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HUDSON RIVER PCBs REASSESSMENT RI/FS
RESPONSIVENESS SUMMARY FOR
PHASE 2 - HUMAN HEALTH RISK ASSESSMENT SCOPE OF WORK

APRIL 1999

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.
and
Gradient Corporation

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

April 27, 1999

To All Interested Parties:

The U.S. Environmental Protection Agency (USEPA) is pleased to release the Responsiveness Summary for the Phase 2 Human Health Risk Assessment Scope of Work (HHRASOW) for the Hudson River PCBs Reassessment Remedial Investigation/Feasibility Study (Reassessment RI/FS).

This Responsiveness Summary contains USEPA's responses to the public comments received on the July 1998 HHRASOW. The HHRASOW presented USEPA's general approach for conducting the Human Health Risk Assessments for the Upper Hudson River and for the Mid-Hudson River. The Upper Hudson River Human Health Risk Assessment will be completed in Summer 1999. The Mid-Hudson River Human Health Risk Assessment will be completed following USEPA's review of the revised Thomann-Farley model developed for the Hudson River Foundation.

If you have any questions regarding this Responsiveness Summary or the Reassessment RI/FS in general, please contact Ann Rychlenski, the Community Relations Coordinator for the site, at (212) 637-3672.

Sincerely yours,

William McCabe

Richard L. Caspe, Director
Emergency and Remedial Response Division

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ACRONYMS

ATSDR	Agency for Toxic Substances and Disease Registry
CDI	Chronic Daily Intake
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CIP	Community Interaction Program
CSF	Carcinogenic Slope Factor
FS	Feasibility Study
GE	General Electric Company
HI	Hazard Index
HHRA	Human Health Risk Assessment
HHRASOW	Human HeaLth Risk Assessment Scope of Work
HROC	Hudson River PCBs Oversight Committee
HQ	Hazard Quotient
IRIS	Integrated Risk Information System
LOAEL	Lowest Observed Adverse Effect Level
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NPL	National Priorities List
NOAA	National Oceanic and Atmospheric Administration
NYSDEC	New York State Department of Environmental Conservation
NYSDOH	New York State Department of Health
OSWER	Office of Solid Waste and Emergency Response
PCB	Polychlorinated Biphenyl
RfC	Inhalation Reference Concentrations
RfD	References Dose
RI	Remedial Investigation
RI/FS	Remedial Investigation/Feasibility Study
ROD	Record of Decision
RM	River Mile
RME	Reasonably Maximally Exposed
RI/FS	Remedial Investigation/Feasibility Study
SARA	Superfund Amendments and Reauthorization Act of 1986
SCEMC	Saratoga County Environmental Management Council
SOW	Scope of Work
STC	Science and Technical Committee
TAGM	Technical and Administrative Guidance Memorandum
TCDD	2,3,7,8-Tetrachlorodibenzo-p-dioxin
TEF	Toxicity Equivalency Factor
TSCA	Toxic Substances Control Act
UCL	Upper Confidence Limit
USDOI	United States Department of Interior
USEPA	United States Environmental Protection Agency
USFWS	US Fish and Wildlife Service

Introduction

HUDSON RIVER PCBs REASSESSMENT RI/FS
RESPONSIVENESS SUMMARY
PHASE 2 - HUMAN HEALTH RISK ASSESSMENT SCOPE OF WORK

APRIL 1999

I. INTRODUCTION AND COMMENT DIRECTORY

1. Introduction

The U.S. Environmental Protection Agency (USEPA) has prepared this Responsiveness Summary to address comments received during the public comment period on the Phase 2 Human Health Risk Assessment Scope of Work (HHRASOW) for the Hudson River PCBs Reassessment Remedial Investigation/Feasibility Study (Reassessment RI/FS), dated July 1998.

For the Hudson River PCBs Reassessment RI/FS, USEPA has established a Community Interaction Program (CIP) to elicit on-going feedback 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.

Because of the large number of CIP participants and associated costs of reproduction, the HHRASOW is incorporated by reference and is not reproduced herein. No revised HHRASOW will be published. The comment responses and revisions noted herein are considered to amend the HHRASOW. For complete coverage, the HHRASOW and this Responsiveness Summary must be used together.

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

The second part, entitled, "Responses to Comments on the Upper Hudson and Mid-Hudson River Human Health Risk Assessment Scope of Work," contains USEPA's responses to all significant comments that are relevant to both risk assessments. Responses are grouped according to the section number of the HHRASOW to which they refer. For example, responses to comments on Section II.2.A. of the HHRASOW are found in Section II.2.A. of the Responsiveness Summary. Additional information about how to locate responses to comments is contained in the Comment Directory.

The third part, entitled, "Responses to Specific Comments on the Mid-Hudson River Human Health Risk Assessment Scope of Work," contains USEPA's responses to all significant comments on the Mid-Hudson that are not addressed in the second part. Responses are grouped according to the section number of the HHRASOW to which they refer. For example, responses to comments on Section III.2.A. of the HHRASOW are found in Section III.2.A. of the

Responsiveness Summary. Additional information about how to locate responses to comments is contained in the Comment Directory.

The fourth part, entitled, "Additional References," contains a listing of references cited in this Responsiveness Summary that are not listed in the HHRASOW.

The fifth part, entitled, "Comments on the HHRASOW," contains copies of the comments submitted to USEPA. The comments are identified by commenter and comment number, as further explained in the Comment Directory.

1.1 Recent Developments

USEPA received the revised Thomann-Farley model on April 27, 1999. USEPA will review the model to determine its appropriateness for use in performing the Mid-Hudson HHRA. In the HHRASOW (p. 1), USEPA noted that the Upper Hudson and Mid-Hudson HHRAs may be developed at different times.

2. Commenting Process

This section documents and explains the commenting process and the organization of comments and responses in this document. Readers interested in finding responses to their comments may skip this section and go directly to the tab labeled "Comment Directory."

2.1 Distribution of HHRASOW

The HHRASOW, issued in July 1998, 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 HHRASOW were also made available for public review in 17 Information Repositories, as shown in Table 2 and on the USEPA Region 2 internet webpage, entitled "Hudson River PCBs Superfund Site Reassessment," at www.epa.gov/hudson.

2.2 Review Period and Public Availability Meetings

Review of and comment on the HHRASOW occurred from July 23, 1998 to August 31, 1998. During this period, USEPA held a Joint Liaison Group meeting at the Marriott Hotel in Albany, New York on July 23, 1998. Subsequently, USEPA sponsored an availability session to answer questions on August 19, 1998 at the Holiday Inn at Latham, New York and on August 20, 1998 at Marist College in Poughkeepsie, New York. These meetings were conducted in accordance with USEPA's "Community Relations in Superfund: Handbook, Interim Version" (1988). 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 HHRASOW, all citizens were urged 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 HHRASOW were received in two ways: letters to USEPA and oral statements made at the Joint Liaison Group meetings on July 23, 1998. USEPA's responses to comments made at the Joint Liaison Group meeting are provided in the meeting minutes.

All significant written comments received on the HHSOW are addressed in this Responsiveness Summary. Comments were received from five commenters. Total comments numbered over 100.

2.4 Distribution of Responsiveness Summary

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

TABLE 1
DISTRIBUTION OF REPORTS

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
- NYSDOH representative
- GE representative
- Liaison Group Chairpeople
- Scientific and Technical Committee representative

SCIENTIFIC AND TECHNICAL COMMITTEE MEMBERS

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

Dr. Daniel Abramowicz
Dr. Donald Aulenbach
Dr. James Bonner, Texas A&M University
Dr. Richard Bopp, Rensselaer Polytechnic Institute
Dr. Brian Bush, New York State Dept. of Health
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
Mr. 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, Jr., The Reilly Group

Dr. John Sanders
Ms. Anne Secord, U.S. Fish and Wildlife Service
Dr. Ronald Sloan, New York State Dept. of Environmental Conservation

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 HHRASOW 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 E. Schumer
- The Hon. John E. Sweeny
- 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

17 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

* ^ United States Military Academy Library
Building 757
West Point, NY 10996

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 HHRASOW Comments and Responsiveness Summary

3.1 Identification of Comments

Each submission commenting on the HHRASOW was assigned "H" for HHRASOW 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 - GE.

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 F1. Each different comment within a submission was assigned its separate subnumber. Thus, if a federal agency submission contained three different comments, the comments would be designated HF1-1, HF1-2, and HF1-3.

Comment letters have been reprinted following the fourth tab of this document. The exception to this is the more than 300 pages of appendices to the comment letter submitted by GE. These appendices are materials that do not contain specific comments on the HHRASOW and have not been reprinted in this Responsiveness Summary because of the volume of paper. GE's comment letter containing 95 pages of comments specific to the HHRASOW is reprinted herein.

The alphanumeric code associated with each reprinted comment letter is marked at the top right corner of the first page of the comment letter and the subnumbers designating individual comments are marked in the margin next to the comment. Comment submissions are reprinted by letter code in the following order: HF, HS, HL, HP and HG.

3.2 Location of Responses to Comments

The Comment Directory, following this text, contains a complete listing of all commenters and comments. This directory allows readers to find responses to comments and provides several items of information.

The first column lists the names of commenters. Comments are grouped first by: HF (Federal), HS (State), HL (Local), HP (Public Interest Group or Individual) or HG (GE).
The second column identifies the alphanumeric comment code (for example, HF1-1) assigned to each comment.
The third column identifies the location of the response by the HHRASOW Section number. For example, comments raised in Section II.2.A of the HHRASOW can be found in the

corresponding Section II.2.A of the Responsiveness Summary, following the third tab of this document.

The fourth, fifth, and sixth columns list key words that describe the subject matter of each comment. Readers will find these key words helpful as a means to identify subjects of interest and related comments.

4. COMMENT DIRECTORY

4.1 Guide to Comment Directory

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

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 HHRASOW section.	Find the responses following the Responses tab. Use the Table of Contents to locate the page of the Responsiveness Summary for the HHRASOW section.
Key to Comment Codes:		
Comment codes are in the format HX1-a H=HHRASOW Y=Commenter group (F=Federal, S=State, L=Local, P=Public Interest Group or Individual, G=GE) a=Submittal number within the commenter group b=Numbered comment		

Example:

COMMENT RESPONSE ASSIGNMENT FOR THE HHRASOW

AGENCY/ Name	Comment CODE	REPORT SECTION	KEY WORDS		
			1	2	3
NOAA /Rosman	HF1-1	1.1	Fish Tissue		

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Comment Directory

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4.2 COMMENT DIRECTORY

AGENCY/ NAME	COMMENT CODE	REPORT SECTION	Key Words		
			1	2	3
NOAA/Rosman	HF1-1	II General	Fish Tissue	PCB Data	Data Sources
NOAA/Rosman	HF1-2	II General	Human Health	Upper Hudson	Lower Hudson
NOAA/Rosman	HF1-3	II.1	Ingestion	Food	PCBs
NOAA/Rosman	HF1-4	II.2.A	Fishing	Fish Advisories	PCBs
NOAA/Rosman	HF1-5	II.2.D	Exposure Duration	Fishing	Mid-Hudson River
NOAA/Rosman	HF1-6	II.2.E	Exposure	Cooking	PCBs
NOAA/Rosman	HF1-7	II.2.E	Exposure Assessment	Cooking	Head/Tail
NOAA/Rosman	HF1-8	II.1	Exposure Assessment	Meat /Vegetables	Floodplains
NOAA/Rosman	HF1-9	II.4	Exposure Duration	Latent Risks	Exposure Levels
NOAA/Rosman	HF1-10	III.2.D	Fishing Limitations	-	-
NOAA/Rosman	HF1-11	III.2.F	Trend	Fish Tissue	PCBs Concentrations
NYSDEC/Ports	HS1-1	II.2.A	Fishing Ban	Fish Ingestion	Health Advisory
NYSDEC/Ports	HS1-2	II.2.D	Exposure Duration	Fish Ingestion	Lifetime Exposure
NYSDEC/Ports	HS1-3	II.3	Reference Dose	Cancer Risk	Non-Cancer Risk
NYSDEC/Ports	HS1-4	II.3	Reference Dose	Inhalation	Ingestion
SCEMC/Balet	HL1-1	II.3	Cancer Slope Factors	Congener Specific	-
SCEMC/Balet	HL1-2	II.2.B	Fishing	Upper Hudson River	Angler Survey
SCEMC/Balet	HL1-3	II.2.E	Cooking Fish	Inhalation	Assumptions
SCEMC/Balet	HL1-4	III.1	Information Repositories	-	-
Scenic Hudson	HP1-1	II.2.B	Subsistence Fishing	Creel Count	Fish Consumption
Scenic Hudson	HP1-2	II.2.B	Fish Consumption	Fisherman Families	Exposure
Scenic Hudson	HP1-3	II.2.B	Exposed Populations	Survey of Families	Exposure Assessment
Scenic Hudson	HP1-4	II.3	Toxicity Values	Reference Dose	PCB Mixtures
Scenic Hudson	HP1-5	II.1	Fish Consumption	Ingestion Pathway	Multiple Exposures
Scenic Hudson	HP1-6	II.2.A	Fish Consumption	Fishing Bans	Recreational Fishing
Scenic Hudson	HP1-7	II.2.B	Angler Surveys	Biased Sampling	Deterministic Methods
Scenic Hudson	HP1-8	II.2.D	Exposed Populations	Exposure Duration	-
Scenic Hudson	HP1-9	II.4	Historical Exposure	Latent PCB Effect	Fish Consumption
GE	HG1-1	II General	Monte Carlo Modeling	Fish Consumption	Risk Analysis
GE	HG1-2	II General	Qualitative	Vague	Proposed Approach
GE	HG1-3	II.2.B	PCB Cancer Risks	Conservative Approach	Extrapolations
GE	HG1-4	II.3	Risk Estimates	Decision Making	-
GE	HG1-5	II General	Federal Dam	Mid-Hudson River	Site Size
GE	HG1-6	II.2.A, II.4	Fish Consumption	Catch and Release	Exposure Limitation
GE	HG1-7	II.2.B	Exposure Assessment	Point Estimates	-
GE	HG1-8	II.4.C	Monte Carlo Modeling	Uncertainty	Variability
GE	HG1-9	II.2.B	Fish Consumption	Exposure Factors	Fish Advisories
GE	HG1-10	II.2.B	Subsistence Anglers	Not Relevant	Hudson River Anglers
GE	HG1-11	II.2.B	Monte Carlo Modeling	Micro-Exposure	Input Parameters
GE	HG1-12	II General,II.2.A	Bioaccumulation	Time Variable	PCB Concentrations
GE	HG1-13	II.2.E	Cooking	PCB Losses	-
GE	HG1-14	II.3	Aroclor Based Toxicity	Toxicity Assessment	-
GE	HG1-15	II.3	Cancer Slope Factors	Extrapolation	-
GE	HG1-16	II.3	Reference Dose	IRIS	Conservative
GE	HG1-17	II.3	Effects Assessment	TEQ	Aroclor Toxicity
GE	HG1-18	II.3	Endocrine Disruption	-	-
GE	HG1-19	II.3	Background	Risk Management	-
GE	HG1-20	II.4	RME	Probabilistic Analysis	Deterministic Analysis
GE	HG1-21	II.4	HHRA	No Action	Remedial Decision
GE	HG1-22	II General	Monte Carlo Modeling	Fish Consumption	Agreement
GE	HG1-23	II.2.A	Qualitative	Vague	Proposed Approach
GE	HG1-24	II.3	Toxicity Studies	Uncertainty	-
GE	HG1-25	II General	Troy Dam	the Site	Lower Hudson River
GE	HG1-26	II General	Data Sources	Additional Data	Usability

AGENCY/ NAME	COMMENT CODE	REPORT SECTION	Key Words		
GE	HG1-27	III.1	Thomann-Farley Model	Peer Review	-
GE	HG1-28	II.4	Hypothetical	Administrative Control	Approach Outline
GE	HG1-29	II.2	Angler Exposure	Fish Consumption	Exposure Limitation
GE	HG1-30	II.2.B	Exposure Assessment	Worst Case	Default Values
GE	HG1-31	II General	SOW	-	-
GE	HG1-32	II.2.A	Fish Concentrations	Fish Consumption	Predatory Fish
GE	HG1-33	II.2.B	Conceptual Model	Model Inputs	Model Structure
GE	HG1-34	II.4.C	Monte Carlo Modeling	Micro-Exposure	Time Varying Models
GE	HG1-35	II.3	Uncertainty Analysis	Uncertainty Sources	-
GE	HG1-36	II.4.C	Monte Carlo Modeling	Input Values	-
GE	HG1-37	II.4.C	Variability	Ingestion Rates	Exposure Limitation
GE	HG1-38	II.4.C	Angler Exposure Model	Uncertainty	Micro-Exposure
GE	HG1-39	III.2.A	Risk Management	Background Exposure	Dosages
GE	HG1-40	II.3	Toxicokinetic Models	Human Body Burdens	Background Exposure
GE	HG1-41	II.2.B	Fish Ingestion Rates	PCB Exposure	Site Specific
GE	HG1-42	II.2.B	Fishing Restriction	Exposure Limitations	Exposure Dosages
GE	HG1-43	II.2.B	Angler Population	Consumption Rates	-
GE	HG1-44	II.2.B	Angler Subpopulation	Fish Consumption	Ingestion Rates
GE	HG1-45	II.2.B	Species Specific	Ingestion Rates	-
GE	HG1-46	II.2.B	Surrogate Fish Species	-	-
GE	HG1-47	II General	Bioaccumulation Models	Average Concentration	RME
GE	HG1-48	II General	Food Chain Model	BSM	Fish Tissue Levels
GE	HG1-49	III.2.A	High End Exposure	Migratory Fish	Mid-Hudson River
GE	HG1-50	II.2.D	Exposure Duration	Receptor Population	-
GE	HG1-51	II.2.E	PCB Losses	Cooking	Fish Advisories
GE	HG1-52	II.2.E	Inhalation	Air Data	PCB Concentrations
GE	HG1-53	II.3	Aroclor Based Toxicity	Toxicity Assessment	TEQ
GE	HG1-54	II.3	Cancer Slope Factors	Dose Response	IRIS
GE	HG1-55	II.3	Epidemiological Studies	Human Exposure	PCB Toxicity
GE	HG1-56	II.3	Epidemiological Studies	PCB Induced Cancer	PCB Exposure
GE	HG1-57	II.3	Epidemiological Studies	PCB Induced Cancer	PCB Exposure
GE	HG1-58	II.3	Epidemiological Studies	PCB Induced Cancer	PCB Exposure
GE	HG1-59	II.3	Non-Negative Effects	PCB Induced Cancer	-
GE	HG1-60	II.3	Adverse Effects	Weight of Evidence	Conservative
GE	HG1-61	II.3	Cancer Slope Factors	Epidemiological Studie	Extrapolations
GE	HG1-62	II.3	Cancer Slope Factors	Cancer Toxicity	Epidemiological Studies
GE	HG1-63	II.3	Non-Cancer Toxicity	Reference Dose	Uncertainty Factors
GE	HG1-64	II.3	Epidemiological Studies	Reference Dose	-
GE	HG1-65	II.3	Aroclor Based Toxicity	Aroclor 1254	Reference Dose
GE	HG1-66	II.3	Epidemiological Studies	Rhesus Monkey	Clinical Relevance
GE	HG1-67	II.3	Epidemiological Studies	Rhesus Monkey	PCB Metabolism
GE	HG1-68	II.3	Epidemiological Studies	Rhesus Monkey	Dioxins/Furans
GE	HG1-69	II.3	Toxicity Studies	Toxicity Assessment	Toxicological Endpoints
GE	HG1-70	II.3	Study Duration	Uncertainty Factors	Sub-Chronic Exposure
GE	HG1-71	II.3	Endocrine Disruption	Endocrine Effects	Qualitative
GE	HG1-72	II.3	Uncertainty	Toxicity Assessment	Toxicological Criteria
GE	HG1-73	II.3	Averaging Time	Non-Carcinogenic	Exposure Duration
GE	HG1-74	II.3	Background	PCB Concentrations	Exposure Assessment
GE	HG1-75	II.3	Fish Advisories	Exposure Duration	Fish Consumption
GE	HG1-76	II.4	Central Tendency	High End Exposure	Risk Analysis
GE	HG1-77	II.4	Deterministic Exposure	Probabilistic Exposure	Risk Analysis
GE	HG1-78	II.4	Risk Reduction	Risk Management	No Action
GE	HG1-79	II.4	Angler Risk	Hot Spot	Risk Interpretation
GE	HG1-80	II.4	Start Date	Exposure Duration	Risk Analysis

Responses

II. RESPONSES TO COMMENTS ON THE UPPER HUDSON RIVER HUMAN HEALTH RISK ASSESSMENT SCOPE OF WORK

Responses to General Comments

Response to HG1-1 and HG1-22

USEPA acknowledges the comments supporting many aspects of the HHRASOW, including the Agency's decision to use Monte Carlo modeling and its determination that the greatest risk of exposure to PCBs in the Hudson River is likely to result from fish consumption. However, USEPA disagrees with the comment that other possible exposure pathways are not significant or have little bearing on potential risk; such statements are premature until the results of the human health risk assessment are known.

Response to HG1-2 and HG1-31

The level of detail in the HHRASOW is appropriate for a scope of work document. The HHRASOW provides an outline of the procedures that will be used by USEPA in developing the baseline Phase 2 Human Health Risk Assessment. USEPA acknowledges that there are decision points in the process of conducting the risk assessment. The decisions made will be supported in the HHRA.

Response to HG1-26

The only data sets not previously described in the HHRASOW contain air data. The air data will update information presented in the Phase 1 risk assessment and address stakeholders' concerns regarding potential inhalation of PCBs. As part of the evaluation of scientific data for this pathway, the following studies are being reviewed as background for understanding volatilization of PCBs:

1. Buckley, E.H. and T.J. Tofflemire, 1983. Uptake of Airborne PCBs by Terrestrial Plants Near the Tailwater of a Dam. Proc. Natl. Conf. on Environ. Eng., ASCE Specialty Conference, July 6-8, pp. 662-669.

2. Nelson, E.D., L.L. McConnell, and J.E. Baker, 1998. Diffusive Exchange of Gaseous Polycyclic Aromatic Hydrocarbons and Polychlorinated Biphenyls Across the Air-Water Interface of the Chesapeake Bay. Environ. Sci. Technol. 32:912-919.

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5. Hornbuckle, K.C., D.R. Achman, and S.J. Eisenreich, 1993. Over-water and Over-land Polychlorinated Biphenyls in Green Bay, Lake Michigan. Environ. Sci. Technol. 27(1):87-98.

Decisions regarding the use of the air data and their use in the HHRA will be supported and described in the appropriate sections of the HHRA.

Responses to Specific Comments on the Upper Hudson River Human Health Risk Assessment Scope of Work Also Applicable to the Mid-Hudson River Human Health Risk Assessment Scope of Work

Response to HF1-1, HG1-12, HG1-47, and HG1-48

Details of the modeling effort are presented in the Phase 2 Report - Further Site Characterization and Analysis Volume 2B - Preliminary Model Calibration Report, Hudson River PCBs Reassessment RI/FS, and will be presented in the Baseline Modeling Report (due May 1999). As described in Section 3.4 of the Phase 2 Report, all modeling work will utilize the extensive database that was created to support the Hudson River PCBs Reassessment RI/FS (Further Site Characterization and Analysis Database Report, 1995). The database contains measurements for sediments, fish and aquatic biota, surface water flow and surface water quality from the USEPA, the New York State Department of Environmental Conservation (NYSDEC) and General Electric. The database includes a total of approximately 750,000 records. Almost 350,000 of these records contain data acquired as part of the USEPA Phase 2 Work Plan and Sampling Plan. The remaining records contain data from a large number of historical and ongoing monitoring efforts in the Hudson River.

Response to HF1-2

USEPA will conduct HHRAs for the Upper and Mid-Hudson River. USEPA will not conduct a HHRA for the portion of the Hudson River site between Poughkeepsie and the Battery in New York City. USEPA's approach for conducting the baseline HHRA is protective of human health because the risk for individuals who consume fish caught closer to the PCB-contaminated sediments in the Upper Hudson River is expected to be higher than the risk to individuals who consume fish caught farther away.

Response to HG1-5 and HG1-25

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 National

Priorities List (codified at 40 CFR Part 300, App. B). In its September 25, 1984 Record of Decision, USEPA defines the site by reference to three figures which, together, depict the site as the entire 200-mile stretch of the River from Hudson Falls to the Battery in New York City, plus the remnant deposits. In addition, during the Reassessment RI/FS, USEPA has consistently defined the site as including the Upper and Lower River (e.g., the Scope of Work for Hudson River Reassessment RI/FS (December 1990) and the Phase 1 Report for the Reassessment RI/FS (August 1991)). The comment regarding USEPA's use of the results of the HHRA (including the Mid-Hudson HHRA) in evaluating remedial alternatives is a risk management issue, and therefore beyond the scope of the HHRA. USEPA decision-makers will consider risk management following completion of the HHRA.

