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

AUGUST 2000

For

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

Book 1 of 1

TAMS Consultants, Inc.
***Gradient** Corporation*

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

August 29, 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 Mid-Hudson River (Mid-Hudson 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 Mid-Hudson HHRA and this Responsiveness Summary should be used together.

In the Responsiveness Summary, USEPA has responded to all significant written comments received during the public comment period on the Mid-Hudson 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 December 1999 Baseline Ecological Risk Assessment for Future Risks in the Lower Hudson River (ERA Addendum), the August 2000 Responsiveness Summary for the ERA Addendum, and the January 2000 Revised Baseline Modeling Report. This Responsiveness Summary also provides separate calculations for cancer risks and non-cancer hazards to children eating fish from the Mid-Hudson River. Importantly, the overall conclusions regarding the cancer risks and non-cancer hazards due to PCBs in the Mid-Hudson River remain unchanged.

If you need additional information regarding the Responsiveness Summary for the Mid-Hudson HHRA or the Reassessment RI/FS in general, please contact Ann Rychlenski, the Community Relations Coordinator for this site, at (212) 637-3672.

Sincerely yours,

William McCabe

Richard L. Caspe, Director
Emergency and Remedial Response Division

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Section III

ACRONYMS

ATSDR	Agency for Toxic Substances and Disease Registry
BERA	Baseline Ecological Risk Assessment
CDI	Chronic Daily Intake
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CIP	Community Interaction Program
CLH	Chemical Land Holdings
CSF	Carcinogenic Slope Factor
CTE	Central Tendency Exposure
EPC	Exposure Point Concentration
FDA	Food and Drug Administration
FS	Feasibility Study
GE	General Electric Company
HHRA	Human Health Risk Assessment
HHRASOW	Human Health Risk Assessment Scope of Work
HI	Hazard Index
HQ	Hazard Quotient
HROC	Hudson River PCBs Oversight Committee
IRIS	Integrated Risk Information System
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NOAA	National Oceanic and Atmospheric Administration
NPL	National Priorities List
NYSDEC	New York State Department of Environmental Conservation
NYSDOH	New York State Department of Health
PCB	Polychlorinated Biphenyl
RBMR	Revised Baseline Modeling Report
RfD	Reference Dose
RI	Remedial Investigation
RI/FS	Remedial Investigation/Feasibility Study
RM	River Mile
RME	Reasonable Maximum Exposure
ROD	Record of Decision
SARA	Superfund Amendments and Reauthorization Act of 1986
SCEMC	Saratoga County Environmental Management Council
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
USF&W	United States Fish and Wildlife

Introduction

I. INTRODUCTION AND COMMENT DIRECTORY FOR THE HUMAN HEALTH RISK ASSESSMENT FOR THE MID-HUDSON RIVER (MID-HUDSON HHRA)

1. INTRODUCTION

The U.S. Environmental Protection Agency (USEPA) has prepared this Responsiveness Summary for Volume 2F-A Human Health Risk Assessment Report for the Mid-Hudson River (Mid-Hudson HHRA), Hudson River PCBs Reassessment Remedial Investigation/Feasibility Study (Reassessment RI/FS), dated December 1999 (USEPA, 1999a). This Responsiveness Summary addresses comments received during the public comment period on the Mid-Hudson HHRA (USEPA, 1999a).

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 Mid-Hudson HHRA is incorporated by reference and is not reproduced herein. The comment responses and revisions noted herein are considered to amend the Mid-Hudson HHRA. For complete coverage, the Mid-Hudson HHRA and this Mid-Hudson Responsiveness Summary must be used together.

The first part of this Responsiveness Summary is entitled "Introduction and Comment Directory for the Human Health Risk Assessment for the Mid-Hudson River (Mid-Hudson HHRA)." It describes the Mid-Hudson 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 Human Health Risk Assessment for the Mid-Hudson River," contains USEPA's responses to all significant comments. Responses are grouped according to the section number of the Mid-Hudson HHRA to which they refer. For example, responses to comments on Section 2.1 of the Mid-Hudson 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 third part, entitled "Risk Assessment Revisions," presents the revised results for the Mid-Hudson HHRA, incorporating the modified forecast concentrations of PCBs in fish, sediments, and river water. To facilitate comparison to the December 1999 Mid-Hudson HHRA results (USEPA, 1999a), all table and figure numberings have retained their original designations.

The fourth part, entitled "Comments on the Human Health Risk Assessment for the Mid-Hudson River," contains copies of the comments submitted to the USEPA on the Mid-Hudson HHRA. The comments are identified by commenter 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 the Mid-Hudson HHRA

The Mid-Hudson HHRA, issued in December 1999, was distributed to federal and state agencies and officials, participants in the CIP, and General Electric Company (GE), as shown in Table 1. Distribution was made to approximately 100 agencies, groups, and individuals. Copies of the Mid-Hudson HHRA also were made available for public review in 16 information repositories, as shown in Table 2 and on the USEPA Region 2 internet web page, 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 Mid-Hudson HHRA from December 29, 1999 to January 28, 2000. USEPA held a Joint Liaison Group meeting on January 11, 2000 in Poughkeepsie, New York that was open to the public to present the Mid-Hudson HHRA. Subsequently, USEPA sponsored an availability session to answer questions on January 18, 2000 in Poughkeepsie, New York. 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 Mid-Hudson 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 Mid-Hudson HHRA were received in letters sent to USEPA and oral statements made at the January 11, 2000 Joint Liaison Group meeting. USEPA's responses to oral statements made at the Joint Liaison Group meeting are provided in the meeting minutes. Written comments were received from seven commenters; total comments numbered approximately seventy. All significant written comments received on the Mid-Hudson HHRA are addressed in this Responsiveness Summary.

2.4 Distribution of the Responsiveness Summary

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

TABLE 1
DISTRIBUTION OF MID-HUDSON 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 (NYSDOH) representative
- GE representative
- Liaison Group Chair people
- 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, SUNY - Albany
- 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 MID-HUDSON 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 Mid-Hudson 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

INFORMATION REPOSITORIES

Copies of the Mid-Hudson HHRA were placed in 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 COMMENTS AND RESPONSES TO COMMENTS

3.1 Identification of Comments

Each submission commenting on the Mid-Hudson HHRA was assigned the letter "H" for Mid-Hudson HHRA, and one of 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, and HF-1.3. Comment letters have been reprinted in section IV of this document, following the fifth tab.

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 commenters and comments. The comment directory table is organized as follows:

- The first column lists the names of commenters. Comments are grouped in the following order: HF (Federal), HS (State), HL (Local), HP (Public Interest Groups and Individuals) and HG (General Electric Company).

- 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 Mid-Hudson HHRA section number. For example, comments on Section 3.2 of the Mid-Hudson HHRA can be found in the corresponding Section 3.2 of the Responses section.

- 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.

4. COMMENT DIRECTORY

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

4.1 Guide To Comment Directory

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

Example:

COMMENT DIRECTORY FOR THE MID-HUDSON HHRA

AGENCY/ NAME	COMMENT CODE	REPORT SECTION	KEY WORDS		
			1	2	3
NOAA /Rosman	HF-1.6	2.3.1	Carp	Catfish	Eel

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Comment Directory

4.2 COMMENT DIRECTORY FOR THE MID-HUDSON HHRA

AGENCY/NAME	COMMENT CODE	REPORT SECTION	KEYWORDS 1	2	3
NOAA/Rosman	HF-1 1	1.2	Risk assessment	Lower Hudson	Definition of site area
NOAA/Rosman	HF-1 2	2 3	Baseline modeling	Farley model	Uncertainty
NOAA/Rosman	HF-1 3	2 3	Baseline modeling	Supplemental analyses	Incorporation
NOAA/Rosman	HF-1.4	2.4	Start date	1999	Underestimate
NOAA/Rosman	HF-1.5	2.3 1	Carp	Catfish	Eel
NYSDEC/Ports	HS-1.1	2 1 3	Residential exposures	Homegrown crops	Local produce and meat
NYSDEC/Ports	HS-1 2	General	Rogers Island	Risk assessment	Comparison
NYSDEC/Ports	HS-1 3	2 4	Lifetime	Exposure duration	High-end
NYSDEC/Ports	HS-1 4	2 4	Past exposures	Risk assessment	
NYSDEC/Ports	HS-1 5	2 1 2	Children	High-end	Fish consumption
NYSDEC/Ports	HS-1 6	4 2	NCP	Acceptable risk range	Risk Management
NYSDEC/Ports	HS-1 7	General	Cancer risks	Individual	Population
NYSDEC/Ports	HS-1 8	2 3 1	Species fractions	Brown Bullhead	Catfish
NYSDEC/Ports	HS-1 9	2.1 2	Children	High-end	Fish consumption
NYSDEC/Ports	HS-1 10	4	FDA tolerance level	Fish concentrations	Comparison
NYSDEC/Ports	HS-1 11	2 4	Lifetime	Exposure duration	High-end
NYSDEC/Ports	HS-1 12	3 2	Toxicity values	Selection	Cancer slope factors
NYSDEC/Ports	HS-1 13	3	RfD derivation	Uncertainties	New Information
NYSDEC/Ports	HS-1 14	3.1	Aroclor 1016	Aroclor 1254	Comparison
NYSDEC/Ports	HS-1 15	3 1	Toxicity profile	Out of date	New information
SCEMC/Balet	HL-1 1	General	Baseline modeling	Farley model	Availability
SCEMC/Balet	HL-1 2	2.3	Farley model	Review	
SCEMC/Balet	HL-1 3	2 3	PCB loading	Contribution	Lower Hudson
SCEMC/Balet	HL-1.4	General	Upper Hudson HHRA	Earlier comments	
SCEMC/Balet	HL-1 5	2 3	Farley model	Congeners	Extrapolations
SCEMC/ Balet	HL-1 6	General	Exposure assessment	Upper Hudson HHRA	Earlier comments
SCEMC/ Balet	HL-1.7	General	Toxicity assessment	Upper Hudson HHRA	Earlier comments
SCEMC/ Balet	HL-1 8	2 3 1	Striped bass	PCB concentration	
SCEMC/ Balet	HL-1 9	2.3	River Miles	Representative	Averaging
SCEMC/ Balet	HL-1 10	2 3 1	RME	PCB concentration	Fish
SCEMC/ Balet	HL-1 11	General	Risk characterization	Upper Hudson HHRA	Earlier comments
Scenic Hudson	HP-1 1	General	Cleanup		
Scenic Hudson	HP-1.2	General	Timeframe	Cleanup	
Scenic Hudson	HP-1 3	2 4 1	Fish consumption	Advisories	HHRA
Scenic Hudson	HP-1.4	General	Institutional controls	Cleanup	
Scenic Hudson	HP-1 5	General	Cleanup level	FDA level	Inadequate Protection
Scenic Hudson	HP-1 6	General	Cleanup		
AMC/Gardner	HP-2 1	General	Timeframe	Cleanup	
AMC/Gardner	HP-2 2	General	Cleanup level	FDA level	Inadequate Protection
AMC/Gardner	HP-2 3	2 4 1	Fish consumption	Advisories	HHRA
AMC/Gardner	HP-2 4	General	Cleanup		
LeRoy	HP-3 1	2 4.1	Fish ingestion rate	Exposure duration	Conservatisms
LeRoy	HP-3 2	General	Community studies	Epidemiology	Actual vs. hypothetical

4.2 COMMENT DIRECTORY FOR THE MID-HUDSON HHRA

AGENCY/NAME	COMMENT REPORT CODE	SECTION	KEYWORDS 1	2	3
LeRoy	HP-3.3	General	PCB concentrations	Decline with time	Risk management
LeRoy	HP-3.4	General	Fish advisories	NY waterbodies	Risk management
LeRoy	HP-3.5	General	Community studies	Clean-up	Risk management
LeRoy	HP-3.6	General	Fish advisories	NY waterbodies	
LeRoy	HP-3.7	General	Health advisories	Public education	Research
LeRoy	HP-3.8	2.3.1	Fish concentrations	Edible tissues	
LeRoy	HP-3.9	General	Community studies	Epidemiology	
LeRoy	HP-3.10	4	FDA limits	Commercial Food	
LeRoy	HP-3.11	2.4.1	Fish ingestion rate	Conservatisms	
LeRoy	HP-3.12	General	PCB concentrations	Decline with time	Risk management
LeRoy	HP-3.13	General	Community studies	Cancer	
LeRoy	HP-3.14	General	Health advisories	Public education	Risk management
GE	HG-1.1	4	Exposure parameters	High End	Unrealistic
GE	HG-1.2	General	Exposure assumptions	Toxicity assumptions	Unrealistic
GE	HG-1.3	2.3	Baseline modeling	Uncertainties	Unreviewed
GE	HG-1.4	General	Probabilistic analysis	Point estimate	Mid-Hudson
GE	HG-1.5	1.2	Risk assessment	Mid-Hudson	Definition of site area
GE	HG-1.6	3	PCB Toxicity	Animal studies	Epidemiological studies
GE	HG-1.7	3	Kimbrough study	Critique	Cancer slope factor
GE	HG-1.8	2.4.1	Fish consumption rates	Connelly survey	Flaws
GE	HG-1.9	2.4.1	Cooking loss	Probability distribution	Monte Carlo
GE	HG-1.10	2.3.1	Species preference	Connelly survey	Barclay data
GE	HG-1.11	3.1	PCB RfD	Re-evaluation	Uncertainty factors
GE	HG-1 12	3.1	PCB RfD	Uncertainty	Probability distribution
GE	HG-1.13	4.2	Kimbrough study	Critique	Cancer slope factor
GE	HG-1.14	1.2	Risk assessment	Mid-Hudson	Definition of site area
GE	HG-1.15	2.3	Baseline modeling	Predicted PCB conc.	Flaws
GE	HG-1.16	General	Probabilistic assessment	Mid-Hudson HHRA	Monte Carlo
GE	HG-1.17	General	Overview	Comments	Risk assessment
GE	HG-1.18	General	Probabilistic assessment	Mid-Hudson HHRA	Monte Carlo
GE	HG-1.19	3	Kimbrough study	Critique	Re-evaluation
GE	HG-1.20	3	PCB Toxicity	Risks	Overestimate
GE	HG-1.21	General	Exposure assumptions	Risks	Overestimate

Responses

II. RESPONSES TO COMMENTS ON THE MID-HUDSON HHRA

Responses to General Comments

Response to HL-1.4, HL-1.6, HL-1.7, HL-1.11

These comments refer to comments previously submitted on the Upper Hudson HHRA (USEPA, 1999b), that are also applicable to the Mid-Hudson HHRA (USEPA, 1999a). These comments are addressed in the March 2000 Responsiveness Summary for the Upper Hudson HHRA (USEPA, 2000a) and are not repeated here. The reader is referred to pp. 13, 19-22, 26-37, and 41-46 of the March, 2000 Responsiveness Summary for the Upper Hudson HHRA (USEPA, 2000a).

Response to HG-1.2, HG-1.17, HG-1.21

Consistent with the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) (USEPA, 1990) and USEPA policy and guidance (USEPA, 1989a, 1989b, 1991a, 1992, 1995, 1996, and 1997a), the exposure parameters used in the Mid-Hudson 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 Mid-Hudson HHRA. The RME is the maximum exposure that is reasonably expected to occur in the Mid-Hudson River under baseline conditions (e.g., no active remediation of the PCB-contaminated sediments in the Upper Hudson River and no institutional controls, 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, 1989a, 1989b, 1991a, 1997a).

The fish ingestion rates and exposure durations for the Mid-Hudson HHRA 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 six counties surrounding the Mid-Hudson River (see, p. 13, Mid-Hudson HHRA, USEPA, 1999a). The fraction from source was assumed to be 1 (i.e., 100%) (see, pp. 12-13, Mid-Hudson HHRA, USEPA, 1999a), which is reasonable given the length (90 miles) of the Mid-Hudson River and the variety of fish species it can support. The concentrations of PCBs in fish beginning in 1999 were based on modeled PCB concentrations in fish, summarized in the Baseline Ecological Risk Assessment for Future Risks in the Lower Hudson River (ERA Addendum, USEPA, 1999c). The modeled concentrations were subsequently updated for this Responsiveness Summary based on those summarized in the Responsiveness Summary for the ERA Addendum (USEPA, 2000d). The forecast results were based on upstream PCB boundary loads presented in the Revised Baseline Modeling Report (USEPA, 2000b). The toxicity values were taken from USEPA's Integrated Risk Information System or IRIS, which is USEPA's consensus database of toxicity values and 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 of the Upper Hudson HHRA (USEPA, 1999b), and Responsiveness Summary for HHRASOW (USEPA, 1999d), pp. 25-26).

Response to HG-1.4, HG-1.16, HG-1.18

Although a Monte Carlo analysis was originally planned for the Mid-Hudson HHRA (as discussed in the HHRA Scope of Work, USEPA, 1998b), it was subsequently deemed unnecessary. The PCB concentrations in the Mid-Hudson River are lower than the Upper Hudson River, the shape of the exposure distributions for the Mid-Hudson HHRA would be expected to be the same as or similar to those used in the Upper Hudson HHRA, and the results from the Upper Hudson HHRA Monte Carlo analysis were consistent with the point estimate results.

A point estimate approach is not the equivalent of a screening level approach. A point estimate approach can be and was used to develop valid central tendency and high-end estimates of exposure, non-cancer hazards, and cancer risks, and is a common risk assessment practice, consistent with USEPA policy (USEPA, 1989b). While a Monte Carlo analysis can be a useful tool, USEPA guidance does not require the use of a Monte Carlo analysis (USEPA, 1997b).

Note that as recognized in the footnote in comment HG-1.16, there is a typographical error in the last paragraph of Section 4.2 of the Mid-Hudson HHRA (USEPA, 1999a). The statement should read "The cancer risks associated with RME fish ingestion exceed the cancer risk range generally allowed under federal Superfund law."

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 (USEPA, 1999e). Both the Rogers Island and the Mid-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, 1989a, 1989b, 1991a, 1992, 1995, 1996, and 1999f-h). 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 Mid-Hudson River risk assessment evaluated recreational exposure only, over a 90-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 to 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 Regional and Headquarters USEPA staff) recommended a skin adherence factor of 1 mg/cm^2 for adults and children (based on Duff and Kissel, 1996, based on a monolayer). 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 Mid-Hudson River risk assessment (USEPA, 1999i, based on a review and analysis of a number of recent soil adherence studies).

Response to HS-1.7

The comment is acknowledged. The Mid-Hudson HHRA (USEPA, 1999a) calculated increased cancer risk to an adult eating fish (RME) of 4×10^{-4}. However, for purposes of risk communication, the risk was presented in the Executive Summary as its mathematical equivalent of four additional cancers in 10,000 exposed people. Note that based on the Mid-Hudson HHRA revisions (Section III of this report, Table 4-21-RME), the cancer risk to an RME individual (child, adolescent, then adult) eating fish is estimated to be 7×10^{-4}.

Response to HL-1.1

Copies of all USEPA reports relating to the Hudson River PCBs RI/FS, including all modeling reports, are available for public review at the 16 information repositories.

Response to HP-1.1, HP-1.2, HP-1.4, HP-1.5, HP-1.6, HP-2.1, HP-2.2, HP-2.4

These comments pertain to risk management decisions, which are outside the scope of the Mid-Hudson HHRA. The role of the baseline risk assessment is to evaluate current and future risks associated with the site and inform decisions regarding remediation in the FS. Remediation goals (including the relevance of the FDA limit in setting remediation goals), remedial alternatives, and the timeframe for cleanup will be addressed as part of the upcoming FS and Proposed Plan.

Response to HP-3.2, HP-3.5, HP-3.9, HP-3.13

The performance of community health-based epidemiological studies, as suggested in the comment, is beyond the scope of USEPA's Mid-Hudson HHRA, and is more appropriately addressed by NYSDOH and ATSDR. As indicated during USEPA's presentation of the Mid-Hudson HHRA on January 11, 2000, USEPA is aware that NYSDOH is conducting a study of individuals living in Hudson Falls, NY (and Glens Falls, NY as a control) to understand the potential impact of PCBs on neurological functions in adults. The NYSDOH research project, "PCBs and Health: The Hudson River Communities Project," is anticipated to be completed in 2001. Upon completion, USEPA will review the results of these studies.

In its draft Toxicological Profile, ATSDR states that it is not known whether PCBs cause cancer in people, but that PCBs have been shown to cause cancer in animal studies (ATSDR, 1999). Note, however, that ATSDR's draft Toxicological Profile for PCBs is currently being revised based on external comments and the results of a peer review of the document. The USEPA and the International Agency for Research on Cancer have classified PCBs as a probable human carcinogen.

Response to HP-3.3, HP-3.12

The PCB concentrations have declined with time. The models used to derive the exposure point concentrations for the Mid-Hudson HHRA predict a decline in future concentrations. Thus, the exposure point concentrations used in the Mid-Hudson HHRA reflect this expected decline over time and with distance down river.

Response to HP-3.4, HP-3.6

There are numerous fish consumption advisories currently in effect in New York State, including a general, state-wide advisory as well as advisories specific to certain water bodies. This fact does not affect the Mid-Hudson HHRA, because in performing a baseline risk assessment of current and future exposure (*i.e.*, assuming no remediation or institutional controls), USEPA does not consider the effects of fish consumption advisories.

Response to HP-3.7, HP-3.14

The USEPA Office of Research and Development (ORD) supports research to improve risk assessment and the New York State Department of Environmental Conservation monitors contaminant

levels in fish and provides data to the Department of Health (NYSDOH).

The importance of public health education is acknowledged. However, the Mid-Hudson HHRA was performed to evaluate health risks under baseline conditions (i.e., assuming no active remediation of the PCB-contaminated sediments and no institutional controls, such as the fish consumption advisories currently in place). Although USEPA believes that consumption advisories can be effective in limiting exposure to PCBs in Hudson River fish, there is no guarantee that all anglers will abide by the consumption advisories. Several studies provide evidence that a percentage of the angling community may not follow fish consumption advisories, and may continue to consume fish from rivers with fish consumption advisories (Barclay, 1993; NYSDOH, 1999; Connelly *et al.*, 1992; Connelly *et al.*, 1996).

1. OVERVIEW OF MID-HUDSON HHRA

1.1 Introduction

No significant comments were received on Section 1.1.

1.2 Site Background

Response to HF-1.1

USEPA has previously responded to public comment regarding its decision to quantify cancer risks and non-cancer hazards to individuals in 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, 1999d, Responsiveness Summary for the HHRA 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 and Mid-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 site-related PCBs found in fish, water and sediments in the Upper and Mid-Hudson River compared to those in the Lower Hudson River.

Response to HG-1.5, HG-1.14

USEPA has previously responded to comments regarding the extent of the site in the Responsiveness Summary for the HHRA Scope of Work (USEPA, 1999d, pp. 14-15) and the Responsiveness Summary for the Upper Hudson HHRA (USEPA, 2000a, p. 15). The listing of the Hudson River PCBs Site on the National Priorities List (NPL) is not limited to the Upper Hudson; the Lower Hudson clearly is within the "broad compass" of the NPL listing because it is within the areal extent of contamination resulting from the discharge of PCBs to the Upper Hudson River. See Washington State Dept. of Transportation v. EPA, 917 F.2d 1309, 1311 (D.C. Cir. 1990). See also Eagle-Picher Industries v. EPA, 822 F.2d 132 (D.C. Cir.1987).

Moreover, USEPA has consistently defined the site to include the Lower Hudson River since at least April 1984, when the Agency issued its FS for the site and before the site was listed on the NPL (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 (USEPA, 1984). In addition, during the Reassessment RI/FS, USEPA has consistently defined the site as including the Upper and Lower River (*e.g.*, USEPA, 1991b).

USEPA disagrees with the commenter's suggestion that it would be inappropriate for USEPA to consider benefits to the Lower River that may accrue from remediation in the Upper Hudson. Throughout the Reassessment RI/FS, USEPA has maintained – and continues to maintain – that the purposes of the Reassessment RI/FS include an evaluation of the impacts that PCB-contaminated Upper River sediments have on the Site, including the Lower River, and an evaluation of remedial options for the Upper River in light of those impacts, among other factors. USEPA is not at this time evaluating remedial options for the Lower River.

1.3 General Risk Assessment Process

No significant comments were received on Section 1.3.

1.4 Discussion of 1991 Phase 1 Risk Assessment

No significant comments were received on Section 1.4.

1.5 Objectives of Phase 2 Risk Assessment

No significant comments were received on Section 1.5.

2. EXPOSURE ASSESSMENT

2.1 Exposure Pathways

2.1.1 Potential Exposure Media

No significant comments were received on Section 2.1.1.

2.1.2 Potential Receptors

Response to HS-1.5, HS-1.9

Cancer risks and non-cancer hazards using child-specific (ages 1-6) values for all input parameters are presented in the Risk Assessment Revisions (Section III of this report and associated tables). For example, the following exposure assumptions were made for the RME young child: an average daily fish ingestion rate of 10.6 g/day (based on a child meal size of 76 grams, or 2.7 ounces), the high-end PCB concentration in fish (1.4 mg/kg), an exposure frequency of 365 days, an exposure duration of 6 years (ages 1-6 years), and a body weight of 15 kg (or 33 pounds, the average body weight for male and female children aged 1 to 6, USEPA, 1989a). The chronic (i.e., 7 years or more) Reference Dose was used to be protective of children (USEPA, 1993). The resulting RME cancer risk for a child ingesting fish was approximately 2×10^{-4} (2 additional cancers in 10,000 children exposed), compared to the RME total cancer risk for adult, adolescent, and child of 7×10^{-4} (7 additional cancers in 10,000 exposed individuals). The RME non-cancer hazard index for a child ingesting fish was approximately 49, compared to the RME adult non-cancer hazard index of 32 and the RME adolescent non-cancer hazard index of 35. The Mid-Hudson HHRA is amended to reflect this additional information.

Note that this assessment assumed that a young child meal portion is approximately 1/3 that of an adult (227 grams for adults, 76 grams for children). This assumed ratio (0.33) is only slightly less than the 0.36 ratio recommended by the commenter. The assumed child portion size, 76 grams or 2.7 ounces,

falls between the mean fish meal sizes reported by the USEPA for children less than five years old and children aged 6 to 11 years old (67 grams [2.4 ounces] and 89 grams [3.1 ounces], respectively) (USEPA, 1997a).

2.1.3 Potential Exposure Routes

Response to HS-1.1

Consistent with the focus of the Reassessment RI/FS, the Mid-Hudson HHRA calculated cancer risks and non-cancer hazards associated with exposure to PCBs in the sediments, water and fish in the Mid-Hudson River. As discussed in the Upper Hudson HHRA (USEPA, 1999b, 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, and the collection of additional PCB data from vegetables, meat, eggs, and milk is not warranted. Therefore, the Mid-Hudson HHRA does not quantify cancer risks and non-cancer hazards due to uptake of PCBs *via* floodplain soils or the other residential pathways identified (see, p. 6, Mid-Hudson HHRA, USEPA, 1999a).

2.2 Quantification of Exposure

No significant comments were received on Section 2.2.

2.3 Exposure Point Concentrations

Response to HF-1.2, HF-1.3, HL-1.2, HL-1.3, HL-1.5, HG-1.3, HG-1.15

These comments refer to the PCB modeling efforts for fish, water, and sediments. The fate and transport and bioaccumulation models are presented in the Revised Baseline Modeling Report (RBMR) (USEPA, 2000b), and the ERA Addendum (USEPA, 1999c), which contains a summary of the Farley model results. Issues relating to these modeling efforts are addressed in the above referenced reports and their Responsiveness Summaries (USEPA, 2000c; USEPA, 2000d). In addition, the RBMR underwent independent peer review and the majority of the reviewers found the report acceptable with minor revisions (ERG, 2000).

USEPA reviewed the Farley model for use in the ERA Addendum (USEPA, 1999c). The data set available to calibrate a PCB fate and transport model in the Lower Hudson is limited. However, as discussed in the Responsiveness Summary for the ERA Addendum (USEPA, 2000d), other data and analyses independently confirm the conclusions drawn from the Farley modeling analysis. For example, the conclusion that the principal source of PCBs to the Lower Hudson is the Upper Hudson is directly supported by the high-resolution core analysis presented in the Data Evaluation and Interpretation Report (USEPA, 1997c). Similarly, the gradual decline in PCB concentration estimated by the model is supported by the analysis of the high-resolution cores presented in the Data Evaluation and Interpretation Report (USEPA, 1997c). Additionally, earlier versions of the Farley model developed by Thomann and others were peer reviewed and published. It is USEPA's understanding that the authors of the most recent version of the Farley model will submit it for publication in a peer reviewed scientific journal.

Cancer risks and non-cancer hazards to human health for the Mid-Hudson have been revised based on supplemental analyses of the fate and transport and bioaccumulation models. These results for the Mid-Hudson are presented in Section III of this Responsiveness Summary. In general, the overall conclusions from the December 1999 Mid-Hudson HHRA (USEPA, 1999a) remain unchanged for this

revised Mid-Hudson HHRA. The revised calculations for the Mid-Hudson HHRA show that cancer risks and non-cancer health hazards to the RME individual associated with ingestion of PCBs in fish from the Mid-Hudson River are above USEPA levels of concern. In addition, the revised calculations indicate that fish ingestion represents the primary way for people to be exposed to PCBs from the site, and that cancer risks and non-cancer hazards from other exposure pathways are generally below USEPA's levels of concern.

Waiting until after the peer review for the RBMR to use the model output would have unnecessarily delayed issuance of the risk assessments by about one year. The Upper Hudson HHRA was peer-reviewed in May 2000 and generally found to be acceptable with minor revisions. The results of the various independent peer reviews are being considered by USEPA, and the Agency will respond to the peer reviewers' recommendations in written Responsiveness Summaries. 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 HL-1.9

This comment is based on a misinterpretation of the use of the modeled river data. The modeled river data cover the 90 miles of the Mid-Hudson River; the data for a single river mile were *not* used to represent that range. Although each reach of the river was identified by the mile marker at the upstream end of the reach, USEPA used the average PCB concentration for each reach. The Mid-Hudson HHRA assumed a uniform likelihood of fishing at any location within the Mid-Hudson River study area, which is believed to be a reasonable assumption in light of the lack of any information specific to fishing practices in the area. This comment is also addressed in the Responsiveness Summary for the ERA Addendum (USEPA, 2000d).

2.3.1 PCB Concentration in Fish

Response to HF-1.5, HS-1.8

The 1991 New York Angler survey (Connelly et al., 1992) reported fish consumption for six species that are potentially caught in the Mid-Hudson River: bass, bullhead, carp, catfish, eel, and perch (Mid-Hudson HHRA, USEPA, 1999a, Table 2-5). In the ERA Addendum (USEPA, 1999c), USEPA forecast concentrations of PCBs in five fish species commonly consumed by humans: brown bullhead, largemouth bass, yellow perch, striped bass, and white perch. Other species known to exist in the Mid-Hudson region (i.e., carp, catfish, and eel) were reported in the 1991 New York Angler Survey (Connelly et al., 1992) and by Dr. Ronald Sloan of NYSDEC (R. Sloan, personal communication). Because carp, catfish, and eel were not specifically modeled, they were assigned the PCB concentration modeled for brown bullhead, which also spends much of its time at the bottom of lakes, rivers, and streams.

One commenter notes that PCB concentrations measured in 1992 in eel, carp/goldfish, and white catfish (9.1, 9.2, and 8.8 ppm, respectively) are higher than in brown bullhead (3.1 ppm). PCB concentrations (Tri+) for brown bullhead and white catfish from Release 4.1 of the Hudson database were compared. The differences between the PCB concentrations for brown bullhead and white catfish ranged a factor of two to four apart; thus, using modeled PCB concentrations for brown bullhead would underestimate PCB concentrations for carp, catfish, and eel. However, given the relatively low intake percentages for the carp, catfish, and eel (5.9%, 7.4%, and 2.5% of the total fish intake, respectively), the total cancer risks and non-cancer hazards from ingesting fish would not be substantially underestimated (see, Mid-Hudson HHRA, USEPA, 1999a, Table 2-7).

Response to HG-1.10

As discussed in the Mid-Hudson HHRA (USEPA, 1999a, pp. 10), the Mid-Hudson species preferences were based on consideration of both the 1991 New York Angler survey (Connelly et al., 1992) and the Hudson River angler surveys (Barclay, 1993; NYSDOH, 1999). There is some uncertainty associated with the species preferences used; however, this uncertainty is unavoidable. Although ascertaining species preference was not the primary purpose of these studies, there are no studies available relevant to the Hudson River that were designed specifically to determine species preferences. The results from the Hudson River angler surveys (Barclay, 1993; NYSDOH, 1999) are more difficult to interpret due to the fish consumption advisories in effect on the Hudson River, and because the studies report only the amount of each species caught, rather than the amount of each species consumed. 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 Mid-Hudson, excluding fish species not found in the Mid-Hudson, and extrapolating the percent of all fish in flowing water bodies to percent of Hudson species (discussed in more detail in the Upper Hudson HHRA, USEPA, 1999b, Table 3-3) were necessary so that the fish species percentages for the Mid-Hudson totaled 100%.

Furthermore, even if anglers were consuming a greater percentage of striped bass or large-mouthed bass, and a smaller percentage of bottom feeders (brown bullhead, carp, catfish, and eel), the total cancer risks and non-cancer hazards from ingesting fish calculated in the Mid-Hudson HHRA would not be expected to change significantly. The exposure point concentration values for striped bass, large-mouthed bass, and brown bullhead were all similar (RME EPC values were 1.2, 0.87, and 1.2 mg/kg, respectively) (Mid-Hudson HHRA revisions, Table 2-8, in Section III of this report). Because the exposure point concentration values for yellow and white perch were the lowest of the five modeled fish species, increasing the preference for yellow or white perch could potentially lower the species-weighted PCB exposure point concentrations, and the resulting total cancer risks and non-cancer hazards. Although herring and American shad are present in the Mid-Hudson, they were not evaluated in the Mid-Hudson HHRA because forecast concentrations were not available for herring or American shad.

Response to HL-1.8

This comment is addressed in the Responsiveness Summary for the ERA Addendum (USEPA, 2000d).

Response to HL-1.10

An RME value for PCB concentration in fish was used in the calculation of the RME cancer risks and non-cancer hazards. This value was calculated by averaging the species-weighted concentration distribution over the 95th percentile exposure duration estimate (*i.e.* 40 years).

Response to HP-3.8

To clarify, in the Mid-Hudson HHRA, the models that were used to derive concentrations of PCBs in fish were calibrated using PCB concentrations measured in fish fillets, skin on. Therefore, the modeled PCB concentrations represent the edible tissue, and not PCB concentrations in whole fish.

2.3.2 PCB Concentration in Sediment

No significant comments were received on Section 2.3.2

2.3.3 PCB Concentration in River Water

No significant comments were received on Section 2.3.3

2.4 Chemical Intake Algorithms

Response to HF-1.4, HS-1.4

The start date for the exposure of anglers used in both the Mid-Hudson and Upper Hudson HHRAs is 1999 (Mid-Hudson HHRA, USEPA, 1999a, pp. 8-11; see also, USEPA, 1999d, Responsiveness Summary for the HHRASOW, pp. 28 and 29). This is consistent with the goals of the Mid-Hudson HHRA because the Mid-Hudson HHRA evaluates current and future risk, and 1999 is the year in which the Mid-Hudson HHRA was completed. Use of a start date before 1999 would not be consistent with USEPA risk assessment guidance (USEPA, 1989b). In addition, 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; this is because data are not available to determine the appropriate dosimetric for PCB carcinogenicity based on existing PCB body burdens (USEPA, 1996b) (see also, Responsiveness Summary for the HHRA Scope of Work, USEPA, 1999d, p. 28). Therefore, although past exposures are a source of uncertainty, this issue is not addressed quantitatively in the Mid-Hudson HHRA.

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, 1989b) and is more representative of a "worst case" exposure scenario than an RME scenario. The 40-year exposure duration used for the RME scenario is based on a reasonable use of site-specific information. 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, 1989b; USEPA 1997a, as cited in Upper Hudson HHRA, p. 57).

2.4.1 Ingestion of Fish

Response to HG-1.8

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 Upper Hudson HHRA, USEPA compared the central (or average) and high-end fish ingestion rates used in the Mid-Hudson and Upper Hudson HHRAs to the surveys identified in the comment, including the 1993 Maine Angler survey (Ebert et al., 1993), the 1992 Lake Ontario diary study (Connelly et al., 1996), and other surveys (see Upper Hudson HHRA, USEPA, 1999b, p. 44 and Table 3-2). The fish ingestion rates used in the Mid-Hudson 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) (Upper Hudson HHRA, USEPA, 1999b, 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 Upper Hudson HHRA (USEPA, 1999b, p. 42). The specific concerns about the 1991 New York Angler survey raised by the commenter, such as the survey response rate, long-term recall bias, and meal size assumptions, are discussed in Section 3.2.1 of the Upper Hudson HHRA (USEPA, 1999b). Furthermore, the results of the sensitivity

analysis for fish ingestion rate in the Upper Hudson HHRA indicate that adopting a lower fish ingestion rate does not change the results significantly.

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 fish consumption health advisories) (Upper Hudson HHRA, USEPA, 1999b, p. 39). Of the available studies of sport fish ingestion, the 1991 New York Angler survey is considered the preferred study to represent Mid-Hudson River anglers because, among other reasons, it was conducted in New York, included the fish species of concern in the Hudson River, included water bodies with no fish consumption advisories, and included a large sample size (Upper Hudson HHRA, USEPA, 1999b, p. 73).

Response to HG-1.9

In the Upper Hudson HHRA, USEPA summarized laboratory studies of fish preparation and cooking methods conducted to quantify the extent of PCB loss prior to consumption (Upper Hudson HHRA, USEPA, 1999b, Table 3-5). Many of the fish species used in these studies are not found in the Upper (or Mid-) Hudson River. Moreover, the studies were conducted over a period of more than 20 years, and the results may not be comparable to one another due to developments in the sampling 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 (Upper Hudson HHRA, USEPA, 1999b, 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.

Response to HP-1.3, HP-2.3

Consistent with the NCP and USEPA risk assessment guidance (USEPA, 1989B), the Mid-Hudson HHRA evaluates both current and future cancer risks and non-cancer hazards in the absence of any remedial action or institutional controls, such as the fish consumption advisories currently in place (Mid-Hudson HHRA, USEPA, 1999a, p. ES-1).

Response to HP-3.1, HP-3.11

As stated in the Mid-Hudson HHRA, the RME fish ingestion rate used in the Mid-Hudson HHRA was 31.9 g/day, which corresponds to approximately one half-pound fish meal per week. This value is based on the 90th percentile fish ingestion rate in the 1991 New York Angler survey (Mid-Hudson HHRA, USEPA, 1999a, Table 2-19). The RME exposure duration of 40 years is based on the 95th percentile of the fishing duration distribution, generated based on the 1991 New York Angler survey and 1990 population mobility data from the U.S. Bureau of Census (Mid-Hudson HHRA, USEPA, 1999a, Table 2-19). The goal of the selection of the fish ingestion rate is to represent a reasonable maximum exposure for current and future exposures and be protective of human health (USEPA, 1989b, 1990, 1992). Using data from Connelly *et al.* (1992) survey that represents fish ingestion by high-end anglers within New York State achieves this goal.

2.4.2 Ingestion of Sediment

No significant comments were received on Section 2.4.2.

2.4.3 Dermal Contact with Sediment

No significant comments were received on Section 2.4.3.

2.4.4 Dermal Contact with River Water

No significant comments were received on Section 2.4.4.

2.4.5 Ingestion of River Water

No significant comments were received on Section 2.4.5.

3. TOXICITY ASSESSMENT

Response to HG-1.6, HG-1.7, HG-1.19, HG-1.20

Consistent with USEPA risk assessment policy and guidance (USEPA, 1996a, 1992), the Mid-Hudson HHRA uses the current toxicity values in IRIS, the Agency's consensus database of toxicity values. USEPA's evaluations of cancer risks and non-cancer health effects of PCBs were externally peer-reviewed and went through internal Agency consensus review before inclusion in IRIS. The IRIS cancer slope factors were developed during USEPA's 1996 reassessment of PCB carcinogenicity (USEPA, 1996b) and are based on a number of published studies that evaluate the carcinogenic potential of PCBs in both humans and animals. USEPA is currently reassessing the non-cancer toxicity values 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. The results of this Agency reassessment of non-cancer toxicity values are expected in 2001. Consistent with risk assessment policy and guidance, USEPA considered relevant new toxicological information prior to using the existing IRIS toxicity values in the Mid-Hudson HHRA (USEPA, 1999h, Upper Hudson HHRA, USEPA, 1999b, Appendix C, pp. C-1 to C-6).

USEPA used a weight-of-evidence approach to evaluate PCBs (USEPA, 1996b, 1999f-h). 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.), as described in the IRIS Weight of Evidence classification (USEPA, 1996, 1999h). Based on this information, USEPA concluded that the available evidence from human studies is inadequate, but suggests that exposure to PCBs can cause cancer. The expert panel convened for the reassessment of the PCB cancer slope factors (USEPA, 1996b) did not recommend that the epidemiological studies be used to derive CSFs for PCBs, noting inadequacies 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, 1999h). 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 used in the non-cancer assessment are available on USEPA's web site at www.epa.gov/iris/subst/0294.htm and www.epa.gov/ncea/pcbs.htm.

Consistent with USEPA risk assessment policy and guidance (USEPA, 1992; 1996b), the Upper Hudson HHRA also contains a summary of the results of the Kimbrough *et al.* (1999a) study and the USEPA's preliminary analysis of the data and its effect on the characterization of the carcinogenicity of PCBs (see, Upper Hudson HHRA, USEPA, 1999b, pp C2-C3). USEPA has not developed a new CSF for PCBs based on the Kimbrough et al. (1999a) study or any of the other human epidemiological studies because of their inadequacies and limitations as described in the IRIS file. 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, 1999h; USEPA, 1996b).

