36738

51A

COMMENTS OF THE GENERAL ELECTRIC COMPANY

ON THE

AUGUST 1991 REVIEW COPY

OF THE

PHASE 1 REPORT - INTERIM CHARACTERIZATION AND EVALUATION

FOR THE

HUDSON RIVER PCB REASSESSMENT RI/FS

October 24, 1991

COMMENTS OF THE GENERAL ELECTRIC COMPANY ON EPA'S PHASE 1 REPORT

TABLE OF CONTENTS

1.0		1
	1.1 Background	2
	1.2 Overview	<u> </u>
	1 2 1 No linaccentable Pisk	7
		4
	1.2.2 NO New Dieuging Technology	b
	1.2.3 Dreaging will cause Adverse Environmental	
	EIIECTS	7
	1.2.4 An Integrated, Quantitative Approach Shows	
	No Significant Benefit from Dredging	9
-	1.2.5 Biodegradation Is Effective on PCBs 1	0
	1.2.6 Other PCB Sources 1	1
	1.3 Required Actions	3
		-
2.0	OUANTITATIVE MODELING	5
2.0	2 1 The Quantitative Modeling Annroach	-
	2.2 The Manuffactive Modeling Apploach	/
	2.2 The Need for a Quantitative Model of PCB	~
	Fate and Transport in the Hudson River 1	9
	2.2.1 Modeling of Sediment Transport	4
	2.2.2 Modeling of PCB Interactions 2	5
	2.2.3 Modeling of PCBs in Fish	8
	2.3 Sediment Transport	3
	2.3.1 Flood Frequency Analysis	3
	2.3.2 Suspended Sediment Analysis	5
	2.3.3 Sediment Transport Modeling	7
	2.3.4 Additional Data Requirements	ั้ว
	2 A Other Modeling Issues	د ۸
	2.4 Dediny issues	44: ∕a
	2.4.1 Radionacing Dating of Sediment Cores 4	4
	2.4.2 Upstream PCB Source 4	6
	2.4.3 The Effect of Floods on Fish	
	Concentrations 4	7
	2.5 List of References 4	9
3.0	RISK ASSESSMENT	2
	3.1 Current Trends in Hudson River Data	
	All Indicate A Reduced Risk Since 1984	2
	3.1.1 PCB Concentrations in the Water Column 5	3
	3.1.2 PCB Concentrations in Sediments	л Л
	3 1 3 BCB Concentrations in Fich	ч ,
	3 1 4 Journ Diver DCD Concentrations	4 E
	2.2 Under River PCB Concentrations	2
	3.2 Human Health Risk Assessment 5	7
	3.2.1 EPA's Assumption About the Toxicity	
	of PCBs is Incorrect 5	8
	3.2.2 Current or Future Exposures to Hudson River	
	PCBs Will Not Present Unacceptable Risks 10	0

HRP 001 1451

Page

i

Page

		3.2.3	Reass	essme	nt o	f Ri	isks	As	soci	ate	ed 1	√it	h				
			PCBs :	In Hu	dson	Riv	/er	Sed	ımen	ts	•	• •	٠	٠	٠	٠	127
	3.3	Ecolog:	ical R	isk A	sses	smer	nt	• •	• •	•	•	• •	•	•	٠	•	135
		3.3.1	A Syst	tems .	Appr	oach	n Is	The	e Mo	st	Apj	pro	pr:	iat	e		
			One .	• • •	• •	• •	•	• •	• •	•	•	• •	•	•	٠	•	136
		3.3.2	No Imp	pairm	ent	to t	:he	Eco	syst	em	Fre	om	the	9			
			Prese	nce o	f PC	Bs .	•	• •	• •	•	•	•	٠	•	٠	•	137
		3.3.3	Method	lolog	ical	and	l An	aly	tica	1 I	Flay	1S	in				
			The Pl	nase	1 Ec	0100	gica	1 A:	sses	sme	ent	•	•	•	•	•	139
		3.3.4	Insufa	ficie	nt D	ata	is	Pres	sent	ed	to	Al	101	N			
			Evalua	ation	of	Eco]	logi	cal	Imp	act	s I	Dur	ing	3			
			the Re	emedi	al S	elec	ctīc	n P	roce	SS	•		•	•	•	•	142
		3.3.5	PCB Ex	xposu	re A	sses	ssme	nt	•. •		•		•	•	•	•	144
		3.3.6	There	is N	o Va	lid	Sci	ent	ific	Ba	asis	5 f	or	th	ie		
			"Risk	Char	acte	riza	atic	n"	Pres	ent	.ed	in	tł	ne			
			Phase	1 Ec	olog	ical	As	ses	smen	t							161
		3.3.7	List (of Re	fere	nces	5.							-			189
								•••	• •	•	•		•			•	
0	REMO	VAL TECI	HNOLOG	IES .	• •	• •	•	• •	• •	•	• •	• •	•	•	•	•	199,
	4.1	Introdu	uction	• •	• •			• •	· · ·		•	•		•		•	199
	4.2	Site-S	pecific	c Imp	edim	ents	s to	Dr	edgi	ng	•		•	•	•		202
	4.3	Dredgi	ng Tecl	hnolo	gies		• • •		• •	-	•		•	•	•	•	205
		4.3.1	Hydrau	ulic	Dred	gind	۲.	• •		•.	•		•	· .	•	•	209
		4.3.2	Mechai	nical	Dre	dgir	ng	• •	• •		•			•	• .		219
		4.3.3	Specia	altv	Dred	qind	.	• •			•			•			228
		4.3.4	Field	Stud	ies							•	•		•		229
		4.3.5	Insig	nific	ant	Char	naes	in	Tec	hno		v	Si	nce	2		
			1984								•		•				232
		4.3.6	Conclu	usion											·		233
	4.4	Adverse	e Envi	ronme	ntal	Eff	fect	s o	fDr	edo	rind	т.					233
		4.4.1	Effect	ts of	בבים בי תוודי	bidi	itv	and	Res	usr	hen	sio	n			•	234
		4.4.2	Increa	ased	Bioa	vail	lahi	1i+	v of	P	Bs						237
		A A 3	Deetri	uctio	$\frac{1}{n}$ of	Dit	107	Hah	itat			•	•	•	•	•	
			and R	anthi			, i + i	DC.	TCUU	.0							238
				Lorm			11 C I	rff.	· ·	•	•	•••	•	•	•	•	240
		4.4.4	Ding-	Term Free	1001	.0910	Dor		eu La Filon	•	•	•••	•	•	•	•	240
		4.4.5	Nevie	ELUS		anu	Der 	051	c t On	•	•	• •	•	•	•	•	241
		4.4.0	Naviga	acion	ai 1 Tw	mpac	508	• •	• •	•	•	• •	•	•	•	•	240
		4.4./	Aestn	FLTC	Twbg		n	•••	• •	•	•	• •	•	•	•	•	24/
		4.4.8	nealti	n and	Sai	ety	KIS	5KS	• •	•	•	• •	•	٠	•	•	248
	4.5	Utner (conceri	ns .	• •	•	• •	• •	• •	•	•	• •	•	٠	٠	٠	249
	4.6	conclu	510N	• • •	• •	• •	•	• •	• •	٠	•	• •	•	•	•	•	250
	4.7	LIST O	r Keiel	rence	s.	• •	• •	••	• •	٠	•	• •	•	•	•	٠	251

4.

HRP 001

1452

ii

Page

5.0	IN S	ITU BIOREMEDIATION
	5.1	Introduction
	5.2	Anaerobic Dechlorination
		5.2.1 Introduction
		5.2.2 Dechlorination Status of Upper Hudson
		PCBs in 1984
		5.2.3 Dechlorination Status of Upper Hudson
		DCBs in 1990
		$5.2 A \text{Summary and Conclusions} \qquad 260$
	5 2	Sequential Microbial BCB Degradation
	5.5	Specific Comments on FDA Boulow
	3.4.	Specific comments on EFA Review
	5.5	
	5.6	List of References
6.0	OTHE	R PCB SOURCES
	6.1	The Benefits of Potential Remedies Cannot Be
		Assessed Without Adequate Characterization of
		Sources
	6.2	EPA's Emphasis On The Mass Movement Of PCBs
		Primarily From A Single Source Is Inconsistent
		With Sediment Data Demonstrates That The Origin
		And Movement Of Hudson River PCBs Is And Has
		Historically Been Dominated By Multiple Sources 274
		6.2.1 Badionuclide Dating Of Sediment Cores
		The DCP Deak Occurred Refere The 1972 Dam
		The FCB Feak Occurred Berore The 1975 Dam
		6.2.2 Local variability in PCB Levels
		6.2.3 Local variability in PCB composition 280
		6.2.4 Regional Trends in PCB Levels
		6.2.5 Regional Trends in PCB Composition 287
		6.2.6 Regional Differences in Total PCB
		Loading
		6.2.7 PCB Movements in Other Estuaries 293
	6.3	PCBs From The Upper River Accounts, At Most, For
		Only A Small Fraction Of The PCBs Accumulated By
		Lower River Fish
		6.3.1 PCB Concentrations in Lower River Fish 296
		6.3.2 Migration Patterns of Striped Bass 299
		6.3.3 Feeding Habits of Striped Bass
		6.3.4 Composition of PCBs and Other
	•	Contaminants in Striped Bass
	6.4	EPA's Investigation And Estimation Of The
		Contribution Of Other PCB Sources Has Been Grossly
		Insufficient
		6.4.1 Industrial Discharges
		6.4.2 Sewage discharges
		6 4 3 Tributaries 316 O
		6 4 4 Landfill leachates
		ц Х

Page

		6.4.5 Storm Water and Combined Sewer/Storm W	Water						
		Outfalls	•	3	19				
	6.4.6	6.4.6 Atmospheric Deposition	•	3	20				
		6.4.7 Total Lower River Sources Relative	urces Relative						
		To Upper River Transport	•	3	22				
		6.4.8 Upper River Sources	•	3	23				
	6.5	Recommendations	•	3	24				
	6.6	List of References	•	3	26				
7.0	CONC	JUSIONS	•	3	31				

INDEX TO APPENDICES (Separately Bound)

- A. SPECIFIC PAGE BY PAGE COMMENTS
- B. COMMENT ON THE CHARACTERIZATION OF PCBS FOUND IN ENVIRONMENTAL SAMPLES
- C. COMMENT ON "TBC" ITEMS
- D. RECENT SCIENCE ON PCB TOXICOLOGY
 - Institute for Evaluating Health Risks. 1991. Reassessment of Liver Findings in Five PCB Studies in Rats.
 - Comments of the General Electric Company on the Advanced Notice of Proposed Rulemaking of the United States Environmental Protection Agency Concerning Disposal of Polychlorinated Biphenyls, 55 F.R. 26738. 1990.
 - Chase, K.H., J. Doull, S. Friess, J.V. Rodricks and S. Safe. 1989. Evaluation of the Toxicology of PCBs.

E. REFERENCE MATERIALS FOR SECTION 2.0

1. Figures for Section 2.2

a.	Figure 2.2-1	Conceptual Diagram of PCB Interactions in Sediment, Water, Air and Biota
b.	Figure 2.2.2.21	Henry's Constants for PCBs in relation to chlorine number.

- 2. Xu, Y.J. 1991. Transport Properties of Fine-Grained Sediments, Ph.D. dissertation Abstract, UCSB.
- 3. Gailini, J., C.K. Ziegler, W. Lick. 1991a. The Transport of Suspended Solids in the Lower Fox River, J. of Great Lakes Research, in press.
- 4. Ziegler, C.K., J. Lick and W. Lick. 1990. SEDZL: A User-Friendly Numerical Model for Determining the Transport and Fate of Fine-Grained, Cohesive Sediments, UCSB report.

001 1455

HRP

F. REFERENCE MATERIALS FOR SECTION 3.0

1.	Figu	res for Section 3.1	•
	a.	Figure 3.1-1	Time Trend Graph of Decreasing PCB Concentrations in the Upper Hudson River Water Column
	b.	Figure 3.1.3-1	Time Trend Graph of Total Lipid Based PCB Concentration in Large Mouth Bass
2.	<u>Figu</u>	res for Section 3.2	
	a.	Figure 3.2.1-1	Mean and Standard Deviation of Birth Weight: High Dose Group and Controls from Barsotti and Van Miller (1984), and Historical Measurements from Van Wagen and Catchpole (1956)
	b.	Figure 3.2.2-1	Monte Carlo Simulation (10,000 iterations)
3.	Table	es for Section 3.2	
	а.	Table 3.2.2-1	Summaries of Studies Regarding Recreational Angler Fish Consumption
	b.	Table 3.2.2-2	Species-specific Angler Effort
	с.	Table 3.2.2-3	LADD Distributions (mg/kg/day) for Thirty Year Exposure
•	đ.	Table 3.2.2-4	Average Percent Reductions in the Concentrations of PCBs in Fish Filets by Various Cooking Methods

G. REFERENCE MATERIALS FOR SECTION 4.0

- 1. Borah, <u>Scour-Depth Prediction Under Armoring Conditions</u> (1988).
- 2. Palermo, Autumn 1991, <u>Equipment Choices for Dredging</u> <u>Contaminated Sediments</u>, Remediation Journal.

vi

- 3. Rice and White, 1987, PCB Availability Assessment of River Dredging Using Caged Clams and Fish, 6 Env. Tox. 259-274 (1987).
- 4. Swan, <u>Analysis of Dredge Safety Hazards</u>, United States Dept. of the Interior, Bureau of Mines.

H. REFERENCE MATERIALS FOR SECTION 5.0

1. Figures for Section 5.2

a. Figure 5.2.2-1

Figure 5.2.3-1 (A-C)

Figure 5.2.3-2

Figure 5.2.3-3

b.

c.

d.

Dechlorination of PCBs in the Upper Hudson River. Open circles represent samples with the ratio of peak 70 to peak 47 less than or equal to 0.5. Crosses are sample location.

- 5

Capillary column GC chromatograms from sediment samples from site H7 in the Upper Hudson River. Figure A to C represent samples obtained in progressively later years.

Average PCB concentrations in sediment samples from the H7 site in the Upper Hudson River. Based on data obtained in 1990.

Average number of chlorine atoms per biphenyl molecule in sediment samples from the H7 site in the Upper Hudson River. Based on samples obtained in 1990.

2. <u>Tables for Section 5.2</u>

a. Table 5.2.2-1

Dechlorination status of PCBs for different concentration ranges on the Upper Hudson River. Based on an analysis of

chromatographic peaks (70 and 47) from the New York 1984 data base.

b. Table 5.2.2-2

Table 5.2.3-1

C.

Percent of samples from the Upper Hudson River showing significant declorination as a function of concentration. Based on an analysis of chromatographic peaks (70 and 47) from the New York 1984 data base.

Average number of chlorine atoms per biphenyl molecule in selected "Hot Spots" sediment samples in the Upper Hudson River. Based on data obtained during 1990.

- 3. Abramowicz, D.A. (1990) Aerobic and Anaerobic Biodegradation of PCBS: A Review. V. 10 I.3, pp. 241-251 <u>Critical Reviews in Biotechnology</u>.
- Bedard, D.L. (1990) Bacterial Transformation of Polychlorinated Biphenyls. V. 4 <u>Biotechnology and</u> <u>Biodegradation</u>.
- 5. General Electric Company Corporate Research and Development (1990) Research and Development Program for the Destruction of PCBS: Ninth Progress Report
- I. Reference Materials for Section 6.0
 - 1. Tables for Section 6.2
 - a. Tables 6.2-1 and 2

Table 6.2.1-1

Table 6.2.1-2

b.

c.

Data Collected by Harza Engineering Co. (1988-90); Sample location sites.

Bopp et al. (1982) Cs-137/PCB Core Data.

> Annual PCB Environmental Load by Aroclor Type. Versar, at 288 (1976).

