMENT, HUDSON RIVER PCBs REASSESSMENT RI/FS, dated August 1999. Herein are my comments.

Basically I find the report inconclusive and misleading. There are so many "weasel" words (words that one can easily slip out of) that no reliable specific values can be assigned; nevertheless, the report assigns specific values with little indication of variation. These include words such as *may, can, could, might, probably, perhaps, estimate, etc.*, and are especially bad when followed by *nevertheless*. I refrain from citing the report section and page numbers for all these words, since they would fill this response. They appear on nearly every page, but are most predominant in sections 4 and 5 where I easily found 35 "weasel" words. **HP-1.1**

A grammatical comment, the author of the report should be aware that a singular subject (*a total, a combination, etc.*) takes a singular verb and is not changed when followed by a phrase starting with *of*. Thus: *A total of 10,000* still requires the verb *is*. It would be better to spell out the number, or use another subject noun such as *Altogether*. This is a common error of present day writing that should not be continued in this report. Again, this occurs throughout the report, and I shall not cite specific section and page numbers. **HP-1.2**

A glaring question is the relationship between all the fishing studies referred to in the report and the study section (Hudson Falls to the Troy Dam) of the Upper Hudson River. If all the fishing studies relate entirely to the study section, this should be clearly stated. If the fishing studies of the Upper Hudson River include sections of the river above Hudson Falls, this should be so stated, and evaluation should be made of what fraction of those as reported fishing in the entire upper section fish in the study section. I state this as I cannot conceive of 10,310 individuals fishing in the 65 kilometers (40 miles) of the study area, especially during the fishing ban [section 2.1.2, P. 7, and numerous other locations]. If this includes fishing in the Hudson River above Hudson Falls, the numbers become more believable. Page 13 states that "there is insufficient information to quantify fishing preference or frequency at specific locations". Nevertheless, the study assumes that all these individuals will fish in the study area beginning in 1999 in the absence of a fishing ban. **HP-1.3**

In the summary, section 2.3.1. P. 12. and in the corresponding detailed sections. the forecast is for 70 years for the Monte Carlo analysis. In a 70 year period. the risk of injury or dying from other sources is much greater than the risk from ingesting cancer from PCBs in fish. Nowhere in this report is there listed such a comparative risk. HP-1.4

The last sentence on page 12 is confusing. HP-1.5

All of the equations for intake of PCBs (such as section 2.4.1. P. 22) and stated on page 23 make no allowance for discharge of PCB from the body, such as body wastes or breakdown in the body. All exposures are assumed cumulative. Unless this has been proven to be fact, some allowance must be made for diminution after ingestion. This could make a significant difference in the results of this study. HP-1.6

Continuing on page 22. it is assumed that all sportsfish caught by the studied fishing population are caught in the Upper Hudson River. In view of the many lakes in the area within 34 miles [section 3.2.1.1. P. 38] of the Hudson River. including Saratoga Lake. one of the most productive lakes in the state. unless specific information is available. some consideration must be made for sportsfish caught elsewhere. In section 3.2.1. P. 37. "Upper Hudson River anglers are defined as all individuals who would consume self-caught fish from the Upper Hudson River at least once per year". No information is available as to how many times they consumed self-caught fish from other nearby bodies of water. The selection of the high amount of fish consumed from the study area represents a high bias in the results. HP-1.7

In selecting a loss of PCBs in cooking [section 2.4.2, P. 25] the high cooking loss of 74% was thrown out. but the low value of 0% was included "to include the *possibility* [emphasis mine] that pan drippings are consumed". Since most cooking losses were between 10 and 40 percent. the midpoint should be 25%. The assumptions chosen represent a high bias in the results. HP-1.8

In defining the *Dermal Absorption Fraction (DA)* in section 2.4.3 on P. 27, the word *rate* should be changed to *fraction*. Rate implies a time factor. HP-1.9

Section 3.1. P. 35 explains that the 2-D analysis was not performed due to insufficient information available. This brings into question the sufficiency of the information available to conduct the other analyses. HP-1.10

In section 3.2.1.1, P. 40, it indicates that in the Maine survey it was assumed that the fish caught were not shared by other household members. My wife would not hear of that, but again this assumption was chosen to bias the results of this study on the high side. HP-1.11

Section 3.2.1.2, P. 42 states that 42.7% of the responses indicated they ate none of their fish. while section 3.2.1.1, P. 40 states that this was 92%. Pick a number to back up your conclusions. HP-1.12

Although I cannot locate the exact number, apparently the Connelly, *et al.* (1992) report identified more than one respondent who claimed eating more than 1,000 meals of fish per year [section 3.2.1.2. P 42]. If they have been consuming fish from the study area for the 40 years that PCBs have been discharged to the river, if the statistics of this report are accurate we should dig up their bodies and see if they died of cancer. HP-1.13

Much space is devoted to an assessment of the age at which individuals started fishing [section 3.2.4.1, P. 51]. However, since this risk study is based on the number of years, starting in 1999, that an individual consumes fish from the Upper Hudson River, this becomes a moot point. **HP-1.14**

In the Toxicity Assessment [section 4, P. 61], it is notable that all the profiles are based on USEPA studies, while summarily throwing out the more comprehensive studies of Kimbrough *et al.* (1999) [page ES-4]. Can we show that the USEPA studies that are designed to support USEPA directives are any less biased than other reports? Again, I refer to the number of "weasel" words in this section. **HP-1.15**

A conclusion of the cancer risk [section 5.1.2, P. 69] is that there would be an *estimated* [emphasis mine] 3.2 additional cases of cancer in a population of 100,000. This means that for the 10,000 individuals who presumably fish in the study section of the Hudson River, there would be less than one case of cancer over a 70 year lifetime. Please note that this is *case*, not *death*. With medical advances, even considering no advances in treatment over the next 70 years, there would not be a statistically determined increase in cancer deaths due to consumption of Hudson River Fish. **HP-1.16**

The conclusion of this report is that there is no public health hazard from consuming fish from the study area of the Upper Hudson River either now or in the future. **HP-1.17**

Thank you for the opportunity to share my evaluation with you.

Sincerely,

Donald B. Aulenbach, PhD, P.E., DEE

HP-2

Dr. Brian Bush

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518-463-8250

August 25, 1999

Alison A. Hess C.P.G, (HHRA Comments)
USEPA Region 2
290 Broadway - 19th Floor
NY NY 10007-1866

Dear Alison,

My comments refer to neurological effect of PCB in mammals, which were not evaluated in the phase 2 report Volume 2F.

My colleagues and I have published serious effects on brain catacholamines in the rat[1] and monkey[2] caused by PCB. We have evaluated a large number of individual congeners with cells in culture and the most potent congener is clearly 2,2-dichlorobiphenyl[3]. Unfortunately your analytical method may not be measuring 2,2-dichlorobiphenyl correctly, since your spokesperson at the Albany August 4th meeting stated that Aroclor 1242 was the least chlorinated Aroclor mixture used in the analysis. HP-2.1

Another unrelated comment is that I have evidence that your estimated PCB concentration in air is an order of magnitude too low. Bopp and Tofflemire's work was probably and "3Cl+" measurement, so that the major components of upper Hudson River water: 2-chloro- and 22-dichlorobiphenyl were not measured. My data will be reported to the NY HP-2.2

Community Trust by Dr Barry Commoner, CBNS, Queen's College, early next month.

Finally I should like to congratulate all at Region 2 for exquisitely presented and well researched investigations. HP-2.3

Sincerely yours,

Brian Bush

Brian Bush Ph.D., F.R.S.C.

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Rensselear NY 12144-3456

1. Seegal, R.F., Brosch, K.O., and Bush, B. (1986). Regional alterations in seratonin metabolism induced by oral exposure of rats to polychlorinated biphenyls. Neurotoxicology 7(1):155-166.
2. Seegal, R.F., Bush, B., and Brosch, K.O. (1991) Comparison of effects of Aroclor 1016 and Aroclor 1260 on non-human primate catecholamine function. Toxicology 66, 145-163.
3. Shain, W., Bush, B., and Seegal, R. (1991) Neurotoxicity of polychlorinated biphenyls: Structure-activity relationship of individual congeners. Toxicol. Appl. Pharmacol. 111:33-42.

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HP-3

September 7, 1999

Sent via facsimile

Alison A. Hess, C.P.G
USEPA Region 2
290 Broadway – 19th Floor
New York, NY 10007-1866

RE: Upper Hudson River HHRA Comments

Dear Alison:

Due to the limited time frame with which we had to review the Human Health Risk Assessment (HHRA) Scenic Hudson's comments on this document are brief. The technical advisor, retained under Scenic Hudson's Technical Assistance Grant (TAG), received this document two weeks after it was released which was not enough time for a thorough review. In addition, our technical advisor did not receive a copy of the **Baseline Modeling Report** that was released this past May, thereby furthering limiting his ability to review the Human Health Risk Assessment. Once Dr. Nisbet has received the Baseline Modeling Report we may submit additional comments based on that review.

Our comments are as follows:

Risk to Non-Anglers – As we have previously pointed out, we are concerned about the usefulness of the HHRA in addressing the risk to those individuals that consume Hudson River fish that were shared by the angler. Clearwater's angler survey indicated that 58% of anglers gave fish to their families for consumption. Those consuming fish, not necessarily the anglers themselves, may be members of at risk populations, such as women and children. Risks to the non-angler consuming Hudson fish must be thoroughly addressed. HP-3.1

Potentially Exposed Populations – While the USEAP has indicated that a HHRA will be conducted for the mid-Hudson Region, it is unclear why a HHRA will not be conducted for the lower Hudson where a sizeable segment of the potentially affected population exists. Although the primary PCB contamination is in the Upper Hudson, the Superfund site incorporates 200 miles of the Hudson River and the USEPA has indicated HP-3.2

in the **Data Evaluation and Interpretation Report** that fifty percent of the PCBs in the New York Harbor are GE PCBs.

Exposure Scenarios - While fish ingestion is likely to dominate risks, as the HHRA indicates, it is important to acknowledge that exposed individuals are likely to be exposed via multiple pathways. Total risks and hazards should be added together across pathways. HP-3.3

We question EPA's conclusion that exposure to PCBs through inhalation of PCBs in the air does not present a risk that is of concern. Recent studies from the Chicago area document an "urban plume" of PCB contamination. Based on these findings and other evidence that PCBs are transported by air depositions, EPA should reevaluate this route of exposure. In addition, EPA should evaluate the feasibility of estimating the food chain pathway risks as a route of exposure via air deposition, such as through milk and meat ingestion. HP-3.4

PCB Toxicological Profile - Scenic Hudson is concerned that the PCB Toxicological Profile (Appendix C) is out of date and probably understates both the hazards posed by PCBs to human health and the degree of certainty that these hazards exist and are applicable to low-level exposures. We appreciate that EPA's procedures for updating RfDs, cancer slope factors, and other peer-reviewed information incorporated into IRIS is deliberate, cumbersome, and time-consuming. Nevertheless, Appendix C already contains reference to several 1999 studies of PCBs, and we believe that it should incorporate reference to other important recent publications. HP-3.5

Carcinogenicity. Appendix C devotes nearly a page to the study by Kimbrough et al. (1999). If this much attention is given to a study which is identified as of poor quality and little value for risk assessment, at least equal attention should be given to recent studies in the general population, including Rothman et al. (Lancet 350: 240-244, 1997) and Moysich et al. (Cancer Epidemiol. Biomarkers Prev. 7: 181-188, 1998). These studies used rigorous case-control designs, with prospective identification of individual exposures through sampling of blood; both showed significant associations between site-specific cancers and exposure to PCBs within the ranges of exposure found in the general population. HP-3.6

Developmental Toxicity. One paragraph on page C-4 refers to reports by Patandin (1999) and Lanting (1999). Both these reports are unpublished theses, not yet subjected to peer review. Citing them as though they were the only basis for inference of health effects from consumption of PCB-contaminated fish, as this paragraph appears to do, does not carry much weight. It should be made clear that these studies are not isolated findings, but extend and confirm a considerable body of other information (peer-reviewed and published) indicating that PCBs do cause these effects at levels of exposure similar to those in the Hudson River. HP-3.7

Dioxin-like Toxicity. Although Section 5.1.3 of the text (pp. 69-70) assesses cancer risks for PCBs based on the Toxic Equivalency Quotients (TEQs) for "dioxin-like" toxic HP-

effects. We agree that this approach to risk assessment for PCBs is appropriate and reasonable, but the basis for this procedure is not given in Appendix C. We recommend that Appendix C should be amended to include a section summarizing the basis for the approach, the procedure for calculating TEQs for PCBs, and the basis for EPA's toxicity criteria for 2,3,7,8-TCDD (CSF and RfD). We further recommend that the text should include a section assessing *non-cancer* risks of PCBs based on dioxin-like toxicity. We believe that EPAs toxicity criteria for 2,3,7,8-TCDD also need updating. We specifically draw EPA's attention to the fact that IARC has recently re-classified 2,3,7,8-TCDD as a multi-site carcinogen in humans (McGregor et al., Environ. Health Perspec. 106, Suppl. 2: 755-760). We believe that this requires reconsideration of EPA's classification of PCBs as B2 carcinogens. Risk calculations for PCBs based on their "dioxin-like" toxicity should reflect the fact that it is now appropriate to classify dioxins as A carcinogens.

Endocrine Disrupters - With regards to PCBs acting as "endocrine disrupters", it is indicated in Appendix C (C-5) that EPA believes that "there is little knowledge of or agreement on the extent of the problem," and "further research and testing are needed." HP-3.9

However the recent **National Research Council Report on Hormonally Active Agents in the Environment** made some definitive statements regarding the affect of these chemicals on human health.

Statements from the report include:

"Adverse reproductive and developmental effects have been observed in human populations, wildlife, and laboratory animals as a consequence of exposure to HAAs [hormonally active agents]" p.3.

"Human dietary intake of synthetic HAAs remains substantial, even intake of HAAs that have not been used commercially for years...Although concentration were found to be greatest in older individuals, even children were not immune to exposure." p.76.

While the science may not be conclusive regarding the connection between HHAs or endocrine disrupters and health effects such as breast cancer, it is clear that these chemicals cause problems with reproduction and development, the nervous system and the immune system. We advocate that the USEPA take a precautionary approach to prevent further harm from Hudson River PCBs although some scientific uncertainty may remain regarding the complete range of affects of PCBs on the endocrine system.

Thank you for the opportunity to provide these comments.

Respectfully submitted by:

Rich Schiafo
Environmental Associate

1015 Belleville Turnpike
Kearny, New Jersey 07032

CLH
CHEMICAL LAND HOLDINGS, INC.

HAND DELIVERED

September 7, 1999

HP-4

Ms. Alison A. Hess, C.P.G.
USEPA Region 2
290 Broadway -19th Floor
New York, New York 10007-1866
Attn: Upper Hudson River ERA/HHRA Comments

Dear Ms. Hess:

Chemical Land Holdings, Inc. (CLH)is pleased to submit the following technical memorandum entitled "Comments on the U.S. Environmental Protection Agency's Hudson River Ecological and Human Health Risk Assessments." The comments provided in this memorandum represent CLH's position on the technical approaches that were used by U.S. Environmental Protection Agency (USEPA) to assess risks to humans and ecological receptors from polychlorinated biphenyls (PCBs) in the Hudson River.

We view the Hudson River risk assessments as an example of how USEPA is going to evaluate ecological and human health risks due to organochlorines and other persistent chemicals in large river systems. We submit these comments to help ensure that USEPA assesses these risks in a technically sound manner, in keeping with applicable regulations and guidance, and in a fashion that is useful to facilitate effective risk management and decision making.

In USEPA's August 4, 1999 memorandum regarding the release of the Hudson River risk assessments, USEPA stated that "comments...should include the report section and page number for each comment." To the extent possible we have tried to provide specific section and page numbers for each of our comments. However, it was not CLH's desire to provide comments on the site-specific details of the Hudson River risk assessments. Rather, the comments contained in this memorandum are focused on the "big picture" technical approaches used by USEPA to assess chemical risks in a large riverine system, and that will likely become the basis for other riverine risk assessments conducted by USEPA in the future. For this reason, the comments are not all specifically targeted towards a page and/or paragraph of the risk assessments. Rather, several comments deal with a more general technical approach that is contained within an entire section of the assessment. We have tried to be as specific as possible in referencing either the page or section number that a comments is targeted towards.

We hope that USEPA will strongly consider these comments and re-think several of the technical approaches used to conduct the Hudson River risk assessments.

Sincerely,

Alex Pittignano

Alex Pittignano
Senior Project Engineer

Technical Memorandum

Comments on the U.S. Environmental Protection Agency's Hudson River Ecological and Human Health Risk Assessments

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Chemical Land Holdings, Inc., (CLH) is pleased to submit these comments to the August 1999 *Hudson River PCBs Reassessment RI/FS Baseline Ecological Risk Assessment* and *Human Health Risk Assessment* report . We view this assessment as an example of how the U.S. Environmental Protection Agency (USEPA) is going to evaluate ecological and human health risks due to organochlorines and other persistent chemicals in large river systems. We submit these comments to help ensure that USEPA assesses these risks in a technically sound manner, in keeping with applicable regulations and guidance, and in a fashion useful to facilitate effective risk management decision making.

Baseline Ecological Risk Assessment (ERA) (Volume 2E)

Exposure Assessment (Section 3 of Volume 2E)

Comment 1

The exposure analysis in the ERA is conducted by simply averaging data from water, sediment, benthic invertebrate, and forage fish samples taken in various locations representing relatively long reaches of the river (Volume 2E Sections 2.3.2 and 3.2). The ERA states (Volume 2E, Section 2.3.2, page 15) that the river segments represented in this scheme are "large enough to encompass the foraging areas of local populations of fish and wildlife, and provide information at an appropriate scale...[to] capture changes in spatial concentrations of PCBs."

This approach to ecological exposure analysis is inadequate for assessing chemical risks in large river systems. Risk Assessment Forum Guidelines for Ecological Risk Assessment ("the Guidelines," USEPA 1998, Section 4.2.1) clearly state that "Exposure

is contact or co-occurrence between a stressor [chemical] and a receptor. The objective is to describe exposure in terms of intensity, space, and time units that can be combined with the effects assessment...A complete picture of how, when, and where exposure occurs or has occurred is developed by evaluating sources and releases, the distribution of the stressor in the environment, and the extent and pattern of contact or co-occurrence." River systems are highly heterogeneous, and heterogeneity is not captured by simply treating vertical river reaches as if they were uniform exposure habitat (which is what the ERA does). There is substantial and important horizontal structure in river systems (NRC 1992). For example, deep mid-channel environments have quite different levels and kinds of biological activity from shallow, near-shore sediments. Riffles differ from pools. Shoreline characteristics, aquatic vegetation types and substrate features typically determine the relative value of near-shore habitats for a variety of aquatic organisms.

The distribution of receptors in a river is largely a function of these habitat differences. For example, many fish-eating birds feed on small forage fish in very shallow waters. These fish are exposed to sediments and food only in limited areas of the river. Consequently, the bird exposure derives from those sediments, and not from others. Thus, the approach taken in the ERA (simply lumping habitats within river reaches as if they were equivalent from an exposure standpoint) is inadequate and does not reflect the guidance.

In general, key parameters are habitat type (e.g., foraging, breeding, loafing, and migrating), distribution, and quality. If there is no habitat for particular receptors in a particular watershed or river system, or river reach, there can be no exposure for those receptors. Because organochlorine compounds do not impact habitat *per se*, habitat conditions are the exposure baseline. If some habitat areas are present, but of relatively poor quality for particular receptors, exposure will be less in those poor quality areas. The more urbanized and degraded a watershed or river reach is, the less important it is as an exposure area. In the ERA, exposure area was by river reach with no consideration of habitat. Quantitative consideration of habitat is important for the technical and regulatory

credibility of the assessment, and should be incorporated to reflect the ecological reality of exposure in this large river system.

Therefore, we recommend that USEPA conduct a habitat assessment of the River, and then conduct a realistic evaluation of exposure for each receptor of interest based on their relative use of specific areas of the River. This type of analysis can be done using tools such as geographic information system (GIS) to map and quantify habitat types, and then evaluate the likely and relative use of each habitat or habitat type by the receptors of interest. ...is type of analysis is key to conducting a realistic assessment of exposure in aquatic systems.

Comment 2

The exposure analysis in the ERA implicitly assumes that all polychlorinated biphenyl (PCB) molecules within a river reach have an equal likelihood of contacting ecological receptors. This is simply not true for PCBs or any other chemical contained in river sediments.

As described in Comment 1, exposure is properly quantified by overlaying the spatial and temporal distributions of chemicals and different types of habitats for representative receptors. In other words, not all organochlorine molecules in a river system are equal—some are more important in the exposure pool than others.

A substantial portion of the PCB in the sediments are bound and have no or limited bioavailability. Others are buried beneath the biologically active surface zone of the sediments, or are in habitats or microhabitats (Resh et al. 1996) that limit or eliminate bioaccessibility. In a particular river system, a relatively large proportion of organochlorine molecules may be in sediments that are not bioavailable or bioaccessible, and thus cannot drive ecological risks. USEPA should evaluate and document the

particular areas in the River that contain PCBs at levels that may pose ecological risk, based on a realistic exposure assessment as described above.

Comment 3

The exposure analysis in the ERA fails to account adequately for life history characteristics of particular receptors. Among the receptors identified for ecological risk assessment, there is a wide range of life history parameters that affect exposure in important ways, and should, therefore, be accounted for in the analysis. Some species (including anadromous fish like striped bass and shortnose sturgeon and migratory birds including tree swallow, mallard, belted kingfisher, great blue heron, and bald eagle) may acquire substantial doses and/or body burdens of PCBs in areas remote from the Hudson. For example, the birds migrate to the southern United States and/or to Central and South America, where they feed actively in preparation for the return migration in the spring (Welty 1982). Striped bass and shortnose sturgeon leave the Hudson and migrate along the coast to deeper and/or more southern waters.

In both cases, there is substantial likelihood that these species acquire PCBs from sources unrelated to the Hudson. Source is an important exposure parameter (USEPA 1998, Section 4.2.1.1). Yet the ERA treats all PCBs as if the source of exposure was the Hudson River system. Relatively simple tools are available to evaluate the ecology of fish and bird movement, and many readily available sources (including published information on bird and fish migration routes and wintering ground populations) track the time spent in summer vs. winter habitats. In addition, if resident subpopulations of some species (such as the striped bass) are present, ecological risks should be quantified separately for this subpopulation because the exposure sources will differ. The potential for exposure in other areas (e.g., waterfowl and tree swallows migrate to Central and South America) should be addressed and, to the extent possible, quantified in the Hudson River risk assessment.

Effects Assessment (Section 4 of Volume 2E)

Comment 4

The ERA relies on a deterministic method for identifying toxic effects thresholds for the ecological receptors that is, a yes/no description of the likelihood of response. This is a common and widely accepted approach to conducting ecological risk assessments. However, for assessments as complex as those involving organochlorine compounds in large river systems, probabilistic analysis of toxicity may be as important for credible risk assessment as is probabilistic analysis (on a habitat basis) of exposure. This is particularly critical for risk assessments involving organochlorine compounds for which susceptibility of organisms is known to differ enormously (by several orders of magnitude across major taxa, by more than an order of magnitude within a single class such as fishes).

By not employing a probabilistic analysis of toxicity, the risk assessment necessitates the application of arbitrary and unjustified "uncertainty factors" (see separate comments below) that hinder utility of the entire risk assessment. The Guidelines (USEPA 1998, Section 4.3.1.1) state that "Point estimates may be adequate for simple assessments or comparative studies of risk...," neither of which is the case for the Hudson River ecological risk assessment. Furthermore, when point estimates are used for ecological risk assessment, they should be derived based on the slope of the dose-response curve (Chapman et al. 1998), and the ERA fails to provide any information whatsoever on quantitative aspects of the dose-response relationship for PCBs. The ERA should consider probabilistic alternatives to the deterministic toxicity thresholds.

Risk Characterization (Section 5 of Volume 2E)

Comment 5

The ERA identifies a number of site-specific field investigations of population-level parameters for some receptors, but then dismisses these studies or gives them little or no weight in the "weight of evidence" analysis. This is a serious shortcoming.

Site-specific population-level data (such as field studies of reproductive impairment and population parameters) are the most relevant and useful data for risk assessment. The Guidelines (USEPA 1998, Section 4.3.1.3.2) clearly state: "Risks to organisms in field situations are best estimated from studies at the site of interest. However, such data are not always available." For the Hudson River, such data are available, and should therefore be used and given appropriate weight in the risk assessment. It is not appropriate for the ERA to discard such data, particularly when the results (such as the findings of tree swallow field studies) are consistent and credible.

Comment 6

The ERA includes screening thresholds for water and sediment quality explicitly as a component of the definitive risk characterization. This is inappropriate from both a scientific and regulatory viewpoint.

Screening thresholds are applied only to guide quantitative risk characterization. Such thresholds are "...based on generic assessment endpoints (e.g., protection of aquatic communities from changes in structure or function) and are assumed to be widely applicable to sites around the United States" (USEPA 1997). Such generic thresholds include water quality criteria and sediment effects thresholds, both of which are designed to identify chemical concentrations below which adverse effects are unlikely. These thresholds are not intended to and cannot be used to quantify risk in an remedial investigation and feasibility study (RI/FS) context for supporting risk management

decision making. USEPA (1997) clearly states that "...requiring cleanup based solely on [the information developed during risk screening assessment] would not be technically defensible." The ERA should be modified to eliminate screening thresholds from the definitive risk characterization.

Comment 7

The ERA consistently misapplies toxicological effect thresholds. In calculating hazard quotients, it is appropriate to use highest no-observed-adverse-effect-level (NOAEL) when a range of choices is available. USEPA (1997) states: "For those contaminants with documented adverse effects, one should also identify the highest exposure level that is a NOAEL." Yet the ERA, without explanation, uses the lowest NOAEL. This fundamental toxicological error should be corrected in a revised version of the ERA.