Response to HS-1.13

In the Mid-Hudson HHRA, USEPA used the current toxicity values in IRIS. As mentioned in Chapter 3 of the Mid-Hudson HHRA (USEPA, 1999a, p. 18), the Upper Hudson HHRA provides an overall discussion on the toxicity of PCBs and identifies some additional information available since USEPA last reassessed cancer toxicity and non-cancer toxicity. In particular, the Upper Hudson HHRA noted the two studies (i.e., Arnold et al., 1995; Rice, 1999) that were mentioned by the commenter (see, Upper Hudson HHRA, USEPA, 1999b, pp. 76-77 and C-4 to C-6). The USEPA is currently reassessing the non-cancer toxicity values for PCBs on an Agency-wide basis, with the results of this reassessment expected in 2001. This reassessment will evaluate the studies mentioned in the comment along with the other available human and animal studies, evaluate the appropriate application of uncertainty factors, and determine whether the RfDs require modification.

3.1 Non-cancer Toxicity Values

Response to HS-1.14

As mentioned in Chapter 3 of the Mid-Hudson HHRA (USEPA, 1999a, p. 18), the critical studies, critical effects, and uncertainty factors for the RfDs for Aroclor 1016 and Aroclor 1254 are discussed in the Upper Hudson HHRA (see, USEPA, 1999b, pp. 62 and C5-C6).

Response to HS-1.15

The Mid-Hudson HHRA did not include a Toxicological Profile for PCBs, but referenced the Toxicological Profile in the Upper Hudson HHRA Appendix C (USEPA, 1999b). This comment regarding information in the Toxicological Profile in the Upper Hudson HHRA (Appendix C) was addressed in the Responsiveness Summary for the Upper Hudson HHRA (USEPA, 2000a).

In the Upper Hudson HHRA, USEPA used the current toxicity values in IRIS. The Upper Hudson 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, 1999f-h). USEPA is currently reassessing the non-cancer toxicity values for PCBs on an Agency-wide basis, with completion expected in 2001. PCB non-cancer toxicity and carcinogenicity is recognized as an area of widespread research, and many articles on PCB non-cancer toxicity and carcinogenicity have been published recently. Nonetheless, it is beyond the scope of the HHRAs for the Hudson River PCBs site to present a detailed evaluation of all the available scientific literature on PCBs, particularly in view of ongoing Agency-wide reassessment of the non-cancer toxicity values. However, USEPA is continually reviewing and evaluating new studies and research as they are published. The comment regarding the Lanting/Patandin studies is acknowledged.

Response to HG-1.11

Consistent with the hierarchy of toxicity information recommended in USEPA risk assessment guidance (USEPA, 1989b), USEPA used the current toxicity values from IRIS in the Mid-Hudson HHRA. USEPA is currently reassessing the non-cancer toxicity values for PCBs on an Agency-wide basis, with the results of this reassessment expected in 2001. This reassessment will evaluate the studies mentioned in the comment along with the other available human and animal studies, evaluate the appropriate application of uncertainty factors, and determine whether the RfDs require modification.

As discussed in the Responsiveness Summary for the HHRA Scope of Work (USEPA, 1999d, pp. 26-27), the health effects in Rhesus monkeys (used as the basis for USEPA's RfD for Aroclor 1254) are relevant to assessing human noncancer risks. Today, similar tests to determine serum IgG and IgM levels are widely used in hospitals and clinical laboratories to diagnose immune deficiencies in suspected immuno-compromised patients (Bakerman, 1994, ABC's of Interpretative Laboratory Data, 3rd edition, Interpretive Laboratory Data, New York). Animal or human IgG and IgM antibody responses to sheep red blood cells or similar multi-antigens systems are routinely and widely used in defining immunocompromised diseases. In addition, the toxicology research community, as evidenced by presentations and audience attendance at immunotoxicology sessions of the annual Society of Toxicology meetings, has expanded its presentations and acceptance of immunotoxicology papers that use similar methods from a wide variety of animal research studies (e.g., Proceedings of the Society of Toxicology Meeting, New Orleans, LA, March, 1999).

The fact that the dermal and ocular effects observed in Rhesus monkeys have not been observed in humans may be due to the well-controlled dosing of the monkeys, whereas the exposure in the human epidemiological studies is not well characterized. With regard to metabolism of PCBs in Rhesus monkeys and humans, USEPA notes that slight differences in metabolic processes have been observed by one research group, but that differences in the critical adverse effects have not been demonstrated by other research groups.

Response to HG-1.12

USEPA did not conduct a Monte Carlo Analysis for the Mid-Hudson HHRA (see responses to HG-1.4, HG-1.16). Regardless, had a Monte Carlo analysis been performed, at present it is USEPA policy to perform a Monte Carlo analysis using distributions only for exposure parameters, while using IRIS values for toxicity parameters (USEPA, 1997b). This approach is consistent with other risk assessments performed by USEPA for other sites as the Agency continues to evaluate the science associated with developing distributions for toxicity values.

The USEPA RfD values were derived to be protective of human health. Uncertainties associated with non-cancer toxicity values were qualitatively addressed in the Upper Hudson HHRA in the Toxicity Assessment (see, Upper Hudson HHRA, USEPA, 1999b, pp. 61-62 and 65-66), the uncertainty section of the risk characterization (see, Upper Hudson HHRA, USEPA, 1999b, pp. 35 and 76-77), and Appendix C: PCB Toxicological Profile (see, Upper Hudson HHRA, USEPA, 1999b, p. C-5). Uncertainties in the non-cancer toxicity values could result in an over- or under-estimation of non-cancer hazards.

3.2 PCB Cancer Toxicity

Response to HS-1.12

In the Mid-Hudson HHRA, USEPA selected cancer slope factors based on the environmental medium being evaluated, which is consistent with IRIS and current USEPA guidance (USEPA, 1996b; USEPA, 1999 f-h). 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 Upper or Mid-Hudson River) or to conduct a supplemental analysis of dioxin TEQs (which was performed in the Upper Hudson HHRA) (see, USEPA, 1999b, pp. 69-70).

4. Risk Characterization

Response to HS-1.10, HP-3.10

The modeled PCB concentrations, by species and location, are shown in Figures 2-1 through 2-10. Consistent with USEPA guidance, the Mid-Hudson HHRA calculated 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 of the Upper Hudson HHRA (USEPA, 1999b, p. C-7).

The FDA tolerance level for PCBs in fish was based on weighing the results of a risk assessment against the magnitude of potential food loss resulting from a lowered tolerance level. The FDA risk assessment was performed assuming that the tolerance level of 2 ppm would be the maximum PCB concentration encountered by a frequent commercial fish consumer, and that PCB concentrations in commercial fish consumed would be distributed below 2 ppm in a manner reflecting a mix of fish from diverse sources. This methodology precludes application of the FDA tolerance level to the Mid-Hudson HHRA for fish ingestion. The FDA specifically states that the tolerance level is intended to apply to fish entering interstate commerce, and that this level may not be protective for locally caught fish from contaminated areas. Note that the FS will contain a discussion and determination of applicable or relevant and appropriate requirements of federal and state environmental laws.

Response to HG-1.1

The Mid-Hudson HHRA found cancer risks and non-cancer hazards for recreational (wading and swimming) and residential (consuming river water) exposure pathways to be below levels of concern. However, the cancer risks and non-cancer hazards presented for ingestion of fish, for both the central tendency (average fish consumption rate) and the high-end estimate, are above USEPA's levels of concern (see, Mid-Hudson HHRA revisions, Section III of this report).

Consistent with the NCP (USEPA 1990) and USEPA policy and guidance (USEPA, 1989a, 1989b, 1991a, 1992, 1995, 1996a, and 1997a), the exposure parameters used in the Mid-Hudson HHRA are appropriately protective of human health and do not reflect "a combination of unrealistic circumstances," as claimed by the commenter. Specifically, USEPA evaluated both high-end (RME) and central tendency exposure (average) cancer risks and non-cancer hazards in the Mid-Hudson HHRA. The RME is not a worst case scenario and 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, 1989a, 1989b, 1990b).

In the Mid-Hudson HHRA, it was not assumed, as claimed in the comment, that anglers ate the same species of fish (eel and carp) from the same part of the river. The cancer risks and non-cancer hazard assessment for ingestion of fish, for the high-end estimate, assume consumption of a number of different fish species (only 2.5% eel and 5.9% carp). In addition, PCB concentrations were averaged over all locations in the Mid-Hudson, assuming a uniform likelihood of fishing at any location within the Mid-Hudson River (Mid-Hudson HHRA, p. 9).

4.1 Non-cancer Hazard Indices

No significant comments were received on Section 4.1.

4.2 Cancer Risks

Response to HS-1.6

The statements in the Mid-Hudson HHRA regarding the acceptable risk range are drawn from the NCP 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" (USEPA, 1990).

Response to HG-1.13

USEPA performed a preliminary review of the Kimbrough *et al.* (1999a) study and identified aspects of the study (discussed in the Upper Hudson HHRA, USEPA, 1999b, pp. C2-C3) that limit its usefulness for Superfund risk assessments. The primary limitation, which is shared by other similar epidemiological studies, is that the degree of exposure is not well characterized. Other scientists have identified this and other limitations of the Kimbrough *et al.* (1999a) study (see, Bove et al., 1999; Frumkin and Orris, 1999, see also Kimbrough et al., 1999b).

Based on the limitations of the Kimbrough *et al.* (1999a) study, USEPA expects that the study will not provide sufficient information to change the Agency's conclusions regarding the weight of evidence of the human PCB data or the health effects of PCBs in general. For these reasons, in the Mid-Hudson HHRA, USEPA used the IRIS cancer slope factors and did not attempt to develop new cancer slope factors based on the Kimbrough et al. (1999a) study.

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Risk Assessment
Revision

III. RISK ASSESSMENT REVISIONS

1. SUMMARY

This section of the Responsiveness Summary presents the revised baseline Human Health Risk Assessment results for the Mid-Hudson River (Mid-Hudson HHRA). The revision reflects sediment, water column, and bioaccumulation modeling as summarized in the Baseline Ecological Risk Assessment for Future Risks in the Lower Hudson River (ERA Addendum, USEPA, 1999c) and the Responsiveness Summary for the ERA Addendum (USEPA, 2000d), which in turn result from the revised PCB boundary load into the Lower Hudson River that was presented in the Revised Baseline Modeling Report (RBMR) (USEPA, 2000b). This section also compares the revised cancer risks and non-cancer hazards and associated conclusions with those of the December 1999 Mid-Hudson HHRA.

The overall conclusions from the December 1999 Mid-Hudson HHRA (USEPA, 1999a) remain unchanged. The revised calculations for the Mid-Hudson HHRA show that cancer risks and non-cancer health hazards to the reasonably maximally exposed (RME) and central tendency (CT) individuals associated with ingestion of PCBs in fish from the Mid-Hudson River are above USEPA levels of concern. In addition, fish ingestion represents the primary pathway for PCB exposure and for potential adverse health effects, whereas the risks and hazards from other exposure pathways are below levels of USEPA concern.

1.1 Introduction

Part III of this Responsiveness Summary summarizes the modifications made to the exposure parameter estimates and presents the results of the revised risk calculations for the Mid-Hudson HHRA. All tables and figures contained in the December 1999 Mid-Hudson HHRA are presented herein. 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 December 1999 Mid-Hudson HHRA results (USEPA, 1999a), all tables and figures have retained their number designations.

1.2 Revisions to Exposure Parameter Estimates

The only exposure parameter modifications made were to the fish, sediment, and river water exposure point concentrations (EPCs). The revised EPCs were calculated using the forecasts from the revised bioaccumulation and fate and transport models, as presented in the ERA Addendum (USEPA, 1999c) and the Responsiveness Summary for the ERA Addendum (USEPA, 2000d). The revised model forecasts were based on revised PCB loads to the Lower Hudson as summarized in the RBMR (USEPA, 2000b).

In addition, to estimate cancer risks and non-cancer hazards to children for the fish ingestion pathway, USEPA has added separate calculations for a young child and an adolescent, based on age-appropriate ingestion rates and body weights.

1.2.1 Fish

Revised Tri+ PCB annual averages for brown bullhead, yellow perch, largemouth bass, striped bass, and white perch are summarized in the Responsiveness Summary for the ERA Addendum (USEPA, 2000d). Consistent with the December 1999 Mid-Hudson 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 to the December 1999 EPCs is shown in the box below. In general, the revised forecast PCB concentrations in the largemouth bass, striped bass, and white perch declined from the earlier results, while the concentration in brown bullhead and yellow perch increased. When averaged over the three locations, the RME concentration increase is approximately 1.5-fold for brown bullhead and 1.1-fold for yellow perch. The RME concentration for largemouth bass, striped bass, and white perch decreased by 5%, 14%, and 7%, respectively. The species weighted RME (40-year) concentration in fish increases from 0.8 mg/kg in the 1999 Mid-Hudson HHRA, to 1.0 mg/kg, approximately a 1.25-fold increase. A discussion of the reasons for the change in the forecasts is provided in the Responsiveness Summary for the ERA Addendum (USEPA, 2000d).

Comparison of 1999 and Revised PCB Concentration in Fish (mg/kg)
Reasonable Maximum Exposure (RME) Over 40 Years

	River Mile 152		River Mile 113		River Mile 90		RME Average Over 3 Locations[2]	
Fish	**1999**	**Revised**	**1999**	**Revised**	**1999**	**Revised**	**1999**	**Revised**
Brown Bullhead	0.96	1.4	0.79	1.2	0.61	0.89	0.79	1.2
Yellow Perch	0.38	0.45	0.31	0.33	0.24	0.25	0.31	0.34
Largemouth Bass	1.4	1.0	1.1	0.90	0.26	0.68	0.92	0.87
Striped Bass	3.6	2.6	0.56	0.47	0.13	0.35	1.4	1.2
White Perch[1]	NA	NA	NA	NA	NA	NA	0.61	0.57

[1] *White Perch were modeled over the entire Mid-Hudson region in the Farley model; thus, concentrations were not predicted at specific River Miles.*
[2] *As summarized in Table 2-8.*

1.2.2 Sediment and River Water

The Responsiveness Summary for the ERA Addendum provides revised forecasts of Total PCB annual averages in sediment and river water for the Mid-Hudson River (USEPA, 2000d). As was the case for the Upper Hudson HHRA, the modeled sediment and river water data assumed a constant upstream boundary condition of 10 ng/L. PCB concentrations in sediment and river water were forecast through the year 2046. The EPCs were calculated by averaging the forecasted results over the appropriate exposure durations for adult, adolescent, and child (i.e. for the cancer assessment: 22, 12, and 6 years, respectively; and for the non-cancer assessment: 7, 7, and 6 years, respectively).

Revised sediment EPCs were approximately 1.1-fold higher than the December 1999 EPCs. Revised river water EPCs were approximately the same for central estimate EPCs, whereas the corresponding revised RME values were approximately 1.1-fold lower than the 1999 results.

2. RESULTS

For known or suspected carcinogens, such as PCBs, 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). Central tendency cancer risks are provided to more fully describe the health effects associated with average exposure.

For an individual consuming fish, the RME estimate of the increased risk of an individual (as child, adolescent then adult) developing cancer averaged over a lifetime is about 7×10^{-4}, or seven additional cancers in 10,000 exposed people. This risk is 700 times USEPA's goal of protection and 7 times greater than the highest risk level generally allowed under the federal Superfund program. The central tendency (average) estimate of risk is about 1×10^{-5}, or one additional cancer in 100,000 exposed people.

For an adult consuming fish, the RME estimate of the increased risk of an individual developing cancer averaged over a lifetime is about 3×10^{-4}, or three additional cancers in 10,000 exposed people. The central tendency (average) estimate of risk is about 6×10^{-6}, or six additional cancers in 1,000,000 exposed people.

For an adolescent consuming fish, the RME estimate of the increased risk of an individual developing cancer averaged over a lifetime is about 2×10^{-4}, or two additional cancers in 10,000 exposed people. The central tendency (average) estimate of risk is about 3×10^{-6}, or three additional cancers in 1,000,000 exposed people.

For a child consuming fish, the RME cancer risk estimate is about 2×10^{-4} or 2 additional cancers in 10,000 exposed children. The central tendency (average) estimate of risk is about 5×10^{-6} or 5 additional cancers in 1,000,000 exposed children.

Estimated cancer risks relating to PCB exposure in sediment and water while swimming or wading, or from consumption of PCBs in drinking water 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 cancer risk calculations is presented below.

Cancer Risk Summary

Pathway	Central Tendency Risk	RME Risk
Ingestion of Fish		
Total*	1×10^{-5} (1 in 100,000)	7×10^{-4} (7 in 10,000)
Adult	6×10^{-6} (6 in 1,000,000)	3×10^{-4} (3 in 10,000)
Adolescent	3×10^{-6} (3 in 1,000,000)	2×10^{-4} (2 in 10,000)
Child	5×10^{-6} (5 in 1,000,000)	2×10^{-4} (2 in 10,000)
Swimming/Wading Exposure to Sediment*	2×10^{-8} (2 in 100,000,000)	2×10^{-7} (2 in 10,000,000)
Swimming/Wading Exposure to Water*	9×10^{-9} (9 in 1,000,000,000)	5×10^{-8} (5 in 100,000,000)
Consumption of Drinking Water*	2×10^{-8} (2 in 100,000,000)	1×10^{-7} (1 in 10,000,000)

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

The evaluation of non-cancer health effects 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.

Adult ingestion of fish resulted in a Hazard Index (HI) of about 32 for the RME exposure and an HI of about 3 for the central tendency exposure. Adolescent ingestion of fish resulted in an HI of about 35 for the RME and an HI of about 4 for the central tendency exposure. Child ingestion of fish resulted in an HI about 49 for the RME exposure and an HI of about 5 for the central tendency exposure.

The total HIs for exposure to sediment and water are all below one. A summary of the estimate for non-cancer hazards is presented below.

Non-Cancer Hazard Summary

Pathway	Central Tendency Non-Cancer Hazard Index	RME Non-Cancer Hazard Index
Ingestion of Fish		
Adult	3	32
Adolescent	4	35
Child	5	49
Exposure to Sediment*	0.002	0.004
Exposure to Water*	0.005	0.007
Consumption of Drinking Water*	0.01	0.02

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

2.1 Comparison/Discussion

This revised Mid-Hudson HHRA provides separate cancer risk estimates for children (young child aged 1-6 and adolescent aged 7 to 18) based on age-appropriate exposure assumptions for ingestion rate and body weight. Previously, in the December 1999 Mid-Hudson HHRA, USEPA approximated the risk to a young child based on a fish meal size of 1/3 the adult portion.

Compared to the RME cancer risk for the adult ingesting fish that was presented in the 1999 Mid-Hudson HHRA (4×10^{-4}), the revised cancer risks for total RME (child, adolescent, then adult) ingesting fish, the pathway with the highest risks, increased approximately 1.75-fold, to 7×10^{-4}. The revised RME non-cancer hazard index for an adult ingesting fish increased approximately 1.1-fold, to 32 compared to 30 in the 1999 Mid-Hudson HHRA. This modest increase in the risk assessment results does not alter the overall conclusions for the Mid-Hudson River. That is, the revised results indicate that cancer risks and non-cancer health hazards to the RME individual associated with ingestion of PCBs in fish from the Mid-Hudson River are above USEPA levels of concern for both cancer risks and non-cancer health hazards.

The calculations show that a child consuming fish from the Mid-Hudson River would be exposed to PCBs above USEPA's levels of concern. Eating one approximately 3 ounce fish meal per week (RME exposure) would increase a child's risk of cancer by 2×10^{-4} (two additional cancers in 10,000 exposed children), which is about 200 times greater than USEPA's goal for protection. The same ingestion rate yields an HI for non-cancer health effects of 49, which is 49 times greater than USEPA's level of concern. A child eating one approximately 3 ounce fish meal every two months (central tendency, or average exposure) would result in an increased cancer risk of 5×10^{-6}, which is 5 times greater than USEPA's goal for protection. This child's fish ingestion rate would result in an HI of non-cancer health effects that is 5 times greater than USEPA's goal for protection. The risks and hazards for children exposed to PCBs from other pathways (swimming, wading, and drinking river water) are below USEPA's levels of concern.

In summary, the revised Mid-Hudson HHRA indicates that fish ingestion represents the primary pathway for children, adolescents, and adults to be exposed to PCBs and experience potential adverse health effects, whereas cancer risks and non-cancer hazards from exposure to PCBs through other exposure pathways are below USEPA levels of concern.

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TABLE 2-1 (Revised)

SELECTION OF EXPOSURE PATHWAYS – Phase 2 Risk Assessment

MID-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	Mid-Hudson Fish	Angler	Adult	Ingestion	On-Site	Quant	PCBs have been widely detected in fish
					Adolescent	Ingestion	On-Site	Quant	
					Child	Ingestion	On-Site	Quant	
	Sediment	Sediment	Banks of Mid-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	Mid-Hudson River	Resident	Adult	Ingestion	On-Site	Quant	Considered in Phase 1 Risk Assessment and determined to have de minimis risk. Included to address public concerns
					Adolescent	Ingestion	On-Site	Quant	
					Child	Ingestion	On-Site	Quant	
		River Water	Mid-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	Mid Hudson River (River and near vicinity)	Recreator	Adult	Inhalation	On-Site	Qual	Considered in Phase 2 Upper Hudson River HHRA and determined to have insignificant risk. Concentrations in Upper Hudson River approximately four times higher than Mid-Hudson region, therefore, not evaluated further in this HHRA.
					Adolescent	Inhalation	On-Site	Qual	
					Child	Inhalation	On-Site	Qual	
				Resident	Adult	Inhalation	On-Site	Qual	Considered in Phase 2 Upper Hudson River HHRA and determined to have insignificant risk. Concentrations in Upper Hudson River approximately four times higher than Mid-Hudson region, therefore, not evaluated further in this HHRA
					Adolescent	Inhalation	On-Site	Qual	
					Child	Inhalation	On-Site	Qual	
	Home-grown Crops	Vegetables	Mid-Hudson vicinity	Resident	Adult	Ingestion	On-Site	Qual	Limited data, studies show low PCB uptake in forage crops Qualitatively assessed in Upper Hudson River HHRA
					Adolescent	Ingestion	On-Site	Qual	
					Child	Ingestion	On-Site	Qual	
	Beef	Beef	Mid-Hudson vicinity	Resident	Adult	Ingestion	On-Site	Qual	Limited data, studies show non-detect PCB levels in cow's milk in NY Qualitatively assessed in Upper Hudson River HHRA.
					Adolescent	Ingestion	On-Site	Qual	
					Child	Ingestion	On-Site	Qual	
	Dairy Products	Milk, eggs	Mid-Hudson vicinity	Resident	Adult	Ingestion	On-Site	Qual	Limited data, studies show non-detect PCB levels in cow's milk in NY Qualitatively assessed in Upper Hudson River HHRA.
					Adolescent	Ingestion	On-Site	Qual	
					Child	Ingestion	On-Site	Qual	

"Quant" = Quantitative risk analysis performed "Qual" = Qualitative analysis performed

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

Scenario Timeframe Current/Future
Medium. Fish
Exposure Medium Fish
Exposure Point Mid-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 21	N/A	2 3	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 1999-2046 (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

MID-HUDSON RIVER - Sediment

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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)	0.31	N/A	0.67	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 segment-averaged modeled concentration between 1999-2046 (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
MID-HUDSON RIVER - River Water

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-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)	3.3E-06	N/A	1.9E-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 segment-averaged modeled concentration between 1999-2046 (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 (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	38.4%	14%
Bullhead	23	1,092	558	24.3	61.9	300	36.7%	14%
Carp	2	[b]	90	45.0	42.4	75	5.9%	2%
Catfish	11	158	113	10.3	15.5	50	7.4%	3%
Eel	4	38	38	9.5	10.6	25	2.5%	0.9%
Perch	17	833	139	8.2	12.5	51	9.1%	3%
Subtotal		3,963	1,522				100%	37%
Salmon	35	559	193	5.5	5.3	25		5%
Trout	130	3,099	1,230	9.5	15.7	133		30%
Walleye	36	333	134	3.7	4.2	20		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	40%	14%
Bullhead	53	1,200	634	12.0	21.5	100	25%	8%
Carp	4	7	29	7.3	6.7	14	1.1%	0.4%
Catfish	10	46	46	4.6	6.9	20	1.8%	0.6%
Eel	2	2	3	1.5	0.7	2	0.1%	0.04%
Perch	51	2,289	816	16.0	32.4	200	32%	11%
Subtotal		6,914	2,560				100%	34%
Salmon	55	538	480	8.7	15.2	80		6%
Trout	152	2,428	1,400	9.2	18.3	150		18%
Walleye	112	2,292	1,054	9.4	14.2	75		14%
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	45%	17%
Bullhead	55	2,374	1,099	20.0	43.2	225	44%	16%
Carp	5	16	11	2.2	1.6	5	0.4%	0.2%
Catfish	4	40	17	4.3	2.8	7	0.7%	0.3%
Eel	5	9	13	2.6	2.5	7	0.5%	0.2%
Perch	24	338	222	9.3	21.7	100	9%	3%
Subtotal		6,783	2,472				100%	37%
Salmon	14	139	120	8.6	7.3	20		2%
Trout	148	2,836	1,319	8.9	16.8	157		20%
Walleye	34	389	206	6.1	8.8	40		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 2-6 (Unchanged)
Mid-Hudson River Perch and Bass

Species	Species Intake[1]	Mid-Hudson Species	Relative Percentage Species Caught[2]	Relative Percentage Species Intake
Perch	9%	White Perch	85%	7.6%
		Yellow Perch	15%	1.4%
Bass	38%	Largemouth Bass	40%	15%
		Striped Bass	60%	23%

[1] *From 1991 New York Angler Survey, see Table 2-5.*
[2] *From 1991/92 and 1996 NYSDOH study of Hudson River anglers (NYSDOH, 1999).*

Table 2-7 (Unchanged)
Species-Group Intake Percentages

Group 1		Group 2		Group 3		Group 4		Group 5	
Brown bullhead	36.7%	White Perch	7.6%	Yellow Perch	1.4%	Largemouth Bass	15%	Striped Bass	23%
Carp	5.9%								
Catfish	7.4%								
Eel	2.5%								
Species Group Totals	53%		7.6%		1.4%		15%		23%

Sources:
1991 New York Angler Survey (Connelly et al, 1992)
1991/92 and 1996 NYSDOH study of Hudson River anglers (NYSDOH, 1999).

TABLE 2-8 (Revised)
MEDIUM-SPECIFIC MODELED EXPOSURE POINT CONCENTRATION SUMMARY
MID-HUDSON RIVER FISH

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-Hudson Fish

Chemical of Potential Concern	Units	Arithmetic Mean (3)	95% UCL of Normal Data	Maximum Concentration (3)	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	**	1 6	N/A	mg/kg wet weight	1 2	Mean-N	Averaged over RME ED	1 4	Mean-N	Averaged over CT ED
In Yellow Perch	mg/kg wet weight	0 32	**	0 65	N/A	mg/kg wet weight	0.34	Mean-N	Averaged over RME ED	0.49	Mean-N	Averaged over CT ED
In Largemouth Bass	mg/kg wet weight	0 82	**	1 7	N/A	mg/kg wet weight	0 87	Mean-N	Averaged over RME ED	1 3	Mean-N	Averaged over CT ED
In Striped Bass	mg/kg wet weight	1 1	**	2 3	N/A	mg/kg wet weight	1.2	Mean-N	Averaged over RME ED	1.7	Mean-N	Averaged over CT ED
In White Perch	mg/kg wet weight	0 53	**	1 4	N/A	mg/kg wet weight	0 57	Mean-N	Averaged over RME ED	0.97	Mean-N	Averaged over CT ED
Species-weighted for adult exposure (1)	mg/kg wet weight	0 91	**	1 6	N/A	mg/kg wet weight	1.1	Mean-N	Averaged over RME ED	1.4	Mean-N	Averaged over CT ED
Species-weighted for adolescent exposure (1)	mg/kg wet weight	0 91	**	1.6	N/A	mg/kg wet weight	1 3	Mean-N	Averaged over RME ED	1.5	Mean-N	Averaged over CT ED
Species-weighted for child exposure (1)	mg/kg wet weight	0 91	**	1 6	N/A	mg/kg wet weight	1.4	Mean-N	Averaged over RME ED	1.5	Mean-N	Averaged over CT ED
Species-weighted for chronic exposure (2)	mg/kg wet weight	0 91	**	1.6	N/A	mg/kg wet weight	1.4	Mean-N	Averaged over RME ED	dependent on receptor (4)	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 fish data was modeled, not measured.

ED = Exposure Duration

CT = Central Tendency

(1) PCB concentrations for each species were weighted based on species-group intake percentages (Connelly et al., 1992; NYSDOH, 1999) and averaged over the central tendency adult, adolescent, and child exposure durations (6, 3, and 3 years, respectively) to calculate the CT EPCs, and over the RME adult, adolescent, and child exposure durations (22, 12, and 6 years, respectively) to calculate the RME EPCs for cancer risks.

(2) PCB concentrations for each species were weighted based on species-group intake percentages (Connelly et al., 1992; NYSDOH, 1999) and averaged over 7 years to calculate the RME EPC for non-cancer hazards

(3) Mean/maximum modeled concentration between 1999-2046 (USEPA, 2000).

(4) CT EPC for chronic exposure is dependent on exposure duration for each receptor (1 4 mg/kg adult; 1.5 mg/kg adolescent/child).

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

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

Chemical of Potential Concern	Units	Arithmetic Mean (1)	95% UCL of Normal Data	Maximum Concentration (1)	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure (2)			Central Tendency (2)		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
PCBs	mg/kg	0.4	**	0.7	N/A	mg/kg						
Adult							0.53	Mean-N	Averaged over RME ED	0.65	Mean-N	Averaged over CT ED
Adolescent							0.59	Mean-N	Averaged over RME ED	0.66	Mean-N	Averaged over CT ED
Child							0.64	Mean-N	Averaged over RME ED	0.66	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

(1) Mean/maximum of segment-averaged modeled concentration 1999-2046 (USEPA, 2000)

(2) EPC values were averaged over 23 yrs RME and 5 yrs CT for adults, 12 yrs RME and 3 yrs CT for adolescents, 6 yrs RME and 3 yrs CT for children, for a total of 41 yrs RME and 11 yrs CT exposure.

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

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-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 (2)			Central Tendency (2)		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
PCBs	mg/L	6 4E-06	**	1 9E-05	N/A	mg/L						
Adult							8 8E-06	Mean-N	Averaged over RME ED	1.5E-05	Mean-N	Averaged over CT ED
Adolescent							1 1E-05	Mean-N	Averaged over RME ED	1.6E-05	Mean-N	Averaged over CT ED
Child							1 4E-05	Mean-N	Averaged over RME ED	1.6E-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

(1) Mean/maximum of segment-averaged modeled concentration 1999-2046 (USEPA, 2000)

(2) EPC values were averaged over 23 yrs RME and 5 yrs CT for adults, 12 yrs RME and 3 yrs CT for adolescents; 6 yrs RME and 3 yrs CT for children; for a total of 41 yrs RME and 11 yrs CT exposure.

Table 2-11 (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							
						Total	From						
Age Group							Albany	Columbia	Dutchess	Greene	Rensselaer	Ulster	
5 to 9	8,638	9,002	228	8,774	2,318	6,456	5,795	42	14	63	536	6	2,546
10 to 14	10,128	6,482	226	6,256	1,607	4,649	4,253	28	21	36	304	7	1,833
15 to 19	11,284	9,642	236	9,406	4,983	4,423	3,713	45	133	64	428	40	5,219
20 to 24	8,012	19,788	428	19,360	11,201	8,159	6,188	83	367	311	995	215	11,629
25 to 29	5,515	18,568	640	17,928	6,882	11,046	9,111	143	94	221	1366	111	7,522
30 to 34	8,196	17,658	558	17,100	5,691	11,409	10,256	86	37	149	840	41	6,249
35 to 44	24,243	20,419	407	20,012	6,094	13,918	12,533	149	53	160	980	43	6,501
45 to 54	20,091	7,999	277	7,722	2,234	5,488	4,866	36	27	72	458	29	2,511
55 to 64	20,764	4,837	97	4,740	1,271	3,469	3,099	34	48	62	222	4	1,368
65 to 74	19,380	4,189	78	4,111	928	3,183	2,867	34	32	34	179	37	1,006
75 to 84	10,929	2,914	22	2,892	653	2,239	1,984	16	0	23	190	26	675
85+	3,670	1,746	0	1,746	367	1,379	1,227	13	0	22	117	0	367

Notes:

a. The Mid-Hudson Region consists of Albany, Columbia, Dutchess, Greene, Rensselaer, and Ulster Counties.

Source: 1990 U.S. Census.

Table 2-12 (Unchanged)
County-to-County In-Migration Data for Columbia County, NY

	No Move	Move In											Total from Outside Region[a]
		Total	From Abroad	Domestic									
				Total	Outside Region[a]	Inside Region							
						Total	From						
Age Group							Columbia	Albany	Dutchess	Greene	Rensselaer	Ulster	
5 to 9	2,143	2,284	91	2,193	506	1,687	1,341	48	165	47	77	9	597
10 to 14	2,399	1,583	20	1,563	433	1,130	900	28	103	35	34	30	453
15 to 19	2,644	1,587	15	1,572	539	1,033	849	31	44	48	41	20	554
20 to 24	1,591	2,024	44	1,980	415	1,565	1,314	23	86	8	118	16	459
25 to 29	1,242	3,246	52	3,194	864	2,330	1,819	97	228	38	122	26	916
30 to 34	1,663	3,144	77	3,067	922	2,145	1,678	80	217	48	91	31	999
35 to 44	6,034	3,896	84	3,812	1,332	2,480	1,859	85	165	103	230	38	1,416
45 to 54	4,979	1,932	38	1,894	622	1,272	1,060	60	80	25	24	23	660
55 to 64	4,756	1,170	4	1,166	388	778	674	34	25	19	16	10	392
65 to 74	4,650	1,075	3	1,072	370	702	613	11	30	11	29	8	373
75 to 84	2,721	823	2	821	192	629	521	10	30	8	51	9	194
85+	725	315	0	315	81	234	182	6	5	15	17	9	81

Notes:

a. The Mid-Hudson Region consists of Albany, Columbia, Dutchess, Greene, Rensselaer, and Ulster Counties.

Source: 1990 U.S. Census.

Table 2-13 (Unchanged)
County-to-County In-Migration Data for Dutchess County, NY

	No Move	Move In											Total from Outside Region[a]
		Total	From Abroad	Domestic									
				Total	Outside Region[a]	Inside Region							
						Total	From						
Age Group							Dutchess	Albany	Columbia	Greene	Rensselaer	Ulster	
5 to 9	9,052	8,557	224	8,333	3,749	4,584	4,363	0	72	0	0	149	3,973
10 to 14	9,868	5,878	135	5,743	2,249	3,494	3,367	16	33	0	0	78	2,384
15 to 19	10,981	7,671	347	7,324	4,313	3,011	2,833	24	40	9	25	80	4,660
20 to 24	7,992	12,027	461	11,566	6,472	5,094	4,675	30	61	25	31	272	6,933
25 to 29	5,622	16,195	497	15,698	7,645	8,053	7,221	166	82	12	46	526	8,142
30 to 34	8,384	15,794	409	15,385	7,156	8,229	7,578	144	90	2	13	402	7,565
35 to 44	23,706	18,091	400	17,691	7,774	9,917	9,255	41	136	8	22	455	8,174
45 to 54	21,703	7,320	180	7,140	2,865	4,275	4,049	8	32	15	4	167	3,045
55 to 64	17,443	4,503	98	4,405	1,885	2,520	2,469	0	9	5	2	35	1,983
65 to 74	13,686	3,394	74	3,320	1,496	1,824	1,727	0	20	0	0	77	1,570
75 to 84	7,236	2,331	52	2,279	984	1,295	1,220	10	33	0	0	32	1,036
85+	2,149	889	0	889	379	510	446	0	0	0	0	64	379

Notes:

a. The Mid-Hudson Region consists of Albany, Columbia, Dutchess, Greene, Rensselaer, and Ulster Counties.

Source: 1990 U.S. Census.

Table 2-14 (Unchanged)
County-to-County In-Migration Data for Greene County, NY

	No Move	Move In											Total from Outside Region[a]
		Total	From Abroad	Domestic									
				Total	Outside Region[a]	Inside Region							
						Total	From						
Age Group							Greene	Albany	Columbia	Duchess	Rensselaer	Ulster	
5 to 9	1,491	1,496	20	1,476	593	883	712	120	1	16	0	34	613
10 to 14	1,706	1,074	2	1,072	383	689	571	79	0	21	0	18	385
15 to 19	1,713	1,145	19	1,126	495	631	525	27	19	20	5	35	514
20 to 24	1,229	1,971	57	1,914	991	923	719	81	31	33	0	59	1,048
25 to 29	967	2,594	65	2,529	1,165	1,364	1111	79	21	14	9	130	1,230
30 to 34	1,216	2,540	33	2,507	992	1,515	1169	171	49	57	12	57	1,025
35 to 44	3,742	2,816	21	2,795	1,109	1,686	1328	137	53	78	27	63	1,130
45 to 54	3,503	1,228	18	1,210	500	710	503	104	15	20	18	50	518
55 to 64	3,195	1,095	3	1,092	518	574	498	25	7	16	0	28	521
65 to 74	3,142	813	3	810	356	454	370	43	17	15	0	9	359
75 to 84	1,979	464	1	463	148	315	279	24	10	0	0	2	149
85+	480	254	0	254	127	127	120	7	0	0	0	0	127

Notes:

a. The Mid-Hudson Region consists of Albany, Columbia, Dutchess, Greene, Rensselaer, and Ulster Counties.

Source: 1990 U.S. Census.

Table 2-15 (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							
						Total	From						
Age Group							Rensselaer	Albany	Columbia	Duchess	Greene	Ulster	
5 to 9	5,577	4,769	80	4,689	1,046	3,643	2,902	656	64	0	4	17	1,126
10 to 14	6,155	3,608	73	3,535	666	2,869	2,283	438	58	21	13	56	739
15 to 19	6,820	5,126	213	4,913	2,304	2,609	2,084	368	46	33	47	31	2,517
20 to 24	4,911	8,940	436	8,504	3,564	4,940	3,777	776	175	157	26	29	4,000
25 to 29	3,763	8,867	435	8,432	2,331	6,101	4,713	1,211	113	40	0	24	2,766
30 to 34	5,236	7,976	221	7,755	2,053	5,702	4,076	1,419	139	42	14	12	2,274
35 to 44	14,632	9,049	130	8,919	2,112	6,807	5,030	1,503	170	11	39	54	2,242
45 to 54	10,930	3,214	40	3,174	685	2,489	1,951	495	39	0	0	4	725
55 to 64	11,355	2,125	46	2,079	487	1,592	1,303	264	10	2	0	13	533
65 to 74	10,010	1,712	5	1,707	369	1,338	1,101	216	9	4	0	8	374
75 to 84	5,613	1,146	7	1,139	190	949	730	205	0	0	5	9	197
85+	1,522	520	0	520	101	419	328	75	9	0	0	7	101

Notes:

a. The Mid-Hudson Region consists of Albany, Columbia, Dutchess, Greene, Rensselaer, and Ulster Counties.

Source: 1990 U.S. Census.

Table 2-16 (Unchanged)
County-to-County In-Migration Data for Ulster County, NY

	No Move	Move In											Total from Outside Region[a]
		Total	From Abroad	Domestic									
				Total	Outside Region[a]	Inside Region							
						Total	From						
Age Group							Ulster	Albany	Columbia	Duchess	Greene	Rensselaer	
5 to 9	5,911	4,990	73	4,917	1,619	3,298	2,990	14	13	250	31	0	1,692
10 to 14	6,285	4,019	43	3,976	1,340	2,636	2,368	5	17	223	19	4	1,383
15 to 19	6,544	4,059	165	3,894	1,915	1,979	1,741	12	15	190	9	12	2,080
20 to 24	4,651	7,370	229	7,141	3,553	3,588	2,980	76	0	454	68	10	3,782
25 to 29	3,959	10,262	293	9,969	3,921	6,048	4,864	75	21	1004	65	19	4,214
30 to 34	5,824	9,224	226	8,998	3,238	5,760	4,916	92	18	663	56	15	3,464
35 to 44	15,066	11,368	209	11,159	3,839	7,320	6,542	45	23	629	66	15	4,048
45 to 54	13,465	4,510	65	4,445	1,602	2,843	2,504	7	18	272	31	11	1,667
55 to 64	12,045	2,774	49	2,725	832	1,893	1,722	17	9	122	23	0	881
65 to 74	10,090	2,122	28	2,094	790	1,304	1,241	0	11	37	15	0	818
75 to 84	5,884	1,307	0	1,307	350	957	890	8	0	54	5	0	350
85+	1,664	494	0	494	181	313	284	0	0	29	0	0	181

Notes:

a. The Mid-Hudson Region consists of Albany, Columbia, Dutchess, Greene, Rensselaer, and Ulster Counties.

Source: 1990 U.S. Census.