1. Plan, Synopsis & Objectives

Response to HP1-5

USEPA acknowledges this comment supporting evaluation of multiple pathways.

Response to HF1-3 and HF1-8

Routes of exposure not quantified in the Phase 1 HHRA will be reviewed in the Phase 2 HHRA to determine if adequate current data exist to support a quantitative assessment (HHRASOW, p. 13). One such route of exposure, noted in the comment, is potential ingestion of vegetables, meat, eggs, and cow's milk from farms located on the flood plains of the Upper Hudson River. USEPA has determined not to evaluate this route of exposure based on existing data on milk samples and forage crops ingested by cattle, as described below.

For the past 25 years, the New York State Department of Agriculture and Markets has analyzed more than 18,200 samples of cow's milk within the state and has not found any detection of PCBs above the detection limit of 0.6 parts per million (ppm) (lipid normalized) (Rudnick, 1999, personal communication). This detection limit is significantly less than the U.S. FDA limit of 1.5 ppm (lipid normalized). Moreover, in the 1980s, Dr. Buckley from the Boyce Thompson Institute at Cornell University collected data on PCBs in forage crops (corn and hay) grown in an area with PCB-contaminated soil along the Upper Hudson River (Buckley and Tofflemire, 1983). The levels of PCBs on these crops were below the U.S. Department of Agriculture regulatory level of 0.2 mg/kg for forage crops. A back-calculation of the level of PCBs in air required to fall on forage crops to attain the U.S. Department of Agriculture level would be in the range of approximately 40 to 60 nanograms/cubic meter. Based a review of air sampling data collected by GE in 1991 on the Hudson River, air concentrations at these levels are typically much lower. Based on this information, USEPA has determined that collection of additional PCB data from vegetables, meat, eggs and milk is not warranted and that the risk via air deposition and ingestion from foods other than fish are likely to be minimal. As stated in the HHRASOW (pp. 14 and 21), the HHRA will present total risks and hazards added together across pathways.

2. Exposure Assessment

Response to HG1-29

USEPA acknowledges the commenter's agreement with the use of site-specific data and appropriate assumptions to develop estimates of exposure, as well as the specific aspects of USEPA's approach listed by the commenter.

A. Concentration of PCBs in Fish

Response to HG1-23

As described in the HHRASOW (p. 7), the HHRA will use estimated future PCB concentrations in fish based on the analytical data and results presented in the Database Report (USEPA, 1995a), the Preliminary Model Calibration Report (USEPA, 1996a), the Data Evaluation and Interpretation Report (USEPA, 1997d), and the Baseline Modeling Report (due May 1999). Specifically, the HHRA will consider annual fish concentrations for Total PCBs as a function of river location and fish species. Concentrations will be adjusted to reflect standard fillet portions. Any distributional information provided by the modelers (reflecting uncertainty and/or variability) will be incorporated into the Monte Carlo analysis.

Response to HF1-4, HP1-6, and HG1-6

As described in the HHRASOW (p. 1) and consistent with USEPA guidance (USEPA, 1989), the HHRA will present an analysis of the potential adverse health effects (current or future) caused by hazardous substance releases from the site in the absence of any remedial actions or institutional controls. The existing health advisories on fish consumption are institutional controls that control or mitigate the exposure to PCB-contaminated fish from the river. As such, the HHRA will discuss the existence of the health advisories, but will assess the risk posed by the site in the absence of the health advisories.

Response to HS1-1

The clarification is acknowledged and will be incorporated into future reports, as applicable.

Response to HG1-12

USEPA will use fate and transport models to describe the distribution of PCBs in the Hudson River. These modeled concentrations of PCBs in sediment and water will be used as initial concentrations in several bioaccumulation models, including a Gobas-type time-variable mechanistic model. Using the fish concentration output from the Gobas model, USEPA will model human ingestion of PCBs in fish. The HHRA will be peer reviewed (see also, response to HG1-39 and HG1-49).

Response to HG1-32

As discussed in the HHRASOW, exposures and risks for the high-end angler will be calculated using the 95th percentile of the fish ingestion rate distribution (HHRASOW, p. 7), the high end of the distribution for exposure duration (HHRASOW, p. 11), and mean body weights consistent with appropriate toxicity assessment assumptions. USEPA is still assessing the most representative value for cooking losses (HHRASOW, pp. 11 and 12). Consistent with USEPA guidance (USEPA, 1989 and 1992c), for the high end exposure point concentration of PCBs in fish, USEPA will use the mean PCB concentration, weighted by fish species preferences and averaged over time and location. The possibility that individual anglers may preferentially fish in the most contaminated stretches of the river, or select only the most contaminated species, will be considered in the uncertainty analysis.

B. Fish Consumption Rates for the Upper Hudson River

Response to HL1-2

Anglers in the Upper Hudson River are required to have fishing licenses and are subject to the "catch and release" fishing advisory. Consistent with USEPA guidance (USEPA, 1989), the expected fish consumption rates for the Upper Hudson River will be evaluated in the absence of institutional controls. Specifically, fish consumption rates from similar rivers that also require fishing licenses but are not subject to fishing advisories or bans will be evaluated. The HHRASOW (pp. 7 and 8) identified the Connelly surveys (Connelly et al., 1990, 1992, and 1996) as a source of comprehensive information about thousands of anglers within New York State, including anglers from similar rivers with similar types of fish. A survey of anglers within 50 miles of the Upper Hudson River, as suggested by the commenter, would include anglers from water bodies that are not similar to the Upper Hudson River and who may consume different fish species. Therefore, the proposed approach is not appropriate for use in the HHRA.

Response to HP1-1, HP1-7, HG1-10

The distribution of fish consumption rates for the Mid-Hudson and Upper Hudson River will include data from the Connelly surveys (Connelly et al., 1990, 1992, and 1996), the Clearwater surveys (Barclay, 1993) and, to a lesser extent, ChemRisk (1991). The distribution of fish ingestion rates will represent the full range of the Hudson River angler population, from those who may eat as little as one fish meal from the Hudson River per year to very frequent anglers, for whom Hudson fish would account for a significant portion of their protein intake, regardless of whether these anglers fish for recreation or sustenance (HHRASOW, p. 9). Uncertainties associated with underreporting of subsistence anglers will be addressed in the uncertainty section of the HHRA.

Consistent with USEPA guidance, both "high end" and "central tendency" risks and hazards (point estimates) will be evaluated in the HHRA. In addition, the Monte Carlo analysis will consider the full distribution of risk and hazards for Hudson River anglers. The point estimate risk

assessment will provide calculated high end and central tendency risks and hazard estimates, while the Monte Carlo analysis will provide a distribution of risks and hazards for all anglers.

Response to HP1-2

As stated in the HHRASOW (p. 9), "a hypothetical study population will be defined as any individual who would consume self-caught fish from the Hudson River at least once per year in the absence of a fishing ban." Based on the available fish consumption studies, the HHRA will evaluate risks and hazards to a high-end fish consumer based on a range of body weights that do not specifically identify gender. The fish consumption rates will incorporate information on males and females, and adults and children, to the extent that each group is represented in the existing datasets of angler populations.

Another factor that must be considered is the toxicity of the chemical. The toxicity values that will be used in the HHRA are protective of both males and females. For cancer health effects, the cancer slope factors (CSFs) that will be used are based on an increased incidence of liver tumors in female rats, reflecting the potential greater sensitivity of this gender. The CSFs generated based on female rats are higher than those generated for tumors found in male rats. Because risk is a function of exposure and hazard, use of the higher CSF based on data from the female rats is more protective of human health than one based on data from male rats.

For non-cancer health effects, the toxicity value is expressed as a Reference Dose (RfD). The RfDs for PCBs are based on several studies in monkeys where females were exposed through ingestion perinatally and as adults. The studies found reduced birth weights in offspring of the perinatally-exposed monkeys and immune effects in adult female monkeys exposed for longer periods of time. The RfDs that will be used have been adjusted lower to account for several factors, including sensitive human subpopulations, such as children and the elderly. The use of these RfDs in assessing potential non-cancer health effects is protective for the populations identified in the comment.

Response to HP1-3, HG1-3, HG1-7, and HG1-30

The HHRA will evaluate current and potential risks and hazards to the reasonably maximally exposed (RME) individual. The calculated risks and hazards for the RME individual are compared to specific criteria of 10^{-4} to 10^{-6} for cancer health effects and a Hazard Index (HI) of 1 for non-cancer health effects.

Consistent with USEPA's guidance which requires evaluation of the risk to an individual (USEPA, 1989), the HHRA will not estimate the total number of anglers that consume their catch or the total number of women of child-bearing age exposed through consumption of fish. In addition, for the current exposure scenario, it would be difficult to identify the number of anglers who are consuming fish in the presence of fishing bans and health advisories, because of the potential for underreporting and the threat of fines for anglers keeping fish from the Upper Hudson.

For the future scenario, it is not possible to project with any certainty the number of potential anglers within various stretches of the river who would consume fish if bans and or health advisories were lifted.

The comments that USEPA intends to use unrealistically high or worst case exposure assumptions are not accurate. Consistent with USEPA guidance (USEPA, 1989 [e.g., see p. 14]), the HHRA will calculate point estimates for central tendency (average) and high-end (RME) exposures (HHRASOW, pp. 14, 15, 21, and 22).

Response to HG1-9 and HG1-42

The HHRA will describe all fishing bans and restrictions currently in place. For example, the New York State Department of Health (NYSDOH) issues numerous health advisories on eating sportfish from New York State for rivers, lakes and streams. The NYSDEC's fishing regulations guide, which incorporates fishery guidelines set out by the Atlantic Marine Fisheries Commission, outlines general angling regulations, such as daily limits, minimum lengths of fish, and dates of open season. These general regulations are not health based, but presumably are established to prevent depletion of fisheries.

However, fish advisories are not 100% effective in preventing or limiting fish consumption, as described in numerous studies of compliance with fish consumption including Connelly's studies (1990, 1992, and 1996). This assumption is supported by EPA's preliminary analysis of the 1992 Connelly data, which showed no significant difference in the mean number of freshwater fish meals eaten when comparing NY water bodies with full, partial, or no advisories, despite the expectation that the fishing advisories would likely suppress fish ingestion rates to some degree.

Therefore, to be protective of public health, the HHRA will evaluate Hudson River fish ingestion rates in the absence of any Hudson-specific fishing bans. The effect of general, non-specific NYSDEC and NYSDOH fishing regulations that would be in effect regardless of PCB contamination levels in the Hudson River inherently will be taken into account through use of fish ingestion rates from the Connelly et al. (1992) data.

The Maine study (Ebert et al., 1993) cited by the commenter will be considered in the HHRA (HHRASOW, p. 8). However, this study is not the best source for angler consumption rates for the Hudson River. In addition to the differences between New York and Maine anglers due to differences in climate, fish species present, general fishing regulations etc., the Maine data set is further limited in that survey respondents were asked only about total fish consumption from all flowing water bodies, and not from individual water bodies separately. As a result, it was not possible to screen to Maine data for more "Hudson-like" rivers and streams, as was possible with the Connelly data.

Response to HG1-11 and HG1-33

The HHRA will not use the ChemRisk (1995) approach for assessing species-specific consumption, as requested by the commenter. As described in the HHRASOW (pp.7- 9), the HHRA will identify species preferences based primarily on the Clearwater studies (Barclay, 1993) and supplemented by the Connelly data (Connelly et al., 1990, 1992, and 1996). Although the ChemRisk (1995) approach is based on an analysis of fish species preferences from the Connelly data, several of the assumptions are flawed. For example, more than 40% of the fish species identified in the ChemRisk (1995) original distribution were salmon and trout species, which are not present in the Hudson River; these fish species were arbitrarily assigned to be bass. In addition, the authors arbitrarily apportioned the almost 20% of fish that fell into an "other" category evenly among seven species found in the Hudson but not listed in the Connelly survey. Considered together, more than 60% of the ChemRisk (1995) distribution was inconsistent with the data set upon which it was reportedly based.

Furthermore, for the reasons noted in the response to HP1-1, HP1-7, and HG1-1, USEPA will use a probabilistic risk analysis as outlined in the HHRASOW (p. 15) and not conduct the micro-exposure event analysis described by ChemRisk (1995).

Response to HG1-41

USEPA acknowledges the commenter's agreement with its approach regarding fish ingestion rates for recreational anglers.

Response to HG1-43

USEPA has chosen to define the angler population as those individuals who consume self-caught fish from the Hudson at least once per year to reflect the RME individual. While some anglers may consume fish at frequencies less than once per year and some friends or family members of anglers may consume "gift fish" at infrequent intervals, there are no data to quantify the risks or hazards to these individuals. Moreover, USEPA's approach is protective of human health because the exposure to these individuals would be lower than those calculated for the angler population. USEPA will address the risk to these less exposed individuals in the uncertainty section of the HHRA.

The commenter also suggested that Mid-Hudson fish ingestion rates consider short-lived fishing activities, such as fish tournaments or short fish runs. However, there are no data to quantify consumption rates due solely to these events. Nevertheless, to the extent that such fishing events take place in various NY flowing water bodies, they are already accounted for in the Connelly data sets and will be reflected in the fish ingestion rates used in the HHRA.

Response to HG1-44

USEPA acknowledges the commenter's agreement with USEPA's approach not to distinguish subpopulations of highly exposed or lesser exposed anglers. As stated in the HHRASOW (pp. 9 and 18), these subpopulations will be represented in the distributions of risk generated in the Monte Carlo analysis for the Upper Hudson River HHRA and in the uncertainty analysis, Monte Carlo analysis or in species-specific consumption rates for the Mid-Hudson River HHRA. However, USEPA notes that the HHRASOW does not specify that the angler ingesting self-caught fish is a recreational angler; he or she could be a subsistence angler.

Response to HG1-45

The HHRA will identify species preferences based primarily on the Clearwater studies (Barclay, 1993) and supplemented by the Connelly data (Connelly et al. 1990, 1992, and 1996) (HHRASOW, pp. 9-10 and 18). After determining average species preferences, these proportions will be used to weight PCB concentration data for the point estimate analyses. The fact that not all anglers consume the same species in the same proportions will be addressed in the uncertainty analysis by evaluating the potential for anglers to consume only the most contaminated fish species. The HHRA will clearly describe the exposure assumptions used for each population to aid the risk management decision.

Response to HG1-46

As described in the HHRASOW (p.18), the risk assessment intends to characterize species-specific intake rates for anglers fishing in the Mid-Hudson River using an approach similar to that used for the Upper Hudson. There is no intention to combine surrogate fish species preferences for the Mid-Hudson with those of the Upper Hudson. Clearly, there are different fish species present in the Upper and Mid-Hudson River, and therefore the potential for different angler preferences exists.

D. Exposure Duration

Response to HF1-5

The concentrations of PCBs found in fish and sediment of the Upper Hudson are higher than those found in the Mid-Hudson. USEPA will evaluate the risk to anglers in the Upper Hudson and to anglers in the Mid-Hudson. An evaluation of the potential risks to the anglers who consume fish from both the Upper and Mid-Hudson River would fall somewhere between the estimates for the Upper and Mid-Hudson River and can be estimated from the proportion of fish consumed from each location. Therefore, explicit quantification of this third exposure scenario is not necessary.

Response to HS1-2 and HP1-8

The HHRASOW (pp. 10-11 and 18-19) addresses the potential for individuals to move from one county to another and continue to fish sections of the Hudson River. As indicated in the discussion, the HHRA will evaluate the distance anglers are willing to travel to fish. Data includes information from the U. S. Bureau of Census on In- and Out-Migration Special Project Files and the national census. The combination of datasets will enable a full evaluation of the potential exposures of these anglers.

Use of a lifetime exposure (e.g., 70 years), as suggested by the commenters, is inconsistent with the USEPA guidance (USEPA, 1989) and is more representative of a maximum exposure than an RME scenario.

Response to HP1-6

The effect of Hudson River fishing bans and advisories on fish consumption rates will be addressed by using data from angler surveys, such as the Connelly database for New York State (Connelly et al., 1990, 1992, and 1996) to determine fish consumption rates in the absence of site-specific fishing restrictions. As stated in the HHRASOW (p. 11), to the extent possible, the HHRA will attempt to incorporate the likelihood that an angler may voluntarily choose to stop fishing, based on an analysis of the percent of licensed anglers in the general New York State population in each age group. The presence of a fishing ban would not be considered a voluntary decision.

Response to HG1-50

USEPA acknowledges the commenter's agreement with USEPA's decisions to characterize exposure duration rather than use default values and to consider angler mobility between counties (HHRASOW, pp. 10-11 and 18-19).

USEPA will consider the risk to anglers who move from one county adjacent to the river to another county adjacent to the river (HHRASOW, pp. 10 and 18). However, the HHRA will not consider the risk to an angler who moves from one specific reach of the Upper or Mid-Hudson to another. Such an approach would involve too many possible combinations and would not be protective of human health.

USEPA agrees with the comment that cessation of angling should be a factor considered, to the extent possible, in exposure duration. USEPA will use a number of factors to fully evaluate angler population activities over the exposure period (HHRASOW, pp. 11 and 19). The lifespan of the population of anglers at the time of the "start date" will be addressed inherently through a distribution of ages at which anglers stop fishing, whether due to voluntary fishing cessation, as described above, or other reasons.

E. PCB Cooking Losses

Response to HL1-3, HG1-13, HG1-51, and HG1-52

USEPA acknowledges the comment agreeing with USEPA's decision to evaluate the available literature on the losses of PCBs during cooking in the Upper Hudson and Mid-Hudson HHRAs (HHRASOW, pp. 11-12 and 19). As stated in the HHRASOW (pp. 11-12), USEPA's determination whether these losses should be assessed qualitatively or quantitatively will be based on a review the available literature (see response to HF1-6, below). As described in the HHRASOW (p. 12), the preliminary recommendation is to assume no loss from cooking when calculating point estimates, which is protective of human health, and to use a uniform distribution of the range of observed cooking losses described in the published studies to represent cooking losses in the uncertainty analysis (the second phase) of the Monte Carlo analysis.

In addition, inhalation of volatilized PCBs will be evaluated for the Upper Hudson and Mid-Hudson HHRAs using data collected from near the Hudson River, where available (HHRASOW, pp. 13 and 21). All data sets evaluated for the inhalation exposure pathway will be identified in the HHRA. Based on the low vapor pressure of PCBs, the low concentration levels found near the Hudson River, and a comparison of the CSFs for inhalation and oral exposure, volatilization is not expected to be a major exposure route compared to fish ingestion.

Response to HF1-6

The commenter asked if PCBs lost in cooking increase exposure through volatilization. Since issuance of the HHRASOW, USEPA reviewed the scientific literature available on National Library of Medicine's Database MedLine and failed to find any scientific studies that have measured the levels of contaminants volatilized during cooking. In the absence of any scientific studies in this area, it is not possible to quantify the potential risks or hazards from this exposure route. Based on a qualitative assessment of the cooking frequency for fish, the temperatures used in the cooking, the various cooking practices used, and the relative toxicity of inhalation versus ingestion of PCB-contaminated fish, the risks from inhalation while cooking are not considered to be a major exposure route compared to the ingestion of fish.

Response to HF1-7

Although the Toxicological Profile for PCBs (ATSDR, December 1998) indicated that PCBs are found in the organ meats and heads of PCB-contaminated fish, USEPA's review of the available literature did not identify any studies that provide concentrations of PCBs in soup made from contaminated fish heads. In the absence of information on the concentration of PCBs in soup associated with levels found in fish, it is not possible to address this quantitatively. Therefore, USEPA will address the exposure to PCBs from consumption of fish soup qualitatively in the uncertainty section of the HHRA.

3. Toxicity Assessment

Response to HS1-3, HL1-1 and HP1-4

As stated in the HHRASOW (p. 14), in the point estimate portion of the HHRA, USEPA will use the Aroclor-specific RfDs and the Total PCBs CSFs that have been established by USEPA to assess non-cancer and cancer health effects, respectively (USEPA, 1996c, 1998a-c). Currently, there are individual RfDs for Aroclors 1016 and 1254 (but none for 1242 or 1260) and three upper bound and central estimate CSFs for Total PCBs. For samples containing a mixture of PCB congeners, USEPA will evaluate the analytical data and use the CSFs and Aroclor-specific RfDs that are most similar to the sample and most appropriate for the exposure scenario being modeled. USEPA will not use distributions of toxicity values in the probabilistic (Monte Carlo) portion of the HHRA (HHRASOW, pp. 14 and 21).

The tiered approach for partitioning and bioaccumulation mentioned by one of the commenters is specific to congener data only, and therefore is not appropriate for the non-cancer assessment of risk in the HHRA. Instead, USEPA will use its current guidance for evaluating cancer risks associated with PCB exposure (USEPA, 1996c) to identify the most appropriate CSF. This guidance is similar to the tiered approach for congeners, in that the highest applicable CSF is applied for those PCBs bioaccumulated in the environment, including food chain exposures, sediment or soil ingestion, dust or aerosol inhalation, and the potential for early lifetime exposure. For fish consumption, a CSF of 2 $(mg/kg\text{-}day)^{-1}$ will be used. For inhalation of volatilized PCBs, a CSF of 0.4 $(mg/kg\text{-}day)^{-1}$ will be used. Because more than 1/2% of the total PCBs sampled have more than 4 chlorines and these higher chlorinated PCBs may be accumulated in the food chain, the use of the less protective CSF of 0.07 $(mg/kg\text{-}day)^{-1}$ is inappropriate.

Response to HS1-4

USEPA's guidance on development of inhalation reference concentrations (RfCs) does not support the use of oral RfDs to calculate inhalation RfCs (USEPA, 1990). Such route-to-route conversion is inappropriate for PCBs because the partitioning, transformation, and bioaccumulation of PCBs are route-specific and there is no evidence to suggest that they would be the same for inhalation as they are for ingestion.

Response to HG1-14 and HG1-53

USEPA acknowledges the comment supporting its use of Aroclor-based toxicity values presented in the USEPA Integrated Risk Information System (IRIS) (USEPA, 1998a-c). USEPA guidance (USEPA, 1996c) recommends that when congener concentrations are available, the mixture-based approach (i.e., selection of one of the tiers of PCB slope factors based on the type of environmental mixture) can be supplemented by analysis of dioxin Toxicity Equivalency Factors (TEFs) to evaluate dioxin-like toxicity. Consistent with this guidance, the carcinogenicity of dioxin-

like PCBs will be presented as an uncertainty and discussed in the risk characterization section of the HHRA.

Response to HG1-4, HG1-15, HG1-24, HG1-54 through HG1-62

USEPA acknowledges the comments supporting its current CSFs for PCBs (USEPA, 1996c) based on rat studies. USEPA's policies for reviewing human and animal data in evaluating potential cancer health effects associated with exposure to carcinogens are provided in its current and proposed guidelines for cancer assessment (USEPA, 1986, 1996b). These guidelines address approaches to extrapolate animal data to humans. USEPA's evaluation of potential cancer health effects includes evaluation of existing positive and negative epidemiological studies. The epidemiological studies cited by the commenter were considered by USEPA in developing its current CSFs for PCBs (USEPA, 1996c). Moreover, in May 1996, USEPA held a peer review workshop, at which its current CSFs for PCBs were reviewed by independent experts on the carcinogenicity of PCBs (USEPA, 1996d). The expert panel approved USEPA's approach and recommended that CSFs for PCBs be based on the comprehensive rat study of Aroclors 1016, 1242, 1254, and 1260 (as described in USEPA, 1996c). The expert panel did not recommend that the epidemiological studies be used to derive CSFs for PCBs, as proposed by the commenter. The expert panel noted inadequacies in the epidemiological data with regard to limited cohort size, problems in exposure assessments, lack of data on confounding factors, and the fact that occupational exposures may be to different congener mixtures than those found in environmental exposures, as well as other limitations and complications associated with interpreting human data (USEPA, 1996d, p. 9).

The commenter proposed that USEPA use the lowest CSF submitted in comments on the Water Quality Guidance for the Great Lakes System (TERRA, 1993). TERRA's approach combined estimates of PCB intake by capacitor workers with estimates of cancer mortality by Brown (1987) or Taylor (1988). The commenter proposed a CSF calculated from the Taylor (1988) study, stating it is "the largest epidemiological study performed to date and is highly relevant to the Hudson River." A CSF based on this approach is flawed for the following reasons:

1. Although the Taylor (1988) study is large, it is not necessarily better for the Hudson River PCBs site than other studies of PCB exposure. Taylor's cohort included employees who worked in offices and manufacturing areas where PCBs were not used.
2. Although the Taylor (1988) study was conducted in New York State, occupational exposures of employees in capacitor plants are not highly relevant to those of residents living near the Hudson River. TERRA (1993) estimated that occupational exposure was largely by inhalation, while USEPA expects residential exposures for the Hudson River PCBs site to be through ingestion of contaminated fish, soil, or sediment. This is a critical difference, as inhalation involves mostly the more volatile congeners of low chlorine content, while contaminated fish, soil, and sediment contain more persistent and less volatile congeners of high chlorine content. Thus workers and residents are exposed to different fractions of the PCB mixtures.