Uncertainty Analysis (Section 6 of Volume 2E)

Comment 8

In the uncertainty analysis of the risk assessment USEPA uses the "uncertainty factor approach" to estimate safe concentrations of PCBs. Unfortunately, there is no foundation in the technical literature for applying "uncertainty factors" of 10 to toxicity thresholds. In fact, comparative toxicological studies have clearly established that the "analogy with human health risk assessment" on this is outdated and indefensible (Chapman et al. 1998). Indeed, this was reflected in USEPA's decision to not apply uncertainty factors when applying toxicity data developed for gallinaceous birds to fish eating birds in deriving the Great Lakes water quality criteria (USEPA 1995).

We suggest that it is important for USEPA to revise this document to properly address this issue. When inappropriate factors-of-ten uncertainty factors are applied, it is difficult or impossible to tell whether risk management decisions are being made to reduce real potential risks or analytical uncertainty. Unless uncertainty bounds can be quantified so

that risk management decisions can be understood in the context of risk and uncertainty (which is impossible with the "factors of 10" approach employed in the Hudson risk assessment), effective risk management decisions cannot be made. The ERA should be revised to provide uncertainty bounds or technically defensible "uncertainty factors," and not rely on simplistic, outdated "factors of 10".

Comment 9

For estimating effects levels on a toxicity equivalence (TEQ) basis to fish, the ERA repeatedly applies Walker et al. (1994) studies on Lake Trout, generally including "factors of 10" uncertainty divisors. This approach is simplistic, not credible, and scientifically indefensible. Salmonids like Lake Trout are highly sensitive to organochlorine compounds, and the application of salmonid studies, particularly with an uncertainty factor of 10 to non-salmonid fishes is inappropriate. For example, in determining effluent quality under the Clean Water Act, USEPA guidance provides a "resident species recalculation" procedure for water bodies lacking certain receptors (such as sensitive salmonids) on which generic standards may be based. The intent of this procedure is to assure that risk management decisions are not made to inappropriately stringent standards. The same procedure should be followed in risk assessments.

For watersheds, water bodies, or river reaches where only warm water fish communities exist, salmonids toxicity thresholds should not be applied. The ERA should be modified to identify areas of the Hudson supporting only warm-water fish communities, and apply a separate and appropriate toxicity threshold for these areas. A salmonid-based threshold should be applied only to areas supporting a cold-water fish community, and then without "factors of 10" uncertainty divisors.

Conclusions

Comment

The Hudson risk assessment cannot be used to support effective risk management decision making for the Hudson River. This is not in keeping with applicable USEPA requirements under Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA); the National Contingency Plan (NCP); and implementing guidance such as that for conducting RI/FSs. It is clear that risk assessments are one of the most critical decision tools to be applied to risk management at Superfund sites (e.g., USEPA 1988, NCP at 300.430(d) and (e)). For the Hudson, the result of not using appropriate exposure and toxicological analyses to develop an accurate characterization of risk, renders this document nothing more than a broad-brush and generic risk assessment.

Sediment parcels that might be associated with higher levels of exposure or toxicity cannot be identified or prioritized for risk management. Given the gross importance of "uncertainty factors of 10" in the technical conclusions, risk managers cannot even know if they would be managing real risks or simply analytical uncertainty if actions were to be taken. Given that the job of risk assessment is to support sound risk management decision making, a risk assessment that concludes, on a generic basis, that risks are "everywhere and all the time" is useless and unacceptable. The ERA should be revised to reflect the realities of exposure and toxicology in such a way that clear, credible, and defensible risk management decisions can be made. Otherwise, the entire exercise is a waste of time and effort.

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Human Health Risk Assessment (HHRA) (Volume 2F)

Comment 1

The USEPA's approach to assessing health risk in the Upper Hudson HHRA is atypical in that only one chemical is considered: PCBs. The focus on a single chemical is not consistent with the Superfund risk assessment process, the primary intent of which is to provide the regulatory decision-makers with information necessary to make consistent decisions regarding protection of public health. The USEPA virtually always requires the preparation of a detailed "screening analysis" that evaluates all detected chemicals whenever a private sector potentially responsible party (PRP) submits a Superfund risk assessment. It is unclear why the USEPA is not held to the same standard and no explanation is given in the HHRA as to why the USEPA has bypassed the standard risk assessment approach in their preparation of this analysis. At best, the assessment gives the reader a very limited understanding of the potential health risks associated with the Upper Hudson. **HP-4.1**

A more accurate analysis of the risks associated with the Upper Hudson River would consider all chemical and nonchemical constituents, regardless of the source. Given the industrialized nature of the Upper Hudson, and the possible sources upstream of the assessment area, it is almost a certainty that other constituents are present in sediments, fish tissues, and other media at concentrations that exceed background. These could include chemical (e.g., metals) and nonchemical constituents (e.g., bacteria, pathogens, pH, dissolved oxygen). Indeed, it is entirely possible that constituents other than PCBs could contribute significantly to total risk, and it is even plausible that some constituents could pose a greater theoretical risk than PCBs. In summary, the narrow focus on one chemical may result in uninformed and incomplete risk management decisions.

We suggest that if the USEPA has conducted an analysis that supports a conclusion that PCBs are the only potential health issue in the Upper Hudson, they should discuss this

analysis in an introductory section of the HHRA. Otherwise, we suggest that the USEPA revise their assessment to consider ALL potential threats to human and ecological health.

Comment 2

Some key assumptions regarding source terms do not appear to be justified. Specifically, the USEPA has assumed that that there are no other historical or ongoing sources of PCBs to the Upper Hudson, and it is implied that they have, therefore, adequately characterized the "source term" necessary for making accurate prospective estimates of PCB concentrations. However, the USEPA offers no discussion of the evidence supporting this assumption. Given the fact that the alleged discharges occurred over 20 years ago, there is certainly sufficient reason to suggest that a significant portion of the PCBs in Upper Hudson surface sediments may be from sources other than those mentioned in the HHRA. For example, PCBs could have been (and perhaps still may be) introduced to the Upper Hudson via surface water runoff, direct discharge from regulated and unregulated sources, atmospheric deposition, spills and leaks from watercraft, etc. HP-4.2

We suggest that, if the EPA has conducted an analysis (e.g., a fingerprinting analysis) that demonstrates that 100 percent of the PCBs in the Upper Hudson are from the sources they identify, then that analysis should be presented or cited in the HHRA. If no such analysis exists, the HHRA should clearly state this fact and acknowledge that there could be numerous other PCB sources.

Comment 3

Unequal attention is devoted to the results of the Monte Carlo vs. deterministic analyses. We support the USEPA's use of probabilistic analysis in the HHRA. However, it is clear that the deterministic results are given far greater emphasis in the conclusions sections. HP-4.3

For example, only the deterministic results are discussed in the "Major Findings of the HHRA" in the Executive Summary.

We suggest that the probabilistic results at least be given equal, if not more, emphasis in these important sections.

The fact that the deterministic reasonable maximum exposure (RME) risk for fish ingestion (1×10^{-3}) is virtually equivalent to the "base estimate" of the probabilistic 95th percentile (9×10^{-4}) is counter-intuitive; there is typically a 10–100 fold difference between these values, and such a margin would certainly be expected in an assessment such as the HHRA where upper-bound point estimates were used for every exposure factor in calculating the deterministic RME (as described in detail below). These findings suggest that the data distributions used in the probabilistic analysis may be overly conservative.

We suggest that the USEPA examine the nature of the distributions used in the HHRA to ensure they are not highly skewed to conservative values at the upper bounds.

Comment 4

There is an inadequate discussion of the uncertainty in the 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) slope factor. The HHRA employs the use of a TCDD slope factor of 156,000 $(mg/kg\text{-}day)^{-1}$ to assess the health risks associated with the "dioxin-like" coplanar PCB congeners. This slope factor is based on an extrapolation from a two-year chronic toxicity and oncogenicity study of Sprague-Dawley rats conducted by Kociba et al. (1978). The USEPA (1985) used the linearized multistage non-threshold dose-response model and an animal-to-human scaling factor based on surface area to derive the slope factor (USEPA 1994). HP-4.4

The HHRA fails to note that this slope factor is a highly controversial value that has been seriously questioned within the regulatory and scientific communities. Specifically, the methods used to derive the value have been continuously and openly challenged since USEPA first proposed the slope factor in 1994. As a result, the slope factor has failed to garner the agency support necessary for verification and inclusion into USEPA's Integrated Risk Information System (IRIS), and there is no indication that the value will be included in IRIS anytime soon. At best, USEPA's interpretation of the Kociba et al. (1978) data should be considered as a proposed value that may change in the future.

Furthermore, there are several published re-interpretations of the Kociba et al. (1978) animal data that offer far more refined estimates of the carcinogenic potency of TCDD. For example, an independent reevaluation was conducted by the Pathology Working Group (PWG) and the results published in 1991 (Keenan et al. 1991) that examined the tumor classification system from Kociba et al. (1978). The PWG used the National Toxicology Program tumor classification system to revise the results from the Kociba et al. study and update the cancer slope factor. This analysis corrected the tumor misclassification error in USEPA's original interpretation. Based on the results of the reanalysis, Keenan et al. (1991) reported a revised cancer slope factor of 9,700 $(mg/kg\text{-}day)^{-1}$.

As described in the Federal Register notice of April 12, 1994, the U.S. Food and Drug Administration (FDA) also relied upon the PWG reevaluation to calculate two unit risk values: one based on body weight scaling and the other based on surface area scaling to the ¾ power. Using the results of the PWG reevaluation and body weight scaling, FDA calculated a unit risk value (9×10^{-6}) that correlates to a dose of 1 pg/kg-day. This unit risk value is equivalent to a cancer slope factor of 9,000 $(mg/kg\text{-}day)^{-1}$. Also using the PWG reevaluation, but surface area scaling to the ¾ power, FDA calculated a unit risk value (3×10^{-7}) that correlates to a dose of 1 pg/kg-day, which is equivalent to a cancer slope factor of 30,000 $(mg/kg\text{-}day)^{-1}$.

Several regulatory agencies have chosen to use these refined slope factor estimates rather than the value suggested by USEPA. For example, the State of Georgia has employed the slope factor of 9,700 (mg/kg-day) calculated by Keenan et al. (1991) to establish surface water quality criteria. In addition, the FDA employed a slope factor of 9,000 $(mg/kg\text{-}day)^{-1}$ in a risk assessment of paper products, and concluded that 2 parts per trillion (ppt) of TCDD in paper correlated to a increased cancer risk estimate of 3×10^{-7}. FDA indicates on page 17,387 of the April 12, 1994, Federal Register notice that they "will use the 3×10^{-7} risk as the best estimate of what the upper-bound lifetime risk from TCDD toxic equivalents would be when all bleached food-contact paper products meet the paper industry's voluntary specification of 2 ppt."

The current version of the HHRA makes no mention of the shortcomings associated with USEPA's TCDD slope factor analysis nor does it cite the refined values that have been published and subsequently used by other agencies to protect public health. In fact, the *Toxicity Assessment* Section of the HHRA, which purports to describe the basis (and IRIS verification) of all toxicity criteria used in the analysis, completely omits any discussion of these issues as they relate to the TCDD slope factor.

Given the fact that the results of the HHRA are "driven" by the TCDD slope factor, and the fact that this single value arguably contains more uncertainty than the aggregate uncertainty in the entire exposure assessment, we suggest that a detailed discussion of the shortcomings inherent in this value, and the alternative values that have been published, is appropriate. This discussion should appear in the *Toxicity Assessment* and the *Uncertainty* sections. We would also suggest that the *Uncertainty* Section describe the range of risks that would be associated with the use of these refined values.

Comment 5

There is inadequate justification for rejection of site-specific angler survey data. Perhaps the most significant refinement of the risk assessment process over the last ten years has HP-4.5

been the increased reliance on the use of site-specific data wherever possible. Current USEPA risk assessment guidance emphasizes a strong preference for using site-specific fish consumption rates and other exposure parameters in risk assessments (USEPA 1989, 1997a,b). Site-specific data are particularly important when estimating a fish consumption rate due to the fact that recreational angling and fish consumption habits are extremely dependent upon location.[1] Failure to obtain site-specific data on fish consumption rates will inevitably lead to a high degree of uncertainty in the health risk estimates.

It is therefore surprising to note that the USEPA has disregarded their own guidance on this issue. Specifically, the HHRA states that the Hudson-specific surveys of fish consumption (e.g., NYSDOH 1999; Barclay 1993) cannot be used because fish consumption advisories were in place on the Upper Hudson River at the time of these surveys (p. 41). As a basis for excluding these data, the USEPA presumes that the advisories had a significant impact on the fish consumption rates of the surveyed anglers. However, no evidence is offered in support of this assertion. In fact, the existing evidence suggests otherwise. Statewide surveys have found that many recreational anglers in New York state are unaware of consumption advisories; and further, that the anglers who are aware of the advisories often perceive the risks to be overstated (NYSDEC 1990; Connelly et al. 1992; Vena 1992; Vena and Jadd 1997). For example, the data from the 1991 New York Angler Survey (Connelly et al. 1992) indicated that "there was no significant difference in the mean number of freshwater fish meals eaten when comparing New York waterbodies with full, partial, or no advisories, despite the expectation that the fishing advisories would likely suppress fish ingestion rates to some degree." Other researchers have noted that many New York anglers ignore consumption

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[1]According to USEPA, the Combined 1989, 1990, and 1991 Continuing Survey of Food Intake by Individuals conducted by USDA reveals the following freshwater and estuarine fish consumption rates: mean, 5.6 g/day; median, 0 g/day; 90th percentile, 17.80 g/day; 95th percentile, 39.04 g/day; 99th percentile, 86.30 g/day. 63 Fed. Reg. at 439803.

advisories and have expressed the opinion that self-caught fish are safer than that purchased in the store (Pflugh et al. 1999). In short, the USEPA has ignored the significant evidence that the advisories on the Upper Hudson most likely have had no effect on fish consumption rates in the area.

Furthermore, even if it was shown that the advisories did have some influence on consumption rates, the site-specific data would arguably still be preferable to literature-based estimates that may bear little or no semblance to actual consumption rates. In short, it is inappropriate to reject the site-specific fish consumption data out of hand and substitute literature estimates based on an assumption of bias that is in fact inconsistent with the available evidence.

We suggest that it might be far more appropriate to use the site-specific rates and discuss the weight-of-evidence regarding their potential bias. If the weight-of-evidence suggests they are biased low, some estimate as to the degree of bias (and the possible impact on the consumption rates) should be discussed in the *Uncertainty* Section. Similarly, if the weight-of-evidence is inconclusive or suggests that the advisories had no influence on consumption rates, then this should be acknowledged in the *Uncertainty* Section.

Comment 6

The HHRA uses an excessive estimate of fish consumption. As noted above, we suggest that USEPA should rely on the site-specific fish consumption data collected in the Upper Hudson, rather than literature-based estimates. In addition, we believe there are several reasons to suggest that the literature-based estimates developed by USEPA (from Connelly et al. 1992) are not representative of the Upper Hudson and are likely to far overstate actual consumption rates. Connelly et al. (1992) conducted a statewide mail survey of anglers who fished in New York waterbodies. However, *it is unknown whether any of the respondents fished in the Hudson River.* Specifically, the survey only asked respondents to identify the county in which they fished. It is, in fact, possible that none

HP-4.6

of the respondents fished in the Upper Hudson River, which obviously raises questions regarding the representativeness of these data. In addition, the study was based on a one-year recall, and it has been well-established that one-year recall data tend to overestimate the number of fishing trips. For example, Chase and Harada (1984) found that respondents to self-administered surveys tended to over-report their actual participation in recreational activities or activities perceived as pleasurable. Westat, Inc. (1989) reported that a one-year recall period produced "substantial overestimates" of fishing statistics. In summary, it is likely that the data reported by Connelly et al. (1992) overestimated actual number of fishing trips.

Also, Connelly et al. (1992) collected information only on the *number* of fish caught and consumed (per year) by each survey participant. No information on sample size was collected. As a result, the authors of the HHRA were forced to make assumptions to fill this significant data gap. The assumption used in the HHRA, that each fish meal was a half-pound in size, significantly over-estimates the typical amount of sport-fish eaten at a single meal. For example, the USEPA recommends an average meal size estimate of 129 g/meal, which is half of that used in the HHRA (one-half pound is equivalent to 227 g). As discussed elsewhere in these comments, these overly conservative assumptions resulted in upper-bound daily fish consumption rates that are well beyond those that have been actually measured in site-specific angler surveys

In summary, we believe that use of Connolly et al. (1992) may be inappropriate due to the uncertain nature of the survey (one-year recall) and the fact that the most critical data endpoint (size of consumed meals) were not even collected.

The *Uncertainty Analysis* Section of the HHRA should be very explicit regarding the above shortcomings associated with the Connelly et al. (1992) data, and should indicate that 1) it is unknown whether the dataset includes anglers from the Upper Hudson, 2) there is no data to support USEPA's assumption that advisories influence fish ingestion rates in the Upper Hudson, 3) published data regarding fishing advisories indicates that they do not significantly influence fish ingestion rates, 4) the Connelly et al. (1992)

dataset contains no information on sample size, and 5) the Connelly et al. (1992) data is based on a one-year recall survey and these types of surveys are known to result in overestimates.

Comment 7

The HHRA eliminates insignificant pathways. We support the USEPA's use of deterministic analyses to eliminate exposure pathways that are not of concern. We would suggest that additional steps could be taken to streamline the assessment. Specifically, because PCB concentrations in the surface water were below drinking water standards, there is no reason to quantify dermal exposure to water; all water contact pathways should be eliminated based on this observation alone. HP-4.7

Comment 8

Quantitative consideration of ongoing PCB sources is required. While we support the USEPA's efforts to accurately account for changes in chemical concentrations that might occur over time, there is one potentially significant factor that was not considered: current chemical inputs to the assessment area via ongoing sources. It has been well-documented that atmospheric deposition and outfall from storm sewers and combined sewer overflows (CSOs) can introduce significant amounts of PCBs and other chemicals into surface water bodies and sediments. HP-4.8

We believe that consideration should be given in the HHRA to ongoing sources. Specifically, inputs from ongoing sources should be quantified to the extent possible and the contributions from these sources to future surface water and sediment concentrations should be clearly described. It is theoretically possible that ongoing sources will contribute chemical inputs to such a degree that future sediment concentrations have been *underestimated* in the HHRA. If so, failure to account for the manner in which ongoing

sources influence future chemical concentrations will lead to uninformed decision making. Also, this information is of critical importance in the feasibility study; remediation of sediments may not be a practicable alternative if ongoing sources (e.g., sewer outfalls) will eventually re-contaminate the sediments to pre-feasibility study conditions.

Comment 9

A more balanced and thorough discussion of epidemiological evidence is required. Recent USEPA guidance indicates that epidemiological data should be presented to provide a perspective and "reality check" on the validity of the health risk estimates derived in the risk assessment (USEPA 1996). If epidemiologic data are of sufficient quality and quantity, the predictive value of the dose response relationship can be far more accurate than results derived from animal studies. Ideally, consideration of epidemiologic data would include a scholarly weight-of-evidence discussion of the published quality studies that have evaluated exposed cohorts. HP-4.9

Unfortunately, the HHRA falls far short in this regard. There are at least 12 published studies involving worker exposure to PCBs. The HHRA chooses to provide only brief summaries of three of these studies and leaves the impression that the weight of evidence indicates a causal relationship between PCB exposure and cancer. However, a careful review of the epidemiological evidence—far from supporting assertions that PCBs cause cancer—suggests just the opposite. The most recent analysis (Kimbrough et al. 1999) clearly concluded that there was little to no evidence supporting a causative relationship between PCB exposure and cancer. These findings are consistent with those of four previous studies of the same cohort or related cohorts (Brown and Jones 1981; Brown 1987; Nicholson 1987; Taylor 1988). Numerous other authors have concluded that the epidemiological evidence either has not shown an association between PCBs and cancer in humans or that the evidence is inconclusive (Danse et al. 1997; Kimbrough 1988;

James et al. 1993; Kimbrough 1995; Longnecker et al. 1997; Swanson et al. 1995; Chase et al. 1989).

In the final element to its review of the epidemiological studies on cancer and PCB exposures, the HHRA "addresses" the recent study of Kimbrough et al. (1999). It is disappointing to find that, unlike the other epidemiological studies summarized in the HHRA, the actual conclusions of Kimbrough et al. (1999) are never mentioned. Rather, the HHRA offers a critique of the study. This obviously biased "review" of the epidemiological studies serves no purpose other than to suggest a poorly hidden agenda. In additional to being peer-reviewed by the editorial board of the journal in which it was published, the Kimbrough et al. (1999) study was reviewed and endorsed by a five-member advisory committee convened by the Institute for Evaluating Health Risks.

We suggest that a more thorough and balanced discussion of the epidemiological weight-of-evidence is required. All of the studies should be described, including those of Kimbrough et al. (1999).

Comment 10

There is an inappropriate use of upper-bound estimates in the RME calculation. The HHRA states on page 5 "An estimate of the RME can be obtained by determining estimates of likely "high-end" exposure factors and then combining these high end factors with average factors...." We agree that this is the appropriate methodology for calculating an RME risk estimate. However, it appears that the HHRA has instead relied completely on the use of upper-bound point estimates for the deterministic RME calculation. Table 1 summarizes the point estimates used to calculate the deterministic RME risk for fish ingestion (the primary exposure pathway in the HHRA). As indicated in the table, each and every parameter used is at least the 90th percentile or greater. In particular, the fish ingestion rate is several-fold greater than the recommended values from USEPA's *Exposure Factors Handbook* (EFH) (USEPA 1997a) for the general

HP-4.10

population and recreational anglers. EFH recommends 6.6 g/day for freshwater and estuarine fish for the general population. For recreational anglers, the recommended intake rates are 8 g/day and 25 g/day for the average and upper-bound exposures, respectively. As mentioned previously in these comments, the angler data relied upon in the assessment is not site-specific and, therefore, may not accurately represent the fishing activities for the Hudson River.

The shortcomings associated with repeated use of upper-bound assumptions have been well established. The RME point estimate approach used in the HHRA repeatedly uses upper-bound or 95th percentile values, which ultimately leads to unrealistic and overly conservative risk estimates. This is because it is highly unlikely that the worst-case of all conditions will occur together. Thus, multiplying the 95th percentiles of exposure parameters together results in a dose approximating the 100th percentile. Burmaster and Harris (1993) have discussed this phenomenon at length. It is interesting to note that the USEPA chose to use a central tendency value only for the body-weight factor, which, because it is in the denominator of the equation, serves to increase the risk estimate. It is possible that some readers of the HHRA may interpret this as a disingenuous attempt to claim that upper-bound and central tendency estimates were used in the analysis.

We recommend that the assessment be revised by balancing the use of both upper-bound and average exposure parameters in the RME calculations for ALL exposure pathways so that the report more closely adheres to Agency guidance and provides a more accurate representation of potential upper-bound risk.

Comment 11

The risk characterization is inadequate. We believe the risk characterization discussion fails to place the estimated exposures into proper perspective. For example, we would recommend a discussion of Travis et al (1987), which reports the results of a survey of health risk estimates that have and have not triggered regulatory action at federal

HP-4.11

Superfund sites. This survey makes it clear that regulatory actions associated with the size of the potentially exposed population at the Upper Hudson never occur unless the risks are above 1×10^{-4}.

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Table 1. Upper-bound exposure parameters used in the Hudson River PCBs reassessment RI/FS

Exposure Parameter	Value	Percentile of Distribution
Fish Ingestion Rate	31.9 mg/day	90
Cooking Loss	0	100
Exposure Duration	40 years	95
PCB Concentration	28.7	95
Fraction from Source	100%	100
Bioavailability	100%	100
Exposure Frequency	365 days/year	100
Body Weight	70 kg	50

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HG-1

Hand Delivered

September 7, 1999

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RE: HUDSON RIVER HUMAN HEALTH RISK ASSESSMENT – COMMENTS

Dear Ms. Hess:

Enclosed are the comments of the General Electric Company (GE) on the U.S. Environmental Protection Agency's (EPA) "Phase 2 Report – Review Copy, Further Characterization and Analysis, Volume 2F – Human Health Risk Assessment, Hudson River PCB Reassessment RI/FS" (HHRA).

As described more fully in our letters of last month requesting additional time to comment, given the significance of this document and the amount of time EPA required to prepare it, the comment period was remarkably short. As a result, it is likely we will supplement these comments to address areas where we could not complete our analysis due to the lack of time, particularly those related to the additional information transmitted by EPA on September 2, 1999.

Overall, we find that the assessment was communicated to the public in a way that resulted in severe misunderstanding of what the current risks to human health really are. EPA properly concluded that there was no unacceptable risk from PCB exposure while swimming, wading or drinking water, but this important finding was completely overshadowed by the assessment of risks to hypothetical future anglers which is based on assumptions of exposure and PCB toxicity that are not factually or scientifically credible. The report and associated press statements failed to make clear that the calculations of risk resulting from fish consumption were hypothetical and, given the fish consumption bans that have been in effect for over 20 years in the Upper Hudson River, not pertinent to conditions today.

The assessment of risk for the future consumption of fish from the Upper Hudson River needs to be substantially revised to correct the problems detailed in the attached comments if it is to be of use in making informed decisions about what remedy, if any, is needed for the sediments in the Upper Hudson River.

Please place a copy of this letter and associated comments in the site administrative record.

If you have any questions on these comments, please let me know.