> > HRP 001 1458

viii

	d.	Table 6.2.4-1	Regression Analysis of Lower River sediment surveys.
	e.	Table 6.2.5-1	Compositions of US PCB Production, of Standard and Evaporated Aroclors, and of Upper Hudson River Reach 9 Deposits and Reach 8 Redeposits.
2.	<u>Tabl</u>	es for Section 6.3	
	Tabl	e 6.3.1-1	PCB concentrations in Striped Bass from the Hudson River in 1990. NYSDEC (1991)
3.	Tabl	es for Section 6.4	
	a.	Table 6.4.1-1	End-Uses of PCTs and PCBs by Aroclor Type. Versar, at 206 (1976).
	b.	Table 6.4.1-2	PCB Concentrations in the Effluents of the Machinery & Mechanical Products Manufacturing. Versar, at 311 (1976).
4.	<u>Figu</u>	res for Section 6.2	
	a.	Figure 6.2.1-1	Domestic Sales of PCBs by Aroclor Type. Versar, at 203 (1976).
	b.	Figure 6.2.1-2	PCB Sediment Concentrations in Lake Ontario
	с.	Figure 6.2.4-1	Average PCB Concentrations in Upper Hudson PCB Sediments. Zafflemere (1979)
	d.	Figures 6.2.4-2 and 3	PCB Levels in Hudson River Gammarus. O'Connor, J.M. (1978-81).
	e.	Figure 6.2.4-3	Graph of Four Lower Hudson and One Upper Hudson Sediment Surveys.

COMMENTS OF THE GENERAL ELECTRIC COMPANY ON EPA'S PHASE 1 REPORT

1.0 INTRODUCTION

The General Electric Company (GE) submits these comments on the August 1991 Review Copy of the Phase 1 Report issued by the U.S. Environmental Protection Agency (EPA) for its Reassessment Remedial Investigation and Feasibility Study (RI/FS) of the Hudson River PCBs Site.

GE believes that the compilation of existing data contained in the Phase 1 Report demonstrates that there is no basis for modifying the Agency's 1984 decision not to dredge Upper Hudson River sediments. These data establish that conditions in the Hudson River have steadily and substantially improved since 1984. The river is cleaning itself naturally, and PCB levels in water, sediment, and fish have been declining.

The simple facts are: (1) natural dechlorination processes are continuously and significantly reducing the impact of PCBs in the Hudson; (2) important new information relating to the toxicity of PCBs establishes that EPA's assumptions in its preliminary risk assessment are scientifically invalid; (3) no harm to human health or the ecosystem has occurred from PCBs in the Upper Hudson, and there will be no future unacceptable risk; (4) no new relevant dredging technologies have been developed since 1984; (5) dredging will be ecologically destructive with no corresponding benefit; and (6) EPA has not evaluated the sources, fate, and transport of PCBs in the Hudson River to adequately characterize the site, to assess risk, or to screen remedial alternatives.

Despite the strong evidence on these points, GE is concerned that EPA may draw incorrect conclusions if it continues to use an inadequate, qualitative approach to data analysis and continues to accept old, faulty assumptions without adequate scientific review. These comments address EPA's method of analysis and assumptions.

1.1 <u>Background</u>

In deciding to perform this Reassessment RI/FS, EPA does not write on a blank slate. Indeed, EPA's 1984 Record of Decision (1984 ROD), based on the results of an NUS Feasibility Study, contains an extensive assessment of remedial alternatives pertaining to the Hudson River, including the no-action alternative and both full-scale and selective "hot spot" dredging.

Significantly, EPA concluded after a detailed analysis of remedial alternatives that the no-action alternative was the most appropriate way of dealing with PCBs in the Hudson River sediments (1984 ROD, pp. 5-9). As stated in the 1984 ROD: "Natural on-going sediment transport mechanisms within the river have covered many of the PCB contaminated areas (hot and cold spots) with a less contaminated sediment layer, which significantly reduces the migration of PCBs in the water column and exposure to aquatic life" (1984 ROD, p. 8). In addition, EPA found that "the natural assimilative capacity of the river will continue the downward trend in the levels of PCBs found in the river" (1984 ROD, p. 8). EPA also noted that "[i]f present

HRP 001 1461

conditions continue, the amount of PCB passing into the estuary will continue to decrease with time" (1984 ROD, p. 9).

Based on these findings, EPA determined that "both the modeling and sampling data collected to date indicate a decreasing threat to public health and the environment" (1984 ROD, p. 9). In light of this decreasing risk, and because "the actual reliability and effectiveness of current dredging technologies in this particular situation is subject to considerable uncertainty" (1984 ROD, p. 9), EPA correctly issued a "no action" ROD with respect to Upper River sediments.

In undertaking the present RI/FS, EPA is determining whether it should reverse its 1984 decision. The 1984 ROD itself states that that decision "may be reassessed in the future if, during the interim evaluation period, the reliability and applicability of in-situ or other treatment methods is demonstrated, or if techniques for dredging of contaminated sediment from an environment such as this one are further developed" (1984 ROD, p. 9). Accordingly, the Agency is not free to reverse its position and to require some action in the Upper Hudson without a clear change of conditions.

Under fundamental principles of administrative law, the burden is upon EPA to establish that the facts have changed. As outlined below and in the detailed comments that follow, the Phase 1 Report provides no basis for EPA to reverse its 1984 decision. If anything, recent evidence <u>confirms</u> the correctness of EPA's 1984 decision.

HRP 001 1462

1.2 <u>Overview</u>

1.2.1 <u>No Unacceptable Risk</u>

The fundamental purpose of a remedial investigation is to determine whether the site poses an unacceptable risk to human health and the environment, and if so, to determine whether an effective remedial option exists to address the identified risk (40 CFR § 300.430 (a)(1)). A careful review of the information contained in the Phase 1 Report, as presented in Section 3.0 of these comments, demonstrates that the PCBs in upper Hudson sediments have not harmed human health or the environment and do not pose a future unacceptable risk. EPA has preliminarily come to a contrary conclusion because EPA has relied on out-dated science, unreasonable exposure assumptions, and a flawed analysis of the existing data.

New evidence since 1984 demonstrates that any risk present at the site in 1984 has decreased even further:

- PCB levels in Hudson River water have declined significantly, and PCB concentrations in fish tissue have also generally declined (pp. B.3-35, B.4-30, B.4-42). The 1991 NYSDEC report on PCB concentrations in striped bass is the most recent evidence of these improvements.
 - Recent scientific evidence based on animal, as well as human, studies shows that the types of PCBs found in the Upper Hudson River are not carcinogenic. This new information significantly reduces the estimated upper-bound risk at the site. The Phase 1 Report inexplicably and unjustifiably fails to use this information in its risk assessment.

HRP

100

- PCBs in the sediments of the Upper Hudson River have been substantially altered, thereby rendering them not only more amenable to complete natural destruction, but also resulting in PCBs that have markedly reduced toxicity and that are less prone to being concentrated in biota.
- A thorough analysis of fish consumption rates and River use patterns shows that real world conditions result in significantly reduced exposure factors for whatever PCBs remain in the River.

All of these changes indicate that whatever risk existed in 1984 is diminished today and will continue to diminish in the future. The 1984 ROD found that any risk at the site did not justify remedial action with respect to the sediments. Even stronger evidence exists today to compel the same conclusion.

EPA's regulations and guidance require that any Superfund risk assessment be a "baseline" assessment of the risks posed only by the site that the Agency intends to remedy -- in this case, the sediments of the Upper Hudson River. The Phase 1 Report, however, combines the risks posed by all PCBs in the Hudson River, including PCBs discharged by other sources. The Phase 1 Report also fails to isolate the effect of the remnant deposits on fish concentrations. GE recently expended \$15 million to remediate the remnant deposits in accordance with 1984 ROD. A risk assessment that does not thoroughly take into account the potential beneficial effect of such remedial work is not a proper baseline risk assessment under EPA's own regulations and the NCP.

5

100

146.

1.2.2 <u>No New Dredging Technology</u>

The Phase 1 Report does not identify any advances in dredging technology that mitigate or eliminate the problems delineated in the 1984 ROD as the basis for disqualifying dredging as a suitable remedy. In particular, the 1984 ROD concluded:

> "Dredging activities by their nature tend to result in some degree of disturbance of the highly contaminated sediments, and thus result in some short-term problems, in the form of elevated PCB concentrations in the water and air, as well as increased fish contamination. . . Therefore, it is difficult to conclude at this time that the technology can be considered feasible or reliable" (1984 ROD, p. 7).

The Phase 1 Report addresses these concerns by simply reciting that "[d]redging systems identified in the literature fall into the hydraulic, mechanical and specialty-type categories" and then by superficially describing the various categories (p. C.4-7). But these dredging technologies all existed in 1984. The Phase 1 Report also suggests that recent field studies at the much smaller and less dynamic New Bedford site prove that the cutterhead hydraulic dredge is the most successful in limiting sediment resuspension into the water column (p. C.4-8). As further discussed in Section 4.0, however, those field studies are not in any way applicable to Hudson River conditions and do not provide evidence overcoming the 1984 ROD's conclusion that dredging was not a feasible remedial technique.

The Phase 1 Report assumes the feasibility of sediment removal through dredging and then spends most of its discussion

HRP 001 1465

(Sections C.1 through C.7) on the screening of treatment technologies. Thus, it passes over one of the important findings of the 1984 ROD without in any way addressing whether there have been any technological developments that make it practical or feasible to dredge the bank areas of a 40-mile stretch of the Upper Hudson River.

1.2.3 Dredging Will Cause Adverse Environmental Effects

In addition to its failure to address the practicality or feasibility of dredging, the Phase 1 Report makes no mention of any adverse environmental and human health impacts of largescale dredging. The 1984 ROD, by contrast, specifically rejected bank-to-bank dredging as an appropriate remedy, because it "could be environmentally devastating to the river ecosystem and cannot be considered to adequately protect the environment" (1984 ROD, p. 6). Nothing in the Phase 1 Report suggests that these adverse environmental effects are any less serious now than they were in 1984. Indeed, the adverse environmental risks in this situation are so great that Congress took specific note of them in 1986 during consideration of the Superfund Amendments and Reauthorization Act:

> "[A] cleanup of PCBs in contaminated rivers like the Hudson, to achieve a cleanup envisioned by [the Toxic Substances Control Act], could require dredging. This, in turn, could result in greater exposure and threat to public health from the disturbed PCBs. . . <u>Such an illogical remedy</u> could also cause serious harm to the river's ecosystem." H.R. Rep. No. 253, 99th Cong., 1st Sess., pt. 1, at 57 (1986) (emphasis supplied).

HRP

I00

1466

Additionally, dredging of PCB containing sediments is not an isolated activity. The massive volume of removed material must go somewhere. The risks associated with removal and disposal must be, but have not been, evaluated to yield a fair comparative risk analysis. The 1984 ROD, in the course of evaluating the effectiveness of excavating the remnant deposits, noted for example that "there may be some adverse short-term impacts on public health" due to the likelihood of PCB releases to the air, the health hazards caused by truck trips through residential areas, and the increase in erosion and resuspension of PCBs into the river (1984 ROD, p. 11). For the much more complex and significant remediation of the Hudson River sediments, the Phase 1 Report does not even attempt such a superficial qualitative impact analysis.

Finally, as noted in the 1984 ROD, after the sediments are removed, they must be deposited in a new landfill either for the short term or long term (1984 ROD, p. 8). No such landfill existed in 1984, and none exists today.

Section 4.3 of these comments takes a more detailed look at the environmental effects of dredging and spoils handling. By contrast, the Phase 1 Report neither compiles data on these effects nor identifies a program for Phases 2 and 3 to develop information which could serve as a basis for changing EPA's own 1984 conclusions on this issue.

HRP 001 1467

1.2.4 An Integrated, Quantitative Approach Shows No Significant Benefit from Dredging

The recent Thomann study, discussed at length in the Phase 1 Report (section A.4), determined that dredging of Upper Hudson sediments will provide, at most, negligible benefits and that PCB concentrations in the Lower Hudson and Lower Hudson fish will improve nearly as rapidly without dredging. If EPA desires to go beyond the determinations of its 1984 ROD and the Thomann study, there is a crucial need for an integrated and quantitative approach toward site characterization and remedial alternative assessment.

This is not exclusively a problem of lack of data, although the Phase 1 Report acknowledges and GE agrees that serious data gaps do exist and preclude such an analysis. The problem also stems from EPA's currently incomplete and flawed methodology for drawing conclusions from the existing data. In particular, the Phase 1 Report fails to recognize the many complex interactions of PCBs in various media in the Hudson River. For example, any scientifically defensible assessment of remedies must understand the relationship between sediment PCB concentrations and water concentrations, between water and biota, between sediment and biota, and ultimately between all three media and fish, the primary route of exposure to humans. Those relationships must also be understood for various types of sediments and biota, different species of fish, varying flow conditions, over both long and short distances and over time. Given these interactions, a quantitative, integrated framework Ξ

9

for understanding the fate and transport of PCBs in the Hudson River, as discussed in Section 2.0 below, is essential. Yet none is currently planned by EPA.

Instead, the Agency intends to conduct a simplistic qualitative analysis of the available data. This is not a sound scientific approach to a large and complex river system. It is a methodology that will inescapably produce indefensible conclusions.

1.2.5 Biodegradation Is Effective on PCBs

Since the EPA 1984 decision, numerous researchers including EPA have found that PCBs, previously believed to be indestructible, can be degraded in an environment, like the Hudson River, by naturally occurring organisms. Despite national emphasis by EPA headquarters on new technologies to address remedial problems, the Phase 1 Report dismisses this important research.

Biodegradation research has established that two separate and complementary biological degradation processes are at work in the Hudson River to degrade PCBs. First, anaerobic bacteria, naturally present in river and lake sediments, remove chlorine from highly chlorinated PCBs. The resultant lightly chlorinated compounds are not carcinogenic and accumulate in organisms to a lesser extent than more highly chlorinated PCBs. These lightly chlorinated compounds are then further and totally degraded by aerobic bacteria found in the Upper Hudson River as well.

HRP 001 1469

The results of this research on natural PCB biodegradation have been widely published. They are critical to the matter addressed in the Phase 1 Report, and there is absolutely no justification for the Report's failure to properly appreciate and take that research into account. The transformation and destruction of PCBs by biological means is a critical process that must be understood if the fate and transport of PCBs in the River are to be evaluated in a scientifically defensible manner. This process is as important as volatilization, partitioning, and others affecting PCBs. Unless and until biodegradation affecting PCBs in the Upper. Hudson is thoroughly evaluated by EPA in this RI/FS, a proper analysis of risks and remedies cannot be made in a credible fashion.

1.2.6 Other PCB Sources

The Phase 1 Report acknowledges that there are significant current sources of PCBs in the Lower Hudson that are not related to PCB transport from the Upper River. EPA's investigation of these and other PCB sources, however, is insufficient to characterize the site. Without identification of the significant PCB sources, it is impossible to predict what impact, if any, potential remedies will have on reducing exposure to contamination. In short, any selected remedy may not address the actual source of the problem.

Furthermore, when addressing the issue of other sources, EPA accepts the assumption that historical and present contamination of the Hudson is dominated by the massive movement

11

of PCBs from two GE facilities after the 1973 dam removal. A thorough review of sediment data, as presented in Section 6.0, demonstrates that this assumption is false. In fact, the peak PCB concentration in lower Hudson sediments occurred in 1971, coincident with the peak in national PCB use and releases to the environment. This same pre-1973 peak has been observed by other researchers in other bodies of water. A full review of fish data also confirms that the Hudson is impacted by many sources of PCBs, not just one Upper River source. Resident fish species vary in PCB concentrations independent of their distance from the Upper River. They are impacted by local PCB sources. Likewise, migratory striped bass accumulate PCBs that did not originate in the Upper Hudson and did not originate with GE.

The importance of reassessing the fundamental assumption about massive movement of PCBs in the Hudson cannot be overstated. If historically no massive movement of PCBs occurred, EPA must seriously re-evaluate what quantity of PCBs could possibly be transported today over long distances from the Thompson Island Pool to other parts of the River. Concerns about the scour impacts of future floods must be examined in this new light.

Finally, EPA must consider focusing its limited resources on controlling these other PCB sources with local impact rather than pursuing a potentially devastating, expensive, and ultimately ineffective remedy that requires the dredging of Upper Hudson sediment. There is no shortage of information for EPA to begin the process of identifying these other sources.

12

Rather, it is up to EPA to use its investigative tools and resources.

1.3 <u>Required Actions</u>

The data presented in the Phase 1 Report demonstrate that EPA's 1984 decision was correct. EPA should at this time recognize the deficiencies in the Phase 1 analysis of the existing data and perform an analysis of the information that leads to scientifically defensible conclusions. To accomplish this, EPA must, at a minimum, do the following:

- Use important new scientific information on PCB toxicity;
- 2. Employ realistic site-specific exposures in the risk assessment;
- 3. Use the results of current research on the naturally occurring biodegradation of PCBs;
- 4. Collect sufficient data to understand
 - processes affecting PCBs in the river;
 - the spatial and temporal variations of the processes;
 - background levels of PCBs;
 - impediments to and adverse environmental effects of dredging;
- 5. Analyze the data (existing and to be collected) in a quantitative framework that allows complexities of the river system to be understood and simulated;
- 6. Investigate the sources of PCBs to the Lower River and reject erroneous assumptions concerning Upper River PCB sources; and
- 7. Analyze the implications of the finding that striped bass do not receive significant levels of PCBs from the Upper and Lower Hudson River.