3. Not only are workers and residents exposed to different fractions of the PCB mixtures, but inhaled PCBs are likely to be less persistent and less carcinogenic than PCB congeners ingested through contaminated fish, soil, or sediment. USEPA's current CSF (USEPA, 1996c) explicitly recognizes the large differences in persistence and toxicity for the different mixture fractions encountered by different exposure pathways.
4. TERRA (1993) used six studies to estimate occupational PCB exposure. Only one, however, involved the same set of PCB mixtures (Aroclor 1254, then 1242, then 1016) used in the plants studied by Brown and Jones (1981), Brown (1987) and Taylor (1988). There is great uncertainty in combining mortality in the plants studied by Brown and Taylor with exposures to other mixtures in other manufacturing plants, some of these from other countries.
5. TERRA (1993) also used a pharmacokinetic approach to estimate occupational PCB exposure. It relied, however, on so-called half-life estimates for different Aroclor mixtures. In presenting its new CSFs, USEPA (1996c) discussed the fallacy of ascribing a single half-life to a mixture as variable as PCBs. Further, these half-life estimates tend to be underestimates; this tends to overstate occupational exposures and understate the CSFs.

Therefore, for the HHRA for the Hudson River PCBs site, USEPA will use its current CSFs, including the current oral CSF of 2 $(mg/kg\text{-}day)^{-1}$ for ingestion and will not use the less protective oral CSF proposed by the commenter.

Response to HG1-16 and HG1-63 through HG1-70

In the HHRASOW (pp. 14 and 21), USEPA stated that it would use the non-cancer RfDs established for PCBs in IRIS (USEPA, 1998a-c), which are the Agency's consensus toxicity values. The commenter objected to the USEPA's oral RfD for Aroclor 1254, asserting that: (1) the clinical relevance of immunotoxicity end points has not been demonstrated, (2) the ocular, dermal, and nailbed changes observed in the rhesus monkey study do not correlate to such changes in humans, (3) there are differences in metabolism of PCBs in monkeys and humans, and (4) the values for uncertainty factors that were applied in extrapolating from the rhesus monkey studies to humans were overly conservative. With respect to each of these points raised by the commenter:

1. The oral RfD for Aroclor 1254 developed by USEPA is appropriate for use in the Hudson River PCBs Site HHRA. USEPA disagrees with the commenter's statement that the clinical relevance of the immunologic changes used as one of the critical adverse effects has not been demonstrated. Today, tests very similar to those employed by Tryphonas et al. (1989, 1991a,b) to determine the levels of serum IgG and IgM (produced by the body's immune system to fight infection and disease) in rhesus monkeys are widely used in hospitals and clinical laboratories to diagnose immune deficiencies in suspected immuno-compromised patients (Bakerman, 1994, ABC's of Interpretive Laboratory Data, 3rd edition, Interpretive Laboratory Data, New York). In addition, the toxicology research community, as evidenced by presentations and audience attendance at immunotoxicology sessions of the annual Society for Toxicology meetings, has expanded its presentation 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). 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.

2. The commenter stated that the rhesus monkey is not an appropriate model for the dermal, ocular, and nailbed effects of PCBs in humans based on a study by Gillis and Price (1996), who reported lower doses for observed dermal, ocular, and nailbed effects in the rhesus monkey than in workers exposed to PCBs. However, the effects of PCBs exposures in rhesus monkeys reported by Gillis and Price (1996) were the result of precise, consistent daily dosing that was monitored and very well characterized, while the effects reported for workers exposed to PCBs were based on sporadic doses, may not have been monitored, and were very poorly characterized. Because of these differences in dosing, monitoring, and reporting, one cannot rely on the Gills and Price (1996) study to conclude that the rhesus monkey is an inappropriate model for the effects of PCBs on humans. The data from the rhesus monkey study result from well-controlled dosing, which is more desirable for a quantitative assessment than exposure that is not well characterized.

3. With regard to metabolism of PCBs in rhesus monkeys and humans, USEPA notes that, while slight differences in metabolic processes have been observed by one research group, differences in the critical adverse effects have not been demonstrated by other research groups.

4. In developing the consensus RfD for Aroclor 1254, which is contained in the IRIS database, USEPA used an uncertainty factor of 300 applied to the Arnold and Tryphonas studies, which includes an uncertainty factor of three out of 10 to account for greater sensitivity in humans than in rhesus monkeys, an uncertainty factor of three out of 10 to account for the longer life of humans when compared to the rhesus monkey and the persistence of PCBs, and an uncertainty factor of 10 out of 10 to account for sensitive humans, such as children and the elderly, and a partial factor to account for the use of a minimal LOAEL (Lowest Observed Adverse Effect Level) because the changes in the periocular (around the eye) tissues and nail beds seen at the 0.05 mg/kg-day dosage are not considered to be of marked severity (USEPA, 1998b). The total uncertainty factor of 300 was developed consistent with USEPA guidelines and current risk assessment practices.

Response to HG1-17 and HG1-71

The commenter stated that a qualitative assessment of potential endocrine effects is unwarranted. The USEPA disagrees with the comment. Estrogenic effects as well as effects upon the thyroid gland and resultant metabolic changes has been observed and documented in many mammalian species, including humans (USEPA, 1997b, c). In addition, the expert panel that peer-reviewed the USEPA's CSFs for PCBs (USEPA, 1996d) recommended that USEPA discuss endocrine effects. Therefore, consistent with the USEPA policies for assessment of endocrine effects (USEPA, 1997b, c), the comprehensive summary of the relevant scientific literature on the PCB toxicity contained in the HHRA will include the endocrine effects of PCBs (HHRASOW, pp. 14 and 21). A very extensive database is available from animal studies regarding the potential endocrine effects that may be attributed to PCBs and the number of these reports increases every

year. In May 1998, the National Institutes of Environmental Health Sciences convened an international panel of experts to discuss the available literature on endocrine effects of PCBs. The results of this meeting will be presented in a future issue of the journal *Environmental Health Perspectives*. The expert panel concluded that the endocrine effects of PCBs are important and require additional research.

Response to HG1-18, HG1-35, and HG1-72

The USEPA agrees with the comment that its current policy for conducting probabilistic risk analysis (USEPA, 1997a) and the associated guiding principles do not apply to dose response evaluations for human health risk assessments. Because this issue is still under evaluation by USEPA, the HHRA will not evaluate the uncertainty associated with toxicological data as proposed by the commenter.

Response to HG1-19, HG1-40, HG1-73, HG1-74, and HG1-75

The commenter proposed using an averaging time based on the half-life of PCBs in humans, to distinguish between background exposure of PCBs in angler body burdens and the increase in body burdens related to the site. The evaluation of internal exposure based on half-life of PCBs in humans was specifically addressed by the expert panel that reviewed USEPA's current CSFs for PCBs. The expert panel did not support adjusting for internal dose at this time because the data are not yet available to determine the appropriate dosimetric for PCB carcinogenicity (USEPA, 1996d, p. 11). Consistent with USEPA's current CSFs for PCBs (USEPA, 1996c), the HHRA will be conservatively protective of human health and no adjustments to exposure duration or averaging time will be made in the HHRA based on consideration of the half-life of PCBs in the body.

4. Risk Characterization

Response to HG1-6 and HG1-28

As described in the HHRASOW (pp. 14 and 22), the risk characterization section of the HHRA will present the current and future risks posed by the site and will explain the assumptions used in the calculation of the risks. These assumptions include no institutional controls such as the "catch and release" program for fishing in the Upper Hudson River. The risk characterization will also address the uncertainties that are inherent in the various components of a risk assessment. However, USEPA notes that NYSDEC's issuance of tickets for violation of the fishing restriction indicates that any risks presented in the HHRA may not be entirely hypothetical, as suggested by the commenter.

Response to HG1-20 and HG1-77

As stated in the HHRASOW (p. 6), the HHRA will contain both point estimate and probabilistic (Monte Carlo) risk analyses. The purpose of providing both the point estimate and the

probabilistic analyses is to provide clarity to the information presented (USEPA, 1992b, 1995b). It also provides a means to compare the results of the two approaches.

Response to HG1-21, HG1-76, HG1-78, and HG1-79

As stated in the HHRASOW (p. 2), the HHRA is a tool to characterize the site contaminants, evaluate the toxicity of the chemicals, assess the potential ways in which an individual may be exposed to the contaminants, and characterize the risks posed by the site (OSWER Directive 9355.0-30, Memorandum from Don Clay to Hazardous Waste Management Directors, entitled "Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions"). The HHRA will calculate both point estimate and probabilistic (Monte Carlo) estimates of risk (HHRASOW, p. 6).

USEPA will prepare an FS that evaluates remedial alternatives, solicit public comment on a Proposed Plan that presents its preferred alternative, and select a remedy based on the nine criteria set forth in the NCP (§300.430(e)(9)). The use of the HHRA by risk managers in the remedy selection process is beyond the scope of the HHRA and therefore is not described in the risk characterization section of the HHRASOW, as proposed by the commenter.

Response to HF1-9, HG1-80, and HP1-9

As noted in the HHRASOW (p. 11), the start date for the exposure of anglers will be 1999. This is appropriate because the HHRA evaluates current and future risk, and 1999 is the year in which the HHRA will be completed. The suggestion that USEPA conduct a separate risk assessment to evaluate the risks that might remain following implementation of various remedial alternatives and, consequently, to change the start date to no earlier than 2002, is inconsistent with the purpose of the HHRA, which is to evaluate current and future risk in the absence of remediation. Similarly, use of a start date before 1999 would not be consistent with USEPA risk assessment policies and guidance (USEPA, 1989).

C. Monte Carlo Analysis

Response to HG1-8, HG1-34, and HG1-38

The Monte Carlo portion of the HHRA will model variability and uncertainty separately, in two stages (HHRASOW, p. 15). Details of the Monte Carlo modeling are not contained in the HHRASOW, which is a general overview of USEPA's approach, but will be presented in the HHRA itself. USEPA has reviewed the commenter's submittals in developing the Monte Carlo modeling approach for the HHRA.

Response to HG1-36

The comment addresses the use of the 90th percentile in Monte Carlo modeling (HHRASOW, p. 15). The >90th percentile mentioned on p. 15 of the HHRASOW is part of the point estimate

calculation of risk and is not part of the Monte Carlo analysis. The 90th percentile mentioned on pp. 15-16 of the HHRASOW is a typographical error. Consistent with Monte Carlo procedures, all exposure parameters for which distributions are available will be randomly selected from the distribution.

Response to HG1-37

The commenter agrees with USEPA that there are no data on actual year-to-year fish ingestion rates for anglers in the published literature (HHRASOW, p. 16). To address this uncertainty, the commenter suggests varying the intake rates within a fixed range of percentiles. USEPA disagrees with this approach because there is no basis for selecting the range and because it is reasonable to assume that an avid angler would remain an avid angler for his or her entire exposure duration. USEPA's approach is protective of human health. For these reasons, the Monte Carlo modeling will assume that each angler's fish ingestion rate remains constant from year to year.

III. RESPONSES TO SPECIFIC COMMENTS ON THE MID-HUDSON RIVER HUMAN HEALTH RISK ASSESSMENT SCOPE OF WORK

Many of the issues raised in the comments are applicable to both the Mid- and Upper Hudson River Risk Assessments. These issues were addressed in Sections II of this Responsiveness Summary. The responses to issues specific to the Mid-Hudson River are presented below.

1. Plan, Synopsis & Objectives

Response to HL1-4 and HG1-27

As stated in the HHRASOW (p. 17), the model for food-chain uptake in the Mid-Hudson River is being developed by Drs. Thomann and Farley for the Hudson River Foundation. These researchers will determine how the model will be peer-reviewed and made available in the public domain. The USEPA's use of the Thomann-Farley model will be presented in the Mid-Hudson River HHRA. The Mid-Hudson HHRA will be made available for public comment and USEPA expects that the Mid-Hudson HHRA will undergo peer review.

4. Exposure Assessment

A. Fish Concentration Data

Response to HG1-39 and HG1-49

The USEPA has consistently defined the site to include the entire Hudson River from Hudson Falls to the Battery in New York City. It is not appropriate to divide the total dose to a Mid-Hudson

River angler ingesting migratory fish into three separate doses (one each for the Upper Hudson, Mid-Hudson, and Lower Hudson), as proposed by the commenter. The risk to an angler eating migratory fish would not be affected by whether the uptake of PCBs into these fish occurred in the Upper Hudson, the Mid-Hudson, or the Lower Hudson River. Further, evaluating the reduction of risk to human health from implementing various remedial alternatives in the Upper Hudson is part of risk management in the remedy selection process, which is beyond the scope of the HHRA.

D. Exposure Duration

Response to HF1-10

The fishing limitations that were in effect in the Mid-Hudson River at the time of the Connelly surveys (HHRASOW, p. 18) are comparable to the fishing advisory currently in effect. The NYSDOH advisories are available at its website at www.health.state.ny.us/nysdoh/fish or by contacting NYSDOH.

F. PCB Concentrations for Deterministic and Monte Carlo Analyses

Response to HF1-11

USEPA agrees with the comment. The intent of the sentence is to convey that if PCB levels are declining with time, these individuals will have lower risk than the original exposed population.

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Federal

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U.S. DEPARTMENT OF COMMERCE
National Oceanic and Atmospheric Administration
National Ocean Service
Office of Ocean Resources Conservation and Assessment
Hazardous Materials Response and Assessment Division
Coastal Resources Coordination Branch
290 Broadway, Rm 1831
New York, New York 10007

August 28, 1998

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

Dear Doug:

Thank you for the opportunity to review the July 1998 Hudson River PCBs Reassessment RI/FS Phase 2 Human Health Risk Assessment Scope of Work. The following comments are submitted by the National Oceanic and Atmospheric Administration (NOAA).

Summary

The July 1998 Hudson River Human Health Risk Assessment (HHRA) Scope of Work (SOW) indicates that a separate assessment will be performed for the Upper Hudson River (from Hudson Falls to the Federal Dam in Troy) and the Mid Hudson River (from Albany to Poughkeepsie). The assessment will evaluate carcinogenic and noncarcinogenic health effects. Total risks and hazards from PCBs will be developed for the average exposed and reasonably maximally exposed individual.

Comments

Pages 1 and 17: Data included in the risk assessment: all relevant fish tissue PCB data should be used, including the most current NYSDEC fish tissue database, data collected by EPA and NOAA as part of the 1993 Ecological Risk Assessment sampling, and fish tissue data collected by NOAA in 1995. It is still not clear from Section I.1. Paragraph 4 what the fish tissue dataset is. The description seems to also ignore fish tissue data collected between the early 1980's and 1990. 1

A HHRA will be performed for the Upper and Mid Hudson River but not for the Lower Hudson between Poughkeepsie and the Battery. Since the Phase I HHRA concluded that ingestion of fish in the Lower Hudson River would produce similar risks to those determined for the Upper Hudson River, it is unclear why the scope of the proposed activities does not include either a quantitative or qualitative assessment for the Poughkeepsie to Battery stretch of the Hudson River. 2

Page 4: Risks from ingestion of vegetables, meat, etc. were not quantified during the Phase I Risk Assessment due to insufficient data. These risks should be evaluated in the proposed HHRA. Numerous farms are located within the floodplain of the Upper Hudson River; hence exposure from vegetables, meat, eggs, and milk (including dairy products) raised on potentially PCB-contaminated soils should be assessed. If there is insufficient data to evaluate this risk, then samples should be collected to aid in the determination. 3

Pages 7 and 18: A decrease in fishing activity or fish consumption due to consumption advisories/bans should not influence an assessment of human health risk. Advisories are issued based on NYS Department of Health recommendations due to contaminant concentrations and exceedances of FDA, EPA or DOH limits. 4

Pages 10 and 18: Exposure duration assumes a mean distance of 34 miles travelled to fish. Neither the Upper or Lower Hudson HHRA account for the possibility that someone living near the Federal Dam could fish both the Upper and Mid Hudson River and represent a third exposure scenario. 5

Page 11: Do potential losses of PCBs in fish during cooking increase exposure to volatilized PCBs? 6

Page 12: Modeled PCB concentrations for fish fillets will be used in the deterministic and Monte Carlo analyses. The models should also take into account the potential for using fish heads/tails in soups. 7

Page 13: Other exposure pathways should be subpopulation consumption of meat, vegetables, eggs, and dairy raised in potentially PCB-contaminated floodplains of the Upper Hudson. 8

Page 15: If the starting point for exposure is 1999 and it is assumed that no exposure occurred prior to that date, then the model could underestimate risk. Baseline should therefore be set at various exposure levels due to potential long-term exposure prior to 1999. 9

Page 18: In paragraph 4, it is stated that fishing limitations were in effect during the Connelly surveys. "Fishing limitations" should have been defined and compared to present conditions. 10

Page 19: In paragraph 3, it is stated that "Since PCB levels in fish seem to be declining with time..." yet on page 11 it is stated that "If PCB levels in fish decline with time...". It appears that EPA has concluded that PCB fish levels are declining with time in the Mid Hudson River but not the Upper Hudson River. The trend in fish tissue PCB concentrations can only be resolved with continued monitoring. The Reassessment RI/FS was prompted in part by the lack of significant declines in fish PCB concentrations that were predicted during the original RI/FS. The sentence on page 19 should be replaced with the one on page 11: "If PCB levels in fish decline with time...". 11

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: Michael Clemetson, DESA/HWSB
Robert Hargrove, DEPP/SPMM
Anne Secord, USFWS
Anton P. Giedt, NOAA
Gina Ferreira, ERRD/SPB
Charles Merckel, USFWS
William Ports, NYSDEC

State

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New York State Department of Environmental Conservation
Division of Environmental Remediation
Bureau of Central Remedial Action, Room 228
50 Wolf Road, Albany, New York 12233-7010
Phone: (518) 457-1741 FAX: (518) 457-7925

John P. Cahill
Commissioner

August 31, 1998

Mr. Douglas Tomchuk
United States Environmental Protection Agency
Region II
290 Broadway - 20th Floor
New York, NY 10007-1866

Dear Mr. Tomchuk:

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

The New York State Department of Environmental Conservation (NYSDEC) and the New York State Department of Health (NYSDOH) have reviewed the July 1998 Hudson River PCBs Reassessment RI/FS reports entitled "Volume 2C-A Low Resolution Sediment Coring Report Addendum to the Data Evaluation and Interpretation Report," and "Phase 2 Human Health Risk Assessment Scope of Work." This letter provides the State's comments on the two documents.

The Low Resolution Sediment Coring Report (LRSCR) presents four major findings. Following are the State's general comments corresponding to each of these findings.

Finding 1

"There was little evidence found of widespread burial of PCB-contaminated sediments by clean sediment in the Thompson Island Pool. Burial was seen at some locations, but more core sites showed loss of PCB inventory than showed PCB gain or burial." [Page ES-3]

State Comment

The State agrees that, based on the data contained in the LRSCR, much of the PCB-contaminated sediments in the Thompson Island Pool are not being buried with significant amounts of clean sediment.

Finding 2

"From 1984 to 1994, there has been a net loss of approximately 40 percent of the PCB inventory from the highly contaminated sediment in the Thompson Island Pool." [Page ES-4]

State Comment

The State agrees that, based on the data contained in the LRSCR, there has been an identifiable PCB inventory loss from the sediments of the Thompson Island Pool. However, based on the data contained in the report, it is difficult to closely quantify the degree of sediment losses. It may be more appropriate for the report to present a range of estimates rather than a single number. This same concern was discussed at the Scientific and Technical Committee meeting on August 18, 1998.

Finding 3

"From 1976-1978 to 1994, between the Thompson Island Dam and the Federal Dam at Troy, there has been a net loss of PCB inventory in *hot spot* sediments sampled in the low resolution coring program." [Page ES-4]

State Comment

The State agrees that, based on the data contained in the LRSCR, there has been an identifiable PCB inventory loss from the hot spots between the Thompson Island Dam and the Federal Dam at Troy.

Finding 4

"The PCB inventory for *Hot Spot 28* calculated from the low resolution coring data is considerably greater than previous estimates. This apparent "gain" in inventory is attributed to significant underestimates in previous studies rather than actual deposition of PCBs in *Hot Spot 28.*" [Page ES-4]

State Comment

The State agrees with this finding based on the data contained in the LRSCR. This inaccuracy in past data gathering efforts may also be present in the PCB inventory estimates in other areas where the core depths were not sufficient in the past. However, NYSDEC believes the USEPA evaluation of sediment PCB inventory gain or loss is valid, and not impacted by the earlier data gathering efforts.

The State also has the following specific comments regarding two other findings of the LRSCR:

1. Page ES-5 and Section 4.1.4 second paragraph — The finding that areas within the Thompson Island Pool (TIP), outside the known hot spot areas of the TIP, have exhibited a large net gain in PCB inventory (up to a 100% increase) is significant because the PCBs are more readily available to fish and other biota.

2. Section 4.4.3 The revised, sediment PCB concentration estimates for the near shore areas are noteworthy. This portion of the river environment has not been well characterized in past investigations, and this information will be useful to both the ecological and human health risk assessments for the site.

The following are the State's comments , including the NYSDOH, on the Phase 2 Human Health Risk Assessment Scope of Work:

1. The first sentence of the first full paragraph of page 9 refers to a hypothetical study population being defined as any individual who would consume self-caught fish from the Hudson River "in 1

the absence of a fishing ban." This passage should be revised for accuracy to read "...in the absence of a fish possession ban and health advisory."

2. The number of years that a person may eat contaminated fish from the Hudson River is estimated in Section II,2.D entitled "Risk Characterization from the Consumption of Fish." Data on how long people live in a county along the river before moving are used to estimate the number of years a person may eat contaminated fish. A significant number of people are likely to move from one county along the river to another county along the river, thus increasing their length of exposure. The number of years that a person may eat contaminated fish from the Hudson River will be underestimated if this possibility is not considered in estimating exposure. Furthermore, a lifetime exposure should be considered in the exposure distribution. 2

3. In evaluating risks, both cancer and non-cancer, the reference dose or cancer potency factor for the Aroclor (e.g. Aroclor 1016, Aroclor 1260, etc.) that is most similar to the PCB mixture in the environmental samples should be used. This approach is more scientifically defensible than automatically using default values as suggested in the Integrated Risk Information System guidance. 3

4. Non-cancer risks are evaluated by comparing exposures to reference doses (ingestion exposure) or reference concentrations (inhalation exposure).Since reference concentrations are not available for the Aroclors, inhalation exposures should be evaluated using reference doses. The risk characterization section of a risk assessment includes a discussion of the uncertainties and limitations of the risk assessment and the uncertainties and limitations, if any, of using reference doses instead of reference concentrations should be included in that section. 4

As additional information becomes available to the parties, the State would welcome the opportunity to provide comments. The State views the completion of the LRSCR and the Risk Assessment Scope of Work as important Hudson River Reassessment milestones, and is pleased that USEPA is adhering to its Reassessment schedule.

Sincerely,

William Davis for

William T. Ports
Remedial Section A
Bureau of Central Remedial Action

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

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Local

SARATOGA COUNTY

ENVIRONMENTAL MANAGEMENT COUNCIL

PETER BALET
CHAIRMAN

GEORGE HODGSON
DIRECTOR

August 28, 1998

Mr. Douglas Tomchuk, Project Manager
Hudson River PCB Reassessment
USEPA, Region 2
290 Broadway, 20th Floor
New York, N.Y. 10007- 1866

Attn: HHRA SOW Comments

Dear Mr. Tomchuk:

Below you will find comments from the Saratoga County Environmental Management Council relative to the Hudson River PCB Reassessment Phase 2, "Human Health Risk Assessment Scope of Work, July 1998:

COMMENTS ON PHASE 2 HUMAN HEALTH RISK ASSESSMENT SCOPE OF WORK, JULY 1998

Prepared for the Saratoga County Environmental Management Council
by D.D. Adams, Member-at-Large

1. P. 3.: Are any data available which indicate a difference in the cancer slope factor (CSF) for lower chlorinated PCBs vs higher chlorinated PCBs? If such data are available, does EPA intend to include different CSF values in the Risk Assessment? The SCEMC believes this difference could be significant and should be considered by EPA. 1

2. P. 8. Rather than rely on a survey of anglers from areas outside the Upper Hudson River Valley, a survey should be made of anglers in the Upper Hudson River Valley only. To circumvent the problem created by the ban on retaining fish caught in the Upper Hudson River, the survey could look primarily at fishing in areas within 50 miles of the Hudson River and consumption of these fish and at catch and release fishing in the Hudson River. It could be assumed that fish caught & released would likely be eaten, if allowed, if fish caught in other areas were eaten. Since the amount of fish consumption is a key part of the Human Health Risk Assessment, it is not acceptable for EPA to say there is no time for doing such a survey or that doing such a survey would delay the final Reassessment decision. It should be paramount in EPA's mind to be sure that the final Reassessment decision is predicated upon the soundest scientific basis possible. The potential 2

50 WEST HIGH STREET BALLSTON SPA, N.Y. 12020 (518) 884-4778

consequences of an unsound final decision are too great for EPA to proceed on any other basis.