Table 2-17 (Unchanged)
County-to-County In-Migration Data for the Mid-Hudson River Region

	No Move	Move In											Total from Outside Region[a]
		Total	From Abroad	Domestic									
				Total	Outside Region[a]	Inside Region							
						Total	From						
Age Group							Albany	Renssalaer	Columbia	Dutchess	Greene	Ulster	
5 to 9	32,812	31,098	716	30,382	9,831	20,551	6,633	3,515	1,533	4,808	857	3,205	10,547
10 to 14	36,541	22,644	499	22,145	6,678	15,467	4,819	2,625	1,036	3,756	674	2,557	7,177
15 to 19	39,986	29,230	995	28,235	14,549	13,686	4,175	2,595	1,014	3,253	702	1,947	15,544
20 to 24	28,386	52,120	1,655	50,465	26,196	24,269	7,174	4,931	1,664	5,772	1,157	3,571	27,851
25 to 29	21,068	59,732	1,982	57,750	22,808	34,942	10,739	6,275	2,199	8,601	1,447	5,681	24,790
30 to 34	30,519	56,336	1,524	54,812	20,052	34,760	12,162	5,047	2,060	8,594	1,438	5,459	21,576
35 to 44	87,423	65,639	1,251	64,388	22,260	42,128	14,344	6,304	2,390	10,191	1,704	7,195	23,511
45 to 54	74,671	26,203	618	25,585	8,508	17,077	5,540	2,466	1,200	4,448	646	2,777	9,126
55 to 64	69,558	16,504	297	16,207	5,381	10,826	3,439	1,543	743	2,682	607	1,812	5,678
65 to 74	60,958	13,305	191	13,114	4,309	8,805	3,137	1,309	704	1,845	430	1,380	4,500
75 to 84	34,362	8,985	84	8,901	2,517	6,384	2,241	971	580	1,304	320	968	2,601
85+	10,210	4,218	0	4,218	1,236	2,982	1,315	462	204	480	157	364	1,236

Notes:

a. The Mid-Hudson Region consists of Albany, Columbia, Dutchess, Greene, Rensselaer, and Ulster Counties.

Source: 1990 U.S. Census.

Table 2-18 (Unchanged)
Computation of 1-Year Move Probabilities for the Mid-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]	$p_{k,l}$[f] (Mid-Hudson)	$p_{k,l}$ (Upper Hudson)	Difference Mid-Hudson vs. Upper Hudson
5 to 9	(1)	10,547	32,812	36,541	6,818	15.7%	3.1%	2.5%	-0.6%
10 to 14	(2)	7,177	36,541	39,986	3,732	8.5%	1.7%	1.6%	-0.1%
15 to 19	(3)	15,544	39,986	28,386	27,144	48.9%	9.8%	9.5%	-0.3%
20 to 24	(4)	27,851	28,386	21,068	35,169	62.5%	12.5%	11.8%	-0.7%
25 to 29	(5)	24,790	21,068	30,519	15,339	33.4%	6.7%	5.9%	-0.8%
30 to 34	(6)	21,576	30,519	43,712[g]	8,383	16.1%	3.2%	3.5%	0.3%
35 to 44	(7)	23,511	87,423	74,671	36,263	32.7%	6.5%	7.5%	1.0%
45 to 54	(8)	9,126	74,671	69,558	14,239	17.0%	3.4%	2.2%	-1.2%
55 to 64	(9)	5,678	69,558	60,958	14,278	19.0%	3.8%	3.2%	-0.6%
65 to 74	(10)	4,500	60,958	34,362	31,096	47.5%	9.5%	9.5%	0.0%
75 to 84	(11)	2,601	34,362	10,210	26,753	72.4%	14.5%	14.0%	-0.5%
85+	(12)	1,236	10,210	NA[h]	11,446		100%[i]	100%[i]	0.0%

Notes: a. *Taken from the column labeled, "Total from Outside Region" in Table 2-14.*

b. *The Mid-Hudson Region consists of Albany, Columbia, Dutchess, Greene, Rensselaer, and Ulster Counties.*

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* $(Out_{1985\text{-}90,k})/(Start_{1985\text{-}90,k} + In_{1985\text{-}90,k})$.

f. *Set equal to 1/5 x 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 2-19a (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

MID-HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-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	1.1	See Table 2-8	1.4	See Table 2-8	Average Daily Intake (mg/kg-day) =
	C_{fish}-NC	PCB Concentration in Fish (Non-cancer)**	mg/kg wet weight	1 4	See Table 2-8	1.4	See Table 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 Mid-Hudson.	1	Assumes 100% fish ingested is from Mid-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	22	derived from 95th percentile value, based on 1991 NY Angler and 1990 US Census data	6	derived from 50th percentile value, based on 1991 NY Angler and 1990 US Census data	
	ED	Exposure Duration (Noncancer)	years	7	see text	6	derived from 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	2,190	ED (years) x 365 days/year.	

** Species-weighted PCB concentration averaged over river location

TABLE 2-19b (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

MID-HUDSON RIVER FISH - Adolescent Angler

Scenario Timeframe Current/Future
Medium Fish
Exposure Medium Fish
Exposure Point Mid-Hudson Fish
Receptor Population Angler
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_{fish}-C	PCB Concentration in Fish (Cancer)**	mg/kg wet weight	1 3	See Table 2-8	1 5	See Table 2-8	Average Daily Intake (mg/kg-day) =
	C_{fish}-NC	PCB Concentration in Fish (Non-cancer)**	mg/kg wet weight	1.4	See Table 2-8	1 5	See Table 2-8	C_{fish} x IR_{fish} x (1 - Loss) X FS x EF x ED x CF x 1/BW x 1/AT
	IR_{fish}	Ingestion Rate of Fish	grams/day	21 3	2/3 of RME adult ingestion rate.	2 7	2/3 of RME adult ingestion rate	
	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 Mid-Hudson.	1	Assumes 100% fish ingested is from Mid-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	12	derived from 95th percentile value, based on 1991 NY Angler and 1990 US Census data	3	derived from 50th percentile value, based on 1991 NY Angler and 1990 US Census data	
	ED	Exposure Duration (Noncancer)	years	7	see text	3	derived from 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	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	2,555	ED (years) x 365 days/year	1,095	ED (years) x 365 days/year.	

** Species-weighted PCB concentration averaged over river location

TABLE 2-19c (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

MID-HUDSON RIVER FISH - Child Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-Hudson Fish
Receptor Population: Angler
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_{fish}	PCB Concentration in Fish**	mg/kg wet weight	1.4	See Table 2-8	1.5	See Table 2-8	Average Daily Intake (mg/kg-day) =
	IR_{fish}	Ingestion Rate of Fish	grams/day	10.6	1/3 of RME adult ingestion rate.	1.3	1/3 of CT adult ingestion rate	$C_{fish} \times IR_{fish} \times (1 - Loss) \times FS \times EF \times ED \times CF \times 1/BW \times 1/AT$
	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 Mid-Hudson	1	Assumes 100% fish ingested is from Mid-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	years	6	derived from 95th percentile value, based on 1991 NY Angler and 1990 US Census data.	3	derived from 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	15	Mean child body weight (USEPA, 1989b).	15	Mean child body weight (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	

** Species-weighted PCB concentration averaged over river location for both cancer and non-cancer calculations.

TABLE 2-20 (Revised)
VALUES USED FOR DAILY INTAKE CALCULATIONS
MID-HUDSON RIVER SEDIMENT - Adult Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid 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	0 53	See Table 2-9	0 65	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 Mid-Hudson	1	Assumes 100% sediment exposure is from Mid-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 Mid-Hudson Counties (see text)	5	derived from 50th percentile of residence duration in 5 Mid-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	0 53	See Table 2 9	0 65	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 2-21 (Revised)
VALUES USED FOR DAILY INTAKE CALCULATIONS
MID-HUDSON RIVER SEDIMENT - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid 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	0.59	See Table 2-9	0.66	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)	$C_{sediment} \times IR_{sediment} \times FS \times EF \times ED \times CF \times 1/BW \times 1/AT$
	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 Mid-Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Mid-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	0.59	See Table 2-9	0.66	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)	$C_{sediment} \times DA \times AF \times SA \times EF \times ED \times CF \times 1/BW \times 1/AT$
	AF	Adherance Factor	mg/cm²	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	cm²/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 Mid-Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Mid-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 2-22 (Revised)
VALUES USED FOR DAILY INTAKE CALCULATIONS
MID-HUDSON RIVER SEDIMENT Child Recreator

Scenario Timeframe Current/Future
Medium Sediment
Exposure Medium Sediment
Exposure Point Banks of Mid-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	0 64	See Table 2-9	0 66	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)	$C_{sediment} \times IR_{sediment} \times FS \times EF \times ED \times CF \times 1/BW \times 1/AT$
	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 Mid-Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Mid-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	0 64	See Table 2-9	0 66	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)	$C_{sediment} \times DA \times AF \times SA \times EF \times ED \times CF \times 1/BW \times 1/AT$
	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 Mid Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Mid 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 2-23 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

MID-HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe Current/Future
Medium River Water
Exposure Medium River Water
Exposure Point. Mid-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	8 8E-06	See Table 2-10	1 5E-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 Mid-Hudson Counties (see text)	5	derived from 50th percentile of residence duration in 5 Mid-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 2-24 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

MID-HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe Current/Future
Medium River Water
Exposure Medium River Water
Exposure Point Mid-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	1 1E-05	See Table 2-10	1.6E-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 Mid-Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Mid-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 2-25 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

MID-HUDSON RIVER WATER - Child Recreator

Scenario Timeframe Current/Future
Medium River Water
Exposure Medium: River Water
Exposure Point: Mid-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	1 4E-05	See Table 2-10	1.6E-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 Mid-Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Mid-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 2-26 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

MID-HUDSON RIVER WATER - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-Hudson River
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
Ingestion	C_{water}	Chemical Concentration in River Water	mg/L	8.8E-06	See Table 2-10	1.5E-05	See Table 2-10	Average Daily Intake (mg/kg-day) =
	IR	Ingestion Rate	L/day	2.3	90th percentile drinking water intake rate for adults (USEPA, 1997c)	1.40	Mean drinking water intake rate for adults (USEPA, 1997c)	C_{water} x IR x EF x ED x 1/BW x 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 Mid-Hudson Counties (see text)	5	derived from 50th percentile of residence duration in 5 Mid-Hudson Counties (see text)	
	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 2-27 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

MID-HUDSON RIVER WATER - Adolescent Resident

Scenario Timeframe: Current/Future
Medium River Water
Exposure Medium River Water
Exposure Point Mid-Hudson River
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
Ingestion	C_{water}	Chemical Concentration in River Water	mg/L	1.1E-05	See Table 2-10	1 6E-05	See Table 2-10	Average Daily Intake (mg/kg-day) =
	IR	Ingestion Rate	L/day	2 3	90th percentile drinking water intake rate for adults (USEPA, 1997c)	1 40	Mean drinking water intake rate for adults (USEPA, 1997c)	C_{water} x IR x EF x ED x 1/BW x 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 Mid-Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Mid-Hudson Counties (see text)	
	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 2-28 (Revised)

VALUES USED FOR DAILY INTAKE CALCULATIONS

MID-HUDSON RIVER WATER - Child Resident

Scenario Timeframe Current/Future
Medium. River Water
Exposure Medium River Water
Exposure Point Mid-Hudson River
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
Ingestion	C_{water}	Chemical Concentration in River Water	mg/L	1.4E-05	See Table 2-10	1 6E-05	See Table 2-10	Average Daily Intake (mg/kg-day) =
	IR	Ingestion Rate	L/day	1 5	90th percentile drinking water intake rate for children, ages 3-5 (USEPA, 1997c)	0.87	Mean drinking water intake rate for children, ages 3-5 (USEPA, 1997c)	C_{water} x IR x EF x ED x 1/BW x 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 Mid-Hudson Counties (see text)	3	derived from 50th percentile of residence duration in 5 Mid-Hudson Counties (see text)	
	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 3-1 (Unchanged)

NON-CANCER TOXICITY DATA -- ORAL/DERMAL

MID-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 0E-05 (2)	mg/kg-d	--	--	--	LOAEL	300	IRIS	6/1/97
Aroclor 1016		7.0E-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 3-2 (Unchanged)

CANCER TOXICITY DATA -- ORAL/DERMAL

MID-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)	--	--	$(mg/kg-d)^{-1}$	B2	IRIS	6/1/97
	2 (3)	--	--	$(mg/kg-d)^{-1}$	B2	IRIS	6/1/97
	0 3 (4)	--	--	$(mg/kg-d)^{-1}$	B2	IRIS	6/1/97
	0.4 (5)	--	--	$(mg/kg-d)^{-1}$	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 ingestion and dermal contact (if no absorption factor is applied) with water soluble congeners in river water.

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

TABLE 4-1a-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-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	1.4	mg/kg wt weight	1.4	mg/kg wt weight	M	6.4E-04	mg/kg-day	2.0E-05	mg/kg-day	N/A	N/A	32
												Total Hazard Index Across All Exposure Routes/Pathways	32

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

TABLE 4-1a-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-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	1.4	mg/kg wt weight	1.4	mg/kg wt weight	M	6.4E-05	mg/kg-day	2.0E-05	mg/kg-day	N/A	N/A	3
												Total Hazard Index Across All Exposure Routes/Pathways	3

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

TABLE 4-1b-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER FISH - Adolescent Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-Hudson Fish
Receptor Population: Angler
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	1.4	mg/kg wt weight	1.4	mg/kg wt weight	M	6.9E-04	mg/kg-day	2.0E-05	mg/kg-day	N/A	N/A	35
												Total Hazard Index Across All Exposure Routes/Pathways	35

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

TABLE 4-1b-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER FISH - Adolescent Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-Hudson Fish
Receptor Population: Angler
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	1.5	mg/kg wt weight	1.5	mg/kg wt weight	M	7.5E-05	mg/kg-day	2.0E-05	mg/kg-day	N/A	N/A	4
												Total Hazard Index Across All Exposure Routes/Pathways	4

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

TABLE 4-1c-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER FISH - Child Angler

Scenario Timeframe Current/Future
Medium: Fish
Exposure Medium. Fish
Exposure Point Mid-Hudson Fish
Receptor Population Angler
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	1 4	mg/kg wt weight	1 4	mg/kg wt weight	M	9 9E-04	mg/kg-day	2.0E-05	mg/kg-day	N/A	N/A	49
												Total Hazard Index Across All Exposure Routes/Pathways	49

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

TABLE 4-1c-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER FISH - Child Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-Hudson Fish
Receptor Population: Angler
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	1.5	mg/kg wt weight	1.5	mg/kg wt weight	M	1.0E-04	mg/kg-day	2.0E-05	mg/kg-day	N/A	N/A	5
												Total Hazard Index Across All Exposure Routes/Pathways	5

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

TABLE 4-2-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER SEDIMENT- Adult Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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	0.53	mg/kg	0.53	mg/kg	M	1.3E-08	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.00019
Dermal	PCBs	0.53	mg/kg	0.53	mg/kg	M	6.9E-08	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0010
										Total Hazard Index Across All Exposure Routes/Pathways			0.0012

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

TABLE 4-2-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER SEDIMENT- Adult Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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	0.65	mg/kg	0.65	mg/kg	M	8.9E-09	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.00013
Dermal	PCBs	0.65	mg/kg	0.65	mg/kg	M	4.5E-08	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.00065
										Total Hazard Index Across All Exposure Routes/Pathways			0.00078

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

TABLE 4-3-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER SEDIMENT- Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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	0.59	mg/kg	0 59	mg/kg	M	7 3E-08	mg/kg-day	7 0E-05	mg/kg-day	N/A	N/A	0 0010
Dermal	PCBs	0.59	mg/kg	0 59	mg/kg	M	2.2E-07	mg/kg-day	7 0E-05	mg/kg-day	N/A	N/A	0 0031
										Total Hazard Index Across All Exposure Routes/Pathways			0.0042

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

TABLE 4-3-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER SEDIMENT- Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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	0.66	mg/kg	0.66	mg/kg	M	4.2E-08	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.00060
Dermal	PCBs	0.66	mg/kg	0.66	mg/kg	M	1.3E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0018
												Total Hazard Index Across All Exposure Routes/Pathways	0.0024

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

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

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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	0.64	mg/kg	0.64	mg/kg	M	1.5E-07	mg/kg-day	7 0E-05	mg/kg-day	N/A	N/A	0.0022
Dermal	PCBs	0.64	mg/kg	0 64	mg/kg	M	1 2E-07	mg/kg-day	7 0E-05	mg/kg-day	N/A	N/A	0.0017
												Total Hazard Index Across All Exposure Routes/Pathways	0.0039

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

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

Scenario Timeframe Current/Future
Medium Sediment
Exposure Medium Sediment
Exposure Point Banks of Mid-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	0 66	mg/kg	0 66	mg/kg	M	8 4E-08	mg/kg-day	7 0E-05	mg/kg-day	N/A	N/A	0.0012
Dermal	PCBs	0 66	mg/kg	0.66	mg/kg	M	6.6E-08	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0009
										Total Hazard Index Across All Exposure Routes/Pathways			0.0021

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

TABLE 4-5-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe. Current/Future
Medium: River Water
Exposure Medium. River Water
Exposure Point. Mid-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	8.8E-06	mg/L	8.8E-06	mg/L	M	1.0E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0014
											Total Hazard Index Across All Exposure Routes/Pathways		0.0014

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

TABLE 4-5-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe Current/Future
Medium River Water
Exposure Medium River Water
Exposure Point Mid-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	1 5E-05	mg/L	1.5E-05	mg/L	M	9.3E-08	mg/kg-day	7 0E-05	mg/kg-day	N/A	N/A	0.0013
												Total Hazard Index Across All Exposure Routes/Pathways	0.0013

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

TABLE 4-6-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-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	1.1E-05	mg/L	1.1E-05	mg/L	M	4.5E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0064
												Total Hazard Index Across All Exposure Routes/Pathways	0.0064

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

TABLE 4-6-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-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	1 6E-05	mg/L	1 6E-05	mg/L	M	3 3E-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 4-7-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Child Recreator

Scenario Timeframe: Current/Future
Medium. River Water
Exposure Medium. River Water
Exposure Point Mid-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	1 4E-05	mg/L	1.4E-05	mg/L	M	2 9E-07	mg/kg-day	7 0E-05	mg/kg-day	N/A	N/A	0 0041
												Total Hazard Index Across All Exposure Routes/Pathways	0.0041

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

TABLE 4-7-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Child Recreator

Scenario Timeframe Current/Future
Medium River Water
Exposure Medium River Water
Exposure Point Mid-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	1 6E-05	mg/L	1.6E-05	mg/L	M	1.8E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0025
												Total Hazard Index Across All Exposure Routes/Pathways	0 0025

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

TABLE 4-8-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-Hudson River
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
Ingestion	PCBs	8.8E-06	mg/L	8.8E-06	mg/L	M	2.8E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0040
												Total Hazard Index Across All Exposure Routes/Pathways	0.0040

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

TABLE 4-8-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-Hudson River
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
Ingestion	PCBs	1 5E-05	mg/L	1 5E-05	mg/L	M	2.9E-07	mg/kg-day	7 0E-05	mg/kg-day	N/A	N/A	0.0041
												Total Hazard Index Across All Exposure Routes/Pathways	0 0041

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

TABLE 4-9-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Adolescent Resident

Scenario Timeframe: Current/Future
Medium River Water
Exposure Medium: River Water
Exposure Point Mid-Hudson River
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
Ingestion	PCBs	1.1E-05	mg/L	1 1E-05	mg/L	M	5 6E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0081
												Total Hazard Index Across All Exposure Routes/Pathways	0.0081

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

TABLE 4-9-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Adolescent Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-Hudson River
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
Ingestion	PCBs	1.6E-05	mg/L	1.6E-05	mg/L	M	5.0E-07	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.0071
												Total Hazard Index Across All Exposure Routes/Pathways	0.0071

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

TABLE 4-10-RME (Revised)
CALCULATION OF NON-CANCER HAZARDS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Child Resident

Scenario Timeframe Current/Future
Medium: River Water
Exposure Medium River Water
Exposure Point. Mid-Hudson River
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
Ingestion	PCBs	1 4E-05	mg/L	1.4E-05	mg/L	M	1 3E-06	mg/kg-day	7.0E-05	mg/kg-day	N/A	N/A	0.019
												Total Hazard Index Across All Exposure Routes/Pathways	0.019

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

TABLE 4-10-CT (Revised)
CALCULATION OF NON-CANCER HAZARDS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Child Resident

Scenario Timeframe Current/Future
Medium River Water
Exposure Medium River Water
Exposure Point Mid-Hudson River
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
Ingestion	PCBs	1.6E-05	mg/L	1 6E-05	mg/L	M	8 9E-07	mg/kg-day	7 0E-05	mg/kg-day	N/A	N/A	0 013
												Total Hazard Index Across All Exposure Routes/Pathways	0.013

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

TABLE 4-11a-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-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	1.1	mg/kg wt weight	1.1	mg/kg wt weight	M	1.6E-04	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	3.2E-04
										Total Risk Across All Exposure Routes/Pathways	3.2E-04

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

TABLE 4-11a-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER FISH - Adult Angler

Scenario Timeframe: Current/Future
Medium. Fish
Exposure Medium. Fish
Exposure Point: Mid-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	5.5E-06	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	5 5E-06
										Total Risk Across All Exposure Routes/Pathways	5.5E-06

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

TABLE 4-11b-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER FISH - Adolescent Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point. Mid-Hudson Fish
Receptor Population: Angler
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	1.3	mg/kg wt weight	1.3	mg/kg wt weight	M	1.1E-04	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	2.2E-04
										Total Risk Across All Exposure Routes/Pathways	2.2E-04

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

TABLE 4-11b-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER FISH - Adolescent Angler

Scenario Timeframe: Current/Future
Medium. Fish
Exposure Medium: Fish
Exposure Point: Mid-Hudson Fish
Receptor Population: Angler
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	1.5	mg/kg wt weight	1 5	mg/kg wt weight	M	3.2E-06	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	3.2E-06
										Total Risk Across All Exposure Routes/Pathways	3.2E-06

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

TABLE 4-11c-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER FISH - Child Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-Hudson Fish
Receptor Population: Angler
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	1.4	mg/kg wt weight	1.4	mg/kg wt weight	M	8.5E-05	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	1.7E-04
										Total Risk Across All Exposure Routes/Pathways	1.7E-04

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

TABLE 4-11c-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER FISH - Child Angler

Scenario Timeframe: Current/Future
Medium: Fish
Exposure Medium: Fish
Exposure Point: Mid-Hudson Fish
Receptor Population: Angler
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	1 5	mg/kg wt weight	1.5	mg/kg wt weight	M	4.5E-06	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	4.5E-06
										Total Risk Across All Exposure Routes/Pathways	4.5E-06

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

TABLE 4-12-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER SEDIMENT- Adult Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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	0.53	mg/kg	0 53	mg/kg	M	4.4E-09	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	8.9E-09
Dermal	PCBs	0.53	mg/kg	0.53	mg/kg	M	2.3E-08	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	4.5E-08
										Total Risk Across All Exposure Routes/Pathways	5.4E-08

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

TABLE 4-12-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER SEDIMENT- Adult Recreator

Scenario Timeframe. Current/Future
Medium: Sediment
Exposure Medium Sediment
Exposure Point Banks of Mid-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	0.65	mg/kg	0 65	mg/kg	M	6.4E-10	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	6.4E-10
Dermal	PCBs	0.65	mg/kg	0.65	mg/kg	M	3.2E-09	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	3.2E-09
										Total Risk Across All Exposure Routes/Pathways	3.9E-09

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

TABLE 4-13-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER SEDIMENT- Adolescent Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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	0 59	mg/kg	0.59	mg/kg	M	1.3E-08	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	2.5E-08
Dermal	PCBs	0.59	mg/kg	0.59	mg/kg	M	3.7E-08	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	7.5E-08
										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 4-13-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER SEDIMENT- Adolescent Recreator

Scenario Timeframe. Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point. Banks of Mid-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	0 66	mg/kg	0.66	mg/kg	M	1.8E-09	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	1.8E-09
Dermal	PCBs	0 66	mg/kg	0.66	mg/kg	M	5.4E-09	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	5.4E-09
										Total Risk Across All Exposure Routes/Pathways	7.2E-09

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

TABLE 4-14-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER SEDIMENT - Child Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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	0 64	mg/kg	0.64	mg/kg	M	1.3E-08	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	2 6E-08
Dermal	PCBs	0.64	mg/kg	0.64	mg/kg	M	1.0E-08	mg/kg-day	2	$(mg/kg\text{-}day)^{-1}$	2.0E-08
										Total Risk Across All Exposure Routes/Pathways	4.6E-08

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

TABLE 4-14-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER SEDIMENT - Child Recreator

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure Medium: Sediment
Exposure Point: Banks of Mid-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	0.66	mg/kg	0.66	mg/kg	M	3.6E-09	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	3.6E-09
Dermal	PCBs	0.66	mg/kg	0.66	mg/kg	M	2.8E-09	mg/kg-day	1	$(mg/kg\text{-}day)^{-1}$	2.8E-09
										Total Risk Across All Exposure Routes/Pathways	6.4E-09

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

TABLE 4-15-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-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	8.8E-06	mg/L	8.8E-06	mg/L	M	3.3E-08	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	1.3E-08
										Total Risk Across All Exposure Routes/Pathways	1.3E-08

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

TABLE 4-15-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Adult Recreator

Scenario Timeframe: Current/Future
Medium. River Water
Exposure Medium· River Water
Exposure Point: Mid-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	1 5E-05	mg/L	1.5E-05	mg/L	M	6.6E-09	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	2.0E-09
										Total Risk Across All Exposure Routes/Pathways	2.0E-09

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

TABLE 4-16-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe: Current/Future
Medium. River Water
Exposure Medium: River Water
Exposure Point: Mid-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	1 1E-05	mg/L	1.1E-05	mg/L	M	7.7E-08	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	3.1E-08
										Total Risk Across All Exposure Routes/Pathways	3 1E-08

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

TABLE 4-16-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Adolescent Recreator

Scenario Timeframe Current/Future
Medium: River Water
Exposure Medium River Water
Exposure Point: Mid-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	1.6E-05	mg/L	1.6E-05	mg/L	M	1.4E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	4.3E-09
										Total Risk Across All Exposure Routes/Pathways	4.3E-09

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

TABLE 4-17-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-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	1 4E-05	mg/L	1.4E-05	mg/L	M	2 4E-08	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	9.8E-09
										Total Risk Across All Exposure Routes/Pathways	9 8E-09

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

TABLE 4-17-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Child Recreator

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-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	1.6E-05	mg/L	1.6E-05	mg/L	M	7.5E-09	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	2.3E-09
										Total Risk Across All Exposure Routes/Pathways	2.3E-09

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

TABLE 4-18-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point. Mid-Hudson River
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-
Ingestion	PCBs	8 8E-06	mg/L	8.8E-06	mg/L	M	9.1E-08	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	3.6E-08
										Total Risk Across All Exposure Routes/Pathways	3.6E-08

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

TABLE 4-18-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Adult Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-Hudson River
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
Ingestion	PCBs	1 5E-05	mg/L	1 5E-05	mg/L	M	2.1E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	6.2E-09
										Total Risk Across All Exposure Routes/Pathways	6.2E-09

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

TABLE 4-19-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Adolescent Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-Hudson River
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
Ingestion	PCBs	1.1E-05	mg/L	1.1E-05	mg/L	M	9.7E-08	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	3.9E-08
										Total Risk Across All Exposure Routes/Pathways	3.9E-08

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

TABLE 4-19-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Adolescent Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-Hudson River
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
Ingestion	PCBs	1 6E-05	mg/L	1.6E-05	mg/L	M	2.1E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	6.4E-09
										Total Risk Across All Exposure Routes/Pathways	6.4E-09

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

TABLE 4-20-RME (Revised)
CALCULATION OF CANCER RISKS
REASONABLE MAXIMUM EXPOSURE
MID-HUDSON RIVER WATER - Child Resident

Scenario Timeframe: Current/Future
Medium: River Water
Exposure Medium: River Water
Exposure Point: Mid-Hudson River
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
Ingestion	PCBs	1.4E-05	mg/L	1.4E-05	mg/L	M	1.2E-07	mg/kg-day	0.4	$(mg/kg\text{-}day)^{-1}$	4.6E-08
										Total Risk Across All Exposure Routes/Pathways	4.6E-08

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

TABLE 4-20-CT (Revised)
CALCULATION OF CANCER RISKS
CENTRAL TENDENCY EXPOSURE
MID-HUDSON RIVER WATER - Child Resident

Scenario Timeframe. Current/Future
Medium. River Water
Exposure Medium: River Water
Exposure Point: Mid-Hudson River
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
Ingestion	PCBs	1 6E-05	mg/L	1.6E-05	mg/L	M	3.8E-08	mg/kg-day	0.3	$(mg/kg\text{-}day)^{-1}$	1.1E-08
										Total Risk Across All Exposure Routes/Pathways	1.1E-08

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

TABLE 4-21a-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

MID-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	Mid-Hudson Fish	PCBs	3.2E-04	--	--	3 2E-04	PCBs	LOAEL	32	--	--	32
						Total Risk Across Fish	3 2E-04					Total Hazard Index Across All Media and All Exposure Routes	32
						Total Risk Across All Media and All Exposure Routes	3.2E-04						

Total LOAEL HI = 32

TABLE 4-21a-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

MID-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	Mid-Hudson Fish	PCBs	5.5E-06	--	--	5.5E-06	PCBs	LOAEL	3	--	--	3
						Total Risk Across Fish	5.5E-06					Total Hazard Index Across All Media and All Exposure Routes	3
						Total Risk Across All Media and All Exposure Routes	5.5E-06						

Total LOAEL HI = 3

TABLE 4-21b-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

MID-HUDSON RIVER - Adolescent Angler

Scenario Timeframe: Current/Future
Receptor Population: Angler
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
Fish	Fish	Mid-Hudson Fish	PCBs	2.2E-04	--	--	2.2E-04	PCBs	LOAEL	35	--	--	35
						Total Risk Across Fish	2.2E-04					Total Hazard Index Across All Media and All Exposure Routes	35
						Total Risk Across All Media and All Exposure Routes	2.2E-04						

Total LOAEL HI = 35

TABLE 4-21b-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

MID-HUDSON RIVER - Adolescent Angler

Scenario Timeframe: Current/Future
Receptor Population: Angler
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
Fish	Fish	Mid-Hudson Fish	PCBs	3 2E-06	--	--	3 2E-06	PCBs	LOAEL	4	--	--	4
						Total Risk Across Fish	3 2E-06					Total Hazard Index Across All Media and All Exposure Routes	4
						Total Risk Across All Media and All Exposure Routes	3 2E-06						
												Total LOAEL HI =	4

TABLE 4-21c-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

MID-HUDSON RIVER - Child Angler

Scenario Timeframe: Current/Future
Receptor Population: Angler
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
Fish	Fish	Mid-Hudson Fish	PCBs	1.7E-04	--	--	1.7E-04	PCBs	LOAEL	49	--	--	49
						Total Risk Across Fish	1.7E-04					Total Hazard Index Across All Media and All Exposure Routes	49
						Total Risk Across All Media and All Exposure Routes	1.7E-04						

Total LOAEL HI = 49

TABLE 4-21c-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

MID-HUDSON RIVER - Child Angler

Scenario Timeframe	Current/Future
Receptor Population	Angler
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
Fish	Fish	Mid-Hudson Fish	PCBs	4 5E-06	--	--	4 5E-06	PCBs	LOAEL	5	--	--	5
						Total Risk Across Fish	4.5E-06					Total Hazard Index Across All Media and All Exposure Routes	5
						Total Risk Across All Media and All Exposure Routes	4.5E-06						
												Total LOAEL HI =	5

TABLE 4-21c-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

MID-HUDSON RIVER - Child Angler

Scenario Timeframe: Current/Future
Receptor Population: Angler
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
Fish	Fish	Mid-Hudson Fish	PCBs	1 7E-04	--	--	1.7E-04	PCBs	LOAEL	49	--	--	49
						Total Risk Across Fish	1 7E-04					Total Hazard Index Across All Media and All Exposure Routes	49
						Total Risk Across All Media and All Exposure Routes	1 7E-04						

Total LOAEL HI = 49

TABLE 4-21c-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

MID-HUDSON RIVER - Child Angler

Scenario Timeframe	Current/Future
Receptor Population.	Angler
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
Fish	Fish	Mid-Hudson Fish	PCBs	4 5E-06	--	--	4 5E-06	PCBs	LOAEL	5	--	--	5
						Total Risk Across Fish	4 5E-06					Total Hazard Index Across All Media and All Exposure Routes	5
						Total Risk Across All Media and All Exposure Routes	4.5E-06						

Total LOAEL HI = 5

TABLE 4-23-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

MID-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 Mid-Hudson	PCBs	2.5E-08	--	7.5E-08	1.0E-07	PCBs	NOAEL	0 0010	--	0 0031	0.0042
River Water	River Water	Mid-Hudson River	PCBs	--	--	3.1E-08	3.1E-08	PCBs	NOAEL	--	--	0 0064	0.0064

Total Risk Across Sediment	1.0E-07
Total Risk Across River Water	3 1E-08
Total Risk Across All Media and All Exposure Routes	1 3E-07

Total Hazard Index Across All Media and All Exposure Routes	0.011
Total NOAEL HI =	0.011

TABLE 4-23-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

MID-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 Mid-Hudson	PCBs	1 8E-09	--	5 4E-09	7.2E-09	PCBs	NOAEL	0 00060	--	0.0018	0 0024
River Water	River Water	Mid-Hudson River	PCBs	--	--	4.3E-09	4.3E-09	PCBs	NOAEL	--	--	0.0048	0 0048

Total Risk Across Sediment	7.2E-09	Total Hazard Index Across All Media and All Exposure Routes	0.0072
Total Risk Across River Water	4.3E-09		
Total Risk Across All Media and All Exposure Routes	1.1E-08	Total NOAEL HI =	0 0072

TABLE 4-24-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

MID-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 Mid-Hudson	PCBs	2 6E-08	--	2 0E-08	4 6E-08	PCBs	NOAEL	0.0022	--	0 0017	0 0039
River Water	River Water	Mid-Hudson River	PCBs	--	--	9 8E-09	9 8E-09	PCBs	NOAEL	--	--	0 0041	0.0041

Total Risk Across Sediment	4.6E-08	Total Hazard Index Across All Media and All Exposure Routes	0 0079
Total Risk Across River Water	9.8E-09		
Total Risk Across All Media and All Exposure Routes	5 6E-08	Total NOAEL HI =	0.0079

TABLE 4-24-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

MID-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 Mid-Hudson	PCBs	3 6E-09	--	2 8E-09	6 4E-09	PCBs	NOAEL	0 0012	--	0.0009	0 0021
River Water	River Water	Mid-Hudson River	PCBs	--	--	2.3E-09	2.3E-09	PCBs	NOAEL	--	--	0 0025	0 0025
						Total Risk Across Sediment	6.4E-09					Total Hazard Index Across All Media and All Exposure Routes	0.0047
						Total Risk Across River Water	2.3E-09						
						Total Risk Across All Media and All Exposure Routes	8.7E-09					Total NOAEL HI =	0.0047

TABLE 4-25-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

MID-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	River Water	Mid-Hudson River	PCBs	3 6E-08	--	--	3.6E-08	PCBs	NOAEL	0.0040	--	--	0.0040
				Total Risk Across All Media and All Exposure Routes			3 6E-08		Total Hazard Index Across All Media and All Exposure Routes				0.0040

Total NOAEL HI = 0 0040

TABLE 4-25-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

MID-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	River Water	Mid-Hudson River	PCBs	6 2E-09	--	--	6.2E-09	PCBs	NOAEL	0.0041	--	--	0.0041
				Total Risk Across All Media and All Exposure Routes			6.2E-09		Total Hazard Index Across All Media and All Exposure Routes				0.0041

Total NOAEL HI = 0.0041

TABLE 4-26-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

MID-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	River Water	Mid-Hudson River	PCBs	3 9E-08	--	--	3.9E-08	PCBs	NOAEL	0 0081	--	--	0 0081
						Total Risk Across All Media and All Exposure Routes	3 9E-08					Total Hazard Index Across All Media and All Exposure Routes	0 0081

Total NOAEL HI = 0.0081

TABLE 4-26-CT (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

CENTRAL TENDENCY EXPOSURE

MID-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	River Water	Mid-Hudson River	PCBs	6 4E-09	--	--	6 4E-09	PCBs	NOAEL	0 0071	--	--	0 0071
						Total Risk Across All Media and All Exposure Routes	6.4E-09					Total Hazard Index Across All Media and All Exposure Routes	0 0071

Total NOAEL HI = 0 0071

TABLE 4-27-RME (Revised)

SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs

REASONABLE MAXIMUM EXPOSURE

MID-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	River Water	Mid-Hudson River	PCBs	4 6E-08	--	--	4 6E-08	PCBs	NOAEL	0.019	--	--	0.019
						Total Risk Across All Media and All Exposure Routes	4.6E-08					Total Hazard Index Across All Media and All Exposure Routes	0 019

Total NOAEL HI = 0.019

Figure 2-1 (Revised)
Average PCB Concentration in Brown Bullhead
Mid-Hudson River

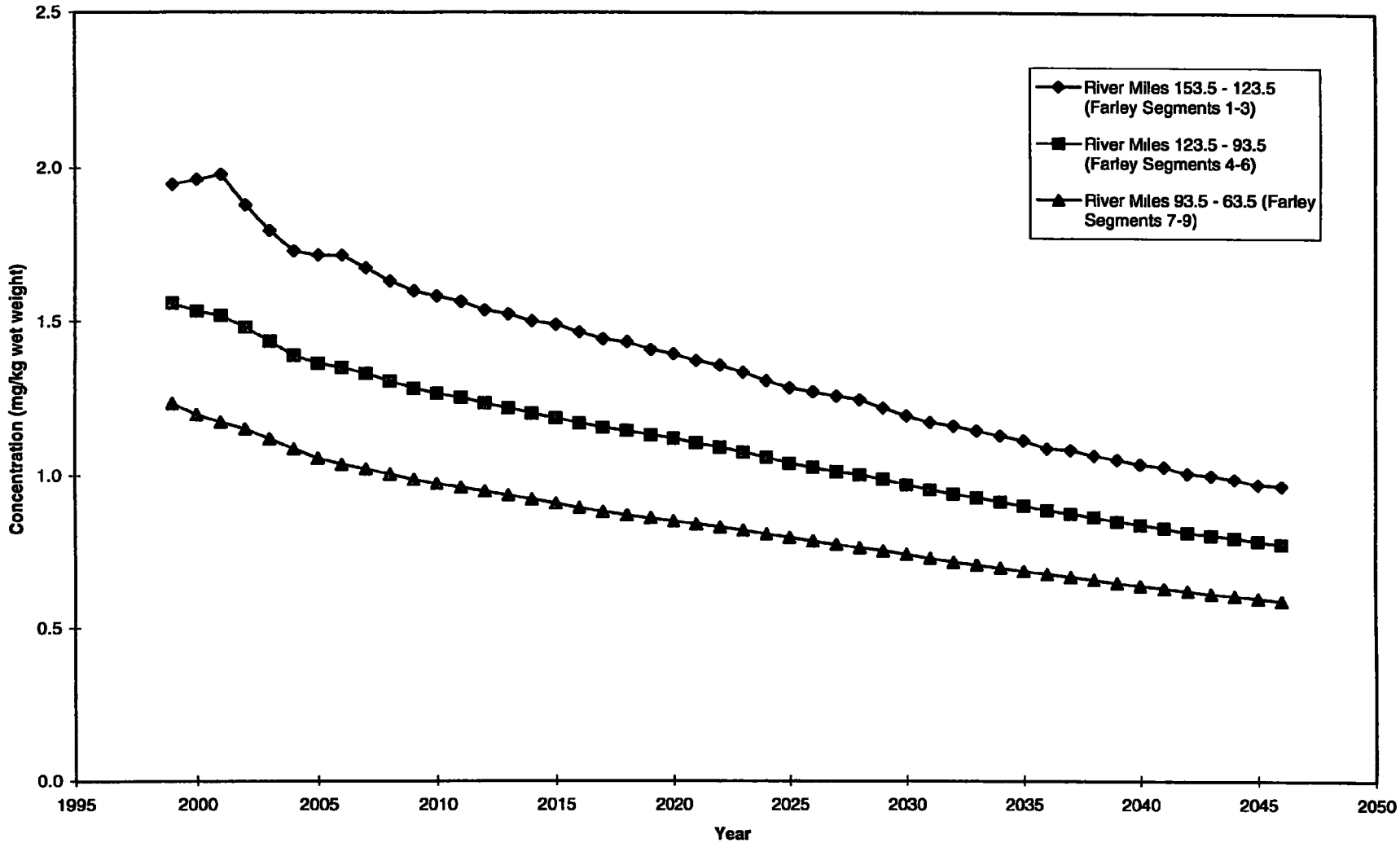


Figure 2-2 (Revised)
Average PCB Concentration in Yellow Perch
Mid-Hudson River

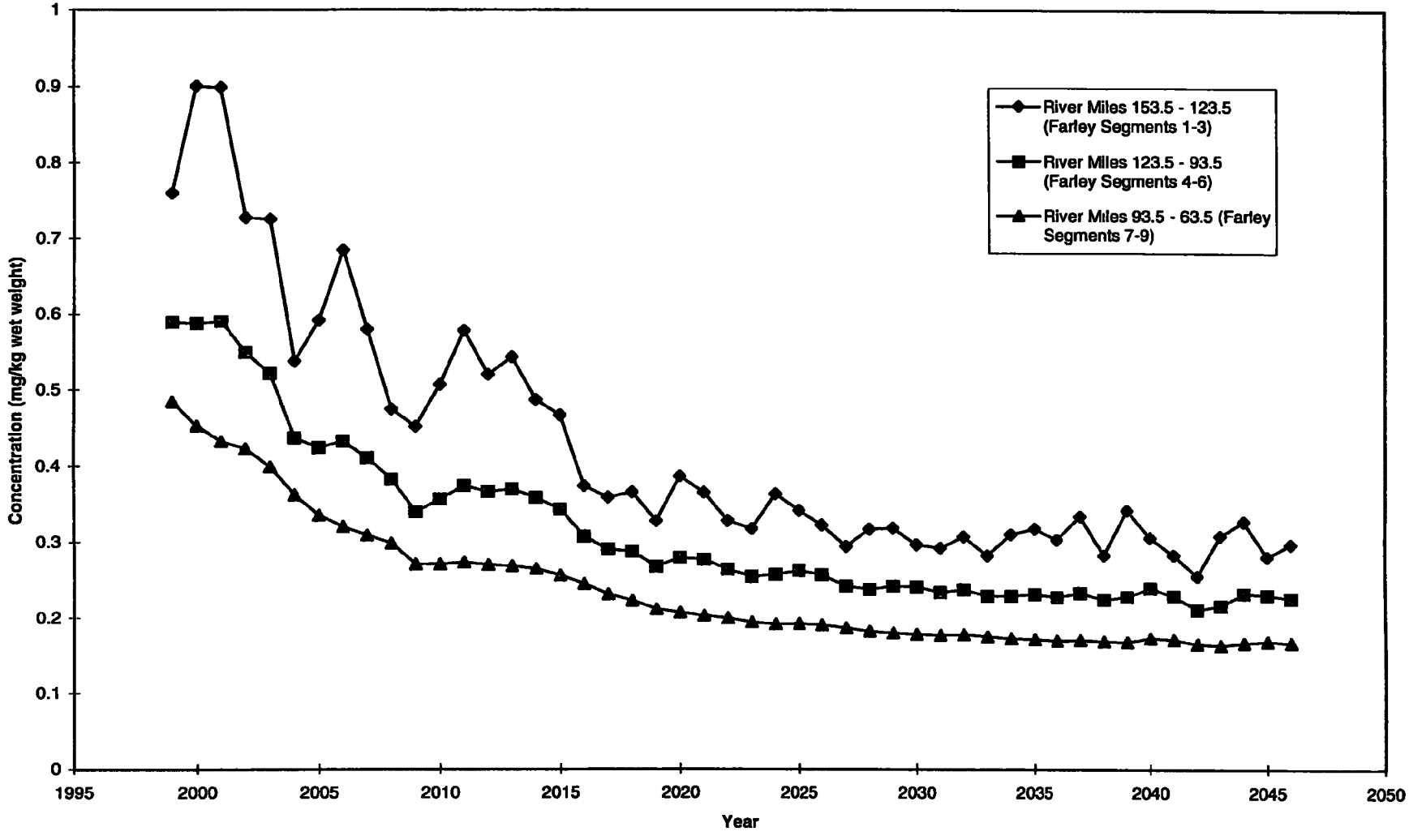


Figure 2-3 (Revised)
Average PCB Concentration in Largemouth Bass
Mid-Hudson River