A rough, qualitative approach to the complexities of the site and PCB fate and transport is unacceptable. When EPA

13

disregarded its national policy of having potentially responsible parties perform the RI/FS and refused to allow GE to perform the Hudson River Reassessment RI/FS, EPA promised that this would be a state-of-the-art effort. If in fact GE or any other PRP had prepared and submitted to EPA the Phase 1 Report, the Agency would have returned it with a demand for extensive revisions. Fundamental fairness and the public interest require that EPA hold itself up to the same high standard.

Finally, GE is concerned that EPA is violating legally mandated procedural requirements thereby unfairly prejudicing GE and the public. For example, at no time prior to the issuance of the Phase 1 Report did EPA create an administrative record for this Hudson River matter. GE and the public have had no opportunity to evaluate the information being considered by EPA as it prepared its Phase 1 Report. As a consequence, EPA has deprived GE and the public of an effective right to comment. What is required, therefore, is a truly open process where scientific information used by the Agency is available to all parties for review and discussion on a timely basis, not wedged in a comment period after EPA has already reached conclusions that it feels compelled to defend.

HRP 001 1473

QUANTITATIVE MODELING

Summary: EPA must construct an integrated, quantitative model of PCB fate and transport in the Upper Hudson to characterize the site adequately and to assess remedial alternatives meaningfully. Proper characterization of the site requires an integrated understanding of the numerous complexities of PCB interactions in Hudson River sediment, water, air, and biota. The assessment of remedial alternatives requires a quantitative tool for analyzing the existing data so that predictions of future PCB conditions under various assumptions may be reliably made. Absent such an integrated understanding and quantitative tool, EPA's qualitative analysis of the existing data will lead to a faulty understanding of PCB dynamics at the site and to an erroneous assessment of the impact on risk reduction by remedial alternatives.

The purpose of an RI/FS is "to assess site conditions and evaluate alternatives to the extent necessary to select a remedy" (40 C.F.R. § 300.430(a)(2)). In performing this RI/FS, EPA is therefore required to "[d]evelop a conceptual understanding of the site based on the evaluation of existing data" (40 C.F.R. § 300.430(b)(2)).

GE is deeply troubled by the qualitative approach used by EPA in the Phase 1 Report to develop a conceptual understanding of the Hudson River site. A qualitative approach fails to account for the real and important complexities of PCB fate and transport in the Hudson River system and will potentially lead to a flawed understanding of: the risks posed by PCBs in the Hudson River, the effectiveness and feasibility of removal technologies in the Hudson River, the potential for

15

100

natural bioremediation at the site, and the importance of the panoply of PCB sources in the Hudson River.

Specifically, in analyzing and synthesizing the historical data concerning PCBs in the Hudson River, the Phase 1 Report acknowledges that significant data gaps and limitations exist, but nevertheless proceeds to derive conclusions from the data regarding the dynamics of PCB transport and the fate of PCBs in the River. The qualitative and compartmentalized approach adopted by the Phase 1 Report to draw these conclusions is grossly inadequate, however, because it does not explicitly examine the specific mechanisms that control PCB fate and transport in a complex riverine system. In the absence of a quantitative understanding of these mechanisms and the constraints imposed by mass balance considerations and data quality limitations, interpretation of the historical data is subjective and open to considerable uncertainty.

As discussed in greater detail below, the roles that sediment transport processes (e.g., scouring, armoring, and suspension), sediment-water interactions (e.g., diffusion in pore water and partitioning on particulate matter), and volatilization play in controlling PCB fate, and the complex interrelationships among sediment, water column, and biota PCB concentrations cannot properly be assessed from a merely qualitative analysis that reduces complex chemical, physical, and biological processes to non-physically based measures. Indeed, reliance on such a qualitative analysis is likely to result in a remedial action

HRP 001 1475

that neither produces significant environmental benefits nor reduces public health or environmental risks.

2.1 The Quantitative Modeling Approach

An integrated fate and transport model -- *i.e.*, a model that defines PCB fate by reference to the physical, chemical, and biological mechanisms that affect PCBs -- is necessary to answer questions about the historical transport of PCBs in the system, the accumulation of PCBs in biota, and the future response of the system under various alternative remedial scenarios. The use of an integrated fate and transport model is therefore an essential tool for the quantitative evaluation of costs and benefits of potential remedial actions and for developing "a conceptual understanding of the site based on the evaluation of existing data" (40 C.F.R. § 300.430(b)(2)).

The use of quantitative fate and transport models for assessing water quality in both fresh water and marine systems is well-established. Over three decades of experience with such models has shown that the modeling approach provides two distinct yet complementary benefits: First, quantitative modeling allows numerous complex processes to be simulated and thus provides important scientific insights into the fundamental transfer and reaction mechanisms that affect the temporal and spatial distribution of the constituent in a water body. Second, quantitative modeling provides a practical and effective method of evaluating, in a meaningful way, various alternatives for addressing a specific problem.

17

Indeed, over the past thirty years, EPA and many state and regional agencies have extensively employed the quantitative modeling approach to address specific water-quality issues. As an indication of EPA's own support of the modeling approach, EPA's Office of Solid Waste and Emergency Response recently issued a draft "Report on the Usage of Computer Models in Hazardous Waste/Superfund Programs" (U.S. EPA 1990) summarizing various administrative approaches toward promoting the effective use of mathematical models by the Agency. EPA's Region II recently sponsored a modeling study for the analysis of nutrient removal for effluents to the Long Island Sound. Integrated, quantitative models have also been developed and refined over the past decade to analyze the fate and transport of contaminants in the James River; the Saginaw River; Green Bay, Wisconsin; and New Bedford Harbor, Massachusetts.

The analysis of kepone in the fresh and marine stretches of the James River, for example, included models of hydrodynamic suspended bed solids, physical and chemical mechanisms, and food chain analyses. The models were then incorporated in an overall framework to address environmental issues similar to those relating to the present conditions in the Hudson River. The James River model, incidentally, indicated that no-action was the most appropriate remedial alternative.

One of the more recent examples of a quantitative framework that relates sources of PCBs to the concentrations in fish is the model constructed by Thomann *et al.* for the Lower Hudson (Thomann *et al.*, 1989). Thomann's analysis incorporates

18

mass balances and estimates significant PCB transfer and loss mechanisms to calculate PCB homolog concentrations in water, sediment, and biota -- including striped bass -- over time and in space. The calculated concentrations were then compared to observed data to provide a quantitative indication of the level of understanding of cause-and-effect relationships. The model has been used to compare, quantitatively, the changes in striped bass PCB concentrations over time for no-action and removal alternatives.

Although the Phase 1 Report raises a series of issues (pp. A.4-5 to A.4-9) in connection with the level of uncertainty in the Thomann model, these issues do not detract from the overriding benefits derived from a quantitative understanding of the site. Because of the compelling need for a quantitative analysis of PCB fate and transport, Phase 2 of the Reassessment RI/FS should not substantially modify or abandon the Thomann model without replacing it with tools that are at least as consistent (*i.e.*, constrained by mass and energy balance considerations), capable of quantitative projections, and testable (via comparisons of independent calculations and observed data).

2.2 The Need for a Quantitative Model of PCB Fate and Transport in the Hudson River

The need for an integrated, quantitative framework to provide an adequate understanding of PCB fate and transport in the Hudson River is clear. For each of the three areas of investigation identified in the Phase 1 Report (p. B.4-1) -- $\bigcap_{i=1}^{n}$

19

migration and redeposition of PCBs in sediment; transfer of PCBs in sediment to water; and the effect of such transfers on bioaccumulation of PCBs in fish -- an integrated and quantitative model of PCB fate and transport is an essential means of drawing conclusions from the existing data in a scientifically valid manner. Indeed, a quantitative model that predicts future PCB concentrations in fish is directly relevant to the risk assessment to be performed by EPA as part of this RI/FS.

As the Environmental Engineering Committee of EPA's Science Advisory Board has urged (U.S. EPA, 1989): "In some cases involving more complex issues, future projections of environmental effects, larger geophysical regimes, inter-media transfer, or subtle ecological effects," all of which characterize the Hudson River site, "mathematical models of the phenomena provide [in addition to adequate data] an *essential element* of the analysis and understanding" (emphasis supplied). This Committee has also recommended (in the same document) that quantitative models should incorporate, "to the extent possible, the state-of-the-art scientific understanding of the environmental problem." EPA's apparent "willingness to abandon fundamental, scientific approaches" therefore should not be excused "simply because the required research and data are too difficult to obtain in a short time span."

Figure 2.2-1 shows, conceptually, a number of significant interactions of PCBs in the various "compartments" within the River. Although some of these processes (e.g., partitioning, biological degradation, and solution) can be

20

described by simple empirical relationships (e.g. partitioning), many are complex processes that do not lend themselves to simple relations (e.g., resuspension of cohesive sediments). More important, the movement of PCBs between compartments (e.g., sediment to fish) may involve a number of complex processes.

Attempts to simplify the description of the system result in interpretations that become less and less connected to reality. An example of this is the approach presented by EPA to understand what is arguably the most difficult compartment to understand, PCBs in fish. As shown in Figure 2.2-1, fish obtain PCBs in a very complex way (Figure 2.2-1 does not even include bioenergetic issues that need to be understood). In the Phase 1 Report, EPA discusses apparent bioaccumulation factors (BAFs), which are simple linear relationships between PCB levels in fish and PCB levels in water. As discussed below, EPA's use of BAFs is flawed, and the apparent linear relationship does not exist for the entire range of PCB concentrations.

There are two additional levels of complexity that are not presented in Figure 2.2-1. The first is that PCBs are not a single compound but rather are a unique group of chemicals with widely varying physical, chemical, and biological properties. One approximation that can be used to describe this large group of chemicals is to classify PCBs into 10 separate compounds, based on the number of chlorines per biphenyl molecule (*i.e.*, homologs). Even with this simplification, such an analysis of PCB fate and transport adds an order of magnitude to the complexity of the task. Indeed, the need for a homolog-specific

21

analysis is clear even from a qualitative review of the data, which show that such a differential treatment of PCBs is needed to help understand the changes in PCB composition in fish tissue (as measured by Aroclors) over time.

The second complicating factor is that spatial and temporal changes in PCBs within the various compartments must also be understood. It is essential to develop a framework for simulating changes in time and space. For example, a fundamental question to be answered by the RI/FS is to compare the changes in PCB concentrations and compositions in various fish species (a) if natural processes are permitted to occur, or (b) if PCBcontaminated sediment is removed from a section of the River (i.e., "hot spot" dredging).

The only credible way to make such a projection or to answer such questions is to integrate each of the processes affecting PCBs into a complete and comprehensive quantitative model. A piecemeal approach that relies on a combination of empirical and qualitative descriptions of the system is not appropriate and will not offer reliable or defensible results.

GE strongly urges EPA to develop such an approach. This will be neither simple nor inexpensive. Significant amounts of data will need to be collected, and GE is prepared to discuss this more fully with EPA. The following is a basic framework of a sophisticated, state-of-the-art, computer-based model of PCB fate and transport in the Hudson River that EPA should develop:

> A two-dimensional, time-variable hydrodynamic model of the Hudson River. This will supply a number of inputs for the rest of the model (e.g.,

> > HRP 001 1481

spatial and temporal distribution of flow velocities).

- 2. A two-dimensional sediment transport model that accounts for both cohesive and non-cohesive sediment transport. The model output will include suspended sediment levels in the water column as well as identification of sediment erosion and deposition areas.
- 3. PCBs will be transported between the sediment and the water column (and air). A time-variable model should be constructed from the first two models and incorporate the important physical, chemical, and biological processes that affect PCBs. This model will provide projections of homolog-specific PCB levels over time and space in the water column, sediment, and air.
- 4. The final component will need to incorporate the PCB dynamics within fish. This will be a timevariable model since fish PCB levels can be a function of prior exposure conditions; it will also need to incorporate bioenergetics theory.

Given the demonstrated effectiveness and necessity of integrated fate and transport models for the analysis of complex, riverine systems, GE views with alarm EPA's lack of commitment to develop and use an appropriate fate and transport model for the Upper Hudson. If the Agency fails to construct an appropriate model of the Upper Hudson, it will be left to analyze numerous data points without any unifying mechanism to interpret the data within a quantitative analytical framework. In short, EPA will be making a decision potentially involving hundreds of millions of dollars -- about a highly complex, dynamic system -- on the basis of what are essentially quasi-scientific guesses.

2.2.1 Modeling of Sediment Transport

The Phase 1 Report's approach toward the analysis of PCB migration and redeposition in sediment is limited by its failure to analyze the significant effects of cohesive sediment transport processes. Detailed comments concerning the proper modeling of sediment transport in the Upper Hudson appear in Section 2.3 below.

The importance of understanding the movement of sediments in the Hudson River cannot be overstated. First, the presence of PCBs in the sediment in the Upper River has raised concerns regarding the potential mobilization of these contaminated, yet buried, sediments as a result of a large flood event. To properly evaluate the potential impact of a large flood, EPA must assess the potential of these sediments to scour. As discussed in Section 2.3.3, the most accurate and scientifically defensible method is to model sediment movement using the theory of cohesive sediment transport, coupled with either a two or three dimensional hydrodynamic model of flow in the Upper River.

Second, because PCBs tend to adhere to particulate matter, assessing PCB transport requires consideration of not only PCBs dissolved in the water column, but also PCBs absorbed to suspended sediment in the water column. Under certain conditions, the latter mode of PCB transport may account for the bulk of PCB movement. EPA must therefore develop a framework for determining the amount of suspended solids that will be transported in the River under a range of flow conditions.

HRP 001 1483

Moreover, to understand the effects of various remedial alternatives, EPA must also predict the suspended sediment load under varying bed geometry conditions. At a minimum, this task requires a two-dimensional cohesive-sediment transport model that accounts for not only the partitioning of PCBs in dissolved and particulate form, but also the different PCB homologs in the system.

2.2.2 Modeling of PCB Interactions

The need for an integrated, quantitative model is perhaps most acute in light of the many complex interactions among PCBs in different environmental media in the Hudson River occurring over time and space. In addition, because PCBs are a group of 209 different chemical compounds, EPA's analysis must recognize that different PCBs behave slightly differently in different media. Even if the 209 congeners are treated in homolog classes, PCBs must still be treated as 10 different compounds.

Among the principal interactions that must be fully understood before the site is properly characterized are: (1) interactions between PCBs in sediment and PCBs in water (partitioning), and (2) interactions between PCBs in water and PCBs in air (volatilization).

2.2.2.1 Sediment-Water Interactions

There are two different, yet equally significant, interactions between PCBs in sediment and PCBs in the water column. First, PCBs in the water column may either be dissolved in the water or absorbed on suspended particulate water. The

distribution of PCBs in the two phases (dissolved and particulate) can be determined by equilibrium partitioning theory. Second, for PCBs that are buried in the sediment, partitioning between PCBs in the sediment and PCBs in the pore water will occur. PCBs in the pore water can be transported by diffusion of advection into the overlying water column.

Laboratory and field data indicate that the partitioning between PCBs in particulate and dissolved phases is a function of PCB chlorination, suspended solids concentration, organic carbon content, and dissolved organic carbon concentration (e.g., O'Connor and Connolly, 1980; DiToro, 1985; Caron, 1988; Capel and Eisenreich, 1990). Homolog-specific partition coefficients, for example, have been calculated from suspended solids concentrations and water column field data collected as part of the New Bedford Harbor RI/FS (Battelle Ocean Sciences, 1990).

An integrated approach is the best way to account for the different characteristics of different PCB homologs in different media. Partition coefficients, for instance, decline with increasing solids concentration and increase with increasing chlorination. A further complication in the analysis of PCB adsorption and desorption is the difference in partitioning between the water column and the sediment. For example, partition coefficients calculated from PCB congener concentrations measured in sediment cores from New Bedford Harbor (Brownawell and Farrington, 1986) are one to three orders of magnitude lower than the water column values. Additionally,

HRP 001 1485
these partition coefficients do not appear to be related to the classically predicted partition coefficient, given by the product of the sediment fraction organic carbon (f_{∞}) and the octanol-water partition coefficient (K_{∞}) . When the partition coefficients are corrected for the dissolved organic content of the sediment pore water, however, they do conform to partitioning theory.