Yours truly,

John G. Haggard

Encl:

cc: Richard Caspe, U.S. EPA
William McCabe, U.S. EPA
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Comments of General Electric Company on Hudson River PCBs Superfund Site Reassessment RI/FS Phase 2 Human Health Risk Assessment

September 7, 1999

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Risk Assessment in the Human Health Risk Assessment

1.0 Introduction and Executive Summary

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

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

1. No Unacceptable Present Risk.

The crucial central conclusion of EPA's assessment of risk to human health is that there is no unacceptable risk today from the PCBs in the sediments of the Upper Hudson River.[1] There is no such risk to those who swim, wade or boat on the River or to those who drink the River water. There is no such risk from breathing the air near the River. Under the present catch and release fishery, EPA did not find any such risk to anglers or fishermen on the Upper Hudson. The Agency did not contend that the catch and release fishing regulations were being violated in any material manner. These are important findings: the present conditions on the Upper Hudson River do not present any unacceptable risk to human health. HG-1.1

2. Hypothetical Risk Relies On Highly Implausible Assumptions

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

- catch and release fishing is abandoned
- anglers, or at least a few anglers, kill and eat extraordinarily large amounts of fish for extraordinarily long periods of time HG-1.2
- these anglers only eat fish from the Upper Hudson River

[1] The Upper Hudson River is the 40 mile stretch between Hudson Falls and the Federal Dam at Troy. For reasons explained previously to the Agency, GE maintains its position that the Hudson River PCBs Superfund Site encompasses only these 40 miles and does not extend to the Lower Hudson River. H ..3

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

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

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

In the second:

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

These scenarios are beyond credibility.

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

The Agency adopts a view on the toxicity of PCBs that discounts the latest and most thorough study of the workers in GE's capacitor plants which shows that more than 7000 workers who were highly exposed to PCBs are now as healthy as the general public. Among these 7000, there were fewer cancer deaths than expected from national or local rates. The mortality rates did not exceed the national and local rates for any other disease. These are the facts that count: people actually exposed – at high concentrations – to the PCBs now found in the Upper Hudson River are healthy. More than twenty years after the use of PCBs stopped at the two GE plants there is no evidence of adverse health effects in the exposed population. HG-1.4

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

- There is no unacceptable risk today from PCBs in the Upper Hudson River. One can drink, swim, wade and boat on the River without fear. Catch and release fishing is protective of human health. HG-1.5

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

- The calculation of possible risk, based on animal studies rather than human epidemiology, is built on a series of highly implausible assumptions of how fishermen would behave if catch and release fishing were abandoned. HG-1[illegible]

Moreover, in making its calculations of risk, EPA's assessment was poorly and inadequately designed, contains calculation errors, and relies on inaccurate or inappropriate assumptions to such an extent that the risk calculations are vastly overstated and unreliable. As a result, the assessment is so seriously flawed that it should not serve as the scientific basis for decision-making for the Hudson River. HG-1

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

EPA acknowledges that it found that PCBs in the Hudson River present no material risk to those who use the river for swimming, wading, boating and other recreational uses. These findings are downplayed in the assessment and in EPA's public statements. EPA chose to emphasize the sole hypothetical risk it identified – a flawed conclusion that someone who eats large amounts of fish from the Upper Hudson River for many years may face an elevated health risk. This faulty conclusion was the heart of EPA's public presentations -- which did not fairly and directly state that this risk does not exist today because for twenty years it has been illegal to keep fish from the Upper Hudson River. HG-1.9

In fact, EPA does not contend that anyone is presently eating fish, or has eaten fish, from the Upper Hudson River in the amounts and for the number of years assumed in its risk calculations. This is supported by data from the conservation officer patrolling the Upper Hudson; from mid-1995 to mid-1998, he checked more than 1400 anglers and issued only nine tickets and three warnings. EPA's risk result is based on implausible and incorrect assumptions, some of which do not pass simple common sense tests. EPA's risk result relies on the highly improbable scenario that someone will eat one-half pound or more of fish he caught in the River every week of every HG-1.1[illegible]

year for forty years.

EPA's Assessment Overstates the Toxicity of PCBs.

EPA's assessment uses excessively high toxicity values based on animal studies and improperly rejects persuasive evidence from more than 20 human epidemiological studies. EPA's preference for animal studies and default assumptions in the face of the actual human data is arbitrary and capricious. There have been studies of the worker populations in GE's Hudson River plants over the past 20 years that demonstrate that the cancer and non-cancer toxicity of PCBs is significantly lower than EPA estimates. These studies focused not on laboratory animals but on the very workers who were exposed to PCBs daily – the PCBs that were discharged to the Hudson River and are that are now in river fish. Studies by a broad array of experts – from Dr. Renata Kimbrough to scientists from the New York State Department of Health and NIOSH - have demonstrated that these workers are just as healthy as the rest of the general population. A weight-of-evidence assessment of the epidemiological and clinical studies shows that there is no credible evidence that PCBs cause cancer in humans. HG-1.11

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

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

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

- EPA improperly relies on preliminary and flawed models to project PCB levels into the future despite EPA's acknowledgement that these models are undergoing significant revisions and have not been peer reviewed. HG-1.1

- EPA's assessment improperly relies on a study to derive fish consumption rates that was not designed for that purpose. Indeed, EPA's own guidance does not categorize this as a "key" study. The appropriate studies, designed to measure how much fish anglers eat, show much lower rates of fish consumption than the study used by EPA which shows some fishermen eating up to 1000 fish meals a year. It is not plausible to assume that only fish caught in the Upper Hudson River will be eaten for breakfast, lunch, and dinner. HG-1.13

- Finally, the assessment improperly defines the angler population, miscalculates and underestimates the annual mobility rates of anglers and does not take full account of the literature on cooking losses of PCBs. These errors collectively lead to an overestimate of potential exposure to PCBs. HG-1.1

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

The study of workers in GE's capacitor plants on the Hudson River found that, despite the high PCB levels to which these workers were exposed and that were reflected in high blood levels, death rates from cancer or other diseases were no higher than national or local rates. This is the latest in a series of studies that consistently reached similar results. EPA produces no evidence that these workers actually exhibited any unusual adverse health effects. Nevertheless, EPA erroneously dismisses the findings of the study because of alleged limitations: HG-1.1

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

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

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

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

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

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

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

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

- Although acknowledging the importance of modeling angler exposures as a series of separate annual events, the model used in the assessment fails to incorporate this approach, instead modeling angler doses as single events that often last more than 40 years. As a result, the model assumes that anglers consume unrealistic amounts of fish harvested from the same locations, cooked in the same fashion, and composed of the same mixture of species every year for periods longer than 40 years. **HG-1.16**

- The model inappropriately evaluates non-cancer risks to anglers exposed for only one or two years as if those exposures occurred over seven or more years. This leads to a significant overestimate of non-cancer risks to these anglers. **HG-1.17**

- The assessment does not adequately describe the Agency's probabilistic model. The failure to document the model properly, including presenting the model code, information on the random number generator used in the model, information on post-analysis manipulation of model output, and information on key model inputs, effectively impeded GE's ability to review and comment on the model. GE has recently received additional information from EPA and will submit supplemental comments following the company's review. HG-1.18

- The model fails to meet the standards established by EPA guidance for Monte Carlo models, including deficiencies in model design and documentation. HG-1_

- The Agency fails to separate uncertainty and variability in its risk estimate and does not provide a quantitative analysis of uncertainty although methods are available for doing so. The Agency's "sensitivity analyses" are useful for identifying factors that contribute to the uncertainty in risk estimates, but are no substitute for a quantitative characterization of the uncertainty associated with the Agency's estimate of risk. HG-1.2^

- The Agency's selection of factors to consider in its sensitivity analyses is arbitrary. The Agency failed to consider important sources of uncertainty in these analyses (e.g., uncertainty in toxicity, angler recall bias, inter-year variation in fish consumption rates and use of consumption data from multiple water bodies), while evaluating sources of uncertainty that were not appropriate (e.g., location) or of minor importance. HG-1.21

Although EPA's analysis is flawed, it is nevertheless apparent that the future risks of eating fish from the Upper Hudson River are clearly limited. It is important to retain focus on the central issue of whether a remedy will materially accelerate the time at which people can eat fish from the Upper Hudson. Nothing in this risk assessment alters the basic facts that natural recovery will lead to edible fish in the not too distant future and a remedy such as dredging will not materially accelerate that date. HG-1.22

2.0 The HHRA Overstates the Toxicity of PCBs

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

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

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

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

HG-1.2

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

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

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

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

Application of the weight-of-evidence approach to studies of the noncancer human health effects

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

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

Finally, EPA's application of the IRIS-derived value in the HHRA is contrary to Agency guidance on the use of IRIS values in Superfund Risk Assessments (EPA, 1993), which explains that using

IRIS values in Superfund risk assessments is not mandatory and that the Agency must consider other available credible and relevant toxicological information. The epidemiological information not considered in the development of the IRIS PCB toxicological values falls squarely within the type of information that the guidance requires EPA to consider.

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

HG-1.24

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

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

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

- The death rate due to all types of cancer combined was at or significantly below the expected

level. Based on national cancer death rates, 699 and 420 deaths were expected among the hourly male and female workers, respectively. Based on regional cancer death rates, 713 and 449 deaths would have been expected among hourly male and female workers, respectively. Only 586 and 380 cancer deaths were observed for the men and women, respectively.

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

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

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

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

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

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

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

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

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

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

> While the industrial hygiene data that are available are extremely limited, they suggest that the time weighted average work place air exposures of electrical capacitor manufacturing workers ranged from concentrations in excess of 1 mg/m^3 in the high exposure areas to general plant-wide concentrations of 0.05 - 0.1 mg/m^3. There is no evidence for substantially different airborne concentrations in the different plants here reviewed.

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

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

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

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

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

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

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

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

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

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

HG-1.25

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

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

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

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

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

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

2.4 EPA Improperly Excluded Uncertainty in Measures of Chemical Toxicity 2.4.1.1.1.1.1.1

HG-1.26

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

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

EPA's decision not to consider uncertainty in toxicity is unreasonable and arbitrary. Current

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

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

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

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

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

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

evaluation of uncertainty in non-carcinogenic and carcinogenic risks as a means of improving EPA decision making:

> Eventually, the majority of the Panel would like to see the whole NOAEL/uncertainty factor framework replaced by a more quantitative risk assessment approach in which all of the safety factors are replaced by distributions based on the best available data from well studied cases. The results of this would ideally be fully quantitative analyses for non-cancer effects as well as cancer risks with an understanding of both uncertainty and variability. Standards would then need to be set for safety goals. (EPA, 1999b; page 37)

> The dilemma above arises because the 10-fold factors are hard to interpret as adjustments for the means of distributed extrapolation factors or as allowances for the worst-case tail of these distributions. A distributional approach to noncancer risk analysis would resolve the dilemma by specifying the whole distribution of the factors in question. If different components of an aggregation have different uncertainties, the distributional approach easily accommodates calculation of the uncertainty of their sum, with the mean of the output distribution making the necessary extrapolation adjustments without conservatism and its spread providing a measure of the uncertainty, providing a basis for risk managers to apply allowances for uncertainty as they see fit. (EPA, 1999b; page 45)

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

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

HG-1.27

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

species; and (5) the dose-response curve for TCDD is parallel to that for individual PCB congeners. Exceptions to all of these assumptions have been reported in the literature (Safe, 1994; Pohjanvirta et al., 1995; Putzrath, 1997; Starr et al., 1997; WHO, 1997).

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

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

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

3.0 EPA's Selection of Conservative Exposure Assumptions Overestimates Risks

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

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

EPA misused the results of Connelly et al. (1992) study on which it based the fish consumption rates used in the HHRA. This study has significant limitations, causing the Agency to overestimate fish consumption rates and adding considerable uncertainty to these estimates. HG-1.2

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

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

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

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

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

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

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

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

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

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

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

substantial uncertainty to the resulting fish ingestion rates.

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

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

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

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

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

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

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

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

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

- Fish consumption advisories did not substantially affect the Maine angler results. At the time that

the survey was conducted, such advisories applied to only 200 miles of Maine's 37,000 miles of river and stream fisheries.

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

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

HG-1.29

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

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

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

There is a third and better option. One can use the available information on inter-year variation to model fish consumption rates. The data include Boyle et al. (1990), who found that 30 percent of

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

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

3.3 EPA Inconsistently Defined the Angler Population

HG-1.3

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

EPA's first definition would include those anglers who might fish the "Upper Hudson River" but might do so with less regularity than one meal per year. As documented by Boyle et al. (1990), Connelly et al. (1992), Phillips et al. (1990), Ebert et al. (1993), and Connelly et al. (1999), a substantial portion of anglers do not fish every year. This fraction may be as high as seventy-five percent of all anglers (Connelly et al., 1999). Excluding those anglers who do not fish every year

results in overestimates of fish consumption per capita and therefore, the distribution of doses is biased towards overestimation of risk. This is not appropriate. EPA should include all individuals who might consume fish from the "Upper Hudson River", including those who eat less than one meal per year.

3.4 EPA Incorrectly Calculated Exposure Duration

HG-1.31

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

$$M_1 = 1 - (1-M_5)^{1.5}$$

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

3.5 EPA Improperly Accounted for Cooking Loss

HG-1.3

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

Percent loss of PCBs can be related to cooking methods, and the method used to prepare the fish can be linked to fish species. EPA acknowledges that "[o]verall, studies support the conclusion that some PCBs are lost during cooking....but quantitative estimates of cooking losses remain uncertain." HHRA at 48. At issue is the inconsistency in the way the authors of the available

studies have reported their results. Authors have reported reductions as the amount of PCBs lost per gram of fat, per gram of fish wet weight, per gram of fish dry weight, or the total mass of PCBs lost. This inconsistency can hamper comparisons and compilations of results and increases the uncertainty associated with the determination of a single cooking loss value or a percentage loss of PCBs resulting from each of the different cooking methods. Sherer and Price (1993) developed a methodology to convert the results of cooking loss studies to a percent loss of PCBs on a total mass basis. Conversion of the results to the same units allows one to determine an average PCB loss for different cooking methods.

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

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

HG-1.3

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

Because many relevant species were omitted from the species list, a large number of responses to the survey listed meals of "Other" species. According to EPA (1999a Table 3-3), 25 percent of

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

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

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

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

with the information in the Connelly et al. (1996) survey, in which bass represented 58 percent of species preference.

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

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

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

HG-1.34

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

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

corrected and peer review completed.

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

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

For example, the modeled PCB levels of fish at Stillwater presented in the BMR exceed the actual data in the 1990s, indicating that the model is not a reliable predictor of fish PCB levels and will overpredict PCB exposure. Moreover, the projected PCB concentrations in fish presented in the HHRA differ from projected concentrations presented in the BMR. Concentrations in largemouth bass from Stillwater in 1998, presented in HHRA Figure 2-5 (approximately 7 ppm wet weight), differ from those in the BMR (Figure 7-14; approximately 5 ppm wet weight). Second, the drop seen in PCB concentrations in largemouth bass from Stillwater in 1999 (BMR Figure 7-14) is not observed in the projections in the HHRA (Figure 2-5). The HHRA references the BMR as the source for the results, which is obviously wrong. The reason for this discrepancy needs to be explained. **HG-1.35**

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

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

- EPA's model fails to satisfy the criteria established in its guidance for use of Monte Carlo analyses (EPA 1997b). This guidance sets out a number of criteria for the acceptance of probabilistic analyses. The probabilistic analysis in the HHRA fails to meet many of the criteria, including deficiencies in the model design and in documentation of the assessment. HG-1.

- Although the modeling approach outlined in the HHRA is generally sound, the actual model used by EPA is fundamentally different from and substantially more limited than the HHRA's general description of the model. On page 36 of the HHRA, EPA acknowledges that modeling PCB exposures to anglers must be performed as a series of separate annual exposure events. Unfortunately, EPA does not model anglers' doses as separate events but instead models them as single blocks of time that last for periods ranging from one year to longer than 30 years. This approach greatly limits the Agency's ability to model temporal changes in inputs and prevents the correct determination of chronic and lifetime doses. HG-1.37

- EPA failed to provide in a timely manner, an adequate description of the probabilistic model used to evaluate angler exposures, impairing the public's opportunity to analyze and comment on matters highly germane to this entire risk assessment. (As requested by GE, EPA provided additional information on September 3, 1999) HG-1.3

- The design of the model forces the Agency to assume that anglers consume unrealistic amounts of fish harvested from the same locations, cooked in the same fashion, and composed of the same mixture of species every year for more than 30 years. People's behavior does vary over time. HG-1.39

- The method used to characterize chronic non-cancer endpoints incorrectly identifies certain anglers with short-term exposures as having very high chronic doses. These anglers only fish for one or two years but are assumed to have the highest chronic doses. This assumption biases the estimates of the hazard quotient for the higher percentiles of the distribution of chronic risks. HG-1.40

- EPA's failure to separate uncertainty and variability weakens its analysis of risk. HG-1.41

- To address uncertainty in model inputs, the Agency performed a sensitivity analysis but presented the results as if it had performed a more sophisticated discrete probability analysis (DPA) (Morgan and Henrion, 1990). Although DPA can be used to evaluate the range and distribution of uncertainty, a sensitivity analysis cannot. Sensitivity analysis can only identify the most significant sources of uncertainty but cannot quantify the significance of that uncertainty. As a result, the Agency's uncertainty assessment does not support the HHRA's conclusions that 1) the findings of significant cancer and noncancer risks occur no matter what assumptions are made for model inputs, and 2) the findings of the probabilistic assessment support the point estimates. HG-1.42

- The Agency made a number of inappropriate choices in the sensitivity analysis. EPA includes sources of uncertainty (e.g., location) that are not appropriate or are of minor importance (e.g., cooking loss and mobility rates). EPA excludes factors that have a major impact on the risk estimates, including uncertainty in the cancer slope factor and the reference dose, angler recall bias, inter-year variation in fish consumption rates, and use of consumption data from multiple waterbodies. Finally, the Agency considers a fish consumption study (West et al., 1989a,b) that is irrelevant to the evaluation of risks at this site. The Agency provides no information on how it selected the sources of uncertainty considered in the sensitivity assessment. As a result, the sensitivity analysis has little or no meaning. HG-1.43

- EPA asserts that the data were insufficient to characterize uncertainty and variability jointly using a two-dimensional Monte Carlo analysis but never justifies this decision. The Agency states that it views uncertainty in distributions in terms of parametric uncertainty but does not attempt to actually define the uncertainty in the parameters of the distributions of variability. In addition, the Agency does not identify what factors or data gaps prevent it from defining the uncertainty in parameters. HG-1.44

5.0 Conclusions

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

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

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

- EPA did a poor job of communicating the fact that the risks from fish consumption calculated by EPA are hypothetical. This leads to mischaracterization of the risk to citizens using the Upper Hudson River. HG-1
- EPA's critique of Kimbrough et al. (1999) is superficial and the claim of limitations is unfounded. EPA needs to complete an objective and scientific evaluation of this groundbreaking study. HG-1
- EPA grossly overestimates the toxicity of PCBs and as a result overstates potential risks. Based on a weight-of-evidence appraisal, there is no credible information that PCBs cause cancer in humans. Additionally, there is little, if any, evidence that PCBs cause adverse effects in humans at environmental exposure levels. HG-1
- The exposure assumptions made to estimate risks to the hypothetical angler materially overstate potential exposures. Key problems include:
  - Use of the results of a flawed PCB food chain model for estimating fish PCB levels HG-1
  - Implausibly high estimates of fish consumption rates and the duration of high fish consumption. HG-

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

HG-1.50

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

HG-1.51

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

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ATTACHMENT A
A Weight-of-Evidence Assessment of the Human Health Risks of PCBs

Preface

The HHRA assesses human health risk resulting from the presence of PCBs in the Upper Hudson River using extremely conservative estimates of PCB toxicity that are based solely on the results of laboratory studies of animals. For estimating cancer risk, the HHRA uses a "cancer slope factor," or "CSF," derived from studies in which particular strains of laboratory rats have been fed massive doses of PCBs. For estimating noncancer risk, the HHRA uses "reference doses," or "RfDs," derived from laboratory studies on rhesus monkeys. Thus, the HHRA's assessment of the health risks of PCBs relies primarily on data from animal studies and does not adequately consider the human epidemiological data as well as data that would assist in assessing the relevance of the animal studies to the potential effects of PCBs on people.

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

The following is a summary of the major points made in this document:

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

- Although laboratory studies indicate that PCBs promote tumors in certain strains of rats, the weight of evidence from the human epidemiology studies demonstrates that there is no credible or consistent evidence that PCBs cause cancer in humans. This view is shared by numerous respected scientists and is apparently supported by ATSDR. In addition, this finding has recently been confirmed by the results of the largest PCB epidemiological study

yet performed (Kimbrough et al. 1999). This study found no association between high dose human exposure to PCBs and deaths from cancer or any other disease.

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

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

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

1. Background

The HHRA uses estimates of PCB toxicity that are developed using Agency guidance that emphasizes the Agency's desire to be protective of human health (IRIS, 1999). This policy has translated into a system for evaluating toxicological information and favoring evidence that suggests high levels of toxicity. This includes focusing on the species that have displayed the greatest sensitivity, use of conservative models of low dose extrapolation (used in setting the cancer slope factor), and conservative values for uncertainty factors (used in setting the reference dose). This approach may be reasonable where there are limited data on chemicals or when "screening" assessments are performed. However in the case of PCBs, where extensive information is available, it leads to a systematic exclusion of highly relevant data, including epidemiology studies, that demonstrate that PCBs have minimal toxicity in humans.

The HHRA uses the IRIS cancer slope factor (CSF) and Reference Doses (RfDs) for estimating the human health risk posed by PCBs in the Hudson River. As described in EPA's IRIS database, the CSF and RfDs were derived solely from animal studies using the highly conservative approach described above. Thus, by using the IRIS CSF and RfDs to estimate risks in the HHRA, EPA has applied an approach that ignores highly probative and persuasive evidence from the epidemiological studies of the human health effects of PCBs.

EPA has stated that the epidemiological evidence that PCBs cause cancer in humans is inadequate (IRIS, 1999) and has taken the position in several rulemakings that the human PCB clinical and epidemiological evidence is not useful in assessing the human carcinogenicity of PCBs. For reasons discussed later in this Attachment, GE strongly disagrees with EPA's position. In fact, the clinical and epidemiological data provide important information to be considered in a weight-of-evidence assessment of PCB cancer risk.

Notwithstanding the ascendancy of the weight-of-evidence approach in the scientific community for assessing human health risks, the Agency persists in disregarding the growing body of human epidemiological evidence of the effects of PCBs on humans. The HHRA's approach to assessing the risks posed by PCBs is thus inconsistent with the modern risk assessment practice.

This document discusses the weight-of-evidence approach to risk assessment and urges EPA to use this approach to assess the human health risks posed by PCBs. In addition, this document applies the weight-of-evidence approach to assess PCB cancer and non-cancer risks and derives a CSF and RfD for PCBs consistent with a sound application of this approach.

2. The Weight-of-Evidence Approach

As EPA has recognized, the weight-of-evidence approach is strongly justified by three scientific considerations. Foremost is the now well-recognized fact that not all positive findings in animal bioassays predict that a chemical poses human health hazards (Goodman and Wilson, 1991 and Gold et al., 1997, 1998). It is now known that chemicals may have different mechanisms of action and pharmacokinetics in different species. Thus, a chemical that has a particular effect in the tissues of one species of laboratory animal may not have that effect in the same type of tissue in other species or, for that matter, may have no similar effect in any other species (Gold et al., 1998). It is also well known that, for species that exhibit similar responses to a chemical, the dose at which the effect occurs may differ widely between species (Peterson et al., 1993). In addition, it is now known that chemicals that exhibit carcinogenic effects under animal bioassay conditions (typically dosing at the "maximum tolerated dose," or "MTD") may not exhibit such effects at lower doses. Even in the case of chemicals that cause cancer responses at doses well below the MTD, those doses may still be well above doses that would occur under reasonable environmental exposure conditions.[1] Finally, it is now known that cancer response is not linear with respect to dose for many chemicals that are in fact carcinogens.[2]

The second scientific underpinning of the weight-of-evidence approach is the fact that human epidemiological studies should be used to place in context the relevance of animal bioassays. It is widely appreciated that different mechanisms of action of a chemical in different species can dictate that a chemical effect in one species will differ from others (Dietrich and Swenberg, 1991; Hard and Whysner, 1994). EPA has recognized that not all animal carcinogens are human carcinogens and that the carcinogenic potency of a compound in humans will differ from its potency in animals. Both positive and negative epidemiology studies allow a direct determination

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[1] Many chemicals that have been proven to be carcinogenic at high doses in animal bioassays have not been shown to be carcinogenic in humans at or near environmental or occupational exposure levels. As an example, over 50 percent of approximately 400 to 500 chemicals have tested positive in at least one rodent species at high doses (Ames, 1989). However, only approximately 40 chemicals are known to cause cancer in humans (Doll, 1984; Paustenbach et al., 1990).

Compounds classified as tumor promoters are particularly troublesome in this regard, because they often produce rodent liver tumors in long term bioassays, but are not generally known to cause cancer in humans (1995; Schulte-Hermann, 1985; Butterworth et al.). Tumor promoters like PCBs enable increased growth of pre-cancerous cells, but do not interact with cellular DNA to cause the initial heritable change which begins the multi-stage process of cancer. The drug phenobarbital is a classic example of a rodent liver tumor promoter that has not been shown to cause cancer in humans taking this drug for many years (Butterworth et al., 1995).

[2] Differences in pharmacokinetics and susceptibility to organ toxicity complicate the issue of interspecies extrapolation (MacDonald et al., 1994). The problem exists because of the need to use a model for extrapolation from high doses in animals to low doses in humans. EPA typically estimates the human carcinogenic potency of a chemical which causes tumors in animals at high doses by using the linear default method presented in EPA (1996e). Originally, the assumption of linearity was based on an elementary theory of the mechanism of chemical carcinogenesis, in which a single chemical molecule can form an adduct to DNA, and thereby result in cancer. Tumor promotion, however, is characterized as a reversible process and the dose response relationship is expected to be nonlinear, including both a threshold dose level and a maximal response (Pitot and Dragan, 1991). EPA's recent cancer guidelines (EPA, 1996) allow for nonlinear low dose extrapolation in cases where the available data support a nonlinear mode of action (e.g., non-genotoxic agents).