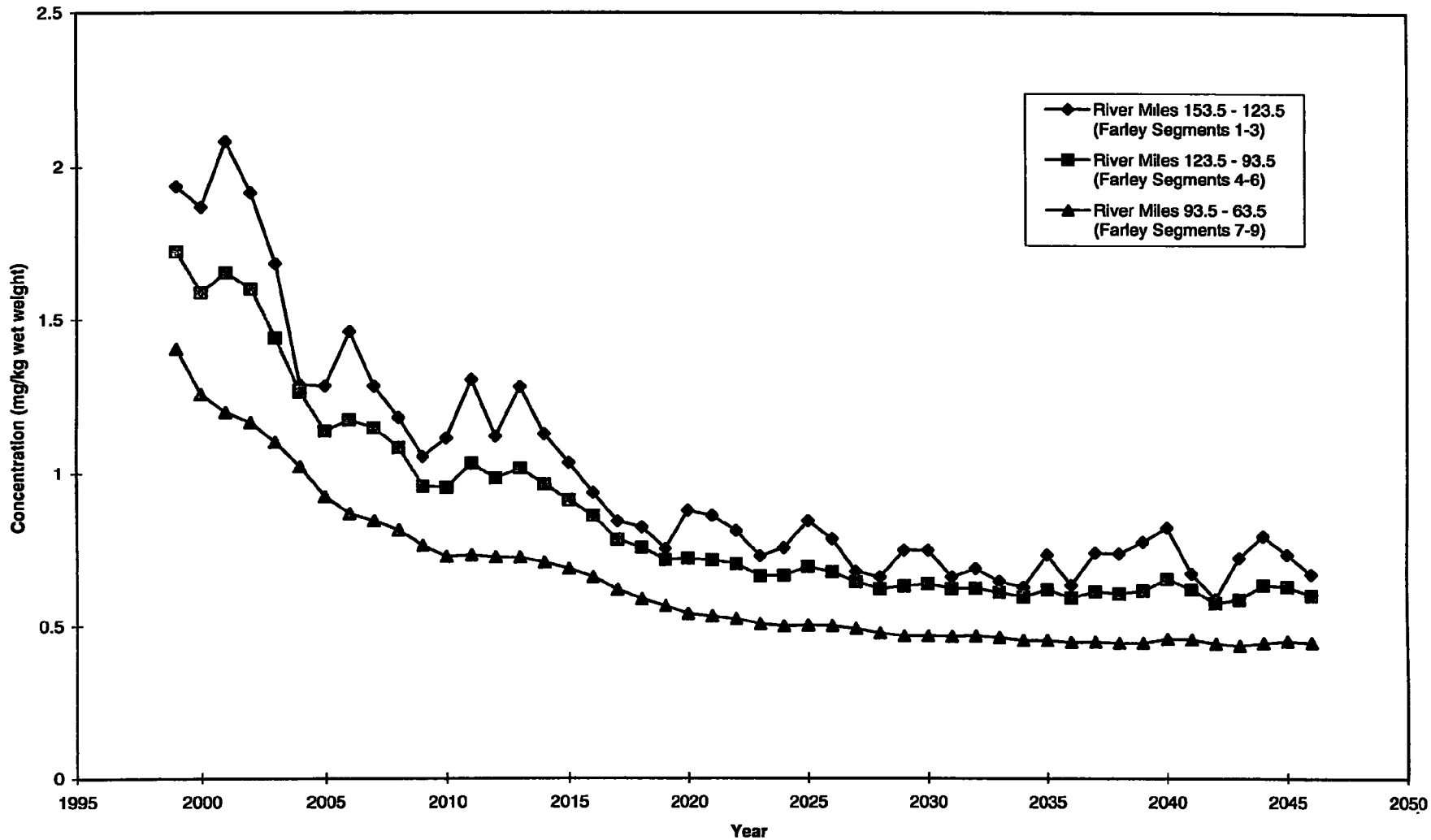


Figure 2-4 (Revised)
Average PCB Concentration in Striped Bass
Mid-Hudson River

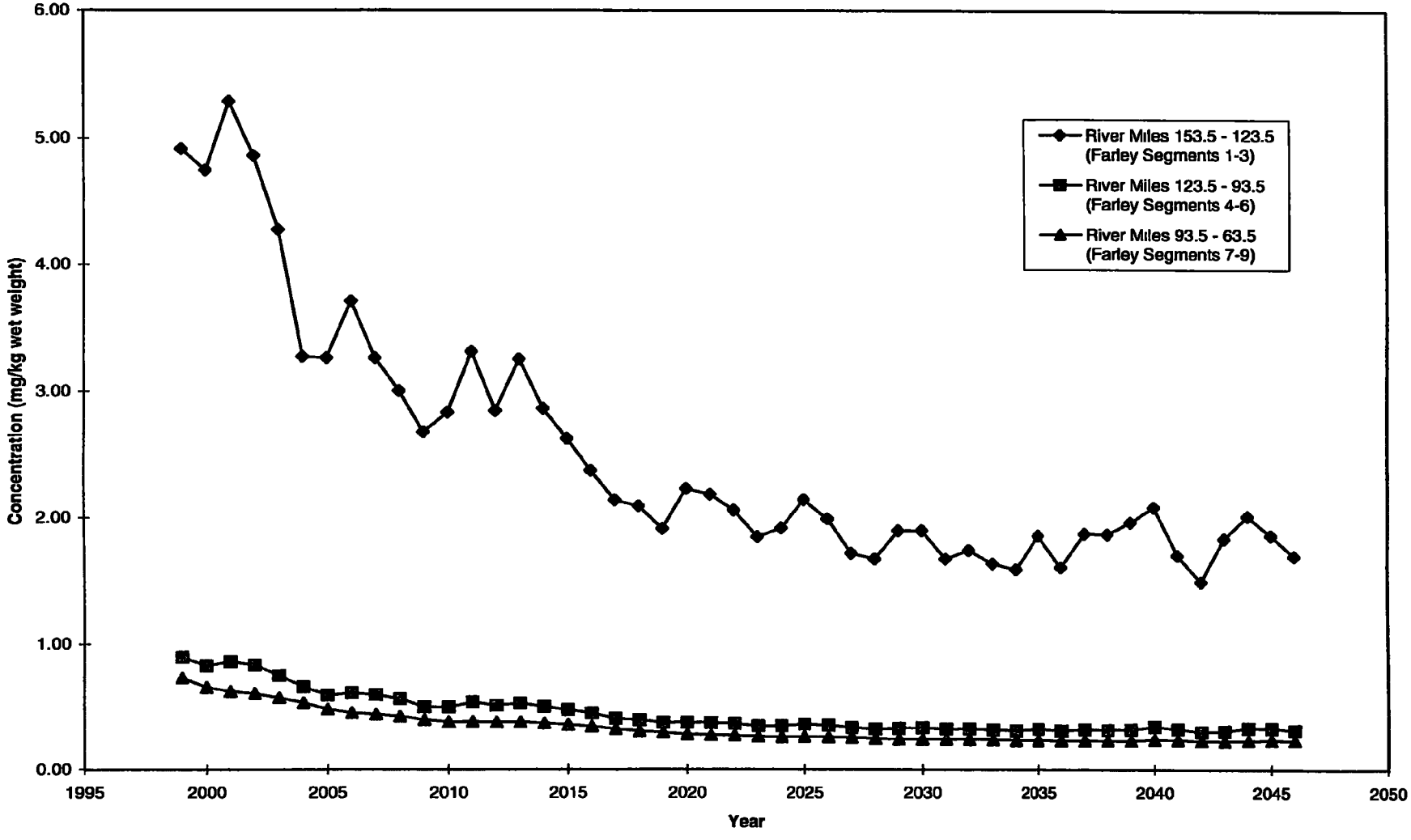


Figure 2-5 (Revised)
Average PCB Concentration in White Perch
Mid-Hudson River

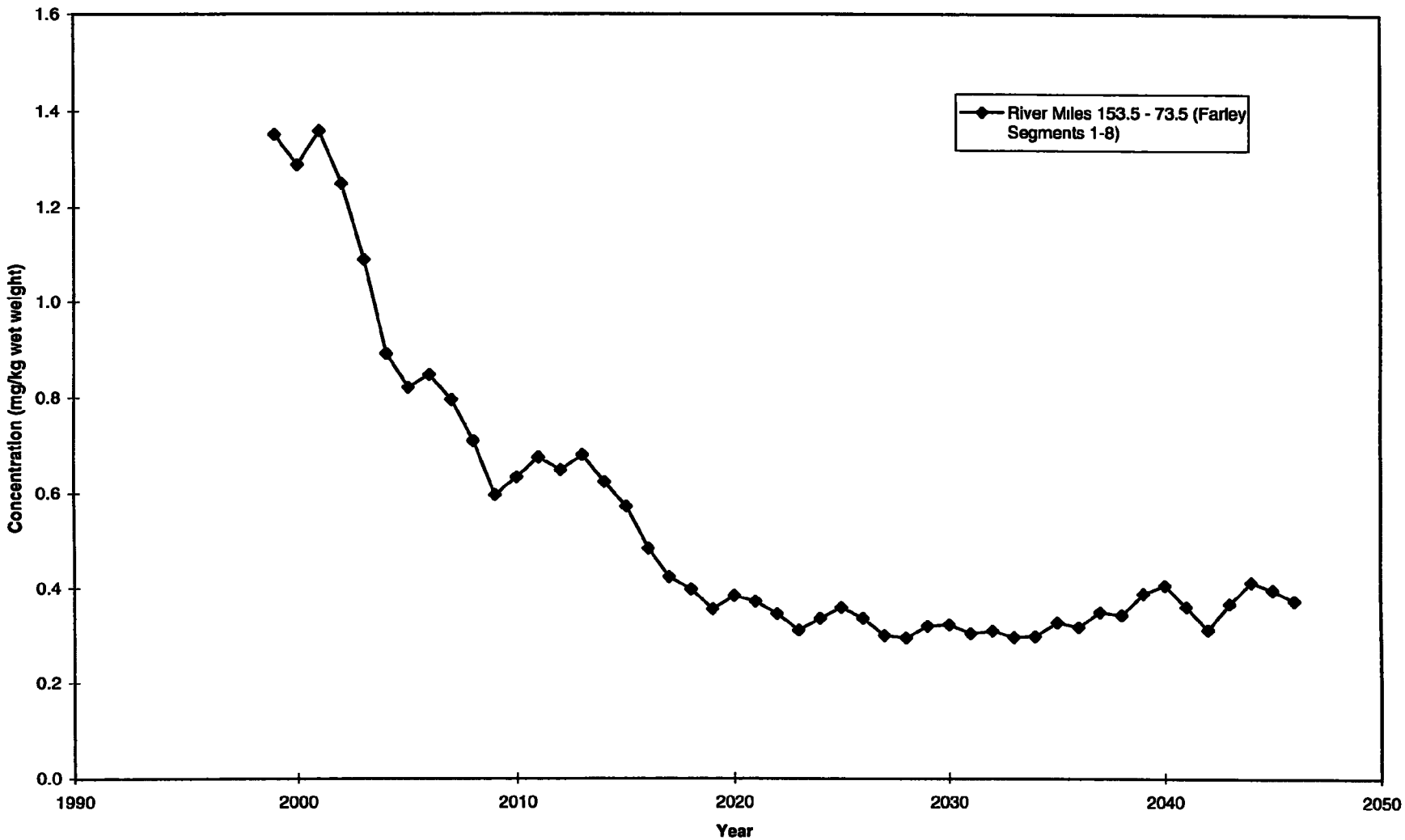


Figure 2-6 (Revised)
Average PCB Concentration by Species (averaged over location)
Mid-Hudson River

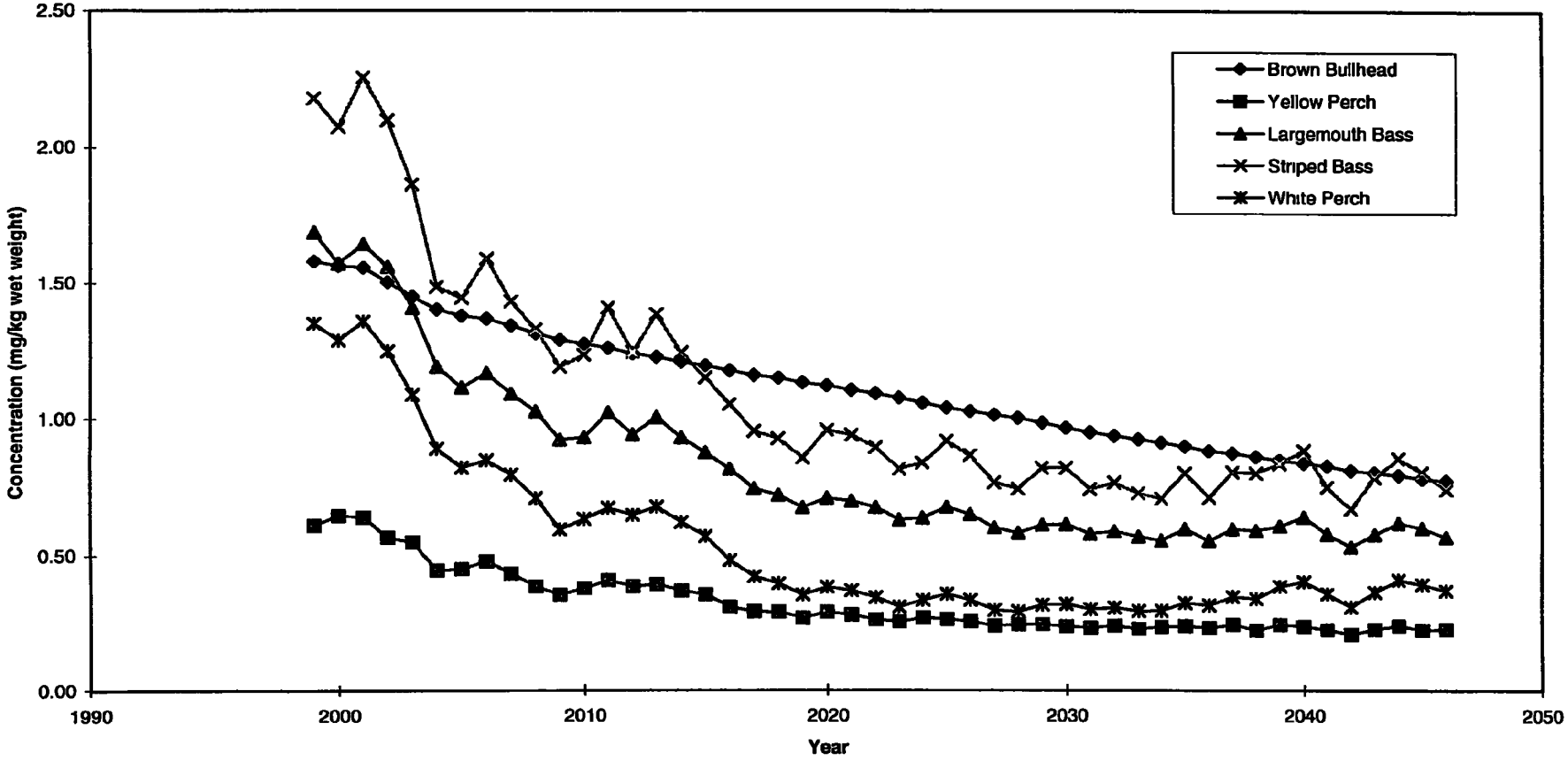


Figure 2-7 (Revised)
Average Total PCB Concentration in Sediment
Mid-Hudson River

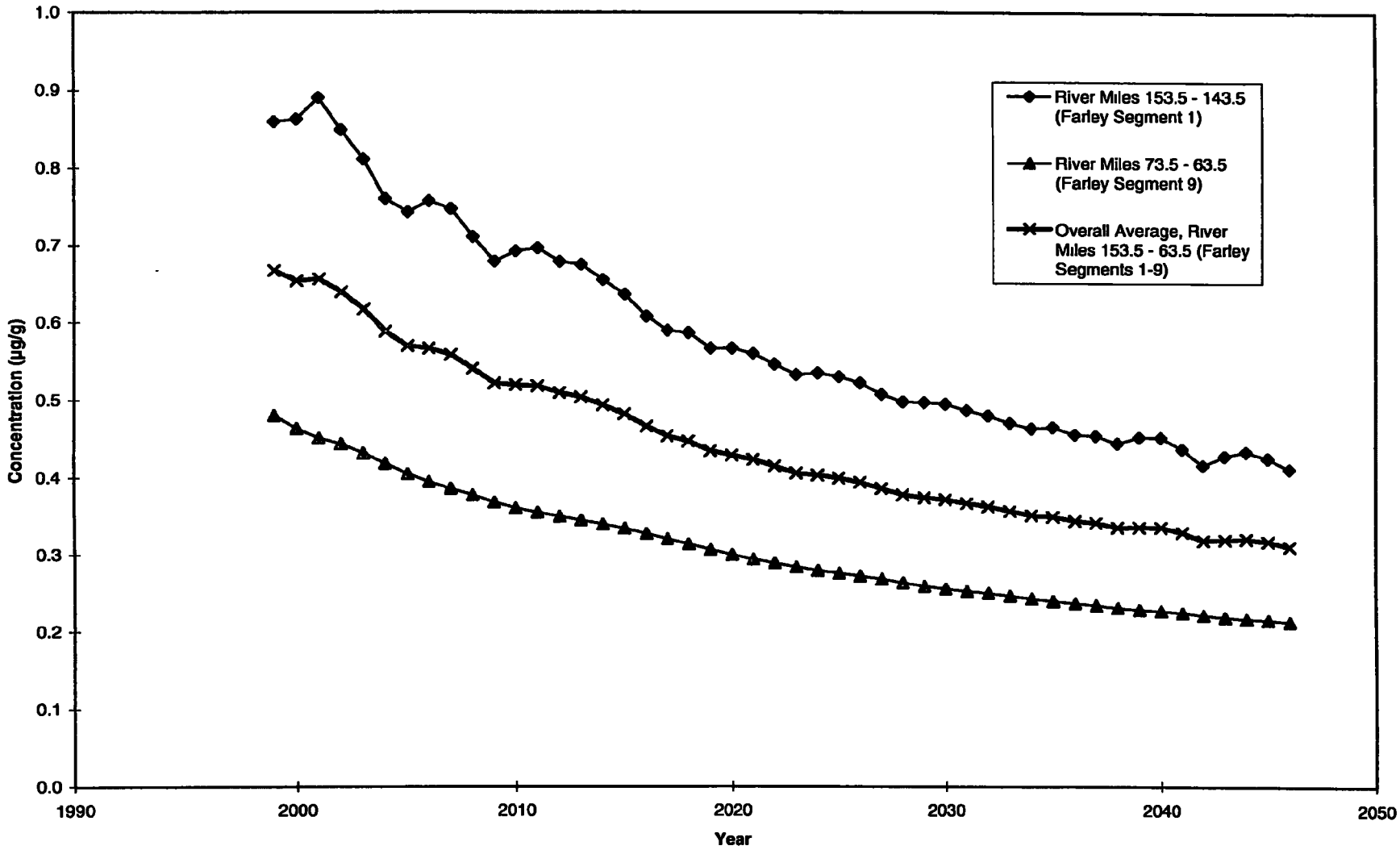
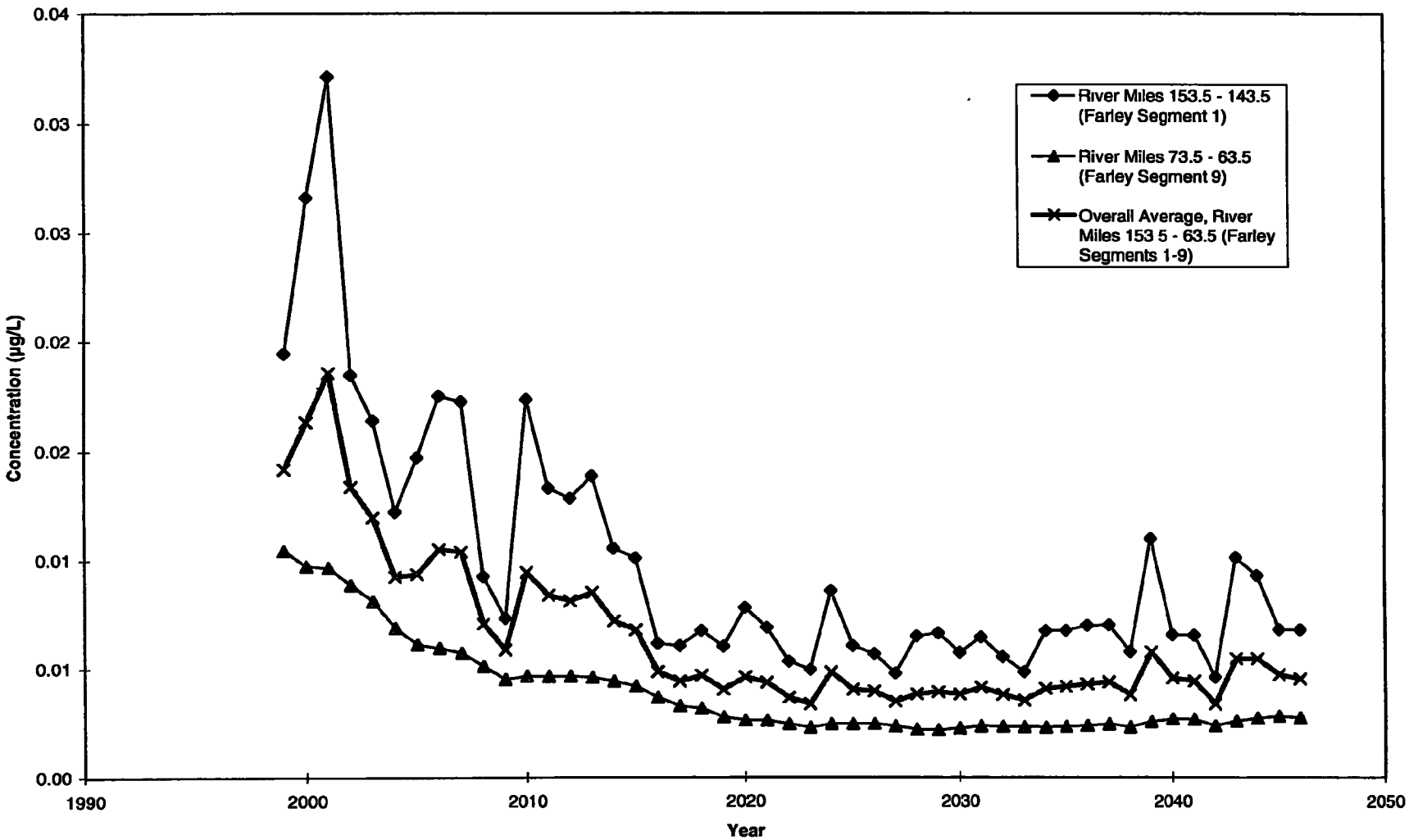


Figure 2-8 (Revised)
Average Total PCB Concentration in River Water
Mid-Hudson River



Comments

Federal

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

January 28, 2000

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 December 1999 Phase 2 Report - Review Copy, Further Site Characterization and Analysis, Volume 2F- Human Health Risk Assessment for the Mid-Hudson River, 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 for the Mid-Hudson River (Mid-Hudson HHRA) assessed exposures and risks to children, adolescents and adults from PCBs between the Federal Dam at Troy to just south of Poughkeepsie. 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 Mid-Hudson 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, consumption of drinking water, and dermal contact with sediment and river water. Inhalation of volatilized PCBs in air was not evaluated since it was shown to be insignificant for the Upper Hudson and concentrations are lower in the Mid-Hudson study area. Species-weighted PCB fish concentration distributions (brown bullhead, largemouth bass, white perch, striped bass and yellow perch), area-weighted sediment and area-weighted water concentration were derived from the Baseline Modeling effort (Farley's fate and bioaccumulation model and EPA's bioaccumulation (FISHRAND) model).

Ingestion of fish resulted in the highest cancer risk (i.e. adult, 9×10^{-6} central tendency, 4×10^{-4} high end; child, 3×10^{-6} central tendency, 1×10^{-4} high end) with the high end or reasonably maximally exposed (RME) more than 100 times greater than EPA's goal of protection. Exposure from sediment 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 (adult Hazard Index (HI)=3) and RME (adult HI=30; child HI=10) point estimates exceeded acceptable levels.

Lifetime cancer risks for exposure to sediment or water, or inhalation of air ranged from 10^{-8} the 10^{-9} for central tendency risk and 10^{-7} the 10^{-8} for RME risk. For non-cancer effects, the HQ associated with exposure to sediment and water was significantly less than one.

General Comments

Two HHRAs were performed during the RRI/FS. Neither the August 1999 baseline HHRA for the Upper Hudson River nor the December 1999

The baseline HHRA for the Mid-Hudson River represents the second component of the human health risk assessment for the Hudson River Superfund site. The risk assessment will not be complete until there is an evaluation of the human health risk for the entire site, including the Lower Hudson River between Poughkeepsie and the Battery, the southern site boundary. The Mid-Hudson HHRA concludes that ingestion of fish is the primary pathway for humans to be exposed to PCBs and that risk for cancer and noncancer health effects exceed EPA's goals of protection. The decision to limit determination of human health risk to the Upper and Mid-Hudson to the exclusion of the Lower Hudson means that potential human health risks associated with the consumption of PCB-contaminated fishery resources and the potential effect of remedial decisions will not be fully evaluated. HF-1.1

NOAA submitted extensive comments (dated 7/1/99, 1/28/00) on the fate and transport and bioaccumulation components of the baseline modeling effort. These comments should be reviewed and their implications to the Mid-Hudson 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, which may result in significant underestimation of resuspension of sediments and/or PCB loading to the river. Furthermore, calibration of the Farley model was not performed. 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 mid-Hudson HHRA since the modeled sediment and water concentrations drive the fish exposure concentrations that are used to derive risk to the public. Moreover, results of supplemental work on the fate and transport and bioaccumulation models will be released at the end of January 2000. 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 Mid-Hudson HHRA. HF-1.2 HF-1.3

Specific Comments

Page 9 and elsewhere: The exposure assessment assumes a start date of 1999. The assumption that no exposure occurred prior to that date, could underestimate risk. HF-1.4

Page 10: "Carp, catfish, and eel were assigned the same PCB concentration as brown bullhead". NYSDEC fish collections include sizable samples of carp, American eel and white catfish. Data for carp, catfish and eel should have been examined for comparability prior to assigning brown bullhead concentrations to these three fish species. HF-1.5

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

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
Kathryn Jahn, USFWS
William Ports, NYSDEC
Ron Sloan, NYSDEC
Sharon Shutler, 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
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HS-1

Environmental Conservation 30 YEARS
John P. Cahill
Commissioner

February 4, 2000

Allison A. Hess
Project Manager
U.S. Environmental Protection Agency
Region 2
290 Broadway, 19th Floor
New York, New York 10007-1866

Dear Ms. Hess:

Re: Hudson River PCB Reassessment RI/FS
Site No. 5-46-031

Enclosed are comments prepared by the New York State Department of Health on the Phase 2 Report - Further Site Characterization and Analysis, Volume 2F - A Human Health Risk Assessment for the Mid-Hudson River, Hudson River PCBs Reassessment RI/FS, dated December 1999.

If you have any questions regarding the comments please contact this office at 518-457-5637.

Sincerely,

William T. Ports

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

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

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

January 28, 2000

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
Mid-Hudson River PCBs
Saratoga County
Site #546031

Dear Mr. Ports:

We have reviewed the United States Environmental Protection Agency's (US EPA) December 1999 "Phase 2 Report - Review Copy, Further Site Characterization and Analysis, Volume 2 F – A Human Health Risk Assessment for the Mid- Hudson River, Hudson River PCBs Reassessment RI/FS." This human health risk assessment (HHRA) is specific for exposure to PCBs in the mid-Hudson River which extends from the Federal Dam at Troy, New York to just south of Poughkeepsie, New York. It is a companion to EPA's August 1999 HHRA for exposure to PCBs in the upper Hudson. Both of the assessments are based on the same methodology and toxicity evaluation, although more detail and discussion is found in the August 1999 HHRA. For these reasons, almost all of our September 7, 1999 comments on the upper Hudson HHRA apply to the mid-Hudson HHRA.

We agree with the overall conclusion of the assessment that the highest estimated human health risk due to PCBs in the mid- 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 technical comments and concerns that should be addressed before finalizing 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

HS-1.1

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.

2. New York State Department of Health (NYS DOH) staff has compared elements of the assessments prepared by US EPA's consultants for the Hudson River and Rogers Island sites. There are numerous differences in the approaches used in the 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 Hudson River and Rogers Island risk assessments unless there are valid technical reasons for not doing so. HS-1.2

3. In a May 20, 1998 letter from Robert Montione to William Ports of the NYS Department of Environmental Conservation, NYS DOH staff provided comments on the US EPA Scope of Work for the Hudson River HHRA. Two comments not addressed in the mid-Hudson HHRA are: 1) The point estimates for high-end risk should include lifetime Hudson River fish consumption (comment 3) and 2) The HHRA should address the effects of past exposures on current and future health risks (comment 4). HS-1.3 HS-1.4

Addressing these issues would provide valuable information to risk managers.

EXECUTIVE SUMMARY

1. Page ES-3 -- The statement that for the fish ingestion pathway, "Both cancer and non-cancer health hazards to an adult angler and a child were calculated" is incorrect. Such calculations only appear in the Executive Summary. The child receptor for the fish ingestion pathway must be incorporated into Chapter 2 - Exposure Assessment and Chapter 4 - Risk Characterization. Furthermore, the risks to children from fish ingestion (pages ES-4 and ES-5) are calculated by simply dividing the adult cancer risk or hazard index by 3, based on the assumption that a child's meal size is approximately 1/3 of an adult's meal size (no reference provided). This calculation fails to account for differences in body weight that would result in higher estimates of daily exposure for children than adults. The approach taken to calculate the child's cancer risk is also flawed because cancer risk estimates are based on 12 years exposure (central tendency) and 40 years exposure (RME), while a person has a child's body weight and meal size for only a fraction of these time periods. Due to the shorter duration of exposure assumed for noncarcinogenic risk (e.g., the assessment assumed that chronic exposures are those which exceed 7 years), the assessment should evaluate exposures and noncarcinogenic risk for at least the high-end child fish consumer. See our comments on Chapter 2 - Exposure Assessment for additional information. HS-1.5

2.. Page ES-4 and Chapter 4 (page 26) -- Statements about an 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 HS-1.6

state (e.g., US EPA, 1991, Risk Assessment Guidance for Superfund: Volume 1 – 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.

3. Page ES-6 (second bullet) -- The HHRA calculates increased cancer risks to individual receptors. Thus, it is recommended that the first sentence be changed to "Under the RME scenario for eating fish, the calculated increased risk is approximately 4 in 10,000". HS-1.7

CHAPTER 2 - EXPOSURE ASSESSMENT

1. The PCB Concentration Weighted by Species-Consumption Fractions section on page 10 and Table 2-7 describe how the assessment classified eight species of fish consumed by Mid-Hudson River anglers into five groups. For Group 1, the assessment uses PCB levels in brown bullhead to represent PCBs in carp, catfish and eel "because, like bullhead, they tend to spend much of their time at the bottom of lakes, rivers, and streams." This is inappropriate because brown bullhead generally have lower PCB levels than American eel, carp or white catfish; for example, 1992 collections of brown bullhead, American eel and carp/goldfish at Albany/Troy and white catfish at Catskill had average PCB levels of 3.1, 9.1, 9.2 and 8.8 ppm, respectively. HS-1.8

2. As discussed in our comments on the Executive Summary, PCB exposures and noncarcinogenic 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 Chapter (Chapter 4) should include a comparison of the modeled fish concentration over time for the different sections of the Mid- 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 - TOXICITY ASSESSMENT

1. As in the HHRA for the upper Hudson, the assessment for the mid-Hudson maintains an artificial dichotomy between the toxicity values for the cancer and non-cancer effects of PCBs. For example: HS-1.12

- The toxicity values used to evaluate the cancer and non-cancer human health risks of the same exposure (water ingestion, sediment ingestion, dermal contact with sediment, dermal contact with water) are based on different 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	1242	1016
fish ingestion	1254/1260	1254
sediment ingestion	1254/1260	1016
dermal contact with sediment	1254/1260	1016
dermal contact with water	1242	1016

2. On page 23, 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. We suggest, however, that the same scientific reasoning be applied to the selection of

cancer slope factors (CSFs) to evaluate the cancer risks of exposure to sediments and water. 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 in the upper Hudson HHRA is consistent with this advice. If the CSFs used to assess sediment and water 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 water should be discussed in the Chapter on Risk Characterization.

CHAPTER 4 - RISK CHARACTERIZATION

1. As in the upper Hudson HHRA, the discussion (pages 25-27) 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. HS-1.13

- 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." 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 not be adequate.

- The average daily dose for an adult high-end angler is 0.6 ug/kg/day. The LOEL used to derive the Aroclor RfD is 5 ug/kg/day. Thus, the adult angler's dose is only about 8 times lower than the animal 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 discussion on pages 76-77 of the upper Hudson HHRA 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 in the upper Hudson HHRA) 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. A comparative summary of the information (critical studies, critical effects, and uncertainty factors) for the Aroclors 1016 and 1254 would provide useful information for the reader and risk manager.. HS-1.14

APPENDIX C - TOXICITY PROFILE (UPPER HUDSON HHRA)

[The comments below were provided on the Upper Hudson HHRA and should be considered when finalizing the mid-Hudson HHRA]

1. 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-1.15

- 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).

- 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.

- 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).

- 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.

- 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.

I hope that our comments and suggestions will assist EPA in finalizing the HHRA. 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

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

HL-1

SARATOGA COUNTY

ENVIRONMENTAL MANAGEMENT COUNCIL

PETER BALET
CHAIRMAN

GEORGE HODGSON
DIRECTOR

January 26, 2000

Alison A. Hess, CPG
USEPA, Region 2
290 Broadway, 19th Floor
New York, N.Y. 10007-1866

Dear Ms. Hess:

Enclosed you will find the Saratoga County Environmental Management Council's (SCEMC's) comments on the **Baseline Ecological Risk Assessment For Future Risks in the Lower Hudson River** and the **Human Health Risk Assessment for the Mid-Hudson River** prepared by the Council's chief technical advisor, David Adams.

Many of the SCEMC's previous comments on the Hudson River Reassessment's Phase 2 Human Health Risk and Ecological Risk Assessment Reports transmitted to you on September 2, 1999 apply to these reports as well. The Council believes these latest Ecological and Human Health Assessments also reflect an unrealistic and excessive degree of "scientific" over-conservatism in calculating the human health and ecological risks.

In the enclosed comments, David Adams makes a number of appropriate and what we feel are valid observations relating to the unavailability and inconsistencies of important modeling information not being provided to the public for its review prior to its being used by EPA in these reports. The unavailability of EPA's revised baseline modeling information and EPA's lack of agency/peer review of the Farley model are important areas of methodological concern as these tools are crucial in determining the magnitude of the Reassessment's risk assessments. The SCEMC requests, at this time, a copy of EPA's revised modeling information for our review and comment. This information should also be provided to all Reassessment public information repositories.

Once again, it becomes apparent that **EPA has not developed an adequate overall methodological framework for the Reassessment** when it relies on a model (Farley's) to assess mid and lower river risks which requires PCB monitoring information on a homolog basis rather than a congener basis which was the type of data collected during the Reassessment monitoring period. This lack of adequate pre-project planning now requires the need for data conversion which introduces yet "another undefined level of uncertainty into the calculated risks". The Council also feels it is inappropriate to utilize a limited number of striped bass samples to draw what we believe to be erroneous conclusions in regarding PCB concentrations found in largemouth bass populations. Again, the need for additional PCB Homolog sampling for

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representative fish species found in the mid and lower Hudson River should have been anticipated and is indicative of the poor methodological planning inherent throughout EPA's Hudson River PCB Reassessment process.

Sincerely,

Peter M. Balet
Chairman

Enc.
cc: Doug Tomchuk, USEPA, Region 2
SCEMC Members
Darryl Decker, Chr., Government Liaison Committee, CIP
The Honorable John Sweeney
John Wanska, USGAO
Dr. George Putman, Scientific & Technical Committee, CIP
William Ports, NYSDEC
Ned Sullivan, Scenic Hudson

SARATOGA COUNTY

ENVIRONMENTAL MANAGEMENT COUNCIL

PETER BALET
CHAIRMAN

GEORGE HODGSON
DIRECTOR

COMMENTS ON PHASE 2 - VOLUME 2E A BASELINE ECOLOGICAL RISK ASSESSMENT FOR FUTURE RISKS IN THE LOWER HUDSON RIVER AND ON VOLUME 2F A HUMAN HEALTH RISK ASSESSMENT FOR THE MID-HUDSON RIVER HUDSON RIVER PCB'S REASSESSMENT RI/FS DECEMBER, 1999

Prepared By: David D. Adams, Member, Saratoga County EMC and Government Liaison Committee, January 2, 2000

General Comments

HL-1.1

1. Both of these risk assessments and the revised EPA FISHRAND Model for the Upper Hudson River are based on the revised EPA PCB Fate and Transport Model and the Farley, et. al. Model for the Lower Hudson River. Reports describing these models and the model results were not made available by EPA with the risk assessment reports. It is improper for EPA to present reports to the public for review and comments when information vital to the review is not available to the general public. Before presenting these reports, EPA should have made the revised EPA model reports and the Farley, et. al. Model report available in the designated PCB Reassessment repositories for review along with the risk assessment reports. I was able to obtain a copy of the Farley, et. al. Model report through the courtesy of Alison Hess of EPA. Results of my review of the Farley Model are presented as appropriate in the comments on the Risk Assessment Reports. My review was constrained, however, by not having the model revisions made after March, 1999. EPA is requested to forward information on these revisions. I still await the revised EPA model reports which have not yet been issued.