These data suggest complex and significant differences in PCB transport in the different fractions, i.e., on suspended solids and in the water phase. A thorough understanding of these differences is required for a proper characterization of the site, because it is otherwise impossible to assess the relative importance of various transport mechanisms and to predict the relative effect of various remedial alternatives. The consequence of these observable differences on PCB fate in Hudson River sediment and water can only be properly evaluated through an integrated, quantitative modeling framework.

2.2.2.2 Water-Air Interactions

Volatilization of PCBs is also a significant complicating factor in the understanding of PCB fate and transport in the Hudson River. For example, the Upper Hudson contains two regimes -- flowing water and water flowing over dams -- that must be treated differently to assess volatilization. The Phase 1 Report fails to account for the enhanced volatilization that results during the free fall of water over a dam. Because different PCB homologs exhibit different fate and transport properties, it is critically important for any acceptable model

1 1486

of PCB fate and transport to account explicitly for changes in PCB homolog distributions as a function of environmental medium, space, and time.

Moreover, for the determination of Henry's Law constants, EPA should perform a critical appraisal of the literature rather than rely solely on the results of one study. Henry's Law constants for individual PCB congeners have been reported by Burkhard et al. (1985), Murphy et al. (1987), Dunnivant and Elzerman (1988), Dunnivant et al. (1988), Hawker (1989), and Brunner et al. (1990), as well as Bopp (1983). Figure 2.2.2.2-1 shows the mean and range of Henry's Law constants for different homologs, as reported by Murphy et al. (1987), Brunner et al. (1990), and Bopp (1983). This figure illustrates declining Henry's Law constants with increasing chlorination and compares the differences between the three studies. The declining trend indicates the importance of distinguishing between lower chlorinated and higher chlorinated PCBs when assessing PCB transport. The differences between the studies indicates that a critical evaluation of the data must be performed so that appropriate values of this parameter may be determined.

2.2.3 Modeling of PCBs in Fish

The need for an integrated, quantitative model of PCB fate and transport is also evident in light of the difficulties in understanding and predicting PCB levels in Hudson River fish. Data in the Phase 1 Report (Table B.4-5; Figures B.3-14 to B.3-17; pp. B.3-29, B.3-34, and B.3-35), for example, suggest

28

that Aroclor 1254 concentrations in fish are not declining as rapidly as they are in other media. Examination of the information in Table B.4-5 indicates that PCB concentrations in fish will be reduced only if the PCBs associated with Aroclor 1254 are reduced in the fish. To achieve this goal, (1) the factors that contribute to PCB homolog concentrations in fish must be identified, and (2) this information must then be used in the evaluation of remedial alternatives to ensure that the relevant PCB homologs are being reduced.

2.2.3.1 Factors Affecting PCB Concentrations in Fish

The complexity of the interactions (over time as well as space) among PCBs in the water, sediment, and fish in the Hudson River can only be understood through an integrated and quantitative analysis. As the Phase 1 Report states (p. B-4.32): "Estimates of removal rate or half-life depend on multiple factors, many or most of which may be unknown or unquantified."

This complexity is exemplified by the equivocal statements in the Phase 1 Report concerning the relative effects of sediment and water concentrations on fish concentrations. On the one hand, the Phase 1 Report employs the bioaccumulationfactor (BAF) approach to derive a linear correlation between water concentrations and fish concentrations (pp. B.4-37 to B.4-38, B.4-42; Figure B.4.25), at least for data from the summer low flow seasons. On the other hand, the Report suggests that the fish concentrations may be declining more slowly than the water

29

concentrations (contrary to the assumption of linearity) "perhaps via a benthic food chain pathway" (p. B.4-40).

Even though these statements are sufficiently qualified to avoid any direct contradiction, the Phase 1 Report plainly reveals a lack of any precise understanding of how PCB concentrations in fish are affected by PCB concentrations in other environmental media. In particular, the BAF approach is very simplistic and has no physical basis. The BAF approach not only fails to represent or explain the data, it also fails to provide any meaningful way of assessing the effects of various remedial alternatives.

Moreover, EPA's reliance on the use of simple time trends to extrapolate from the historical data is unwarranted by data limitations (as defined by the data quality objectives of the various studies). The use of extrapolations of time trends without an understanding of the underlying causal relationships, particularly the relationships between sources of PCBs and concentrations in fish, is unsound and can lead to serious errors. Here again, an integrated and quantitative model, rather than qualitative suppositions, will significantly further an accurate understanding of the system that will permit a more rational and defensible assessment of remedial alternatives.

EPA must also develop a food web model to understand PCB movements in relevant species. A food web model based on bioenergetic theory, for example, can provide an understanding of the sources and fate of PCBs in the fish. If such a model is combined with a time-variable PCB transport model, EPA will be

30

able to evaluate the effect of various remedial alternatives on PCB concentrations in fish. Indeed, EPA followed this procedure at the New Bedford Harbor Superfund site.

Finally, any integrated understanding of the site must account for the significant and widespread biodegradation of PCBs in Upper Hudson sediments. PCBs that have been biologically altered as a result of natural processes have less of a tendency to bioaccumulate in biota. As discussed in Section 5.0, EPA must consider the impact of biodegradation to achieve an adequate understanding of the site.

2.2.3.2 PCB Concentrations in Fish and the Evaluation of Remedial Alternatives

The finding that Aroclor 1254 is the most abundant PCB in Upper Hudson fish also has significant implications for the determination and definition of remedial alternatives. For a remedial action to be effective, it must be shown to reduce PCB concentrations in Upper Hudson fish. This means a reduction in the penta-chlorinated and hexa-chlorinated homolog PCBs as characterized by the Webb and McCall Aroclor 1254 measurements. Appropriate remedial actions are, therefore, those that address sources of the particular PCBs that affect the fishery, i.e., the penta-chlorinated and hexa-chlorinated PCBs. In other words, remedial actions that reduce PCB sources that are not substantial contributors to the concentrations of PCBs in fish should not be HRP considered effective remedial actions that will improve the fishery or reduce a perceived potential health risk. 100

31

To illustrate this point, consider (by analogy) the discussion in the Phase 1 Report (p. B.3-39) concerning PCB concentrations in Chironomids in the Upper Hudson. Assume for the sake of the analogy that Chironomids are the organism to be protected and that concentrations of the tetra-chlorinated homolog, which is the most abundant in Chironomids, must be decreased to meet PCB standards. If remedial action evaluations are based on total PCB removal, then a remedial alternative that reduces di-chlorinated and tri-chlorinated PCB homologs with very little reduction of tetra-chlorinated homologs could be selected. This alternative will lower water column PCB concentrations (because di-chlorinated and tri-chlorinated PCB homologs are the most abundant in the water column), but will have little or no effect on the tetra-chlorinated PCB homolog concentrations in Chironomids. Thus, upon proper analysis, such a remedial action would not be an effective method for reducing PCB concentrations in Chironomids.

Analogously, in situations such as the Upper Hudson, evaluations of remedial alternatives that do not consider individual homologs, mass balances, and fundamental mechanisms, or that consider all PCBs alike, are likely to result in the selection of ineffective remedial actions. From this example it is also apparent that, when concentrations in biota are controlled by a limited number of congeners or homologs (as they are in the Upper Hudson), the failure to perform a homolog-

32

remedial actions. The integrated analysis discussed above removes this bias.

2.3 Sediment Transport

2.3.1 Flood Frequency Analysis

An accurate estimate of the peak flood flows in the Upper Hudson River is essential for reliable predictions of the erosional effects of a 100-year flood. As noted in the Phase 1 Report (p. B.4-6), previous investigators have significantly overestimated the peak flow rate of the 100-year flood in the Thompson Island Pool. GE therefore agrees with EPA's conclusion (p. B.5-6) that prior estimates of sediment bed erosion due to the 100-year flood are probably significantly higher than the actual erosion that would occur under EPA's estimate of the 100-year flood.

The impact of EPA's estimate of the 100-year flood flow rate on erosion in the Thompson Island Pool can be assessed from results of Zimmie's application of HEC-6 (Zimmie, 1985). Although EPA's estimate of the 100-year peak flow (44,300 cfs) is lower than Zimmie's 10-year flood peak of 46,000 cfs, results of Zimmie's model indicated that sediment bed elevation changes at the 46,000 cfs flow rate were "judged to be relatively insignificant with respect to erosion of sediment." In fact, erosion was predicted in only 14 of the 32 model elements with the median erosional depth being about 0.9 inches and the maximum being 1.8 inches. As discussed below, GE believes that even this conclusion overestimates the actual scour, because Zimmie's model

P 001 1492

does not employ proper sediment transport theories (i.e., those that account for cohesive sediment transport).

The Phase 1 Report's flood frequency analysis does contain one minor anomaly. Estimates of daily average flood flow rates for the Hudson below Sacandaga are presented on page B.4-3 and equivalent estimates at Fort Edward are listed in Table B.4-1. A comparison of these tables reveals that daily average flood flows at Fort Edward are lower than the same flows at the Hudson below Sacandaga, which is upstream from Fort Edward. Due to the significant increase in drainage area between Sacandaga and Fort Edward, the daily average flood flow rates should be higher at Fort Edward than at the upstream station. The source of the difference between these two tables is unknown and should be examined.

In addition, the Phase 1 Report omits one source of data that may prove useful for further refinement of the flood frequency analysis. Average daily flow rates at Spiers Falls have been measured by the Hudson River-Black River Regulating District since 1930 (Lawler et al., 1978). Spiers Falls is approximately 17.4 miles upstream from Fort Edward. The confluence of the Sacandaga and Hudson Rivers is about 10.2 miles upstream from Spiers Falls. The Spiers Falls data could be used to determine the accuracy of the Report's present analysis. The proximity of Spiers Falls to Fort Edward would tend to reduce any error caused by downstream translation of estimated flood flow $\frac{H}{20}$ rates.

34

2.3.2 <u>Suspended Sediment Analysis</u>

Although the Phase 1 Report generally contains an adequate analysis of suspended sediment concentration data, some interpretations of the data need to be reexamined. The Phase 1 Report and others (Zimmie, 1985) assert that a breakpoint in suspended sediment concentration exists at a flow rate of 10,000 to 12,000 cfs at Fort Edward. Under this theory, suspended sediment concentrations remain at a low, constant level until the river flow rate reaches the breakpoint, above which concentrations increase as a function of the flow rate. The Phase 1 Report concludes (p. B.4-9) that "[s]uch behavior is thought to represent an approximate critical shear stress for sediments in the river."

This statement should be qualified by two factors that may alter this interpretation of the data. First, no empirical evidence presently exists to establish a direct correspondence between suspended sediment concentrations and sediment bed erosion in the Thompson Island Pool; direct measurements of sediment bed erosion or deposition have been neither carried out nor correlated with sediment concentrations. Second, the suspended sediment in the river primarily results from two sources: erosion of the sediment bed and wash load from tributaries. It is therefore possible that the breakpoint could correspond to an increase in sediment load from tributaries. Again, no data are currently available to differentiate between the portion of the suspended sediment load due to bed erosion and the portion derived from the tributary wash load. The Phase 1

Report's conclusion that the breakpoint, at about 10,000 cfs, approximates a critical shear stress for sediments should therefore be recognized as a conclusion supported by neither an empirical model nor any other independent data.

The Phase 1 Report also presents an empirical trend analysis of suspended sediment concentration at Fort Edward, Schuylerville, and Stillwater. Although a correlation between concentration and flow rate has a sound physical foundation, the attempt to establish a functional dependence of concentration over time may be flawed. The Phase 1 Report's analysis asserts that the suspended sediment concentration in the Upper Hudson River is an exponentially decreasing function of time, with an average rate constant of -0.03 year⁻¹. A half-life of 23 years for sediment concentration decline is derived from this analysis. The Phase 1 Report then attempts to justify this correlation by postulating that the river sediment bed is gradually returning to an equilibrium condition after removal of the Fort Edward Dam in 1973.

Although removal of the dam certainly affected the sediment transport processes in the Thompson Island Pool, other factors may have also caused the apparent temporal decrease of suspended sediment concentrations in the Thompson Island Pool. EPA's unexplained use of an exponential curve to fit the data collected after 1973 may not adequately determine whether the sediment bed is returning to equilibrium after the dam removal.

001 1495

HRP

2.3.3 <u>Sediment Transport Modeling</u>

As the Phase 1 Report appears to recognize, the Upper Hudson River has a heterogeneous sediment bed that is composed of fine-grained, cohesive sediments (i.e., silts, clays, and organic matter) and coarse-grained, non-cohesive sediments (i.e., sands and gravels). Any sediment transport model that is applied to this river must therefore be capable of realistically modelling the transport processes of both cohesive and non-cohesive sediments in order to make predictions.

Previous attempts to model the sediment transport processes in the Upper Hudson River (Lawler et al., 1978; Zimmie, 1985) have used the HEC-6 model (U.S. Army Corps of Engineers, 1977). GE agrees with the Phase 1 Report's criticisms (section B.5.2) of the HEC-6 model. As the Phase 1 Report notes (p. B.5-3), HEC-6 has significant limitations that render the applicability of that model to the Upper Hudson questionable. Specifically, HEC-6 primarily simulates the transport of non-cohesive sediments and does not explicitly model cohesive sediment transport. As the Phase 1 Report correctly recognizes (p. B.5-3), cohesive sediments "may play an important role in Hudson River PCB transport."

In addition, the HEC-6 model is a one-dimensional model that accounts for neither lateral variations in the composition of the sediment bed nor hydrodynamic effects due to water depth changes. HEC-6 is therefore incapable of realistically simulating variations in a river that has a deep, central channel composed of sands and gravel as well as shallow, nearshore areas

001 1496

HRP

containing fine-grained, cohesive sediments -- a typical sediment bed structure in the Thompson Island Pool.

The Report identifies DYNHYD5 as the hydrodynamic model and STREAM as the sediment transport model to be applied to the Thompson Island Pool. Although these models are improvements over HEC-6, they are nevertheless constrained by serious limitations that call into question their ability to simulate sediment transport processes in the Thompson Island Pool in an accurate and realistic manner.

DYNHYD5 is a one-dimensional hydrodynamic model that essentially solves the same equations of motion and continuity as The Phase 1 Report proposes to use DYNHYD5 to model HEC-6. hydrodynamics in the Thompson Island Pool in a guasi-twodimensional manner by creating a link-node network with a maximum of three lateral channels. Although this application of the model does provide a rough approximation of the lateral variability in sediment bed structure and current velocities, it does not produce a true two-dimensional model. This is so because the link-node network, which determines the structure of the flow field, is still constructed externally and has no a priori theoretical basis. In addition, certain flow conditions may be incorrectly represented by the defined linkage. Only a true two-dimensional, vertically-integrated hydrodynamic model is capable of properly simulating lateral velocity variations. the very least, the Phase 1 Report should acknowledge the model's 100 weaknesses and identify possible sources of error.

The sediment transport model identified by the Phase 1 Report (STREAM) presents more serious problems that cannot be ignored. The Phase 1 Report correctly emphasizes the importance of cohesive sediment transport in the Thompson Island Pool throughout section B.5. Contrary to this recognition, the model selected by EPA is a non-cohesive sediment transport model and has no capability for explicitly modeling cohesive sediment processes. Although the Report states that a sediment erodibility parameter, e, in Equation (24) "represents the resistance to erosion due to cohesion or other bonding properties," the STREAM model is simply not designed to handle cohesive sediments. In short, EPA's simplified model does not contain an appropriate physical basis for modeling cohesive sediments and is therefore wholly inadequate for the important task at hand. To establish its scientific and technical credibility, EPA must consider the use of a more sophisticated and rigorous cohesive sediment transport model.

Several other problems exist with the STREAM model, although these deficiencies are minor compared with the model's inability to simulate cohesive sediment transport processes. For example, the details of the sediment bed model are presented in section B.5.4 but several key points are omitted. No mention is made of the specific transport capacity formula that will be used to calculate T_i in Equation (17). A large number of formulations are available, with different equations producing varying degrees of success, depending on the problem being examined (Garcia and Parker, 1991; Yang and Wan, 1991). Choosing the proper transport

capacity formula for the Thompson Island Pool is a critical issue and should be addressed. Another detail requiring discussion is the sediment size class distribution chosen for use in the calculations.