EPA concedes that there are a number of chemicals which produce a carcinogenic response by mechanisms that may exhibit a nonlinear dose response curve at low doses (EPA, 1996). See also Butterworth and Slaga (1987). The increased acceptance of the nonlinearity of dose and effect at low doses is evidenced by a growing consensus among risk assessment practitioners that the linear model is inappropriate for an increasing number of non-genotoxic chemicals, e.g. dioxin, thyroid-type carcinogens, nitrilotriacetic acid, trimethylpentane (Paynter et al., 1988; Andersen and Alden, 1989; Paustenbach, 1989, EPA, 1992, 1996).

of these differences.

Although it has been stated that epidemiologic studies are not as statistically robust as animal studies and, therefore, not as useful (Silbergeld et al., 1988), this is not universally true and the direct relevance of human epidemiological studies further mitigates this concern. In many cases human epidemiology studies can and should be used to validate, confirm, or set upper bound estimates of cancer and non-cancer potency.

In general, when epidemiology data are available, it is simply not appropriate to accept only the results of mathematical models that analyze rodent data without giving serious consideration to the human experience (Cook, 1982; Dinman and Sussman, 1983; Layard and Silvers, 1989). This is exemplified dramatically by the case study of ethylene dibromide (EDB). In 1982, it was claimed that workers exposed for 8 hours per day for 40 years to the OSHA threshold limit value (TLV) for EDB of 20 ppm would incur a cancer risk of nearly 100% (999 in 1,000) based on animal studies. However, epidemiological evidence of actual cancer incidence in these workers did not show any increase in the cancer rate (Hertz-Piciotto et al., 1988; Cook, 1993).

A third important scientific underpinning of the weight-of-evidence approach is the fact that all studies -- including animal bioassays, human clinical studies and human epidemiological studies -- vary in quality and statistical power. Where results of studies are inconsistent, studies of higher quality and power should be given greater weight in assessing risk.

There are sound scientific reasons for using a weight-of-evidence approach to assess the toxicity and risks of chemicals. In most evaluations, the weight-of-evidence test applies what has become known as "causation analysis." The methodology is well recognized within EPA (EPA, 1992, 1996), although its application appears to have been limited. At least ten criteria have been proposed for establishing cause and effect relationships (Hill, 1965; Evans, 1976; Hackney and Linn, 1979, Doll, 1984; Guidotti and Goldsmith, 1986; Mausner and Kramer, 1985; Monson, 1988, Hernberg, 1992). However, as typically applied, the scientific convention applied in weight-of-evidence evaluation of either (i) a single study associating a chemical with an effect, or (ii) the universe of studies on whether a chemical causes a particular effect, requires:

- a specific effect endpoint, and
- satisfaction of all or most of six fundamental criteria (Hill 1965; Mausner and Kramer 1985; USEPA, 1985, 1996e; Monson, 1988; Rothman, 1988; Hernberg 1992; IARC 1987)

The six fundamental criteria are

- strength of association;
- consistency of association;
- temporally correct association;

- dose-response relationship;
- specificity of the association; and
- coherence with existing information (also called "biological plausibility").

None of the criteria, with the exception of temporality, should be considered necessary to establish causation. Each of the criteria, however, is important, and causation is established by the weight-of-evidence and the degree to which all six criteria are satisfied by the available data. However, the rejection of causation may be made with a high degree of confidence when three of the criteria -- temporality, consistency, and biological plausibility -- are not met (Rothman, 1988; EPA, 1996).

3. A Weight-of-Evidence Assessment of the Cancer Risks of PCBs

PCBs promoted liver tumors in three of the four strains of rats studied in chronic bioassays (EPA, 1996). The largest and most comprehensive PCB animal-feeding study ever conducted, in which the toxicity and cancer potency of Aroclors 1260, 1254, 1242 and 1016 were compared in male and female Sprague-Dawley rats, was recently reported by Mayes et al. (1998). Using a linear-quadratic multistage model, EPA (1996) calculated CSFs using the results of this study, as follows:

Sex	Aroclor	Central-tendency CPF $(mg/kg/day)^{-1}$	Upper-bound CSF $(mg/kg/day)^{-1}$
Female	1260	0.4	0.5
Female	1254	1.2	1.5
Female	1242	0.3	0.4
Female	1016	0.04	0.07
Male	1260	0.1	0.2
Male	1254	0.06[a]	0.1 [A]
Male	1242	0.03 [A]	0.08 [A]
Male	1016	0.02 [A]	0.04 [A]

Based on the results of this study, as well as the reevaluation of previous animal-feeding studies by Moore et al. (1994), EPA's current guidance recommends CSFs substantially lower than those previously recommended. EPA guidance now recommends that risk assessments should be performed using CSFs in the range of 0.07 to 2.0 $(mg/kg/day)^{-1}$, depending on the route of exposure and chemical composition (EPA, 1996).

[a] Based on a group with statistically insignificant tumor incidences.

Although PCBs promote tumors in some strains of rats in chronic bioassays, the weight-of-evidence approach requires that all available evidence be evaluated, and weighed, in assessing the carcinogenicity of PCBs in humans. To perform such an assessment, three fundamental questions must be answered: (1) Do PCBs cause cancer in humans? (2) If so, how should the degree of PCB human carcinogenicity be characterized? (3) Is there a threshold below which PCBs do not elicit a carcinogenic response?

In the case of many chemicals, such questions are difficult to answer since little epidemiological information exists. In the case of PCBs, a large and growing body of epidemiological information can be consulted to attempt to answer these questions. As discussed below, the large epidemiological database for PCBs demonstrates that there is no credible evidence that PCBs cause cancer in humans. Nevertheless, that database can also be used to conservatively assess the cancer risk of PCBs and calculate an upperbound CSF.

a. An Assessment of the PCB Epidemiological Data

As this section demonstrates, a weight-of-evidence assessment of the PCB epidemiological studies reveals that there is no credible evidence that PCBs cause cancer in humans. This conclusion is shared by numerous eminent scientists and is apparently supported by the Agency for Toxic Substances and Disease Registry. (See ATSDR 1999, at 227 "The weight of evidence does not support a causal association for PCBs and human cancer at this time.".

Over 20 epidemiological or clinical studies have investigated whether PCBs may cause cancer in humans. These studies have investigated five major cohorts and several small cohorts. Based on the findings and other characteristics of the studies, they can each be weighed for importance in contributing to the weight-of-evidence determination. The majority of the studies are negative for all types of cancers investigated -- that is, no statistically significant correlation was found between exposure to PCBs and increased cancer. In some studies, a statistically significant *negative* correlation between exposure to PCBs and cancer was found. Other studies must be regarded as clearly inconclusive because of inadequate cohort size, clear defects in study design or implementation, and/or statistical flaws. Finally, a few studies have reported a statistically significant increase in one or more types of cancer, or increased cancer mortality, but contain significant limits on their usefulness which in many instances have been acknowledged by their authors and/or EPA. We refer to these studies as "positive" for purposes of discussion, while pointing out that none of the "positive" studies provide credible evidence that PCBs cause cancer in humans.

Below, we discuss the human cancer studies to determine what relevant evidence, if any, each study provides. We then analyze, using weight-of-evidence techniques, what the studies in total tell us about the potential human carcinogenicity of PCBs. In the discussion below, the studies have been grouped into those that do not support an association between PCBs and human cancer risk (negative studies), studies that are clearly inconclusive (inconclusive studies) and studies that have reported a statistically significant elevation of a response for a cohort over the control ("positive" studies).

Negative Studies

Kimbrough et al. (1999)

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

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

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

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

Significantly, the GE workers studied in Kimbrough et al. (1999) were also studied in four previous epidemiological studies, Taylor (1988), Nicholson (1987), Brown and Jones (1981), and Brown (1987). These studies are discussed below. In each case, the PCB exposure of the GE cohort was not associated with increased cancer mortality. It is important to note that the "positive" results reported in the Brown (1987) study involved the cohort from the second plant studied by these authors, not the GE cohort.

Taylor (1988)

One of the largest studies of PCB exposed workers, this study showed no significant increases in mortality or cancers. Taylor (1988) involved a cohort of 6,292 persons employed for at least three months during the period 1946-1976 at the GE Hudson Falls and Ft. Edward capacitor plants. It

was initiated by Dr. Taylor on assignment from NIOSH to NYSDOH in collaboration with NYSDOH scientists. The study was completed at Harvard University as Dr. Taylor's Ph.D. thesis. This study showed no increase in cancer mortality or in overall mortality compared to national averages. Deaths due to malignant melanoma, lymphopoietic cancers, or the combination of liver, gallbladder and biliary cancers were not significantly elevated, and brain cancers were well below the expected value. PCB exposure was shown to be negatively associated with cancer mortality (all types combined) and lung cancer (the only cancer outcome with numbers of cases sufficient to permit a regression analysis). In other words, as PCB exposure increased the numbers of overall cancer deaths and lung cancer deaths *decreased*. As noted above, the results of Taylor (1988) are consistent with Kimbrough et al. (1999).

Nicholson (1987)

This study investigated cancer mortality in 788 employees at the GE capacitor manufacturing facilities (these plants were also studied by Kimbrough et al. (1999) and Taylor (1988); one of the plants was studied by Brown and Jones (1981) and Brown (1987)). In Nicholson (1987), the cohort was selected to improve the possibility of detecting latent cancers among workers with long exposure histories -- the criteria for selection were employment beginning before 1954 for a period of at least five years. At each facility, Aroclors 1254 and 1242 were used prior to 1970; Aroclor 1016 and, occasionally, Aroclor 1221 were used after that date. Industrial hygiene surveys in 1977 indicated that certain job locations involved PCB exposures ranging from 300 to 1,000 ug PCB/m^3 air.

The numbers of deaths attributed to all causes and all cancers were less than expected. The cohort was also divided into low, medium and high exposure groups and the results of the analysis revealed no association between PCB exposure and mortality for any cause. In fact, no cancer deaths occurred in workers having 30 or more years since first exposure, i.e., no deaths were observed in the "30 or more year" category when 8.3 cancer deaths would have been expected.

Hoyer et al. (1998)

This study found no relation between PCBs and breast cancer. Hoyer et al. (1998) was a large and well-conducted case control and prospective study of 268 women who contracted cancer out of 7,712 women whose blood serum was sampled 17 years earlier. Each of the 268 women was carefully matched with two cancer-free women from the same cohort, and serum samples were taken in 1996-1997 from 240 of the women with breast cancer and from 477 of the controls. The samples were analyzed for 28 PCB congeners and 18 organochlorine pesticides. Statistical analyses were performed by multiple logistic regression for each PCB congener, total PCBs, and the pesticides. Adjustments were then made for potential confounding factors, except age, which was the main matching variable. Hoyer et al. (1998) found that although cancer risk was associated with serum levels of dieldrin and possibly beta-HCH, "no association was apparent for total DDT, p,p'-DDE, p,p'-DDT, total PCB or any specific congeners, kepone, lindane, or chlordane metabolites."

Hunter et al. (1997)

This study found no association between PCBs and breast cancer. The authors tested the hypothesis that higher blood levels of DDE and PCBs are associated with an increased risk of breast cancer by measuring the levels of organochlorines in 240 women with breast cancer and 240 matched control women using blood samples prospectively collected from 1989 to 1990. The mean level of PCBs was 5.08 ± 2.51 ng/ml in the women with breast cancer and 5.16 ± 2.26 ng/ml in the controls. After extensive analysis, the authors concluded that their "data do not support the hypothesis that exposure to DDE and PCBs increases the risk of breast cancer." Hunter et al. (1997) has been acknowledged as a definitive result showing no link whatsoever between PCB exposure and breast cancer, see, e.g., Safe and Zacharewski (1997).

Guttes et al. (1998)

Guttes et al. (1998) investigated whether concentrations of PCB congeners and certain other chlorinated organic compounds were higher in surgically removed breast tissue from women whose tumors were found to be malignant than in breast tissue from women whose tumors were benign. The authors' conclusion was as follows: "In light of these observations and taking into account the contradictory results of measurements of chlorinated hydrocarbons in human tissue, blood serum, and plasma . ., it must be assumed that there is no correlation between concentrations of these substances in the human body and breast cancer."

Mussalo-Rauhama (1990)

This study reported on the concentrations of residues of polycyclic aromatic hydrocarbons and neutral organochlorine compounds in breast fat of 44 breast cancer patients and 33 controls from the Helsinki, Finland area. Tissue samples were evaluated for organochlorine compounds including DDT, hexachlorobenzene (HCB), hexachlorocyclohexane (HCH), and PCBs. The only risk factor identified was a statistically significant increase in the concentration of beta-HCH in breast cancer patients. No correlation was found between PCB concentrations and breast cancer incidence. In fact, the mean concentration of PCB was less in breast cancer patients (1.05 mg/kg fat) than in controls (1.30 mg/kg fat).

Krieger et al. (1994)

Krieger et al. (1994) conducted a nested case-control study of serum DDE and PCB levels in 150 patients with breast cancer. Serum DDE and PCB levels were not significantly different between the case and matched control groups and the authors concluded that their work "did not support the hypothesis that exposure to DDE and PCBs increases the risk of breast cancer."

Greenland et al. (1994)

This study was undertaken to address earlier reports of excess cancer mortality associated with employment at a large transformer manufacturing plant. Subjects were restricted to 1821 white males because nonwhites and females were too few to allow for adequate statistical power.

Exposures to several industrial compounds were evaluated for possible association with cancer mortality including Pyranol (a mixture of PCBs (45-80%) and tri and tetrachlorobenzene (20-55%)), benzene, trichloroethylene, other solvents, machining fluids, asbestos, and resin systems. The authors concluded that the only unequivocally positive association involved polymer resins (containing asbestos) and lung cancer.

Unger and Olsen (1980); Unger et al. (1984)

Unger and Olsen (1980) examined 71 samples of abdominal adipose tissue for PCB and DDT content. Twenty seven samples were from individuals with cancer and 44 were from individuals without cancer. At least 11 types of cancer were represented in the study. The authors concluded that there was a possible correlation between the concentrations of PCBs and DDT and cancer, but that the association was not conclusive. In Unger et al. (1984), breast fat tissue samples where examined to determine if a correlation could be demonstrated between the concentrations of PCBs and DDT and breast cancer. They evaluated fat samples from 32 women with cancer and 56 non-cancer controls. The authors reported no significance and concluded that "it seems that the accumulation of PCB and DDT measured in breast fat tissue do not relate to the occurrence of mammary cancer".

Wolff et al. (1993)

This study is one of the few that controlled for known breast cancer risk factors. The study used a subset of the 14,290 women who participated in the New York University Women's Health Study. Blood serum concentrations of DDE, DDT, and PCB from 58 women who developed breast cancer were compared with 171 randomly chosen controls. An association between DDE and breast cancer was found. With PCBs, the nominal trend for increased breast cancer risk with increasing levels of PCBs was not statistically significant, and when both DDE and PCBs were modeled together, the association with PCBs further decreased.

Zach and Musch (1982)

In this study, mortality rates were reported for 89 workers involved in the manufacture of PCBs in a plant in Illinois. Among the 30 deaths in this cohort, no statistically significant increase in mortality from all cancers or any cancer type was noted.

Bertazzi et al. (1982, 1987) and Tironi et al. (1996)

These studies present a series of investigations on workers in an Italian capacitor manufacturing plant which used a series of PCB mixtures from 1946 to 1980. Between 1946 and 1964, 54% chlorine PCB mixtures were used. Starting in 1965, the plant began to phase out use of the 54% mixture, replacing it with a 42% mixture. By 1970, only the 42% mixture was used.

As discussed below, the Bertazzi et al. (1982) and (1987) studies were previously considered inconclusive. With the publication of Tironi et al. (1996), the finding for this cohort must now be considered negative.

The progression of the "Bertazzi" studies from 1972 to 1996 points out the problems of drawing conclusions from epidemiology studies having very low number of deaths. For example, the International Agency for Research on Cancer cited the earlier Bertazzi results prominently in their decision to reclassify the human mortality data from the classification "possibly carcinogenic" (2B) to "probably carcinogenic" (2A). Based on the latest data, this reliance was unfounded.

Bertazzi (1982) presents the initial findings of the mortality study of workers in the capacitor manufacturing plant studied by Tironi et al. (1996). The authors analyzed the 27 deaths that occurred between 1954 and 1978, and reported a statistically significant increase in total cancers among males and in all cause mortality among females, compared to local population mortality rates. Statistical significance was not achieved, however, for any specific cancer type.

Bertazzi et al. (1987) was a follow up study in which the cohort selection criteria were changed from six months employment to only one week of employment and both production and non-production workers were included in the cohort. The new cohort had 2,100 members. The authors followed the cohort's mortality experience from 1946 to 1982. Sixty-four deaths (3% of the cohort) were reported. Mortality due to all cancers (14 observed vs. 5.5 national and 7.6 local) and due to cancer of the gastrointestinal tract (6 observed vs. 1.7 national and 2.2 local) was significantly increased among male workers. Death rates from hematologic neoplasms and from lung cancer were also elevated, but not to a statistically significant degree. Overall mortality was significantly increased above local rates (34 observed vs. 16.5 local) in the female population. Total cancer deaths (12 observed vs. 5.3 local) and mortality from hematologic neoplasms (4 observed vs. 1.1 local) were also significantly elevated over local rates in the female population.

Due to limitations in study design, the excess cancers reported in Bertazzi et al. (1987) should not be given serious consideration even if Tironi et al. (1996) had not concluded that, over a longer latency period, cancer deaths were not elevated.[3] The authors themselves conceded that their study "did not permit a causal association to be either proved or dismissed." Two of the six gastrointestinal cancers were in workers who had no reported direct PCB exposure and a third was in a worker who began employment, and thus exposure, at an advanced age. Further, if all cancers not likely to be related to the male workers' work history are subtracted (those with no known PCB exposure and those with one or less years of exposure), the mortality rate from all cancers is not different from the national or local mortality rate. Similarly, the statistical excess of hematologic cancer is lost if those with short latencies are excluded. Therefore, it does not appear that the excesses reported by Bertazzi et al. (1987) are related to PCB exposure.

[3]The results of the Bertazzi et al. (1987) study are also limited by several factors, including the small number of cancer cases observed, the limited latency period, the lack of a pattern or trend when the data were analyzed by duration of exposure, and some deaths in males with low potential for direct PCB exposure (Kimbrough, 1987; ATSDR, 1998; IRIS, 1999). A major problem in the study design was the one week minimum period of employment required for inclusion in the study and the inclusion in the cohort of workers who had no PCB exposure. This makes it difficult to assume that excess cancer cases are attributable to PCB exposures rather than to other factors. This study also did not show a dose-response relationship or any direct relationship between latency and the disease. EPA appears to consider the results of the study inconclusive because of lack of a dose-response relationship and the small number of cancers in the cohort (IRIS, 1999).

[4] EPA has failed to consider this paper in its most recent assessment of PCBs (IRIS, 1999). A copy of the paper and an English translation is attached.

Tironi et al. (1996)[4] is an updated report on the same cohort examined by Bertazzi et al. (1987). The mortality rate was studied for the period 1954-1991. The article noted the exceptionally high air measurements of Aroclor 1254 ranging from 5.2 to 6.8 mg/m^3 in 1954, reduced to 0.048 to 0.275 mg/m^3 in 1977. Similar levels persisted until 1982 even though PCB usage ended in 1980. The authors noted the ubiquitous contamination of surfaces in the plant, including offices located in the same building.

The cohort comprised all workers in every department of the plant, including administration, who worked in the plant a minimum of one week, but the focus was on women workers because most of the capacitor manufacturing work was performed by women. Thus, a total of 1556 women were studied, for a total of 44,328.5 person-years. Follow-up reached 99% participation – only 16 workers could not be traced. Local mortality data were used for comparison.

In the updated results, previously reported excesses of cancer (based upon very small numbers of deaths) were no longer evident. The only significant excesses reported for women were for all causes of mortality where 47 deaths were observed versus 34.4 expected, a number that was driven by the excess deaths due to accidents and traumas (ICD8 codes 800-999) where 12 deaths were observed versus 3.7 expected. Total deaths due to malignancies were 19 observed versus 16.1 expected – not statistically significant. For the men, there was no statistically significant excess for any classification of death. Total deaths due to malignancies were 20 observed versus 18.4 expected

EPA Incorrectly Dismissed the Kimbrough et al. (1999) Study

The HHRA sets forth several alleged "limitations" of the Kimbrough et al. (1999) study and states that the study is undergoing peer review. Prejudging the outcome of the peer review, the HHRA then states that the Kimbrough et al. (1999) study will likely not lead EPA to reassess its views regarding the cancer potency of PCBs. HHRA, Appendix C, at C-2 to C-3 Each of the "limitations" cited by EPA is based on a misunderstanding of either the extent of the workers' exposure to PCBs or to the length and latency of that exposure. EPA's perceived limitations are addressed below.

Alleged Limitation: "More than 75% of the workers in the study never worked with PCBs."

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

All employees at the plants were exposed to PCBs to varying extents well above the environmental background levels. This is because the way PCBs were handled at the plants resulted in elevated PCB concentrations in the workplace air. PCBs were heated to better impregnate the thin paper that was placed between the sheets of aluminum foil in the capacitors.

[4] EPA has failed to consider this paper in its most recent assessment of PCBs (IRIS, 1999) A copy of the paper and an English translation is attached

After the canisters were filled by immersion in open tanks containing PCB Aroclors, the uncovered canisters were placed into vacuum ovens, thus increasing the rate of volatilization of the PCBs. When the ovens were opened, PCBs both as a gas and as an aerosol, were released into the air around the ovens and were circulated by the ventilation system of the building. As pointed out by Kimbrough et al. (1999), the same air ventilating system served the entire building in which capacitor filling was performed, including the shipping and winding areas, the offices, and the break rooms. A similar finding was reported by Bertazzi et al. (1986) and Tironi et al. (1996).

Dermal exposure was obviously highest for workers employed in filling capacitors. However, due to the dispersion of PCB aerosols through the shared building ventilation system, virtually all surfaces within the plant became contaminated with PCBs. Therefore, all plant employees had dermal exposure (Nicholson, 1987).

These conclusions are confirmed by blood measurements of persons who worked in capacitor plants where PCBs were being used but who had no direct exposure to PCBs. At the GE capacitor plants, serum PCB levels of persons of no direct exposure averaged 50.4 ppb Aroclor 1242 and 11.3 ppb Aroclor 1260, at least an order of magnitude above today's levels for persons exposed to environmental sources. The continuing exposure of persons working in the plants is demonstrated by average measurements of 28.3 ppb of lower chlorinated PCBs (low Cl) and 7.8 ppb of higher chlorinated PCBs (high Cl) for persons employed after the usage of PCBs was discontinued in the plants (Lawton, et al., 1981). Wolff, et al., (1992) also reported blood levels for persons with low or indirect exposure in the same plants studied by Lawton (1981). She reported a geometric mean of 39 ng/ml (ppb), which dropped by only 14% to 33 ng/ml, 46 months later. Similarly, Smith et al. (1982) reported mean serum levels of 89 (low Cl) and 22 (high Cl) ng/ml (ppb) for capacitor plant workers who had never worked in impregnation or maintenance jobs at the Bloomington, IN plant.

These findings document that the exposure of persons working in the GE and other capacitor plants were exposed to PCB levels well above those experienced by the general population, and that their exposure persisted for extended periods.

Furthermore, as pointed out by Kimbrough et al. (1999), workers did not always hold the same jobs and, therefore, the number of workers with the highest exposure to PCBs is much larger than the number of workers involved with filling capacitors at any one point in time. Workers rotated through jobs with high exposure, with undefinable exposure (which may have involved high exposure, low exposure, or both), and with low exposure. The four groups of workers in the study -- male hourly workers, female hourly workers, male salaried workers, and female salaried workers -- were always analyzed separately.

Finally, it is disingenuous and inconsistent for EPA to suggest that the GE plants provide a poor cohort for an epidemiological study of the health effects of PCBs. The same cohort was studied in Brown (1987), a study cited with approval by EPA in the HHRA (Appendix C, at C-2) and in IRIS (1999). Concerns about the exposures of this cohort to PCBs is evidenced by the fact that four other studies have been performed by NIOSH, NYSDOH, Mt. Sinai Hospital, and Harvard

University on these workers (Brown and Jones, 1981; Brown 1987, Nicholson 1987, and Taylor (1988). For EPA to suggest now--after the last two studies of the cohort have been negative--that the cohort was not appropriate for study in the first place, is preposterous.

Alleged Limitation: "The actual level of PCB exposure in the remaining workers could not be confirmed."

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

Nicholson (1987) investigated PCB concentrations in workplace air at several capacitor plants using PCBs, including plants that were the subject of studies that EPA cites with approval. The GE plants were also included in this evaluation. The exposure descriptions in Nicholson (1987) go into great detail. Nicholson (1987) arrived at the following conclusion: "While the industrial hygiene data that are available are extremely limited, they suggest that the time weighted average work place air exposures of electrical capacitor manufacturing workers ranged from concentrations in excess of 1 mg/m^3 in the high exposure areas to general plant-wide concentrations of 0.05 - 0.1 mg/m^3. There is no evidence for substantially different airborne concentrations in the different plants here reviewed." The PCB air concentrations reported by Nicholson (1987) are quite consistent with the concentrations cited in Kimbrough et al. (1999), which were measured in the winding area and shipping area where workers did not have the highest exposure to PCBs.

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

Response: GE does not understand the basis for EPA's estimate of workers employed for less than one year. It is clear that even workers who were employed for relatively short periods of time carried body burdens of PCBs much higher than those carried by members of the general population.