HL-1.2

2. In EPA's public presentation of the Risk Assessment Reports, EPA stated that EPA does not plan to review the Farley Model. The reason given was that the Reassessment and subsequent remediation decision being done by EPA is for the Upper Hudson only. The logic of this position is difficult to understand. If the risk assessments of the Mid and Lower Hudson are of no significance to EPA's study of the Upper Hudson, then why were the risk assessments done? If the results of the risk assessments may have bearing on EPA's decision about remedial action in the Upper Hudson, then EPA owes the public the assurance that the risk assessments have been done on a sound basis. This assurance requires EPA's review of the Farley Model and also review by an appropriate independent review panel. EPA is requested to respond as to the use of these risk assessments and based on that response, as to whether the Farley Model will be reviewed. While overall the Farley Model appears

to be a good and credible model, the following are some of my questions/concerns that arose from my review of the report by Farley, et. al. which illustrate why review of the Farley Model is needed:

a. The very sharp concentration gradient shown in Fig. 1-1 for di PCB's between RM159 and RM144 is suspect as it is not clear what could cause such a gradient. Also, there is no explanation for the second bar graph at RM159. If this bar graph is selected, the sharp gradient for di disappears. Is it possible there is something wrong with the data presented in the first bar graph?
b. In many places, values of parameters are stated or assumed with little or no justification. Examples are the sediment thicknesses assigned to each model segment (p. 19); the use of the 1989 Mohawk River and Upper Hudson River flows as a constant yearly flow repeated annually throughout the PCB simulations (P. 24); sedimentation rates, suspended solids concentrations, settling velocity, suspended sediment loads from the Upper Hudson and Mohawk River during high and low flow periods, sediment loads from the Lower Hudson Watershed and their distribution in the model segments (P. 26); production rate of solids by phytoplankton, the stoichiometric conversion factor, the decomposition percentage for phytoplankton, and average-annual sedimentation rates (P. 27); fraction of organic carbon in sediments (P. 30); the values for a_{DOC} (P. 56); use of Mohawk River PCB concentrations for Passaic, Hackensack, and Puritan Rivers (P. 40).
c. The specification rather than modeling of hydrodynamic, organic carbon, and sediment transport (P. 18).
d. The lack of data to support model calculated values (see P. 28 & Fig. 2-5 where data are lacking above RM25 for low flow and RM12 for high flow and P. 55 & Fig. 3-1 where data are lacking below RM80).
e. The assignment of PCB initial conditions for sediments for model segments missing sediment cores. Based on the distribution of cores, it appears only 6 or 7 segments out of 26 segments in the model have core data (PP. 41 & 45).
f. There seems to be a very large number of parameter adjustments required to calibrate the bio-accumulation model (P. 54).
g. The rather poor fit in several instances of the data to the model calculations for PCB homologue concentrations in surface sediments (P. 59 & Fig. 3-5).
h. The apparent over prediction of total PCB's in perch (P. 75 & Fig. 3-14).

HL-1.3

3. EPA also stated in its public presentation that the only PCB source considered to the Lower Hudson was the PCB's coming over the Troy Dam. While I could not find an explicit statement in the model discussion in the Ecological Risk Assessment Report to this effect, the presentation in the Report appears to be based on the Upper Hudson as the only source to the Lower Hudson. Farley, et. al. state on P. 41 of their report that while the Upper Hudson dominated the loading to the Lower Hudson in the early 1990's, the Upper Hudson loads continued to decrease in the 1990's and by 1997 are estimated to be slightly less than one-half of the total PCB load to the Lower Hudson. EPA is requested to justify assuming all the PCB loading comes from the Upper Hudson in view of the position stated by Farley, et. al. As a minimum, EPA should provide values for the risks assuming that the Upper Hudson load is eliminated and 50% of the PCB load to the Lower Hudson remains into the future as no action to remove these loads appear to be underway. These risk values would put into proper perspective the possible contribution of PCB loads from the Upper Hudson to risks in the Lower Hudson.

HL-1.4

4. Much of the information in the December, 1999 reports regarding such items as exposure and toxicity assessment is a copy of similar information in the August, 1999 Risk Assessment Reports for the Upper Hudson. Comments were previously submitted on these sections for the Upper Hudson in the Saratoga County EMC's letter to EPA of September 2, 1999 as corrected by the EMC letter of October 1, 1999. Therefore, the earlier comments will not be repeated here but will be referenced as appropriate.

HL-1.5

5. The need to convert EPA model Upper Hudson PCB inputs to the Farley Model from tri+congeners of the EPA model to the homologue distribution of the Farley Model, as discussed in App. A of the Ecological Risk Assessment, is another example of the lack of planning which has plagued EPA's investigation since the beginning. The need for evaluation of the Lower Hudson should have been seen at the start of the study and plans made to obtain data and a model which would fit together without the manipulations of App.A which introduce another undefined level of uncertainty into the calculated risks. Comments on the procedure EPA used to make the extrapolation are given later in comments on Appendix A.

Vol. 2E Baseline Ecological Risk Assessment Comments

Section 3.1.1.1; P.15: Please identify the "few changes" needed to make the Farley Model usable by EPA. Also, EPA is requested to provide an evaluation of the potential effects of starting the model over after each 15-year increment with possibly imprecise initial conditions. Is there the possibility of increasing error in the future predictions?

Section 3.1.1.2; P.16&17: The treatment of PCB body burdens for striped bass throughout this report and the comparison Human Health Risk Assessment Report is puzzling and a major source of concern. The discussion starting on P. 16 focuses on predicting striped bass body burdens in Region 1 because the Farley Model only predicted striped bass body burdens as far as Region 2. This focus on striped bass in Region 1 continues throughout both Reports as calculated striped bass body burdens are only reported for RM152 and RM113 whereas calculations are made for other fish species at RM90 and RM50 also. This focus by EPA solely on Region 1 for striped bass is puzzling because apparently Farley, et. al. did not consider striped bass to be significant in Region 1. The Farley report discusses the migratory behavior of striped bass on P. 78 and following pages of the report. This discussion only mentions striped bass as going as far north as Region 2 which ends at RM73.5, implying Farley, et. al. felt no need to consider Region 1. It must be that some striped bass appear in Region 1 as EPA on P. 16 discusses data at RM152 and RM113. However nowhere in the EPA reports are the data for striped bass shown. Comparisons of model results to data for other fish species are shown in Fig. 3-12 but not for striped bass. Therefore, there is no way of evaluating the significance of the data on striped bass for Region 1. EPA is requested to provide an explanation of the basis for considering body burdens in striped bass at RM152 and RM113 while excluding striped bass at RM90 and RM50. The Farley, et. al. report would indicate just the opposite. EPA is also requested to furnish information on the number and age of fish samples of each species sampled at the RM's152, 113, 90 and 50 used in the risk analysis so the size of the data base on which the model is based can be evaluated. EPA should also show a comparison of the model results to the data for striped bass as was done for other species of fish.

The EPA focus on RM152 and RM113 for striped bass is a major concern because of the significance of striped bass to the risk assessments. In the Human Health Risk Assessment Report, Tables 2-6 and 2-7 show that striped bass are the second largest species eaten by anglers. The concentration of PCB's in

striped bass are the highest of any of the fish species ranging up to twice the PCB concentration in brown bullheads which represent the major fraction of fish consumed (52% per Table 2-7 of Vol. 2F). Thus, the product of the percent species in the diet times the PCB concentration makes striped bass as significant as brown bullhead in contributing to the human health risk from eating fish.

The situation for avian and mammal populations is less clear. While many include fish in their diet, in most cases, but not all, the fish seem to be smaller than striped bass. Because EPA does not provide definitive information, either in the August, 1999 or December, 1999 reports, it is not possible to determine the fraction of the avian and mammal receptors diet that is assumed to come from striped bass but it is likely striped bass contribute in EPA's analysis to at least some of the avian and mammal receptors.

Because of the major significance of striped bass to the risk assessments, it is very important that proper selection be made of the modeled PCB concentrations in striped bass to be used in the risk assessments. The trend for PCB concentration with decreasing river mile shows declining concentrations with decreasing river mile until New York City is reached. Review of Figure 3-18 for largemouth bass from Vol. 2E (the species EPA uses to estimate striped bass PCB concentrations at RM150 and RM113) indicates this decline is not linear but rather decreases from RM113 to RM90, and finally has a much more gradual decline from RM90 to RM50. This trend is important because of how EPA calculates the future yearly PCB concentrations in each fish species used in the human health risk assessment. While not stated, (see comments on Sect. 2.3.1, P. 9 of Vol. 2F) it appears this average is calculated assuming a linear variation with distance. This assumption would overestimate the PCB concentration in largemouth bass and therefore striped bass. Use of a technique such as graphical integration would seem to be a more appropriate way to calculate the average concentration for these species. It is also of note that EPA provides curves vs. time for all fish species at each river mile except for striped bass. EPA is requested to provide the curve for striped bass. But of more consequence is the fact that EPA has chosen to use striped bass concentrations only at RM152 & 113 in both the ecological and human health risk assessments, while using concentrations at RM152, RM113, RM90 and RM50 for all other species in the ecological risk assessment and RM152, RM113, and RM90 in the human health risk assessment. This is done, despite the fact that Farley, et. al. do not even consider striped bass in this region (Region 1) and the likely sharp drop-off in PCB concentration in striped bass from RM152 to RM90.

The approach EPA has taken for striped bass is certainly overly conservative and likely incorrect in calculating the contribution of striped bass to the risk assessments. EPA should recalculate the risks using a more accurate approach. It is recommended that EPA use striped bass concentrations at RM90 in the human health risk assessment, and that the ecological risk to striped bass be evaluated at RM90 and RM50 as was done for other fish species. Whether the lack of striped bass PCB concentrations for these river miles affects the ecological risk to other species at these locations is unclear because EPA has not identified the amount of striped bass in the diets of receptors. In recalculating the PCB concentrations in striped bass, EPA should also define and account for any size restrictions New York imposes on catching and retaining striped bass. Size is related to age and is important because PCB concentration in striped bass decreases with age due to the migratory nature of striped bass as discussed in the Farley, et. al. report on P. 78 and shown by Figs. 3-16 through 3-19 of the report. It is my understanding that NYS limits keeping striped bass to fish 18" or greater. Fish of this size would be expected to be older than 0-2 yr. Age class which exhibits peak PCB concentrations. The excess conservatism in the EPA calculation of PCB concentration in striped bass is illustrated by comparing Table 3-18 of EPA's Vol. 2E with Fig.

3-16 of the Farley report. Table 3-18 shows median values for the years from 1993 to 1997 of 36 to 24 at RM152 and 5 to 3.5 for RM113. For fish born in 1987, Fig. 3-16 gives a mean of about 3 for Food Region 2. Fig. 3-19 shows data points ranging from 1 to 2 (one year about 5) over this time period for fish 6 to 17-years-old.

The use of largemouth bass, which are a non-migratory fish as a surrogate for striped bass, a migratory fish, is in itself questionable. More uncertainty in the calculation for striped bass arises from the large difference between the ratios of striped bass to largemouth bass PCB concentrations at RM152 (2.5) and RM113 (.52) (see P.17). EPA is requested to provide an explanation for this difference as there is no apparent reason for it. What are the ratios for RM90 and RM50? It is also of interest that the ratios (and also those for White Perch) have dropped considerably in recent years. Shouldn't any ratio, if used to calculate striped bass concentrations, be based on the more recent data for future predictions?

Going back to P. 16, EPA is requested to explain why the FISHRAND Model was used for all fish species except striped bass as again the reasons are not apparent. Would using FISHRAND for striped bass eliminate or reduce some of the concerns discussed above? Also, Farley, et. al. make a distinction between ages of striped bass (2-6 yrs. and 6-16 yrs.). Does EPA modeling do this? If not, why not?

Section 3.1.1.3; PP.17&18: Why is there no discussion of the second part of Table 3-3, the period from 4/91 to 2/96? Table 3-3 does not seem to agree with Fig. 3-2. Table 3-3 shows more penta coming from HUDTOX but Fig. 3-2 shows the opposite. Also, Table 3-3 shows a delta of −18 kg for hexa but Fig. 3-2 shows a delta of about −52 kg. Please explain these differences. It would be helpful if EPA would stick to one set of units as less arithmetic would be required.

Section 3.1.1.4;P.20: The comparison of measured striped bass body burdens to modeled values in Fig. 3-9 is for Region 2 only, whereas EPA uses only modeled values in Region 1 in its health risk assessment. EPA is requested to show a plot of the EPA model results vs. data for Region 1 (RM152 & RM113) so the proper comparison can be made.

Section 3.1.1.5;P.21: Referring to Fig. 3-10, would it make more sense to plot the average of FISHRAND values in Region 1 to compare to the Farley Model as it uses averages for Region 1?

Section 3.1.1.6;P.21: EPA is requested to supply a comparison similar to Fig. 3-12 for striped bass. Why are striped bass often omitted from data comparisons?

Section 3.1.2.2;P.23: Please explain what all the "x's represent on Figs. 3-16 & 3-17. It is also noted Fig. 3-17 shows results only for Region 2 despite the title on the figure.

Section 3.1.2.3;P.24: Comparing Fig. 3-16 to Fig. 3-19, it appears the average value for Region 1 from Fig. 3-19 is about 50% higher for the year 2020 than the value from Fig. 3-16, but for Region 2 it appears Fig. 3-16 gives a somewhat higher value. Please explain why this changeover should occur. Would using the Farley Model throughout give more internally consistent results and thus be preferred over FISHRAND? Again, why is there no forecast for striped bass?

Section 3.2, P.25: The selection of a river mile towards the upper end of each range to represent the range is another example of the excessive conservatism in the EPA assessments. Given the known drop

off of PCB body burden with decreasing river mile, using the body burden at the selected river miles instead of an appropriate average over the river mile segment introduces unnecessary extra conservatism.

Section 3.2.4;P.26: The use of brown bullhead results to represent short-nosed sturgeon makes the risk assessment for the sturgeon very uncertain and of dubious value because of the unknown uncertainty. Also the need to extrapolate the fish PCB concentration data from standard fillets basis to whole body wet weight basis produces more uncertainty of unknown magnitude into the risk assessment, again decreasing the value of the calculated risks.

Section 3.3;PP.27-30: These sections are very similar to those in the August, 1999 Risk Assessment Reports. The comments previously submitted on these items apply to this report as well and will not be repeated here.

Section 4; PP.31-36: These sections are very similar to those in the August, 1999 Risk Assessment Reports. The comments previously submitted on these items apply to this report as well and will not be repeated here. Additional comments come from PP. B-10 & B-11 of Appendix B. The presentation in Section B.2.3.1 on P. B-10 answers the question asked in the EMC's comments to the August, 1999 Risk Assessment Reports as to the amount of chlorine in chlophen compared to PCB's. However, no information is given to justify that the behavior in fish of the chlorine in chlophen duplicates that of PCB's. Page B-11 says "Hatchability was significantly reduced in fish with an average total PCB concentration of 170 mg/kg...." I thought Bengtsson's testing was done with chlophen A50 and not PCB's. This sentence should be corrected to state what was actually tested. The discussion here introduces another factor of about 10 conservatism in the results by not using the 170 mg/kg and 15mg/kg data from Bentgsson study but rather the 15 mg/kg and 1.6 mg/kg data. This further adds to the total excessive conservatism in the EPA risk assessments (also applies to other fish species in Section B.2.3 of Appendix B). Does this new conservatism mean that EPA now considers the ecological risk evaluation of these fish species in the August, 1999 risk assessment to be wrong?

Section 5.;P.37-55: Comments previously made on the August 1999 ERA regarding the over conservatism in EPA's risk characterization apply to the report as well and will not be repeated here.

Section 5.2.1.9;P.43: As previously questioned, EPA is requested to explain why EPA reports Measurement Endpoints for striped bass only for RM152 and 113 and why these river miles should be considered at all for striped bass.

Section 5.2.4.1;P.45&46: In view of the unquantified uncertainty in the calculation of body burdens in the shortnosed sturgeon and the positive statements about the health of the shortnosed sturgeon in the last paragraph on this page, why does EPA insist on putting forth a negative risk evaluation for the shortnosed sturgeon? This question also applies to white perch as the discussion on P. 46 again indicates a healthy situation and the discussion at the end of the paragraph represents speculation based on only extremely conservative calculations and is inconsistent with the facts shown by the field studies.

Section 5.4.3;P.50,Section 5.5.3.1;PP.53&54,Section 5.3.3.1;PP.47&48A: EPA is requested to provide information on what trends were seen in the Christmas bird counts. This information would be helpful in assessing what is happening to the health of birds in the region.

Section 5.7.3.1;P.57: The discussion in this paragraph leads to the conclusion that not enough raccoons would be affected by the PCB's in the Hudson to have an impact on the raccoon population so why is EPA insisting on singling out the potential risk to those few raccoons that might be affected?

Section A.2;P.A-2: It is not clear what is meant by the phrase "duplicate samples are equivalent." Does this mean the PCB data from the duplicate samples are exactly equal? If not the case, why weren't the duplicate GE samples averaged as were the EPA duplicates?

Section A.3;P.A-3: EPA is requested to provide some discussion of what factors could effect the geochemical processes and why these factors are not expected to change to justify the assumption made here. The discussion of the steps taken is confusing in thar it appears the first step described applies to Factor 2 and the second step to Factor 1. Is this correct?

Section A.3;P.A-3 and Figs.A-1toA-5: The EPA mean values shown on these figures for the TID (presumably from years prior to 1996) agree more with GE means (see Fig. A-9) for post 1996 data and not at all with GE means for prior 1996 data. Since the GE data set for the TID is much larger (225 samples prior to 1996 and 293 samples after 1996) than the EPA data set of 4 to 12 samples, the use of the EPA data at the TID to calculate the ratio for homologues at Waterford (or the Troy Dam) is very questionable. Shouldn't the GE data be used to calculate the factors in Table A-2? EPA is requested to address this issue regarding the calculation EPA used to get input to the Farley Model.

Section A.3;P.A-4: EPA is requested to provide the citation of the data used as the basis for the statement that there is little evidence of decline in PCB loads at the TID post-1995. Is this still true based on 1999 data?

Section A.3;P.A-4: See comment above on A-3 and Fig. A-1 - A-5 questioning validity of factors given in Table A-2. Also, why should these factors stay constant for 40 years?

Section A.5,P.A-7: The basis for the statement at the top of the page about releases from Baker Falls is unclear. Weren't the major releases from Baker Falls post 1990? If so, EPA is requested to clarify why the post 1990 releases are not of concern.

Vol. 2F – Human Health Risk Assessment Comments HL-1.6

Section 2;PP.5-21: Comments previously submitted on Section 2 of the August, 1999 Risk Assessment apply to this report as well and will not be repeated here. HL-1.7

Section 3;PP.23&24: Comments previously submitted on the August 1999 risk assessment regarding non-cancer toxicity values and cancer toxicity apply to this report and will not be repeated here. HL-1.8

Section 2.3.1;P.8: Comments given above on the Ecological Risk Assessment regarding the EPA approach to calculating PCB concentrations in striped bass apply here also. HL-1.9

Section 2.3.1;P.9: The comment on Section 3.2, P. 25 of the Ecological Risk Assessment applies here also to the selection of river miles to represent sections of the river as do comments about selecting a more appropriate way to average values than straight linear averages.

HL-1.10

Section 2.4.1;P.14: Please confirm that it is the RME value of PCB concentration in the fish that is used in the cancer risk assessment.

HL-1.11

Section 4; PP.25-27: Comments previously submitted on the August, 1999 risk assessment regarding the over conservatism on EPA's risk characterization apply to this report as well, and will not be repeated here.

SCENIC HUDSON INC.

Protecting the Valley's Environment, Town by Town

HP-1

Sent by Facsimile

January 28, 2000

Alison A. Hess, C.P G.
USEPA Region 2
290 Broadway – 19th Floor
New York, NY 10007-1866

RE. Hudson River HHRA/ERA Addendum Comments

The findings of the Human Health Risk Assessment for the Mid-Hudson River and the Ecological Risk Assessment Addendum: future risks in the lower Hudson River continue to underscore the need for an aggressive PCB cleanup of the upper Hudson River. With human health risks and ecological risks exceeding
HP-1.1 acceptable levels into the foreseeable future, for 200 miles of the Hudson River, it becomes even more critical than ever that the EPA move forward with a cleanup decision as soon as possible.

The EPA has pledged to develop and release a plan by the end of this year that will serve as the basis of a cleanup decision. In light of the most recent findings, this process must continue to move forward and no additional delays will be
HP-1.2 acceptable. Any requests for additional study or "side-by-side" peer review should in no way impede the Reassessment schedule. The EPA should move forward with peer review of EPA documents and EPA documents only, despite pressure for "side-by-side" peer review and work towards a cleanup of Hudson River PCBs.

Due to the limited effectiveness of the fish consumption advisories and the continued need for more education about the PCB contamination of fish in the Hudson River, the EPA should continue to assess the risks in the Hudson assuming that such advisories do not exist. Angler surveys have indicated that the majority of anglers eat their catch or give it to family members. In its 1996 survey, the New York State Department of Health found that "two-thirds of anglers fishing between Catskill and the Tappan Zee Bridge continued to report eating their fish at least
HP-1.3 sometimes and almost half (46%) of anglers gave fish away sometimes or frequently. More than half (57%) of anglers in this area ate more fish than advised by the NYS DOH advisories."[1]

[1]Health Consultation: 1996 Survey of Hudson River Anglers, Hudson Falls to Tappan Zee Bridge at Tarrytown, New York, Public Review Draft, February 1999, New York State Department of Health, Center for Environmental Health, prepared under a Cooperative Agreement with U.S. Department of Health & Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, p. 14.

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HP-1.3 (Continued) In both (the Hudson River Sloop Clearwater Survey and the NYS DOH Survey "the fish that anglers kept were among the most contaminated species in each part of the river."[2]

As EPA has concluded the 1996 NYS DOH Angler Survey also concluded that "Some anglers and others who eat fish from the Hudson River are being exposed to levels of PCBs that are a health concern and are at risk of adverse health effects."[3] Institutional controls, such as the fish advisories, are not a substitution for a cleanup of the Hudson River as has been suggested by HP-1.4 the General Electric Company. It is important to note that due to the PCB contamination of fish, women of childbearing age and children are advised not to eat any fish, from any location along the Hudson.

New scientific information concerning non-cancer health effects of PCBs has shown that the Food and Drug Administration 2 parts per million (ppm) level, on which New York State advisories are based, is not adequately protective of human health. The scientific and public health community now advocates a much lower level. Based on EPA's most recent findings for non-cancer health risks that eating fish from the mid-Hudson results in PCB exposure that is 30 HP-1.5 times higher than EPA Hazard Index Reference level, it is imperative that EPA adopt a much lower level than the FDA level of 2 ppm. The EPA should adopt a level no greater than 0.1 ppm as has been done recently by the State of Connecticut for their fish advisories.

The alarming reality that human health and ecological resources of the Hudson River are threatened from Fort Edward to New York City, reminds us that 200 miles of this great River is and will continue to be severely impacted by the PCB contamination that started some 60 years ago. These most recent reports, in conjunction with other EPA findings, indicate that the HP-1.6 sediments are the dominant source of PCBs to the rest of the river system and that the natural breakdown of PCBs is inappreciable, provides compelling and irrefutable evidence for the need to remove PCB-contaminated sediment from the upper Hudson River.

Respectfully Submitted,

Rich Schiafo

Rich Schiafo
Scenic Hudson

[2] Ibid.
[3] Ibid.

HP-2

January 27, 2000

Jeanne M. Fox
Regional Administrator
United States Environmental Protection Agency
290 Broadway
New York, NY 10007-1866

Re: EPA Baseline Risk Assessments of PCBs in the Hudson River

Dear Ms. Fox:

On behalf of over seven hundred Appalachian Mountain Club members in from the Albany region who live near and enjoy the varied recreational resources within the Hudson River watershed, I am writing to comment on the EPA Baseline Risk Assessment of PCBs in the Hudson River. The Appalachian Mountain Club promotes the protection, enjoyment and wise use of the mountains, rivers and trails of the Northeast. Central to our mission is the belief that mountains and rivers have an intrinsic worth and also provide recreational opportunity, spiritual renewal and ecological and economic health for the region.

The findings of the Human Health Risk Assessment for the Mid-Hudson River and the Ecological Risk Assessment Addendum continue to provide scientific evidence supporting the need for a thorough PCB clean up of the upper Hudson River. The EPA's own reports indicate that PCBs from the Upper Hudson River continue to pose a threat for 200 miles of the river. For some species, the report shows, future concentrations of PCBs in the lower Hudson River will generally exceed levels known to cause adverse ecological effects through 2018. Given the on-going threat posed by the PCBs in the river sediment to the environment and to human beings, it is time to move forward with a clean up decision. The Environmental Protection Agency should move steadily towards releasing a decision on clean up plans by the end of this year. For every delay, human health and ecological well being continue to be jeopardized. For this reason, any requests for additional studies or for "side by side" peer review should not obstruct the Reassessment schedule. The river has waited long enough. HP-2.1

New scientific information concerning non-cancer health effects of PCBs has shown that the Food and Drug Administration's level of 2 parts per million (ppm) does not provide adequate protection of human health. The EPA's own findings for non-cancer health risks from consuming fish from the mid-Hudson river show that PCB exposure HP-2.2

is 30 times higher than the EPA Hazard Index Reference level. Due to the levels of risk and of hazard, the EPA should adopt a level of no greater than 0.1 ppm instead of using the FDA level. This action would be supported by much of the scientific and public health community and would set a standard that is more protective of human health.

Fish Advisories are not a substitute for removing PCBs from the river. There is already substantial evidence that many anglers do not follow or understand the posted warnings and share fish caught from the Hudson with members of their families, thus putting multiple lives at risk of adverse health effects. The EPA must continue to assess the risks in the Hudson River with the assumption that the advisories do not exist. HP-2.3

The evidence is in. It is time to move forward with a full and comprehensive clean up of the Hudson River in order to protect human and ecological health. It is time to stop the continuation of exposure to health risks and ecological hazards caused by the presence of PCBs in river sediments and by the movement of PCBs both through the food chain and through high water events. HP-2.4

Thank you for your efforts. If there is any way that I can be of assistance, please let me know.

Sincerely,

Joseph Gardner
Conservation Chair
Mohawk-Hudson Chapter/Appalachian Mountain Club
68 Carson Road
Delmar, New York 12054

HP-3

US EPA Region 2
Ms. Alison Hess
Remedial Project Manager
290 Broadway
New York, NY 10007-1866

Re: Comments Health Risk Assessment, Lower Hudson River

Dear EPA:

Please refer to the attached documents for supporting technical information regarding my comments. The EPA presented documentation indicating that there "might be" a possible health risk with those citizens which consume at 51 one-half pound meals per year, when the central tendency fish ingestion rate has been determined to be six half-pound meals per year (Connelly et al., 1992). The public health protection/ worst case scenario of 51 half-pound meals per year for 40 years is mathematically conservative and is not supported nor denied by current health data, and cannot be related to any community health based studies which may support or deny this conclusion. HP-3.1

At the recent public meeting I asked the question, "Are there any Community Health Based studies which would show any indications of health effects or higher cancer rate in communities which have been exposed to PCB in any fashion in NY?" The answer, to the best of my memory, was related to the amount of time it would take to accumulate this type of information and this was the best way to get answers quickly. The problem is not easy to understand, so why do we expect the answers to come any easier? I believe that PCB's have been in the Hudson River for many years, we have known about this for years, and only recently has anyone tried to quantify the related health effects and show direct health effects to the communities that live in and around the Hudson River. The NY State DEC keeps record of everyone who has obtained a fishing HP-3.2

license in NY. This would probably show families whom have fished for years in and around the Hudson and someone could utilize the money being spent to find actual health effects in communities and families with the hypothetical exposures being presented by EPA at this time. Again, this problem has been present for many years and even the most vehement environmentalist wanting cleanup of the Hudson would have to agree that the Hudson River is in better shape than it was 20 years ago! Also, according to EPA in 1999, NY State still had 79 Fish Advisories in effect for PCBs, Chlordane, Cadmium, Dioxins, Mirex, and DDT.

HP-3.3

HP-3.4

The process to find out possible health effects from PCB's started at least 10 years ago and only in the past year has anyone actually gone into the homes of people potentially effected by PCB's. The New York State DOH and ATSDR have begun studies to quantify any effects in population along the Hudson in Glens Falls and Fort Edward, NY. This information must be apart of any health based decision making process and the ATSDR must perform a Health Assessment for pre and post treatment alternatives to ensure optimal public health protection. We cannot afford to start a "Clean-up" project based upon limited information on the possible health effects that the "clean-up" could cause in the communities. Example: MTBE was placed into gasoline supplies to help stop air pollution, but nobody wanted to study the effects of MTBE in groundwater prior to its release into the environment. To initiate a clean up without studying all potential impacts is irresponsible and I do not want the Hudson River to be another example of a recommended clean-up project gone wrong (like MTBE in groundwater supplies)!

HP-3.5

Please consider the following information while reviewing the immediate EPA Health Risk Assessment and the need (if any) for Immediate action:

100% of New York State's lake acres and river miles are under fish advisories.	HP-3.6
National Academy of Sciences (NAS) found that FDA and State codes should be strengthened to reduce consumption of organisms with high contaminant levels; agencies should support research to determine the actual risks from consuming organisms with contaminants, and States should continue site closures, health advisories, and continue public education about the risks on specific chemical contaminants.	HP-3.7
NAS found that data evaluating contaminant levels in fish do not consistently focus on the analysis of edible tissue. "These analyses, by their design, offer insufficient insight into contaminant levels in the edible portion of the seafood products." Also, "There is an apparent lack of coordination in the development and use of data on chemicals in the aquatic environment among FDA, EPA, and the NOAA, and other States."	HP-3.8
NAS: "the CDC should develop an active and aggressive program, founded on community-based health surveys, to better determine the level and source of seafood-borne illness in the US population."	HP-3.9
The FDA specifies PCB concentration limits of 0.2 to 3 parts per million in infant foods, eggs, milk fat, and poultry fat. These products (concentrations) can be sold to consumers.	HP-3.10
The 51 half-pound meals = about 25 pounds of recreational fish consumption for the maximum exposed individual. The FDA-NAS reported that in 1991 about 4 pounds of recreational fish were consumed per year in addition to the 15-16 pounds of commercial fish per year. Total = about 20 pounds of fish consumed per year, 5 lbs. less than max.	HP-3.11
In 1993, research has shown that nature has some processes already chemically reducing the PCBs present in the Hudson River.	HP-3.12
The ATSDR does not know whether PCBs causes cancer in people. Also, ATSDR with NYSDOH is currently researching effects of PCB exposure by conducting community-based health surveys.	HP-3.13

In addition, there was a reference during the public meeting that the current health advisories are not acceptable means to prevent exposure to health risks. I would like to remind all of us that Public Education and Awareness programs are the backbone of all HP-3.14

public health programs. The reason the USA is in good health is because of the good public health education! Proper refuse and garbage control prevent vector and rodents, washing hands prevent the spread of foodborne disease and infection in hospitals, and even lead poisoning can be avoided by education to avoid high lead content water and using first flush activities if the situation fits the level of protection.

To say in passing that public health education is not an effective method of public health disease prevention is not accurate.

Thank you.

Scott T. LeRoy, MS, REHS/RS, Soil Scientist
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State of New York
Department of Health

Hudson River PCB Research Project Announced

ALBANY, April 23, 1999 - The State Health Department today announced a new research project, PCBs and Health: The Hudson River Communities Project, that will examine the possible effects of exposure to PCBs on the human nervous system. For the project, the Health Department is recruiting 100 residents, both men and women, between the ages of 55 and 74 who have lived in the villages of Fort Edward or Hudson Falls for at least 25 years. These villages are two areas where PCBs have been used in manufacturing operations. In addition, a control group is being recruited consisting of men and women, also between the ages of 55 and 74, who have resided in the city of Glens Falls for at least 25 years. Glens Falls was selected because it is upriver from where PCBs were used in manufacturing operations.

The focus of the project is current and past exposure through the consumption of PCB-contaminated fish or through airborne PCBs. Therefore, to be eligible for the project, participants must not have worked in a job where they may have been potentially exposed to PCBs. Information collected from the Fort Edward/Hudson Falls group will be compared to information collected from the Glens Falls control group. Department of Health researchers will analyze project data to see if the two groups score differently on the nervous system tests, and whether or not the differences are associated with higher PCB exposures and blood PCB levels.

PCBs are a group of 209 man-made chemicals that were used in many commercial and electrical products until their manufacture was banned in the mid-1970s. The manufacturing of PCBs was halted in the United States because of evidence relating to environmental buildup and its potential harmful effects. Edible portions of sport fish from the Hudson River are also known to contain PCBs.

This project is designed to address whether exposure to PCBs may cause biological changes in the nervous system such as memory loss, decreased muscle coordination and control, and decreased sense of smell.

This two-phase project will include interviews, biological sampling, and nervous system tests in Phase I, and environmental sampling in Phase II. The interviews will include questions about participants' consumption and preparation of fish caught locally, residential histories, and lifestyle characteristics such as cigarette smoking. Biological sampling will include collecting a blood sample from each participant and analyzing the samples to determine blood PCB level. The nervous system tests will measure small changes in short term memory, muscular movement abilities, and sense of smell. They will involve identifying odors, shapes or words and performing simple tasks with hands and fingers.

The environmental sampling in Phase II will involve air testing for PCBs in and near participants' homes. Participants will be paid up to $100, including $50 for the interview, blood sampling, and nervous system tests, and another $50 for the completion of the air sampling. This project is funded by the Agency for Toxic Substance Disease Registry (ATSDR) for three years and will begin this summer.

4/23/99-39 OPA

U. S. FOOD AND DRUG ADMINISTRATION
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Seafood Training Program

National Academy of Sciences Report on Seafood Safety

Executive Summary
Seafood Production Distribution and Consumption
Microbiological and Parasitic Exposure and Health Effects
and
Naturally Occurring Fish and Shellfish Poisons

National Academy Press, Washington, D.C.
1991

DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
FOOD AND DRUG ADMINISTRATION

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Executive Summary

OVERVIEW

Fish and shellfish are nutritious foods that constitute desirable components of a healthy diet. Most seafoods available to the U.S. public are wholesome and unlikely to cause illness in the consumer. Nevertheless, there are areas of risk. The major risk of acute disease is associated with the consumption of raw shellfish, particularly bivalve molluscs. For persons living in areas in which reef fish are consumed (Hawaii, Puerto Rico, the Virgin Islands), there is a risk of ciguatera; other natural toxins (paralytic shellfish poisoning, neurotoxic shellfish poisoning, etc.) have been associated with shellfish from endemic areas. Finally, there are less well-defined risks of acute and chronic disease related to environmental contamination of aquatic food animals. Dealing with such risks on a short-term basis requires improvements in the present system of regulatory control. In the long term, amelioration and eventual elimination of some hazards require strengthening and more effective application of control measures to prevent the disposal of human and industrial waste into offshore marine and fresh waters.

Because of the strong public interest in seafood safety and the declared intention at the congressional level to develop a new inspection system, a clear opportunity exists to introduce innovative methodologies for control that address directly the important health issues associated with seafood consumption.

This report reviews the nature and extent of public health risks associated with seafood, and examines the scope and adequacy of current seafood safety programs. The conclusions and recommendations arrived at are summarized in the following material:

- Most current health risks associated with seafood safety originate in the environment and should be dealt with by control of harvest or at the point of capture. With minor exceptions, risks cannot be identified by an organoleptic inspection system.

- Inspection at the processing level is important to maintain safety of seafoods, but there is little evidence that increased inspection activities at this level would effectively reduce the incidence of seafood-borne disease.
- With currently available data, it is possible to identify the source of much of the acute illness associated with seafood consumption, though the dimensions of the problems are not always known; these data, in turn, can form the basis for national control programs.
- Chronic illness resulting from seafood consumption is associated primarily with environmental contamination; thus, control depends on improved understanding of the occurrence and distribution of the chemical agents involved, the exclusion of contaminated seafood from the market, and increased action to prevent additional pollution of the waters.
- Because well over half the nation's seafood supply is imported and environmental contamination is globally pervasive, it is important that the safety of imported seafood be ensured through equivalent control measures in exporting countries.
- One-fifth of the fish and shellfish eaten in the United States is derived from recreational or subsistence fishing, and these products are not subject to health-based control; there is need to improve protection for consumers of these products by regulation of harvest and by education concerning risks associated with their consumption.
- Because the problems are largely regional, the primary effective control—except for imports—is at the state level, and this effort should be strengthened. However, there is need for federal oversight, general rule setting, and support to ensure the effectiveness of state-based programs and to provide expert assistance and specialized facilities.
- There is a lack of understanding of the nature of seafood hazards in the food service sectors and by the consuming public and health professionals; a vigorous campaign for information dissemination and education in these matters is needed, particularly for high-risk consumers and high-risk products such as raw shellfish.
- An improved national surveillance system should be developed to provide more reliable and comprehensive information on seafood-borne disease incidence. Data will then permit meaningful risk identification and risk assessment as a basis for effective regulation of seafoods (current data on disease occurrence in seafood consumption are too fragmentary to allow reliable risk assessment of microbiological and natural toxin hazards).

A summary of hazards, risks, and their control for the major groups of hazardous seafoods in shown in Table 1-1. They are arranged in order of importance.

Among seafood consumers, the group at greatest risk appears to be consumers of raw molluscs because of environmental contamination and naturally occurring vibrios. Consumers of recreational and subsistence fishery products are the second largest constituency at risk, both from natural toxins and from environmental contaminants.

- Primary regulatory authority should be at the state level, with funding, quality control, and specialized assistance from a federal seafood safety program.
- Imported seafoods must be certified to be free of natural toxins through equivalency arrangements or more effective memoranda of understanding (MOUs) with exporters. An MOU refers to a formal agreement between a U.S. government agency (e.g., FDA) and another government agency (federal, state, local), or an informal agreement with a foreign government or other foreign institution.
- Educational programs on the dangers of natural seafood toxins must be developed for recreational and subsistence fishers, and health providers must be given information to improve the identification and treatment of illness due to seafood toxins.

Chemical Residues

EXTENT OF RISK

Fish and shellfish accumulate chemicals from the environment in which they live, but the extent of accumulation depends on such factors as geographic location, species of fish, feeding patterns, solubility and lipophilicity of the chemicals, and their persistence in the environment. Moreover, whereas land animals used for human consumption are fed mostly food of plant origin, aquatic animals that contribute to the human diet are generally predators of other animals and, in some cases, predators of predators. Because of this, chemicals have an opportunity to become more concentrated through bioaccumulation.

The most difficult area for risk evaluation is the problem of chemical residues because the health effects suspected do not take the form of obvious, distinctive, and acute illnesses. The potential risks of concern (e.g., modest changes in the overall risk of cancer; subtle impairments of neurological development in fetuses and children) are generally quite difficult to measure directly in people exposed at levels that are common for U.S. consumers. Immunoincompetence increases cancer risk. Inferences about the potential magnitude of these problems must be based on the levels of specific chemicals present, on observations of human populations and experimental animals exposed at relatively high doses, and on reasonable theories about the likely mechanisms of action of specific toxicants and the population distributions of sensitivity and human exposure. In nearly all cases the current state of knowledge on these subjects must be regarded as quite tentative.' Additionally, the number and variety of chemical residues are substantial, although a small minority constitute the bulk of the risk that can be assessed quantitatively at this time.

Overall, several chemical contaminants in some species of aquatic organisms in particular locations have the potential to pose hazards to public health that are gre enough to warrant additional efforts at control. Available information suggests that these risks, in the aggregate, are not generally of a magnitude comparable to the high environmental health hazards characterized to date; nevertheless, their control would

significantly improve public health. Some examples of risks that may be significant include reproductive effects from polychlorinated biphenyls (PCBs) and methylmercury; carcinogenesis from selected congeners of PCBs, dioxins, and dibenzofurans (all of which appear to act primarily by binding to a single type of receptor); and, possibly, parkinsonism in the elderly from long-term mercury exposure. Several other metallic and pesticide residues also warrant attention.

PRINCIPAL CONCLUSIONS

- A small proportion of seafood is contaminated with appreciable concentrations of potentially hazardous organic and inorganic chemicals from both natural and human sources. Some examples of the risks that may be significant include reproductive effects from PCBs and methylmercury, and carcinogenesis from selected PCB congeners, dioxins, and chlorinated hydrocarbon pesticides.
- Consumption of some types of contaminated seafood poses enough risk that efforts toward evaluation, education, and control of that risk must be improved.
- Present quantitative risk assessment procedures used by government agencies should be improved and extended to noncancer effects.
- Current contaminant monitoring and surveillance programs provide an inadequate representation of the presence of contaminants in edible portions of domestic and imported seafood, resulting in serious difficulties in assessing both risks and specific opportunities for control.
- Due to the unevenness of contamination among species and geographic sources, it is feasible to narrowly target control efforts and still achieve meaningful reductions in exposure.
- The data base for evaluating the safety of certain chemicals that find their way into seafood via aquaculture and processing is too weak to support a conclusion that these products are being effectively controlled.

PRINCIPAL RECOMMENDATIONS

- Existing regulations to minimize chemical and biological contamination of the aquatic environment should be strengthened and enforced.
- Existing FDA and state regulations should be strengthened and enforced to reduce the human consumption of aquatic organisms with relatively high contaminant levels (e.g., certain species from the Great Lakes with high PCB levels, swordfish and other species with high methylmercury levels).
- Federal agencies should actively support research to determine actual risks from the consumption of contaminants associated with seafood and to develop specific approaches for decreasing these risks.
- Increased environmental monitoring should be initiated at the state level as

part of an overall federal exposure management system.

- States should continue to be responsible for site closures, and for issuing health and contamination advisories tailored to the specific consumption habits, reproductive or other special risks, and information sources of specific groups of consumers.
- Public education on specific chemical contaminant hazards should be expanded by government agencies and the health professions.
- For specific contaminants in particular species from high-risk domestic or foreign geographic areas, government agencies should consider the option of mandatory labeling.
- Additional study of potential chemical contamination risks associated with both domestic and imported aquaculture products is required. Because of different standards for drug or agricultural chemical use and water quality prevailing in other countries, imported aquaculture products should be effectively certified as meeting U.S. standards.

SCOPE AND ADEQUACY OF CURRENT SEAFOOD SAFETY PROGRAMS

Regulatory Guidelines, Monitoring, and Inspection

The current system of governance designed to protect the U.S. seafood consumer is composed of an intricate and complementary system of programs at the federal and state levels of government. Additional programs have been instituted in the private sector that offer a measure of industry self-regulation. At the federal level the principal responsibility for setting regulatory guidelines and for the surveillance and control of seafood safety is divided among the FDA, the Environmental Protection Agency (EPA), and the National Marine Fisheries Service (NMFS).

Within states, responsibility may lie with one or more of their health, environmental, fishery, or agricultural departments. States generally tend to adopt federal regulatory guidelines.

A primary role for the federal government is setting regulatory guidelines designed to promote inspection and enforcement activities both within and outside formal governmental programs. Existing regulatory guidelines can be divided into (1) those designed to reduce acute risk from microbial and natural toxin contaminants, and (2) those designed to reduce long-term or chronic risk due to chemical contamination. Guidelines for microorganisms and natural toxins are determined solely by the FDA and have been set primarily on an as-needed basis, that is, in response to a reported public health problem.