3. P. 12&13: In the EPA Availability Meeting held on August 19, 1998, EPA stated that loss of PCBs during cooking as well as inhalation of PCBs are not expected to be significant parameters in EPA's Health Risk Assessment. If these assumptions turn out to be incorrect, more precise data on these parameters should be obtained for the same reasons as stated in Comment 1. (Note: comment on previous Phase 2 reports have questioned the applicability of volatilization data from areas outside the Hudson River Valley) 3

4. P. 17: EPA should provide the public with information on where & when the work on the Lower Hudson River Model is to be reported. Will comments be accepted on this work & will it be subjected to peer review? 4

Thank you for the opportunity to comment.

Sincerely,

Peter M. Balet

Peter M. Balet GH
Chairman

PB/gh
cc: Ms. Carol Browner, Administrator, USEPA
Ms. Jeanne Fox, Regional Administrator, Region 2, USEPA
Mr. Richard Caspe, Director, ERRD, Region 2, USEPA
Mr. William McCabe, Deputy Director, ERRD, Region 2, USEPA
Ms. Ann Rychlenski, Public Affairs Specialist, Region 2, USEPA
The Honorable Gerald Solomon
The Honorable Alphonse D'Amato
The Honorable Daniel Moynihan
The Honorable George Pataki
Mr. John Cahill, Commissioner, NYSDEC
Mr. Stuart Buchanan, Region 5 Director, NYSDEC
The Saratoga County Board of Supervisors
Mr. David Wickerham, Administrator, Saratoga County
Hudson River PCB Liaison Group Chairs
SCEMC members & staff

Public Interest Groups

SCENIC HUDSON INC
9 VASSAR STREET • POUGHKEEPSIE, NY 12601 • (914) 473-4440 • FAX (914) 473-2648

Scenic Hudson's Comments on
US EPA's Scope of Work for
Phase Two Human Health Risk Assessment -
Hudson River PCBs Reassessment RI/FS

Full characterization of the health effects associated with exposure to Hudson River PCBs is a critical part of the EPA's Reassessment of the Hudson River. While we were pleased to see that the health risk assessment will now quantify both carcinogenic and non-carcinogenic health effects from exposure to PCBs for the Upper and Mid-Hudson River, we have the following concerns:

1. **Accounting for Subsistence Fishing** - It appears that exposure due to subsistence fishing will not be adequately sampled and accounted for in the analysis proposed. Although subsistence fishing is briefly mentioned on page 9, it is not clear that the creel count and license approach planned will give adequate representation of exposure for subsistence anglers and their families. The existing literature on fishing and fish consumption on the Hudson indicates strongly that subsistence fishing is common. Therefore it is important that this segment of the population be more carefully accounted for. 1

2. **Risk to Family Members** - As proposed, the health risk assessment does not address the issue of risk for family members of anglers. Clearwater's angler survey indicated that 58 % of fishermen questioned gave fish to their families for consumption. Risk to these family members - women and children - must be thoroughly addressed. 2

3. **Potentially Exposed Populations** - Somewhere in the assessment, either in the exposure assessment or risk characterization, a characterization of the population or populations exposed to PCB contamination should be included. For example, EPA should estimate the number of anglers that consume their catch. The assessment should acknowledge that the population exposed is larger than the population of anglers because many anglers share their catch. Further, because some PCB health effects are associated with pre-natal exposure, the assessment should estimate the number of women of child-bearing age that consume fish. The characterization of this potentially affected population would contribute to the risk characterization and qualitative discussion of endocrine disruption. In other words, it would be desirable to characterize the size of the total potentially affected population. 3

Although the study area extends only as far south as Poughkeepsie, the Superfund site extends to New York City. A sizeable segment of the potentially affected population is outside the study

email: scenichu@mhv.net

area for the risk assessment. Although individual risks may be greatest for anglers within the study area, EPA can and should acknowledge that the population exposed to PCB contamination from the site is very large. Unlike most Superfund sites, this site has a very large exposed population and the "maximally exposed individual" should not be the sole measure of exposure or determination of acceptable risk.

4. **Toxicity Assessment** - The risk assessment proposes to use toxicity values of PCB arochlors 1016 and/or 1254 because the 99reference doses and cancer slope factors have been established for these two arochlor compounds. It is unclear which value will be used for comparison with PCB mixtures as they occur in the Hudson River or in Hudson River fish. Choosing either 1016 or 1254 may not adequately capture the toxicological profile of 1242, the arochlor discharged in greatest volume to the Hudson and present in Hudson River fish. 4

In a recent article in Environmental Health Perspectives, (Volume 106, Number 6, June, 1998, article attached) entitled "Assessing the Cancer Risk From Environmental PCBs" by James Cogliano (Volume 6, Number 6, June, 1998, article attached), it is indicated that EPA in fact has a method for PCB risk assessment that would address this problem (page 321). According to the article, EPA is using a tiered approach that considers how partitioning and bioaccumulation affect each exposure pathway. Using this approach, the high risk slope factor is applied in situations where: environmental processes tend to increase risk; there is food chain exposure; there is sediment or soil ingestion; there is dust or aerosol inhalation; there is exposure to dioxin like, tumor-promoting, or persistent congeners, and there is early life exposure (all pathways and mixtures). Under this approach, the Hudson River would fall into the "high risk and persistent" category, the highest risk tier. We question why EPA is instead planning to use comparative toxicity values instead of it's own current procedure that would require use of the highest risk tier and attendant slope factors.

5. **Other Exposure Scenarios** - The scope (page 7) states that an objective of the Phase II risk modeling is to confirm the Phase I conclusion that fish ingestion "outweighs" other exposure scenarios. Fish ingestion is likely to dominate risks from other exposure pathways. However, it is important to acknowledge that exposed individuals are likely to be exposed to multiple pathways. We support the proposal (section II(2)(H)(1)) to present total risks and hazards added together across pathways. Pathways with risks above 10-6 should not be excluded because they are small relative to fish ingestion. 5

Recent studies from the Chicago area document an "urban plume" of PCB contamination. Based on these findings and other evidence that PCBs are transported by air deposition, EPA should reevaluate the feasibility of estimating food chain pathway risks via air deposition, and milk and beef ingestion. If data to evaluate this pathway are unavailable, EPA should at least include a discussion of the relevant literature related to this exposure pathway. 300851

6. **Fishing Bans** - The impact of fishing bans on fish consumption rates (discussed on page 7, section 2B) should not be included for baseline risk results. Fishing bans could be factored in as part of the "institutional controls" in a remedial scenario. It is not clear that this is EPA's 6

proposed approach. People who voluntarily stop fishing (discussed on page 11) because of the fishing advisories should not be included in the baseline risk assessment. There are many average recreational anglers that are not aware of the PCB contamination, its potential risks, or the health advisories. The assessment should address these anglers and more highly exposed subpopulations.

7. **Use of Angler Surveys** - The Clearwater angler survey and the Chemrisk survey from Maine should be evaluated based on availability and relevance. The Maine survey is less relevant to the assessment than the Clearwater angler survey, which addresses Hudson River anglers. On page 9 in the discussion on angler surveying it should be noted that interviewers may avoid areas where subsistence fishing may be the greatest due to socio-economic conditions. Also, subsistence anglers may be under-represented in surveys because of language barriers. Mail-in surveys under-represent illiterate anglers. In the same discussion it states that "attempts will not be made to distinguish between subpopulations of highly exposed or lesser exposed...". Does this refer only to the Monte Carlo approach? Highly exposed anglers should definitely be distinguished for the deterministic risk model. 7

8. **Exposure duration** - Exposure duration will be estimated assuming that anglers fish from the Hudson only as long as they live in a county along the Hudson. To estimate the time of exposure, EPA will analyze "information such as the length of time people live in a single residence". This may underestimate exposure duration because some anglers will move to a new residence within the same county or to a residence in another county along the river. For the deterministic model, high end exposure duration should be based on an angler that lives and fishes along the Hudson all of his/her lifetime. This is not an overly-conservative scenario. Later (page 12), the scope says that a high-end estimate of exposure duration will be used for the deterministic model. What will that estimate be? Will it be lifetime or some high-end estimate based on county-level mobility data? The simple and more conservative lifetime assumption is more defensible that a complicated and highly uncertain estimate based on mobility data. 8

9. **Start Date** - The EPA proposes that the risk model will address 1999 onward. It would be more appropriate to select an earlier start date. Most of the population of anglers in 1999 has been fishing from the Hudson in earlier years. As proposed, will pre-1999 exposures be ignored? Some of the 1999 anglers were probably fishing in the 1970s. Fish contamination data are available in great abundance from at least the early 1980s and should be used. Also, the EPA reports so far to have focussed on a modeling period in the early 1990s. Why wouldn't the risk assessment include this period also for consistency and comparison of results? 9

10/6/98

SIDLEY & AUSTIN
A PARTNERSHIP INCLUDING PROFESSIONAL CORPORATIONS

1722 EYE STREET, N.W.
WASHINGTON, D.C. 20006
TELEPHONE 202 736 8000
FACSIMILE 202 736 8711

FOUNDED 1866

CHICAGO
DALLAS
LOS ANGELES

NEW YORK
LONDON
SINGAPORE
TOKYO

WRITER'S DIRECT NUMBER
(202) 736-8161

August 31, 1998

Douglas Tomchuk
U SEPA - Region 2
290 Broadway
20th Floor
New York, NY 10007-1866

Re: HHRA SOW Comments

Dear Mr. Tomchuk:

Enclosed please find General Electric Company's ("GE") comments on the "Hudson River PCBs Reassessment RI/FS Phase 2 Human Health Risk Assessment Scope of Work." These comments provide a detailed critique of the Scope of Work, and I will not repeat that discussion here. One specific issue, however, deserves emphasis.

One of GE's primary concerns is the Scope of Work's vague, muddled, and at times inconsistent description of the Agency's proposed baseline human health risk assessment. This has increased the difficulty of assessing and commenting on the Scope of Work. Consequently, in addition to pointing out the portions of the Scope of Work with which GE agrees or disagrees, GE's comments provide specific recommendations on how the Agency should conduct the baseline risk assessment. GE also urges the Agency to reissue the Scope of Work to provide a more coherent description of the risk assessment and to respond to the issues raised in GE's comments.

GE welcomes the opportunity to discuss its comments with EPA in greater detail.

Sincerely,

Thomas G. Echikson

cc: Richard Caspe
William McCabe
Melvin Hauptman
Douglas Fischer, Esq.
Marianne Olson
John Cahill
Frank Bifera, Esq.
Walter Demick

COMMENTS OF GENERAL ELECTRIC COMPANY
ON

Hudson River Reassessment RI/FS
Phase 2 Human Health Risk Assessment
Scope of Work
July 1998

August 31, 1998

Melvin B. Schweiger
John G. Haggard
General Electric Company
Corporate Environmental Programs
1 Computer Drive South
Albany, NY 12205

Russell Keenan, PhD
Paul Price
Ogden Environment and Energy Services
15 Franklin Street
Portland, ME 04101

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FIGURE

Figure 1. Reprint of Figure 9-12 from the EPA Preliminary Model Calibration Report

EXECUTIVE SUMMARY

EPA's Scope of Work for the Human Health Risk Assessment ("SOW") for the Hudson River PCBs Superfund Site ("Site") sets out the Agency's proposed approach for conducting and preparing the baseline human health risk assessment ("HHRA") for the Site. The SOW proposes two risk assessments – one for the Upper Hudson and one for the Mid Hudson – using deterministic and probabilistic methods and standard, IRIS-derived toxicity values. This information will then be used to present a hypothetical statement of the risks associated with the Site against which the risk reduction achieved by various remedial alternatives can be measured.

There is much in the SOW that General Electric Company ("GE") supports. For instance, GE agrees with the Agency's proposal to use Monte Carlo modeling to develop a probabilistic risk analysis capturing both variability and uncertainty associated with the risk estimate. GE also agrees with the Agency's assessment that the primary route of exposure to PCBs is through fish consumption and that other possible exposure routes have little bearing on potential risks. 1

Despite GE's agreement with much of the general approach described in the SOW, GE has a number of concerns about the Agency's proposal. Many portions of the SOW are vague, internally inconsistent and simply fail to provide enough information to permit one to ascertain the Agency's proposed approach. From the information available, it appears that the Agency intends, in many instances, to use unrealistically high exposure assumptions. Further, the Agency apparently intends to ignore the vast body of epidemiological data that do not show that PCBs cause cancer in humans, instead relying entirely on the uncertain extrapolations derived from laboratory studies of animals. GE's comments point out these and other problems, along with recommendations on how the Agency should conduct the HHRA, convey the risk estimates to the public, and use the results in its remedial decision-making. 2 3 4

Beyond the specific concerns summarized below, there are some general points worth emphasizing. First, because the Site does not extend below the Federal Dam at Troy and in light of the numerous sources of PCBs to the lower River and lower River fish, EPA cannot reasonably rely on the results of the Mid Hudson risk assessment to justify remedial actions in the Upper Hudson. Second, the Agency must emphasize that its risk estimates are hypothetical. The existing catch-and-release requirements in the Upper Hudson, and the consumption advisories in the lower River all significantly limit fish consumption now and into the foreseeable future. If the Agency does not take these facts into account in the baseline HHRA, it must recognize and clearly state that its estimates of risk are completely hypothetical. 5 6

1. Exposure issues

- The SOW's method for selecting point estimates for incorporation into the exposure models is vaguely described and implies that the Agency intends to use unrealistic, worst case exposure assumptions. EPA must use more realistic, site-specific and relevant assumptions. 7

- The SOW's explanation of the Monte Carlo modeling does not clearly explain whether and how the Agency intends to model uncertainty and variability separately. Doing so is necessary to understand the estimated range of exposures as well as the uncertainty in those estimates. EPA should adopt and incorporate the approach set out in GE's previous submissions to the Agency. 8

- At a minimum, the baseline fish consumption rates must account for consumption limitations posed by factors other than PCBs, including the statewide consumption advisories and the conservation-based fishing restrictions imposed by New York State and the Atlantic Marine States Fisheries Commission. Furthermore, given the lack of good consumption rate data from the Hudson or other comparable New York waters, the best source for recreational angler consumption rates is the assessment of such rates in Maine contained in Ebert, et al. (1993). 9

- EPA should not consider hypothetical subpopulations of "subsistence" anglers. The available information demonstrates that income level, ethnic background, and commercial and recreational angler status are not relevant for deriving a subpopulation of highly-exposed Hudson River anglers. 10

- The SOW's proposed approach for assessing species-specific consumption should be replaced with the approach set out in ChemRisk (1995), which provides the appropriate input parameters for the microexposure Monte Carlo analysis. 11

- EPA should use a mechanistic, time-variable bioaccumulation model to compute average future PCB concentrations in fish. Variability in fish PCB levels should be estimated directly from the NYSDEC database. In addition, for the Mid Hudson, EPA must account for sources of PCBs other than the Upper Hudson. 12

- GE agrees with the proposal to account for cooking loss of PCBs, which, contrary to the implication of the SOW, is well-supported in the peer-reviewed literature. 13

2. Toxicity issues

- GE supports the proposal to use Aroclor-based toxicity criteria in light of the more reliable and complete toxicological, epidemiological and analytical databases for Aroclors. Other methods, including congener-based analysis, have critical scientific rely on inconsistencies and inappropriate assumptions. 14

- The Agency's reassessment of the cancer slope response of PCBs, which is based entirely on rat feeding studies is an important advance. In light of the difficulties and uncertainties associated with extrapolation from rats to humans, GE believes that it is feasible and more appropriate to use a cancer slope factor which takes into account the vast epidemiological database which does not support the proposition that PCBs cause 15

cancer in humans. GE 's comments present a method for deriving a human-based cancer slope factor that should be used in the HHRA.

- The IRIS-derived reference dose ("RfD") cited in the SOW is also flawed and overly conservative. It is based on a single study of rhesus monkeys and incorporates numerous uncertainty factors when extrapolating to humans. GE's comments present a more defensible and realistic RfD that EPA should use in the HHRA. 16

- EPA should not proceed with the SOW's proposal to present a qualitative assessment of endocrine disruption in light of the lack of evidence that PCBs have any effects on the human endocrine system. 17

- EPA should incorporate an analysis of the uncertainty associated with the PCB toxicological criteria incorporated into the risk assessment, just as it intends to do with the exposure assumptions. 18

3. **Risk characterization issues**

- Because one goal of the risk management is to determine how exposures for a particular source relate to background, the HHRA must recognize the background levels of PCBs in all individuals. 19

- The assessment of risks to the "reasonably maximally exposed individual" and the average individual should be based only on the findings of the probabilistic analysis, which is much more powerful than the proposed deterministic analysis. EPA should abandon the proposed deterministic analysis, which would lead to overly conservative risk estimates. 20

- The baseline HHRA cannot and should not be used to select a remedial decision. The results of the baseline HHRA can only be used to assess the hypothetical risks associated with the "no-action" alternative and, in the context of remedial decision- 21

making, must be measured against the risks estimated to result after implementation of different remedial options.

SECTION I
INTRODUCTION

General Electric Company ("GE") is pleased to submit these comments on the July 1998 "Hudson River PCBs Reassessment RI/FS Phase 2 Human Health Risk Assessment Scope of Work" ("SOW"). GE supports many aspects of the SOW. For example, GE generally supports the use of Monte Carlo modeling to assess exposure in the Human Health Risk Assessment ("HHRA"). GE also agrees with the Agency's conclusion that the greatest risk of exposure to PCBs in the Hudson River is likely to result from fish consumption and that other exposure routes are not significant. 22

Nevertheless, the SOW is inadequate in several respects. The SOW fails to explain in sufficient detail the methodology EPA intends to use to complete many of the identified tasks. The SOW also contains significant gaps and inconsistencies, as well as confusing statements and terminology. The SOW provides only a vague description of the relationship between the exposure assessment and the Agency's effort to model future levels of PCBs in fish and the Agency's intended use of Monte Carlo modeling. The SOW proposes to rely entirely on toxicity estimates based on animal studies, ignoring the extensive data from epidemiological studies that do not show that PCBs cause cancer in humans. GE's comments focus on these problems and provide recommendations on how EPA should complete the human health risk assessment ("HHRA") for the Site. 23 24

SECTION II
GENERAL COMMENTS

Several broad issues raised by the SOW deserve comment.

A. The site does not extend below the Troy Dam.

First, as GE has previously raised with EPA,[1] the Company disagrees with the Agency's description of the Site as including all 200 miles of the Hudson River between Hudson Falls and the Battery. The documents in the administrative record for the addition of the Site to the CERCLA National Priorities List explicitly limit the reach of the Site to the area above the Federal Dam at Troy, and EPA's post-rulemaking comments to the contrary cannot change this fact. GE's disagreement with EPA on the scope of the Site is particularly important in the context of the HHRA, in light of EPA's proposal to conduct a separate analysis of human health risks from PCBs in the fresh water portion of the lower River, a portion of the River that is not properly considered part of the Hudson River PCBs Superfund Site. From previous correspondence and statements, GE understands that EPA is limiting its analysis to potential remedial actions in the Upper River. Assessing human health risks in the Lower River suggests that the Agency may be attempting to justify a remedial action on the basis of benefits to the Lower River. 25

Justifying any remedial action in the Upper Hudson River on the basis of benefits to the Lower River would have serious consequences to the scope of EPA's present reassessment. In such circumstances, EPA would be obligated to investigate and evaluate remedial alternatives, such as source control in the lower River; consider the greatly increased number of sources of PCBs (and other contaminants) to fish in the Lower Hudson; and identify the much wider group of parties who rightfully should be classified as PRPs. The presence of other dischargers of PCBs in the Lower River is well known to

[1] See Nov. 6, 1997, letter from Angus Macbeth to Richard Caspe; May 5, 1998, letter from Angus Macbeth to Douglas Fischer.

EPA; the Agency has conducted recent studies of PCB discharges into New York Harbor, including sampling outfalls, and of comparative contributions of PCBs into the Harbor. The Agency 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."

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 or two PRPs outside that 150 miles as the sole subjects for remedial consideration. 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.

B. The Agency must provide advanced notice of its intent to use additional data.

The Agency points out in the introduction of each section of the SOW (SOW, at 1, 6, 7) that individual components of the proposed approach may be revised if additional data are identified in the course of preparing the risk assessment. GE agrees that appropriate additional data should be included if they would lead to a more accurate baseline risk assessment. We note, however, that this statement appears to be inconsistent with the Agency's claim that it already possesses all the necessary data to complete the reassessment and, indeed, that no new data will be considered. We trust that the Agency will not ignore new and relevant data in its remedial decision making. Regardless, the Agency should notify the public of any new data it intends to use, indicate how it proposes to incorporate the data in the risk assessment, and, when appropriate, make such data available for review and comment by the public. By releasing this information prior to its use, the Agency will comply with its mandate to include the public in its decision-making process. 26

In that light, the SOW states that the risk assessment for the Mid River will be based on the ongoing modeling by Drs. Thomann and Farley (SOW, at 17). This work is currently not available for external review and comment. If the Agency intends to use the Thomann/Farley model, it must provide the public an opportunity to review and comment on it. The Thomann/Farley model should also be subject to external peer review consistent with the peer review of EPA's own modeling effort for the Upper Hudson. 27

C. The Agency must emphasize that the risk estimates presented in the HHRA are hypothetical.

By EPA policy, baseline risk assessments of Superfund sites generally do not consider the effects of administrative and other types of existing controls on exposure (EPA, 1989). To the extent that the assessments provide a starting point for the decision of what remedies (including administrative controls) are necessary at a site, this approach is understandable. However, baseline risk assessments also communicate to the public an understanding of the fundamental nature of the risks that currently exist at Superfund sites. 28

In the case of the Upper Hudson River, the risks that would occur without fishing restrictions are different from the risks that actually exist today and into the foreseeable future. NYSDEC has established and enforces a ban on keeping of fish in the Upper Hudson.[2] This ban is well publicized and enforced by a conservation officer who patrols the Upper Hudson. This officer interviews each angler he meets, including anglers fishing from shore and from boats. Over a recent three-year period (August 31, 1995 through July 31, 1998), the conservation officer on the Hudson River has checked 1,437 anglers and issued only nine tickets and three warnings for keeping fish (NYSDEC, 1998, attached). This finding confirms that the current ban on keeping fish is extremely effective in controlling exposure to PCBs.

[2] As discussed in Section 3.3 below, consumption of fish from the entire Hudson River is also subject to a general restriction in consumption that is independent of the PCBs in the fish. In addition, stiff, conservation-based restrictions on fishing are in place on the lower River. The advisory and fishing restrictions affect fish consumption by truncating the distribution of consumption rates. Since the advisory and restriction are not functions of PCB contamination, they must be taken into account when assessing fish consumption in the baseline risk assessment.

As to the Lower Hudson, NYSDEC and the Atlantic States Marine Fishery Commission have imposed conservation-based restrictions on keeping fish of many species. These restrictions are imposed to assist in the maintenance of fish stocks, not for reducing exposures to PCBs. Nevertheless, they have the effect of reducing such exposure dramatically. Under the baseline assessment described in the SOW, estimates of fish consumption will be developed under the assumption that there is no ban or restriction on keeping and consuming fish. The findings of an assessment that assumes that all anglers are free to keep and consume as many fish as they desire would not be a fair description of the actual risks facing anglers using the Hudson River. Therefore, the HHRA should clearly state that (1) the fishing restrictions effectively eliminate current and future exposures, thereby eliminating the risks (if any) from the consumption of fish; and (2) the risk estimates produced in the Phase 2 assessment are hypothetical risks that would occur in the absence of the current restrictions on keeping and consuming fish.

SECTION III
EXPOSURE ASSESSMENT

For the exposure assessment, the SOW proposes to develop an exposure scenario using a variety of inputs based both on site-specific data and default assumptions. These will then be used to develop both deterministic and probabilistic estimates of exposure.

GE concurs with many aspects of this approach. For instance, the SOW's proposal for the following aspects of the exposure scenerio are generally sound:

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- The definition of the exposed populat on as those anglers who begin fishing at a specific "start date" ;
- The assumption that an angler fishes from multiple locations along the Upper Hudson River (or Mid Hudson) ;
- The recognition that PCB levels in fish should vary as a function of date, location and species of fish;
- The assumption that an angler catches and consumes a number of different species of fish;
- The consideration of cooking loss;
- The determination of duration of exposure based on site-specific data; and
- Basing fish consumption rates on site-specific information.