Further, each member of the Kimbrough et al. (1999) cohort was employed at one or more of the plants for at least 90 days. The HHRA cites with approval the studies of Bertazzi et al. (1987), and Sinks et al. (1992), Brown (1987). HHRA, Appendix C, at C-2. The Brown (1987) cohort, like the Kimbrough et al. (1999) cohort, used an employment cut-off of 90 days. The Bertazzi et

al. (1987) cohort included workers employed for as little as one week. The Sinks et al. (1992) cohort included workers employed for as little as one day. EPA states no basis whatsoever to suggest that the employment cut-off used by Kimbrough et al. (1999) was in any way unusual or inappropriate.

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

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

Second, EPA's criticism again over-reaches. The average follow-up time for the workers in Kimbrough et al. (1999) was 31 years, providing a very long latency period in which to determine whether there was a statistically significant increase in mortality due to cancer or other causes. The cohort was followed through 1993, providing 120,811 person years of observation for men and 92,032 person years of observation for women. In this cohort 763 (19%) males and 432 (14%) of the females have died. Thus, Kimbrough et al. (1999) has by far the longest latency period and by far the largest number of deaths of any of the PCB epidemiological studies. It is incomprehensible that EPA would criticize Kimbrough et al. (1999) on this ground when all other studies, including studies cited with approval by EPA, had much shorter latency periods and evaluated much smaller numbers of deaths.

Alleged Limitation: "The study did not investigate vulnerable populations such as children, the elderly, or people with existing health problems."

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

Inconclusive Studies

Hay and Tarrel (1997)

The cohort in Hay and Tarrel (1997) comprised 225 individuals who were involved in the spraying of phenoxy herbicides on electrical distribution rights-of-way. The herbicides were mixed with oils, including used PCB transformer oils. The study cannot be used for characterizing potential health effects resulting from exposure to PCBs because exposure was not quantified, and the used oils were presumed to be contaminated with polychlorinated

dibenzodioxins and polychlorinated dibenzofurans.

Rylander and Hagmar (1995)

This study compared the cancer incidence and mortality of Swedish fishermen's wives with the general Swedish population. Dietary surveys revealed that fishermen's wives on average consumed more fish than the general Swedish population and, although the concentrations of organochlorine compounds was not measured in this study, the premise is that fish are a significant source of dietary organochlorine compounds and may be responsible for increased cancer incidence and mortality. The authors conclude that the study supports, but does not prove, the hypothesis of an association between exposure to a mixture of persistent organochlorine compounds through fish consumption and increased risk for breast cancer. Because this study makes no attempt to quantify the concentrations of the various organochlorine compounds (including PCBs) that were presumably present in the fish, the finding of increased risk for breast cancer can not be correlated with any single contaminant.

Wasserman et al. (1976)

Wassermann et al. (1976) measured the concentration of several organochlorine compounds in nine women with breast cancer and compared them with five controls. The authors observed that generally all of the organochlorine compounds were found at elevated concentrations in cancerous breast tissue compared with normal breast tissue or adjacent adipose tissue. However, the authors acknowledge that, due to the small number of cases studied, the data are inadequate for the purpose of drawing conclusions.

Yusho and Yu-Cheng

In 1968, about 1500 persons in Japan became ill after consuming rice oil that was accidentally contaminated with a PCB mixture (the "Yusho" incident) (Amano et al., 1984). A similar incident, known as "Yu-Cheng," occurred in Taiwan in 1979. EPA has suggested that increased incidences of liver cancer may have resulted from consumption of the rice oil (IRIS, 1999).

The cause of the incident was extensively studied and the rice oil was found to contain high levels of polychlorinated dibenzofurans ("PCDFs"), a chemical that is 100 to 1,000 times more toxic than PCBs. After finding that workers exposed to much higher levels of PCBs showed minimal adverse health effects, and after performing dose-response studies on the rice oil mixture, Japanese and Taiwanese scientists concluded that PCDFs were the prime causal factor in the Yusho and Yu-Cheng incidents (Kashimoto et al., 1986).

It is now generally recognized that most of the effects observed in these two outbreaks were caused by the ingestion of the polychlorinated dibenzofurans. The Halogenated Organics Subcommittee of EPA's Science Advisory Board reviewed a PCB health advisory from EPA and concluded that:

> Recent studies indicate that the major etiologic agents in Yusho were polychlorinated

> dibenzofurans rather than polychlorinated biphenyls . . . . Thus, a discussion of the human health effects of polychlorinated biphenyls should not use 'Yusho' as an example. Industrial exposure data more accurately reflect human health effects. (Doull and Abrahmson, 1986).

EPA appears to agree with this reassessment (IRIS, 1999).

"Positive" Studies

Bahn et al. (1977); Lawrence (1977); NIOSH (1977)

Bahn et al. (1976, 1977) evaluated the incidence of tumors occurring among workers at a New Jersey petrochemical facility where Aroclor 1254 had been used from 1949 to 1957. A significantly increased incidence of malignant melanomas was observed among research and development workers (2 of 31) and refinery personnel (1 of 41). In an update of that same study, NIOSH (1977) observed eight cancers in the total study population (5.7 expected). Three of these tumors were melanomas and two were pancreatic cancers. The incidence of these tumor types was reported to be significantly above calculated expectations, although no data were presented.

The results of this study are suspect due to small cohort size, the fact that the workers in this facility were exposed to numerous other chemicals, and the fact that the expected cancer rates were based on U.S. population data rather than on local rates (NIOSH, 1977). EPA states that the results of NIOSH (1977) are inconclusive (IRIS, 1999).

Brown and Jones (1981); Brown (1987)

Brown (1987) found an excess risk of cancer of the liver, biliary tract, or gall bladder in 2,588 workers (1,270 male, 1,318 female) from two capacitor manufacturing plants, a GE plant in New York and an Aerovox plant in Massachusetts. The workers had worked for at least three months in areas where they received heavy exposure to PCBs. Exposure was to Aroclors 1254, 1242 and 1016 (Lawton et al., 1981). The workers were also exposed to other chemicals, including trichloroethylene, toluene, and methyl isobutyl ketone. The first evaluation of this cohort (Brown and Jones, 1981) found increased cancer mortality that was not statistically significant. After an additional seven years of observation (Brown, 1987), two additional cancers of the liver, gall bladder or biliary tract were observed, making the cancer increase in this combined cancer grouping significant. Among the grouped cancers, four of the five occurred in women at the Aerovox plant. There was no increase in the number of rectal cancers from the previous study. For the total cohort, total mortality and cancer mortality were less than expected. Total cancer among the cohort at the GE plant was significantly less than expected (18 observed versus 31 expected).

According to ATSDR (1998), limitations and confounding factors in Brown (1987) include the small number of cases and the fact that PCB blood levels were higher in the plant with the lower incidence of cancer. Moreover, the study failed to account for several factors particular to the

plant where the increased cancer incidence was noted, including ethnicity and life style (the workers were from a harbor/fishing town where alcohol consumption and smoking behaviors were high). Furthermore, of the five liver grouping cancers, four of the workers had worked at the plant 1.5 years or less and the other worker worked at the plant less than 10 years. Of the five cancers, only one was a primary liver tumor (the type of tumor predicted by animal studies) and at least one had metastasized from another site (and was therefore incorrectly identified as a liver tumor). Finally, the lack of consistency in results between the two plants indicates that PCB exposure, which was common in both plants, was not responsible for the excess.

Brown (1987) concluded that his work provided only "limited information" associating PCBs with the liver grouping cancers because: (1) misclassification of the cause of death is quite possible for cancers in this category; (2) most of the cancers were not of the expected type; and (3) the study failed to demonstrate reasonable expected patterns of dose response and latency. In light of these confounding factors and limitations, ATSDR states that "the liver cancer cannot be unequivocally attributed to PCB exposure." EPA suggests that the study is inconclusive because no dose-response relationship was apparent and the number of cancers in the cohort was small (IRIS, 1999).

Hardell et al. (1996)

Hardell et al. (1996) published a case-control study of 27 patients with non-Hodgkin lymphoma ("NHL") to determine if there was a likelihood of higher PCB serum levels in these cases. While there was no significant difference in total PCB levels between the NHL cases and controls, some PCB congeners showed an association. Hardell et al. (1996) concluded that "[s]ince immunosuppression is an established risk factor for NHL, our results are of interest in the etiology of NHL but need to be confirmed in larger studies."

Loomis et al. (1997)

Loomis et al. (1997) conducted a mortality study of 138,905 men who worked for at least six months between 1950 and 1986 at five electrical power companies in the United States. The study reported that mortality due to melanoma was correlated with self-reported PCB exposure (no PCB measurements were taken), although all-cause mortality, total-cancer mortality, mortality due to liver cancer, and incidence of brain cancer did not correlate with PCB exposure.

When Loomis et al. (1997) examined relative risk by occupational category, only one small subgroup of mechanics who had worked zero to five years demonstrated a statistically significant association. No association was seen among mechanics with greater exposure, nor was there an association seen among electricians, linemen and cable splicers, or laborers and material handlers. No association was found between total career exposure to PCBs and melanoma.

Loomis et al. (1997) also divided the cohort into five, ten and 20 year lag periods, then analyzed each of these lag periods by three categories of total career exposure. Of the nine categories (three lag periods by three levels of exposure), three yielded statistically significant findings. However, these significant findings are questionable, as one of the three categories only saw one

death due to melanoma and another saw only two deaths.

The authors note several limitations of the exposure assessment of this study:

- the evaluation of the worker's exposure to PCBs and other chemicals was indirect and based on judgment

- the authors were unable to directly assess the use of personal protective equipment, and

- the quality of information on exposure to sunlight, a potentially important confounder, is of some concern. The authors also lacked information about exposure to sunlight during leisure time. The authors note, "A strong association of melanoma with recreational exposure to sun could distort our results, if that exposure were differential by level of exposure to PCBs. . ."

Moysich et al. (1998)

Moysich et al. (1998) examined the effects of PCBs and a variety of other chlorinated organic chemicals on postmenopausal breast cancer risk. The study included 154 breast cancer cases and 192 controls and sought to correlate breast cancer incidence with PCB serum levels. With respect to PCBs, the study found that higher serum levels of total PCBs, moderately chlorinated PCBs, more highly chlorinated PCBs, and greater number of PCB congener chromatogram peaks were not associated with increased breast cancer risk. However, there was some indication of a modest increase in risk for women with detectable levels of less chlorinated PCBs. Moreover, among parous women who had never lactated, there was some evidence for increased risk associated with higher serum levels of total PCBs, moderately chlorinated PCBs, and greater numbers of detected PCB congeners. The authors concluded that the "results suggest that an increase in risk of postmenopausal breast cancer associated with environmental exposure to PCBs , if at all present, is restricted to parous women who had never breast-fed an infant."

The limited findings of Moysich et al. (1998) are subject to several limitations and confounding factors, some of which were noted by the author, including:

- Organochlorine body burdens may be more accurately measured among women who have never breast-fed, presumably because their body stores of PCBs have not been reduced by nursing. This suggests that the accuracy of extrapolations for estimating body burdens, as well as measurement precision for PCBs, will necessarily be covariant with lactation history Thus, cause and effect relationships cannot clearly be determined.

- The finding with respect to the relation of the less-chlorinated congeners to cancer risk was problematic, because these compounds are metabolized rapidly, and measured levels reflect only recent exposure, not exposure at the time critical for tumorigenesis.

- The low participation rates in both the case and control groups may have introduced error due to

selection bias. Among the breast cancer case group, nonparticipation may have resulted in a case sample that was not representative of all women with breast cancer.

- There is evidence that lactation reduces both the probability of breast cancer and the level of PCBs.

Rothman et al. (1997)

Rothman et al. (1997) published a case-control study examining 74 persons with non-Hodgkin Lymphoma ("NHL"). The researchers' *a priori* hypothesis was that exposure to DDT would be associated with increasing serum concentrations of DDT. No association for this exposure was found. However, in examining the data, a dose-response relationship was found between NHL and lipid-corrected serum PCB concentration. Rothman et al. (1997) noted: "These results should be regarded as hypothesis-generating. Before causal inferences can be made about exposure to PCBs and increased risk of non-Hodgkin lymphoma, our findings require replication and the biological plausibility of the association needs further investigation." The authors also noted that past studies had documented serum levels in occupational studies of at least one order of magnitude greater than those found in his cases -- yet no association was found between NHL and PCBs. Thus, the study concluded that: (1) "it is possible that confounding was present in our studies -- i.e., that an unrecognized risk factor was associated with PCB concentrations and, more strongly, with risk of non-Hodgkin lymphoma;" (2) "the inconsistency between our findings and those from studies of PCB-exposed occupational cohorts needs to be explained"; and (3) "the biological plausibility of this association requires further investigation."

Sinks et al. (1992)

Sinks et al. (1992) conducted a retrospective cohort mortality analysis of 3,588 workers who were employed for at least one day at a Westinghouse capacitor manufacturing plant between 1957 and 1977. Aroclor 1242 was used in this plant through 1970, and Aroclor 1016 was used from 1970 to 1977. Mortality from all causes and from all cancers was less than expected. A significant increase in mortality rate was observed for skin cancer (8 observed[5] vs. 2 expected) and death rates from brain and nervous system cancers were non-significantly elevated over expected rates. No excess deaths were observed from cancers of the rectum, lung, or liver, biliary tract and gall bladder, or from hematopoietic malignancies. Based on a cumulative dose estimate, which incorporated information on job station history, limited PCB environmental sampling data, and serologic data, the authors were not able to establish a clear relationship between exposure to PCBs, latency or duration of employment and risk for malignant melanoma. Sinks et al. (1992) point out that the skin cancer excesses are not consistent with those of similar studies. The authors also point out that mortality may not be the best index of risk for malignant melanoma, as survival can be affected by differences in health care quality. In addition, other limitations include the lack of evaluation of exposures to other chemicals (including isopropyl biphenyl, toluene,

[5]One of the 8 individuals was diagnosed with skin cancer two months prior to employment at the capacitor plant. One cancer was reported as melanoma of the gallbladder, and was obviously misclassified.

xylene, methyl ethyl ketone, trichloroethylene, and methyl chloroform), the relatively short latency period, misclassification of tumors, and the small number of deaths within the cohort. Sinks et al. (1992) concluded that the results of their study should not be interpreted as demonstrating a causal relationship between PCBs and malignant melanoma and cautioned that "the possibility that the results are due to chance, bias, or confounding cannot be excluded." EPA suggests that the study is inconclusive because no dose-response relationship was apparent and the number of cancers in the cohort was small (IRIS, 1999).

Yassi et al. (1994)

This study examined a cohort employed between 1947 and 1975 in a transformer manufacturing plant in Canada. Yassi et al. (1994) reported that only a few transformers were filled with PCBs; the vast majority were filled with mineral oil. No overall increase in cancer deaths was found, but deaths due to pancreatic cancer were increased.

ATSDR (1998) notes "severe limitations" to the study, including the fact that employees were exposed to chemicals other than PCBs and no medical histories of the employees were provided. In addition, the correlation between PCB exposure and pancreatic cancer appears spurious because, in the group with the highest SMR, only three cancers were reported, and two of these were in individuals who worked at the plant for one year or less. One of these individuals died within one year after leaving the plant. Wong (1995) and ATSDR (1999) conclude that these two employees had neither sufficient exposure duration nor latency for the cancers to be attributed to PCB exposure.

Gustavsson et al. (1986), Gustavsson and Hogstedt (1997)

Gustavsson et al. (1986) sought to determine whether there was an excess cancer incidence in 142 Swedish male capacitor manufacturing workers who were exposed to a 42% chlorine PCB mixture between 1965 and 1978, providing a mean exposure duration of 6.5 years and a median latency period of 13 years. The mean exposure measured in 1973 was 0.1 mg PCB/m^3 air, and exposures were likely higher in prior years. Compared to national mortality rates, no excess cancer incidence for all cancers or any specific cancer was found, and overall mortality was slightly below the expected rate. A subgroup of 19 individuals within the highest exposure group was analyzed separately and there was no increase in mortality or cancer incidence in this group.

Gustavsson and Hogstedt (1997) followed up on Gustavsson et al. (1986) by expanding the cohort to include all capacitor workers at the plant regardless of nationality (242 workers) and adding 11 years of follow-up for cancer incidence and nine years for mortality. The authors reported that there was a significantly increased mortality from cardiovascular diseases among those employed for at least five years in high exposure jobs, with a latency of 20 years. The authors stated that the reason for this excess is unknown, given that no other PCB epidemiological study had reported an excess of cardiovascular disease. The total cancer incidence for the cohort was lower than expected, and no statistically significant increase in cancer incidence was found for any tumor type. However, the authors reported two liver cancers in the cohort and found this of special interest because an increase in liver tumors was also seen at

significant levels in Brown (1987)

The authors noted that their study was small and that the two cases of liver cancer are insufficient to draw conclusions regarding the relationship of liver cancer to PCB exposure. The authors also noted smoking is a risk factor for heart disease and that their study had not controlled for this behavior. The study provides no information regarding other chemicals to which the workers had been exposed, and did not control for any risk factors, including alcohol consumption. Given the small size of the cohort, the fact that an increase in cardiovascular disease has not been seen in any other PCB-exposed cohort, and the small number of cases of liver cancer, the study must been deemed inconclusive.

b. Weight-of-Evidence Analysis of Human Cancer Studies

As noted previously, the weight-of-evidence approach can be used to reach conclusions from a variety of data, not all of which is consistent. The weight-of-evidence approach avoids undue conservatism[6] and reflects the modern views of the scientific community and a growing number of regulators that not all scientific data are equal, and that only data of similar quality should be compared when drawing conclusions regarding toxic effects based on multiple studies. The weight-of-evidence philosophy represents an important refinement that should be applicable to both hazard identification and dose response assessment (Sielken, 1985; Anderson, 1989; Gray et al., 1993). EPA's (1996) proposed cancer risk guidelines also embrace this philosophy. The benefit of using a "weight-of-evidence" approach is that the results of several high quality toxicity studies will not be disregarded simply because the results of one or two poorly controlled ones have dissimilar findings.

As discussed previously, the scientific convention applied in weight-of-evidence evaluation of epidemiological studies requires (a) the observation of a specific endpoint, and (b) the meeting of other criteria (i.e., strength of association, consistency of association, dose-response relationship, temporally correct association, specificity of the association, and coherence with existing information (biological plausibility)) before a causal relationship between an agent and a disease can be inferred. None of the criteria, with the exception of temporality, should be considered as necessary to establish causation. Each of the criteria is important, and causation is established by the weight of the evidence and the degree to which all six criteria are satisfied by the available data. However, the rejection of the association may be made with a high degree of confidence when three of the criteria -- temporality, consistency, and coherence with existing information -- are not met (Rothman, 1988; EPA, 1996). In addition to considering the weight-of-evidence, it is important to understand that studies with larger cohorts and numbers of cancer deaths are inherently more important when considering the weight of the evidence than are studies with smaller cohorts and fewer cancer deaths.

[6]Undue conservatism in hazard identification is manifested when regulatory agencies place an emphasis on data suggesting that chemicals might pose adverse effects, and little weight on data suggesting that chemicals fail to cause adverse effects. Emphasizing study data that show adverse health effects in animals while virtually ignoring studies showing no adverse effects in humans does not represent a balance of scientific information (Nichols and Zeckhauser, 1988). Frequently, EPA places extraordinary confidence on a study that suggests that a chemical may pose a particular hazard, while only modest consideration is given to the study's quality

Applying the weight-of-evidence approach to the PCB cancer epidemiological studies results in the conclusion that there is insufficient evidence to show a causal relationship between PCB exposure and the subsequent development of any form of cancer. As is apparent from the above discussion, 13 of the epidemiological studies, of which four involved major cohorts (including the largest PCB epidemiological study to date, Kimbrough et al. (1999)), have been negative for an association between PCB exposure and cancer. Four of the studies are clearly inconclusive because exposure was primarily to chemicals other than PCBs (Hay and Tarrel, 1999; Yassi et al., 1994), PCB exposure was wholly unquantified in the "exposed" and "unexposed groups" (Rylander and Hagman, 1995); the cohort was extremely small and there was no coherence in the results (Wasserman et al., 1976), or effects originally linked to PCBs were subsequently associated with other chemicals (the Yusho and Yucheng studies). All of the nine "positive" studies also suffer from serious problems that render their findings dubious. Among these problems are the following:

- Three of the studies clearly do not establish dose-response relationships (Loomis et al., 1997, the Sinks et al. 1992, and Yassi et al, 1994).

- Five studies involved cohorts too small to draw credible conclusions (the Bahn et al. studies, Hardell et al., 1996, Rothman et al., 1997, Moysich et al., 1998, and Gustavsson et al. studies).

- Seven studies reported results that do not appear to be biologically plausible and have not been confirmed by other human or animal studies (Hardell et al., 1996, Loomis et al., 1997, Rothman et al., 1997, Sinks et al., 1992, the Bahn et al. studies, Gustavsson et al. studies and Yassi et al., 1994).

- One study's findings have been rejected as spurious because there was clearly no relationship between the small number of cancers reported and exposure duration or latency (Yassi et al., 1994).

- Five of the studies are confounded by the documented fact that the cohorts were exposed to many chemicals other than PCBs and/or by other serious confounding factors (the Bahn et al. studies, the Brown studies, Sinks et al., 1992, Loomis et al. 1997, and Yassi et al., 1994).

From the above summary, it is evident that the Bahn et al. studies, Hardell et al. (1996), Moysich et al. (1998), Rothman et al. (1997) Gustavsson et al. studies, and Yassi et al. (1994) involve cohorts that are too small to provide useful information, are very limited in their findings, and/or have methodological problems that make their limited findings unreliable. Although the Brown studies, Loomis et al. (1997), and Sinks et al. (1992) also suffer from serious limitations, they do involve cohorts that are sufficiently large to provide potentially useful information. Thus these four studies, together with the three negative studies that involved major cohorts (Nicholson et al., 1987; Taylor et al., 1988; Kimbrough et al., 1999) are the best sources of information with which to assess the cancer risk of PCBs.

Evaluation of Weight-of-Evidence Criteria of Six Epidemiology Studies Qualifying for Further Analysis

The seven studies involve five separate groups of workers (since Taylor (1988) and Kimbrough et al.(1999) examined essentially a single group of workers as did Brown and Jones (1981) and Brown. (1987)). The following table summarizes the results of the studies by cohort.

Summary of The Major PCB Mortality Studies

Cohort/Study	Total Cancers	Malignant Melanoma	Liver/Biliary Cancers
Brown Cohort			
Brown and Jones (1981) Mortality study of 163 deaths Among 2,567 U.S. capacitor workers	No	No	No
Brown (1987) Updated mortality of Brown and Jones 1981 analyzing 295 deaths among 2,588 workers	No	No	Yes (limited to females, at one plant)
Nicholson Cohort			
Nicholson et al. (1987) Mortality study of 188 deaths among 769 U.S. capacitor workers with 5 years exposure and 10 years latency since first exposure	No	No	No
Loomis Cohort			
Loomis et al. (1997) Mortality study of 138,905 male electrical power workers with > 6 months employment	No	Yes	No
Sinks Cohort			
Sinks et al. (1992) Mortality study of 192 deaths among 3,588 U.S. capacitor workers	No	Yes (limited to males)	No
Kimbrough Cohort			
Taylor et al. (1988) Mortality study of 510 deaths among 6,292 capacitor workers	No	No	No
Kimbrough et al. (1999) Mortality study of 1,195 deaths among 7,075 capacitor workers	No	No	No

Based on the positive results of Brown (1987), Loomis et al. (1997) and Sinks et al. (1992), the relevant questions to be addressed in a weight-of-evidence assessment are whether PCB exposure is associated with an increased incidence of liver/biliary cancer or malignant melanoma.

As noted previously, a weight-of-evidence assessment using causation criteria requires assessment of the following criteria: strength of association; consistency of association; dose-response relationship; temporally correct association; specificity of the association; and coherence with existing information (biological plausibility). Whether these criteria are satisfied by the major

cohorts is addressed below.

Strength of the Association

Liver/biliary cancer

The strength of the association between liver/biliary cancer and exposure to PCBs is very weak. A statistically significant relation between this type of cancer and PCB exposure was reported by Brown (1987) only for females who worked at one of the two plants. The lack of consistency in responses between the two plants indicates that PCB exposure, which was similar in both plants, was not responsible for the excess cancer cases. Moreover, no association between liver/biliary cancer and PCB exposure was identified in any of the other major cohorts. An additional confounding factor is that the women at the plant in question had ethnic backgrounds from Portugal and Cape Verde, where liver cancer rates for women are 10 to 15 fold greater than those for women in the United States. Although the cause of the elevated liver cancer in Portugal and Cape Verde women is unknown, it clearly suggests that the elevated liver cancer rate reported by Brown (1987) had causes other than exposure to PCBs. It should also be noted that Brown (1987) did not control for alcohol consumption.

Malignant Melanoma

From the five major cohorts studied, malignant melanoma has been reported only by Sinks et al. (1992) and Loomis et al. (1997). Although dermal effects have previously been attributed to PCBs (chloracne and hyperpigmentation), especially for the Yusho and Yucheng incidents, these effects have subsequently been attributed to high-temperature breakdown contaminants (polychlorinated dibenzofurans) that existed in the PCB oils consumed in those incidents. Studies of other heavily PCB-exposed worker populations have not suggested an increased risk for malignant melanoma and, therefore, the strength of any association between PCBs and skin cancer is poor. Furthermore, Loomis et al. (1997) has several limitations in the assessment of exposure, including the quality of information on exposure of electrical workers to sunlight, which is a potentially important confounder and is recognized as such by the authors.

Consistency of the Association

Liver/biliary cancer

With respect to liver/biliary cancer, there is no consistency of association because a statistically significant association was seen in only one of six studies of the five major cohorts.

Malignant Melanoma

With respect to malignant melanoma, there is no consistency of association because a statistically significant association was seen in only two of six studies of the five major cohorts.