Properly collated and effectively presented guidelines could provide a stro basis for the production and supply of safe seafood. However, in several areas relatec to new processing techniques and other emerging problems, new guidelines seem both appropriate and necessary. Setting federal guidelines for residual chemical

contaminants is a task shared by EPA and FDA. Their strategy has been to focus on a limited number of chemical contaminants and to set regulatory limits by means of "tolerance levels." Results of various federal and state efforts to monitor contaminant loads in the nation's marine and freshwater environments suggest strongly that several chemicals require a more fundamental review and evaluation.

In terms of assessing and managing risks, the overall posture of relevant federal agencies, particularly FDA, appears to be almost totally reactive. In the committee's judgment, there has been less effort than would be desirable to discover and quantify hazards that are not yet on the public agenda, to evaluate options for reducing risks, and to implement policies that protect both the health of consumers and the stability of commercial markets.

One of the more important activities at both the federal and the state levels is environmental monitoring. Because the majority of seafood is from wild stocks, the quality of harvesting waters is of fundamental concern. The EPA and certain state governments [primarily by way of their involvement in the National Shellfish Sanitation Program (NSSP)] have instituted programs to establish the level of contaminants in seafood harvesting waters.

These efforts have led to important insights into general water quality but, for the most part, do not supply sufficient information on the question of seafood safety. Among other things, they lack (1) sufficient geographic scope, (2) a common methodological approach, and (3) sufficient focus on the edible portion of seafood in order to determine public health, as opposed to environmental health, impacts. This last point is an important one. Except for the monitoring of harvesting waters carried out as part of the NSSP, data evaluating contaminant levels in fish and shellfish do not consistently focus on the analysis of edible tissue. More often the focus is on whole fish or on liver and gallbladder analysis. These analyses, by their design, offer insufficient insight into contaminant levels in the edible portion of seafood products.

Inspection efforts by FDA and various state and local public health agencies are designed to ensure safety, but are insufficient to ensure in all cases that the regulatory guidelines defined by FDA and EPA are not being exceeded. The sampling strategies employed by these various agencies are designed to focus inspection and enforcement activities on areas in which the probability of a problem appears highest. Ongoing governmental efforts to develop new inspection programs, with a focus on the public health aspects of the raw product and the environment from which these products are derived, along with continued control of seafood production and processing, could provide measurable additional benefits in seafood safety.

Given many of the intrinsic attributes of seafood already discussed, it is clear that an approach recognizing the advantages of regional/local control and surveillance is essential. The question of seafood safety should continue to be one in which federal and state roles are viewed as a cooperative partnership. It is also apparent that seafood commerce is taking place within an increasingly interdependent international economy. Many of the major trading partners of the United States are developing or further refining formal regulatory programs for seafood safety. These efforts should

be taken into account in designing a domestic program.

PRINCIPAL CONCLUSIONS

- Federal (mostly FDA) guidelines for microbial and natural toxin contamination should be extended and updated. Those that exist have not been adequately conveyed to the fishing industry and to interested members of the public.
- Federal guidelines on chemical contaminants in seafoods are limited in scope and, in some cases, questionable as to the levels set. There is an apparent lack of coordination in the development and use of data on chemicals in the aquatic environment among FDA, EPA, the National Oceanic and Atmospheric Administration (NOAA), and the states. Better recognition is required of the importance of regional factors in the occurrence of toxic fish and shellfish and of the existence of high at-risk groups (e.g., pregnant women, children, recreational and subsistence fishers).
- The present federal monitoring and inspection system is too limited in frequency and direction to ensure enhanced safety of seafoods. The monitoring process depends too much on evaluation of the product, rather than on safety of raw materials, with the single notable exception of the NSSP. However, even NSSP is not providing adequate protection because molluscan shellfish appear to cause most seafood-borne disease.
- Recreational and subsistence fishing is largely ignored in health and safety monitoring at the federal level. Consumers of seafood from these sources can be at high risk from natural toxins and chemical pollutants in certain regions and in particular species of fish. The health risks include cancer and the subtle impairment of neurological development in fetuses and children.
- The present system of data collection on seafood-borne illness by CDC does not provide an adequate picture of the extent and causes of such disease.
- Seafood advisories warning of local or species-associated health risks are issued mostly by state authorities and vary greatly in both their content and their distribution. Nevertheless, these advisories serve a useful purpose.
- Because of the regional nature of much of the domestic fisheries problems, states seem the logical level at which to tackle seafood control problems. However, help and guidance from the federal level are required.
- State programs for monitoring, surveillance, and control of seafood safety are generally in place in coastal states that use federal guidelines and action levels where these are available. However, the quality and effectiveness of the programs vary greatly as a function of the financial and administrative support available to the responsible state units, and in accordance with the character of the resource. A greater emphasis should be placed on the development of formal arrangements with foreign producers to guarantee that imported seafood has been harvested and processed in noncontaminated environments.

- Present training and education of industry and regulatory personnel are too limited both in scope and in number. Insufficient attention is given to the education of physicians and other health professionals on seafood safety and the characteristics of seafood-borne disease. This is also true of the consuming public.
- The regulation of imported seafoods to ensure safety is largely based on end product inspection and testing, except where MOUs exist. This is ineffective because it involves a mainly reactive process.
- The regulation of imported seafood products is carried out largely without regard to other national or international programs. There is tremendous variance in both regulatory limits for contaminants and inspection protocols in various countries, which leads to excessive and cumbersome inspection strategies for the importing state, and may also lead to a general restriction in the number of countries engaged in international seafood trade in the future.

PRINCIPAL RECOMMENDATIONS

- A more concise, comprehensive, and generally available single source for all FDA guidelines relating to seafood safety should be developed and updated on a regular basis. This information should be disseminated to industry and integrated into state regulatory processes through more routine and uniform training programs.
- The development of an interagency structure with a single focus on seafood safety could contribute significantly toward increasing communication within the federal regulatory system, but the responsibility for primary control should be with the state.
- Federal agencies should develop a set of monitoring and inspection practices focusing more strongly on environmental conditions and on contaminant levels in the edible portion of seafood at the point of capture.
- Strong consideration should be given to creating a marine recreational fishing license system that is linked to the distribution of information characterizing the level and scope of potential risk from eating recreationally caught fish. Strong consideration should also be given to the closure of recreational harvest areas deemed to pose a threat to human health.
- The CDC should develop an active and aggressive program, founded on community-based health surveys, to better determine the level and source of seafood-borne illness in the U.S. population.
- Consideration should be given to the development of agreements with foreign authorities and individual producers to ensure that imported products are treated in a manner consistent with and equivalent to domestic products.
- A more pronounced and consistently defined federal role in the risk characterizations leading to seafood health advisories should be developed. A more consistent and focused effort in determining and communicating public health risks from contaminated seafood should also be developed.

• As more countries require the equivalency of domestic and imported products, it is apparent that the time has come for the international community to begin a process that would minimize the differences existing among national regulatory guidelines and approaches.

OPTIONS FOR REDUCING PUBLIC HEALTH RISKS

Monitoring, Control, and Surveillance Measures

The current system involves (1) surveillance by federal and state agencies to identify seafood-borne disease (e.g., CDC and state health departments); (2) evaluation of risk and setting of guidelines and action levels mostly by federal agencies (e.g., EPA and FDA); (3) control of risk by inspection and testing of edible fish and shellfish (e.g., states, FDA, and NMFS); and (4) action to protect consumers by embargo, detention, seizure, or recall, and by issuance of warning advisories (e.g., states and FDA). This system needs revision and strengthening to develop a truly risk-based regulatory process.

The data base on which regulation depends is inadequate. The disease surveillance system of CDC suffers from inadequate resources and should be refocused to provide a more complete and balanced account of seafood-borne disease. More analytical data on contaminants are needed, which could be obtained by increasing FDA analyses and sponsoring broader integrated studies of marine and fresh waters by EPA and corresponding state agencies.

Inspection and testing should focus on actual problems (as in HACCP systems), and there should be increased efforts to develop rapid, reliable test methods for dangerous microorganisms, toxins, and contaminants. This will require a restructuring of inspectional systems to accommodate newer methodologies and to train personnel in their application. Emphasis on purely sensory evaluation should be decreased.

Problems of interagency jurisdiction, unclear regulations, or poor cooperation among state and federal agencies should be addressed and rectified. This will require added resources.

Characteristics of Control Requirements

Control measures should be applied initially at the earliest stage of seafood production by monitoring of water quality and condition. Such measures would apply to the molluscan shellfish problem and to most natural toxins and chemical contaminants, and would permit the exclusion of potentially dangerous fish or shellfish from markets by fishing closures and use of advisories. Rapid and simple tests should be developed and used to screen potentially hazardous fish or shellfish at the point of harvest to reduce costs to the fishermen and to protect the consumer from toxins and

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Seafood Production, Distribution, and Consumption

ABSTRACT

Consumption of seafood has increased over the last decade, without a concomitant increase in reported illness. This increased consumption trend is expected to continue both for prepared and for fresh or frozen varieties. The 1989 consumption figure was 15.9 pounds of edible meat per person per year. Total commercial landings were a record 8.5 billion pounds in 1989, and imported edible products totaled 3.2 billion pounds. The majority of the seafood supply was harvested from wild populations. The aquaculture portion of this supply will probably increase. A substantial amount of seafood (600 million pounds of finfish and 300 million pounds of shellfish) is caught recreationally. About 70% of commercially produced seafood in the United States is sold fresh or frozen. Canned seafood constitutes approximately 25%, and smoked/cured products 5%, of the seafood consumed. The United States exported 1.4 billion pounds of edible domestic fishery products in 1989. The largest importer was Japan; Canada, the United Kingdom, France, and South Korea also provided good markets. The seafood harvest by industry is fragmented, diversified, seasonal, complex, and difficult to manage. Studies are needed to monitor changing consumption trends and patterns. The processing, distribution and merchandising of finfish and shellfish will require more emphasis to reduce cross-contamination. Attention must be given to aquaculture in order to produce high-quality, consistently available species. Attention must also be focused on the harvesting, handling, distribution, and preparation of recreationally harvested fish to ensure consumer safety. More emphasis should be placed on educating the industry and the consumer about safe-handling practices that can reduce potential food-handling problems.

INTRODUCTION

As Americans become increasingly aware of the relation between diet and good health, the consumption of fishery products will most likely increase. The consumer recognizes that fish and shellfish are nutritious and wholesome foods. They are perceived as an excellent source of high-quality protein, containing lipids with high levels of unsaturated fatty acids, and perhaps contributing to the enhancement of human health by reducing the risk of cardiovascular disease. Likewise, seafood is characteristically tender, easily digested, and a good source of many important minerals and vitamins (NRC, 1989).

Although the attributes of seafood attract a more health conscious consumer, they also enforce expectations for enhanced safety. Consumer consciousness of seafood safety issues has become, as a result, increasingly important. Pollution and environmental issues have further focused people's attention on contamination problems. Concurrent media coverage and public interest groups have heightened the demand for rigorous safety standards in the food industry (Haas et al., 1986; Newton, 1989).

Unlike meat and poultry, which are derived from domesticated sources, the majority of the edible seafood supply in the United States, approximately 12.0 billion pounds including domestic landings and imports in round weight equivalents, was harvested from wild populations in 1989 (NMFS, 1990). The aquacultured portion of this supply is predicted to increase from both foreign and domestic sources (Redmayne, 1989), and recorded commercial landings are further supplemented by a growing portion of recreationally caught seafood destined for consumption.

Because the supplies of many seafoods are relatively small and regional, large numbers of individuals, using a variety of vessels that range from small boats to large factory ships, are involved. The seafood harvesting industry is highly fragmented. The diversity of the industry, the seasonal nature of fishing, the complexity of fish processing operations, and the substantial amount of seafood caught recreationally (600 million pounds of finfish and 300 million pounds of shellfish) make it difficult to manage and regulate these living resources (NOAA, 1990).

Both finfish and shellfish are subjected to contamination and cross-contamination in their natural habitat, as well as at any point during handling, processing, distribution, or preparation (Haas et al.,.1986; Newton, 1989; NOAA, 1990). Seafood-borne illness has been reported due to natural toxins, microbial contamination, parasites, poor seafood handling, and chemical contaminants (CDC, 1981 a-c, 1983a,b, 1984, 1985, 1989; FDA, 1989). Because of the primary reliance on limited data-reporting systems via state departments of public health, and eventually the Centers for Disease Control (CDC), the extent of the public health risk due to cumulative exposure to microorganisms, natural toxins, and chemical contaminants cannot be assessed easily, especially in the context of total dietary exposure. Given this qualification, current data indicate a decrease in the reported incidents of illness from seafood relative to consumption.

The committee has critically examined and evaluated the degree of severity of illnesses, their significance,.and the extent of possible health risks involved. Its findings are documented in subsequent chapters of this report.

DEMOGRAPHICS OF THE SEAFOOD INDUSTRY

In 1989, commercial and recreational fishermen harvested more than 8.5 billion pounds of fish and shellfish from U.S. waters, which includes edible and industrial products. More than 300 major species of seafood were marketed, reflecting the

diversity of the resource base (NMFS, 1990). Over 4,000 processing and distribution plants handled the commercial products of the nation's 256,000 fishermen. Almost 95,000 boats and vessels constituted the fleet (NMFS, 1990).

Although commercial establishments are easily documented, the number of recreational fishermen and their support base are more difficult to quantify. Increasing numbers of anglers for fish from the nation's freshwater, estuarine, and marine waters are producing a growing share of the fresh and frozen seafood in today's diet. The number of recreational harvesters has been estimated to be in excess of 17 million individuals (NOAA, 1990).

Fresh and frozen seafood constitute about 70% of the product consumed in the United States. Canned seafood, particularly tuna, constitutes almost 25% of domestic consumption, and cured/smoked products account for the remaining 5% of per capita consumption.

FISHERY RESOURCES

Commercial landings (edible and industrial) by U.S. fishermen at ports in all the fishing states were a record 8.5 billion pounds (3.8 million metric tons) valued at $3.2 billion in 1989 (NMFS, 1990). This was an increase of 1.3 billion pounds (576,300 metric tons) in quantity, but a decrease of $281.8 million in value, compared with 1988. The total import value of edible fishery products was $5.5 billion in 1989, based on a record quantity of 3.2 billion pounds. Imports of nonedible (industrial) products set a record in 1989, with products valued at $4.1 billion, an increase of $676.1 million compared with 1988 (NMFS, 1990).

The trade deficit in fishery products has not declined. The dollar value of imports was higher in 1989 than in the previous year (NMFS, 1990). Canada is still the largest importer to the United States, sending in more than 700 million pounds of fishery products in 1988. Ecuador was ranked second and Mexico third. Whereas Canada ships finfish products, shrimp is the primary commodity exported by Ecuador and Mexico. Imports from Thailand and China are both increasing due to rising shrimp production from their expanding aquaculture systems.

On a worldwide basis, aquaculture is becoming a major new factor in seafood production. The cultivation of high-value species, popular in the U.S. market, is a major factor in import sourcing. China, for example, along with other Asian nations, is replacing South and Central American countries as a major shrimp supplier to the United States. Aquaculture is expected to determine much of the future fisheries growth, because wild stocks are nearing full utilization (NMFS, 1990; NOAA, 1990).

The total export value of edible and nonedible fishery products of domestic origin was a record $4.7 billion in 1989, an increase of $2.4 billion compared with 1988. The United States exported 1.4 billion pounds of edible products valued at $2.3 billion, compared with 1.1 billion pounds at $2.2 billion exported in 1988. Exports of nonedible products were valued at $2.4 billion. Japan continues to be America's best

export customer. Over 700 million pounds of seafood was sold to the Japanese market, with salmon, crabs, and herring the primary commodities. Canada, the United Kingdom, France, and South Korea were also good markets in 1989, but the value of their imports was small, compared to Japan's purchase of West Coast products (NMFS, 1990).

Consumers in the United States spent an estimated $28.3 billion for fishery goods in 1989, a 5% increase from 1988 (NMFS, 1990). The total included $19.1 billion in expenditures in food service establishments (restaurants, carryouts, caterers, etc.); $9.0 billion in retail stores (for home consumption); and $181.7 million for industrial fish products. In producing and marketing a variety of fishery products for domestic and foreign markets, the commercial fishing industry contributed $17.2 billion in value-added dollars to the gross national product (GNP), an increase of 5% compared to 1988.

Consumption of fish and shellfish in the United States totaled 15.9 pounds of edible meat per person in 1989 (NMFS, 1990). This total was up 0.7 pound from the 15.2 pounds consumed per capita in 1988. Per capita consumption of fresh and frozen products registered a total of 10.5 pounds, an increase of 0.3 pound from the 1988 level. Fresh and frozen finfish consumption was 7.1 pounds per capita in 1989. Fresh and frozen shellfish consumption amounted to 3.4 pounds per capita, with canned fishery products at 5.1 pounds per capita, up 0.4 pound over 1988. The per capita use of all fishery products (edible and nonedible) was 62.2 pounds (round weight), up 2.8 pounds compared with 1988 (NMFS, 1990).

Although most of the fish and shellfish consumed is from commercial production, a significant share is caught recreationally. In 1990, the National Marine Fisheries Service (NMFS) estimated that 17 million marine anglers harvested more than 600 million pounds of finfish (NOAA, 1990). Although statistics are lacking, NMFS suggests that 200-300 million pounds of molluscs and crustaceans was harvested by recreationalists. This catch represents 3-4 pounds of domestic per capita consumption (Krebs-Smith, 1989), outside the commercial figure of over 15 pounds per person. The source, handling, and distribution of the recreational catch are just beginning to draw attention. Indeed, because recreational anglers are not regulated as food producers/manufacturers, there is concern about the use and distribution of this "recreational" resource.

Although it is difficult to give definite numbers for either the commercial or the recreational harvesting sector, some general observations can be made. Commercially, the trend is toward more efficient activity. Consequently, the number of participants in the commercial sector is decreasing. The commercial processing industry appears headed toward consolidation, with increased dependence on imported products and aquaculture. Recreational participation remains strong. Consumption data, as suggested by both the Department of Agriculture and the Department of Commerce, indicate a continued, if not expanding, harvest of sport caught fish and shellfish. More than 20% of all fresh and frozen seafood consumed in the United States, may now be attributed to noncommercial harvest and distribution.

AQUACULTURE

Aquaculture is a rapidly growing mode of production in the seafood industry. Annual production of farmed fish and shellfish in the United States has grown 305% since 1980 (TFTC, 1988). The greatest production is of catfish (Sperber, 1989). Catfish production increased 31% from 1986 to 1987. According to the Catfish Institute, farm-raised catfish increased from 5.7 million pounds in 1970 to 295 million pounds in 1988 and were expected to exceed 310 million pounds in 1989 (Sperber, 1989). Salmon production in the Pacific Northwest and Maine totaled 85 million pounds in 1987. In addition, other fish that are farmed include trout, redfish, sturgeon, hybrid striped bass, carp, and tilapia, as well as shellfish and crustaceans such as oysters and crawfish. Crawfish production acreage has increased 145% to about 160,000 acres. Overall U.S. aquaculture production of fish and shellfish increased from 203 million pounds in 1980 to some 750 million pounds in 1987. It is estimated that by the year 2000, that figure will reach 1.26 billion pounds.

Large amounts of cultured fish and shellfish are also imported annually. Approxi-mately one-half of the 500 million pounds of shrimp imported is cultured (Schnick, 1990); 143 million pounds comes from China and Ecuador, neither of which regulates the use of chemotherapeutic agents in culture. More than 40 million pounds of salmon is also imported annually, often from countries similarly lacking tolerance levels for residues. Of special interest are the use of chloramphenicol in shrimp culture and ampicillin in yellowtail culture (Hawke et al., 1987; Manci, 1990). The Food and Drug Administration (FDA) has not examined imported seafood for drug residues, and there is no information regarding levels that might be ingested (Schnick, 1990).

Aquaculture also produces fish used to stock recreational fishing areas. This procedure is under the control of government agencies that follow FDA regulations, use only approved drugs, and abide by legal withdrawal times.

CONSUMPTION TRENDS

Today's consumer is changing rapidly. Instead of single-income households, it is increasingly more common to have both man and woman working. The size of the family is decreasing. As many as one-fourth of all households are occupied by one person. This means more shoppers and diners, most with little time for home preparation (Davis, 1989).

Most adult men and women now work outside the home. In recent surveys, 7 out of 10 new home buyers noted that they will need two incomes to pay their respective mortgages. Nevertheless, the growth in two-income couples has generally created an increase in disposable income, but with little time to spend it. With as many as 50% of new mothers working outside the home within the first year of childbirth, it is easy to see the revolutionary changes taking place among families. The

working mother or single dweller does not have the time to prepare meals in the traditional sense. In recent Food Marketing Institute (FMI) surveys, more than 30% of the husbands of women who work full-time did as much cooking, cleaning, and food shopping as their wives (Davis, 1989; FMI, 1988).

The population is aging. Going into the next century, the fastest growing groups will be those aged 45 to 54, along with those over age 85. By the year 2000, the proportion of Americans over age 65 will be the same throughout the country as the proportion in Florida today. An aging population means decreased discretionary spending and more demands for healthful and nutritious foods.

Minorities are growing in America. Within 10 years, one-quarter of all Americans will be either black, Hispanic, or Asian. The city of Los Angeles illustrates the trend. At present, Los Angeles is the largest Mexican city outside Mexico, the second largest Chinese city outside China, the second largest Japanese city outside Japan, and the largest Philippine city outside the Philippines (Davis, 1989).

The consumer demand for convenience, gourmet foods, ethnic items, and other services is increasingly evident in the food service and retail food industries. As the number of working women and single dwellers increases, the consumer base continues to change. With reduced leisure time, consumers who once spent two hours per day in the kitchen, now spend less than a half hour. Convenience stores, fast-food restaurants, specialty food service outlets, and prepared items in the supermarket are food industry responses (FMI, 1988; Taylor, 1989).

To illustrate the impact of less preparation time in the home, a quick review of consumer buying habits is in order. In 1973, almost 80% of the food dollar was spent on home-prepared foods. In 1988, this number had fallen to 67%. Many predict that the figure may be as low as 40% by the year 2000. As with all foods, fish and shellfish preparation must be viewed in the manner in which consumers use the product in a contemporary environment. This does not mean that the consumer will be eating at home less but, rather, that less time will be devoted to food preparation. This trend toward "cocooning," in which the family spends more time around the home but utilizes the time more prudently, is central to future consumer patterns (Davis, 1989).

Consumers want more convenience and nutrition. Value-added products, ready-to-eat items, and microwave entrees are examples. Deli departments of the supermarket may soon become food service operations, competing with fast-food and takeout restaurants (FMI, 1989; Taylor, 1989).

Seafood, like other foods, will be placed in a competitive consumer environment. Fish and shellfish must continue to taste good if they are expected to attract more consumers. Further, seafood must stay within the budget of the new consumer. If the industry can respond to the changing consumer base, the opportunity to expand per capita consumption appears good (Taylor, 1989).

The amount of imported product is not yet recognized as a potential problem by the consumer, yet it is of significant concern to regulatory officials. Rising needs place increased pressure on government to protect consumers without the ability to monitor the harvest, processing, and distribution of the hundreds of species in question.

Because of the potential of ever-increasing imports, the safety issue is becoming a matter of international concern. Although agencies routinely sample and require country-of-origin labeling, the consumer is unaware of the complexity of attempting to truly safeguard these foodstuffs.

ACTIVITIES IN OTHER COUNTRIES

A number of countries have endeavored to enhance the value of their seafood products by enacting programs to ensure product quality. Canada, Denmark, and Norway have given high priority to marketing safe, quality seafood items. Canada, for example, inspects vessels, landing sites, and processing facilities on an annual basis. Vessels must meet the same exacting standards as processing facilities or risk losing their certification. Canadian plant registration requires compliance with a posted list of standards. At inspection, plants are rated by use of a Hazard Analysis Critical Control Point (HAACP) approach. Critical findings result in more frequent inspections or the possibility of noncertification.

In Europe, similar programs are in place. Denmark inspects fishing vessels. Each participant must meet certain sanitation requirements, as well as certification for activities such as on-board processing. Distribution centers receive regular inspections that monitor all products entering the marketplace. The advent of the European Economic Community (EEC) has brought forth a host of new regulations, ensuring that member nations comply with the policies of their EEC partners.

Many other countries have seafood inspection programs, but they are often not dedicated programs like those in Canada, Denmark, Norway, Iceland, and New Zealand. Consequently, they do not pay the same rigorous attention to detail. Indeed, most countries have programs centered on seafood as a food group, not as a distinct entity that requires special attention.

CONCLUSIONS AND RECOMMENDATIONS

Based on commercial sources, Americans consumed almost 60% more seafood in 1989 than they did 10 years earlier. This increase in consumption was not accompanied by a concomitant increase in reported seafood-borne illnesses. The total supply of fishery products to fulfill the domestic requirement for seafood was in excess of 8.5 billion pounds in 1989, with over 300 species involved in the catch statistics. Production and consumption trends suggest that domestic seafood demand will continue, with more emphasis on prepared convenience foods along with the traditional demand for fresh and frozen selections. Production will have to be supplemented with more imported and cultured sources. Recreational harvesting, both in the purist sense and as subsistence fishing, continues to contribute a significant portion to the annual per capita intake.

The committee recommends the following:

- Consumer information studies must be conducted to monitor the rapidly changing consumption trends in the United States. Patterns of consumer use and preparation, as well as sources of seafood products used in the home, must be evaluated. By better understanding consumption patterns, fishery managers and food regulators will be more able to influence dietary intake, and reduce potential exposure to fish from contaminated water.
- Changes in consumption patterns necessitate more attention to informing consumers on how to best handle highly perishable products such as seafood. As much as 50% of all reported, acute fish and shellfish problems might be eliminated by more careful handling and proper preparation in the home or in food service establishments. With the advent of more prepared foods, every effort should be made to ensure the safety of the product both in the manufacturing/distribution chain and for the end user.
- The retail and institutional handling of seafood products requires increased attention to control cross-contamination. A number of seafood-related illnesses can be traced to poor sanitation practices by employees or to lack of proper handling via the distribution system. More efforts will be needed to alert all users to the importance of time/temperature relationships, HACCP concepts, good manufacturing practices, and new technology (e.g., live holding tanks).
- Aquaculture promises to produce a larger share of domestically consumed fish and shellfish in the years ahead. Cultured plants and animals hold the promise of being high quality, and generally free of some of the contamination associated with wild species. Care, however, must be taken to avoid the untimely use of antibiotics and other chemicals in these closed or recirculated systems, which are often used to control pathogens in semiclosed systems.
- The safety of recreationally harvested fish and shellfish requires increased vigilance, which means increased focus on the origin, handling, and distribution of recreational products. These harvesting efforts may now account for over 20% of all fresh and frozen seafood consumed in the United States. However, this catch is not well controlled, and users may handle, distribute, and prepare the product in an unsafe manner. Further, much of this product may be harvested from areas not suited for consumption due to natural or induced contamination problems. Increased educational activity is required to protect the consumer with regard to this resource. Fishery managers will have to pay greater attention to the implications of sport caught fish and shellfish on consumer health.

REFERENCES

CDC (Centers for Disease Control). 1981a. *Salmonella* Surveillance, Annual Summary, 1978. HHS Publ. No. (CDC) 81-8219. Public Health Service, U.S. Department of Health and Human Services, Atlanta, Ga. 25 pp.

HEALTH ADVISORY

The following recommendations are based on contaminant levels in fish and shellfish and are updated on a regular basis (see page 44 for instructions on how to get updates). To minimize potential adverse health impacts, the NYS Department of Health (DOH) recommends:

- Eating no more than one meal (1/2 pound) per week of fish from any freshwater, the Hudson River estuary and the area including Upper Bay of New York Harbor north of the Verrazano Narrows Bridge, Arthur Kill, Kill Van Kull, East River to the Throgs Neck Bridge and Harlem River, except as recommended below.
- Women of childbearing age, infants and children under the age of 15 should not eat any fish species from the waters listed below.
- Following trimming and cooking advice.
- Observing the following restrictions on eating fish from these waters and their tributaries to the first barrier impassable by fish.

Water (County)	Species	Recommended
Arthur Kill (Richmond)	*See Hudson River (south of Catskill)*	
Barge Canal (Tonawanda Creek) Lockport to Niagara River (Erie; Niagara)	Carp	1 meal / month
Belmont Lake (Suffolk)	Carp	1 meal / month
Big Moose Lake (Herkimer)	Yellow perch	1 meal / month
Buffalo River & Harbor (Erie)	Carp	Eat none
Canadice Lake (Ontario)	Lake or brown trout over 21"	Eat none
Canandaigua Lake (Ontario; Yates)	Lake trout over 24"	1 meal / month
Carry Falls Reservoir (St. Lawrence)	Walleye	1 meal / month
Cayuga Creek (Niagara)	All species	Eat none
Cranberry Lake (St. Lawrence)	Smallmouth bass	1 meal / month
Delaware Park Lake (Erie)	Carp	1 meal / month
East River (New York City)	American eel	Eat none
	Atlantic needlefish, bluefish, striped bass, white perch	1 meal / month
Eighteen Mile Creek (Niagara)	All species	Eat none
Ferris Lake (Hamilton)	Yellow perch over 12"	Eat none
	Smaller yellow perch	1 meal / month
Fourth Lake (Herkimer; Hamilton)	Lake trout	Eat none
Francis Lake (Lewis)	Yellow perch	1 meal / month
Freeport Reservoir (Nassau)	Carp	1 meal / month
Gill Creek (Niagara) Mouth to Hyde Park Lake Dam	All species	Eat none
Grant Park Pond (Nassau)	Carp	1 meal / month
Grasse River (St. Lawrence) Mouth to Massena Power Canal	All species	Eat none
Halfmoon Lake (Lewis)	Yellow perch	1 meal / month
Hall's Pond (Nassau)	Carp, goldfish	Eat none
Harlem River (New York City)	American eel	Eat none
	Atlantic needlefish, bluefish, striped bass, white perch	1 meal / month
*Herrick Hollow Creek (Delaware)	Brook trout	1 meal / month
Hoosic River (Rensselaer)	Brown trout, rainbow trout	1 meal / month
Hudson River:		
Sherman Island Dam to Feeder Dam at South Glens Falls	Carp	1 meal / month
Hudson Falls to Troy Dam	All species— Catch and release only	Eat none
Troy Dam south to bridge at Catskill	All species except American shad	Eat none
Bridge at Catskill south to and including the Upper Bay of NY Harbor, Arthur Kill and Kill Van Kull	American eel, bluefish, striped bass, Atlantic needlefish, rainbow smelt, white perch, carp, goldfish, white catfish, largemouth bass, smallmouth bass, walleye	1 meal / month
	Blue crab:	Eat no more than 6 crabs per week
	hepatopancreas (mustard, liver or tomalley)	Eat none
	cooking liquid	Discard
Indian Lake (Lewis)	All species	1 meal / month
Irondequoit Bay (Monroe)	Carp	Eat none
Keuka Lake (Yates; Steuben)	Lake trout over 25"	1 meal / month
Kill Van Kull	*See Hudson River (south of Catskill)*	
Kinderhook Lake (Columbia)	American eel	1 meal / month
Koppers Pond (Chemung)	Carp	1 meal / month
Lake Capri (Suffolk)	Carp	1 meal / month
Lake Champlain:		
Entire lake	Lake trout over 25", walleye over 19"	1 meal / month
Bay within Cumberland Head to Crab Island	American eel, brown bullhead, yellow perch	1 meal / month
Lake Erie	See page 43	
Lake Ontario and Niagara River below the falls (See Niagara River for additional advice.)	American eel, channel catfish, lake trout over 25", chinook salmon, brown trout over 20", carp	Eat none
Lake Ontario (Continued)	White sucker, rainbow trout, coho salmon over 25", smaller lake trout and brown trout	1 meal / month
West of Point Breeze	White perch	Eat none
East of Point Breeze	White perch	1 meal / month
Lott's Pond (Nassau)	Carp, goldfish	1 meal / month
Long Pond at Croghan (Lewis)	Splake over 12"	Eat none
Upper Massapequa Reservoir (Nassau)	White perch	1 meal / month
Massena Power Canal (St. Lawrence)	Smallmouth bass	1 meal / month
Meacham Lake (Franklin)	Yellow perch over 12"	Eat none
	Smaller yellow perch	1 meal / month
Mohawk River from Oriskany Creek to West Canada Creek (Oneida, Herkimer)	Carp	Eat none
	Largemouth bass, tiger muskellunge	1 meal / month
Moshier Reservoir (Herkimer)	Yellow perch	1 meal / month
Nassau Lake (Rensselaer)	All species	Eat none
*Neversink Res. (Sullivan)	Smallmouth bass	1 meal / month
New York Harbor	*See Hudson River (south of Catskill) and Marine Waters (See next page)*	
Niagara River above the falls	Carp	1 meal / month
Niagara River below the falls; also see Lake Ontario	White perch	Eat none
	Smallmouth bass	1 meal / month
Onondaga Lake (Onondaga)	All species	Eat none
Oswego River (Oswego) from power dam in Oswego to upper dam at Fulton	Channel catfish	1 meal / month
Ridders Pond (Nassau)	Goldfish	Eat none
*Rondout Res. (Sullivan, Ulster)	Smallmouth bass over 16"	1 meal / month
Round Pond (Hamilton)	Yellow perch over 12"	1 meal / month
St. James Pond (Suffolk)	All species	1 meal / month
St. Lawrence River		
Entire river	American eel, channel catfish, Lake trout over 25", chinook salmon, brown trout over 20", carp	Eat none
	White perch, white sucker rainbow trout, coho salmon over 25", smaller lake and brown trout	1 meal / month
Bay at St. Lawrence-Franklin County line	All species	Eat none
Salmon River (Oswego) Mouth to Salmon Reservoir, also follow Lake Ontario advisories	Smallmouth bass	1 meal / month
Sauquoit Creek Between dam at Clayville and Mohawk River (Oneida)	Brown trout	Eat none
Saw Mill River (Westchester)	American eel	1 meal / month
Schroon Lake (Warren, Essex)	Lake trout over 27"	1 meal / month
Sheldrake River (Westchester)	American eel, goldfish	Eat none 1 meal / month
Skaneateles Creek (Onondaga) Seneca River to dam at Skaneateles	Brown trout over 10"	1 meal / month
Smith Pond at Rockville Centre (Nassau)	White perch	1 meal / month
Smith Pond at Roosevelt Park (Nassau)	American eel	Eat none
	Carp, goldfish	1 meal / month
Spring Pond at Middle Island (Suffolk)	Carp, goldfish	Eat none
Stillwater Reservoir (Herkimer)	Splake,smallmouth bass, yellow perch over 9"	1 meal / month
Sunday Lake (Herkimer)	Yellow perch	1 meal / month
Three Mile Creek (Oneida)	White sucker	1 meal / month
Valatie Kill (Rensselaer) between County Rte. 18 and Nassau Lake	All species	Eat none
Whitney Park Pond (Nassau)	Carp, goldfish	1 meal / month

* Changes from the 1998-99 Fishing Regulations Guide

ADDITIONAL ADVICE

Advisories for Lake Erie—Due to PCB Contamination, women of childbearing age, infants and children under the age of 15 are advised to eat no more than one meal per week of chinook salmon less than 19 inches, burbot, freshwater drum, lake whitefish, rock bass and yellow perch, and EAT NO MORE THAN ONE MEAL PER MONTH of all other fish from Lake Erie. Other people should eat no more than one meal per week of any Lake Erie species.

Marine Bluefish and Eels—The general advisory (eat no more than one meal per week) applies to bluefish and American eel, but not to most other fish from Long Island Sound, Peconic/Gardiners Bays, Block Island Sound, the Lower Bay of New York Harbor, Jamaica Bay and other Long Island South Shore waters.

Marine Striped Bass—Women of childbearing age and children under the age of 15 should eat no striped bass taken from the Upper and Lower Bays of New York Harbor or Long Island Sound west of Wading River. Other people should eat no more than one meal per month of striped bass from these waters. Everyone should eat no more than one meal per week of striped bass taken from Jamaica Bay, Eastern Long Island Sound, Block Island Sound, Peconic/Gardiners Bays or Long Island South Shore waters.

Blue Crab and Lobsters—The hepatopeas (liver, mustard, or tomalley) of crabs and lobsters should not be eaten because it has high contaminant levels.

Hudson River Shad—The advisory for women of childbearing age, infants and children under the age of 15 is EAT NONE for all fish from the lower Hudson River because of PCB contamination. However, shad have lower PCB levels than other species. A few meals of Hudson River shad meat and roe, especially using cooking and trimming methods that minimize PCB content, would not pose an unacceptable risk for women of childbearing age and children, assuming this is their only significant exposure to PCBs.

Deformed or Abnormal Fish—The health implications of eating these fish are unknown. Any grossly diseased fish should probably be discarded.

Health Benefits—When properly prepared, fish provide a diet high in protein and low in saturated fats. Almost any kind of fish may have real health benefits if it replaces a high-fat source of protein in the diet.

Chemicals in Sportfish or Game

Summary

The NYS Department of Environmental Conservation (DEC) routinely monitors contaminant levels in fish and wildlife. The NYS Department of Health (DOH) issues advisories on eating sportfish and game taken in New York State because some of these foods contain potentially harmful levels of chemical contaminants. The health advisories are: (1) general advice on sportfish taken from waters in New York State; (2) advice on sportfish from specific waterbodies; and (3) advice on eating game. The advisories are updated annually.

Contaminants in Fish and Game

Long-lasting contaminants, such as PCBs, DDT and cadmium, build up in your body over time. It may take months or years of regularly eating contaminated fish to build up amounts which are a health concern. Health problems which may result from the contaminants found in fish range from small changes in health that are hard to detect to birth defects and cancer. Mothers who eat highly contaminated fish and wildlife before becoming pregnant may have children who are slower to develop and learn. The meal advice in this advisory is also intended to protect children from these potential developmental problems. Women beyond their childbearing age and men face fewer health risks from contaminants than children do.

Some contaminants cause cancer in animals. Your risk of cancer from eating contaminated fish and wildlife cannot be predicted with certainty. Cancer currently affects about one in every three people, primarily due to smoking, diet and hereditary risk factors. Exposure to contaminants in the fish and wildlife you eat may not increase your cancer risk at all. If you follow this advisory over your lifetime, you will minimize your exposure and reduce whatever cancer risk is associated with these contaminants.

The federal government establishes standards for chemical residues in food. When establishing these standards for fish, the federal government assumes that people eat about one-half pound of fish each month. The contaminant levels are measured in a skin-on fillet which has not been trimmed; this sample is used in determining whether or not the fish exceeds standards. Fish cannot be legally sold if they contain a contaminant at a level greater than its standard. When sportfish from a waterbody contain contaminants at levels greater than federal standards, the DOH issues a specific advisory.

General Advisory

The general health advisory for sportfish is that you eat no more than one meal (one-half pound) per week of fish from the state's freshwaters and marine waters at the mouth of the Hudson River. These waters include the New York waters of the Hudson River including Upper Bay north of the Verrazano Narrows Bridge, Arthur Kill, Kill Van Kull, Harlem River, and the East River to the Throgs Neck Bridge. This general advisory is to protect against eating large amounts of fish that have not been tested or may contain unidentified contaminants. The general advisory does not apply to most fish taken from marine waters.

Specific Advisories for Freshwaters, the Hudson River and Upper Bay of New York Harbor

Over 60 waterbodies in New York State have fish with contaminant levels greater than federal standards. DOH recommendations suggest either limiting or avoiding eating a specific kind of fish from particular waterbodies. In some cases, enough information is available to issue advisories based on the length of the fish. Older (larger) fish are often more contaminated than younger (smaller) fish.

Health advice is also given for **infants, children under the age of fifteen and women of childbearing age.** DOH recommends that they not eat any fish species from the specific waterbodies listed in the advisory. The reason for this specific advice is that chemicals may have a greater effect on developing organs in young children or in the fetus. They also build up in women's bodies and are often passed on in mothers' milk.

Waters which have specific advisories have at least one species of fish with an elevated contaminant level, which means that a contamination source is in or near the water.

When eating fish from waters where cadmium or mercury are listed as primary contaminants, it is important to space out fish meals according to the specific advisory for that waterbody. For example, if you eat a meal of yellow perch from Moshier Reservoir, you should not eat any more fish with the same mercury advisory for the rest of that month.

However, for other contaminants, the total number of meals that you eat during the year is important and many of those meals can be eaten during a few months of the year. If most of the fish you eat are from the "One Meal a Week" category, you should not exceed 52 meals per year. Likewise, if most of the fish you eat are in the "One Meal a Month" category, you should not exceed 12 meals per year. Remember, eating one meal of fish from the "One Meal a Month" group is comparable to eating four meals from the "One Meal a Week" group.

Other Advisories

DOH has also issued special advisories for snapping turtles and waterfowl. Cooking methods are recommended that minimize the amount of contaminants which would be eaten. Advisories for snapping turtles and waterfowl are provided in the Hunting and Trapping Guide.

Reducing Exposure To Chemical Contaminants From Fish

Fish are an important source of protein and are low in saturated fat. Naturally occurring fish oils have been reported to lower plasma cholesterol and triglycerides thereby decreasing the risk of coronary heart disease. Increasing fish consumption is useful in reducing dietary fat and controlling weight. By eating a diet which includes food from a variety of protein sources an individual is more likely to have a diet which is adequate in all nutrients.