Although the Report presents an elaborate streambank erosion sub-model in section B.5.4.2, the need for analyzing the effects of streambank erosion in the Thompson Island Pool sediment transport model is questionable. Significantly, the sub-model has a large number of parameters that are difficult to measure. Moreover, calibration and verification of the streambank erosion sub-model will be extremely difficult. Finally, it is not clear that the banks of the Thompson Island Pool represent a significant source of sediment or PCBs.

Despite the above-described flaws in the Phase 1 Report's discussion of sediment transport modeling, the Phase 1 Report mentions a model that is particularly well-suited for studying the Upper Hudson River. Specifically, the Phase 1 Report cites (p. B.5-5) an application of the Ziegler-Lick sediment transport model (Gailani et al., 1991a) to the Fox River in Wisconsin. The Phase 1 Report acknowledges that this model is able to simulate cohesive sediment transport in a river that is similar to the Upper Hudson River. This model includes effects of flocculation on sediment deposition and bed compaction on Significantly, the erosion, both of which are time-dependent. model uses a true two-dimensional, vertically-integrated hydrodynamic and sediment transport algorithm. The model also $\stackrel{\circ}{\vdash}$ 1499 includes a non-cohesive sediment transport sub-model that has

been shown to produce reasonable results on the Fox River (Gailani et al., 1991b). Due to the successful results of the EPA sponsored Fox River project, the Ziegler-Lick sediment transport model is well-suited for application to the Upper Hudson River.

Of particular importance in determining the erosional effects of a 100-year flood in the Thompson Island Pool are the resuspension properties of fine-grained, cohesive sediments. The Ziegler-Lick model uses an experimentally based formula that predicts the amount of cohesive sediment that can be resuspended for a given sediment bed shear stress. After a finite amount of sediment is resuspended, the bed becomes armored. This armoring process is an important and fundamental difference in the behavior of cohesive and non-cohesive sediments. The amount of cohesive sediment resuspended is given by (Gailani et al., 1991a):

$$\epsilon = \frac{a_o}{t_D^n} \left(\frac{\tau - \tau_o}{\tau_o} \right)^m, \ \tau > \tau_o$$

(A)

effective critical shear stress that varies from approximately 0.1 dyne/cm² for freshly deposited sediments to approximately 1 dyne/cm² for t_D greater than 1 day. Results of the Fox River study (Gailani *et al.*, 1991a) indicate that Equation (A), as utilized in the Ziegler-Lick model, accurately simulates erosion of a cohesive sediment bed in a river during a major flood event.

As previously mentioned, the Phase 1 Report indicates that erosion of fine-grained, cohesive sediments may be simulated by modifying a non-cohesive sediment bed model. EPA proposes to calibrate the model by adjusting the sediment erodibility parameter, e, in Equation (24) of the Report. This approach, however, overlooks a key observed phenomenon that differentiates cohesive from non-cohesive sediments. Cohesive sediments resuspend a prescribed quantity of sediment for a given shear stress and time after deposition. After this resuspension, bed armoring eliminates further erosion unless the shear stress increases. The approach proposed in the Phase 1 Report has no experimental foundation and fails to represent correctly the complex interactions at the cohesive sediment-water interface.

Even if calibration of the proposed model (by the adjustment of e) is possible, e then becomes a lumped model parameter without definable relationships to fundamental mechanisms. The value of the lumped parameter e will vary in unknown ways. Thus, projections of sediment and PCB transport during extreme flow events, an acknowledged critical element of the model, cannot be relied upon. The use of quantitative models developed under EPA's sponsorship -- models that integrate funda-

HRP 001 1501

mental physical, chemical, and biological mechanisms -- can eliminate these problems and provide the basis for a credible analysis of transport during extreme flow events.

The aforementioned difficulties with the STREAM model make its use problematic, especially since the Ziegler-Lick model is unquestionably superior for the modeling of cohesive sediments and has been utilized by EPA on other rivers similar to the Upper Hudson. A documented version of the Ziegler-Lick model (Ziegler et al., 1990) is contained in Appendix E and should be applied by EPA.

2.3.4 Additional Data Requirements

To use this more appropriate sediment transport model, additional data concerning the properties of cohesive sediments in the Thompson Island Pool should be collected. Specifically:

- Shaker studies, similar to those conducted on the Fox River and Buffalo River (Xu, 1991), should be carried out to determine the *in situ* resuspension potential of Thompson Island Pool sediments. These field measurements will determine the *in situ* value of a, in Equation (A) for the Thompson Island Pool. Spatial variability of a, in the Thompson Island Pool could also be investigated.
- 2. The Fox River study (Gailani et al., 1991a) also indicated that inclusion of an easily-resuspendable, surficial sediment layer, i.e., a fluff layer, is necessary to simulate flood events accurately. The presence of a fluff layer in rivers, lakes and estuaries is well-known from field and laboratory studies. Measurements of the thickness and sediment concentration of the fluff layer in the Thompson Island Pool could be made in conjunction with any shaker studies.
- 3. The compaction of fine-grained, cohesive sediment beds, particularly those beds which contain a high fraction of very fine sand such as is found in the Thompson Island Pool, has a significant impact on the resuspension potential of the bed. Laboratory

HRP 001 1502

investigations using an annular flume (Xu, 1991) should be conducted on Thompson Island Pool sediments to determine quantitatively the effects of compaction time on resuspension potential. The value of n in Equation (A) has been experimentally determined to be approximately 2 for sediments deposited in a lake environment. Recent laboratory results (Xu, 1991) indicate that cohesive sediments deposited in a riverine environment compact much differently than lake-deposited sediments; the value of n for river sediments is probably significantly different from 2. Flume studies could be used to determine a realistic value of n for cohesive sediments in the Thompson These field and laboratory studies are Island Pool. essential for the development of accurate and reliable estimates of the parameters that control cohesive sediment bed erosion.

2.4 Other Modeling Issues

2.4.1 Radionuclide Dating of Sediment Cores

The Phase 1 Report relies on radionuclide core dating techniques (pp. A.3-1, A.3-2, B.3-12) for the analysis of PCB fate and transport. Indeed, the Phase 1 Report goes so far as to conclude (p. B.3-12) that the data from the interpretation of cores "demonstrate[s] that the sediments of the Upper Hudson could be used to determine PCB transport history."

Radionuclide core dating techniques were originally developed for ocean and lake sediments. The application of these techniques to river systems, which are characterized by differential sediment settling and scour over both time and space, introduces limitations on the usefulness of the analysis. These limitations make inferences of PCB sources, loadings, or fate and transport unreliable when they are based solely on core analysis. Thus, although the core data (vertical PCB or radionuclide profiles) can be used in an integrated modeling effort as one part of the total calibration and verification

database, and although the data may have limited use on its own terms (e.g., where the radionuclide peaks occur in cores that are close in time and space to the radioactive source), any broader inferences drawn solely from core analysis will likely lead to unknown but potentially large errors.

In addition to the significant limitations inherent with the use of radionuclide dating techniques in a riverine system, the core data on which the Phase 1 Report relies are not representative of the sediment database. In fact, only a very small number of sampled cores produced vertical profiles of radionuclides or PCBs that could be interpreted in the idealized context used to define interpretable cores. Other sampling stations did not have vertical profiles of radionuclides or PCBs that could be used in this context.

This selective use of the totality of the database indicates that the sampling stations that have interpretable cores may be different from the rest of the river in a number of important respects. These differences, of course, may be explained by a number of reasons:

- 1. Observed data from sediment samples indicate a large heterogeneity in types of sediment solids and PCB concentrations.
- 2. This heterogeneity is observed between stations in areas dominated by scour and areas dominated by settling. In addition there is heterogeneity between stations in the same area.
- 3. The rate of sediment accumulation is different between stations in the scour and settling dominated areas and within a given area. The accumulation rates vary with time.

HRP 001 1504

- Sediment from one location may be scoured and re-deposited at different locations depending on the sequence and magnitude of scouring flows.
- 5. The concentrations of PCBs are different between stations in the scour and settling dominated areas and within a given area. The accumulation rates vary with time.
- 6. The organic carbon content is also different between stations in the scour and settling dominated areas and within a given area. Organic carbon deposition rates vary with time.
- 7. The percentages of PCBs in the water column that are deposited are different at stations in the scour and settling dominated areas and within a given area. These rates also vary with time.

Because of these factors, the Phase 1 Report's conclusion that the radionuclide core analysis may be used to determine PCB transport history is questionable. The differences between sediment areas characterized by interpretable cores and areas characterized by non-interpretable cores impose profound limitations on the uses and extrapolations of information developed from analysis of data from interpretable cores. These data limitations render the use of this information for developing conclusions regarding PCB sources, loadings, and fate and transport highly unreliable.

2.4.2 Upstream PCB Source

The Phase 1 Report observes (p. B.4-24) that "it appears that a significant PCB load is in the river upstream of the hot spot areas (see Figure B.4-19)." If this is true, removal of sediment from the Thompson Island Pool will not have as significant an effect on PCB concentrations in the Upper Hudson River system as otherwise assumed.

HRP 001 1505

The existence of an upstream source of PCBs therefore changes or eliminates many of the assumptions held by EPA and others regarding the possible sources of PCBs in Upper Hudson fish. It is incumbent on EPA to understand the impact of this source during Phase 2 of the Reassessment RI/FS as part of proper site characterization and risk assessment. In particular, the contribution of this source to PCB levels in sediment, water, and biota must be investigated to draw a proper conclusion regarding the relative effects of potential remedial alternatives. Failure to do so will result in an overestimation of the risks associated with PCBs in the sediment and the selection of an ineffective and arbitrary remedial action. Indeed, EPA's identification of an upstream source provides yet another compelling reason to construct an integrated framework for a quantitative and homologspecific cause-and-effect analysis that relates PCB sources to PCB concentrations in fish.

2.4.3 The Effect of Floods on Fish Concentrations

The Phase 1 Report suggests (p. B.4-32) that the decline in Upper Hudson fish PCB concentrations during the 1980s may have been caused by low flows during that period, which in turn resulted in reductions in the availability of the lower chlorinated PCBs. The implicit assumption, of course, is that when higher flows occur, fish PCB concentrations will increase because the lower chlorinated homologs will then be scoured and will accumulate in the fish. The report therefore characterizes its projected declines in PCB concentrations as "best case" estimates.

The hypothesis adopted in the Phase 1 Report is only one of many that can explain current PCB trends. As an example of the contradictions that often result from this type of speculation, the Phase 1 Report also observes (p. B.4-24) that "[t]he spring flood in 1983 (35,200 cfs) was even greater than that of 1979[,] and PCB loads increased sharply during this year." The effect of the increase in PCB load in the spring of 1983 is not evident in the fish data, however, because fish do not respond to short term fluctuations in PCB water concentrations. This contradiction illustrates how a "back of the envelope," qualitative analysis can lead to misleading or unreliable conclusions.

In addition, EPA must account for the fact that sediment that is scoured during a flood will contain PCBs that have been naturally biodegraded, *i.e.*, are lightly chlorinated. EPA must therefore consider:

- 1. If the PCB-contaminated sediments are transported downstream, they will be in an aerobic environment. This will facilitate complete biological destruction.
- 2. If these lightly chlorinated PCBs enter the food web, they will have relatively short residence times within biota that tend to bioaccumulate these types of PCBs.
- 3. Because these lightly chlorinated PCBs tend to dissolve more readily into the water column and then volatilize more readily into the air, these PCBs will likely be less available to fish.

An integrated and quantitative cause-and-effect analysis -- one that incorporates mass balances, fundamental physical, chemical, and biological mechanisms, and homolog-specific

HRP 001 1507

differences among PCBs -- is essential to performing an adequate site characterization and to predicting the effectiveness of remedial alternatives. In particular, the sediment erosion model is one part of an overall integrated framework. Such a model can be used to predict, in a quantitative manner, the effects of a given flood and to determine the distribution of sediment and PCBs after the flood for any remedial action. The post-flood distribution of sediment and PCBs can be used in the fate and transport model to make projections over time of PCB conditions after the flood event. These projections can then be used to obtain a quantitative comparison of the relative costs and benefits of the various remedial alternatives under consideration. Given the acknowledged complexity of determining PCB fate and transport in the Hudson River, proper site characterization (as required by the NCP) requires no less.

2.5 List of References

Battelle Ocean Sciences. 1990. New Bedford Harbor Modeling Program Final Report. U.S. EPA, Boston, MA.

Bopp, R.F. 1983. Revised parameters for modeling the transport of PCB components across an air water interface. J. Geophy. Res. 88:2521-2529.

Brownawell, B.J. and J.W. Farrington. 1986. Biogeochemistry of PCBs in interstitial water of a coastal marine sediment. Geochimica et Cosmochin. Acta 50:157-169.

Brunner, S., E. Hornung, H. Santi, E. Wolff, O.G. Piringer, J. Altschuh and R. Bruggemann. 1990. Henry's Law constants for polychlorinated biphenyls: experimental determination and structure-property relationships. Environ. Sci. Technol. 24:1751-1754.

Burkard, L.P., A.W. Andren and D.E. Armstrong. 1985. Estimation of vapor pressures for polychlorinated biphenyls: a comparison of eleven predictive methods. Environ. Sci. Technol. 19:500-507.

001 1508

HRP

Caron, G. 1988. The influence of dissolved organic carbon on the environmental distribution or nonpolar organic compounds. Ph.D. Thesis, Drexel University. 185 p.

Capel, P.D. and S.J. Eisenreich. 1990. Relationship between chlorinated hydrocarbons and organic carbon in sediment and porewater. J. Great Lakes Res. 16:245-257.

DiToro, D.M. 1985. A particle interaction model of reversible organic chemical sorption. Chemosphere 14:1503-1538.

Dunnivant, F.M. and A.W. Elzerman. 1988. Aqueous solubility and Henry's Law constant data for PCB congeners for evaluation of quantitative structure-property relationships (QSPRs). Chemosphere 17:525-541.

Dunnivant, F.M., J.T. Coates and A.W. Elzerman. 1988. Experimentally determined Henry's Law constants for 17 polychlorobiphenyl congeners. Environ. Sci. Technol. 22:448-453.

Gailani, J., C.K. Ziegler and W. Lick. 1991a. The Transport of Suspended Solids in the Lower Fox River, J. of Great Lakes Research, in press.

Gailani, J., C.K. Ziegler, W. Lick and J. Steuer. 1991b. The Transport of Sediments in the Fox River, presented at the 34th Conference on Great Lakes Research, Buffalo, NY.

Garcia, M. and G. Parker. 1991. Entrainment of Bed Sediment into Suspension, ASCE J. of Hyd. Engr., 117(4):414-435.

Hawker, D.W. 1989. Vapor pressures and Henry's Law constants of polychlorinated biphenyls. Environ. Sci. Technol. 23:1250-1253.

Lawler, Matusky and Skelly. 1978. Upper Hudson River PCB No Action Alternative Study, Final Report. Report to NYSDEC. Pearl River, NY.

Murphy, T.J., M.D. Mullin and J.A. Meyer. 1987. Equilibration of polychlorinated biphenyls and toxaphene with air and water. Environ. Sci. Technol. 21:155-162.

O'Connor, D.J. and J.P. Connolly. 1980. The effect of concentration of adsorbing solids on the partition coefficient. Water Res. 14:1517-1523.

Thomann, R.V., J.A. Mueller, R.P. Winfried and Chi-Rong Huang. 1989. Mathematical Model of the Long-term Behavior of PCBs in the Hudson River Estuary. Report prepared for the Hudson River Foundation, June 1989. Grant Nos. 007/87A/030 and 011/88A/030.

HRP 001 1509

U.S. Army Corps of Engineers. 1977. HEC-6 Computer Program: Scour and Deposition in Rivers and Reservoirs, Users Manual. Hydrologic Engineering Center, Davis, CA.

U.S. EPA. 1990. Office of Solid Waste and Emergency Response (OSWER), Information Management Staff, "Report on the Usage of Computer Models in Hazardous Waste/Superfund Programs," OSWER Models Management Initiative, Phase II, draft, November 1990.

U.S. EPA. 1989. Science Advisory Board, Report of the Environmental Engineering Committee, Resolution on Use of Mathematical Models by EPA for Regulatory Assessment, and Decision-Making (EPA-SAB-EEC-89-012), January 13, 1989.