Dose-Response Relationship

Liver biliary cancer

According to Brown (1987), the cancers observed in his study failed to demonstrate any dose-response. Specifically, of the five workers categorized with liver cancers, four had worked in jobs with "PCB exposure" for 1.5 years or less and the other had worked in a high PCB exposure job for less than 10 years. In contrast, workers with high PCB exposure for over 30 years had not contracted this type of cancer.

Malignant Melanoma

Based on a cumulative dose estimate, which incorporated information on job station history, limited PCB environmental sampling data, and serologic data, Sinks et al. (1992) were unable to establish a clear relationship between duration of employment and risk for malignant melanoma. Loomis et al. (1997) found that malignant melanoma correlated only with one small group of workers, and that no correlation occurred in groups of more highly exposed workers. Moreover, malignant melanoma did not correlate with total career exposure to PCBs. Thus, the dose-response criterion is not satisfied.

Temporally Correct Association

Liver biliary cancer

According to Brown (1987), the cancers observed in his study failed to demonstrate reasonable expected patterns of latency. Specifically, two of the five liver cancer deaths occurred with less than 10 years of latency and one of the remaining deaths occurred with less than 20 years of latency. The author considered the latency information uninformative.

Malignant Melanoma

Sinks et al. (1992) were unable to establish a clear relationship between duration of employment, latency and risk for malignant melanoma.

Specificity of the Association

"Specificity of the association" refers to the fact that evidence for cause and effect is increased if a cause (here, PCB exposure) is associated with a unique or rare effect. Because liver/biliary cancer and malignant melanoma are not rare, and because these cancers are known to have other causes, the specificity of the association is not helpful in assessing the likelihood that the excess cancers

observed in Brown (1987), Sinks et al. (1992) and Loomis et al. (1997) are related to PCB exposure.

Coherence with Existing Information (Biological Plausibility)

Liver/biliary cancer

Although a statistically significant increase in liver/biliary cancer and PCB exposure was reported by Brown (1987), two of the cancers are actually metastasis to the liver from other (non-liver) sites of origin, and the remaining three are described as bile duct or biliary system carcinomas. In animal studies, the majority of cancers are primary liver cancers originating from liver cells (i.e., hepatocytes), not bile ducts. Thus, the findings of Brown (1987) are not concordant with existing information.

Malignant Melanoma

Although in some studies animals treated orally or dermally with PCBs have shown dermal abnormalities, no studies have reported cancerous lesions. Thus, the findings of Sinks et al. (1992) and Loomis et al. (1997) are not coherent with existing information.

Based on the above analysis, it is clear that the weight-of-evidence does not support the contention that PCBs are carcinogenic in humans. This conclusion is shared by many other scientists. Chase, Doull, Friess, Rodricks, and Safe (1989), concluded that:

> There is insufficient evidence to show a causal relationship between PCB exposure and the subsequent development of any form of cancer. In light of the long-term and widespread usage of PCBs in the workplace and, in some cases, the extensive exposures of workers, it is likely that evidence of carcinogenicity in humans would have been observed in the various epidemiological studies discussed above if PCBs were in fact potent carcinogens.

Similarly, Kimbrough (1988) concluded that:

> Thus far, no conclusive adverse effects have been demonstrated in people who carry body burdens of PCBs from environmental exposure to trace amounts of PCBs. . . . Even workers with exposures two orders of magnitude greater than environmental exposures show no convincing health effects. . . . Thus, despite positive laboratory animal data and except for chloracne, exposure to PCBs has led to no convincing, clinically demonstrable, chronic health effects in humans.

In her 1995 update, Dr. Kimbrough reached a similar conclusion (Kimbrough 1995).

A recent review of the occupational studies by the American Council on Health and Science also concluded that none of the studies provides evidence that PCB exposure increases cancer risk in humans. (Danse et al., 1997). A review of studies seeking to determine if there was a relationship between environmental exposures to PCBs and any human health effects, including cancer, found

that "none of the 33 studies where exposure had occurred in the natural environment provided positive or suggestive evidence of an association with adverse effect" Swanson et al., 1995). Similarly, Adami et al. (1995) reviewed the epidemiological literature that has investigated the possible link between organochlorine compounds (including PCBs) and estrogen-related cancers in women (i.e., breast and endometrial). The authors conclude that "[i]n reality, . . . it is questionable whether the background levels of organochlorines in the general population will be high enough to elicit any [cancer] effects. In fact, [our analysis] would indicate that this is unlikely."

Finally, ATSDR has come to the same conclusion. According to ATSDR's most recent draft of its PCB Toxicological Profile, "[t]he weight of evidence does not support a causal association for PCBs and human cancer at this time," see ATSDR (1998) at 227.

Thus, a fair and careful review of the existing PCB epidemiological studies leads to the conclusion that there is no credible evidence that PCBs cause cancer in humans, even at exposures that are orders of magnitude greater than environmental exposures. This information can, and should, be used in assessing the cancer risks of PCBs.

EPA should use the Negative Epidemiology Findings to Characterize the Upper Bound of PCB Cancer Potency.

As described in GE's comments on the HHRA Scope of Work (GE, 1998), negative epidemiology studies can be used to characterize the plausible upper bound of cancer potency in humans for animal carcinogens. EPA has long recognized that not all animal carcinogens are human carcinogens and that the carcinogenic potency of a compound in humans may differ from its potency in animals.

Epidemiology studies of workers exposed to animal carcinogens allow a direct determination of the potential for cancer risks to humans (EPA, 1996, 1986). According to EPA (1986):

> If available, estimates based on adequate human epidemiologic data are preferred over estimates based on animal data. If adequate exposure data exist in a well-designed and well-conducted negative epidemiology study, it may be possible to obtain an upperbound estimate of risk form that study.

EPA in its response to GE's comments on the HHRA Scope of Work did not dispute this point (EPA, 1999).

GE recognizes that major concerns with the use of epidemiology data in carcinogenic risk assessment are the lack of statistical power of some epidemiology studies and difficulties in estimation of the doses received by the cohort. If an epidemiology study has insufficient power due to too small a cohort, or other limitations, then its findings may be too uncertain to justify replacing an animal-based estimate of potency. In addition, if the exposures were too low, then the predictions of potency from epidemiology studies can be overestimated. As a result, many negative epidemiology studies will not warrant modifying an estimate of carcinogenic potency

derived from animal studies.

This is not true in the case with PCBs. There are a number of studies with large cohorts, long latency periods, and well characterized exposures. In addition, the persistent nature of PCBs allows the use of biomarkers (concentrations of PCB mixtures and individual congeners in blood and adipose tissues) to characterize worker's total aggregate doses from all routes of exposure. As a result, it is possible to define worker's exposure based on measurements of PCBs in air and on surfaces and based on body burdens. In recognition of this fact, TERRA (1993) has developed an estimate of carcinogenic potency based on the findings of two of the epidemiology studies (Brown, 1987; Taylor, 1988) using estimates of exposure based on both air monitoring data and measurements of body burdens.

The TERRA study reported estimates of PCBs carcinogenic potency that ranged from 7.7×10^{-4} to 1.9×10^{-2} $(mg/kg\text{-}day)^{-1}$. These estimates are from 100 to 3,000 fold lower than the estimate of PCB cancer potency from rodent studies. These findings demonstrate that both the Brown and the Taylor studies have sufficient statistical power to realistically demonstrate that the potency of PCBs is lower in humans than in rodents. In addition, the level of exposure data for the workers are sufficient to develop meaningful estimates of dose. Doses were estimated using two independent sources of exposure, air data and measurements of body burden. Both sources resulted in similar estimates of risk.

The size of the differences between the cancer potency estimates based on the rodent studies and the potency determined from the human studies are too great to be explained by differences in the composition of the PCBs or any uncertainty in the dose estimates. The potency of all Aroclor mixtures containing higher chlorinated PCBs has been found to fall within a range of potencies of about a factor of four (EPA, 1996). Thus, differences in composition cannot explain the difference in potency. As discussed below, the estimates of dose in the TERRA report are more likely to be underestimates of exposure than overestimates. Therefore, the findings of the TERRA report provide clear evidence that the use of cancer potency estimated from rodent studies will overestimate risks to humans.

As noted previously, the recent findings of Kimbrough (1999) have extended and confirmed the findings of Brown (1987) and Taylor (1988). GE believes that an estimate of potency derived from the negative findings of the Kimbrough study provide the best basis for establishing a conservative estimate of carcinogenic risk. Therefore, GE strongly encourages the Agency to use these recent findings to establish potencies for PCB exposures.

EPA has over the years expressed several concerns over using the epidemiological studies to assess the cancer risks of PCBs. EPA's stated concerns,[7] and GE's responses, are set forth below:

[7] EPA's stated concerns are taken from the following sources: Reassessment: EPA Response to Comments Database in the GLI rulemakings (Code P2654 185, Subject HH CRIT PCBS, Date 9/10/93), and Water Quality Guidance for the Great Lakes System – Supplementary Information Document.

Concern: Some epidemiological studies reported air concentrations, but because skin contact is a major route of exposure, air concentrations would be a poor measure of exposure.

Response: GE agrees that air concentrations would not fully characterize exposure. Rather, air concentrations would significantly underestimate exposure since dermal exposure and incidental ingestion would also form significant exposure routes. Accordingly, any cancer potency calculated from the epidemiological studies would overestimate the cancer risk of PCBs

Concern: Some studies reported blood levels, but for relatively few workers at the end of exposure.

Response: GE agrees that some of the studies reporting blood PCB concentrations had access only to data taken near the end of the exposure period. However, this fact would tend to underestimate exposure, resulting in derivation of cancer potencies that overestimate cancer risk.

EPA is incorrect in asserting that there is little information on blood PCBs levels in the epidemiological studies. For GE capacitor workers, a group on which there is extensive epidemiological data from multiple investigators, blood PCB levels were determined in early 1976 (after 30 years of PCB usage at the plant, and one year before discontinuance of such usage). One hundred and ninety four individuals were selected as having direct or peripheral PCB exposure by the plant physician and nurse. Another 326 individuals, having a wide variety of intra-plant exposures, volunteered for testing. Sampling was performed by doctors at the Mount Sinai School of Medicine. Since 33 individuals participated in both studies, a total of 487 people were examined. Most members of both groups were reexamined three and one-half years later, after the discontinuance of PCB usage, and the more heavily exposed group was examined again in 1983 and 1988, thus permitting definition of clearance rates of all the major PCB congeners. The findings of both groups of investigators have been extensively reported in the peer-reviewed literature. GE does not believe that 487 individuals should be described as "relatively few," nor that measurements made during the period of exposure on individuals with an average service time of 17 years should be considered irrelevant to the question of chronic uptake rates.

Concern: Dermal exposure studies, with regard to PCB exposures, are not considered adequate due to uncertainty in quantifying the dermal absorption of PCBs.

Response: GE shares EPA's concern regarding the difficulties of estimating dermal exposure. However, the use of conservative assumptions, can be used to overcome this problem. GE also notes that recent studies have quantified PCB exposure through the skin (Wester et al., 1987, 1990, 1993).

Concern: Reconstruction of past exposure is problematic because different mixtures had been in use over the years, the distribution of exposure and absorption by route and congener is unknown, and congener persistence in the body varies.

Response: GE agrees that reconstructions of past exposure, including assessment of congener persistence in the body, are difficult. However, the use of conservative assumptions, can be used

to overcome this problem.

GE also agrees that the distribution of exposure and absorption by route and congener are difficult to estimate. However, it is not necessary to quantitatively determine the presence and persistence of each congener in the body in order to make use of epidemiologic data. In the case of the largest epidemiological studies performed to date (Taylor, 1988 and Kimbrough et al., 1999), the Aroclors to which the cohort was exposed are well documented (Lawton et al. 1981). Exposure was primarily to Aroclors 1242 and 1254, with a small amount of exposure to Aroclor 1016 in later years. If one makes the conservative assumption that all exposure was to Aroclor 1242, which is less carcinogenic to rodents than Aroclor 1254, one can derive a cancer potency from Taylor (1988) that must overestimate cancer risk. This conservative cancer potency for Aroclor 1242 can then be used to derive a cancer potency for other Aroclors, or total PCBs, based on the relative cancer risk of Aroclor 1242 to the risk of other Aroclors as determined by rodent studies. Thus, by a set of conservative assumptions, the concern expressed by EPA can be entirely overcome.

Concern: The human carcinogenicity database on PCBs is considered inadequate by EPA. Although there are many studies with regard to occupational exposures, the data are judged inadequate due to confounding exposures. The confounding factors noted in IRIS are population differences in alcohol consumption, dietary habits, ethnic composition, contamination of PCBs by dibenzofurans, and exposure of workers to other carcinogens.

Response: In response to EPA's concern about confounding factors, GE notes that confounding factors must be guarded against in all epidemiological studies. However, EPA appears to ignore the fact that different types of confounding factors can bias different studies in different ways. For example, if a study of PCB-exposed workers who were also smokers concluded that the cohort had a higher than normal incidence of cancer, the fact that the workers were smokers would confound the conclusion that PCBs were the cause of the elevated cancer rate. On the other hand, if the cohort was found not to have an elevated incidence of cancer, the fact that the cohort was composed of smokers would not invalidate the conclusion that exposure to PCBs was not observed to be related to a higher than normal cancer rate. All of the confounding factors referenced by EPA would tend to result in an increased cancer incidence, thereby increasing the cancer potency factor derived from an epidemiological study.

Recently, in responding to the HHRA Scope of Work (GE, 1998) EPA raised two concerns regarding use of the human epidemiological data to assess the cancer risk of PCBs. EPA's concerns, and GE's responses, are as follows:

Concern: Exposure in the employee epidemiological studies was primarily through the inhalation route. Thus, the employees were primarily exposed to the more volatile congeners of lower chlorine content. In the case of the Hudson River, the population is exposed through fish, sediment, and soil ingestion, providing exposure to congeners that are more persistent, less volatile, and of higher chlorine content. The higher chlorine content congeners have a higher potency than the lower chlorine congeners.

Response: The manufacturing operations at the GE capacitor plant used PCBs having both higher (i.e., Aroclor 1254) and lower (i.e., Aroclors 1242 and 1016) chlorine contents, and emitted such PCBs as both vapor and aerosols. This resulted in extensive contamination of both air and surfaces throughout the plant. The median air contamination level in both plants in January 1975, during the period of full PCB use, was about 700 ug/m^3 (GE Report, Sept., 1981) In mid-1978, a year after discontinuance of PCB use, the median air contamination levels were still about 100 ug/m^3 at Hudson Falls and about 42 ug/m^3 at Ft. Edward, indicating the significance of the contamination of the in-plant surfaces. Employee exposure was via both inhalation of PCB vapors, which would be somewhat depleted in higher congeners, and by dermal contact with contaminated surfaces, which would be correspondingly enriched in higher PCBs.

The net results of such exposures for the great majority of employees who did not have jobs involving direct PCB contact may be calculated from the 1976 plasma PCB levels reported by Fischbein et al. (1979). The plasma PCBs in the 158 workers then employed in jobs with "low" relative exposure intensity included 41 ± 75 ppb of "higher" PCB homologs. These higher homologs consisted almost entirely of congeners 118, 138/163, and 153 with small amounts of congeners 105 and 180.

The clearance rates for all of these congeners in the GE capacitor worker population have been determined (Brown et al., 1989, 1994). These rates (in yr^{-1}) are 0.12 for PCB 118, 0.089 for PCB 138/163, and 0.056 for PCB 153. For the minor congeners 105 and 180 the rates are 0.18 and 0.01, respectively. For the major congeners, the clearance rates are close enough to permit use of the mean clearance rate (0.09 yr^{-1}) to calculate the mean uptake rate.

The reported mean plasma concentration of 41 ppb for the "higher" PCB homologs in plasma corresponds to a mean level of 8.2 ppm in plasma neutral lipid which (may be assumed to be equal to that in all body lipid). Since the generally overweight workers in the plant were found to be averaging 22 kg of body fat (Lawton et al., 1985), this corresponds to a mean body burden of 180 mg higher PCBs. Multiplying by the 0.09 yr^{-1} clearance rate indicates a mean uptake rate of 16.2 mg/yr or 44 ug/day of retained higher congeners. Since these retained higher congeners constitute only about 20% of Aroclor 1254, this corresponds to a mean intake rate of 220 ug/day of Aroclor 1254 equivalents for the actively employed but low exposure group.

The retirees examined by Fischbein et al. (1979) had plasma PCB levels about twice as high (84 ± 120 ppb) despite opportunities for clearance. If we assume an average of five years retirement, their original mean higher PCB level would have been 132 ppb, corresponding to a mean intake of about 700 ug/day of Aroclor 1254 equivalents. This higher number presumably reflects the predominant usage of Aroclor 1254 during the first decade of plant operation. Thus, we may estimate general employee Aroclor 1254 uptake rates as about 700 ug/day during the first decade of capacitor manufacture at the plant and 220 ug/day for the second two decades, or very roughly 380 ug/day for the entire period. For the directly exposed group, of course, the intake rates would be considerably greater. Thus, although the majority of the PCB exposure in the plant was to the lower congeners, very substantial exposure to higher congeners also occurred.

Concern: TERRA (1993) used a pharmacokinetic approach to estimate occupational PCB

exposure based on half-life estimates for Aroclor mixtures. The Reassessment discussed the fallacy of ascribing single half-lives to mixtures as variable as PCBs. Further, these half-life estimates tend to be underestimates, thus overstating occupational exposure to PCBs.

Response: GE is well aware of the fallacy of using single half-lives to characterize PCB clearance and has published both the data and equations needed for scientifically correct calculations (Brown, 1994). However, if a mixture of half-lives had been used the results would have been little different. The predominant PCB congener in the group of actively employed GE capacitor workers in 1976 was PCB 28 for which the clearance half-time is 1.4 yr. TERRA's calculations assumed a mean value of 1.8 yr., which would be very close to the initial clearance rate of all PCBs from the Aroclor 1016-exposed group.

Where TERRA made a more serious error was in assuming that the retained congeners represented the entirety of the exposure, whereas actually the retained lower congeners (i.e., PCBs 28, 74 and 99) instead constitute only 9% of either Aroclor 1242 or 1016. Accordingly, using Fischbein's et al. (1978) data for the lower PCB levels in the people with low intensity job exposure one may repeat the previous calculation, assuming a mean clearance rate of 0.35 yr^{-1} ($t_{½}$, 2 yr.) for the lower congeners. Thus, 73 ± 81 ppb lower PCBs in plasma becomes equivalent to a mean of 14.6 ppm in plasma lipids, or 320 mg mean body burden, corresponding to 112 mg/yr or 308 ug/day mean uptake of retained lower congeners, or 3.4 mg/day uptake of Aroclors 1242 or 1016. For the people with "medium" or "high" relative exposure the mean lower Aroclor intake rates for the lower chlorinated Aroclors would have been about 8.0 and 12.5 mg/day, respectively.

With the additions of the higher Aroclor intake rates previously calculated, the mean total PCB intakes for the people in jobs with "low" and "high" exposures become 3.6 and 12.9 mg/day, respectively. Thus, TERRA's calculation actually underestimated rather than overestimated the PCB intake rate.

4. A Weight-of-Evidence Assessment of the Non-carcinogenic Effects of PCBs

As in the case of EPA's assessment of the cancer risks of PCBs, EPA has to date assessed the non-cancer risks of PCBs solely on the basis of animal studies. Specifically, EPA has derived RfDs for Aroclors 1254 and 1016 based on studies on rhesus monkeys (IRIS, 1999). NOAELs or LOAELs from these studies are extrapolated to yield the RfDs using up to four uncertainty factors. The HHRA uses these values to assess non-cancer risks from PCBs in the Hudson River. By relying solely on animal studies to derive RfDs for PCBs, EPA has ignored the important information that is available from a weight-of-evidence approach to assessment of PCB non-cancer risks. Like the cancer epidemiology studies, the non-cancer studies in humans provide no clear evidence of a causal link between PCBs and non-cancer effects. Furthermore, the non-cancer human health studies can be used to: (a) inform the choice of laboratory bioassay to use in deriving an RfD; and (b) assess the relative sensitivity of humans and laboratory animals to the non-cancer effects of PCBs, thus supporting the selection of scientifically valid uncertainty factors and uncertainty factor distributions for use in deriving human RfDs and distribution of bioassay NOAELs and LOAELs.

Section 4 a. of this Attachment discusses what can be learned from the human non-cancer PCB studies. Section 4 b of Attachment critiques EPA's derivation of the RfD for Aroclor 1254[8] in light of the information that can be gleaned from the human non-cancer studies, and responds to EPA's comments on similar arguments presented by GE in its comments on the HHRA Scope of Work (GE, 1998).

a. Epidemiological Evidence Regarding PCB Non-cancer Human Health Effects

The HHRA provides surprisingly little information regarding studies of the non-cancer effects of PCBs on humans. The HHRA's only references to human non-cancer effects are: (i) a short discussion of chloracne and other mild clinical conditions based on ATSDR's Toxicological Profile for PCBs; (ii) references to findings reported in Pantandin et al. (1999) and Lanting et al. (1998) of possible associations between perinatal exposure to PCBs and dioxins and adverse effects on growth, immunologic parameters, and neurodevelopmental and behavioral effects; and (iii) a short discussion of PCB's potential role as "endocrine disrupters." HHRA, Appendix C at C-4. Overall, the HHRA might well be read to suggest that EPA does not believe that there is much evidence of PCB non-cancer effects in humans. However, in other fora, EPA has expressed serious concern about such effects.

Organizations and scientists who have reviewed the extensive literature on PCB non-cancer effects have come to the conclusion that there is little, if any, convincing evidence that PCBs have adverse health effects at either current environmental exposure levels or historical workplace exposure levels. ATSDR, the author of the most recent comprehensive review of the literature on PCB toxicology, has stated following specific conclusions regarding the various potential health effects of PCBs:

> Death "No studies were located regarding death in humans after exposure to PCBs by any route. . . . The acute lethality data do not suggest that PCBs would be acutely toxic in humans." ATSDR (1997) at 154, 155; ATSDR (1998) at 205, 206.
>
> Respiratory effects "[Respiratory] effects cannot be definitely attributed to PCBs due to study limitations such as lack of control data, co-exposure to other chemicals, insufficient corroboration, and lack of confirmation in follow-up evaluations. . . . Overall, there is inconclusive evidence that the respiratory tract is a target of PCBs in humans." ATSDR (1997) at 155; ATSDR (1998) at 206.
>
> Cardiovascular effects "Evidence of increased blood pressure or an association between serum levels of PCBs and hypertension in populations with occupational or environmental exposure to PCBs is negative or inconclusive. . . . The existing data are insufficient to infer possible cardiovascular toxicity of PCBs in humans." ATSDR (1997) at 156; ATSDR (1998) at 207.

[8] This document does not discuss the Agency's RfD for Aroclor 1016 because the HHRA determined that the exposure pathways evaluated using the RfD for Aroclor 1016 did not pose an unacceptable risk.

Hepatotoxicity "Considering the generally small increases, inconsistencies, and other issues associated with the serum enzyme and lipid data, and the uncorroborated report of hepatomegaly, there is weak evidence that occupational inhalation exposure to PCBs causes hepatotoxicity in humans." ATSDR (1997) at 25; ATSDR (1998) at 30

Gastrointestinal effects "Nonspecific symptoms such as a loss of appetite, nausea, epigastric distress and pain, and intolerance to fatty foods have been experienced by workers exposed to PCBs . . . . No apparent gastrointestinal effects were reported in environmentally exposed populations . . . . The human data for gastrointestinal effects of PCBs are inconclusive and the relevance of the animal data seems questionable since most animal studies used doses much higher than current background levels for the general population and presumably also higher than those experienced by workers exposed to PCBs for months or years." ATSDR (1997) at 156; ATSDR (1998) at 207.

Hematological effects "Conclusive hematological alterations have not been observed in workers who were chronically exposed to PCBs . . . or in individuals environmentally exposed . . . . " ATSDR (1997) at 156 ATSDR; (1998) at 208.

Musculoskeletal effects "The only information regarding musculoskeletal effects in humans exposed to PCBs is the report of joint pain in 11% of the workers exposed to a variety of Aroclors at concentrations of 0.007-11 mg/m^3 . . . . Information on the cause of this pain or whether it is related to duration of exposure was not provided in the study. . . . Based on the existing data, it is not possible to infer that similar skeletal effects could occur in exposed humans." ATSDR (1997) at 157; ATSDR (1998) at 215.

Renal effects "There is no evidence of an association between PCB exposure and renal toxicity or kidney disease in occupationally . . . or environmentally . . . exposed subjects. . . . The relevance of the renal effects observed in animals treated with high doses of PCBs to human health is unclear since the exposure levels were much higher than current background levels for the general population and higher than those to which workers may have been exposed." ATSDR (1997) at 154; ATSDR (1998) at 215.

Dermal effects "Dermal lesions including skin irritation, chloracne, and pigmentation of nails and skin have been observed in humans following occupational exposure to PCBs . . . . Overall, the existing evidence suggests that it is unlikely that adverse dermal effects will appear in the general population due to background exposure to PCBs. Exposure to PCBs through contaminated fish consumption . . . , contaminated sludge use . . . , or residence near a PCB waste site . . . have not shown any significant dermal effect or chronic skin disease." ATSDR (1997) at 164, 165; ATSDR (1998) at 216, 217.

Body weight effects "No information was located regarding body weight effects in humans after exposure to PCBs. Body weight loss and/or reduced body weight gain are commonly seen effects of PCB exposure in animals. . . . The relevance of the animal data to body weight effects in humans is unknown." ATSDR (1997) at 166; ATSDR (1998) at 217.

Immunotoxic effects "Although the limited data on humans are inconclusive, the available evidence does not suggest that occupational exposures to PCBs were immunotoxic, and no association has been found between PCB exposure and excess mortality from infectious diseases." ATSDR (1997) at 169; ATSDR (1998) at 220.