Although eating fish has some health benefits, fish with high contaminant levels should be avoided. When deciding whether or not to eat fish which may be contaminated, the benefits of eating those fish can be weighed against the risks. For young women, eating contaminated fish is a health concern not only for herself but also to any unborn or nursing child since the chemicals may reach the fetus and can be passed on in breastmilk. For an older person with heart disease, the risks, especially of long term health effects, may not be as great a concern when compared to the benefits of reducing the risks of heart disease.

Everyone can benefit from eating fish they catch and can minimize their contaminant intake by following these general recommendations:

- Choose uncontaminated species from waterbodies which are not listed in the DOH advisories.
- Use a method of filleting the fish which will reduce the skin, fatty material and dark meat. These parts of the fish contain many of the contaminants.
- Choose smaller fish, consistent with DEC regulations, since they may have lower contaminant levels. Older (larger) fish within a species may be more contaminated because they have had more time to accumulate contaminants in their bodies.
- For shellfish, such as crab and lobster, do not eat the soft green substance found in the body section (mustard, tomalley, liver or hepatopancreas). This part of the shellfish has been found to contain high levels of chemical contaminants, including PCBs and heavy metals.
- Cooking methods such as broiling, poaching, boiling and baking, which allow fats to drain out, are preferable. Pan frying is not recommended. The cooking liquids of fish from contaminated waters should be avoided since these liquids may retain contaminants.
- Anglers who want to enjoy the fun of fishing but who wish to eliminate the potential risks associated with eating contaminated sportfish should consider "catch and release" fishing. Refer to this fishing guide for suggestions on catch and release fishing techniques.

Cleaning and Cooking Your Fish

Many contaminants are found at higher levels in the fat of fish. You can reduce the amount of these contaminants in a fish meal by properly trimming, skinning and cooking your catch. **Remove the skin and trim all the fat:** the belly flap, the line along the sides, the fat along the back and under the skin. (See diagram below.)

Cooking or smoking fish does not destroy contaminants in fish but heat from cooking melts some of the fat in fish and allows some of the contaminated fat to drip away. Broil, grill or bake the trimmed, skinned fish on a rack so that the fat drips away. Do not use drippings to prepare sauces or gravies. If you deep fry the fish, do not reuse the cooking oil.

These precautions will not reduce the amount of mercury or other metals. Mercury is distributed throughout a fish's muscle tissue (the part you eat), rather than in the fat and skin. Therefore, the only way to reduce mercury intake is to reduce the amount of contaminated fish you eat.

To receive an updated, complete version of the advisories, or for more DOH information on health effects from exposure to chemical contaminants, contact:

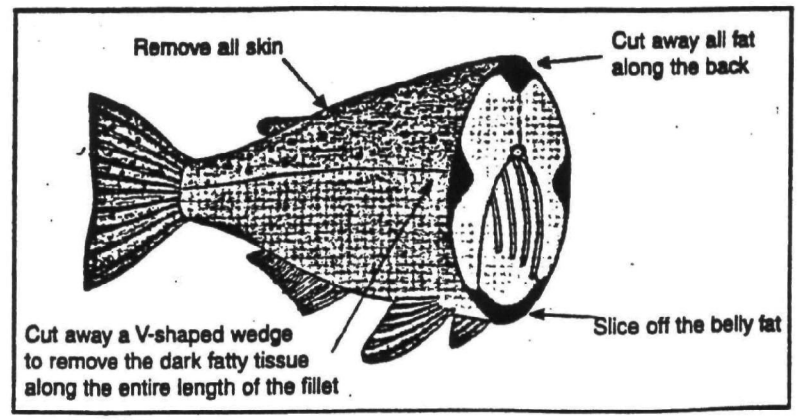
Environmental Health Information
1-800-458-1158
(toll-free number)

Leave your name, number and brief message. Your call will be returned as soon as possible.

The complete, updated advisories are available at
http://www.health.state.ny.us
or can be requested by E-mail:
BTSA@health.state.ny.us.

For more DEC Information on contaminant levels and eating sportfish, contact:

Bureau of Habitat
50 Wolf Road, Albany, NY 12233-4756
(518)457-6178.



United States Environmental Protection Agency | Office of Water 4305 | EPA-823-F-99-005 July 1999

EPA Fact Sheet

Update: National Listing of Fish and Wildlife Advisories

Summary

The 1998 update for the database National Listing of Fish and Wildlife Advisories (NLFWA) is now available from the U.S. Environmental Protection Agency (EPA). This database includes all available information describing state-, tribal-, and federally-issued fish consumption advisories in the United States for the 50 states, the District of Columbia, four United States territories and one tribal organization. It also includes information from 1997 for 12 Canadian provinces and territories. The database contains advisory information provided to EPA by the states as of December 1998. The number of advisories in the United States rose by 205 in 1998 to a total of 2,506, a 9% increase over 1997. The number of waterbodies under advisory represents 15.8% of the Nation's total lake acres and 6.8% of the Nation's total river miles. In addition, 100% of the Great Lakes waters and their connecting waters and 58.9% of the Nation's coastal waters are also under advisory. The total number of advisories in the United States increased for three major contaminants—mercury, PCBs, and DDT—but declined for dioxins and chlordane.

Beginning in 1996, EPA contacted health officials in Canada in an effort to identify fish consumption advisories in effect. The number of Canadian advisories in effect as of December 1997 was 2,625. No updates to information on Canadian advisories were made in 1998. All of the 1997 Canadian fish advisories resulted from contamination from one or more of the following five pollutants: mercury, PCBs, dioxins/furans, toxaphene, and mirex. Provincewide advisories for mercury were in effect for New Brunswick and Nova Scotia in 1997.

The NLFWA is now available for use on the Internet at: *http://www.epa.gov/ost/fish*

Background

The states and the four U.S. territories and Native American tribes (hereafter referred to as states) have primary responsibility for protecting residents from the health risks of consuming contaminated noncommercially caught fish and wildlife. They do this by issuing consumption advisories for the general population, including recreational and subsistence fishers, as well as for sensitive subpopulations (such as pregnant women, nursing mothers, and children). These advisories inform the public that high concentrations of chemical contaminants (e.g., mercury and dioxins) have been found in local fish and wildlife. The advisories include recommendations to limit or avoid consumption of certain fish and wildlife species from specific waterbodies or, in some cases, from specific waterbody types (e.g., all lakes). Similarly, in Canada, the provinces and territories have primary responsibility for issuing fish consumption advisories for their residents.

States typically issue five major types of advisories and bans to protect both the general population and specific subpopulations.

- When levels of chemical contamination pose a health risk to the general public, states may issue a no-consumption advisory for the general population (NCGP).
- When contaminant levels pose a health risk to sensitive subpopulations, states may issue a no-consumption advisory for the sensitive subpopulation (NCSP).
- In waterbodies where chemical contamination is less severe, states may issue an advisory recommending that either the general population (RGP) or a sensitive subpopulation (RSP) restrict their consumption of the specific species for which the advisory is issued.
- The fifth type of state-issued advisory is the commercial fishing ban (CFB), which prohibits the commercial harvest and sale of fish, shellfish, and/or wildlife species from a designated waterbody and, by inference, the consumption of all species identified in the fishing ban from that waterbody.

As shown in Table 1, advisories of all types increased in number from 1993 to 1998.

Table 1: U.S. Advisories Issued from 1993 to 1998 by Type

	1993	1994	1995	1996	1997	1998
No Consumption – General Population	503	462	463	563	545	532
No Consumption – Sensitive Subpopulation	555	720	778	1,022	1,119	1,211
Restricted Consumption – General Population	993	1,182	1,372	1,763	1,843	2,062
Restricted Consumption – Sensitive Subpopulation	689	900	1,042	1,370	1,450	1,595
Commercial Fishing Ban	30	30	55	50	52	50

Advisories in Effect

The database includes information on

- Species and size range of fish and/or wildlife
- Chemical contaminants identified in the advisory
- Geographic location of each advisory (including landmarks, river miles, or latitude and longitude coordinates of the affected waterbody)
- Lake acreage or river miles under advisory
- Population for whom the advisory was issued.

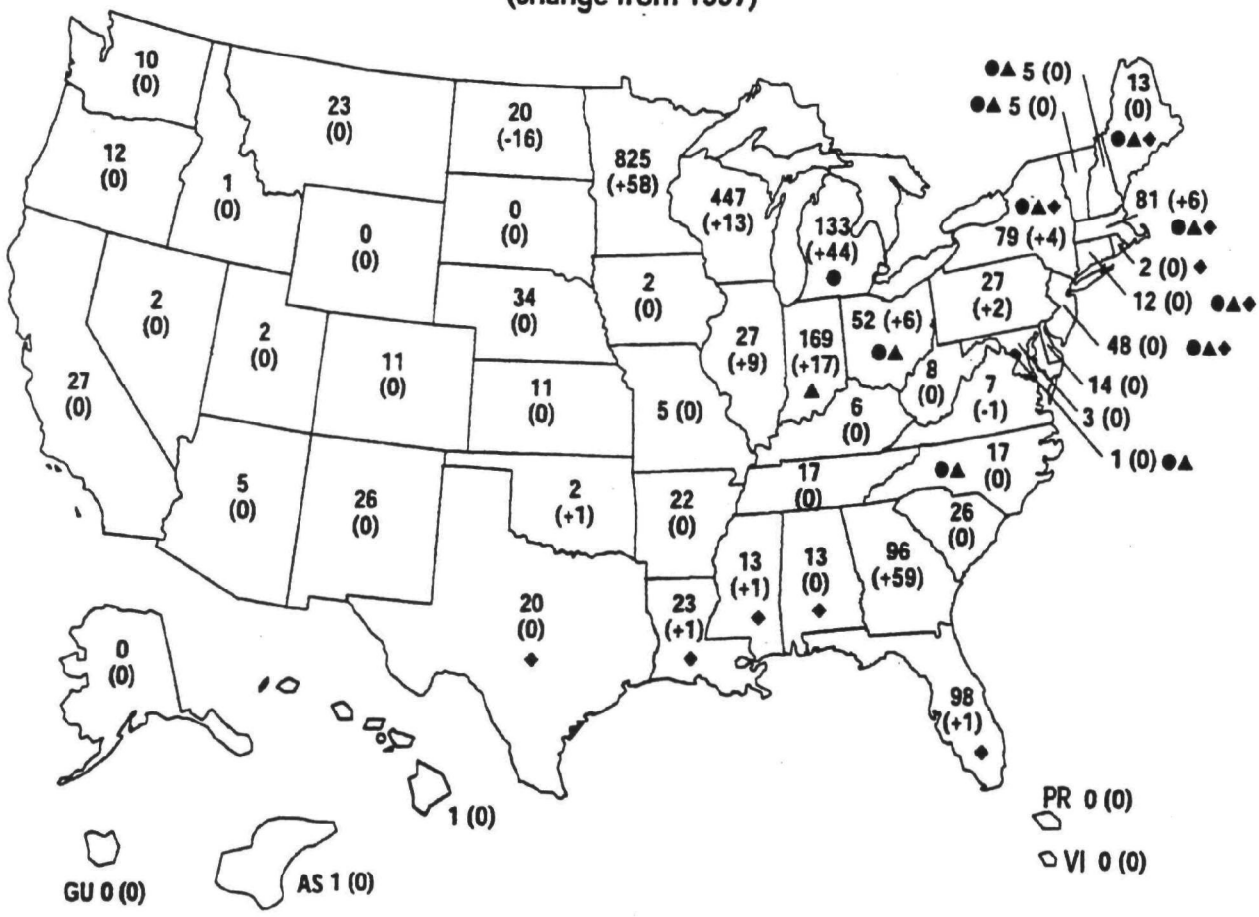
The 1994, 1995, 1996, 1997, and the new 1998 versions of the NLFWA database can generate national, regional, and state maps that illustrate any combination of these advisory parameters. In addition, the 1996 through 1998 versions of the database can provide information on the percentage of waterbodies in each state that is currently under an advisory and the percentage of waters assessed. A new feature of the 1998 database provides users access to fish tissue residue data for those waterbodies under advisory in 16 states. The name of each state contact, a phone number, FAX number, and e-mail address are also provided so that users can obtain additional information concerning specific advisories. Comparable advisory information (excluding tissue residue data) and contact information for 1997 are provided for each Canadian province or territory.

Advisory Trends

The number of waterbodies in the United States under advisory reported in 1998 (2,506) represents a 9% increase from the number reported in 1997 (2,299 advisories) and a 98% increase from the number of advisories issued since 1993 (1,266 advisories). Figure 1 shows the number of advisories currently in effect for each state and the number of new advisories issued since 1997. The increase in advisories issued by the states generally reflects an increase in the number of assessments of the levels of chemical contaminants in fish and wildlife tissues. These additional

Figure 1

Total Number of Fish Advisories in Effect in Each State in 1998 (change from 1997)



assessments were conducted as a result of the increased awareness of health risks associated with the consumption of chemically contaminated fish and wildlife. Some of the increase in advisory numbers, however, may be due to the increasing use of EPA risk assessment procedures in setting advisories rather than Food and Drug Administration (FDA) action levels developed for commercial fisheries.

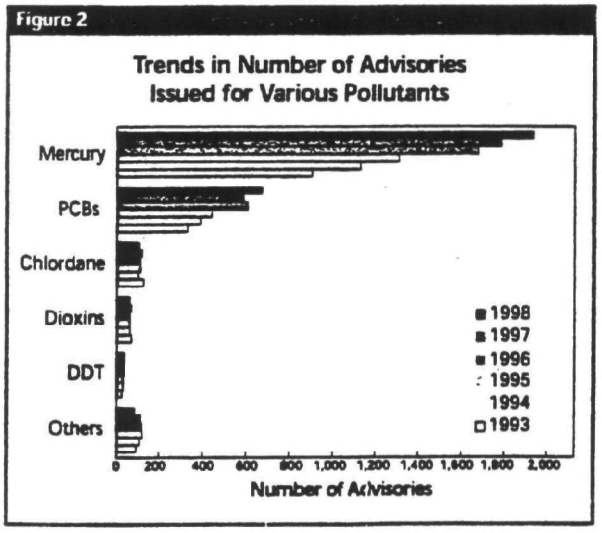
Bioaccumulative Pollutants

Although advisories in the United States have been issued for a total of 46 chemical contaminants, most advisories issued have involved five primary contaminants. These chemical contaminants are biologically accumulated in the tissues of aquatic organisms at concentrations many times higher than concentrations in the water. In addition, these chemical contaminants persist for relatively long periods in sediments where they can be accumulated by bottom-dwelling animals and passed up the food chain to fish. Concentrations of these contaminants in the tissues of aquatic organisms may be increased at each successive level of the food chain. As a result, top predators in a food chain, such as trout, salmon, or walleye, may have concentrations of these chemicals in their tissues that can be a million times higher than the concentrations in the water. Mercury, PCBs, chlordane, dioxins, and DDT (and its degradation products, DDE and DDD) were at least partly responsible for 99% of all fish consumption advisories in effect in 1998. (See Figure 2.)

Mercury

Advisories for mercury increased 8% from 1997 to 1998 (1,782 to 1,931) and increased 115% from 1993 to 1998 (899 to 1,931). The number of states that have issued mercury advisories also has risen steadily from 27 in 1993 to 40 in 1997 and remained at 40 in 1998. The rise in the number of mercury advisories in 1998 can be attributed primarily to issuance of new mercury advisories in 11 states. The majority (80%) of these new advisories, however, were issued in three states: Minnesota (61), Georgia (57), and Indiana (17).

Figure 2



It should also be noted that 10 states (Connecticut, Indiana, Maine, Massachusetts, Michigan, New Hampshire, New Jersey, North Carolina, Ohio, and Vermont) have issued statewide advisories for mercury in freshwater lakes and/or rivers. Another five states (Alabama, Florida, Louisiana, Mississippi, and Texas) have statewide advisories for mercury in their coastal waters. To date, 90% of the 1,931 mercury advisories in effect have been issued by the following 11 states: Minnesota (821), Wisconsin (402), Indiana (126), Florida (97), Georgia (80), Massachusetts (58), Michigan (53), New Jersey (30), New Mexico (26), South Carolina (24), and Montana (22).

PCBs

Advisories for PCBs increased 15% from 1997 to 1998 (from 588 to 679) and increased 112% from 1993 to 1998 (319 to 679). The number of states that have issued PCB advisories increased only slightly from 31 to 35 from 1993 to 1994, declined to 34 states in 1995 and 1996, and increased to 35 states in 1997 and up to 36 states in 1998 with the addition of Hawaii. The majority (77%) of the new PCB advisories in 1998 were issued by four states: Michigan (48), Illinois (11), Indiana (5), and Minnesota (5). To date, 79% of the of the 679 PCB advisories in effect have been issued by 10 states: Indiana (125), Michigan (104), Minnesota (83), Wisconsin (54), New York (47), Ohio (37), Georgia (25), Nebraska (22), Pennsylvania (22), and Massachusetts (20). Three states (Indiana, New York, and District of Columbia) have issued statewide freshwater (river and/or lake) advisories for PCBs. Six other states (New Jersey, Connecticut, New York, Rhode Island, New Hampshire, and Massachusetts) have issued PCB advisories for all of their coastal marine waters.

Other Pollutants

The total number of advisories for DDT (and its degradation products, DDE and DDD) increased from 33 in 1997 to 34 in 1998. The total number of advisories for dioxins rose from 54 in 1993 to 63 in 1994, held steady at 63 in 1995, declined to 60 in 1996, increased to 65 in 1997, and fell to 59 in 1998, a 9% decrease from the previous year. Dioxins are one of several chemical contaminants for which advisories have been rescinded by many states, in part because many pulp and paper mills have changed their processes. In 1998, three states (Arkansas, Michigan, and Virginia), rescinded a total of four dioxin advisories. The number of chlordane advisories also decreased, by 11%, from 117 in 1997 to 104 in 1998.

Wildlife Advisories

In addition to advisories for fish and shellfish, the database also contains several wildlife advisories. Four states have issued consumption advisories for turtles: Arizona (3), Massachusetts (1), Minnesota (8), and New York (statewide advisory). One state (Massachusetts) has an advisory for

frogs. New York has a statewide advisory for waterfowl (including mergansers), Arkansas has an advisory for woodducks, and Utah has an advisory for American coot and ducks. Maine issued a statewide advisory for moose liver and kidneys due to cadmium levels.

Table 2. Summary of Statewide Advisories by Waterbody Type

State	Lake	River	Coastal Waters
Alabama			Mercury
Connecticut	Mercury	Mercury	PCBs
Dist. of Columbia	PCBs	PCBs	
Florida			Mercury
Indiana		Mercury PCBs	
Louisiana			Mercury
Maine	Mercury	Mercury	Dioxins
Massachusetts	Mercury	Mercury	PCBs organics
Michigan	Mercury		
Mississippi			Mercury
New Hampshire	Mercury	Mercury	PCBs
New Jersey	Mercury	Mercury	PCBs Cadmium Dioxins
New York	PCBs Chlordane Mirex DDT	PCBs Chlordane Mirex DDT	PCBs Cadmium Dioxins
North Carolina	Mercury	Mercury	
Ohio	Mercury	Mercury	
Rhode Island			PCBs
Texas			Mercury
Vermont	Mercury	Mercury	

1998 Advisory Listing

The 1998 database lists 2,506 advisories in 47 states, the District of Columbia, and the U.S. Territory of American Samoa. Some of these advisories represent statewide advisories for certain types of waterbodies (e.g., lakes, rivers, and/or coastal waters). An advisory may represent one waterbody or one type of waterbody within a state's jurisdiction. Statewide advisories are counted as one advisory. The database counts one advisory for each waterbody name or type of waterbody regardless of the number of fish or wildlife species that are affected or the number of chemical contaminants detected at concentrations of human health concern. Eighteen states (Alabama, Connecticut, District of Columbia, Florida, Indiana, Louisiana, Maine, Massachusetts, Michigan, Mississippi, New Hampshire, New Jersey, New York, North Carolina, Ohio, Rhode Island, Texas, and Vermont) currently have statewide advisories in effect (see Table 2). Missouri rescinded its statewide advisories for lakes and rivers in 1998, and Mississippi added a statewide coastal advisory for mercury. A statewide advisory is issued to warn the public of the potential for widespread contamination of certain species of fish in certain types of waterbodies (e.g., lakes, rivers and streams, or coastal waters) or certain species of wildlife (e.g., moose or waterfowl). In such a case, the state may have found a level of contamination of a specific pollutant in a particular fish or wildlife species over a relatively wide geographic area that warrants advising the public of the situation.

Table 3. Fish Advisories Issued for the Great Lakes

Great Lakes	PCBs	Dioxins	Mercury	Chlordane
Lake Superior	●		●	●
Lake Michigan	●		●	●
Lake Huron	●	●		●
Lake Erie	●			
Lake Ontario	●	●		

The statewide advisories and 2,506 specifically named waterbodies represent approximately 15.8% of the Nation's total lake acreage and 6.8% of the Nation's total river miles. In addition, 100% of the Great Lakes waters and their connecting waters are also under advisory (see Table 3). The Great Lakes waters are considered separately from other lakes, and their connecting waters are considered separately from other river miles. The percentages of lake acres and river miles in each state that are currently under a fish advisory are shown in Figures 3 and 4, respectively.

In addition to the Great Lakes, many other Great Waters of the United States are currently under fish consumption advisories for various pollutants. The Great Waters include not only the Great Lakes but also Lake Champlain (which is under advisories for PCBs and mercury), the Chesapeake Bay, 28 National Estuary Program (NEP) Sites, and 23

Figure 3

Percentage of Lake Acres Currently Under Advisory

Eleven states have 100% of their lake acres under fish advisories (these include some states with statewide advisories), another 8 states have 10% to 50% of their lake acres under advisories, 21 states have <10% of their lake acres under advisories, and 15 states have no lake acres under advisories.

Figure 4

Percentage of River Miles Currently Under Advisory

Eleven states have 100% of their river miles under fish advisories (these include states with statewide advisories), 30 states have <10% of their river miles under advisories, and 13 states have no river miles under advisories.

National Estuarine Research Reserve System (NERRS) Sites (see Table 4). Although the Chesapeake Bay itself is not under any advisories, the Potomac, James, Black, and Anacostia rivers, which connect to the Chesapeake, are all under advisories. All of these rivers, with the exception of the James River (which is under advisory for kepone), are under chlordane advisories. The Anacostia River is also listed for PCBs, and the Potomac River is listed for PCBs and dioxins in addition to chlordane. Baltimore Harbor, which also connects to the Chesapeake, is under advisory for chlordane contamination in fish tissue.

A number of the major estuaries listed in the NEP and/or designated as NERRS sites are under fish and/or shellfish advisories for a range of chemical contaminants (see Table 4). Sixty-three percent of the total number of NEP, NERRS, and combined sites are under fish consumption advisories. There are 18 sites that have no current fish consumption advisories.

Several states have issued fish advisories for all of their coastal waters. Using coastal mileages calculated by the

Table 4. Fish Consumption Advisories Issued for NEP and NERRS Sites

Waterbody	PCBs	Dioxins	Mercury	Cadmium	Chlordane	Others
Casco Bay, ME *		●				
Wells, ME #		●				
Great Bay, NH #	●					
Great Bay, Little Bay, and Hampton Harbor, NH *	●					
Massachusetts Bay *	●					●[1]
Buzzards Bay, MA *	●					●[1]
Waquoit Bay, MA #	●					●[1]
Narragansett, RI * #	●					
Long Island Sound, NY/CT *	●	●		●		
Peconic Bay, NY *	●	●		●		
Hudson River, NY #	●			●	●[2]	
New York/New Jersey Harbor *	●	●		●	●	
Barnegat Bay, NJ *	●	●		●	●	
Jacques Cousteau-Great Bay and Mullica River, NJ #	●	●		●	●	
Delaware Estuary, DE/NJ/PA * #	●	●		●	●	
Albemarle-Pamlico Sounds, NC *		●				
Ashepoo-Combahee-Edisto Basin, SC #			●			
Indian River Lagoon, FI *			●			
Charlotte Harbor, FL *			●			
Rookery Bay, FI #			●			
Sarasota Bay, FL *			●			
Tampa Bay, FL *			●			
Apalachicola Bay, FL #			●			
Mobile Bay, AL *			●			
Casco Bay, ME*						
Wells, ME #						
Great Bay, NH #	●					
Weeks Bay, AI #			●			
Barataria-Terrebonne Estuarine Complex, LA *			●			
Galveston Bay, TX *		●	●			
Corpus Christi Bay, TX *			●			
Puget Sound, WA *	●	●	●			●[3]
Columbia River, OR/WA *	●	●				●[4]
San Francisco Bay, CA *	●	●	●		●	●[5]

[1]Organic compounds.

[2]For waterfowl.

[3]Specific embayments of Puget Sound are listed for the following pollutants: creosote, pentachlorophenol, volatile organic compounds (VOCs), tetrachloroethylene, arsenic, metals (unspecified), vinyl chloride, polyaromatic hydrocarbons (PAHs), polynuclear aromatics, and pesticides (unspecified).

[4]DDT.

[5]DDT, dieldrin, other unspecified pesticides.

*NEP site.

#NERRS site.

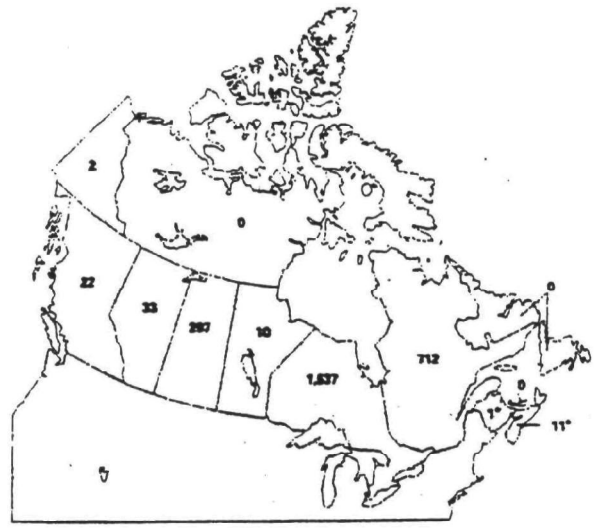
National Oceanic and Atmospheric Administration, an estimated 58.9% of the coastline of the contiguous 48 states currently is under advisory. This includes 61.5% of the Atlantic Coast and 100% of the Gulf Coast. No Pacific Coast state has issued a statewide advisory for any of its coastal waters although several local areas along the Pacific Coast are under advisory. The Atlantic coastal advisories have been issued for a wide variety of chemical contaminants including mercury, PCBs, dioxins, and cadmium, while all of the Gulf Coast advisories have been issued for mercury.

Summary of Canadian Advisories

No new information was collected regarding fish advisories in Canada for 1998. Beginning in 1996, EPA contacted health and environmental officials in the 12 Canadian provinces and territories to obtain narrative and geographic information system (GIS) information on advisories throughout Canada. Figure 5 shows the number of waterbodies under advisory in 1997 for each of the Canadian provinces. The number of Canadian advisories in effect in 1997 was 2,625. Provincewide advisories for mercury were also in effect in 1997 for Nova Scotia and New Brunswick. With respect to chemical contaminants, advisories in Canada have been issued for a total of five bioaccumulative chemical contaminants including mercury (2,572), PCBs (59), dioxins/furans (68), toxaphene (16), and mirex (9). More than 97% of all Canadian advisories have been issued for mercury.

Figure 5

Total Number of Fish Advisories in Effect in Canada



*Provincewide advisories in effect in 1997 for Nova Scotia (all rivers and lakes) and New Brunswick (all lakes).

Database Use and Access

The NLFWA database was developed by EPA to help federal, state, and local government agencies and Native American tribes assess the potential for human health risks associated with consumption of chemical contaminants in noncommercially caught fish and wildlife. The data contained in this database may also be used by the general public to make informed decisions about the waterbodies in which they choose to fish or harvest wildlife; the frequency with which they fish these waterbodies; the species, size, and number of fish they collect; and the frequency with which they consume fish from specific waterbodies.

EPA will make this 1998 update of the NLFWA database available on the Internet at:

http://www.epa.gov/ost/fish

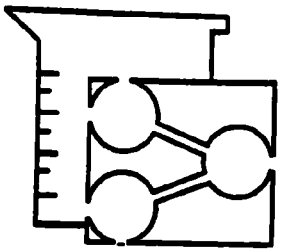
Further information on specific advisories within a particular state is available from the appropriate state agency contact listed in the database. This is particularly important for advisories recommending that consumers restrict their consumption of fish from certain waterbodies. State health departments provide more specific information for restricted consumption advisories (RGP and RSP) on the appropriate meal size and meal frequency (number of meals per week or month) that is considered safe to consume for a specific consumer group (e.g., the general public versus pregnant women, nursing mothers, and young children). For further information on Canadian advisories, contact the appropriate provincial contact given in the database.

For more information concerning the National Fish and Wildlife Contamination Program, contact:

U.S. Environmental Protection Agency
Office of Science and Technology
401 M Street SW, Maildrop 4305
Washington, DC 20460

U.S. EPA contact: Jeffrey Bigler
Phone 202 260-1305 FAX 202 260-9830
e-mail: Bigler.Jeff@epa.gov

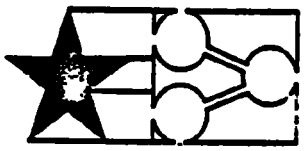
What levels of exposure have resulted in harmful health effects?



Figures 1.1, 1.2, and 1.3 on the following pages show the relationship between exposure to PCBs and known health effects. Other PCBs may have different toxic properties. In the first set of graphs, labeled "Health effects from breathing PCBs," exposure is measured in milligrams of PCBs per cubic meter of air (mg/m^3). In the second and third sets of graphs, the same relationship is represented for the known "Health effects from ingesting PCBs" and "Health effects from skin contact with PCBs." Exposures are measured in milligrams of PCBs per kilogram of body weight per day (mg/kg/day). It should be noted that health effects observed by one route of exposure may be relevant to other routes of exposure.

In all graphs, effects in animals are shown on the left side, effects in humans on the right. The first column on the graphs, labeled short-term, refers to known health effects from exposure to PCBs for 2 weeks or less. The columns labeled long-term refer to PCB exposures of longer than 2 weeks. The levels marked on the graphs as anticipated to be associated with minimal risk of developing health effects are based on information generated from animal studies; therefore, some uncertainty still exists. Based on evidence that PCBs cause cancer in animals, the Environmental Protection Agency (EPA) considers PCBs to be probable cancer-causing chemicals in humans and has estimated that ingestion of 1 microgram of PCB per kilogram per day for a lifetime would result in 77 additional cases of cancer in a population of 10,000 people or equivalently, 77,000 additional cases of cancer in a population of 10,000,000 people. These risk values are plausible upper-limit estimates. Actual risk levels are unlikely to be higher and may be lower.

What recommendations has the federal government made to protect human health?



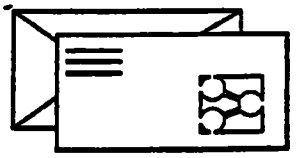
For exposure via drinking water, EPA advises that the following concentrations of PCB 1016 are levels at which adverse health effects would not be expected: 0.0035 milligrams PCB 1016 per liter of water for adults and 0.001 milligrams PCB 1016 per liter of water for children.

EPA has also developed guidelines for the concentrations of PCBs in ambient water (e.g., lakes and rivers) and in drinking water that are associated with a risk of developing cancer. The guideline for ambient water is a range, 0.0079 to 0.79 nanograms of PCBs per liter of water, which reflects the increased risk of one person developing cancer in populations of 10,000,000 to 100,000 people. The guideline for drinking water is a range, 0.005 to 0.5 micrograms of PCBs per liter of water, which also reflects the risk of one person developing cancer in populations of 10,000,000 to 100,000 people.

The Food and Drug Administration (FDA) specifies PCB concentration limits of 0.2 to 3 parts per million (milligrams PCB per kilogram of food) in infant foods, eggs, milk (in milk fat), and poultry (fat).

The National Institute for Occupational Safety and Health (NIOSH) recommends an occupational exposure limit for all PCBs of 0.001 milligram of PCBs per cubic meter of air (mg/m^3) for a 10-hour workday, 40-hour workweek. The Occupational Safety and Health Administration (OSHA) permissible occupational exposure limits are 0.5 and 1.0 mg/m^3 for specific PCBs for an 8-hour workday.

Where can I get more information?



If you have more questions or concerns, please contact your state health or environmental department or:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
1600 Clifton Road, E-29
Atlanta, Georgia 30333

Scott LeRoy

From: Scott LeRoy <sleroy@bestweb.net>
To: sleroy@bestweb.net
Subject: Defining and Demonstrating Injury
Date: Saturday, January 22, 2000 1:49 PM

Response to Public Comments on the Draft Scope for the Hudson River Natural Resource Damages Assessment Plan June 1999 NYSDEC, Wednesday, November 17, 1999

The Hudson River Natural Resource Trustee Council (Trustee Council) received numerous comments on the Draft Scope for the Hudson River Natural Resource Damages Plan. This document is designed to address issues and questions raised in the public comments and provide a general overview of the range of topics identified relative to the Hudson River natural resource damage assessment (NRDA).

Defining and demonstrating injury

Numerous comments related to the way we define and measure injuries. The DOI regulations provide guidance on this topic: they describe the requirements for assessing injuries to natural resources that result from the release of a hazardous substance. The process involves determining a pathway from the source of the hazardous substance(s) to the injured resources, and then determining whether services normally provided by the resource have been reduced as a result of the release. The DOI rule defines injury in terms of direct biological impacts as well as exceedences of federal and state drinking water standards, surface water quality standards and criteria, and relevant Food & Drug Administration action and tolerance levels. A tolerance level exceedence occurs when concentration of a contaminant in an organism(s) is sufficient to exceed levels for which a State health agency has issued limits or bans on their consumption.

Agency for Toxic Substances and Disease Registry

How can PCBs affect my health?

Animal testing is sometimes necessary to find out how toxic substances might harm people or to treat those who have been exposed. Laws today protect the welfare of research animals and scientists must follow strict guidelines. People exposed to PCBs in the air for a long time have experienced irritation of the nose and lungs, and skin irritations, such as acne and rashes. It is not known whether PCBs may cause birth defects or reproductive problems in people. Some studies have shown that babies born to women who consumed PCB-contaminated fish had problems with their nervous systems at birth. However, it is not known whether these problems were definitely due to PCBs or other chemicals. Animals that breathed very high levels of PCBs had liver and kidney damage, while animals that ate food with large amounts of PCBs had mild liver damage. Animals that ate food with smaller amounts of PCBs had liver, stomach, and thyroid gland injuries, and anemia, acne, and problems with their reproductive systems.

Skin exposure to PCBs in animals resulted in liver, kidney, and skin damage.

How likely are PCBs to cause cancer?

It is not known whether PCBs causes cancer in people. In a long-term (365 days or longer) study, PCBs caused cancer of the liver in rats that ate certain PCB mixtures. The Department of Health and Human Services (DHHS) has determined that PCBs may reasonably be anticipated to be carcinogens.

Is there a medical test to show whether I've been exposed to PCBs?

There are tests to find out if PCBs are in your blood, body fat, and breast milk. Blood tests are probably the easiest, safest, and best method for detecting recent exposures to large amounts of PCBs. However, since all people in the industrial countries have some PCBs in their bodies, these tests can only show if you have been exposed to higher-than-normal levels of PCBs. However, these measurements cannot determine the exact amount or type of PCBs you have been exposed to or how long you have been exposed. In addition, they cannot predict whether you will experience any harmful health effects.

Has the federal government made recommendations to protect human health?

The EPA has set a maximum contaminant level of 0.0005 milligrams PCBs per liter of drinking water (0.0005 mg/L). The EPA requires that spills or accidental releases into the environment of 1 pound or more of PCBs be reported to the EPA. The Food and Drug Administration (FDA) requires that milk, eggs, other dairy products, poultry fat, fish, shellfish, and infant foods contain not more that 0.2–3 parts of PCBs per million parts (0.2–3 ppm) of food.

Environmental Health Perspectives Volume 103, Supplement 5, June 1995

[Citation in PubMed] [Related Articles]

Aerobic and Anaerobic PCB Biodegradation in the Environment

Daniel A. Abramowicz

Environmental Laboratory, GE Corporate Research and Development, Schenectady, New York

Abstract

Studies have identified two distinct biological processes capable of biotransforming polychlorinated biphenyls (PCBs): aerobic oxidative processes and anaerobic reductive processes. It is now known that these two complementary activities are occurring naturally in the environment. Anaerobic PCB dechlorination, responsible for the conversion of highly chlorinated PCBs to lightly chlorinated *ortho*-enriched congeners, has been documented extensively in the Hudson River and has been observed at many other sites throughout the world. The products from this anaerobic process are readily degradable by a wide range of aerobic bacteria, and it has now been shown that this process is occurring in surficial sediments in the Hudson River. The widespread anaerobic dechlorination of PCBs that has been observed in many river and marine sediments results in reduction of both the potential risk from and potential exposure to PCBs. The reductions in potential risk include reduced dioxinlike toxicity and reduced carcinogenicity. The reduced PCB exposure realized upon dechlorination is manifested by reduced bioaccumulation in the food chain and by the increased anaerobic degradability of these products. -- **Environ Health Perspect** 103(Suppl 5):00-00 (1995)

Key words: aerobic PCB biodegradation, anaerobic PCB dechlorination, dioxinlike toxicity, carcinogenicity, PCB biotransformation

This paper was presented at the Conference on Biodegradation: Its Role in Reducing Toxicity and Exposure to Environmental Contaminants held
26-28 April 1993 in Research Triangle Park, North Carolina.

Address correspondence to Dr. Daniel A. Abramowicz, Manager, Environmental Laboratory, GE Corporate Research and Development, P.O. Box 8, Schenectady, NY 12301-0008. Telephone (518) 387-7072. Fax (518) 387-7611.

[Table of Contents] [Full Article] [Citation in PubMed] [Related Articles]

Last Update: September 24, 1998

HG-1

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February 4, 2000

Alison A. Hess, C.P.G.
U.S. Environmental Protection Agency
290 Broadway, 19th Floor
New York, NY 10007-1866

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 – A Human Health Risk Assessment For the Mid-Hudson River" (HHRA, December 1999).

The central conclusion of the mid-Hudson Human Health risk assessment is that PCBs pose no unacceptable risk to people who swim, wade, or boat in or drink water from the mid-Hudson river, or breath the air in the vicinity of the river. The sole risk of concern to EPA was to the hypothetical person who consumes extraordinary large amount of fish over a large period of time. Even in this case the calculated risks were very near to the level deemed to be acceptable by EPA. All-in-all this should have come as very good news particularly considering that the analysis was based on assumptions that grossly overestimated exposure to and toxicity of PCBs.

We were disappointed to see that comments we submitted to EPA on September 7, 1999 on your upper Hudson Human Health Risk Assessment were not considered and as a result this risk assessment suffers from the same flaws.

While we have attempted to work within the stringent comment deadlines you imposed on commentors, we found it impossible to complete our review since we only just received the EPA Baseline Modeling Report that provides one of the key inputs into the risk assessment. As a result we reserve the right to supplement these comments.

Please place a copy of this letter and associated comments in the site administrative record.

If you have any questions on these comment, please let me know

Yours truly,

John G. Haggard

JGH/bg

Enclosure

cc: Richard Caspe, U.S. EPA
William McCabe, U.S. EPA
Douglas Fischer, U.S. EPA (ORC)
Marion Olsen, U.S. EPA
Michael O'Toole, NYDEC
Walter Demick, NYDEC
Nancy Kim, NYDOH
Anders Carlson, NYDOH
Bob Montione, NYDOH

COMMENTS OF GENERAL ELECTRIC COMPANY ON

Mid-Hudson River Human Health Risk Assessment
Hudson River PCBs Superfund Site Reassessment RI/FS

February 4, 2000

General Electric Company
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Albany, NY 12203

Ogden Environmental and Energy Services
15 Franklin Street
Portland, ME 04101

TABLE OF CONTENTS

1.0 Introduction and Executive Summary

General Electric Company submits these comments on EPA's Mid-Hudson River Human Health Risk Assessment (Mid-HHRA). In September, 1999, GE submitted comments on EPA's Human Health Risk Assessment for the Upper Hudson River (Upper-HHRA). In light of the similarities between the two documents, it is apparent that EPA failed to consider GE's earlier comments in preparing the Mid-HHRA. The Mid-HHRA thus suffers from many of the same problems as the Upper-HHRA.

Despite the ominous language that EPA favors, the central conclusion in the Mid-HHRA is that PCBs pose no unacceptable risk to people who swim, wade, boat in or drink water from the mid-Hudson River or breathe the air near the river. EPA asserts there is a remote risk of an additional case of cancer in 10,000 among people who eat extraordinarily large quantities of fish (a half pound a week for 40 years), whose diet inexplicably tilts toward some of the most unpopular and unappetizing fish (eel and carp), and who, for no clear reason, eat the same species of fish from the same part of the river each week for 40 years — a combination of unrealistic circumstances. HG-1.1

GE's comments on the Mid-HHRA focus on several problems:

- The same unrealistic exposure and toxicity assumptions that GE and others identified with respect to EPA's Upper-HHRA are repeated in the Mid-HHRA and result in significant overestimates of potential risk. HG-1.2
- The predictions of water, sediment and fish PCB concentrations that form the foundation of the Mid-HHRA are highly uncertain, are based on unvalidated and unreviewed models, and fail to account properly for other PCB sources. HG-1.3
- Unlike the Upper-HHRA, EPA does not bother to conduct a probabilistic analysis of risks from fish consumption for the Mid-HHRA. EPA's basis for this decision – that PCB levels are lower in the Mid-Hudson - is nonsensical and is inconsistent with EPA guidance. As a result, the Mid-HHRA relies entirely on a flawed, screening-level, point estimate analysis. HG-1.4

- EPA inappropriately treats the Mid-Hudson as part of the reassessment of the Hudson River PCBs Superfund Site which is limited to the stretch of river between Fort Edward and Troy. HG-1.5

2.0 The Flaws in the Upper-HHRA Are Repeated in the Mid-Hudson Assessment and Result in Overestimates of Risk

In September 1999, GE submitted substantial comments on EPA's Upper-HHRA. These comments identified a number of significant flaws in that risk assessment. Although EPA had nearly four months to address the issues identified in GE's comments, the Mid-HHRA neither acknowledges these comments nor attempts to address them in any fashion. As a result, the Mid-HHRA repeats many of the flaws GE had previously identified, flaws that result in significant overestimates of risk to the Mid-Hudson angler. Rather than repeat GE's earlier comments, we incorporate them by reference and summarize them below.