Xu, Y.J. 1991. Transport Properties of Fine-Grained Sediments, Ph.D. dissertation, UCSB.

Yang, C.T. and S. Wan. 1991. Comparisons of Selected Bed-Material Load Formulas. ASCE J. of Hyd. Engr., 117(8):973-989.

Ziegler, C.K., J. Lick and W. Lick. 1990. SEDZL: A User-Friendly Numerical Model for Determining the Transport and Fate of Fine-Grained, Cohesive Sediments, UCSB report.

Zimmie, T.F. 1985. Assessment of Erodibility of Sediments in the Thompson Island Pool of the Hudson River. Report to NYSDEC.

RISK ASSESSMENT

<u>Summary</u>: PCB concentrations in Hudson River water, sediment and fish have significantly declined since the 1984 ROD. Whatever risk existed then is less today and continues to decline. In addition, new science about the relevant types of PCBs in the Hudson River demonstrates they are neither carcinogenic in humans nor the etiological agents for any significant noncarcinogenic human health effects. Even if adverse health effects are assumed to be caused by PCBs, a properly conducted risk assessment shows that the baseline condition of the Upper Hudson sediments does not present an unacceptable risk to human health or the ecosystem. The Phase 1 Report fails properly to (a) account for the trends, (b) identify the baseline conditions, (c) evaluate PCB toxicity, (d) use realistic exposure scenarios, and (e) appreciate the current biological integrity of the Upper Hudson ecosystem.

3.1 Current Trends in Hudson River Data All Indicate A Reduced Risk Since 1984

The Phase 1 Report concludes (pp. A.3-5, B.4-16) that PCB concentrations in the water column in both the Upper and Lower Hudson have declined significantly over time since 1984. GE agrees with this conclusion (Figure 3.1-1). In addition, the Phase 1 Report correctly recognizes (pp. B.3-12, B.3-14) that the historical data for PCBs in Upper Hudson sediments are inconsistent and difficult to quantify (see Appendix B), but also notes that there has been an apparent decline in PCBs in the sediment samples since 1978. The Phase 1 Report also acknowledges (pp. B.3-35, B.4-30, B.4-37, B.4-42) that PCB concentrations in fish are not rising and in fact have generally **1**00 been declining over time.

HRP

It would appear, then, that the risk in the Upper Hudson associated with PCBs in the water column, sediments, and fish has declined below the risk present in 1984 when EPA decided that the risk was acceptably low and no action was warranted. If the risk in 1984 did not justify undertaking remedial action, current conditions compel the same result with greater confidence because exposure to PCBs and associated risk is declining. The lower PCB concentrations in water, sediments, and fish, and the associated lower risk to health and the ecosystem, today support reaffirmation of EPA's 1984 no action decision.

3.1.1 PCB Concentrations in the Water Column

The Phase 1 Report states that "there has been a statistically significant downward trend in concentration during the period of monitoring signifying a negative correlation between concentration and year" (p. B.4-16). This trend is illustrated in Figure B.3-12 in the Phase 1 Report.

Based on data provided by the U.S. Geological Service (USGS), the average PCB concentrations in the River (from mile posts 194 to 160), during summer average flow periods, decreased from about 0.5 μ g/l in the late 1970s to about 0.03 μ g/l in the late 1980s (Table B.3-13). The data show a significant and steady decline in summer average water column PCB concentrations to well below the detection limit of 0.1 μ g/l (p. B.3-24; Table B.3-13). Indeed, although year-to-year variations exist, the general trend is a 50 percent reduction in total PCB loading every three years (Figure 3.1-1). A similar trend is observed during high flow events.

According to USGS data, concentrations at the Waterford monitoring station declined from 0.40 μ g/l in 1970 to 0.033 μ g/l in 1989, and since September 1982, no PCB concentration greater than the detection limit of 0.1 μ g/l was found in either raw intake samples or treated water samples taken from the Waterford water treatment plant (p. B.3-25). In addition, monitoring at Schuylerville showed a decline from 0.66 μ g/l in 1977 to 0.038 μ g/l in 1989; monitoring at Stillwater indicated that PCB concentrations had declined from 0.74 μ g/l in 1977 to 0.045 μ g/l in 1989; and monitoring at Rogers Island at Fort Edward showed a decline from 0.22 μ g/l in 1978 to 0.026 μ g/l in 1989 (Table B.3-13).

3.1.2 PCB Concentrations in Sediments

The Phase 1 Report documents (pp. B.3-12, B.3-14) the decline in PCB concentrations in sediments since 1978. Figure A.3-1 in the Phase 1 Report shows that total PCB levels in dated Hudson River sediment cores have declined since the early 1970s. Likewise, Figure A.3-3 in the Report illustrates the decrease in PCB levels in the Hudson River sediment over time.

3.1.3 PCB Concentrations in Fish

The Phase 1 Report further states (p. B.4-30; Tables B.3-16 to B.3-19; Figures A.3-4 to A.3-7) that PCB levels in fish have declined exponentially over the last ten years, with some stabilization in recent years. Specifically, EPA concludes that "[p]lots of concentrations versus time for fish in the Upper Hudson indicate that PCB levels in all fish species appear to have declined in recent years" (p. B.4-30), and that "[a]verage

001 1513

HRP

lipid-based PCB concentrations in brown bullhead show a regular exponential decline for Aroclor 1016 components and a less dramatic decline for Aroclor 1254" (p. B.4-42).

Moreover, according to the Phase 1 Report, the upper 95 confidence limits of the projected 30-year average (1991 - 2020) PCB concentration of largemouth bass and brown bullhead are already at or below the 2 ppm FDA action limit (p. B.4-37).

The Report's analysis of PCBs in fish nevertheless has deficiencies. The use of the 1980 through 1988 fish data to determine time trends for extrapolation to the future is significantly flawed. The Report states (p. B.4-33) that lipidbased PCB concentrations in large mouth bass increased slightly between 1981 and 1988. GE's analysis, however, indicates a slight decline (Figure 3.1.3-1). EPA's results reflect the inappropriate use of a simple arithmetic average of the data, rather than the more appropriate log-normality analysis.

3.1.4 Lower River PCB Concentrations

The Phase 1 Report also states (p. A.3-3; Figure A.3-3) that the exponential decay rate of PCB concentrations in Lower Hudson River sediments appears to be the same as that in Upper River sediments.

Similarly, water column monitoring by USGS between 1978 and 1981 shows consistently lower PCB levels in the Lower River (p. A.3-6). As EPA concludes:

> "Like the Upper Hudson, the PCB levels in the Lower Hudson water column showed a declining trend in time over the monitoring period" (p. A.3-5).

HRP

100

1514

In addition, a time series trend of total PCBs on a ppm wet weight basis in the spring-collected striped bass from the Lower Hudson shows a large decline from 1978 to 1979. The geometric mean shows a decline from 1979 to 1987 (p. A.3-10).

Finally, the declining PCB trend in striped bass observed from 1978 to 1987 has continued. Recently, the State of New York released the results of its 1990 striped bass survey as a follow up to its 1987 sampling. The 1990 survey concluded: "overall, PCB concentrations are <u>significantly</u> lower then they were in 1978" (NYSDEC, 1991 (emphasis supplied)).

In sum, PCB concentrations in all relevant media -water, sediment, and fish -- in all parts of the Hudson River have significantly declined since 1984. These favorable trends will continue. As a result, the potential for human exposure to PCBs has decreased and continues to decrease. Whatever risk existed from such potential exposure has thus been diminished by natural processes.

HRP 001 1515

3.2 <u>Human Health Risk Assessment</u>

Section B.6 of the Phase 1 Report contains a preliminary human health risk assessment. This assessment concludes that there are unacceptable human health risks from the PCBs currently in the Upper Hudson River. This conclusion is inconsistent with the conclusion reached in the 1984 ROD. Because all of the trends since 1984, as noted above, point toward reduced human exposures to PCBs, the conclusion of the Phase 1 Report is inexplicable. The Phase 1 Report makes no attempt to reconcile these different findings.

GE believes that there are three principal reasons for the Phase 1 Report's erroneous conclusion:

First, EPA's failure to characterize the site accurately has led to an overestimation of the PCB concentrations at the point of exposure attributable to the Upper River sediments. A correct use of the techniques explained in Section 2.0 will eliminate this error in the future.

Second, EPA has not performed a proper toxicity assessment, has used outdated science on the carcinogenicity of PCBs, and has summarily derived an <u>ad hoc</u> PCB Reference Dose without any valid scientific basis.

Third, EPA has not used proper exposure pathway assumptions and has failed to develop realistic exposure scenarios.

A properly conducted health risk assessment shows that there are no unacceptable risks from the Upper Hudson sediments.

HRP 001 1516

57 -

3.2.1 EPA's Assumption About the Toxicity of PCBs is Incorrect

In its assessment of PCB toxicity in the Phase 1 Report, EPA relies heavily on outdated information and assumptions concerning PCB toxicity. Appendix D of these comments contains a full discussion of recent science on PCB toxicology.

The Phase 1 Report properly takes note of this new science (p. B.6-2), but the Report then fails to use the new science in its human health risk assessment, deferring to some unspecified "scientific review process." This dodge is clearly improper. The EPA staff responsible for the RI/FS has an affirmative obligation to respond to the information it has received that casts undeniable scientific doubt on the PCB toxicity information it is using.

The applicable guidance (RAGS I, p. 7-14) requires the "regional staff" to consult the EPA IRIS coordinator and establish a verification workgroup when confronted with information demonstrating that IRIS toxicity values for PCBs are outdated or inapplicable. The "it's not my job" or "it's out of my hands" attitude expressed in the Phase 1 Report is improper and, if continued, will perpetuate the errors contained in the Phase 1 risk assessment. More importantly, it will use inaccurate risk conclusions to drive a decision-making process to an incorrect and inappropriate result.

As discussed in more detail below, the major errors in the Phase 1 toxicity assessment are:

HRP

1517

- 1. It assumes that all of the 209 PCB congeners have identical toxicological characteristics; this is not true. The congeners GE discharged into the Upper Hudson have been shown to be noncarcinogenic.
- 2. It relies on an assessment of carcinogenic potential that is now known to be incorrect.
- 3. It fails to consider the epidemiological evidence demonstrating that exposure to PCBs do not result in elevated cancer risks in humans.
- 4. It neglects to account for the effect of natural PCB biodegradation on the cancer potency of PCBs in the environment.
- 5. It uses an unconfirmed and technically flawed PCB Reference Dose to characterize non-cancer risks and misuses the literature on the non-carcinogenic effects of PCBs.

3.2.1.1 Carcinogenicity of PCBs

In the assessment of carcinogenic potential of PCBs, EPA relies on outdated information. In July 1991, GE submitted a report to EPA (Moore, 1991) demonstrating that the PCB mixtures similar to those found in the sediments of the Upper Hudson River are not carcinogenic in rats and that other, more highly chlorinated PCB mixtures have a lower carcinogenic potential than assumed by EPA (see Appendix D). The cancer potency factor used in the Phase 1 Report is incorrect in light of this new scientific information, and the human health risk assessment performed using this erroneous factor comes to invalid conclusions.

3.2.1.1.1 1988 EPA Assessment

EPA's interim risk assessment in the Phase 1 Report uses estimates of the carcinogenic risks posed by PCBs currently set forth in IRIS and based on the revised carcinogenic potency

59

HRP 001 1518

assessment developed in the Drinking Water Criteria (U.S. EPA, 1988). In that assessment, EPA considered five studies of the carcinogenicity of PCBs in rodents. Published reports of these studies indicated that mixtures of PCBs with 42 and 60 percent chlorine were carcinogenic, but that those with 54 percent were not. (In the case of the 42 percent mixtures the carcinogenicity was based on an increase in benign tumors).

EPA's actual estimate of carcinogenic potency for PCBs as a group was based on only one of these studies: the Norback and Weltman (1985) study of Sprague-Dawley rats. This study found that female rats exposed to a commercial mixture of PCBs containing 60 percent chlorine by weight demonstrated the greatest carcinogenic response of any PCB mixture tested. The carcinogenic potency (or cancer slope, q_1 *) was estimated using the Global 86 linearized multistage low-dose response model and a "body surface area factor" to scale the animal potency to humans. Based upon this analysis, the potency of all PCBs was estimated to be 7.7 (mg/kg/day)⁻¹ (U.S. EPA, 1988).

3.2.1.1.2 New Findings

Recently, the liver tissue slides from each of the five original studies were screened by a panel of expert pathologists using current guidelines for interpreting liver lesions. These guidelines were developed by the National Toxicology Program (Maronpot et al., 1986; McConnell et al., 1988) and have been $\frac{H}{2}$ endorsed by EPA. The panel's proceedings were observed by representatives from EPA, FDA, Experimental-Pathology

60

Laboratories, Inc., the Institutes for Evaluating Health Risks, and participants in the original studies (Moore, 1991).

Although this review confirmed that the rats exposed to 60 percent chlorine mixtures developed tumors, the expert panel found that the number of animals with benign or malignant liver tumors was less than originally reported. More important, the review resulted in a reversal of the original conclusions of the Clophen A30 (a mixture containing about 42 percent chlorine) study (Schaeffer, 1984), concluding that the results were negative as to the carcinoginity of this PCB mixture. Finally, the panel confirmed that the study of Aroclor 1254 (a mixture containing 54 percent chlorine) performed by the National Cancer Institute was negative (NCI, 1978).

The basic conclusions of this 1991 review were that different PCB mixtures have significantly different carcinogenic effects and that some mixtures were not carcinogens. Therefore, the appropriate regulation of PCBs requires distinguishing between different PCB mixtures.

3.2.1.1.3 Reassessment of the Potency of PCBs on a Percent Chlorine Basis

1520

It has been a basic policy of EPA to assume that individual chemicals in a chemical class will differ in their carcinogenic potential. In the OSTP guidelines on chemical carcinogens, it was concluded that "Ordinarily, not all chemicals belonging to any class are carcinogenic, nor are all those compounds within a class which exhibit carcinogenicity equally potent" (OSTP, 1985).

EPA has recognized the need to adjust potency estimates for certain members of chemical classes. For example, EPA's rule on incidental generation of PCBs in manufacturing operations recognizes the difference between very lightly chlorinated PCBs and other PCBs by applying discounting factors of 50 and 5, respectively, for the toxic potential of mono- and di-chlorobiphenyls. Thus, for purposes of determining if a chemical mixture containing incidentally generated PCBs reaches the regulated level of 50 ppm, the concentration of monochlorinated biphenyl is divided by 50, and the concentration of di-chlorinated biphenyl by 5.

In addition, in recent policy decisions pertaining to the PCDD (polychlorinated dibenzo dioxin) and PCDF (polychlorinated dibenzo furan) families, the EPA has determined that approximately ten percent of the individual PCDD and PCDF congeners are considered toxic enough to be measured for risk assessment purposes. In performing risk assessments involving exposures to PCDDs or PCDFs, EPA has developed a system to account for the differing potencies of the different members of these chemical classes.

Thus, a clear policy and precedent exists for treating different PCBs differently.

Nonetheless, in its assessment of PCBs, EPA selected the study by Norback and Weltman (1985) for estimating the RP potency of all PCBs. Based on this study, EPA decided in its 1988 assessment that it could not apply its policy of 100 differentiating between chemical classes to PCBs, but instead 1521
would assume that all PCBs had the same carcinogenic potential as the most highly chlorinated mixture, Aroclor 1260, for which it had bio-assay results.

The 1991 reread, using current scientific methodology, clearly indicates that the EPA 1988 conclusions are not valid. The Schaeffer (1984) study of Clophen A30 (42 percent chlorine) is now clearly known to be negative. Thus, the only positive animal studies remaining in EPA's 1988 reassessment are those using PCB mixtures containing 60 percent chlorine, and, even in those studies, the estimate of carcinogenic potency was significantly overestimated.

3.2.1.1.4 Implications for the Upper Hudson River

The issue of selecting the most appropriate potency for PCBs is critical for a proper analysis of the Upper Hudson, since the PCBs released from the GE facilities had less than 60 percent chlorination. These included Aroclor 1254 (54 percent chlorine), Aroclor 1242 (42 percent chlorine) and Aroclor 1016 (<40 percent chlorine). Sales records for the period 1957 to 1977 indicate that 98% of GE's purchases of PCBs for use in the manufacture of capacitors at Hudson Falls and Fort Edward, NY, were Aroclors 1242 and 1016 (~42% chlorinated PCB). The balance was Aroclor 1254. Although Aroclor 1260 is commercially used in the manufacture of transformers, GE did not use Aroclor 1260 in the manufacture of the capacitors produced at the two Hudson River plants.