The SOW's failure to include details on how the Agency intends to implement these principles is troubling and suggests that the Agency may intend to default to "worst case" exposure assumptions (for example, SOW, at 13). 30

The SOW's failure to explain the proposed approach for the HHRA adequately also affects other important aspects of the exposure assessment. In particular, the SOW's description of the proposed Monte Carlo modeling is muddled and confusing. We point out these problems below and make recommendations about how the probabilistic modeling should be implemented. We also present recommendations concerning other 31

aspects of the proposed exposure assessment, including estimation of fish consumption rates, future PCB concentrations in fish, duration of exposure and cooking loss.

A. The proposed approach for determining fish tissue concentrations for the high-end angler is flawed.

The SOW's proposal for selecting fish tissue concentrations for the high-end angler is invalid. As indicated in EPA's Phase 1 Report, fish tissue concentrations increase with the trophic level of the fish. As a result, the fish with the highest levels of PCBs tend to be predator fish, such as the northern pike. These fish species make up a relatively small fraction of the edible fish in the Upper Hudson. It is highly implausible that an angler could catch a sufficient number of these fish to support high levels of fish consumption. It is far more likely that the anglers with high fish consumption rates will consume the more readily available species and will consume multiple species. Thus, the high level of intake will be associated with the average fish concentrations of the more available species. 32

The HHRA should follow the Guidelines for Exposure Assessment's approach for selecting inputs for the determination of the high-end exposed individual (EPA, 1992). This guidance recommends that values be selected for one or two inputs based on the 95th percentile and that the remaining values be assigned values for typical individuals. Use of extended worst case or even reasonable worst case values for all inputs of a risk assessment results in implausible results (McKone and Bogen, 1991).

Therefore, GE recommends that the inputs to the high-end angler be revised as follows:

- The values for fish intake and duration should be set at the 95th percentile values.
- The median value of body weight (age adjusted) should be used.
- Cooking loss should be based on the most likely estimate of reduction.

- The value for fish concentration should be based on a weighted-average of the best estimates (not UCL) of the mean concentrations of PCBs in the most available species.

B. Monte Carlo Modeling

The SOW provides limited information on EPA's proposed use of Monte Carlo modeling of exposure. GE supports the Agency's proposal to use Monte Carlo modeling, but the SOW's failure to present a thorough and transparent description of the modeling that the Agency intends to use inhibits a detailed critique. Consequently, we present GE's recommendations on how the Agency should proceed.

B.1 The SOW does not provide an adequate description of the model.

The portion of the SOW that discusses the modeling is limited to a few scattered paragraphs and sentences. These fragments contain inconsistent and confusing terminology. Moreover, the limited information provided in the SOW concerning the proposed Monte Carlo modeling suggests that a number of fundamental decisions on the model structure and the inputs to the model have been made, but the details of these decisions have been not been disclosed.

The lack of coherent description is surprising in light of the extensive discussions between EPA and GE concerning this topic and the information that GE has previously provided to the Agency. GE has developed an advanced Monte Carlo model of exposure to PCBs from the consumption of fish in the Upper Hudson River (ChemRisk, 1995e). In October 1995, GE met with EPA and provided a conceptual description of the model, a printout of the computer code, copies of ancillary materials, and electronic copies of the working model. This material outlined the conceptual issues and provided technical material to assist in the evaluation of essential scientific questions in modeling. A peer-reviewed article based on this work has been published on the topic of modeling of exposure to PCBs from the consumption of fish (Keenan et al., 1996). GE urges EPA to

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consider, and where appropriate, incorporate these materials into its Monte Carlo modeling effort.

B.2 The SOW does not explain whether and how the Agency intends to model variation and uncertainty.

Monte Carlo modeling is fundamentally the same for any type of equation, involving the repeated use of an equation to produce a range of answers. However, the process of Monte Carlo modeling becomes more complex when models separate uncertainty from variability (Frey, 1993; Hoffman and Hammonds, 1994), and when models consider time-varying exposures such as microexposure event models (Price et al., 1996). 34

Several statements in the SOW suggest that such complex approaches will be used in the Hudson River assessment. However, the discussion is so scattered and incomplete that the approach the Agency intends to follow is not clear. For example, the SOW states that the exposure assessment portion of the risk assessment will consist of two parts: the first, a standard exposure assessment, and the second, a Monte Carlo analysis (SOW, at 6). The discussion continues that the probabilistic (Monte Carlo) analysis will attempt to capture information on uncertainty and variation. Yet, the discussion only includes a brief description of the steps needed to develop a Monte Carlo model of exposure variation across individuals; it contains no discussion of uncertainty. From this discussion, it is unclear whether EPA intends to assess uncertainty in the Monte Carlo modeling.

Similarly, there are various references in the SOW that allude to "two tier" (SOW, at 10, 11, 12) and "two-stage" (SOW, at 13) Monte Carlo analysis. It is not clear whether the Agency intends these statements to refer to separate analyses of uncertainty and variability or some other sort of analysis. In contrast, in the discussion of risk characterization (SOW, at 15), the SOW states that "[a]n enhanced Monte Carlo analysis will be performed to evaluate variability and uncertainty in exposure parameters, using two phases to distinguish the impacts of variability and uncertainty, where appropriate."

Although this implies separate analyses of uncertainty and variability, the description of the Monte Carlo modeling is insufficient to allow an understanding of how many of the elements will be performed.

GE urges EPA to analyze variability and uncertainty separately. EPA guidance has emphasized the value of separating variability and uncertainty in probabilistic analysis (EPA, 1997a,b). The Agency must recognize, however that this level of analysis poses significant challenges for the Agency. First, such analyses require the use of nested loops in programming (Hoffman and Hammonds, 1994). Since the current modeling recommended by GE requires the use of nested loops (one for each year, and inside of each year a loop for each fish consumed) (ChemRisk, 1995e), the addition of another layer of nested loops poses a significant computational challenge.

Second, all inputs with variability must jointly characterize uncertainty and variability. This may be relatively minor for inputs such as body weights but will pose a significant challenge for factors such as fish consumption, duration, and fish concentration.

Third, the Agency should not arbitrarily reject the consideration of certain sources of uncertainty. If the Agency includes quantitative analysis of uncertainty, then it should strive to include all sources of uncertainty. These sources include:

- uncertainty in the dose response measurements; 35
- uncertainty in intake rates that results from the use of data on the consumption of fish from multiple bodies of water;
- uncertainty in the stability of fish consumption rates over time;
- uncertainty in modeling fish tissue concentrations; and
- uncertainty in duration of exposure when the effects of cessation are not considered.

B.3 Other issues concerning Monte Carlo modeling

There are several other issues concerning Monte Carlo modeling that require comment.

First, the SOW suggests that an individual's exposure will take into consideration the time-varying nature of exposure concentrations (SOW, at 13). GE supports the consideration of time-varying exposures.

Second, the statement in the SOW that the "90th percentile will be always used" (SOW, at 15) is contradictory to the fundamental nature of Monte Carlo modeling. Monte Carlo modeling requires a random selection of input values from a distribution. Limiting the selection to a single percentile is clearly wrong. 36

Third, the SOW correctly states that there are no data on an angler's year-to-year variation in fish intake rates in the published literature (SOW, at 15). This raises an important issue on how to account for such potential variation. The SOW correctly states that intake rates over time will not be perfectly correlated because variations in weather, productivity of a fishery, vacation choices, and other factors will influence anglers annual fishing rates on a yearly basis. GE does not agree, however, with EPA's rationale for not allowing the model to vary the ingestion rate from year to year -- namely that such an approach "would assume that there is no correlation between yearly ingestion rates and effectively average the high-end consumers out of the analysis" (SOW, at 16). There are a number of methods by which an angler's year-to-year variation in intake rates can be modeled without assuming that anglers' annual intakes are not correlated. For example, intake can be correlated by allowing the intake rates to vary within a fixed range of percentiles (Price et al., 1997). The uncertainty in annual intake variation is a legitimate issue that should be addressed in modeling long-term dose rates. The Agency's adoption of a modeling approach where the intake rate is fixed as a worst case assumption unnecessarily adds conservatism and fails to use the strengths of Monte Carlo techniques. 37

Finally, the SOW states that the modeling of fish tissue concentrations may or may not include information on the variation in PCBs in fish of the same species, date, and location (SOW, at 13). This inter-fish variation can be shown to average out in anglers with high levels of fish intake. That is, this source of variation averages out and does not contribute to the variation in doses in anglers consuming large amounts of fish. As a result, all that may be necessary for modeling of fish tissue concentration is an estimate (with uncertainty) of the mean. This factor should be taken into consideration in the development of data on fish tissue levels.

B.4 Recommended approach

As noted above, GE provided EPA with detailed information on a modeling approach for characterizing exposures to anglers (ChemRisk, 1995 a-e) (attached). Based on discussions with the Agency, it was apparent at that time that EPA contractors generally agreed with the approach. GE still believes that this modeling approach is fundamentally correct and should be applied to the Hudson River. This modeling approach includes: 38

- modeling each fish consumed by each angler;
- defining the angler population of interest as those anglers who would begin using the river at a certain "start date";
- accounting for the temporal changes in the concentration of PCBs in fish;
- modeling exposure duration as a function of the demographics of the angler population;
- accounting for cooking loss;
- modeling the species preference;
- developing estimates of the average daily dose using a PCB specific averaging time; and
- accounting for temporal changes in angler body weight and behavior.

In addition, GE proposes the following additional comments on modeling angler exposures. First, the modeling approach should separate uncertainty from variability. This

should be done using a nested loop approach (Hoffman and Hammonds, 1994; Price et al., 1995). In the outer loop, values are selected from distributions that characterize the uncertainty in the inputs. As discussed above, this uncertainty should include all sources of uncertainty.

Within this outer loop, the model should consist of a microexposure model of variation in the anglers, as described by ChemRisk (1995c). EPA should investigate whether the model can be simplified by replacing distributions of inter-fish variation in concentrations (species, location and date specific) with the mean concentration.

In the case of the Mid Hudson, EPA should revise the model to discriminate between the exposures that hypothetically occur as a result of eating fish that have accumulated PCBs from the Upper Hudson River and those that have accumulated PCBs from other sources (in the Mid River and elsewhere). It will be critical for the Agency to track these two doses separately. This is necessary in order to provide risk managers with a clear understanding of the relationship between PCBs in the river and potential exposures in order to assess the potential effectiveness of remedial options in the Upper River. 39

EPA should also extend the models of dose to track how exposures from fish affect the human body burdens of PCBs. This analysis will require the incorporation of simple toxicokinetic models of PCB intake and retention into the microexposure Monte Carlo model (Keenan et al., 1997; Avantagio et al., 1998) and data on the background levels of PCBs in anglers. The goal of this analysis is to determine whether the consumption of fish from the Hudson River will change the body burdens of anglers (see Section 5). 40

C. Fish consumption rates

C.1 Development of a fish consumption rate distribution for recreational anglers

The amount of fish that anglers consume is an important parameter in the estimate of exposure to PCBs from Hudson River sediments. GE agrees with the SOW's conclusion that: 41

> *Fish ingestion rates are waterbody specific and depend on a number of factors including weather, available fish species, angler (man, woman or child who fishes), preference for specific species, impact of fishing bans, and distance of the angler from the water body.* (SOW, at 7)

This recognition is a significant improvement over the flawed approach taken by EPA (1991) in the Phase 1 Report, where a marine fisheries value of 30 g/day, subject to avidity bias problems (Price et al., 1994), was adopted as an estimate of fish consumption for Hudson River anglers. As GE has explained, the amount of fish consumed by a population of anglers depends on the numbers and types of waterbodies fished and the characteristics of the angler population (ChemRisk, 1995c; 1995e, attached). Fish consumption also depends on factors such as climate, fish species present, productivity, access, and the size of the angler population.

Consumption of fish is constrained by New York State fishing restrictions.

The most important factors affecting consumption of fish from the Hudson River are New York State's strict and effective restrictions on fish consumption in the Hudson River, which result in a significantly lower consumption rate than might be assumed under default exposure scenarios. It can be argued that the baseline HHRA should consider these restrictions in order to provide a site-specific and realistic estimate of exposure. The following paragraphs elaborate on this topic in light of EPA science policy and guidance. 42

Even apart from the Hudson-specific restrictions and advisories, New York State has issued a statewide general health advisory for eating sportfish from any of New York's freshwaters, "to protect against eating large amounts of fish that haven't been tested or [that] may contain unidentified contaminants" (NYSDOH, 1997). This advisory urges individuals to consume no more than one meal per week (32 g/day) of sportfish taken from any of the state's freshwaters (NYSDOH, 1997). This statewide general health advisory is different from the consumption restrictions placed on the Upper Hudson River. In addition, NYSDEC and the Atlantic States Marine Fisheries Commission impose strict, conservation-based fishing restrictions for various species, including striped bass. Even if EPA disregards the effect of the Upper River consumption ban in developing its rate of fish consumption for use in the baseline HHRA, EPA must address the statewide consumption advisory and the conservation-based fishing restrictions in this context. EPA must do so in light of the fact that these advisories and restrictions were issued independent of the presence of PCBs in the Hudson River. Specifically, any distribution of fish consumption rates selected for use in the baseline Hudson River HHRA must be truncated at 32 g/day to reflect the maximum consumption rate allowed by New York's advisory.

It is appropriate and consistent with EPA guidance for the exposure assumptions in the baseline risk assessment to reflect real-world, current conditions – especially those like the statewide general health advisory that are unrelated to the risk management of the Hudson River PCB Superfund Site. As a general rule, EPA's Risk Assessment Guidance for Superfund ("RAGS") favors the use of site-specific information instead of generic, standardized assumptions. See Risk Assessment Guidance for Superfund, Human Health Evaluation Manual Part A (Interim Final) (July 1989). For example, RAGS recommends examining actual and potential "land use" when characterizing the potentially exposed populations, id. at 6-7, and evaluating a number of site-specific factors that affect the ingestion of chemicals through consumption of fish. Id. at 6-43. Other Superfund guidance provides that, when available, site-specific exposure information should be used in the baseline risk assessment in lieu of standardized, default exposure assumptions. OSWER Directive 9285.6-03, RAGS Volume I: HHEM Supplemental Guidance "Standard Default Exposure Factors" (Interim Final) (March 25, 1991)(Standard exposure

assumptions are to be used only where site-specific values arenot available.). Similarly, the EPA Science Advisory Board ("SAB") recommends the use of site-specific information in Superfund risk assessments where site-specific conditions may be unique, as on the Hudson, and limiting the use of default information to circumstances where exposure parameters are unlikely to vary significantly from site to site. An SAB Report: Superfund Site Health Risk Assessment Guidelines (February 1993, at 2).

Indeed, the whole tenor of EPA's risk assessment approach, as articulated in the above references, is to rely on site-specific information to the greatest extent possible. EPA's Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish: A Guidance Manual (September 1989) (EPA-503/8-89-002) recommends "that local or regional assessments of fishery consumption be performed whenever possible to avoid errors inherent in extrapolating standard values for the U.S. population to distinct subpopulations." (Id., at 54). EPA's Exposure Assessment Guidelines recommend the use of distributional methods of exposure analysis, such as Monte Carlo analysis, because they rely on site-specific data to provide a more precise understanding of the range of actual exposures to an affected population. (57 Fed. Reg. 22889, 22922, May 29, 1992). The SAB similarly recommends that distributional exposure approaches be used in Superfund risk assessments in order to reflect the actual behavior and exposures of those who visit or live near a site because it is "more consistent with the exposure assessment guidelines, and ...in the spirit of the Exposure Factors Guidelines." (Id at 17). In fact, the Commission on Risk Assessment and Risk Management established under the Clean Air Act Amendments of 1990 advocates the use of distributional exposure assessment methods because they incorporate population-specific exposure (receptor-based) information, and not merely assumptions about exposure derived from sources and models. "Risk Assessment and Risk Management in Regulatory Decisionmaking: Volume 2" (1997, at 774-75). The Commission noted that source-based exposure information can be "seriously misleading" as compared to personal measurement results, where individuals in a particular population are monitored for exposure. (Id., at 191).

The distinction between source-based exposure and receptor-based exposure is an important one for EPA to consider, where the pathway from a contamination source (*i.e.*, fish) to a potentially affected population is markedly reduced from that of the generic consumption assumptions on account of New York's statewide health advisory and the conservation-based fishing restriction. Unless the advisory's maximum daily rate of fish consumption (32 g/day) is used to truncate the regional fish consumption distribution selected for use in the Monte Carlo analysis, the risk assessment will fail to reflect the actual receptor-based exposure levels of the potentially affected fishing population.

Using standard fish consumption assumptions would also be contrary to Agency policy. EPA's 1992 Exposure Assessment Guidelines state that, for fish tissue, the following site-specific data are required to characterize exposure: relationship of samples to food supply for individuals or population of interest, consumption habits, and preparation habits (57 Fed. Reg., at 22910). This information is unknown for the Hudson River, largely because of the consumption ban (and subsequent enforcement) on the Upper Hudson, the consumption advisory on the Lower Hudson, the conservation-based fishing restrictions on the lower Hudson, and the statewide general health advisory pertinent to all freshwaters of the state. As it is reasonable to assume that the latter two factors will remain in place for the foreseeable future, regardless of what remedial decision is made for the Hudson River, their effects must be incorporated into exposure information used in the baseline HHRA.

<u>The data from Ebert et al. (1993) should be used to calculate hypothetical fish consumption rates for the Upper Hudson.</u>

If, in spite of the Upper River fish consumption ban and the Lower River advisories, the Agency nevertheless assumes that fish consumption is occurring, then significant changes need to be made to the SOW's proposed approach for estimating consumption rates for recreational anglers. Most importantly, EPA should use the data from Ebert et al. (1993) to calculate hypothetical consumption rates for the Hudson River. Furthermore, the consumption rate distribution derived from Ebert et al. should be

truncated at a maximum value of 32 g/day to reflect the New York statewide health advisory level for freshwater fish consumption (NYSDOH, 1997).

GE disagrees with the SOW's assessment of the surveys that might be used for estimating fish consumption in the Hudson River in the absence of the current restrictions. Of the three surveys of angler behavior on the Hudson River, two are mail surveys of New York anglers in general (NYSDEC, 1990; Connelly et al., 1992), and the Clearwater creel survey (Barclay, 1993) was performed on Hudson River anglers. None of these surveys focused exclusively on fish consumption from the Hudson River. NYSDEC (1990) evaluated fish consumption from all recreational and commercial sources, including self-caught fish from the Hudson. Connelly et al. (1992) evaluated self-caught fish consumption but did not estimate consumption from individual waterbodies. Barclay (1993) collected data on the frequency of self-caught fishmeals but did not calculate a fish consumption rate. In addition, this survey does not contain sufficient information to allow the calculation of a meaningful fish consumption rate for the Upper Hudson River.[3]

Because the available surveys are flawed and cannot be used to assess hypothetical consumption rates in the absence of restrictions, the SOW should base the Hudson River estimates on data from similar bodies of water or from regional data. The selection of a surrogate study depends on the characteristics of the population under consideration and the type of waterbody being evaluated. Specifically, it is critical that the study be focused on the consumption rates of self-caught, freshwater fish over long periods of time. These criteria must be met to ensure that the fish consumption rate closely approximates hypothetical consumption from the Hudson. It would be preferable to use a study that evaluated consumption from a single river that was similar to the Hudson. If a specific waterbody with appropriate characteristics cannot be identified, it would be more appropriate to use estimates generated for flowing waters only. The selected study should

[3] The SOW implies that the Agency intends to create a distribution of fish consumption for the Hudson River by merging data from multiple studies. Such a process is complex, with important logical and statistical issues that must be addressed before proceeding. It is not clear from the SOW that EPA intends to do so. Moreover, the rationale for using data from these angler surveys appears to be founded on the idea that if their results are consistent, EPA can have greater confidence in selecting the Connelly et al. data as its *a priori* favorite (SOW, 8).

have collected data from regionally appropriate waterbodies. In addition, there should be a metric that demonstrates the appropriateness of selection and the uncertainties associated with it.

There are a limited number of studies available in the New York/New England area that provide information on consumption of sport-caught fish from freshwater rivers and streams. The Ebert et al. (1993) and Connelly et al. (1992) studies most closely approximate hypothetical consumption from the Hudson River.[4] Both of these studies evaluated consumption of self-caught freshwater fish by recreational anglers using a mail recall survey. Given these similarities, it is not surprising that both studies reported very similar fish consumption rates. The results of Connelly et al. (1992) indicated that the average New York angler consumes 11 meals per year of self-caught fish from New York's freshwater fisheries. If it is assumed that each meal is 227 grams in size (1/2 pound) (West et al., 1989; NYSDEC, 1990), it can be estimated that the average New York angler consumes self-caught freshwater fish at a rate of 7 g/day. This estimate is very similar to the mean rate of freshwater fish consumption by Maine anglers of 6.4 g/day from all waters reported by Ebert et al. (1993).

Although the Connelly et al. (1992) study is specific to New York State, there are several factors that would require the analyst to make additional assumptions to use the data as the basis for the Hudson River assessment. First, Connelly et al. (1992) only presents a single point estimate value for fish consumption. The use of a distribution of consumption rates is necessary in order to characterize interindividual variability and realistically assess the potential risks to recreational anglers. With only an average consumption rate value, it is not possible to represent the range of recreational anglers accurately, including those anglers who ingest higher amounts of fish. While it may be possible to develop a distribution of consumption rates by going back to the original raw data, additional analysis will be required to complete this task.

[4] Connelly et al. (1996) surveyed Lake Ontario anglers to evaluate the effect of Lake Ontario health advisory recommendations and therefore this study may not be directly relevant to fish consumption in the Upper Hudson.

Second, the mean fish consumption rate determined by Connelly et al. (1992) represents fish eaten from all freshwaters in the State (i.e., lakes, ponds, rivers, and streams). As pointed out in Ebert et al. (1993), intake from rivers and streams is only a fraction of the intake from all freshwaters. In addition, the rate of intake from multiple waterbodies is higher than that from a single water system (Ebert et al., 1994). Given these factors, it is highly likely that the fish consumption rate in Connelly et al. (1992) overestimates the hypothetical fish consumption rate on a single portion of the Upper Hudson River.

Finally, the purpose of the Connelly et al. (1992) study was not to identify a consumption rate for New York anglers. Although questions were asked in the survey regarding fish consumption behaviors, those questions were aimed at estimating how the effect of health advisories altered the consumption behavior of recreational anglers.

While the data from Ebert et al. (1993) are not specific to New York State, these data are readily useable and may provide a more appropriate surrogate for Hudson River anglers than the Connelly et al. (1992) data. Angler demographics and fishing opportunities are similar in Maine and New York, and the mean fish consumption rates are similar for both studies (NYSDEC, 1990; Connelly et al., 1992; Ebert et al., 1993). In addition, Ebert et al. (1993) provides a complete distribution of fish intake rates for flowing waters, i.e., streams and rivers. The Ebert et al. (1993) survey also addresses each of the criteria identified in the SOW (SOW, at 9) to evaluate angler surveys.[5] Thus, the best region-specific data on fish consumption rates are available from Ebert et al. (1993), and GE urges EPA to use these data in the Hudson River risk assessment.

[5] The selection of the most appropriate fish consumption rate is discussed more fully in the paper entitled *Estimating Fish Consumption Rates for the Upper Hudson River* (ChemRisk, 1995c) and in the peer-reviewed journal articles, *The Effect of Sampling Bias on Estimates of Angler Consumption Rates in Creel Surveys* (Price et al., 1994), *Selection of Fish Consumption Estimates for Use in the Regulatory Process* (Ebert et al., 1994), and *Estimating Consumption of Freshwater Fish among Maine Anglers* (Ebert et al., 1993).

The SOW's definition of study population is arbitrary and must be changed.

GE disagrees with the SOW's arbitrary definition of the study population. The SOW truncates the full distribution of hypothetically exposed anglers by defining the lower end of the study population as those anglers "who would consume self-caught fish from the Hudson River at least once per year in the absence of a fishing ban." Many anglers, however, will consume fish at frequencies much less than once per year (ChemRisk, 1991a; Ebert et al., 1993). The SOW's omission of such frequencies thus biases upwards the resulting risk estimates. A better approach would be to define the population as those anglers who consume fish once in the "start year" (the first year of exposure) but not require them to consume fish in every subsequent year. This would allow the model to include anglers who consume fish at lower frequencies than once per year. 43

For the Mid Hudson, the HHRA should also evaluate its selection of angler consumption studies in the context of potentially short-lived fishing activities, such as fish tournaments or short fish runs (e.g., shad run in the Mid and Lower Hudson River), to determine the potentially exposed populations. Because of these events, it is possible that the estimates of fish consumption for the Upper Hudson cannot be applied categorically to the Mid Hudson. Therefore, surrogate surveys of fish consumption that consider such events are needed to accurately select a distribution of fish consumption rates for this section of the River.

C.2 Angler Subpopulations

The SOW states that no attempt will be made to distinguish subpopulations of "highly exposed or lesser exposed anglers." GE agrees with this approach, which is supported by the available data suggesting that recreational anglers are the appropriate population for the HHRA. 44

Historically, concerns have been raised over hypothetical subpopulations of anglers who consume greater amounts of self-caught fish than the general recreational angler population, due to their reliance on fishing as a major or sole source of dietary protein for their families (USEPA, 1998); Abraham et al., 1995; Becher et al., 1995; McCormack and Cleverly 1990; West et al., 1991). The term subsistence anglers has been applied to this population.