Developmental effects "Results from some studies in the United States in which exposure to PCBs was assumed to have been by consumption of contaminated fish have raised the possibility that exposure to PCBs causes developmental effects in humans. . . . The overall evidence suggesting that PCBs may represent a developmental hazard for human health is inconclusive." ATSDR (1997) at 172, 174; ATSDR (1998) at 223, 225.

Neurological effects. "Limited information exists regarding neurological effects in adult humans following exposure to PCBs. . . . The toxicological significance of the reported neurological effects in rats is unknown, in particular since no apparent clinical signs of neurological damage were observed in the chronic study. The information is insufficient to assess the potential for neurological effects in adult humans exposed to PCBs." ATSDR (1997) at 169, 170; ATSDR (1998) at 220, 221.

Genotoxic effects "The generally negative results of *in vitro* and *in vivo* genotoxicity studies suggest that the PCB mixtures tested do not pose a genotoxic threat to humans." ATSDR (1997) at 177; ATSDR (1998) at 227.

Reproductive effects "Conclusive information on reproductive effects of PCBs in humans was not located. . . ." ATSDR (1997) at 170; ATSDR (1998) at 221, 223.

Ocular effects "Eye irritation, burning sensation, conjunctivitis, and eye discharge have been reported in occupationally exposed individuals. . . . Overall, the existing evidence suggests a potential for adverse ocular effects in humans repeatedly exposed to low levels of PCBs." ATSDR (1997) at 166; ATSDR (1998) at 217.

Thus, ATSDR has concluded that there is no conclusive evidence that PCBs are responsible for any human non-cancer effect, other than clinically minor ocular effects.[9] It is, however, questionable whether ocular effects have resulted from workplace exposure to PCBs and highly unlikely that environmental exposure to PCBs could lead to such effects. ATSDR (1997, 1998) does not cite, nor is GE aware of, any study indicating that ocular effects have ever been observed in humans exposed to PCBs other than workers exposed to PCBs at elevated levels. Thus, GE believes that the only sound conclusion that can be drawn regarding potential ocular effects of PCBs are that: (1) in the past, high occupational exposures to PCBs may have caused eye irritation and other temporary adverse effects on the eyes; but (2) at current exposure levels, there is no evidence that PCBs are causing any such effects in humans.

The conclusion reached by ATSDR (1997, 1998) regarding the non-cancer human health effects

[9] ATSDR has also concluded that "[t]he weight of evidence does not support a causal association for PCBs and human cancer at this time." ATSDR (1998) at 227.

of PCBs is shared by many eminent scientists. For example, Kimbrough (1988) concluded:

> Thus far, no conclusive adverse effects have been demonstrated in people who carry body burdens of PCBs from environmental exposure to trace amounts of PCBs. . . . Even workers with exposures two orders of magnitude greater than environmental exposures show no convincing health effects. . . . Thus, despite positive laboratory animal data and except for chloracne, exposure to PCBs has led to no convincing, clinically demonstrable, chronic health effects in humans.

In her 1995 update, Dr. Kimbrough reached similar conclusions (Kimbrough 1995).

Moreover, a recent review of the occupational studies by the American Council on Health and Science also concluded that there is little evidence that PCBs cause adverse human health effects Danse et al. (1997):

> No conclusive evidence exists that background levels in the general population, or even the very high levels that occurred among some occupational groups, resulted in acute or carcinogenic effect. In humans, the only adverse health effects that are strongly associated with PCB exposures are skin and eye problems (chloracne, changes in skin pigmentation, and chronic skin and eye irritation). These effects have only been reported following exposures to unusually high levels of PCBs, along with other chemicals. None of these effects have been observed in populations exposed through the consumption of fish. . . .
>
> Recent reports suggesting a correlation between exposure to PCBs *in utero* (from maternal consumption of contaminated fish) and impaired intellectual development are not supported by other studies on prenatal development and are limited by deficiencies in exposure assessment and control of confounding variables.

Similarly, a review of studies seeking to determine if there was a relationship between environmental exposures to PCBs and any human health effects found that "none of the 33 studies where exposure had occurred in the natural environment provided positive or suggestive evidence of an association with adverse effect" (Swanson et al., 1995). Finally, James et al. (1993) conducted a thorough literature review of health studies of the population most highly exposed to PCBs—electrical capacitor and transformer workers, which centered on potential adverse effects on the liver, lungs, skin, cardiovascular system, nervous system, endocrine systems, the blood/immune system, and the gastrointestinal and urinary tracts. The authors came to the following conclusion:

> Studies of PCB-exposed populations collectively suggest that the only adverse health effects attributable to PCBs in humans are dermal: chloracne, hyperpigmentation, and sequelae of chronic and ocular irritation. These conditions occurred only in workers with relatively high dermal and/or inhalation exposures.

It is therefore apparent that respected groups and individual scientists have concluded that PCBs have been shown to have only limited human health effects, and that these effects are associated

only with historical occupational exposures. Recently, however, EPA and others have cited studies of three human cohorts as supporting the proposition that PCBs may have adverse developmental and neurological effects in children at environmental dose levels. The cohorts are commonly referred to as: (1) the "Michigan" (or "Jacobson") cohort; (2) the "North Carolina" (or "Rogan") cohort; and (3) the "Netherlands" (or "Dutch") cohort.

A large number of respected scientists have reviewed the studies of these cohorts and have generally concluded that a number of methodological flaws render these studies inconclusive, at best, regarding the health effects of PCBs on humans at environmental dose levels. (Paneth, 1991; WHO, 1993; Expert Report on Polychlorinated Biphenyls, 1994; Schantz, 1996; Buck, 1996; Seegal, 1996; Kimbrough and Doemland,[10] 1996; Danse et al., 1997; Middaugh and Egeland, 1997; TERRA, Karch and IEHR,[11] 1999). In particular, there are very serious limitations associated with the studies of the Michigan cohort. These include serious problems with the definition of the "high" and "low" exposure groups within the cohort, inadvertent selection bias, potentially selective attrition rate, analytical problems in quantifying and interpreting PCB concentrations in fish and blood samples from the cohort, failure to quantify other potential neurotoxicants (including lead, mercury and chlorinated pesticides), failure to control for important confounding factors, lack of internal consistency, methodological problems related to measuring developmental effects in children (including use of tests not designed for that purpose), unavailability of critical statistical data for peer review, implausibility of results, and serious discrepancies between the findings of the Michigan studies and the North Carolina studies.

The Expert Report on Polychlorinated Biphenyls (1994) provides a concise summary of the views of many scientists on just a few of the problems and limitations of the studies of the Michigan cohort. The following are some representative quotations from that report:

> The authors suggested that PCBs in the fish were the cause of the reported effects although no correlation was observed between fish consumption and concentrations of PCBs in umbilical cord blood serum. . . . The concentrations of PCBs in cord serum at birth, as reported by Fein et al. (1984), were correlated with decreased cognitive performance in the children at 4 years of age; however, there was no correlation between concentrations of PCBs in cord serum and maternal fish consumption . . . (the proposed source of PCBs).
>
> [T]he number of inconsistencies in the reported analyses for PCBs in maternal and cord blood sera, and in breast milk, affect the confidence of the overall evaluation of possible dose-response relationships. It appears that analyses for PCBs were not conducted on serum samples with lipid concentrations less than 200 mg/dl. This decision could bias the interpretation of subsequent correlations with adverse effects in an undetermined manner since the PCBs in blood are associated with blood lipids. . .
>
> The deficiency in the criteria for a dose-response relationship, a basic requirement for establishing causality (Hill 1965; Fox 1991), and the lack of substantial differences

[10] Copies of these reports are attached.

between the concentrations of PCBs in the study and in the general population, indicate that the effects reported on human development in the populations studied were not causally related to exposures to PCBs.

[A] factor affecting the strength of the proposed association is that the women from the elevated fish consumption group also reported significantly greater consumptions of alcohol, caffeine, tobacco, and cold remedy prescription use during pregnancy than those consuming less fish. ... All these confounding factors seriously detract from the biological plausibility of an association between the amount of exposure to PCBs, as indicated by the concentrations of PCBs in blood, and effects on infant development.

[A] factor affecting the strength of the proposed association is the lack of plausibility and consistency of association that are required (Hill, 1965; Fox, 1991) to establish a causal relationship. Greater umbilical cord serum concentrations of PCBs were marginally associated with cognitive performance parameters (e.g., poorer verbal and memory scores on the McCarthy Scales performance tests and lower scores in the verbal and numerical memory subtests) . . . in the same children 4 years later (Jacobson et al., 1990a). Scores on such tests are affected by socioeconomic status of the subjects and the age at which the tests were conducted. Scores for other components of the McCarthy Scales performance tests (perceptual performance, quantitative, motor, and general cognitive index) were unrelated to *in utero* exposure to PCBs. Also, no correlation was observed between fish consumption and concentrations of PCBs in umbilical cord serum. As a result, no correlation can be made between contaminated fish and scores on the McCarthy Scales performance tests. These factors seriously weaken the plausibility of associations between the effects reported by Fein et al. (1984) and exposures to PCBs.

The plausibility of the results of the Jacobson et al. (1990a) study is further eroded by the report that certain marginal deficits observed in some clusters of the McCarthy tests were associated with greater concentrations of PCBs in the serum of the mother, but not with greater exposures to PCBs through lactation.

[I]t was not apparent that the scores obtained in the tests of any of the children were outside the ranges of normal since no such ranges were given.

[A] total of approximately 38 behavioral and neurological tests were conducted on the children, even though the results of only 2 tests were reported to be affected. Some of the association based on chance alone would be expected from this large number of tests. In addition, the assessment of the test results was based on a "clustering" approach, which is not a standard procedure in evaluating neurological test results from children.

Based on the above analysis, and considering the marginal significance of the observations, the information reported by Fein et al. (1984), Rogan et al. (1986a,b) and Jacobson et al. (1990a,b) do not meet the criteria for the establishment of a causal association for an effect of PCBs on growth and behavior in human populations.

Due to the numerous problems with the studies of the Michigan cohort, it is not surprising that studies of the North Carolina cohort have been unable to duplicate the findings of the Michigan studies. Schantz (1996) provided the following comparison of the studies of the two cohorts:

> Despite the early deficits in psychomotor performance on the Bayley Scales, there was no indication of a relationship between PCB exposure and scores on the McCarthy Motor Scale. This scale is not an exact analogue of the Bayley Psychomotor Scale, but it is similar in that it uses common, age-appropriate tasks to assess motor function. These findings suggest that the initial delay in psychomotor development associated with transplacental PCB exposure does not persist beyond 2 years of age. There was no indication of a relationship between PCB exposure and scores on the McCarthy Memory Scale. Thus, these authors were not able to confirm the relationship between prenatal PCB exposure and short-term memory deficits reported by Jacobson, Jacobson, and colleagues.
>
> In summary, Rogan and Gladen did not confirm the Jacobson's findings of decreased birth size and weight, or impaired short-term memory function, but did find evidence of a delay in psychomotor development in their most highly PCB-exposed children.

Decreased birth weight and head circumference and deficits in short-term memory functioning were observed in the Michigan cohort, but not in the North Carolina cohort. It has been suggested that these differences may be due to differences in exposure. Unfortunately, different analytical techniques were employed in the two studies, making it impossible to directly compare exposure levels. The reported exposure levels for the North Carolina cohort were nearly double those for the Michigan cohort, but there is good evidence that the analytical technique used in the North Carolina study overestimated PCB exposure by a factor of about two. If the North Carolina data are corrected for this overestimation, the actual exposure levels are very similar to the exposure levels in the Michigan study, not lower as has been previously suggested. Thus, it is unlikely that the differences in the level of PCB exposure can account for the differences in outcome. A related possibility is that the effects observed in the two Michigan cohorts were associated with other, unmeasured contaminants that were present in fish and covaried with PCBs. That is, perhaps PCBs were merely a marker for some other highly lipophilic compound such as methylmercury or chlorinated dibenzodioxins. This seems plausible when one considers the effect on birth weight. The effect seen in the Michigan cohort was of a size (160g) that should have been easily detectable in the larger North Carolina cohort, yet there was not even a suggestion of an effect on birth weight in that cohort. Finally, it is possible that inadequate control of confounding variables such as maternal prepregnancy weight, alcohol consumption, and smoking in the Michigan study resulted in spurious findings.

Studies of the Dutch cohort also do not confirm the results of the studies of the Michigan cohort. The Dutch studies are following the neurodevelopment of 418 children born in either semi-urban Groningen or industrialized Rotterdam in the Netherlands. Prenatal exposure was defined as the summed natural log concentrations of four PCB congeners in maternal plasma that together represented only 50% of the total plasma PCB. These children were either breast-fed or fed commercial formula. Lactational PCB and dioxin exposure was assessed from breast milk concentrations and duration of breast-feeding. Current PCB body burdens in the children were

estimated from plasma concentrations of the same four congeners taken when they were assessed for neurodevelopmental progress (3, 7, 18, and 42 months). Although small but transient neurodevelopmental effects were associated by the authors with estimated PCB and dioxin exposure at earlier time points, more recent reports have concluded that neither prenatal nor postnatal exposure to PCBs and dioxins was found to be correlated with neurological condition at 42 months (Lanting et al., 1998).

In the latest report on the same cohort, Patandin et al. (1999) reported that there were no differences between the two cities in PCB or dioxin concentrations in before-birth maternal plasma, 42 month-old plasma, or breast milk PCB. Thus, the initial premise that the two cities presented different exposure scenarios was disproved. The authors also showed that there were no associations at 42 months between either lactational PCB and dioxin exposure or current PCB body burden and the overall cognitive abilities of these children as measured by the Kaufman Assessment Battery for Children. In all, 395 children from the cohort were tested and all scores were within or above the mean population score of 100 ± 15.

Although slightly lower overall test score means (110 *vs.* 114 points) were associated with higher maternal plasma PCB concentrations for the entire cohort, these differences disappeared for the breast-fed group when separately analyzed. Furthermore, no negative effects of prenatal TEQ or PCB exposure were found in the breast-fed group. Lactational exposure to nondioxin-like PCBs, dioxin-like PCB-TEQs, and dioxin-TEQs was also not related to performance.

The studies of the Dutch cohort are subject to several limitations. According to the authors, maternal age, education, verbal IQ, HOME environment index, and alcohol use during pregnancy were generally higher in the highest exposed group. Given that prenatal exposure to alcohol has been negatively correlated with neurodevelopment, this observation could be highly significant. The authors questioned whether the observed effects are due to PCBs or other contaminants but did not address this issue in their study. The authors also suggested that substances in breast milk or factors associated with breast-feeding might be important during the prenatal period of cognitive development. Lastly, the authors stated that the difference in results between Patandin et al. (1999) and those reported by Lanting et al. (1998) could be explained by differences in the testing procedures.

From these and other studies, it is clear that before any valid conclusions regarding the effects, if any, of PCBs on the cognitive and intellectual development of children can be made, the following factors must be established:

- The exact determination of the extent of exposure of the children to PCBs and other environmental contaminants during both *in utero* and postnatal periods.
- The nature of the influence of socio-economic factors and other poorly defined influences on the cognitive and intellectual development of children.
- The degree of uncertainty inherent in the intellectual testing procedures that are used.

It is thus apparent that there is little, if any, evidence that PCBs cause adverse non-cancer effects in humans, with the possible exception of minor dermal and ocular effects at high doses. As

discussed in the next section of these comments, this information is important to the derivation of an appropriate RfD for PCBs.

b. Animal Studies Relating PCBs to Non-Cancer Human Health Effects and Derivation of a Non-Cancer RfD for PCBs

As noted above, EPA used the RfD for Aroclor 1254 to assess certain non-cancer human health risks for the Hudson. The RfD for Aroclor 1254 is 0.0002 mg/kg/day and was derived from a LOAEL of 0.005 mg/kg/day using an uncertainty factor of 300 (IRIS, 1999).

(i) EPA's Derivation of the RfD for Aroclor 1254

The rhesus monkeys used in the study were exposed to 0, 0.005, 0.02, 0.04, or 0.08 mg/kg/day Aroclor 1254 in the diet for five years (Tryphonas et al., 1989, 1991a,b; Arnold et al., 1993a,b) (collectively referred to as the "Arnold/Tryphonas study"). Systemic, reproductive, and immunological effects were investigated during the pre-breeding phase of the study. Results of immunologic evaluations after 23 and 55 months of exposure were reported by Tryphonas et al. (1989, 1991a,b). General health and clinical pathology results following 37 months of exposure were reported by Arnold et al. (1993a,b).

Dose-related dermal, nail, and ocular effects, including ocular exudate, inflamed Meibomian glands, and changes in fingernails and toenails, were reported in all dose groups. Arnold et al. (1993a,b). In the immunologic portion of the study, Tryphonas et al. (1989) reported a statistically significant ($p<0.05$) dose-related decrease in antibody titers of IgM and IgG isotypes to sheep red blood cells (SRBCs) for all dose groups after 23 months of exposure. A significant dose-related decrease in IgM production was also observed after 55 months of exposure. Tryphonas et al. (1991a).

EPA derived an RfD of 0.0002 mg/kg/day from the LOAEL of 0.005 mg/kg/day by applying a total uncertainty factor of 300. The total uncertainty factor included a factor of 3 for interspecies extrapolation, a factor of 3 for use of a LOAEL rather than a NOAEL, a factor of 10 for interindividual human variability, and a factor of 3 for the subchronic nature of the study. The resultant uncertainty factor of 270 was rounded to 300.

There are several reasons why EPA should not have used the Arnold/Tryphonas studies to derive an RfD for Aroclor 1254. As indicated above, the LOAEL and subsequent RfD are based on immunological changes and dermal, nail and ocular effects. The immunologic changes should not be used to derive an RfD because the clinical relevance of these changes has not been demonstrated. Moreover, the dermal, nail and ocular effects should not be used to derive an RfD because these effects have been shown to occur in monkeys at much lower doses than those required to produce similar effects in humans. The following sections critique EPA's interpretation of the immunologic assessment and the clinical health data for these studies.

i. (A) Immunologic Assessment

Immunologic responses to PCB exposure have been evaluated in a limited number of epidemiologic studies, none of which establishes a causal link between PCB exposure and immunologic toxicity (Kimbrough, 1987; Emmett et al., 1988a,b; Kuratsune, 1989). A number of immune parameters have also been measured in rodents exposed to PCBs (Vos and de Roij, 1972, Loose et al., 1978; Thomas and Hinsdill, 1978; Truelove et al., 1982; Smialowicz et al., 1989). However, the results of these studies are mixed and inconclusive.

As noted above, in a series of three papers, Tryphonas et al. (1989, 1991a,b) studied the immunotoxicity of PCBs in primates. Five groups of rhesus monkeys (16 animals each) received long-term doses of Aroclor 1254 at either 0, 0.005, 0.02, 0.04, or 0.08 mg/kg/day in their diet. Tryphonas et al. (1989) reported on the immunological health of the animals after 23 months. A statistically significant ($p<0.05$) dose-related decrease in antibody titers of the IgM and IgG isotypes to SRBCs was reported for all dose groups. Lymphocyte counts were measured in the control and 0.08 mg/kg-day dose groups only. A significant increase in T-suppressor (T_s) cells (CD8 cells), a significant reduction in relative number of T-helper (T_H) cells (CD4 cells), and a significant reduction in the T_H/T_s ratio when compared to controls were reported. B-lymphocyte and total lymphocyte counts were not different from controls. Stimulation by standard mitogens produced no significant differences in lymphocyte transformation rates. No statistically significant differences were observed in total serum immunoglobulin levels, serum protein levels, or hydrocortisone levels when compared to the control group.

Tryphonas et al. (1991a) continued their investigations by examining additional nonspecific and specific immune parameters in the same group of rhesus monkeys after 55 months of exposure. A statistically significant dose-related decrease in IgM antibodies was observed in the monkeys that were immunized with SRBCs to measure their anamnestic or secondary response, but pairwise comparisons between treatment groups and controls showed that IgG levels did not decrease significantly. Antibody response to the pneumococcal antigen was similar in treated and control groups. Lymphocyte proliferation from stimulation by standard mitogens was similar in treated and control groups. A significant change was observed in the relative number of CD2 cells (to total lymphocytes) in all dose groups as compared to controls. Results for all other lymphocyte subpopulation analyses, including absolute number of total CD2 cells, were not significantly different from control levels. Results of a mixed lymphocyte culture assay (one-way) and a monocyte activation assay were similar in control and treated groups (Tryphonas et al., 1991a). Measures of the levels of hydrocortisone and interleukin-1 were not significantly different from control levels (Tryphonas et al., 1991a).

The effects of PCBs on nonspecific immune parameters in the rhesus monkey were reported by Tryphonas et al. (1991b). Serum complement (CH_{50}) activity was significantly higher in all treated groups than in the control group. This effect did not appear to be dose-related, however. A significant trend toward increased natural killer cell activity was observed at the adjusted effector (E) to tumor target (T) cell ratio (E:T) of 75:1. No difference in activity was found when a pairwise comparison was made between the treatment and control group. No significant differences were reported at the 50:1 or 25:1 ratios. Interferon (IFN) levels increased significantly from controls in the 0.02 and 0.08 mg/kg/day dose groups, IFN levels significantly decreased from controls in the 0.04 mg/kg/day group, and no difference was observed in the

0.005 mg/kg/day group when compared to controls. Hence, no dose response relationship was observed for this effect. No treatment-related effects were reported for tumor necrosis factor production.

The clinical relevance of the immunological responses reported by Tryphonas et al. (1989, 1991a,b) has not been demonstrated. In fact, the responses observed in the PCB-treated monkeys could be considered indicative of a healthy and intact immune system. To support this conclusion, it is necessary to outline the sequence of events in a typical immune response.

After exposure to an antigen (a molecule that stimulates a specific immune response, e.g., SRBCs or pneumococcus), phagocytosis or ingestion of the antigen by macrophages occurs. During phagocytosis, the antigen breaks down and antigen fragments move to the surface of the macrophage (antigen presentation). Antigen presentation leads to stimulation of the T-cell and B-cell systems, which are responsible for the immune response. Interleukin-1 (Il-1) is involved in the T-cell system and is responsible for activating resting T cells. Once stimulated, T cells will proliferate with the aid of Interleukin-2 (Il-2), previously called the T-cell growth factor. Activated T cells within the T-cell system will bind and kill target cells completing the cell-mediated immune response (Roitt et al., 1989; NRC, 1992). A complex network of humoral mediators (cytokines) modulates the response.

Antigen presentation also stimulates the B-cell system. Antigen fragments react with specific T lymphocytes called helper T cells (CD4 cells). An activated helper T cell will recognize processed antigen fragments in association with B lymphocytes and elaborate lymphokines that are stimulatory for B lymphocytes. A B cell, when stimulated, multiplies and differentiates into antibody-secreting plasma cells. The antibodies combine with the antigen to complete the humoral (antibody) immune response (Roitt et al., 1989; NRC, 1992).

The results reported by Tryphonas et al. (1991a,b) indicate that macrophage response and antigen presentation are intact in treated animals. Macrophages (monocytes) were stimulated *in vitro* with zymosan or phorbol myristate acetate (PMA). Monocyte activation was similar in the treated and control groups (Tryphonas et al., 1991a). Production of interleukin-1 (Il-1) and tumor necrosis factor (TNF) were unaffected by PCB treatment (Tryphonas et al., 1991a,b). As described previously, antigen presentation stimulates T-cell or B-cell systems. Several parameters measured by Tryphonas and coworkers documented the health of these systems in PCB-treated monkeys

T lymphocytes isolated from treated and control monkeys were stimulated by standard mitogens (phytohemagglutinin (PHA-P) and concanavalin A (Con A)). Lymphocyte proliferation was unaffected by PCB treatment (Tryphonas et al., 1989, 1991a). Con A is a strong T-cell mitogen. If T-cells are truly suppressed by PCB treatment as Tryphonas et al. (1989; 1991a) contend, then fewer T-cells should be able to respond to Con A. In fact, lymphocyte proliferation in Tryphonas et al. (1991a) was equivalent or greater in treated animals when analyzed using the parameter "Stimulation Index," as recommended by Luster et al. (1988) and Mishell and Shiigi (1980).

When lymphocyte populations were measured, total lymphocytes and total T-helper lymphocytes

(CD4 cells) did not differ between treated and untreated animals (Tryphonas et al., 1989, 1991a). An increase in T-suppressor (T_s) cells was reported in Tryphonas et al. (1989). However, the T_s nomenclature is outdated and misleading. T-suppressor cells are a subset of CD8 cells and exclude consideration of T_c cells (cytotoxic T-lymphocytes) which also are a subset of CD8 cells. In a normal immune response, one would expect to see an increase in cytotoxic T cells. In addition, the T_H/T_s ratio reported to be lower in treated animals Tryphonas et al. (1989) may be skewed since cytotoxic T lymphocytes were excluded. In fact, CD8 cells and the CD4/CD8 ratio were unaffected by PCB treatment when measured in Tryphonas et al. (1991a).

The Tryphonas studies report evidence that B cells remained healthy in PCB-treated monkeys. The levels of available B lymphocytes (CD20 cells) were unaffected by PCB treatment. Tryphonas et al. (1989; 1991a). Lymphocyte proliferation induced by pokeweed mitogen, a B-cell mitogen, was similar in treated and control animals (Tryphonas et al., 1991a). Total serum immunoglobulin levels were unaffected by PCB treatment as well (Tryphonas et al., 1989).

After inoculation with SRBCs, a reduction in the number of IgM and IgG antibodies was observed. Tryphonas et al. (1989). While a statistical significance between control and treated levels was reported, the difference is not clinically significant. At least a 4-fold change in titer count is necessary to be considered clinically significant. Paul and White (1973). When conducting an immunological assessment, a positive control group is normally included by treating animals with cyclophosphamide, which results in significant immunosuppression and provides an indicator of relative "immunotoxic potency" (Luster et al., 1988). A positive control group was not included in the work conducted by Tryphonas et al. (1989, 1991a,b). The reduction in antibodies reported in Tryphonas et al. (1989) may be within the range for variation and not comparable to that observed for a truly suppressive chemical like cyclophosphamide.