HRP 001 1522

3.2.1.1.5 Proposed Approach

On the basis of the recent scientific studies described above, a clear and sufficient scientific basis is now available to warrant regulation of PCBs by their degree of chlorination ("closest Aroclor" approach).

With respect to the studies of the lower chlorinated PCB mixtures, the results do not show a statistically significant increase in tumor incidence over control groups (Moore, 1991). Therefore, under current risk assessment guidelines, these compounds should not be regarded as carcinogens (OSTP, 1984). This position has been taken by the Science Advisory Panel of the State of California in its regulation of PCBs under Proposition 65.

3.2.1.1.6 Reevaluation of the Rat Liver Model for Determination of Human Risk

A review of the PCB animal studies also shows that:

- The PCB-exposed rats, including those with liver tumors, lived significantly longer than the controls (unexposed rats).
- The PCB-exposed rats had significantly fewer cancers of all types, *i.e.*, sum of all cancers, than did the controls (unexposed rats).
- The liver tumors, although formally classified as cancers, did not metastasize to other organs or invade blood vessels.

In other words, PCB exposure in rats appears to produce non-invasive, non-life-threatening rat liver tumors and indeed may well produce beneficial effects (significant life extension and reduction in number of other cancers relative to the controls). These conclusions seriously call into question the

relevance of the rat liver tumors to human risk. They provide additional assurance that a declassification of PCB mixtures having less than 60 percent chlorination as animal carcinogens can be made without endangering human health.

Results of several PCB experiments (Bandiera et al., 1982, Poland and Knutson, 1982; Safe et al., 1985) support previous in vitro mechanistic PCB studies which suggest that doses below a certain threshold should not activate the Ah receptor or induce enzymatic activity. Based on PCB structureactivity relationships, the most active congeners are the para and meta positions of both phenyl rings (Goldstein et al., 1977; Safe, 1989). These studies suggest that a PCB exposure level that produces neither a positive Ah receptor response nor induction of the cytochrome P450 system may be defined.

3.2.1.1.7 Evidence from Epidemiology Studies

After stating that epidemiological studies of human exposure to PCBs are "inconclusive" (p. B.6-31), the Phase 1 Report illogically goes on to conclude that PCBs cause cancer and a variety of other undesirable endpoints in humans (p. B.6-32). This conclusion is supported by an inaccurate and misleading tabular summary of epidemiological studies (Tables B.6-7 and B.6-8).

In fact, recent human epidemiology studies do not support the conclusion that exposures to large concentrations of PCBs result in elevated cancer risks in humans. Data from these studies have failed to demonstrate any consistent tumorigenic effect among populations exposed to high concentrations of PCBs.

HRP 001 1524

The Phase 1 Report's treatment of these studies misinterprets them and produces an alarming, but incorrect, summary of PCB's carcinogenic potential.

Perhaps the most shocking inaccuracy is the Phase 1 Report's repeated reference to the so called "Yusho incident." No responsible epidemiologist or toxicologist continues to believe that PCBs were the etiological agents responsible for the health effects observed in the Yusho incident population. In the Yusho incident, about 1,500 persons in Japan in 1968 became ill after consuming rice oil accidently contaminated with a PCB mixture known as Kanechlor 400 (48 percent chlorine) (Amuno et al., 1984; Kuratsune 1986). Numerous adverse short-term health effects were noted in the exposed persons, and studies suggested possible long-term effects, including increased cancer. However, recent re-evaluations of the Yusho incident have led to the conclusion that it was not a case of PCB poisoning but probably poisoning by polychlorinated dibenzofurans. The scientific community's consensus on this new conclusion was reported by Drs. Kimbrough and Goyer of the National Institutes of Health in 1985 and confirmed in 1986 by the Halogenated Organics Subcommittee of EPA's Science Advisory Board, which concluded that:

> "a discussion of the human health effects of polychlorinated byphenyls should not use Yusho as an example."

Subtracting Yusho from Table B.6-7 in the Phase 1

1986; Davidoff and Knupp, 1979; Brown, 1987; and Zack and Musch 1979) reported no incidence of cancer significantly elevated above calculated endpoints. EPA interprets the other four studies (Bahn et al., 1976, 1977; Bertazzi et al., 1987; Sinks et al., 1990; and Liss, 1990) as presenting evidence that exposure to PCBs causes cancer. This interpretation is not consistent with good science, as the following discussion shows.

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 (1977b as cited in ATSDR, 1988) observed 8 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 (ATSDR, 1988). The results of this study were further confounded by the small cohort size and the fact that the workers in this facility were exposed to numerous other chemicals (Bahn et al., 1977; Lawrence, 1977).

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 1 week in the manufacture of PCBimpregnated 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

HRP 001 1526

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. The results of the Bertazzi et al. (1987) study are limited by the small number of cancer cases observed and the limited latency period (ATSDR, 1988; Kimbrough, 1987). A major problem in the study design was the one week minimum period of employment required for inclusion in the study and the inclusion in the cohort of workers who had no PCB exposure. This makes it difficult to assume that excess cancer cases are attributable to PCB exposures rather than to other factors. This study also did not show a dose-response relationship or any direct relationship between latency and the disease.

Liss (1989 [unpublished]) conducted a retrospective cohort mortality and cancer incidence study of 1073 workers employed between 1960 and 1976 at a transformer manufacturing plant (Ferranti-Packard Ltd.) in Ontario. Cohorts were defined in this study by exposure intensity and frequency to characterize those who had worked, and those who had never worked, in a job considered to be "exposed." Among females, there were few

001 1527

HRP

deaths; one each occurred due to cancer of the lung and of the breast in the "ever exposed" group, and one death from lung cancer occurred among the "nonexposed" group. Overall mortality among males was less than expected when compared to the population of Ontario. Mortality due to all malignant neoplasms was elevated, but not significantly so, in "ever exposed" workers. This elevation was due primarily to statistically significant increases in deaths from cancer of the brain and nervous system (4 observed vs. 0.8 expected) and prostate (5 observed vs. 1.2 expected). The brain cancer incidence rate among "ever exposed" males was significantly elevated over the expected rate (4 observed vs. 0.9 expected) and the prostate cancer incidence rate was elevated, but not significantly so. Α separate analysis of 159 men who had ever worked in the "highest exposure" jobs indicated that deaths from all malignancies were fewer than expected, and no deaths due to cancer of the brain or prostate were observed. In this "highest exposure" group, no significant increase in cancer incidence rates were observed. Among male workers not known to have been exposed, deaths from malignant neoplasms were less than expected, and deaths due to cancer of the gallbladder or bile ducts were significantly elevated (2 observed vs. 0.11 expected).

From these results, the author (Liss, 1989 [unpublished]) concluded that, because no brain or prostate cancers were observed in the "highest exposure" group, the relationship of these excesses to PCB exposure is not confirmed. In addition, no liver, biliary tract or gall bladder cancers were

69

observed among workers in exposed jobs, nor were deaths or incident cases from tumors of the lymphatic and hematopoietic tissue significantly elevated above expected rates.

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 were 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. (Table B.6-7 of the Phase 1 Report erroneously reports that brain cancer was significantly elevated). No excess deaths were observed from cancers of the rectum or lung, liver biliary 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. Though an excess of malignant melanomas was reported by Bahn et al. (1976; 1977), HRP there were a number of problems with that particular study (discussed above) which confound the results. The authors also point out that mortality may not be the best index of risk for 1529

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.

By contrast, the largest study of PCB exposed workers involved a cohort of 6292 persons employed for at least three months during the period 1946-1976 at the GE Hudson Falls and Ft. Edward facilities (Taylor, 1988). These plants are the alleged source of the PCBs in the Upper River which the Phase 1 Report human health risk assessment is supposed to be about. This study showed no increase in cancer mortality or in overall mortality compared to national averages. Neither deaths due to malignant melanoma, lymphopoietic cancers or the combination of liver, gallbladder and biliary cancers were significantly elevated and brain cancers were well below the expected value. PCB exposure was shown to be negatively associated with cancer mortality (all types combined) and lung cancer (the only cancer 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. This study was initiated when Dr. Taylor, an employee of NIOSH, was assigned to the New York State Department of Health (NYSDOH), and involved collaboration with other scientists at NYSDOH. It is astonishing that Table B.6-7 of the Phase 1 Report fails even to mention the largest and most relevant epidemiological report in existence!

71.

None of cancer incidence and mortality studies cited by the Phase 1 Report, as reviewed in this section, demonstrates a cause-effect relationship between PCB exposure and cancer. Not only do the individual studies fail to show causation, but the weight of the evidence from the studies taken collectively fails to establish any such relationship.

The scientific convention applied in weight-of-theevidence evaluation of epidemiological studies requires (a) the observation of a specific cancer endpoint, and (b) the meeting of other criteria (strength of association, dose-response relationship, temporally correct association, specificity of the association, and biological plausibility) before a causal relationship between an agent such as PCBs and cancer can be inferred (Hill, 1965; Mausan and Kremer, 1985; OSTP, 1985; Kelsey et al., 1986; IARC, 1987). In the PCB studies, small increases in a wide variety of cancer endpoints were seen in different populations with no common thread, and many 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, other than by inferring that PCBs were the causal agent. The statement in the Phase 1 Report that the epidemiological "findings are usually consistent with those from animal research" is not supported by an objective review of these data. Little evidence exists that PCBs are human $_{\rm III}$ RP carcinogens, and the weight of the evidence fails to establish a 100 definitive causal relationship between exposure to PCBs even in high concentrations, and the incidence of cancer in humans. 1531

3.2.1.1.8 Reality Check

The Phase 1 Report itself contains a reality check that demonstrates that the EPA methodology of calculating PCB cancer risk is incorrect. Page B.6-36 of the Phase 1 Report shows that if the EPA cancer slope factor is applied to the maximum allowed OSHA PCB exposure limit in the workplace, an estimated cancer risk of 3.4 in an exposed population of 10 would exist. Since the literature contains numerous epidemiological studies of capacitor worker cohorts having significant long-term high exposures to 42 percent and 54 percent chlorinated PCBs in the workplace, and no virulent cancer epidemic such as would have been predicted by the current EPA approach has been discovered, this is a further demonstration that the Phase 1 Report's treatment of all PCBs as probable human carcinogens is unsupported by empirical evidence and good science.

3.2.1.1.9 Effect of Biodegradation on the Carcinogenic Potency of Hudson River Sediments

As discussed above, the revised analyses of the rodent bioassays indicate that PCBs with an average of 6 chlorines per biphenyl (Aroclor 1260, Clophen A60) are carcinogenic, whereas mixtures that have an average of 3 or 5 chlorines (Clophen A30, Aroclor 1254) are not carcinogenic (Moore, 1991). The correlation of carcinogenicity with the degree of chlorination strongly implies that a conversion of PCBs with 5 or more chlorines to PCBs with 3 or less will reduce the carcinogenicity of the mixture.

Anaerobic degradation processes (see Section 5.0 of these comments) will significantly reduce the carcinogenic risks associated with PCBs in Hudson River sediments. During anaerobic degradation, PCBs sequentially lose chlorines. By this process, highly chlorinated PCBs are reduced to a mixture of mono- and dichlorinated PCBs and eventually primarily to mono-chlorinated byphenyl. In the Upper Hudson River, the most studied system to date, natural anaerobic dechlorination is widespread and nearly ubiquitous. Indeed, anaerobic microorganisms have been shown to have significantly reduced the average number of chlorines per biphenyl in the anaerobic sediments of the Hudson (Abramowicz, 1991).

Anaerobic PCB dechlorination is particularly effective in removing the <u>meta</u> and <u>para</u> chlorines (Abramowicz, 1990). Indeed, one of the signatures of anaerobic degradation is the relative enrichment of mono- and di-ortho substituted PCBs in environmental samples. However, recent studies have suggested that anaerobic dechlorination may remove <u>ortho</u>-chlorines as well (Van Dort and Bedard, 1991). Anaerobic microbial dechlorination alone has the potential, therefore, to reduce not only the degree of chlorination but also the total amount of PCBs. Recent studies have demonstrated that both the number of chlorines and the total level of PCBs tend to decrease with sediment depth. Currently the average number of chlorines per biphenyl for PCB in sediments in the Hudson is less than 3 (Abramowicz, 1991). Over time this degree of chlorination is expected to decrease even

HRP 001 1533

further. Natural biodegradation is therefore reducing any conceivable cancer risk.

When it comes to carcinogenity, the Phase 1 Report ignores the data and the new science and accepts, without question, the cancer slope factor contained in IRIS. When it comes to alleged noncarcinogenic effects of PCBs, the Phase 1 Report is even less scientific, rational, and consistent with EPA guidances.

3.2.1.2 Non-Carcinogenic Effects of PCBs

As the Phase 1 Report notes (p. B.6-25), no Reference Dose for PCBs exists in IRIS. RAGS I set forth a procedure for developing a Reference Dose where none is provided in IRIS. Not only does the Phase 1 Report fail to use this procedure, but it also (a) misrepresents the literature on the noncarcinogenic effects of PCBs, and (b) adopts a Reference Dose that is not supported by either the literature or by any valid science.

Numerous agencies and researchers have examined the association between exposure to PCBs and noncarcinogenic effects in human populations (ATSDR, 1989; EPA, 1988; Kimbrough, 1987; Swain, 1991). The effects attributed to PCB exposures have included chloracne, skin irritation, burning eyes and skin and effects on the liver (Alvares et al., 1977; Baker et al., 1980; Brown and Jones, 1981; Drill et al., 1981; Emmett, 1985; Fishbein et al., 1979, 1982, 1985; Guzelian, 1985; Kimbrough, 1987; Kreiss, 1985; Lawton et al., 1985; Maroni et al., 1981a; Meigs et al., 1954; NIOSH, 1977; Ouw et al., 1976; Smith et al., 1981a, 1981b, 1981c).

HRP 001 1534

Because PCBs are sometimes contaminants in, or are contaminated by, other halogenated aromatic compounds, the interpretation of both animal toxicity and human health effects studies has been difficult. The first commercial use of PCBs was as a low-level additive in chlorinated napthalenes, which are known to be chloracnegenic and to cause liver toxicity. These mixtures were used as solid electrical insulating compounds called "Halowax" or "Chlorowax." Exposure to these mixtures during their manufacture and use resulted in reports of chloracne and liver disease.

Following one such occurrence, Bennett, Drinker, and Warren (1938) conducted studies of rats given doses of individual components of the Halowax compound and reported that "chlorinated diphenyl gave evidence of being the most toxic." A year later, Drinker reported that this compound had been erroneously labeled as chlorinated diphenyl. An authentic sample of 68-percentchlorinated biphenyl proved to be "almost non-toxic" (Drinker, 1939). As noted by NIOSH in 1977, "[t]hese animal experiments reported by Drinker and by Bennett have continued to be erroneously cited" (NIOSH, 1977).

Following a review of the studies that reported toxic effects on the liver, ATSDR (1991) concluded that the effects are not consistent, that they may be within the normal range for the population, and that they have not been shown to be associated with hepatic dysfunction.

With respect to chloracne and PCB exposure, the first incident was reported in 1936 (Jones and Alden, 1936). After ω

performing skin patch tests with suspect chemicals, including PCBs, on PCB-exposed workers, the authors of this report concluded that the cause was an impurity in the benzene used to make the biphenyl, and that "the chlorinated diphenyl can absolutely be absolved as the irriating agent."

The second episode involving PCBs and chloracne occurred in 1950 and 1951, when 14 people were exposed to PCB vapors (reported at 100 μ g/m³) from a leaky heat exchanger, and seven of the 14 developed chloracne (Meigs *et al.*, 1954). A third episode was noted in the early 1960s when 13 of 16 people exposed to vapors from an oven in which PCB-plasticized enamels were being baked were similarly affected (Birmingham, 1964). Other occurrences of chloracne have involved PCB usage abroad, where data on conditions of use or contaminant concentrations do not permit reliable conclusions to be drawn about the cause of the health effect.