The Mid-HHRA Overstates the Toxicity of PCBs: As with the Upper-HHRA, the Mid-HHRA relies entirely on animal-based estimates of PCB toxicity and fails to adequately consider the available human epidemiological data, including the findings of Kimbrough, et al. (1999). GE's Upper-HHRA Comments include a detailed critique of these animal studies, their relevance to humans, and a methodology for considering the epidemiological data. See Appendix A to GE's Upper-HHRA Comments. HG-1.6

The Mid-HHRA Improperly Dismisses the Kimbrough Study: EPA's presumptive conclusion that this study will not result in a change in the Agency's cancer slope factor for PCBs, coming before the Agency completes its own, internal review of Kimbrough et al. (1999), is ill-considered and has no support in the record. Indeed, GE's earlier comments responded in detail to the Agency's earlier criticisms of Kimbrough et al. (1999), but, as with GE's other comments, the mid-HHRA appears to ignore the substance of these comments in its unfounded dismissal of this study. HG-1.7

The Mid-HHRA Relies on the Wrong Study to Estimate Fish Consumption Rates: The Mid-HHRA uses the same problematic study – Connelly et al. (1992) – to estimate fish consumption rates that was used in the Upper-HHRA. These limitations are set out in detail in Appendix B to GE's Upper-HHRA Comments. Problems include in-compatibility of results with other surveys of northeastern anglers, low survey response rate, incorrect weighting of non-respondents, long-term recall bias, lack of information on meal sizes, and the need to make uncertain assumptions from survey results about the fish caught and consumed. For the reasons described in our earlier comments, the Agency should have used the Connelly et al. (1996) and/or the Ebert et al (1993) surveys. HG-1.8

The Mid-HHRA Improperly Accounts for Cooking Loss: Although acknowledging that PCBs are removed during cooking, the Mid-HHRA, as with the Upper-HHRA, underestimates these losses. Instead of using 20 percent loss for the central tendency and zero percent loss for the RME, EPA should have done a Monte Carlo analysis using the probability distribution. HG-1.9

- The Mid-HHRA Improperly Relies on Connelly et al. (1992) Data to Establish Species Preference: Because the Connelly et al. (1992) study was not designed to ascertain species preference, but instead was intended to measure anglers' understanding and compliance with consumption advisories, it should not be used to establish species preference. Further, the species listed in the survey are different from those that would be expected to be caught in the mid-Hudson. When combined with the significant uncertainty required to extrapolate from the survey results, Connelly et al. (1992) is the wrong study to use for species preference. HG-1.10

  These problems are exacerbated in the Mid-HHRA by the manner in which EPA used the Connelly et al. (1992) study, in conjunction with Barclay (1993), to conclude that more than 50% of the species the average angler targets and eats are comprised of bottom-feeders, such as catfish, brown bullhead, and eel. This result is not only contrary to common sense, it is inconsistent with the Barclay and NYSDOH data, as well as the available abundance data which show that the anadromous species, such as striped bass and members of the herring family are the primary fish in the Lower Hudson River.

  The Barclay (1993) data (as presented in NYSDOH, 1999) show that the bottom feeders (brown bullhead, carp, catfish, and eel) comprise only 24% of the catch, significantly lower than the species preference of 52% estimated by EPA. Conversely, the Barclay (1993) data show a species preference of 26% for white perch, which is substantially greater than EPA's estimate of 7.6%. Preference for yellow perch is also higher than the preference used by EPA. Barclay (1993) also demonstrated that there is a substantial species preference for herring and American shad. These species are not considered at all by EPA.

In addition to the issues identified above, there are several additional issues that require further discussion.

2.1.1 EPA Should Reevaluate its Current RfD for PCBs

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 Mid-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 HG-1.11

study clearly do not demonstrate clinically significant effects (see Paul and White, 1973; ATSDR, 1993; Kimbrough, 1995). Furthermore, studies of PCB-exposed workers showed no adverse immunological effects or clinical signs of immunocompromise, even when the workers' blood concentrations of PCBs were more than ten-fold greater than the levels measured in the Rhesus monkeys (Emmett et al., 1988a; 1988b). 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. For example, none of the studies of highly exposed workers have reported finding the pattern of nail, dermal, and ocular effects seen in the primates (Ouw et al., 1976; Smith et al., 1982; Wolff et al., 1982; Lawton, 1985; Emmett et al., 1988a,b; Taylor et al., 1988). The reasons for this are apparent from the differences in metabolism between Rhesus monkeys and humans (Brown, 1994). In fact, the data indicate that humans are many 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 that demonstrate that the RfD is based on a gross exaggeration of the potential human health risks of PCBs.

If EPA continues to rely on the monkey study as the critical study to derive a deterministic RfD, EPA should apply uncertainty factors based on recent data regarding exposure and toxicity of PCBs in humans and experimental animals. These uncertainty factors (UF) should be as follows: (1) a subchronic-to- chronic UF of 1 based on the fact that the monkeys were dosed for more than 25 percent of their lifetimes and pharmacokinetic equilibrium had been reached between PCB concentrations in adipose tissue and blood; (2) an interspecies UF of 1 based on evidence that demonstrates that humans are less sensitive to the effects of PCBs than are Rhesus monkeys; and (3) consistent with EPA practice, UFs of 10, 3, 1 for interindividual variability, minimal LOAEL to NOAEL extrapolation, and database uncertainty, respectively. Application of these appropriate UFs results in a chronic RfD for Aroclor 1254 of 2×10^{-4} mg/kg-day, which is ten times higher than the value currently used by EPA.

While a deterministic 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 HG-1.12

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.

2.1.2 EPA Incorrectly Dismissed the Findings of the Kimbrough Study

GE's comments on the Upper-HHRA responded to several purported "limitations" of the Kimbrough et al. (1999) epidemiological study of capacitor workers identified by EPA. Rather than address GE's comments, the Mid-HHRA summarily dismisses the Kimbrough et al. (1999) study on the grounds that these "limitations," combined with those identified in two Letters to the Editor in the Journal of Occupational and Environmental Medicine (in which Kimbrough et al. (1999) was originally published), lead the Agency to conclude now that the "study will not lead to any change in its CSFs for PCBs." Mid-HHRA at 24. HG-1.13

The record provides no basis for EPA to reach this conclusion. EPA has not completed its "internal" peer review (which will supplement the two rounds of pre-publication peer review to which the study was subject), and it is premature to guess at what conclusions that review might reach about the value of the study. Nor do the Letters to the Editor raise new and substantial issues about the Kimbrough et al. (1999) study. Kimbrough et al. responded to all these criticisms in detail, demonstrating why they do not undermine the validity of the study's conclusions (A copy of these letters and Kimbrough et al.'s response is attached to GE's comments in Appendix A). Simply citing EPA's earlier "criticisms" and these letters as purported evidence of controversy about the study is not a valid basis for rejecting it and does not substitute for an unbiased, reasoned and detailed assessment of the study itself.

Rather than rehash the controversy surrounding the Kimbrough et al. (1999) study, EPA should turn its attention to determining how the study can be used to improve the validity of and certainty associated with EPA's CSFs for PCBs. A critical element of this effort should be to focus on determining the "dose" of PCBs to which the studied workers were exposed. With a proper reconstruction of the dose, one can use the valuable data from the Kimbrough et al. (1999) study to test and, if appropriate, revise the CSFs for PCBs.

3.0 EPA Inappropriately Treats The Mid-River As Part Of The Hudson River PCB Superfund Site

EPA continues its fallacious claim that the Hudson River PCBs Superfund Site extends below the Federal Dam at Troy to the Battery in New York City. GE has addressed this issue in the past. The Site is limited to approximately 40 miles of the River between the Federal Dam and Fort Edward. This conclusion is consistent with the administrative record on which the listing of the Site on the National Priorities List, is based. EPA's post-rulemaking statements to the contrary cannot modify the promulgated extent of the Site. United States v. ASARCO, Inc., 28 F.Supp.2d 1170 (D.Idaho, 1998) (post-rulemaking statement cannot expand scope of Site). In any event, many post-rulemaking statements of the Agency are from the site boundaries set out in the NPL. Indeed, EPA's singular remedial focus on the sediments in the Upper Hudson River underscores the fact that the Agency still treats the Upper Hudson as the Superfund site. HG-1.14

This point has more than academic interest. In the Mid-HHRA, EPA "evaluates both current and future risks . . . in the absence of any remedial action and institutional controls" in order to "establish acceptable exposure levels for use in developing remedial alternatives for PCB-contaminated sediments in the Upper Hudson River." Mid-HHRA at ES-1, ES-2. In other words, EPA intends to use the results of the Mid-HHRA to provide justification for remedial action in the Upper Hudson. It would be reasonable to look at the effect of potential remedial measures in the upper river to assure that a possible remedy will not adversely impact the lower river. On the other hand, in light of the fact that the Site does not extend to the lower river and EPA is not examining potential remedies or PRPs in the lower river, it is unreasonable to seek to justify upper river remedial action on the basis of purported benefits to those who consume lower river fish.

The impropriety of such an approach is obvious. The presence of sources of PCBs in the lower river is well known to EPA; EPA, New York and New Jersey, in fact, are engaging in an extensive effort to identify and reduce such sources. The Agency also made the importance of other contaminants plain in its 1984 ROD, concluding "that detectable levels of dioxin,

dibenzofurans, mercury and chlordane (from known and unknown sources) have also been identified in Hudson River fish, and that even if PCBs decrease to an acceptable level, the fishing bans would continue on the basis of these other types of contaminants." Many of the most desirable fish in the lower Hudson, such as striped bass, are migratory and thus are exposed to many potential sources of PCBs and other contaminants. Despite these facts, EPA's remedial focus remains fixed on the PCBs in the sediments of the upper river and, effectively on a single PRP. The Agency is not examining potential remedial alternatives in the lower river to determine their potential benefits to lower river fish consumers or even comparing the effect of such remedies with the actions it is considering in the upper river. Simply put, the Agency can not rely on benefits to the lower river, where numerous PCB sources exist and other contaminants may be of concern, to justify remediation in the upper river without looking at alternatives that directly address those lower river sources.

In short, EPA cannot have it both ways. The Agency cannot describe the site as encompassing the 150 miles from Troy to the Battery and then address only one contaminant and one area outside that 150 miles as the sole subjects for remedial consideration. Quite apart from the legal requirements, if one expands a Superfund site by 150 miles to take in a diversely populated estuary exhibiting contamination from a large array of sources and chemicals, one cannot continue to consider only one area, one chemical, and one PRP as the target of remediation. Superfund did not legalize vendettas.

The scope of EPA's Superfund activity at the Site is circumscribed by the characterization and definition of the site, which EPA promulgated in its rule making many years ago.

4.0 The Predictions of Water, Sediment and Fish PCB Concentrations that Form the Foundation of the Risk Assessment are Highly Uncertain and Fail to Properly Account for All PCB Sources

HG-1.15

The Mid-HHRA relies on predictions of fish, water and sediment PCB concentrations made by the Farley et al. (1999) fate and bioaccumulation model, EPA's bioaccumulation model (FISHRAND), and EPA's fate and transport model (HUDOX). As discussed in our comments

on the baseline ecological risk assessment for the Lower Hudson River (GE, 2000), the validity of these predictions is questionable because of inaccurate descriptions of the processes controlling PCB fate and bioaccumulation. Of particular concern for the HHRA is that the inaccuracy and uncertainty of the predictions increases with the length of the prediction. Thus, the 40-year predictions used in the HHRA are subject to a large, but unknown, degree of inaccuracy. A significant issue in this regard is the impact of the incorrect specification of the migratory behavior of striped bass (and movements of motile species such as white perch). The assumption that striped bass are exposed to PCBs only in the mid-Hudson results in a failure to account for the substantial contribution from the lower estuary and New York Harbor. This contribution increases with time in the model projections as the PCB load from the Upper Hudson River declines and the PCB load from the metropolitan NY/NJ PCB sources remains constant (an assumption in the model predictions).

5.0 EPA Failed to Conduct a Probabilistic Model of Potential Exposure to Anglers on the Mid-Hudson River

HG-1.16

Although EPA conducted a probabilistic assessment of risk for the Upper-HHRA, it failed to include such an analysis for the Mid-Hudson reasoning that "a Monte Carlo analysis of cancer risks and non-cancer hazards for the fish ingestion pathway was not warranted for the Mid-Hudson HHRA, because the concentrations of PCBs in the Mid-Hudson River are lower than in the Upper Hudson." [Mid-HHRA, page ES-2] This rationale is nonsensical and inconsistent with EPA guidance.

EPA's justification for not performing a Monte Carlo analysis is inadequate. It is clear from the Phase 2 Scope of Work (EPA, 1998) and the Phase 2 Responsiveness Summary (EPA, 1999) that EPA intended to conduct a Monte Carlo analysis for the Mid-Hudson. Perhaps the most compelling examples are a subsection in the Scope of Work entitled "Monte Carlo Analysis", where EPA states "as in the Upper Hudson Risk Assessment, the Monte Carlo analysis will evaluate annual exposures on a year by year basis..." (EPA, 1998), and in response to comments on fish consumption rates in the Responsiveness Summary, EPA (1999) states "in addition, the Monte Carlo analysis will consider the full distribution of risk and hazards for Hudson River

anglers." EPA gives no hint in either document of a situation where a Monte Carlo analysis would not be warranted for the Mid-Hudson River.

EPA (1997) in its guiding principles for Monte Carlo analysis, describes several situations where a Monte Carlo analysis is warranted. It is this same guidance that EPA (1998) cites in the Phase 2 Scope of Work when describing the presentation of the results of the Monte Carlo analysis – "the Monte Carlo analysis information will be presented following the recommendations outlined in the Policy for Use of Probabilistic Analysis in Risk Assessment" (EPA, 1997). Thus, EPA fails to follow its guidance by not conducting a Monte Carlo analysis for the Mid-Hudson.

According to EPA's guidelines for probabilistic analysis, a Monte Carlo analysis is useful when screening-level risk estimates are above levels of concern. In addition, a Monte Carlo analysis is useful "when it is necessary to disclose the degree of bias associated with point estimates of exposure; when it is necessary to rank exposures, exposure pathways, sites or contaminants; when the cost of regulatory or remedial action is high and the exposures are marginal; or when the consequences of simplistic exposure estimates are unacceptable." (EPA, 1997). A Monte Carlo analysis does not add value only when screening risk estimates are clearly below levels of concern or when the costs of remediation are low (EPA, 1997). Low contaminant concentrations are not a valid basis for not performing a Monte Carlo analysis.

All the factors favoring application of Monte Carlo techniques are present here. EPA's Mid-Hudson point estimate analysis purports to show that risks from fish consumption are unacceptable. Only a Monte Carlo analysis can begin to characterize the degree of bias associated with these point estimates.

Accordingly, EPA should conduct a Monte Carlo analysis for the Mid-Hudson River. EPA has previously developed a Monte Carlo exposure model for the Hudson River. Although this model is flawed (as noted in GE's comments on the Upper-HHRA), no additional development time would be required to implement the model. Whether this model or GE's more sophisticated time-dependent two-dimensional model, as detailed in GE's comments on the Upper HHRA

(GE, 1999) is used, we believe the results will demonstrate that fish consumption in the Mid-Hudson is unlikely to pose unacceptable risks[1].

6.0 Conclusions

The purpose of the Mid-HHRA is to characterize current risks and their associated uncertainties.

HG-1.17

In some regards the Agency has performed well, and in others it has not. The Mid-HHRA concludes that the only material human health risk is the potential consumption of fish from the Mid-Hudson River. EPA, however, poorly characterizes the fish consumption pathway and arrives at hypothetical risk estimates that are unrealistically overstated. Furthermore, the risk assessment poorly communicates the findings and uncertainties. The major problems include:

- The Mid-HHRA follows a screening-level, point estimate approach. A Monte Carlo analysis, even a limited one like EPA's model of the Upper Hudson River, would result in reduced risk estimates and different risk conclusions. HG-1.18

- 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-1.19

- 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-1.20

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[1] We assume that EPA's statement that cancer risks from fish consumption "are within the upper bound of the cancer risk range generally allowed under the federal Superfund law" [Mid-HHRA at____] is a typographical error, but if not, EPA must clarify its conclusions about the cancer risks posed by fish consumption.

- The exposure assumptions made to estimate risks to the angler materially overstate potential exposures. HG-1.21

As a result, it is apparent that EPA needs to redo the calculations of potential risk to the angler in the Mid-Hudson River to correct these errors. Using a Monte Carlo analysis, the cancer risks would be acceptable, even if EPA uses its flawed model.

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Attachments

Attachment A: Copies of the Letters to the Editor, *Journal of Occupational and Environmental Medicine* 41(9): 739-745. September 1999.

Letters to the Editor

Readers are invited to submit letters for publication in this department. Submit them to The Editor, Journal of Occupational and Environmental Medicine, PO Box 370, Bryn Mawr, PA 19010. Letters should be typewritten and double spaced and should be designated "for Publication."

Evidence of Excess Cancer Mortality in a Cohort of Workers Exposed to Polychlorinated Biphenyls

To the Editor: To further explore previously reported excesses in cancer-specific mortality in workers who have been occupationally exposed to polychlorinated biphenyls (PCBs), Kimbrough et al[1] reported a retrospective cohort mortality study of 7075 male and female workers exposed to PCBs during the capacitor-manufacturing process at two General Electric (GE) plants in upstate New York. Kimbrough et al concluded that the study results failed to show any association between occupational PCB exposure and cancer-related mortality. We interpret their study findings differently. Although limitations in the study approach (outlined below) tend to dilute any excesses in cancer mortality resulting from PCB exposure, the findings *still* suggest a relationship between PCB exposures and excess cancer in humans.

First, this study demonstrated once again that modern industrial workers are healthier than the general population. Known as the "healthy worker effect" (HWE), this bias results in standardized mortality ratios (SMRs) that are considerably less than expected (eg, SMR < 90) for all mortality and cancer mortality[2–4] when workers are compared with a general population. Consistent with the HWE bias, Kimbrough et al found that all cancer mortality was significantly below that expected in male hourly workers (SMR = 81), male salaried workers (SMR = 69), and female salaried workers (SMR = 75). However, despite the HWE, female hourly workers had elevated SMRs for all cancer mortality (SMR = 110) and for three (intestinal [SMR = 157], rectal [SMR = 169], and melanoma [SMR = 144]) of the six cancers of a priori interest. Melanoma mortality was also elevated for male hourly workers (SMR = 130). Although the elevations in cancer-specific SMRs did not achieve statistical significance, they were consistent with elevations found in other studies of PCB-exposed workers.[5–6] Given the HWE, these elevations are particularly noteworthy.

Second, when looking at cancer mortality rates, it is customary to include a latency period to adjust for the time lag between exposure and clinical evidence of disease (or, in this study, cancer death).[7] However, Kimbrough et al included a latency period only for all cancer mortality and for intestinal cancer mortality among female hourly workers. When female hourly workers with at least 20 years of follow-up were evaluated (ie, with a sufficient latency period), the SMR for all cancers increased from 110 to 117* ($P = 0.058$). The SMR for intestinal cancers increased from 157 to 189, thus becoming statistically significant ($P < 0.05$).

Third, proper assessment of exposure should have accounted for the dates (calendar years) of employment, the intensity of exposure for each type of job, and the specific Aroclor PCB used. For example, in the earlier years of plant operation (1946 to 1954), any exposures would have been to Aroclor 1254, whereas exposures in the 1970s would have been to the less toxic Aroclor 1016.[8,9] Industrial hygiene procedures at the plant probably improved over time as well. Therefore, length of employment alone was an inadequate surrogate of exposure and a likely source of exposure misclassification bias that could have led to an underestimate of effect and distortion of exposure-response relationships.

Kimbrough et al assembled the largest cohort of hourly PCB workers studied to date, including a large number of female workers. However, most of the hourly workers had exposures that were comparable with exposures among the general US population. From the data provided, it appears that approximately one fourth of the person-years contributed by male hourly workers, and approximately 10% of the person-years contributed by female hourly workers, were contributed by workers who had been employed for at least 6 months in high-exposure jobs. Only 112 (3.8%) male hourly workers and 12 (0.5%) female hourly workers were employed exclusively in high-exposure jobs. The majority of the hourly workers never worked in high-exposure jobs. Only a small percentage of hourly workers had evidence of PCB exposure that was appreciably greater than that of the US population. Therefore, relatively small elevations in cancer mortality would be expected for this group, even if PCB cancer potency were alarmingly high.

Fourth, although one of the goals of this study was to evaluate six specific cancers of a priori interest (ie, melanoma, liver, rectal, gastrointestinal tract, brain, and hematopoietic cancers), the study focused almost entirely on all cancer mortality. In planning the study, the researchers should have realized that the size and age distribution of the hourly work-

*Note: There is an error in Table 6 of the study report. The SMR for "all cancers" in female hourly workers with ≥20 years' latency over all lengths of employment should be "117" and "96" as reported.

TABLE 1
Calculations of Statistical Power to Detect Varying Standardized Mortality Ratios (SMRs) for the Six Cancers of A Priori Interest

Cancer	Expected Number	SMR = 150	SMR = 200	SMR = 300
Male hourly workers				
Melanoma	3 8	12%	35%	80%
Liver	2 5	9%	24%	62%
Rectum	3 4	14%	37%	80%
GI*	14 0	36%	85%	100%
Brain	5 1	15%	44%	89%
Blood	14 1	37%	86%	100%
Female hourly workers				
Melanoma	2 0	8%	22%	55%
Liver	2 2	12%	28%	65%
Rectum	1 6	10%	22%	52%
GI	12 7	36%	83%	100%
Brain	3 7	11%	32%	78%
Blood	10 5	32%	77%	100%

* GI, Gastrointestinal tract

force would result in poor statistical power to evaluate the cancers of a priori interest Table 1 shows the expected number of deaths for each of these cancers for male and female hourly workers and the resulting statistical power for SMRs from 150 to 300, using the study's method for determining statistical significance (ie, the 95% confidence interval) Because of the biases in the study and the low percentage of highly exposed workers, an SMR of 150 might be as high as would be expected for these cancers As seen in Table 1 for an SMR of 150, the study had less than a one in five chance of obtaining a statistically significant result for four of the six cancers Given the sample size and the numbers of expected cancers, the study did not have sufficient statistical power (>80%) to detect an SMR of 300 for most of the cancers of interest.

Kimbrough et al examined and reported SMRs for categories of increasing length of employment and years of latency only when " . there was an elevated total SMR with two or more observed deaths and for which the lower boundary of the 95% confidence interval (CI) was 90 or above "[1] The impact of this decision can be seen in Table 2 Given

TABLE 2
Number of Observed Deaths and the SMR Required for ≥90 as the Lower Limit of the 95% Confidence Interval

No. of Deaths	SMR
2	744
3	437
4	331
5	278
6	245
7	224
8	209
9	197
10	188
11	180
12	174
13	169
14	165
15	161
16	157
17	154
18	152
19	150

the biases mentioned previously, it is understandable that just one of the six a priori cancers met these requirements Furthermore, accounting for a latency period should be a prerequisite for calculating any adult cancer SMR. Otherwise, the SMR is biased toward or below 100. For all six cancers of a priori interest, analyses accounting for latency and for length of employment should have been done and presented, allowing the reader to decide whether or not the results were meaningful

In summary the Kimbrough et al study suffered from HWE bias, failure to account for latency, exposure misclassification potentially insufficient dosage differences between exposed and comparison groups, and poor statistical power Nevertheless, the study did find excesses in three of the six cancers of interest. Future research should include analyses made with internal comparisons (to minimize biases from HWE) of sufficient numbers of highly exposed workers, as well as analyses accounting for cancer latency periods This might require an additional decade or more of follow-up on this cohort and the addition of exposed workers from other PCB plants (eg, workers at the Massachusetts plant included in Brown[5]), before a definitive statement about the association between PCB exposure and specific cancers can be made

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To the Editor. We were glad to see the recent article on mortality among workers exposed to polychlorinated biphenyls.[1] At a time when fewer and fewer companies are funding occupational epidemiological studies, we commend the sponsor, General Electric, for this initiative. The completeness of case ascertainment was outstanding. In addition, this report was a model of clear writing and clear display of results.

However, two issues, sample size and exposure, raise significant concern. First, the study population was very small. Over 7000 workers contributed over 200,000 person-years of observation, more than in prior PCB mortality studies. But when attention is restricted to those workers with high exposure, moderate- to long-duration employment, and adequate person-time after a latency period, the numbers are dramatically reduced. For example, only one third of the cohort worked for longer than 5 years. (We note in passing that Table 2, the source of these data, shows 7178 workers in the upper panel and 7075 workers in the lower panel, a disparity the authors do not explain.) Similarly, less than one fourth of the cohort was classified as highly exposed, and the median period of high exposure was less than 2 years. Although data are not presented to support exact calculations, it appears that fewer than 10 cancers of any type, and more typically fewer than three, were expected in any sex-salary stratum with high exposure, more than a year of employment, and more than 20 years of latency. Could this be why the article is conspicuously silent on the issue of statistical power?

The problem of small number could have been addressed. A company as large as GE presumably had other capacitor plants and could have supported a multisite study. Alternatively, an industry-wide study would have been informative as we have seen in the semiconductor, rubber, petrochemical, automobile, and other industries. Indeed, we wonder why restricting a cancer mortality study to only two plants should not be viewed as a willful effort to avoid a positive finding.

The second major concern lies with exposure assessment. As with many historical cohort studies, the authors created a matrix to characterize each individual's exposure. If the designated "high exposure" jobs did not actually entail high exposure, then misclassification occurred and could have introduced substantial bias toward the null. Were the exposures accurately assessed?

The article makes reference to a readily available way to validate the exposure assessment, serum PCB levels obtained during the 1970s on a sample of several hundred cohort members. Where are these measurements? Did the authors check their exposure assignments against the past serum measurements? If not, why not? If so, why was this comparison not reported?

Another difficulty with exposure in this article is the admixture of various types of PCBs. More carcinogenic forms, such as Aroclor 1254, were used in the early years, and less carcinogenic forms, such as Aroclor 1016, were used later. By combining the two rather than focusing on the early exposures, the authors may have obscured a true effect.

Overall, these concerns significantly limit the conclusions that can be drawn from the study. The authors conclude that their results "would suggest a lack of an association." This conclusion is overstated. These results do offer some evidence that PCBs are not highly potent carcinogens causing relative risks above 10 or 20, a conclusion that was already fairly well established. But they provide little reassurance that PCBs do not double or triple the risk of some cancers after significant exposure.

For this reason, we were especially concerned that the results of the study were not interpreted and presented more carefully. The authors might have noted, in their conclusion, that PCBs are serious health hazards irrespective of carcinogenicity,[2] with effects that include decreased birth weight,[3] neurodevelopmental abnormalities,[4–6] and interference with both estrogen[9] and thyroid[10] hormone function. Accordingly, even negative findings in a cancer study would not reassure us of safety. That omission in the *JOEM* article, in turn, may have contributed to overtly misleading journalistic coverage, such as the *New York Times* headline: "Study Finds Little Risks [sic] From PCB's."[11]

The authors of this study note that our knowledge of PCB health effects is "limited." On the path to a more complete understanding, the current study results represent a great leap sideways.

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The Authors Reply: Thank you for giving us the opportunity to reply to the letters by Bove et al and Frumkin and Oris commenting on our mortality study of PCB-exposed capacitor workers.[1] We disagree with the statement by Bove et al that "... limitations in the study approach tend to dilute any excesses in cancer mortality resulting from PCB exposure . . ." These assertions are speculative and not supported by the data. Although some degree of misclassification in observational studies is unavoidable, it is usually not possible to determine whether this misclassification is differential or non-differential. Furthermore, non-differential misclassification does not always result in bias toward the null hypothesis. Neither the type nor the effect of the misclassification can be determined by Bove et al. In our article, we do, however, discuss at length the measures taken to limit misclassification, and we feel strongly that we were successful in doing so.

Bove et al assert that the healthy worker effect (HWE) results are an underestimate of the SMRs for all-causes mortality and cancer mortality. This is partially true. The HWE is most pronounced for cardiovascular deaths and thus affects all-causes mortality.[2] It has much less of an effect on cancer deaths.[3]

The presentation by Bove et al of the all-cancers SMRs and selected cancer-specific SMRs without confidence intervals (CIs) gives incomplete information and is misleading. Had the confidence intervals been reported, the lack of significance for these SMRs would have been immediately obvious to the reader. Bove et al selected the female hourly employees all-cancers SMR of 110 (95% CI, 93 to 129), intestinal cancer (SMR = 157; 95% CI, 96 to 242), rectal cancer (SMR = 169; 95% CI, 46 to 434), melanomas (SMR = 144; 95% CI, 30 to 421), and melanomas in male hourly employees (SMR = 130; 95% CI, 42 to 303). Notably absent from this list of SMRs considered by Bove et al are the male hourly SMRs for intestinal and rectal cancer (SMR = 57; 95% CI, 25 to 112; and SMR = 87; 95% CI, 18 to 255, respectively).

Bove et al suggest that the male all-cancers SMRs of 81 (hourly employees; 95% CI, 68 to 97) and 69 (salaried employees, 95% CI, 52 to 90) are largely due to the HWE. A careful examination of Table 4 in our article suggests that the statistically significantly low all-cancers SMRs in both the hourly and salaried males result primarily from the lower than expected lung cancer SMR (for hourly workers 42 observed/54.5 expected, SMR = 77, 95% CI 56 to 104, and for salaried workers 12 observed/29.6 expected SMR = 41, 95% CI, 21 to 71).

The statement by Bove et al that these elevations were consistent with elevations found in other studies of PCB-exposed workers is not correct.[4–6] In addition to the three studies cited by Bove et al, there is the Bertazzi cohort and its update by Bertazzi et al[7] and Tironi et al.[8] The results of the Brown[4] and Sinks et al[5] studies are inconsistent with each other. The Loomis et al[6] study of utility workers not capacitor workers, did report an elevation in melanomas in some subsets of the cohort that were presumed to have had exposure to PCBs while working outdoors. Exposure to sunlight was not adequately accounted for by Loomis et al.[6] Brown and Jones[9] and Brown[4] found an excess of liver and rectal cancers. Neither Sinks et al[5] nor Loomis et al[6] reported such increases. Sinks et al[5] reported a non-significant elevation in brain and nervous system cancers. Neither Brown and Jones,[9] Brown,[4] Bertazzi et al,[7] or Tironi et al[8] found an elevation in brain cancer. These inconsistencies were discussed in our article.

Bove et al state that we only included a latency-period analysis for all cancers and for intestinal cancer. This was done primarily because of space limitations. Cumulative exposure and latency tables were computed and evaluated for many other causes of death, including all of the cancers of interest. The interpretation by Bove et al that the intestinal cancer SMR increases to a significant level for women with ≥20 years of latency ignores the importance of examining the trend associated with latency and length of employment. Furthermore, it might be worth noting that for women employed for 10

years or longer with a latency period ≥20 years, the SMR was 100 The individual category-specific SMRs cannot be interpreted as meaningful without examination of the trend across cumulative exposure categories. Although the intestinal cancer SMR for latency ≥20 years was significantly elevated, there was no significant trend indicating an increase in risk with cumulative exposure or latency, as discussed in our article Furthermore, comparison with the regional population resulted in a much-reduced SMR (SMR = 120; 95% CI, 74 to 186) for intestinal cancer in female hourly workers The regional comparison is more representative because higher rates of intestinal cancer are observed among the white population of the northeastern part of the United States.

Bove et al raise concerns about our exposure assessment Several factors need to be recognized when assessing the propriety of our exposure assessment and our use of length of employment as a surrogate of exposure Workers accumulate PCB body burdens over time, which persist for many years even after their occupational PCB exposure is discontinued. To suggest that PCB body burdens among capacitor workers were comparable to those found in the general population is unjustified and is not supported by previously published data.[10-13] The fact that workers in capacitor plants had significantly higher body burdens than the general population has been demonstrated in other capacitor plants [14] As reported in our article, average serum PCB levels in the general population between 1976 and 1979 were 5 to 7 parts per billion (ppb, μg/L) [14] Geometric mean serum PCB levels in GE workers in 1979 (2 years after PCBs were no longer used) were 277 ppb (μg/L) reported as Aroclor 1242 and 55 ppb (μg/L) reported as Aroclor 1254 In 1983, 5 years after termination of the use of PCBs, geometric mean serum levels were 116 ppb (μg/L) for Aroclor 1242 and 34 ppb (μg/L) for Aroclor 1254 In 1988, the geometric mean serum PCB levels were 90 ppb (μg/L) quantitated as Aroclor 1242 and 32 ppb (μg/L) quantitated as Aroclor 1254 [15] Workers preferentially retained the more persistent congeners so that the gas chromatographic pattern of their body burden gradually approached that observed in the general population, with primary retention of the more highly chlorinated, poorly metabolized congeners [12] The half-lives of the major PCB congeners retained in these workers were as follows for 2,4,4' trichlorobiphenyl, 1.4 years for 2,4,4'5 tetrachlorobiphenyl, 3 2 years, for 2,3',4,4',5 pentachlorobiphenyl, 5.8 years, and for 2,2',4,4',5,5' hexachlorobiphenyl, 12.4 years.[16] Even though different commercial mixtures of PCBs were used in the capacitor plants, the congeneric composition on a qualitative basis is similar [17] Production began in 1946 with the highly chlorinated Aroclor 1254, and small amounts of Aroclor 1254 were used in the plant at least through 1971

The statement that length of employment alone was an inadequate surrogate for exposure and a likely source of exposure misclassification bias leading to an underestimation of the effect and a distortion of the exposure-response relationship is not supported by the toxicokinetics of PCBs, nor is it an accurate representation of the data analyses conducted on our cohort and reported in the article

Bove et al report that the majority of hourly workers never worked in a high-exposure job, when in fact 1268 of the 2984 male hourly employees (42 4%) did work in a high-exposure job Only 13.8% of the female hourly employees worked in a high-exposure job, not an uncommon occurrence in an industrial setting To suggest that the remaining portion of the cohort experienced PCB exposure similar to that of the general population is not an accurate representation of the facts This is presented in the exposure-assessment section of our article

Bove et al state in the opening sentence that although the goal of the study was to evaluate six specific cancers, we focused almost entirely on all-cancers mortality Table 4 in the article presents SMRs and 95% CIs not only for the six cancers of interest but for 32 other causes of death, including 15 additional cancers The issue of statistical power is raised by Bove et al and two tables were provided These tables were not properly referenced nor was the methodology used to generate these calculations explained It is unclear why an SMR of 150 should be considered the "highest expected" for these cancers, when previous publications on smaller cohorts reported statistically significant SMRs well above 150 Our study was an attempt to evaluate these earlier observations in a larger study with a longer follow-up period

Bove et al question the decision to limit the latency by length of employment calculations to cancers with more than two observed cases and a lower boundary of the 95% CI of 90 or above. This decision was made by the investigators to limit the multiple comparison problem and to provide more meaningful data, rather than to obscure data. Additionally, the lack of presentation of data should not be interpreted as the data not having been analyzed. All six a priori cancers of concern were examined carefully; however, publication space is limited and presenting a table of latency by cumulative exposure for liver cancer, for instance, with two deaths was deemed unwarranted

In their summary statement, Bove et al dismiss our study findings because of the HWE effect, failure to account for latency, exposure misclassification, potentially insufficient dosage differences between exposed and comparison groups, and poor statistical power, yet they still insist that we did find excess cancer risk for three of the six a priori cancers of interest and give credence to those findings It is inconceivable to the

investigators of this study how Bove et al given this litany of problems, were able to differentiate the impact and direction of these biases with such certainty and specificity

The authors take exception to the tone of the letter by Frumkin and Orris and find statements such as "conspicuously silent" and "willful effort to avoid a positive finding" inflammatory and suggest that such statements do little to advance the understanding of PCBs and cancer risk.

Most of the issues raised by Frumkin and Orris have been addressed earlier Their suggestion to include more capacitor plants to increase power has merit, however. The General Electric Company had only the two facilities in upstate New York (Hudson Falls and Fort Edward) where capacitors were made using PCBs.

Frumkin and Orris question whether high-exposure jobs actually entailed high exposure and raise concerns about misclassification The exposure misclassification suggested by Frumkin and Orris is highly improbable, given the distinction between jobs with direct dermal and inhalation exposure and those with only inhalation exposure to PCB air levels in the plant, as explained and referenced in our article Additionally, the characterization of this bias as substantial is unwarranted and is an overstatement of the potential effect. Assignment of exposure for specific job categories was done before determination of vital status. At both plants, workers were located in the same building, and the same air-ventilating system served the entire building We verified the physical layout by conducting a walk through the building and by talking to present and former employees. Many workers had different jobs in the different exposure categories (high, undefinable, and low). All workers, including those in low-exposure jobs, had significantly higher exposures than the general population, on the basis of PCB serum levels reported by Lawton et al,[11] Brown et al [5,10] and Brown [18]

The PCB blood levels (from 194 and 290 workers) mentioned by Frumkin and Orris were of limited value in validating an exposure job matrix for 7075 workers Although the job histories and the exposure assignment did confirm that workers in high-exposure jobs had high PCB blood levels, these workers were selected either because of their known high-exposure job[11] or they were self-selected [10] The high-exposure jobs were readily identified by plant personnel and were confirmed by PCB air-level readings and PCB blood levels Misclassification of jobs into the high-exposure category or misclassifying high-exposure jobs as lower-level exposure jobs was extremely unlikely

Frumkin and Orris suggested that PCBs are serious health hazards, irrespective of carcinogenicity with effects that include decreased birth weight, neurodevelopmental effects, and interference with thyroid and estrogen hormone function It has not been shown that PCBs interfere with estrogen-hormone function in humans Studies conducted to examine the effects of PCBs in infants and children have been critically reviewed[19-22] or could not be supported.[20] Results from thyroid function tests performed in infants were within the normal range Furthermore, Kimpman-Esseboom et al[27] stated, "The mean dioxin-like PCB toxic equivalent levels and the mean total PCB and dioxin toxic equivalent levels of the neurological normal infants were significantly higher ($p = 0.04$ for both) compared with the levels of the neurologically (mildly or definitely) abnormal infants There was no relationship between the TT3 (serum total triiodothyronine), TT4 (serum total thyroxine), FT4 (free thyroxine), and TSH (thyroid stimulating hormone) levels in maternal, umbilical, or infant plasma (collected in the second week after birth) and the results of the neonatal neurological examinations We conclude that overt abnormalities found in the neonatal period are not caused by either direct effects of PCB or dioxin exposure or lowered thyroid hormone levels." According to the National Center for Health Statistics,[28] birth weight is affected by education of the mother, mother's age, birth order, interval between births, gender, inadequate prenatal nutrition, alcohol consumption, smoking, lack of prenatal care, incidence of elective induction contraceptive utilization, out-of-wedlock births, metropolitan areas (lower), and race. The body size of the parents and maternal illnesses such as diabetes also play a role. These many variables exemplify the difficulties of appropriately designing studies to examine a single factor affecting birth weight Given these uncertainties and the published criticisms of studies reporting "other health effects of PCBs" it has not been conclusively shown that PCBs cause other "serious" health problems in humans.

We disagree with the final comment by Frumkin and Orris that this study was a great leap sideways on the path to a more complete understanding of the health effects of PCBs. The issue of PCBs and potential health effects has been a significant public health concern for more than 30 years The lack of consistent findings in the previous cohort studies was assumed to have resulted from small cohort sizes and short follow-up periods Given the disparate findings in these smaller capacitor cohorts, the appropriate next step was to assemble a larger cohort of PCB-exposed workers and examine them throughout a longer follow-up period. The fact that we were unable to confirm any of the previously reported findings is important and adds to the knowledge about PCBs and health effects The assumption that a negative study does not provide valuable information imposes significant restrictions on the scientific process and the ability to ade-

quately and objectively assess all data.

Errata: The correct number of female salaried workers with a length of employment of 10 to <15 years in Table 2 is 27; 5.8% is the correct percentage. In Table 6, line 2, last column, total SMR for ≥20 years of latency should be 117. The total number of workers in the upper panel of Table 2 should be 7075

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Investigation of Elevated Urine Beta-2-Microglobulin in a Cohort of Cadmium Workers

To the Editor Prior to the issuance of the 1993 Occupational Safety and Health Administration Cadmium Standard, urine testing for beta-2-microglobulin (β_2m) was not frequently performed Testing for β_2m was an esoteric laboratory test performed only on workers whose cadmium levels had been found to be elevated The Cadmium Standard mandated that all employees exposed to greater than 2 5 $\mu g/m^3$ cadmium dust or fumes be tested at least annually for urine β_2m, as well as for blood cadmium (CdB) and urine cadmium (CdU) At a nickel-cadmium battery manufacturing facility, approximately 1000 employees, some of whom had been exposed to cadmium and some of whom had not, were evaluated for β_2m levels, most for the first time.

Elevated β_2m was defined as a β_2m level higher than 300 $\mu g/g$ creatinine[1]; expectations were that approximately 10% of workers with cadmium levels higher than 10 $\mu g/L$ blood or 10 $\mu g/g$ creatinine would also show an elevated β_2m level [2,3] Because 54 employees had such elevated cadmium levels in 1993, it was expected that approximately five or six would also show elevated β_2m levels. It was not known how many employees with other conditions