Tryphonas et al. (1991a) investigated the anamnestic or secondary response to SRBCs and reported a significant reduction in IgM antibodies but not IgG antibodies. The anamnestic response to an antigen is primarily by the IgG isotype (Roitt et al., 1989; Amdur et al., 1991). In such a response, IgM antibody is inconsequential, because only small amounts are produced and are of lesser affinity/avidity than the IgG produced. This suggests that the extended exposure to PCBs in the monkeys had little effect on the elaboration of memory cells and, in fact, a classic IgG dominated, anamnestic response was elicited (Roitt et al., 1989).

Unlike the artificial nature of the SRBC antigen, immunization with the pneumococcal vaccine in Tryphonas et al. (1991a) provided a "real" antigen. Results of the pneumococcus challenge demonstrated that B-cells received proper signals from helper T-cells and that B-cell populations were healthy and intact.

Overall, the results of immune response testing reported by Tryphonas et al. (1989, 1991a,b) have not demonstrated an immunotoxic response and are, in fact, indicative of a healthy immune system in both the PCB-treated and untreated monkeys. Other reviewers have reached similar conclusions. For example, Letvin (1993) has concluded that there is no evidence that any of the subtle changes in immune function reported by these investigators have any clinical significance. Similarly, in a review of the human health effects of PCBs, Kimbrough (1995) noted that the

decreases in immunoglobulin IgM and IgG isotypes to SRBCs reported by Tryphonas et al. (1989, 1991a, b), though statistically significant, are not clinically relevant. She also points out that (as discussed above) a number of other immunological assays (unrelated to SRBC induction of antibodies) conducted by these investigators showed no evidence of adverse effects related to PCBs. ATSDR (1993) acknowledged this lack of confirmation when it stated that "the interpretation of the toxicological significance of the effects reported by Tryphonas et al. (1989; 1991a) is complicated because other immunological parameters monitored were not significantly affected . . . ." In short, as Kimbrough (1995) concluded, the immunological responses reported by these investigators do not suggest immunotoxicity.

In the Response to Comments document for the Hudson River Scope of Work (EPA, 1999), EPA defended its consideration of the immunological findings reported by Tryphonas et al. (1989, 1991, a,b). Stating that tests very similar to those used by Tryphonas et al. "are widely used in hospitals and clinical laboratories to diagnose immune deficiencies in suspected immuno-compromised patients." GE strongly disagrees with this position.

The appropriateness of using the immune response to the SRBC antigen in PCB-treated monkeys, or any other test species, in determining the possible impact of PCB exposure on human immune system responsiveness is questionable at best. First, there is no documentation that indicates that the human immune system is sensitive to PCBs at the exposure levels typically encountered. And secondly, there is evidence that the effect of PCBs on the immune response is variable across test species and, therefore, any PCB effects in one species may not be predictive of PCB effects in humans. Smialowicz et al. (1994) reported that in contrast to the immunosuppressive effects seen in mice, TCDD significantly enhanced this response in rats administered doses as low as 10 ug/kg. Furthermore, they later reported in a study with mice that PCB 153 also enhanced the SRBC response (Smialowicz et al., 1997). When PCB 153 and TCDD were co-administered, they found that the immunosuppressive effect of TCDD was completely eliminated and a normal response was observed. Clearly, these findings indicate that any use of this approach to evaluate the risk of immune impairment in humans by this class of compounds is not justified and will lead to serious miscalculations of risk.

In addition, the available human data do not provide evidence of immunologic effects from PCB exposure. As noted above, epidemiologic studies that have evaluated immunotoxicity of PCBs have not shown a causal association between PCB exposure and immunological effects in humans. For example, in studies conducted by Emmett et al. (1988a,b), PCB-exposed transformer maintenance and repair workers (serum PCB levels of up to 300 mg/L) did not differ significantly from non-PCB exposed, matched controls (serum PCB levels of up to 15 mg/L) in delayed hypersensitivity reaction tests which measure T-cell and macrophage response. Moreover, although average PCB levels in the PCB-exposed workers (geometric mean concentration of 9.4 mg/L) were comparable to the steady state serum PCB levels (10 mg/L) measured in the Tryphonas et al. (1991a) rhesus monkeys, no adverse immunologic effects or clinical signs of immunocompromise were observed in the PCB-exposed workers (Kimbrough, 1995).

(B) Differences Between Rhesus Monkeys and Humans for PCB-Induced Dermal, Ocular and Nail Effects

As previously stated, EPA finds additional support for its RfD for Aroclor 1254 in the findings of adverse dermal, ocular and nail effects in rhesus monkeys. However, comparison of the laboratory studies of rhesus monkeys and human epidemiologic studies suggests that PCBs produce nail changes, ocular effects, and dermal effects in rhesus monkeys with significantly lower body burdens than have been measured in the general population and in workers occupationally exposed to PCBs who have generally shown no adverse effects. As discussed below, these findings suggest that rhesus monkeys are significantly more sensitive to these effects than humans exposed to PCBs.

Arnold (1993a,b) reported that rhesus monkeys receiving 0.005 mg/kg/day doses of Aroclor 1254 displayed a number of readily observable effects including: swelling of the Meibomian glands, swelling around the eyes, nail discoloration, and nail loss. The PCB blood concentrations associated with these effects in the test animals were 10 mg/L in plasma; the level in adipose tissue was approximately 2.7 mg/L (wet weight). In another study of systemic effects of Aroclor 1016 in rhesus monkeys conducted by Barsotti (1980), swelling around the eyes, ocular exudate, alopecia, and acne were reported in rhesus monkeys exposed to Aroclor 1016. The body burden of PCBs in the Barsotti (1980) study was measured at 33 mg/L, i.e., 10-fold higher than the animals in the low dose group of Arnold et al. (1993a,b).

By contrast, human epidemiological studies have not shown dermal, ocular, or nail effects in humans resulting from higher body burdens of PCBs. One of the most serious dermal conditions of concern for human exposure to halogenated aromatic compounds is chloracne. A number of reports have been published on the incidence of chloracne in two populations in Asia who had significant concurrent exposure to PCBs, elevated levels of polychlorinated dibenzofurans (PCDFs), and other chlorinated compounds via consumption of contaminated rice oil (Kunita et al., 1984, Kuratsune, 1989). These accidental poisonings occurred in Japan and Taiwan in what are known as the "Yusho" and "Yu-Cheng" incidents. However, the adverse health effects associated with the Yusho and Yu-Cheng poisoning episodes have been attributed to PCDFs, not PCBs (Kashimoto et al., 1985; Kamrin and Fisher, 1989).

Other, less serious dermal effects of PCBs in humans have also been investigated in numerous epidemiological studies (Ouw et al., 1976; Fischbein et al., 1979, 1982, 1985; Lu and Wong, 1984; Lu and Wu, 1985; Kuratsune, 1989; Rogan, 1989; Wolff et al., 1992). Several studies have reported minor dermal effects (skin rashes, changes in pigmentation of the skin and nails, erythema and thickening of the skin, and burning sensations) following occupational exposure to relatively high levels of PCBs (Ouw et al., 1976; Lu and Wong, 1984; Fischbein et al., 1985; Lu and Wu, 1985; Kuratsune, 1989; Rogan, 1989). In other studies, however, no dermal, ocular, or nail effects were reported in PCB-exposed workers, and in studies where dermal, ocular, and nail effects were observed, no significant correlation was found between dermal findings and PCB body burdens (Ouw et al., 1976; Wolff et al., 1982; Smith et al., 1982; Lawton et al., 1985; Emmett et al., 1988a,b; Taylor et al., 1988).

Although the workers in these epidemiological studies had mean serum PCB levels up to two orders of magnitude greater than the levels in the Aroclor 1254-exposed rhesus monkeys (Arnold

et al., 1993a,b), the adverse dermal effects (chloracne-like lesions, edema of the eyelids, eye exudate, conjunctivitis, and nail loss) observed in monkeys were not observed in workers. As Dr. Renate Kimbrough (1995) commented: "These clinical signs would be easily recognizable had they occurred in the hundreds of workers that have been studied and in the thousands of workers that made capacitors. However, they have not been reported." Kimbrough (1995) further points out that the serum PCB levels (10 mg/L) measured in the offspring of PCB-exposed rhesus monkeys (Arnold et al., 1990) are comparable to levels found in the general U.S. population of adults and infants. However, these effects have not been observed in the general population of infants or in infants born to mothers working in capacitor plants with body burdens much higher than the exposed monkeys, (Taylor, 1988). Kimbrough's finding is further supported by data from an ATSDR (1993) review of published literature on historical body burdens of PCBs in the general population and in specific subpopulations. These data suggest that many groups of adults (particularly those measured during the 1980s) have had average PCB body burdens similar to or in excess of the levels associated with dermal, ocular, and nail effects observed in the Arnold et al. (1993a,b) studies. Yet similar effects were not observed in these populations. (Gillis and Price, 1996)

In the Responsiveness Summary for the Scope of Work EPA (1999), EPA asserts that the data on the exposures to the rhesus monkeys was the "result of precise, constant daily dosing that was monitored and very well characterized" while the effect (or lack thereof) was reported in workers whose exposures "were based on sporadic doses, may not have been monitored, and were very poorly characterized." GE is puzzled by this statement. Gillis and Price (1996) compared measurements of body burdens not doses. The study compared measurement of body burden as reported in ATSDR (1995) Toxicology Profile for PCBs. In addition, since the body burden resulted from workers, (and the general population,) long-term exposures, it is difficult to see the relevance of the timing or degree of control exerted over their exposures. Thus, the measurements of both the monkeys and humans were essentially identical and the Agency's objection to considering the findings of Gillis and Price (1996) appears to be baseless.

In summary, a comparison of PCB body burdens in exposed human populations and rhesus monkeys exposed in the laboratory indicates that the rhesus monkey is more sensitive to PCB dermal, ocular, and nail effects than humans. This suggests that rhesus monkeys are not an appropriate model for the dermal, ocular, and nail effects of PCBs in humans.

In addition to epidemiological evidence indicating that rhesus monkeys are more sensitive than humans to the dermal/ocular/nail effects of PCBs, there is mechanistic evidence suggesting that, as a more general matter, the rhesus monkey may be a poor model for PCB toxicity in humans. Recent studies of PCB metabolism suggest that PCBs are metabolized differently in humans and rhesus monkeys and that the metabolism of PCBs may be critical to the overall expression of PCB toxicity. One indication of this difference is the line of evidence suggesting that the patterns of PCB congeners that accumulate in adipose and hepatic tissues of rhesus monkeys chronically exposed to Aroclor 1254 differ from patterns of congener retention in humans.

Two PCB metabolic pathways and associated enzyme systems have been identified in humans based on patterns in the relative retention of PCB congeners. Humans exposed to PCBs have

been shown to produce patterns of congener retention similar to that observed in the in vitro studies of P450 2B enzyme activity (Brown et al., 1989, 1994). One pattern is referred to as P450 2B-type metabolism and is also exhibited by other terrestrial mammals, birds, and crustaceans (Brown, 1992). PCBs have been shown to induce the P450 2B enzyme activity in humans with PCB body burdens as low as 20 g/kg (lipid basis) in blood (Brown et al., 1994).

A second pattern (Pattern A) (Masuda et al., 1978; Kunita et al., 1984) occurs in humans exposed to mixtures of PCBs and furans, and results from metabolism of PCBs by a combination of P450 2B-type and P450 1A-mediated metabolic pathways. It appears that, in the absence of concurrent exposures to dioxins and furans, PCBs do not induce the P450 1A enzymes in humans. Similar findings were also reported by Kunita et al. (1984) who noted that PCB congener retention patterns in Yusho and Yu Cheng patients exposed to PCBs and PCDFs were different from Japanese occupational workers exposed to PCBs but not to PCDFs. Furthermore, studies of PCB induction of P450 1A in rodents indicate that such induction, if it occurs in humans, would require PCB exposures far higher than have occurred from environmental or occupational settings (Brown et al., 1991).

In contrast to the metabolism of PCBs in humans (in the absence of concurrent exposures to dioxins and furans), a different pattern was observed in rhesus monkeys in that the administered PCBs were metabolized by two pathways (Brown et al., 1994). The first pathway is the P450 1A pathway. The second pathway, known as the P450RH pathway, appears to be unique to the rhesus monkey. Brown et al. (1994) determined patterns of PCB congeners in various organs of the same test animals that were used in the original Tryphonas et al. (1989) and Arnold et al. (1993a,b) studies. The study included animals with body burdens as low as 10 mg/L in blood plasma, suggesting that the metabolism of PCBs in rhesus monkeys differs significantly from metabolism in humans at the lowest doses associated with adverse effects.

The specific enzymes responsible for metabolizing PCBs in the unusual P450RH pattern observed in monkeys are unclear at this time. However, studies of enzymatic activity indicate that Cynomolgus monkeys, a species closely related to the rhesus, have P450 enzymes that differ from humans (Yoshimura et al., 1987; Komori et al., 1992). This finding provides additional evidence that rhesus monkeys may differ from humans in the metabolism of PCBs and thus may be an inappropriate model for PCB toxicity in humans.

Several studies have demonstrated that the pattern of PCB metabolism is critical to the expression of PCB toxicity in humans (Brown et al., 1989, 1991, 1994). For example, induction of P450 1A at low PCB doses is associated with dermal, ocular, and nail effects in animals (Brown et al., 1994). In humans, Yusho victims, who were exposed to both PCBs and furans and experienced many of these effects, also displayed P450 1A metabolism. Conversely, metabolism of PCBs under the P450 2B-type pathway in occupationally exposed human populations is not associated with these effects.

The mechanism by which halogenated aromatic hydrocarbons (HAHs), including PCBs, produce their effects has been the subject of intensive research. One hypothesis is that HAHs cause their effects by stimulating enzyme systems that metabolize both the HAH and certain nonchlorinated

lipophilic compounds that play key roles in hormonal regulation of various organ systems. These compounds may include steroids, retinoids, and possibly thyroxinol. If this is true, then the atypical cytochrome P450 activity observed in monkeys may be indicative of a unique metabolism of other lipophilic hormones, possibly including those which regulate cell growth and differentiation.

EPA, in its "Health Assessment Document for 2,3,7,8-Tetrachloro-*p*-dioxin (TCDD) and Related Compounds," (EPA, 1994) has recognized (a) that a wide variety of the characteristic dermal, hepatotoxic, immunotoxic, and developmental effects induced by sufficiently high doses of dioxin-like compounds are mediated by their binding to, and thence activating, the Ah-receptor (AhR); (b) that the magnitude of these effects correlates with the tissue accumulation of the administered agent, rather than the dose rate; and (c) that there are large interspecies and intertoxicant differences in animals' sensitivity to these AhR-mediated toxic effects.

The available literature shows that the rhesus monkey is unusually sensitive to the induction of dermal, immunological, and reproductive derangements by Aroclor 1254. Notably, Arnold et al. (1990) reported that all female rhesus monkeys administered Aroclor 1254 at 5 ppm in their diet developed inflammation and/or enlargement of the tarsal glands, edema of the eyelids, eye exudates, conjunctivitis, gingivitis, and various nail bed alterations, followed by loss of nails within 2-25 months of dosing. The extensive literature on the Japanese and Taiwanese rice oil (yusho or yu-cheng) PCB/PCDF poisoning incidents shows that all these characteristic AhR-mediated dermal responses, as well as chloracne, may also be exhibited in man – but only if PCDFs are present as sizeable contaminants in the PCBs. (Kunita et al, 1994). In a group of 194 extensively studied capacitor workers carrying geometric mean serum levels of 363 ppb (5%-95% range, 57-2269 ppb lower PCBs (GC retention times < DDE) and 30 ppb (5%-95% range, 6-142 ppb) higher PCBs (GC retention times > DDE; similar to residues from Aroclor 1254, and containing all the congeners with significant AhR-agonist activity), there were no observations of chloracne, fingernail loss, or other unusual dermal symptoms (Lawton et al., 1985). However, the Aroclor 1254-dosed rhesus monkeys that exhibited a 100% incidence of these dermal effects did so after accumulating blood levels of only 10 ppb PCB (about 90% higher PCBs) Mes et al. 1981). From this we must conclude that the human is at least 15-fold less sensitive than the rhesus monkey to the induction of AhR-mediated toxic effects by PCBs, and probably much more so.

In summary, these findings on the metabolism of PCBs suggest that the differences between rhesus monkeys and humans with respect to PCB toxicology may extend beyond dermal, ocular, nail, and immunological effects. As a result, the use of rhesus monkey studies as a model for the evaluation of any potential toxic effects of PCBs in humans is inappropriate and the Arnold/Tryphonas studies should not be used to derive an RfD for Aroclor 1254.

For the reasons discussed above, GE does not believe that the Arnold/Tryphonas studies should be used to derive an RfD for Aroclor 1254. However, if an RfD is derived based on these studies, the RfD should be based on the dermal, nail and ocular effects reported, not on immunotoxicity. As discussed above, the reported immunological responses simply do not suggest immunotoxicity (Kimbrough, 1995).

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ATTACHMENT B

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

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

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

Limitations of the Connelly et al. (1992) Dataset

Survey Design

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

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

8 ounces, the Agency may have substantially over- or underestimated intakes by individual anglers and did not consider the variability associated with this parameter.

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

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

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

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

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

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

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

Response Rate

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

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

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

This approach does not give adequate weight to the remainder of non-respondents. If it is assumed that the subsample of the 919 non-respondents to the survey is representative of the entire non-respondent population, this means that 55 percent of all non-respondents, or 505 individuals, were consumers of fish. According to the data provided by respondents to the survey, 37.6 percent of the respondents who ate fish consumed fish from flowing waterbodies. If this same fraction is applied to the 505 non-respondents who consumed fish, it can be assumed that 190 non-respondents consumed fish from flowing waterbodies during the survey period. These individuals should have been included in the correction for non-response bias to provide a total

sample of 416 anglers (226 respondents plus 190 non-respondents). Inclusion of these additional, non-responding consumers would have resulted in substantially lower estimates of fish consumption for the total angler population.

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

Consistency Among Studies of Similar Populations

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

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

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

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

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

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

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

appropriate to use to determine for the HHRA fish ingestion distribution.

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

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

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

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

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

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

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

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

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

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

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

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

Finally, although Ebert et al. (1993) did not correct for non-response bias, this is not a sound basis

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

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

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

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

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

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

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

Selection of the Most Appropriate Fish Consumption Distribution

Meal Sizes

Using Connelly et al. (1992) required EPA to make an assumption about the size of each meal in order to derive annualized daily consumption rates. Such assumptions are not needed to use

either the Connelly et al. (1996) or the Ebert et al. (1993) surveys. Connelly et al. (1996) required that each respondent record the size of each fish meal consumed as either "less than", "equal to", or "more than" a 0.5 pound meal pictured among the survey materials. While this approach also requires that some assumptions be made as to the actual sizes of the meals, it provides an added degree of precision not possible with the Connelly et al. (1992) data. Indeed, Connelly et al. (1996) assigned 5 ounces to represent meals that were less than 0.5 pound and 12 ounces to represent meals that were larger than 0.5 pound.

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

Species Lists

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

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

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

Segregation of Data by Waterbody Type

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

The Ebert et al. (1993) study does not suffer from this problem because respondents were

required to record fish consumed in one of two categories of waterbodies: flowing or standing waters. Thus, all fish consumed can be attributed to a particular type of waterbody, thereby reducing the uncertainty in these estimates.

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

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

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

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

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

Uncertainty in Fish Meal Estimates

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

The Maine angler survey does not suffer from this problem because anglers were asked to recall all fish consumed from all waterbodies. Although recall and digit bias may introduce some uncertainty, it is likely that responses were more accurate than responses given as a "?". In addition, because both surveys were mail surveys with a one-year recall period, the direction and degree of recall bias is likely to be similar for both. Because long-term recall tends to result in

overestimation of fishing activities (Westat Inc., 1989; West et al., 1989a,b; Connelly and Brown, 1995; Roach et al., 1999), it is likely that any inaccuracies from this type of bias result in an overestimation of fish consumed, providing an additional degree of conservatism in the Ebert et al. (1993) distribution.

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

Sample Size

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

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

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

Response Rate

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

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

The HHRA faulted the Ebert et al. (1993) study for not having completed a follow-up survey of non-respondents that would have allowed an adjustment for non-response bias in the survey results. The findings of other non-response follow-ups in studies of angler participation and consumption have shown that non-respondents tend to have lower participation and consume less fish than do respondents (Brown and Wilkins, 1978; West et al., 1989a,b; Connelly et al., 1990).

This relationship was confirmed by the follow-up results reported by Connelly et al. (1992). As a result, it is likely that the Ebert et al. (1993) survey of Maine anglers overestimates consumption by the total angler population and thus represents a conservative estimate of consumption by freshwater recreational anglers in the Northeast.

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

Summary

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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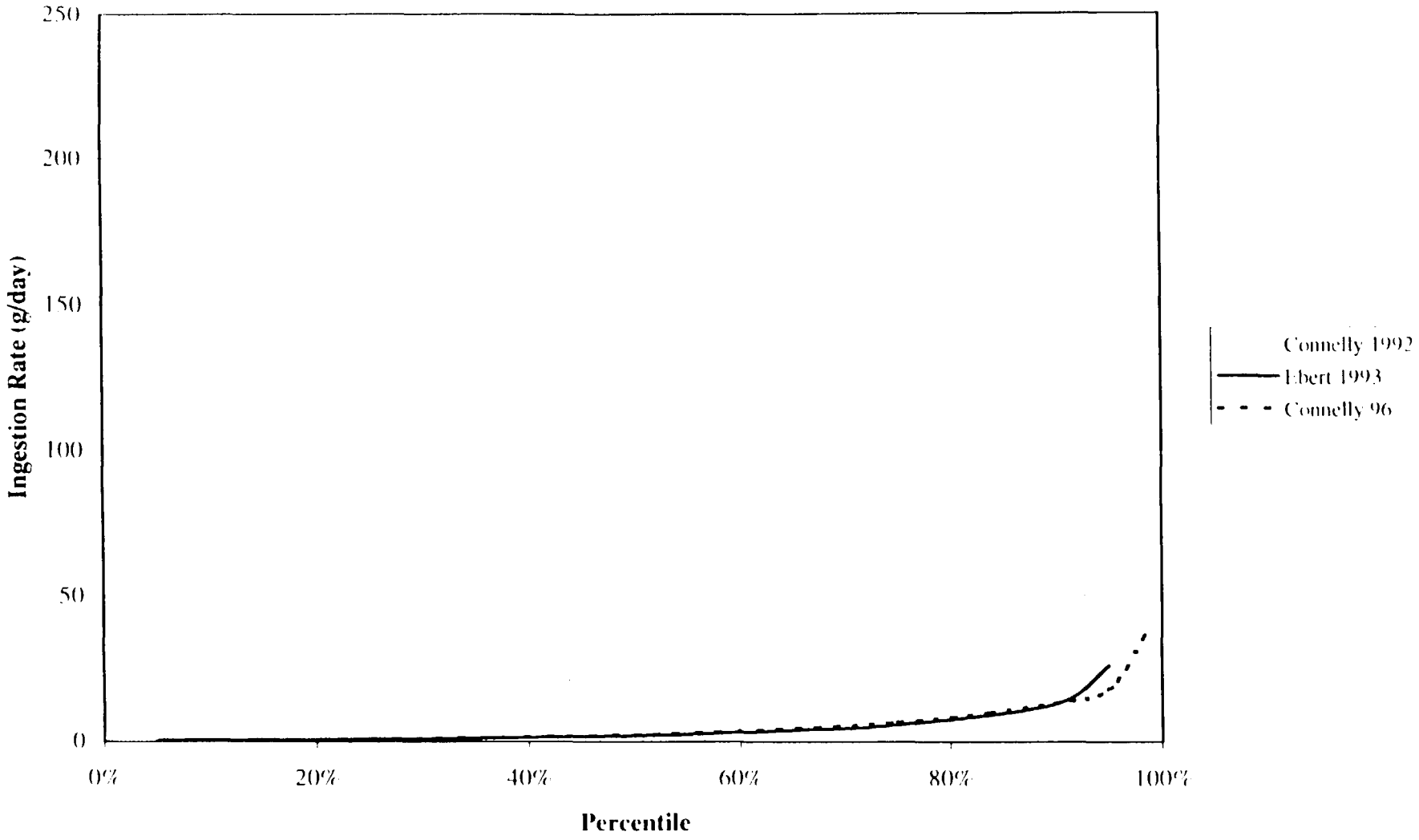
Figure B-1. Comparison of Different Consumption Rate Studies



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

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

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

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

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

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

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

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

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

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

The Probabilistic Assessment Fails to Meet Agency Guidance

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

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

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

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

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

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

3. *Sensitivity analysis results must be presented and discussed.*

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

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

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

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

4. *Information for each input and output distribution must be given in the report including:*

- *Tables and graphs of the distributions,*
- *An explanation for the choice of distributions, and*
- *Differentiate variability and uncertainty for both input and output distributions.*

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

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

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

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

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

Failure to Model Temporal Variation in Model Inputs Properly

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

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

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

Failure to Characterize Variation and Uncertainty Jointly

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

distribution of uncertainty and variability. Numerous techniques exist for merging the results from multiple studies, ranging from meta-analysis to simple systems of weighting the studies, but the HHRA does not discuss such techniques.

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

Failure to Model Chronic Dose Rates Properly

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

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

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

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

Evaluation of Uncertainty in Probabilistic Assessment

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

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

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

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

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

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

spacing across the range of plausible values.

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

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

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

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

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

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

meta-analysis or systems of weights can be used to characterize uncertainty.

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

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

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

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

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

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

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

consumed fish were non-consuming anglers (catch-and-release anglers).

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

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

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

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

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

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

investigate how the estimates of risk would have been impacted by this alternative assumption.

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

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

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