The characterization of this population has been extremely ambiguous. In North America, Native American populations that have subsistence and treaty rights to certain fisheries (CRITFC, 1994) and Arctic Inuits who, because of tradition and their remote location, rely heavily on native foods obtained from the sea (Kinloch et al., 1992; Coad, 1994) appear to be high consumers. Beyond these fairly well defined populations, clear examples of subsistence anglers are difficult to define.

In order for an individual to consume at high rates, that person must have access to large amounts of the fish and must have either a need or preference to consume locally caught fish in large quantities. There are several factors that could define such a population including:

- low income individuals who must rely on fish for their dietary needs,
- native peoples who have cultural traditions of consuming large quantities of fish,
- commercial anglers who have ready access to large amounts of fish, and
- recreational anglers who have a strong preference for fish

The fish consumption habits of these subpopulations, compared with the distributions of consumption rates for the general recreational angler population, are discussed below.

Income level

Low income, in and of itself, does not lead to high levels of fish consumption. The fish consumption survey literature indicates that there are no significant differences in fish consumption rates among different income groups (Ebert et al., 1993; Connelly et al. 1990; West et al., 1991). Wendt (1986) studied the fish consumption habits of low income families

living in New York State to determine how much freshwater fish they consumed from New York State waters. Based on the reported range of meals and an assumed meal size of 1/2 lb. (227 g), it can be estimated that these individuals consumed at a mean rate of 11 g/day and a maximum rate of 60 g/day. This mean is consistent with the means reported in more recent surveys of New York's recreational anglers (Connelly et al., 1990, 1996) and other recreational anglers in the Northeastern U.S. (Ebert et al., 1993; 1996), while the maximum rate is lower. Thus, low-income populations living in New York State do not have higher rates of fish consumption than recreational anglers in the region.

Ethnic background

There are data indicating that certain localized North American ethnic subpopulations may have higher rates of consumption than the general angler population. Studies of native peoples in the Pacific Northwest of the U.S. and Canada indicate that they rely more heavily on fish as a staple of their diets than does the general population. However, these findings are of little relevance to the Upper and Mid Hudson Rivers since the counties bordering these portions of the River do not have such local ethnic populations.

In addition, when individuals from these same ethnic populations reside in a more heterogeneous and economically developed area, these differences diminish (Wolfe and Walker 1987). While mean consumption rates reported for native peoples living in closer proximity to economically developed areas were higher than the mean values reported for the general recreational populations (NYSDOH, 1993; West et al. 1991; Selikoff et al. 1982; Hutchison and Kraft, 1994; Peterson et al., 1994), their maximum rates were similar. Other comparisons of fish consumption by ethnic background have reported no significant differences among consumption rates for those groups (Ebert et al., 1993; Landolt et al., 1985; Anderson and Rice, 1993).

Commercial Anglers

Since at least 1976, commercial angling has not been practiced on the Upper River and has been severely restricted for many species on the lower River. Nevertheless, this group could, in theory, be a population of concern for the Mid River. Individuals who have commercial fishing licenses have unlimited access to their marketable catch and might be assumed to consume more fish than the recreational angler population. Limited data on the fish consumption activities of freshwater commercial anglers show that such anglers do not eat substantial amounts of the fish that they harvest, due to the fact that the sale of those fish is critical to their household income and their ability to pay for other foods and living expenses. For example, Hubert et al., (1975) studied commercial freshwater fishing activities in Upper East Tennessee during 1973 and reported that, of a total of 94,079 kg of fish commercially harvested by 29 anglers, 2,665 kg were retained for personal use. If this amount of fish is divided among the 29 anglers and their families and assumed to have edible portions of 30 percent, the resulting mean consumption rate is 25 g/day. This mean rate is very similar to mean rates reported for recreational anglers fishing large bodies of water (SCCWRP and MBC, 1994; Ebert et al., 1994). Thus, commercial freshwater anglers do not consume substantially more fish than recreational anglers fishing the same types of waterbodies.

Recreational Anglers

Fish consumption rates among recreational anglers are highly variable. Based on available survey data and on a critical review of the relevant literature, high-level fish consumers in North America are a diverse group that cannot easily be defined or identified by socioeconomic characteristics. With the exception of certain native peoples who have continued to promote their cultural dietary traditions, there are no social or economic characteristics that are associated with the presence of a high fish consuming population. Consequently, EPA has made the correct decision in not developing a separate exposure assessment for this population.

C.3 Species-specific fish ingestion rates

In general, GE agrees with the Agency's attempt to address species-specific fish ingestion rates in the exposure assessment. Anglers typically prefer to catch certain desirable species and to reject others. Moreover, many anglers engage in short-lived fishing activities, as mentioned previously. Since PCB levels in fish vary by species, it is important to capture this angler preference in the estimates of exposure to PCBs. The NYSDEC study and the work by Connelly et al. show that New York anglers preferentially select for certain species in both fishing effort and consumption (NYSDEC, 1990; Connelly et al., 1992). In many cases, the species selected were those that accumulate lower levels of PCBs, often because these most desirable species have relatively low lipid contents as compared to other species present in the Upper Hudson. Since the species of fish sampled by EPA or NYSDEC for PCB tissue analysis are not necessarily consumed by recreational anglers in amounts proportional to their sampling frequencies, the risk assessment for the Upper Hudson should consider both interspecies differences in PCB concentration and angler preferences.

Information on species preference specific to the Upper Hudson River is unavailable. However, data on angler preference in freshwater rivers in New York similar to the Upper Hudson River are available from Connelly et al. (1992).[6] Based on these data, it is possible to identify species preferences among New York anglers that can be used as a surrogate for Hudson River anglers. Connelly et al. (1992) collected information on fishing behaviors (e.g., species caught, waterways fished) and fish consuming behaviors (e.g., species eaten, preparation techniques used) of licensed anglers. In order to use these data for the Upper Hudson, it is necessary to identify rivers and streams with characteristics and species similar to the Upper Hudson. Such an analysis results in a list of fish species likely caught in the Upper Hudson and the probability of how often these species are eaten. By taking this approach, a probability distribution that accurately reflects species consumption preferences of Hudson River anglers can be developed. This

[6] EPA should not include the Connelly et al. (1996) study as it was a survey of Lake Ontario fishers.

issue is addressed in ChemRisk (1995a), which recommends the appropriate input parameters for the microexposure Monte Carlo analysis.

GE is concerned about the SOW's proposed approach for considering species-specific consumption (SOW, at 9-10). The SOW states that species-specific fish ingestion rates will be developed from data collected from multiple studies of anglers. The result of this analysis appears to be some sort of species weighting factors that will be applied to all anglers. This use of a single set of factors implicitly assumes that all anglers will consume the same species and in the same proportions. As the SOW acknowledges (SOW, at 10), this assumption is implausible. Anglers can be expected to vary the species they consume based on their choice of fishing location, tackle, and means of fishing. The SOW proposes to address this uncertainty by running a separate analysis in which anglers will be assumed to consume the species with the highest level of PCBs (SOW, at 13). The SOW does not indicate how the results of the two estimates will be used in assessing the baseline analysis. In any event, this approach for assessing the impact of species choice is invalid because it provides the risk manager with results from two implausible sets of assumptions. 45

GE is also concerned with the SOW's proposal to combine surrogate fish species preferences for the Upper and Mid Hudson. This approach is unjustified and scientifically invalid as these anglers would be expected to have very different preferences based on the type of fish present in the respective stretches of the river. 46

D. Determination of future PCB concentrations in fish

D.1 Use of model results

The SOW states that projected PCB concentrations in fish will be determined using the EPA bioaccumulation models (SOW, at 12). There are two components to these predictions: the average concentration and the distribution of concentrations. 47

Average concentration

The Bivariate Statistical Model is the primary model developed by EPA to estimate mean fish total PCB and Aroclor levels. It is subject to several limitations, as described in GE's comments concerning the Preliminary Model Calibration Report (GE, 1996). One particular source of concern is that the BSM overestimates the observed values at low concentrations (see the enclosed reprint of EPA Figure 9-12). PCB levels in fish are declining, as shown by the trends since 1993 in the upper river. Therefore, it is important to be sure that model predictions do not overestimate the true future levels. As discussed in GE (1996), an alternative modeling methodology must be developed; a time-variable mechanistic bioaccumulation simulation is the preferred alternative.

In addition, the BSM has practically no predictive power within each river reach. For example, in EPA Figure 9-12, there is no relationship between predicted and observed largemouth bass total PCB concentrations in Thompson Island Pool (symbol "D" on the Figure). Because of these problems, the PCB levels predicted by the BSM may not reflect the decline in concentrations in largemouth bass in Thompson Island Pool. This will produce an unrealistically high computed human health risk. 48

The probabilistic food chain model (PFCM) is based upon linear steady state relationships between sediment, water and fish, as is the BSM. Therefore, the PFCM should be subject to the same bias and lack of predictive power as the BSM in predicting average PCB levels.

Variability

The SOW states that the high-end exposure point concentration for the HHRA will be determined using the 95% Upper Confidence Limit on the mean PCB concentration. The confidence limits of the mean are dependent upon the variance. Therefore, the distribution of PCB concentrations within each fish subpopulation must be estimated. One goal of EPA's modeling efforts was to compute the population variability using the PFCM.

As described in GE, 1996, the PFCM:

- is improperly constructed, calculating variability from uncertainty. This causes model results to have no physical meaning;
- requires the answer to solve the problem;
- incorrectly assumes sediment PCB levels do not change over time; and
- does not take advantage of the substantial information and data that are available concerning the mechanisms of bioaccumulation.

An alternative already suggested by GE is to use the extensive database collected by DEC over the course of 20 years to estimate the shape and parameters of the distributions of PCB levels in fish.

D.2 Selection of Mid Hudson fish species

The SOW states that high-end exposure point concentrations will be estimated using the most contaminated species and data from the most contaminated stretch of the Mid Hudson River (SOW, at 20). Unlike the Upper River, the Mid River contains migratory fish species (striped bass, eels, shad, etc.) that spend significant time away from the Mid River. During this time, the fish have the potential to accumulate PCBs from other sources than the Upper River. EPA must take care interpreting results from such fish when making remedial decisions related to the Upper Hudson. 49

E. Duration of exposure

The approach proposed to characterize the duration of exposure (SOW, at 10-11, 18-19) is a significant improvement over the approach used in the Phase 1 assessment, which relied on default values. As the SOW notes, mobility is a major consideration in determining the duration of exposure. The Agency's decision to use county mobility as a 50

surrogate for the probability that an angler will cease using the Hudson because of a new residence location is also appropriate.

However, this approach assumes that a move from one county bordering the Hudson to another does not end exposure, which is invalid for model runs in which the location along the river is considered. Moving from Hudson Falls to Albany will affect the probability of fishing the Thompson Island Pool. Therefore modeling of specific reaches should consider inter-Hudson River county moves.

GE supports the effort to take cessation of angling into the assessment of duration. The SOW, however, does not provide any support for the statment that "generally, anglers are highly dedicated to their sport, and few voluntarily stop fishing." GE has previously provided to EPA evidence from the Maine angler survey showing that cessation is an important factor (ChemRisk, 1995b). GE urges EPA to use these data and, as indicated in the SOW, to perform similar analyses of the data in Connelly et al. (1992).

The SOW fails to discuss the role of lifespan in limiting exposure duration. The population of concern is that group of anglers who would use the Hudson River at an appropriate "start date." Such a population at the time of the "start date" would have an age structure spanning from teenagers to individuals over 65. Therefore, the HHRA must take lifespan into account when determining the distribution of duration.

The HHRA also should model duration of exposure using the methodology described in ChemRisk (1995b). Under this approach the duration is not an input to the model but is directly based on age-specific estimates of cessation, mobility and lifespan. The advantage of this approach is that the age structure of the population is handled in a consistent fashion throughout the model.

F. Cooking loss

GE supports the use of the peer reviewed literature to model the loss of PCBs during cooking, as proposed in the SOW (SOW, at 11). Cooking loss of lipophilic compounds such as PCBs is a real and verified phenomenon that has been repeatedly demonstrated in more than 20 publications in the peer reviewed literature (e.g., Sherer and Price, 1993; Wilson et al. 1998) and forms the basis for fish advisories used by the State of New York (NYSDOH 1996; NYFW, 1995). As discussed in Wilson et al. (1998), the data are more than adequate to allow the modeling of cooking loss as a function of cooking method and in turn the type of fish. GE agrees that the estimates of reduction are subject to uncertainty, and that this uncertainty may warrant consideration in the modeling of exposures. 51

G. Inhalation exposures

The SOW proposes that risks from inhalation of ambient air will be computed using a deterministic assessment approach (SOW, at 13, 21). As an initial matter, the Agency should recognize that this route of exposure is insignificant. Studies of PCB blood levels in individuals near other Superfund sites have consistently revealed that such individuals do not have excessively high blood levels. GE urges the Agency to abandon this exposure route. 52

If the Agency still proceeds, it must specify the source(s) of the data it intends to use, when and with what method(s) the data were collected, the quality of the data, or how data will be evaluated with respect to calculating the high-end and central tendency point estimate concentrations. The SOW vaguely describes data collected from sites near the Hudson river, but provides no details concerning these data. The Agency must specify the source(s) of air concentration data and, if not currently publicly available, should make that data available for public review and comment

SECTION IV
TOXICITY ISSUES

A. GE supports the use of Aroclor-based toxicity criteria.

GE supports EPA's use of Aroclor PCBs in lieu of PCB congeners for the human health risk assessment. However, some may claim that an alternative approach should be 53
taken, which we do not support based on critical scientific inconsistencies and inappropriate assumptions. This alternative for evaluating potential risks from exposure to PCBs in environmental matrices consists of the following steps, all of which serve to increase the complexity and the uncertainty of the analysis. First, the concentrations of the 11 "dioxin-like" PCB congeners is converted to 2,3,7,8-tetrachlorodibenzo-p-dioxin toxicity equivalents (TEQs) through the use of one of several TEQ conversion schemes (Ahlborg et al., 1994; EPA, 1989; WHO, 1997) The choice of conversion method is left to professional judgement and can introduce additional uncertainty into the analysis. The carcinogenic risks are then calculated for the TEQs by combining the TEQ concentrations for these congeners with a CSF for 2,3,7,8-tetrachlorodibenzo-p-dioxin of 150,000 $(mg/kg\text{-}day)^{-1}$ or with one of the more recent and more scientifically appropriate values (Keenan et al., 1991). For the non-dioxin-like PCBs, this approach uses the total PCB concentration in conjunction with the CSF for PCBs of 2 $(mg/kg\text{-}day)^{-1}$ to yield the "non-dioxin-like" PCB risk. It then adds these risks together.

To be logically consistent with this approach, the analyst must subtract out the concentrations of the dioxin-like congeners from the total PCB concentrations before making the calculations for the other PCBs. If one fails to do so, then the analysis has additional flaws due to double-counting of the carcinogenic potential of the dioxin-like congeners by including those congeners both in the risk calculation for the TEQs and in the risk calculation for the so-called "non-dioxin-like PCBs." Moreover, even if the analyst subtracts out the concentrations of the dioxin-like congeners in making the risk calculations for the remaining PCBs, this approach would still double-count the carcinogenic potential of the dioxin-like congeners, because those congeners are included

in the CSF for PCBs. The CSF for PCBs of 2 $(mg/kg\text{-}day)^{-1}$ was based on toxicological studies of Aroclor mixtures that contained dioxin-like congeners. Indeed, EPA has attributed much of the so-called carcinogenic potency of PCB mixtures to these congeners (IRIS, 1998). Thus, the CSF of 2 $(mg/kg\text{-}day)^{-1}$ is much too high to represent the carcinogenic potential of the non-dioxin-like congeners. Accordingly, even if the PCB concentrations used for the non-dioxin-like PCBs does not include dioxin-like congeners, the use of a CSF of 2 $(mg/kg\text{-}day)^{-1}$ to calculate the carcinogenic risk of those PCBs represents a double-counting of risks. In fact, unless there were a CSF for non-dioxin-like PCBs, there is no defensible way to use both the TCDD CSF and the PCB CSF in the same assessment.

Furthermore, the toxicological, epidemiological and analytical databases for Aroclor PCBs are more reliable and complete than those for PCB congeners. In summary, the following represent the advantages associated with the use of Aroclor PCBs in lieu of PCB congeners:

- The toxicity studies used to derive the Aroclor PCB CSFs and RfDs include both the coplanar and non-coplanar PCB congeners present in the Aroclor mixtures.

- The concentrations of the coplanar PCB congeners - reputedly the more toxic of the PCB congeners - is known for most of the Aroclor PCBs (e.g., Frame et al., 1996).

- There is a paucity of toxicity data for the non-coplanar PCB congeners.

- The comparability of analytical results can be difficult in PCB congener data since there are inconsistencies in the analytical methods used to quantify coplanar and non-coplanar PCB congeners.

- Aroclor PCB results are the most appropriate to use if there have not been significant changes in the PCB peak patterns.

B. Cancer dose response

GE supports EPA's recent efforts in reassessing the cancer risk of PCBs (EPA, 1996, "the Reassessment") and continues to have an important interest in working with EPA on various issues related to PCB toxicology. Although the Reassessment represents a positive step in evaluating the PCB cancer risk suggested by animal studies, GE believes that EPA needs to conduct additional analyses of existing and forthcoming data if it is to accurately assess and quantify the cancer risk that PCBs pose to humans. 54

Specifically, GE has previously submitted comments in several rulemakings, urging EPA to consider the numerous epidemiological studies that have been performed on populations with extensive workplace exposure to PCBs. Others have asked EPA to use epidemiological studies to establish a human cancer potency factor. EPA's responses to these comments, as well as statements in the Reassessment and the IRIS database, have been sparse and have suggested strongly that EPA has not thoroughly reviewed the epidemiological studies or considered how they can be used in risk assessment. GE believes that the SOW presents the Agency with a good opportunity to consider this matter more thoroughly.

B.1 The rationale for using epidemiological studies to establish environmental standards

To date, EPA has established cancer slope factors for PCBs based on the results of rat feeding studies. EPA's most recent effort in this regard is the Reassessment, which advocates use of a range of cancer slope factors based on the results of rat studies. Risk managers are to choose a slope factor from within the range based on the regulatory context and the pathway by which humans are expected to be exposed. 55

Although EPA has historically viewed all positive findings in animal bioassays as suggesting equally serious human health hazards, in reality chemical carcinogens may have tissue-specific effects, and different mechanisms of action and pharmacokinetics.

Additionally, chemicals may differentially exhibit carcinogenic effects under specific animal bioassay conditions that are unrelated to reasonable human exposures. Moreover, as discussed in the reassessment, studies vary in quality and power.

EPA recognizes the difference in potency of chemical carcinogens tested in animal bioassays, but does not evaluate the probability that such chemicals may not be human carcinogens. Many chemicals that have been proven to be carcinogenic at high doses in animal bioassays have not been shown to be carcinogenic in humans at or near environmental or occupational exposure levels. As an example, over 50 percent of approximately 400 to 500 chemicals have tested positive in at least one rodent species at high doses (Ames 1989). However, only approximately 20 chemicals are known to cause cancer in humans (Doll 1984; Paustenbach et al., 1990). Even after accounting for the typical shortcomings of some epidemiology studies (small sample size and poor quantitative knowledge of relatively small exposures), it is clear that many potent rodent carcinogens do not pose an equivalent cancer hazard in humans. (Houk 1990; Kimbrough 1990).

There are several difficulties in estimating human cancer risks from rodent bioassays. Differences in pharmacokinetics and susceptibility to organ toxicity complicate the issue of interspecies extrapolation (MacDonald et al. 1994). Compounds classified as tumor promoters are particularly troublesome in this regard, because they often produce rodent liver tumors in long term bioassays, but are not generally known to cause cancer in humans (Butterworth et al. 1995; Schulte-Hermann 1985). Tumor promoters like PCBs selectively increase the growth of cancerous cells, but do not interact with cellular DNA to cause the initial heritable change that begins the multi-stage process of cancer. The drug Phenobarbital is a classic example of a rodent liver tumor promoter that has not been shown to cause cancer in humans taking this drug for many years (Butterworth et al. 1995).

Another problem caused by the use of animal studies to predict human cancer risk is the need to use a model for extrapolation from high doses to animals to low doses to humans. In the Reassessment, EPA estimated the carcinogenic potency of PCBs by using the linear default method presented in EPA's Proposed Guidelines for Carcinogen Risk Assessment (EPA 1996). This method is likely to overestimate the low-dose carcinogenic risk of PCBs because it assumes that there is a direct linear relationship between the dose of the chemical and a carcinogenic effect. The rationale given by EPA for using a linear low dose extrapolation in the Reassessment is based on the possibility that PCBs might act in concert with other exposures and processes leading to a background incidence of cancer that would be linear at low doses.

Originally, the assumption of linearity was based on an elementary theory of the mechanism of chemical carcinogenesis, in which a single chemical molecule can form an adduct to DNA, and thereby result in cancer.[7] Tumor promotion, however, is characterized as a reversible process and the dose response relationship is expected to be nonlinear, including both a threshold dose level and a maximal response (Pitot and Dragan 1991). EPA's recent cancer guidelines (EPA 1996) allow for nonlinear low dose extrapolation in cases where the available data support a nonlinear mode of action (e.g., nongenotoxic agents).

EPA concedes that there are a number of chemicals which produce a carcinogenic response by mechanisms that may exhibit a non-linear dose response curve at low doses (EPA 1996; Butterworth and Slaga 1987). The increased acceptance of the nonlinearity of dose and effect at low doses is evidenced by a growing consensus among risk assessment practitioners that the linear model is inappropriate for dioxin, thyroid-type carcinogens, nitrilotriacetic acid, trimethylpentane and, presumably, similar non-genotoxic chemicals, (Paynter et al. 1988; Andersen and Alden 1989; Paustenbach 1989; EPA 1992b). Given

[7] While genotoxic chemicals are assumed to be better modeled by a linear dose response assumption (Weisburger and Williams 1987), this is not a proven scientific fact. Ottobonni (1984) suggested that genotoxic agents might also exhibit thresholds at low doses. These thresholds may result from a number of factors including DNA repair mechanisms, cell death, or lethal mutations. Therefore, there is considerable uncertainty in the assumption of low dose linearity for carcinogens.

the uncertainty in cancer dose response modeling, the Agency should reexamine the evidence for carcinogenic risk that can be derived from human epidemiology studies. It has been stated that epidemiologic studies are not as statistically robust as animal studies and, therefore, not as useful (Silbergeld et al. 1988). Although this can be a legitimate concern in some cases, in many cases human epidemiology studies can and should be used to validate, confirm, or set upper bound estimates of carcinogenic potency. In general, when epidemiology data are available, it is not appropriate to accept only the results of mathematical models that analyze rodent data without serious consideration given to the human experience[8] (Cook 1982; Dinman and Sussman 1983; Layard and Silvers 1989). EPA (1996b) appears to recognize this point in its proposed cancer guidelines:

> *Epidemiologic data are extremely useful in risk assessment because they provide direct evidence that a substance produces cancer in humans, thereby avoiding the problem of species to species inference. Thus, when available human data are extensive and of good quality, they are generally preferable over animal data and should be given greater weight in hazard characterization and dose response assessment, although both are utilized.*

In the case of PCBs, EPA can no longer ignore the many clinical and epidemiological studies that do not support the proposition that PCBs cause cancer in humans. GE realizes that toxicologists must be careful in relying on the results of negative epidemiological studies. However, when, as in the case of PCBs, several excellent epidemiological studies have been performed using large numbers of workers heavily exposed to a chemical over a long period of time, and the results of those studies have been negative, GE submits that such results must be factored into, or used in, the derivation of a human cancer potency factor. As discussed below, the epidemiology studies of

[8] An example of where an animal study yielded implausible results is ethylene dibromide (EDB). In 1982, it was claimed that workers exposed for 8 hrs/day for 40 years to the OSHA threshold limit value (TLV) for EDB of 20 ppm incurred a risk of 999 in 1,000 of developing cancer. However, epidemiological evidence of actual cancer incidence in these workers did not show an increase in the cancer rate (Cook 1993). Although the EDB risks suggested by the low-dose animal models may initially seem plausible, the human epidemiologic evidence makes it clear that these workers are not likely to die prematurely as the model predicted (Hertz-Picciotto et al. 1988).

PCB-exposed cohorts do not indicate that PCB exposure leads to increased mortality, whether based on overall cancer mortality or deaths due to individual cancer types. These findings strongly suggest that human health risks from PCB exposure have been significantly overestimated in current regulations and that EPA should undertake a thorough reevaluation of the actual risks posed by PCB exposures.

In assessing the PCB studies, EPA should use state-of-the-art methodology for interpreting the results of epidemiological studies. This methodology uses a weight-of-the-evidence test and applies what has become known as "causation analysis." The methodology is well recognized within EPA (EPA 1992a; EPA 1996b). At least ten criteria have been proposed for establishing cause and effect relationships (Hill 1965; Evans 1976; Hackney and Linn 1979; Doll 1984; Guidotti and Goldsmith 1986; Mausner and Kramer 1985; Monson 1988; Hernberg 1992). However, as typically applied, the scientific convention applied in weight-of-the-evidence evaluation of epidemiological studies requires (a) the observation of a specific cancer endpoint, and (b) the meeting of six other criteria before a causal relationship between an agent such as PCBs and cancer can be inferred (Hill 1965; Mausner and Kramer 1985; Rothman 1988; Monson 1988; Hernberg 1992; EPA 1985b; IARC 1987; EPA 1996b). The six fundamental criteria are: strength of association; consistency of association; temporally correct association; dose-response relationship; specificity of the association; and coherence with existing information (also called "biological plausibility"). None of the criteria, with the exception of temporality, should be considered as necessary to establish causation. Each of the criteria is important, and causation is established by the weight of the evidence and the degree to which all six criteria are satisfied by the available data. However, the rejection of the association may be made with a high degree of confidence when three of the criteria -- temporality, consistency, and biological plausibility -- are not met (Rothman 1988; EPA 1996b). In addition to considering weight of the evidence, it is important to understand that studies with larger cohorts and numbers of cancer deaths are inherently more important when considering the weight of the evidence than are studies with smaller cohorts and fewer cancer deaths.

B.2 Epidemiological data

The collected evidence from numerous epidemiological studies over the past 20 years fails to demonstrate that PCBs cause cancer in humans, even in populations with much greater exposures than those involved here. A review of the epidemiology data for PCBs is provided below. In general, studies of PCB workers, who were exposed to PCB levels hundreds or thousands of times higher than current environmental levels, have failed to demonstrate a causal association between PCB exposure and cancer. Further, in a recent study by Harvard University researchers, no relationship was found between PCBs and breast cancer among 240 women (Hunter et al., 1997). 56

The most celebrated incident in which PCBs became suspected of causing cancer in humans is the so-called "Yusho" incident. In 1968, about 1500 persons in Japan became ill after consuming rice oil that was accidentally contaminated with a PCB mixture known as "Kanechlor 400" (Amano et al. 1984). A similar incident, known as "Yucheng," occurred in Taiwan in 1979. Typical symptoms were chloracne, swelling of eyelids, eye discharges, brown pigmentation of the nails and skin, and curling of fingernails and toenails. Signs of the disease were also observed in some offspring of affected mothers. Although the major symptoms disappeared over the next sixteen to twenty years, subsequent studies suggested a possible increase of cancer and adverse developmental and behavioral effects in offspring.

The cause of the incident was extensively studied and the rice oil was found to contain high levels of polychlorinated dibenzofurans ("PCDFs"), a chemical that is 100 to 1,000 times more toxic than PCBs. After finding that workers exposed to much higher levels of PCBs showed minimal adverse health effects, and after performing dose-response studies on the rice oil mixture, Japanese and Taiwanese scientists concluded that PCDFs were the prime causal factor in the Yusho and Yucheng incidents (Kashimoto et al. 1986). ATSDR agrees, finding that "there is inconclusive evidence of cancer in people who were exposed to heated non-Aroclor PCBs during the Yusho and Yu-Cheng incidents, but PCDFs were major contaminants" (ATSDR 1997).

In 1985, Dr. Kimbrough and Dr. Goyer of the National Institutes of Health unequivocally concluded that:

> *The scientific community assumes now that most of the effects observed in these two outbreaks were caused by the ingestion of the polychlorinated dibenzofurans.* (Kimbrough et al. 1985)

Likewise, the Halogenated Organics Subcommittee of EPA's Science Advisory Board reviewed a PCB health advisory from EPA and concluded that:

> *The health effects section suggests that the short-term human exposure to Yusho poisoning is [not] representative of polychlorinated biphenyl toxicosis. Recent studies indicate that the major etiologic agents in Yusho were polychlorinated dibenzofurans rather than polychlorinated biphenyls... Thus, a discussion of the human health effects of polychlorinated biphenyls should not use 'Yusho' as an example. Industrial exposure data more accurately reflect human health effects.* (Doull et al. 1986)

A number of years later in her update of PCB exposure and human health effects, Kimbrough (1995) emphatically stated that:

> ***In the poisoning outbreaks, the PCDFs, not the PCBs, caused the adverse human health effects.***

Significantly, this scientific reinterpretation of the Yusho and Yucheng incidents is consistent with data from animal studies that show a relatively low level of acute toxicity — *e.g.*, LD50s ranging from about 1 to 11 g/kg-body-weight in rats, depending on the Aroclor mixture. Moreover, this explanation is consistent with the numerous studies

(discussed below) that show no significant adverse health effects in workers who had been exposed to average levels of PCBs higher than the Yusho patients were.

Subtracting Yusho from the universe of epidemiological studies of the cancer risk of PCBs leaves a number of other studies which can be grouped into three categories: (1) negative studies reporting no statistically significant relationship between exposure to PCBs and cancer (Taylor 1988; Kimbrough et al. 1997, unpublished; Hunter et al. 1997; Zack and Musch 1979; Gustavson 1986; Nicholson et al. 1987), (2) studies that were inconclusive due to small cohort sizes or flaws in study design and data interpretation (e.g., Yassi et al. 1994); and (3) studies which have been cited by some as suggesting a relationship between human exposure to PCBs and cancer (Bahn et al. 1976, 1977; Bertazzi et al. 1987; Brown 1987; Sinks et al. 1991). As will become clear from the following discussion, the studies, whether considered individually or assessed using a weight-of-the-evidence approach, provide virtually no support to the claim that PCBs are human carcinogens.

Inconclusive Studies

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Yassi et al. (1994) examined the mortality of 2222 males employed between 1946 and 1975 at a transformer manufacturing plant in Canada. Although some transformers were filled with PCB-containing fluids, the vast majority were filled with mineral oils refined predominantly from naphthenic base crudes (only 85 of 51,000 transformers filled between 1956 and 1975 contained PCB fluids).

This report concludes that neither overall mortality nor total cancer mortality varied significantly from the expected. There were eleven deaths due to pancreatic cancer, a statistically significant excess, but only three of the affected workers worked in transformer assembly. Five of the affected workers worked at the plant for less than one year, and another worked just two years. There were no liver cancers in the cohort.

This study was heavily criticized by Wong (1995) for methodological flaws. Moreover, the Manitoba Workers' Compensation Board awarded compensation to widows of two of the men who died of pancreatic cancer on the basis that the cancers were linked to mineral oil exposures. ATSDR (1997) concluded that the results of Yassi et al. (1994) "must be regarded as inconclusive due to limitations such as exposure to other chemicals and the fact that no medical history of the workers was provided."

Studies Cited as Linking Human PCB Exposure to Cancer

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Bahn et al. (1976, 1977) evaluated the incidence of tumors occurring in a New Jersey petrochemical facility where Aroclor 1254 had been used from 1949 to 1957. A significantly increased incidence of malignant melanomas was observed among research and development workers (2 of 31) and refinery personnel (1 of 41). In an update of that same study, NIOSH (1977) observed eight cancers in the total study population (5.7 expected). Three of these tumors were melanomas and two were pancreatic cancers. The incidence of these tumor types was reported to be significantly above calculated expectations, although no data were presented. The results of this study were confounded by the small cohort size, the fact that the workers in this facility were exposed to numerous other chemicals, and the fact that the expected cancer rates were based on U.S. population data rather than on local rates (Bahn et al. 1977; Lawrence 1977). ATSDR (1997) states that the findings of this study should be regarded as inconclusive.

Bertazzi et al. (1987) conducted a retrospective cancer mortality study of 544 male and 1,556 female workers who had been employed for at least one week in the manufacture of PCB-impregnated capacitors in an Italian plant between 1946 and 1978. Mortality was examined for that cohort from 1946 to 1982 and was compared to both national and local mortality rates. Mortality due to all cancers (14 observed vs. 5.5 national and 7.6 local) and due to cancer of the gastrointestinal tract (6 observed vs. 1.7 national and 2.2 local) was significantly increased among male workers. Death rates from hematologic neoplasms and from lung cancer were also elevated, but not significantly. Overall mortality was significantly increased above local rates (34 observed vs. 16.5 local) in the female

population. Total cancer deaths (12 observed vs. 5.3 local) and mortality from hematologic neoplasms (4 observed vs. 1.1 local) were also significantly elevated over local rates in the female population.

These results are limited by several factors, including the small number of cancer cases observed, the limited latency period, lack of pattern or trend when data were analyzed by duration of exposure, and some deaths in males with low potential for direct PCB exposure (ATSDR 1997; Kimbrough 1987). A major problem in the study design was the one week minimum period of employment required for inclusion in the study resulted in the inclusion in the cohort of workers who had no PCB exposure. This makes it difficult to assume that excess cancer cases are attributable to PCB exposures rather than to other factors. This study also did not show a dose-response relationship or any direct relationship between latency and the disease. Moreover, as discussed below, the results of this very small study are dissimilar from the results of much larger and statistically more valid studies of similar worker populations in the United States and Canada. ATSDR (1997) found that the results of this study were inconclusive.

Brown (1987) found an excess risk of cancer of the liver, biliary tract, or gall bladder in 2,588 workers (1,270 male, 1,318 female) from two capacitor factories. The workers had worked for at least three months in areas where they received heavy exposure to PCBs. Exposure was to Aroclors 1254, 1242 and 1016 (Lawton et al. 1981). The workers were also exposed to other chemicals, including trichloroethylene, toluene, and methyl isobutyl ketone.

The first evaluation of this cohort (Brown and Jones 1981) found increased cancer mortality that was not statistically significant. After an additional seven years of observation (Brown 1987), two additional cancers of the liver, gall bladder or biliary tract were observed, making the cancer increase in this combined cancer grouping significant. Among the grouped cancers, four of the five occurred in women from one of the plants. There was no increase in the number of rectal cancers from the previous study. For the total cohort, total mortality and cancer mortality were less than expected. Total cancer

among the cohort at one of the plants was significantly less than expected (18 observed versus 31 expected).

According to ATSDR, limitations and confounding factors in Brown (1987) include the small number of cases and the fact that PCB blood levels were higher in the plant with the lower incidence of cancer (ATSDR 1997). Moreover, the study failed to account for several factors particular to the plant where the increased cancer incidence was noted, including ethnicity (dominant Cape Verde background) and life style (the workers were from a harbor/fishing town where alcohol consumption and smoking behaviors are high). Furthermore, of the five liver grouping cancers, four of the workers had worked at the plant 1.5 years or less and the other worker worked at the plant less than 10 years. Finally, of the five cancers, only one was a primary liver tumor (the type of tumor predicted by animal studies) and at least one had metastasized from another site (and was therefore incorrectly identified as a liver tumor).

Sinks et al. (1991) conducted a retrospective cohort mortality analysis of 3,588 workers who were employed for at least one day at an electric capacitor manufacturing plant between 1957 and 1977. Aroclor 1242 was used in this plant through 1970, and Aroclor 1016 was used from 1970 to 1977. Mortality from all causes and from all cancers was less than expected. A significant increase in mortality rate was observed for skin cancer (8 observed vs. 2 expected) and death rates from brain and nervous system cancers were non-significantly elevated over expected rates. No excess deaths were observed from cancers of the rectum, lung, or liver, biliary tract and gall bladder, or from hematopoietic malignancies. Based on a cumulative dose estimate, which incorporated information on job station history, limited PCB environmental sampling data, and serologic data, the authors were not able to establish a clear relationship between latency or duration of employment and risk for malignant melanoma. Sinks et al. (1991) point out that the skin cancer excesses are not consistent with those of similar studies. The authors also point out that mortality may not be the best index of risk for malignant melanoma, as survival can be affected by differences in health care quality. In addition, other limitations include the lack of evaluation of exposures to other chemicals (metals, solvents, etc.), the relatively short

latency period, the small number of deaths within the cohort, and possible misclassification of brain cancer cases. Citing additional deficiencies in the study, ATSDR (1997) found the results of the study inconclusive.

Negative Studies

By contrast to the inconclusive and confounded results of these studies which are sometimes cited to link PCBs with cancer in humans, the largest study of PCB exposed workers (Taylor 1988) showed no significant increases in mortality or cancers. Taylor (1988) involved a cohort of 6,292 persons employed for at least three months during the period 1946-1976 at the GE Hudson Falls and Ft. Edward facilities. This study showed no increase in cancer mortality or in overall mortality compared to national averages. Deaths due to malignant melanoma, lymphopoietic cancers, or the combination of liver, gallbladder and biliary cancers were not significantly elevated, and brain cancers were well below the expected value. PCB exposure was shown to be negatively associated with (not statistically significant to) cancer mortality (all types combined) and lung cancer (the only cancer outcomes with numbers of cases sufficient to permit a regression analysis). In other words, as PCB exposure increased, the numbers of overall cancer deaths and lung cancer deaths decreased. 59

Recently, in a follow-up to Taylor (1988), a retrospective mortality study was conducted of the same cohort. (Kimbrough et al. 1997, unpublished). All workers were followed through the end of 1993. The cohort of 4062 white males and 3013 white females contributed 120,811 and 92,032 person years of observation, respectively. There were 763 (19%) deceased males and 432 (14%) deceased females. Death certificates were available for 98.5% of the decedents and only 1.3% of the cohort was lost to follow-up. For comparison, standardized mortality rates (SMRs) were calculated using both U.S. and local county mortality tables.

Overall mortality for the total cohort was significantly lower than that for the general population, as was the mortality for all cancers. The significantly lower SMRs in

the male workers is partly attributable to the low SMRs in the salaried male workers. The overall mortality and mortality due to all cancers of hourly workers was also significantly lower. The dramatically low SMRs in salaried male workers were not as evident in salaried female workers. However, 71% of the male salaried workers had obtained a college education reflecting a socioeconomic factor that is well correlated with decreased mortality (Sortie et al., 1995). Finally, there were no statistically significant increases in mortality due to any of the *a priori* cancer types. The study concluded that there was no evidence that PCB exposure at this plant had resulted in cancer mortality.

Additional recent studies undermine the often-cited link between PCBs and cancer. In Pittsfield, Massachusetts, PCBs were used in manufacturing over an extended period. The Massachusetts Department of Public Health (MDPH) recently issued a registry of the incidence of cancer mortality in Massachusetts from 1987 through 1994. The registry showed no statistically significant increases in cancer incidence (at any level of statistical significance) in Pittsfield or Berkshire County for any of the 23 types of cancers evaluated. MDPH (1997). In fact, the results for all cancer types showed that total cancer incidence in Pittsfield was 10 percent lower than expected based on the state-wide average, and also showed lower-than-expected total cancer rates for other towns in the area.[9] Last year, in what many in the scientific world describe as a definitive result, Hunter et al. (1997) published in the *New England Journal of Medicine* a study focused on the interaction of endocrine disruption and cancer. The study showed no link between PCB exposure and breast cancer. Similar results were reported by Key and Reeves (1994). As Dr. Steven Safe noted in an editorial accompanying Hunter et al. (1997), this study and others "should reassure the public that weakly estrogenic organochlorine compounds such as PCBs, DDT, and DDE are not a cause of breast cancer." (Safe 1997).

[9] Similarly, in a prior letter in 1980, the MDPH had advised the City of Pittsfield that review of the cancer mortality data for 1969-1978 showed no excess cancer mortality in Pittsfield across all causes of cancer, and further showed no excess cancer mortality in the Lakewood neighborhood adjacent to the GE facility. Parker (1980).

Conservatism in hazard identification is manifested when regulatory agencies place an emphasis on data that chemicals might pose adverse effects, and little weight on data that suggest that chemicals fail to cause adverse effects. Emphasizing study data that show adverse health effects in animals while virtually ignoring studies showing no adverse effects does not represent a balance of scientific information. (Nichols and Zeckhauser 1988) Frequently, extraordinary confidence is placed on a study that suggests that a chemical may pose a particular hazard, while only modest consideration is given to the study's quality.

More recently, the scientific community and some regulators have come to accept that not all scientific data are equal, and that only data of similar quality should be compared when drawing conclusions regarding toxic effects based on multiple studies. This philosophy, known as a "weight of evidence" approach, represents an important refinement that should be applicable to both hazard identification and dose response assessment (Sielken 1985; Anderson 1989; Gray et al. 1993). EPA's (1996b) proposed cancer risk guidelines also embrace this philosophy. The benefit of using a "weight-of-evidence" approach is that the results of several high quality toxicity studies will not be disregarded simply because the results of one or two poorly controlled studies have dissimilar findings.

As is clear from the discussion of the PCB epidemiology studies, none of the cancer incidence and mortality studies demonstrates a cause-effect relationship between PCB exposure and cancer.[10] Not only do the individual studies fail to show causation, but the weight of the evidence from the studies taken collectively also fails to establish any such relationship.

[10] It is acknowledged that Rothman et al. (1997) observed a dose-response relationship between PCB serum levels and non-Hodgkin lymphoma; however, the authors pointed out that their "results should be regarded as hypothesis generating," that "our findings require replication, and biological plausibility," and that the matter "needs further investigation." The authors also noted that studies of highly exposed capacitor workers do not support a relationship between non-Hodgkin lymphoma and PCB exposure.

As discussed previously, the scientific convention applied in weight-of-the-evidence evaluation of epidemiological studies requires (a) the observation of a specific cancer endpoint, and (b) the meeting of other criteria (strength of association, consistency of association, dose-response relationship, temporally correct association, specificity of the association, and coherence with existing information (biological plausibility)) before a causal relationship between an agent such as PCBs and cancer can be inferred. None of the criteria, with the exception of temporality, should be considered as necessary to establish causation. Each of the criteria is important, and causation is established by the weight of the evidence and the degree to which all six criteria are satisfied by the available data. However, the rejection of the association may be made with a high degree of confidence when three of the criteria -- temporality, consistency, and coherence with existing information -- are not met. (Rothman 1988; EPA 1996b) In addition to considering weight of the evidence, it is important to understand that studies with larger cohorts and numbers of cancer deaths are inherently more important when considering the weight of the evidence than are studies with smaller cohorts and fewer cancer deaths.

In the PCB studies, small increases in a variety of cancer endpoints were seen in different populations with no common thread, and several studied populations showed no increases at all. The discrepancies can be explained in innumerable ways, including exposures to other chemicals, population life styles, and even chance. Thus, little evidence exists that PCBs are human carcinogens, and the weight of the evidence fails to establish a definitive causal relationship between exposure to PCBs -- even in high concentrations -- and the incidence of cancer in humans.

In 1993, TERRA, Inc. submitted comments on the Great Lakes Initiative that provided a thorough weight-of-the-evidence assessment of what the authors classified as the four "major" cohorts in the PCB epidemiological studies (the Brown, Nicholson, Sinks and Taylor cohorts (TERRA, 1993). GE incorporates these comments by reference. The following tables from that document provide a summary of TERRA's analysis.

Summary of The Major PCB Mortality Studies

Study/Cohort	Were Significant Increases Noted?			
	SMR for All Cancer Deaths	Total Cancers	Malignant Melanoma	Liver/Biliary Cancers
The Brown Cohort				
Brown and Jones 1981 Mortality study of 163 deaths among 2,567 U.S. capacitor workers	89	No	No	No
Brown 1987 Updated mortality of Brown and Jones 1981 analyzing 295 deaths among 2,588 workers	78	No	No	Yes (but limited to females)
Nicholson et al. 1987 Mortality study of 188 deaths among 769 U.S. capacitor workers with ³5 years exposure and ³10 years latency since first exposure	79	No	No	No
Sinks et al. 1992 Mortality study of 192 deaths among 3,588 U.S. capacitor workers	85	No	Yes (but limited to males)	No
Taylor et al. 1988 Mortality study of 510 deaths among 6,292 capacitor workers	95	No	No	No

Evaluation of the Major Cohorts Using Causation Criteria

	Was This Criteria Satisfied?	
Causation Criteria	Liver	Skin
1. Strength of the Association	No*	No*
2. Consistency of the Association	No	No
3. Temporal Relationships	No	No
4. Dose-Response Relationships	No	No
5. Specificity of Association	No	No
6. Coherence of Evidence	No	No

* Denotes a statistical observation that is considered weak because it is confounded by a lack of confirmation in studies of equivalent or greater size, and it lacks confirmation in a study using greater exposure duration and latency criteria for cohort definition.

From its analysis, TERRA concluded that "the available scientific evidence do not support the contention that PCBs are carcinogenic in humans."

Other scientists have reached similar conclusions. For example, Chase et al. (1989), concluded that:

> *There is insufficient evidence to show a causal relationship between PCB exposure and the subsequent development of any form of cancer. In light of the long-term and widespread usage of PCBs in the workplace and, in some cases, the extensive exposures of workers, it is likely that evidence of carcinogenicity in humans would have been observed in the various epidemiological studies discussed above if PCBs were in fact potent carcinogens.*

Similarly, Kimbrough 1988 concluded that:

> *Thus far, no conclusive adverse effects have been demonstrated in people who carry body burdens of PCBs from environmental exposure to trace amounts of*

PCBs... Even workers with exposures two orders of magnitude greater than environmental exposures show no convincing health effects... Thus, despite positive laboratory animal data and except for chloracne, exposure to PCBs has led to no convincing, clinically demonstrable, chronic health effects in humans.

In her 1995 update, Dr. Kimbrough reaches a similar conclusion (Kimbrough 1995).

A recent review of the occupational studies by the American Council on Health and Science also concluded that none of the studies provides evidence that PCB exposure increases cancer risk in humans (Danse et al. 1997). A recent review of studies seeking to determine if there was a relationship between environmental exposures to PCBs and any human health effects, including cancer, found that "none of the 33 studies where exposure had occurred in the natural environment provided positive or suggestive evidence of an association with adverse effect." Swanson et al. (1995).

A fair and careful review of the existing PCB occupational studies leads to the conclusion that there is no credible evidence that PCBs cause cancer in humans, even at exposures that are orders of magnitude greater than environmental exposures. Therefore, GE urges EPA to reassess the human carcinogenicity of PCBs in light of the epidemiological studies.

B.3 Derivation of a cancer slope factor for PCBs from the epidemiological studies

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Although the weight of the evidence approach results in the conclusion that there is no credible evidence that PCBs cause cancer in humans, it is still possible to derive a cancer slope factor from the epidemiological studies. In TERRA (1993), the authors derived such slope factors using two approaches.

First, the authors assumed that the results of Brown (1987) showing a statistically significant increase in combined liver and biliary cancers reflected a real measure of cancer potency. The authors then used the observed increase in cancer incidence, along with a

conservative estimate of exposure, to generate a human cancer potency factor. That is, it was assumed that the results of Brown (1987) were representative of human cancer risk, even though other studies of comparable or greater size had failed to duplicate the findings of liver cancer.

Second, the authors used the negative results of the largest study completed at that time (Taylor 1988), along with a conservative estimate of exposure, to calculate an upper confidence on the measured zero risk, thereby placing an upper bound on the risk.

The authors' methodology for estimating exposure and calculating cancer slope factors are described in detail in their paper. It is important to note that the authors estimated exposure using two different methods: estimating the daily dose capacitor workers received using reported workplace exposure estimates and known or estimated absorption; and estimating daily doses received by the capacitor workers using basic pharmacokinetic principles and reported body burdens and estimated tissue half-lives. In both methods, the authors used conservative assumptions to assure that there was little possibility that dose would be overestimated. As one example, when assessing exposure through the inhalation route, the authors used the geometric mean of work place air concentrations measured in the mid- to late 1970s, when PCB use was being phased out. The two methods of estimating exposure arrived at very similar dose estimates.

To be conservative in calculating cancer slope factors from the dose estimates and the results of Brown (1987) and Taylor (1988), and in recognition that there is some degree of uncertainty in the incremental risk rates that were calculated, the authors calculated cancer slope factors using both the measured cancer incidence rate and the 95% upper confidence limit on the incremental risk rate. The calculated cancer slope factors are as follows:

Cancer Slope Factors

Study/Method	Cancer Slope Factor ($mg/kg/day^{-1}$)
Brown (1987) – Measured	5.9×10^{-3}
Brown (1987) - 95% UCL	1.9×10^{-2}
Taylor (1988) – Measured	7.7×10^{-4}
Taylor (1988) - 95% UCL	8.9×10^{-3}

For the reasons discussed throughout these comments, GE believes that cancer slope factors calculated from epidemiological studies can be used to establish environmental standards, including a water quality standard for PCBs. Given that Taylor (1988) is the largest epidemiological study performed to date and is highly relevant to the Hudson River, GE recommends using the measured cancer slope factor from this study as the starting point for establishing environmental standards for PCBs based on cancer risk. As discussed above, the workers studied in Taylor (1988) were exposed primarily to Aroclor 1242 and 1254, with minor exposure to Aroclor 1016. Thus, it is conservative to use the measured cancer slope factor from Taylor (1988) as the cancer slope factor for Aroclor 1242.

Accordingly, General Electric proposes that the SOW for the Hudson River Phase 2 Risk Assessment use a CSF of 7.7×10^{-4} $(mg/kg/day)^{-1}$.

B.4 Summary for cancer toxicity assessment

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Although the Reassessment was a positive step in reevaluating cancer risk from exposure to PCBs, GE strongly believes that EPA should use the numerous epidemiological studies that have been performed to date to further assess the true human cancer risk of PCBs. EPA can no longer ignore the many clinical and epidemiological studies that do not support the proposition that PCBs cause cancer in humans. Although

toxicologists must be careful in relying on the results of negative epidemiological studies, when several excellent epidemiological studies have been performed using large numbers of workers heavily exposed to a chemical over a long period of time, and the results of those studies have been negative, such results cannot be ignored.

These comments provide a scientifically valid rationale for using epidemiological studies rather than rodent studies to establish environmental standards for PCBs. They also provide an assessment of the adequacy of the PCB epidemiological studies for evaluating the human cancer risk of PCBs and set forth overall conclusions that can be drawn from those studies using a "weight of the evidence" approach. These comments have further shown how the studies can be used to derive a conservative cancer slope factor for PCBs. GE strongly urges the Agency to use the opportunity presented by the HHRA to consider this matter more thoroughly.

C. Noncancer toxicity values

The noncancer reference dose (RfD) cited in the SOW is flawed and overly conservative, in that it is based on an inappropriate monkey study and overly conservative uncertainty factors. In the SOW, EPA plans to base the noncancer risk assessment using the oral RfD of 2×10^{-5} mg/kg-day (20 ng/kg-day) for Aroclor 1254. This RfD is based on dermal, ocular and immunologic effects in a series of studies of rhesus monkeys reported by Arnold et al. (1993a,b) and Tryphonas et al. (1989; 1991a,b). 63

The Aroclor 1254 reference dose (RfD) is based on the results of a five year feeding study in rhesus monkeys (Arnold et al., 1993a,b; Tryphonas et al., 1989; Tryphonas et al., 1991a,b). Groups of 16 adult female monkeys ingested gelatin capsules containing Aroclor 1254 (in glycerol, corn oil vehicle) at daily doses of 0, 5, 20, 40, or 80 ug/kg-day for over five years. PCB concentrations in the monkeys had achieved steady state pharmacokinetics by 25 months of exposure, as demonstrated by PCB measurements in blood and adipose tissue (Tryphonas et al., 1989; Mes et al., 1989). The general health and clinical pathology findings in the adult female monkeys dosed with Aroclor 1254 were 64

reported by Arnold et al. (1993a,b). Clinical signs of toxicity were limited to eye exudate, inflammation, and/or prominence of the tarsal (Meibomian) glands, and changes in finger and toe nails. Significant dose related trends were reported for these clinical signs (Arnold et al., 1993a). The results of an immunologic assessment of the PCB exposed adult female monkeys was reported by Tryphonas et al. (1989, 1991a,b). The most significant finding was a treatment-related decrease in antibody response (IgG, IgM) to sheep red blood cells (SRBC). The LOAEL for clinical signs and immune system effects was 5×10^{-3} mg/kg-day. A total UF of 300 was applied (ten for sensitive individuals, three for interspecies extrapolation, three for minimal effect LOAEL to NOAEL, three for subchronic to chronic study duration) and a RfD of 2×10^{-5} mg/kg-day was calculated.

A number of questions can be raised about EPA's selection and evaluation of the studies from which the Aroclor 1254 RfD was derived. Two critical shortcomings are most important. First, those studies are not appropriate for deriving an RfD because: (a) the clinical relevance of the immunologic changes reported by the researchers has not been demonstrated; (b) the rhesus monkey is not an appropriate model for dermal, ocular, and nail effects of PCBs in humans; and (c) there is compelling evidence to indicate that rhesus monkeys metabolize PCBs in a significantly different way from humans. Second, EPA used inappropriate and overly conservative uncertainty factors in extrapolating from this set of studies to derive an RfD for humans. These points are explained in greater detail in a memorandum prepared by Dr. Russell Keenan and Ms. Carol Gillis, then at ChemRisk, which was attached as Exhibit 96 to GE's May 1, 1998 comments on EPA's proposal to list the Housatonic River site on the National Priorities List (GE, 1998) and is incorporated by reference herein. 65

Despite changes in immunologic parameters reported by the researchers, clinical relevance of these changes has not been demonstrated. In addition, the rhesus monkey is not an appropriate model for dermal, ocular and nail effects of PCBs in humans. A comparison of effects and body burdens (blood serum levels) seen in workers exposed to PCBs and the effects and associated body burdens reported in rhesus monkey studies indicates that PCBs produce nail changes, ocular effects, and dermal effects at much lower 66

doses in rhesus monkeys than in humans (Gillis and Price, 1996). In fact, PCBs alone do not produce nail changes, chloracne or ocular effects in humans as demonstrated by the fact that the length of exposures in epidemiological studies would have provided adequate opportunity to demonstrate these effects if they were occurring. These findings suggest that rhesus monkeys are significantly more sensitive and respond differently to PCBs than humans.

More importantly, there is compelling evidence to indicate that PCBs are 67
metabolized differently in humans than in rhesus monkeys and that the metabolism of PCBs may be critical to the overall expression of PCB toxicity (Brown, 1994; Brown et al., 1994). One indication of this difference is the line of evidence suggesting that the patterns of PCB congeners that accumulate in adipose and hepatic tissues of rhesus monkeys chronically exposed to Aroclor 1254 differ from patterns of congener retention in humans exposed to PCBs. Humans produce a retention pattern similar to that observed in *in vitro* studies of P4502B enzyme activity, referred to as P4502B-like metabolism (Brown et al., 1989; 1994). Humans produce a second pattern when exposed to mixtures of PCBs and furans (Masuda et al., 1978; Kunita et al., 1984). This pattern results from metabolism of PCBs by a combination of P4502B-like and P4501A-mediated metabolic pathways. It appears that, in the absence of concurrent exposures to dioxins and furans, PCBs do not induce the P4501A enzymes in humans. Furthermore, studies of PCB induction of P4501A in rodents suggest that such induction, if it occurs in humans, would require exposures of PCBs far higher than have occurred from environmental or historical occupational exposures (Brown et al., 1991).

In contrast to the metabolism of PCBs in humans (in the absence of concurrent 68
exposures to dioxins and furans in the toxic range), a different pattern is observed in rhesus monkeys. Metabolism patterns of PCBs in rhesus monkeys indicate that PCBs are metabolized by means of the P4501A pathway and a second pathway known as the P450RH pathway, which appears to be unique to the rhesus monkey (Brown, 1994). The specific enzymes responsible for metabolizing PCBs in the unusual P450RH pattern observed in monkeys are unclear at this time. However, the differences in enzyme systems

support the finding that PCBs are metabolized differently in rhesus monkeys and humans, and suggest that the rhesus monkey is a poor model for endpoints associated with the activation of P4501A.

Several studies have demonstrated that PCB metabolism is critical to the expression of PCB toxicity in humans (Brown, 1994; Brown et al., 1989; 1991; 1994). For example, induction of P4501A at low PCB doses is associated with dermal, ocular, and nail effects in animals (Brown et al., 1994). In humans, Yusho victims, who were exposed to both PCBs and furans and experienced many of these effects, also displayed P4501A metabolism. Conversely, metabolism of PCBs under the P4502B-like pathway in occupationally exposed human populations is not associated with these effects. In summary, the findings on the metabolism of PCBs suggest that the differences between rhesus monkeys and humans with respect to PCB toxicology may extend beyond dermal, ocular, nail and immunological effects.

In addition to EPA's improper selection of the critical study and toxicological endpoints for establishing the RfD, EPA applied inappropriate uncertainty factors for quantitatively deriving the RfD. According to EPA, an uncertainty factor of three was applied to account for interspecies extrapolation due to the similarities in toxic responses and metabolism of PCBs in monkeys and humans and the general physiologic similarities between the species. This uncertainty factor implies that humans are three times more sensitive than the rhesus monkey for the critical effects on which the RfD is based. However, because the evidence indicates that the rhesus monkey is significantly more sensitive than humans, EPA's uncertainty factor of three to account for interspecies sensitivity is inappropriate. An uncertainty factor equal to or less than one would be more appropriate to address interspecies uncertainty. 69

In addition, EPA applied an uncertainty factor of three to adjust for study duration. This factor is intended to account for uncertainties related to less-than-chronic exposure and the assumption that a longer exposure duration may result in more pronounced adverse effects as adverse effects at a low dose. In the case of Aroclor 1254, the monkeys were 70

dosed for greater than 25 percent of their lifetime and steady-state PCB body burdens were achieved (Arnold et al., 1993a,b). This suggests that a longer exposure duration would not result in an increased toxic response. Thus, an uncertainty factor of three is not warranted and should be reduced to a factor of one. In summary, reduction of the uncertainty factors for interspecies sensitivity and study duration from a value of three to one results in a total uncertainty factor of 30, rather than 300. Even if one were to accept EPA's selection of the toxicological study and agree with EPA's evaluation of the critical effects, the necessary adjustments to the total uncertainty factor would argue for a revised chronic RfD no more stringent than 200 ng/kg-day.

D. Endocrine disruption

The SOW currently plans to evaluate "the potential for endocrine effects" in the HHRA. GE disagrees with the need for conducting this evaluation, even if it is limited to "a qualitative assessment of the currently available information on the potential effects of PCBs on the endocrine system," as can be inferred from the SOW. We believe this analysis is unwarranted in light of recent EPA findings on this topic as well as those of independent scientific researchers. For example, it has been suggested that PCBs can interfere with normal endocrine function leading to infertility and other hormone related disorders, although recent reviews suggest that the evidence for these effects is weak and circumstantial (Danse et al., 1997; Golden et al., 1998). Indeed, EPA (1997a) concluded that, with few exceptions (for compounds unrelated to PCBs), "an adverse health effect in humans via endocrine disruption has not been established." 71

Last year, in what many in the scientific world describe as a definitive result, Hunter et al. (1997) published in the *New England Journal of Medicine* a study focused on the interaction of endocrine disruption and cancer. The study showed no link between PCB exposure and breast cancer. Similar results were reported by Key and Reeves (1994). As Dr. Steven Safe noted in an editorial accompanying Hunter et al. (1997), this study and others "should reassure the public that weakly estrogenic organochlorine compounds such as PCBs, DDT, and DDE are not a cause of breast cancer." (Safe 1997).

GE believes that further qualitative or quantitative evaluation of this topic is not necessary or appropriate in the Hudson River HHRA. As EPA and independent scientific researchers have been unable to find a link between PCB exposure and endocrine disruption, it is not worthwhile to invest the level of effort and resources necessary to further elucidate this topic in the context of a Superfund site risk assessment. Furthermore, by raising it as a potential issue in the Hudson River HHRA, which is itself based on a hypothetical exposure scenario, undue alarm or concern may be generated. This would not serve the public interest.

E. Uncertainty in toxicological criteria

The SOW states the Agency does not intend to evaluate the uncertainty in the dose response criteria (the RfD and the CSF) in the human health assessment (SOW, at 14). The basis for this approach is purportedly consistent "with EPA's policies (EPA, 1997a,b)." These documents are not a statement of the limits of Agency policy. Rather they are a set of guiding principles for the use and evaluation of Monte Carlo modeling. The relevant passage in the documents is as follows: 72

> *For human health risk assessments, the application of Monte Carlo, and other probabilistic techniques has been limited to exposure assessments in the majority of cases. The current policy, Conditions for Acceptance and associated guiding principles are not intended to apply to dose response evaluations for human health risk assessments until this application of probabilistic analysis has been studied further.* (EPA 1997a, at 2)

It is clear that the EPA's current policy is limited to the evaluation of exposure assessments in human health risk assessments, and that the Agency does not provide the assessor with guidance for the evaluation of Monte Carlo assessments of dose response (toxicity).[11] This is not the same as a policy that forbids the consideration of quantitative

[11] See the identical language in U.S.EPA's Guiding Principals for Monte Carlo Analysis, (EPA, 1997b).

modeling of the uncertainty in dose response. It merely is a statement that the current policy does not address this type of analysis and that the Agency is not offering guidance on the evaluation of this practice.

Therefore, there is no basis for concluding that the uncertainty in toxicity measurements can be set aside or ignored in the evaluation of the uncertainty in risk estimates. GE is unaware of any legal or technical reason to exclude the uncertainty in toxicity criteria. In fact, the Agency's own technical experts have advocated that the Agency consider the uncertainty in toxicity on a case-by-case basis (SAP, 1998).

GE believes that it is critical to account for the uncertainty in toxicity measurements. It has long been recognized that the toxicity portion of the risk assessment, not the exposure assessment, is the greatest source of uncertainty in risk assessment (McKone and Bogan, 1991). Thus, the decision to exclude consideration of such uncertainty has grave impacts on the assessment of risks.

Given the importance of this issue, the only basis for excluding consideration of uncertainty from the HRA is technical infeasibility. However, a number of technical approaches have been suggested for characterizing uncertainty in toxicity criteria. The uncertainty in cancer slope factors has been investigated by several authors (Crouch et al. 1996; Evans et al. 1996a,b). Uncertainty in the RfD has also be the subject of a number of research publications (Slob, 1997; Baird et al. 1996; Price et al., 1997, and Swartout et al. 1998). Swartout et al. (1998) has established a framework for redefining the RfD in probabilistic terms.

Techniques for the integration of uncertainty in toxicity assessments into assessment of exposure uncertainty have also been developed (Carlson-Lynch et al. 1997; Harvey, et al. 1997; Price et al. 1998). In Price et al. (1998), a Monte Carlo model was constructed of the uncertainty and variation in the PCB dose rates of anglers consuming fish from the Tennessee and Clinch Rivers. This model was combined with information on the uncertainty in the toxicity of PCBs to estimate the probability of exceeding the actual

dose that is "protective of sensitive individuals". The result is an assessment that fully discloses the uncertainty in the risk characterization for PCB noncarcinogenic effects and provides the risk manager with the most appropriate basis for decision making.

As discussed elsewhere, the Agency's approach for Monte Carlo analysis is not entirely clear, however, at several points the Agency has indicated that uncertainty will be explicitly modeled (SOW, at 10, 13). Therefore, there appears to be no technical reason why information on uncertainty on the PCB toxicity criteria cannot be evaluated with the uncertainty in exposure.

F. Averaging time

The SOW does not discuss the issue of averaging time. Averaging time is a term in 73
the equation to estimate the average daily dose rate that is used to evaluate non-carcinogenic effects (EPA, 1989). When the dose rate received from a source of contamination is constant over time, the issue of averaging time does not affect the estimate of average daily dose. However, when the dose rate changes over time, the choice of the duration for the averaging time can have a dramatic impact on the estimate of daily dose (Muir et al. 1998). Since the proposed model will address changes in fish tissue concentrations over time, the selection of an averaging time is an important issue for the HHRA.

GE recommends that the averaging time be established based on the half-life of PCBs in humans. Lipophilic compounds exert their chronic effect as a function of the long-term body burden of the compounds. Studies of PCBs in test animals determine doses that are approximately in equilibrium with the body burdens that are associated with the presence or absence of long tem effects. Therefore, it is important that the averaging period is sufficiently long that the dose can come into some sort of equilibrium with the body burdens of the anglers. This suggests that the averaging time be several multiples of the half-life of PCBs in humans. At a minimum, the averaging time should be greater than 10 years.

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SECTION V
RISK CHARACTERIZATION ISSUES

A. Consideration of background sources of PCBs

PCBs were used in consumer and commercial products from the 1940s through the 1970s. Because of this widespread use and the persistence of the compounds, PCBs still occur at trace levels in many foods and in many households. As a result, all individuals carry trace levels of PCBs in their bodies (ATSDR, 1997).

One criteria in risk management is to determine how exposures from a particular source relate to the background levels and whether exposure from a certain source will significantly raise an individual's body burdens. For example, if an individual has 2 ppb of a contaminant in his or her blood because of background sources, and exposure at a Superfund Site raises the blood level by 0.01 ppb, then there is likely to be little health benefit to the individual from the control of contamination at the Site. If the exposures at the site doubles or triples the individual's body burden then the potential for causing adverse effects is much higher and the control of the site may be warranted.

The impact of a source on background body burdens can be investigated directly by surveying the blood levels of individuals exposed by certain routes and comparing the 74
results to levels in unexposed populations. Such studies have been performed for exposure to PCBs in soil (Chase et al., 1989; ATSDR, 1987) and exposure from the consumption of fish in the Great Lakes, Tennessee River (ATSDR, 1997b), and in Western Massachusetts (Housatonic River). In the latter two studies, the individuals who consumed fish did not have elevated blood levels of PCBs.

Such direct studies cannot be performed for the Hudson River since the ban on keeping fish has eliminated exposures from fish consumption. However, it is possible to 75
take the output of the exposure models proposed in the SOW and determine the incremental change that consumption would cause in background levels of PCB

(Avantaggio et al. 1997; Keenan et al. 1997). Such an analysis would provide EPA with information on whether the control of PCBs from the consumption of fish would have a significant impact on angler's body burdens.

B. Development of the central and high-end exposure risk estimates

The Agency has indicated that risk management decisions will be made based on the current and future risks to average exposed individual and to the reasonably maximally exposed individual (RMEI) (SOW, at 2). These two risk measurements are proposed to be based on the exposure assessment that consists of a deterministic and probabilistic analysis (SOW, at 6). Thus, EPA is proposing to use two estimates of risk for the average individual and RMEI. The SOW does not clearly state how the two estimates will be used in the risk characterization process. 76

Deterministic assessments have been historically used at Superfund sites to consider the need to perform remediation and to select a remedial option. At most small sites the deterministic baseline risk assessment would be representative of the site risks. For larger and more complex sites with dynamic physical features (like the Hudson River), a deterministic model is inadequate even to describe "typical" conditions. Probabilistic models, such as Monte Carlo models, are more appropriate in such cases since they have a better chance to capture the variability and the uncertainty which is inherent as sites become more complex.

Consequently, EPA should abandon its proposal to use the findings of the deterministic exposure assessment. As discussed in EPA's Guiding Principles on Monte Carlo (EPA, 1997), the use of probabilistic techniques provides the decision maker with additional insights that the deterministic methods cannot provide. Therefore, the assessment of risks to the RMEI and average individual should be based only on the findings of the probabilistic analysis. 77

C. The baseline assessment cannot be used to select remedial options.

The baseline risk assessment cannot be used to assess the reduction of risk associated with the implementation of a remedial strategy. Rather, the baseline HHRA examines the hypothetical risk associated with the "no action" scenario. These risks must be measured against the estimated risks that might remain after implementation of various remedial options in order to assess the appropriateness of such actions. Therefore, a separate risk reduction analysis should be completed for each remedial option to focus on the net reduction in risks, if any, associated with the different remedial options. 78

D. Use of risk finding for small number of anglers fishing hot spots

The SOW describes the assessments of risks associated with fish caught in hot spots as one of the analyses that will be performed (SOW, at 13). However, the procedure to evaluate the results of the risk assessment — i.e., how it will be used in the risk management and feasibility study remedial alternatives assessment — is not presented in the SOW. One area of possible misinterpretation is the extreme upper limit risks that will likely be calculated for the localized areas of elevated contamination. It is important that these risks be put into proper context in the overall risk. This assessment should include a discussion of the areal extent of the elevated concentrations, probability of a suitable fishery, probability of anglers in the same area, angler success, and other relevant factors. 79

E. Choice of "start date"

According to the SOW, the baseline risk assessment will focus on a population of anglers who begin fishing in 1999 (SOW, at 11). The selection of this date may not be appropriate for the baseline assessment. As discussed above, a separate risk assessment will be needed to evaluate the risks that might remain after various remedial options. These risks will not exist until the remediation has been completed. Given the current schedule for the Hudson River reassessment, this could happen no earlier than 2002 to 2005. The data used in the remedial decision-making process should represent the 80

conditions that best represent the risks beginning at that time. In order to be consistent with the remedial assessment, the Agency should start the baseline risk assessment clock beginning in 2002 not 1999.

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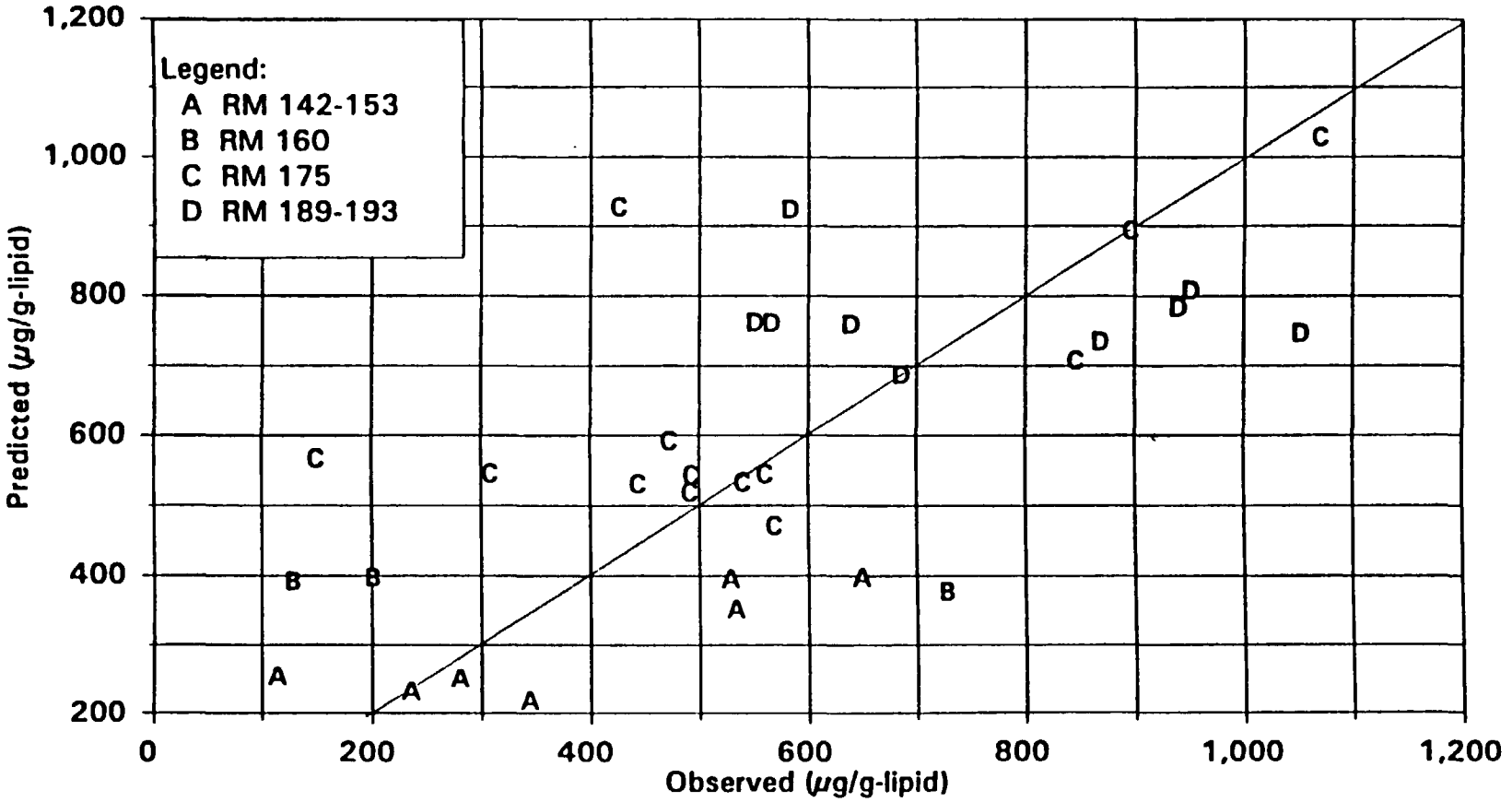
Figure 9-12
Comparison of Observed and Predicted Aroclor 1254 Concentrations in
Hudson River Largemouth Bass (Corrected to NYSDEC 1983 Quantitation Basis)



Source: TAMS/Gradient Database, Release 3.1

300953

Figure 9-12 from EPA, 1996. Preliminary Model Calibration Report.

NYSDEC RIVER ENFORCEMENT SUMMARY OF THE CATCH AND RELEASE FISHING PROGRAM (8/31/95 - 7/31/98)

VIOLATION TYPE	TICKETS ISSUED	WARNINGS ISSUED	TOTAL
KEEPING FISH	9	3	12
BAIT FISH/TIP-UPS	5	74	79
NO LICENSE	72	93	165
NAVIGATIONAL/ OTHER	40	28	68
TOTALS	126	198	324
TOTAL FISHERMEN CHECKED TO DATE	1437		

08/10/98