In light of the circumstances surrounding these isolated PCB incidents, *i.e.*, impurities in the materials and the heating of PCBs under oxidative conditions, it seems reasonable to attribute the chloracne to contamination by polychlorinated dibenzofurans (PCDFs). As demonstrated by the Yusho/Yucheng incidents, and as confirmed in the laboratory, PCDFs also occur in varying concentrations in commercial PCB mixtures, with higher concentrations in Japanese and European products than in Aroclors. As pointed out by NIOSH (1977), "[c]hloracne has frequently been associated with processes where the PCBs were heated."

HRP 001 1536

Perhaps most revealing, however, is the fact that in the three largest and most recent studies of capacitor manufacturing and transformer repair workers, not one case of chloracne was identified (Smith et al., 1982; Lawton et al., 1985; Emmett et al., 1988). This result is particularly significant because the mean PCB serum levels in one of the studies were two orders of magnitude greater than national population mean levels, and because one of the researchers, Dr. E. Emmett of Johns Hopkins University, was a dermatologist and made a special search for signs of chloracne.

In short, much like the initial hypotheses that surrounded the Yusho incident, subsequent study has shown that any relationship between PCB exposure and chloracne is likely spurious. No reliable study has shown that, absent confounding factors, PCB exposure causes chloracne.

3.2.1.2.1 Neurodevelopmental Reproductive Toxicity of PCBs

A number of studies have been conducted to evaluate the impact that PCBs or other environmental contaminants have in uteri (Fein, 1984; Fein et al., 1984; Gladen et al., 1991; Jacobson et al., 1984a, 1984b, 1985; Rogan et al., 1986a, 1986b, 1988; Taylor et al., 1984). The difficulty associated with evaluating the effects of moderate to low PCB exposures is considerable, especially when considering the question of potential adverse neurodevelopmental effects. The following O discussion reviews a number of the more significant human

epidemiology studies that have focused on this toxicological endpoint.

One of the early studies to evaluate the impact of PCBs on reproductive outcome was conducted by Taylor et al. (1984), who reported a slight decrease in mean birth weight and gestational age of 51 infants born to women with a history of high exposure to Aroclors 1254, 1242, and/or 1016. As with many epidemiological studies, the inability to control a variety of confounding factors compromised the study. According to ATSDR (1989), "the results of this study are considered suggestive but inconclusive because the effects were small and confounding factors such as smoking and alcohol consumption, prenatal care, underlying medical conditions, maternal height, and previous history of low birth weight were not considered."

In a recent report, Harold Humphrey, Ph.D, Michigan Dept. of Public Health, discusses the evidence assciating environmental contaminants and reproductive outcomes. He summarizes a series of studies carried out by Fein, Jacobson and himself as follows:

> "In a Michigan study of 242 children born of mothers who ate sport-caught Lake Michigan Fish and 71 comparison children, investigators used maternal fish consumption and maternal serum and cord blood PCB levels to estimate exposure. They found an association between maternal fish consumption and smaller birth size, and an association between cord blood PCB levels and depressed Brazelton scales and poorer visual recognition memory at seven months of age. Like the Bayley scales used in North Carolina, the Brazelton scales represent an indication of poorer cognitive performance that could possibly be related to learning.

When the Michigan children were evaluated again at age four, researchers found that deficits in body size (weight gain) persisted and indicators of poorer cognitive performance (McCarthy verbal and quantitative performance scales) continued to be present and associated with *in utero* exposure as measured by cord blood PCB levels."

In the same publication, Nigel Paneth, MD, MPH of Michigan State Univ. points out numerous shortcomings in the Jacobson, et al., studies, including:

> the difficulty of assessing exposure through interviews of mothers regarding fish consumption, especially individual fish species. The selection of cases and controls. All mothers with intermediate levels of fish consumption were eliminated from the study. The control sample was restricted to one-third the size of the exposed group, placing "enormous weight on the 71 women chosen (as controls) to represent the entire universe of unexposed mothers." A random, rather than a matched sample, of controls was chosen. This decision may have introduced major confounding factors, since a variety of socioeconomic and other maternal characteristics greatly influence such outcomes as birthweight and cognitive For example, powerful factors such function. as increased consumption of alcohol, caffeine and cold medicines, and lower maternal weight were reported for the exposed mothers relative to the controls. This introduces a strong bias toward adverse reproductive/developmental outcomes in the exposed group that may be impossible to correct.

Paneth also points out that fish consumption did not predict PCB exposure based on maternal serum levels. Therefore, if any relationships of adverse outcomes are real, they must be associated with factors other than PCBs. Obvious chemicals for consideration are pesticides, heavy metals, and chlorinated

dibenzofurans and dioxins. (Unfortunately, these chemicals were not evaluated as part of the study.) This possibility was also recognized by Jacobson, who noted "since behavioral deficits are unrelated to cord blood level, it is possible that toxins other than PCBs found in these same contaminated fish are responsible" (Jacobson, et al., 1985a).

In her review of the Fein et al. (1984) and Jacobson et al. (1983, 1984) studies, Kimbrough (1991) concluded that the findings are difficult to evaluate because: (1) exposure in the population was not well defined; (2) dose response relationships were not well established; (3) other potentially confounding factors, such as exposure to heavy metals were not considered; and (4) the mothers' lifestyle, well-being, and genetic make-up were not considered. Kimbrough concluded that while these findings need to be studied further, it appears that if PCBs make any contribution to the factors affecting birth weight, growth, and development, their contribution is likely to be minor.

Rogan et al. (1986b) reported the results of a prospective study of 912 children born between 1978 and 1982. In that study, cord blood PCB levels, maternal milk PCB levels, and formula PCB levels were measured at birth. Maternal milk PCB levels were measured periodically for the duration of lactation. A modified version of the BINBAS (Jacobson et al., 1984b) was administered to all neonates within 31 days of birth. Multiple regression analysis was used to assess the relationships between birth weight, head circumference, and the BNBAS scores to PCB and DDE levels in maternal milk. Although the authors analyzed for

81[.]

HRP 00/ 1540

PCBs in cord and maternal serum, only milk fat PCB levels were used in the statistical analyses. Parameters used as covariates in the BNBAS analysis included mother's age, education, occupation, smoking history, alcohol consumption, and level of fish consumption during pregnancy, as well as the infant's race, sex, birth weight, age at which the BNBAS was administered, and number of hours since the infant was last fed. In contrast to Jacobson et al., Rogan et al. (1986b) found no association between levels of PCB and birth weight or head circumference. The only significant findings for the BNBAS were for tonicity and reflex cluster scores. Within the tonicity cluster, higher PCB levels were found to correlate with reduced muscle tone and activity, but only at the highest PCB levels. Within the reflex cluster, both PCBs and DDE were associated with hyporeflexia. The PCB effect was observed only at the highest PCB levels whereas the effect of DDE increased as dose increased. The authors concluded that although they observed hypotonicity and hyporeflexia associated with PCBs, "there remains the possibility that even the measured amount of PCBs or DDE is a surrogate for some other agent" (Rogan et al., 1986b).

In a follow-up study, Gladen et al. (1988) assessed mental and psychomotor development in 858 children from the earlier Rogan et al. (1986a, 1986b) studies. In this study, the Bailey Scales of Infant Development were applied at age 6 and 12 months. Again, an estimate of the mother's body burden of PCBs and DDE at birth (*i.e.*, breast milk levels expressed as levels in milk fat at the time of birth) was used as a measure of exposure

to the neonates prior to birth. Neither postnatal PCB or DDE exposure were found to be related to either the Mental Development Index (MDI) or the Psychomotor Development Index (PDI) scores. For prenatal exposure, these authors reported decreasing PDI scores with increasing maternal milk fat PCB levels and increasing MDI scores with increasing maternal milk fat DDE levels. Correlation coefficients for both effects were statistically significant (p<0.05). When discussing their findings Gladen et al. (1988) noted that their observed association between the Bailey Scales of Infant Development and exposures to PCB and DDE "is an observation rather than an experimental finding and is seen for the first time at these exposure levels; it is, of course, possible that it is related to some factor that we did not measure, or to residual uncontrolled confounding."

Gladen and Rogan (1991) recently reported the results of a follow-up study to the Rogan et al. (1986a, 1986b, 1988) cohort. These investigators administered the McCarthy Scales of Children's Abilities at 3, 4, and 5 years of age. In addition, report card grades for at least one school year were evaluated for each child. Exposure measurements were identical to those of Rogan et al. (1986a, 1986b, 1988). Gladen et al. (1991) found no association between transplacental PCB exposure and McCarthy scores. For postnatal exposure, there was an insignificant decrease in verbal and memory scores in the mid-exposure group, but not in the high exposure groups in 3-year-old children. No relationships were observed in the same children at 4 and 5 years

83

of age. The authors concluded that "in these data the association of prenatal PCB exposure with delayed development, seen previously up to 2 years of age in these children, does not persist. We were unable to confirm an association between prenatal PCB exposure and scores on the McCarthy Memory and Verbal Scales at 4 years of age."

Upon review of the Gladen, et al. (1988) study, Cole (1991) commented that

> "The association reported between PCBs and PDI is almost certainly attributable to chance, bias or to residual confounding More importantly, the study provides as much or more evidence in refutation of a causal interpretation of this association as it does in favor. This contracausal evidence appears in the paper's Table II which shows PDIs at 6 ad 12 months according to 'Transplacental' PCB exposure divided into 8 levels. The lowest exposure category (0.0--0.9 ppm PCB) has a PDI score (at 6 months) of 118.0 while the highest (4.0+ ppm PCB) has a score of 110.9. However, the PCB-PDI association is, in fact, found only if these two extreme exposure groups are compared with one another. When one looks within the data there is no suggestion of a continuous (or dose-response) relationship. Indeed. excluding the two extreme exposure groups (both of which include relatively small numbers of children) leaves a pattern that suggests that higher PCBs are associated with a higher PDI. For example, children in exposure levels 2 and 3 (1.0--1.4 and 1.5--1.9 ppm PCB) have a PDI score of 115.0 (N=461) while those in exposure levels 6 and 7 (3.0--3.4 and 3.5--3.9 ppm PCB) have a PDI score of 116.4 (N-52). The information at age 12 months also suggests that any overall association derives primarily from findings in extreme categories.

"Despite the statistical significance of the PCB-PDI findings, chance remains a highly credible explanation. For one reason, if 8 independent evaluations of non-existent

associations are made, there is a 50% chance that one statistically significant finding will emerge. In this study there is only one independent finding regarding PCBs. For another reason, we do not know how many comparisons were actually made. The METHODS section of the paper clearly indicates that observations were made at 9 different ages. (It is not clear whether PDI and MDI were assessed at each age.) Why were findings at 6 and 12 months the only ones presented?

"Bias is a substantial possibility as an explanation of these results. Examiners were aware of the children's nursing status and, no doubt, of many other aspects of each child (i.e., in effect, socio-economic status). There could easily be a tendency to score low those children who appeared poorer (of course, such children would tend to have higher PCB levels) and vice-versa. In this regard it is important to keep in mind that a slight, almost trivial, bias of this sort could produce the weak and inconsistent association that was reported.

"Finally, both residual confounding by factors studied (e.g., education) and complete confounding by those not studied (e.g., income) could produce the weak result seen. While good efforts were made to control confounding for some factors, such efforts are always imperfect. Uncontrolled factors, of course, could have enormous effects.

"In conclusion, this study provides some evidence that PCBs and PDI at ages 6 months and 12 months are not inversely related and may even be directly related. The weak inverse association reported can not be interpreted in casual terms."

While numerous epidemiological studies have investigated the potential relationship between PCB exposure and adverse neurodevelopmental effects, the results of these studies are generally inconclusive (ATSDR, 1989; Kimbrough, 1987, 1991; Paneth, 1991). Although maternal milk PCB levels and cord serum

85

PCB levels may be markers of exposure, it is possible that the observed effects may result from confounding factors such as exposure to other environmental chemicals that are not measured rather than from exposure to PCBs that are now measured routinely. (Rogan et al., 1986b, 1988).

3.2.1.2.2 Reference Doses (RfD) of PCBs The Phase 1 Report proposes to use a Reference Dose (RfD) of 1 x 10⁴ mg/kg-day that is based on studies that have not undergone complete evaluation and critique. Additionally, EPA's own publicly available data systems (IRIS and Health Effects Assessment Summary Tables) do not list an RfD for PCBs. The Phase 1 Report, in effect, arbitrarily selects an RfD for PCBs without formal data analysis or interpretation, without peer review, and without verification by an intra-Agency RfD Workgroup. This violates established EPA policy as set forth in RAGS I and elsewhere. The human health risk assessment in the Phase 1 Report is driven by the inapplicable RfD and, therefore, is invalid.

Even the most cursory review of the literature from which the Phase 1 Report's RfD was derived demonstrates how weak the evidence for it is.

An RfD of 1 x 10^4 mg/kg-day was proposed in 1987 as part of the 1988 Drinking Water Criteria Document for PCBs. However, the RfD was not actually used by EPA in the establishment of drinking water criteria. The proposal was based $\stackrel{\circ}{0}$ upon a rhesus monkey study (Barsotti and Van Miller, 1984).

toxicological basis for the RfD was a source of significant controversy. As a result, the RfD was withdrawn in the final document, and the EPA's Office of Environmental Criteria and Assessment ceased advocating the use of the value.

In the study that provides the basis for the Phase 1 Report's RfD value, Barsotti and Van Miller (1984) investigated the effects of Aroclor 1016 on adult female rhesus monkeys that were fed 0, 0.25, or 1.0 ppm in their diets. Breeding was initiated in the seventh month following the start of the experiment. Each attempt to breed consisted of placing the female in the male's cage for 96 to 120 hours. All animals conceived within 3 attempts, carried their infants to full term, and delivered viable offspring. The only difference observed between exposure groups was a statistically significant (p<0.01) lower mean birth weight in the high dose group when compared to controls. Infants in the control group weighed, on average, 512 g with a standard deviation of 64 g whereas the mean birth weight of infants in the high dose group was 422 g with a standard deviation of 29 g. Therefore, the 1.0 ppm exposure level represents the lowest observable adverse effect level (LOAEL) and the 0.25 ppm exposure level represents the no observable adverse effect level (NOAEL) for rhesus monkeys.

These findings suffer from several problems. First, the differences in birth weights could be the result of non-dose related factors such as genetic differences, pre-pregnancy birth weight, length of gestation, maternal age, and sex of the offspring. There is significant reason to expect that control

HRP 001 1546

animals differed from treatment animals for several of these factors. Barsotti and Van Miller (1984) report that all animals were feral and that the control animals were purchased in 1973, whereas the experimental animals were purchased in 1977. Because the control animals had been in captivity longer than the experimental animals, pre-pregnancy maternal weights were likely greater in the control animals due to the extended controlled diet and limited exercise.

It is also possible that significant differences in genetic makeup exist between the two groups of monkeys. Barsotti (1980) reports that feral animals were captured in India, but did not describe the size of the area from which the animals were captured. Animals obtained from different geographic areas may be different strains or of different genetic makeup; these variations may affect the birth weight of offspring. Finally, because control animals and experimental animals were purchased four years apart, the control animals were likely, on average, to be older than the experimental animals. The authors do not report maternal age or individual maternal body weights in the study.

Second, although birth weights of animals in the high dose group and the control group statistically differed, both groups appear to be within the range of historical measurements. Van Wagenen and Catchpole (1956) report on infant birth weights in their study of physical growth in rhesus monkeys. These authors report a mean and standard deviation birth weight of 465 and 70 g for females and 490 and 60 g for males. These data

001 1547

HRP

suggest that normal birth weights within one standard deviation for animals of both sexes range from 395 g to 550 g. The birth weights of infants (both controls and experimental) in the Barsotti and Van Miller (1984) study appear to have ranged from 393 g to 576 g. On the low end of birth weight, nearly all the animals were probably within the normal range of birth weights. On the high end, however, the control animals in the Barsotti and Van Miller (1984) study may have been moderately heavier than normal. Therefore, the difference between the 1.0 ppm group and controls may be the result of control animals that were not truly representative of experimental animals with respect to birth weights. In addition, although there may have been a statistically significant difference within the high dose and the control animals in the Barsotti and Van Miller (1984) study, there appears to be no significant difference between the high dose and historical measurements (Figure 3.2.1-1).

Third, Barsotti and Van Miller (1984) and Barsotti (1980) provide only limited information on other potential cofactors. Neither report includes the individual birth weights or sex of individual offspring. In addition, although the authors note that all animals carried their infants to term, the length of gestation is not reported. As a result of this lack of data, the effects of possible differences in the maternal age, prepregnancy maternal weight, sex of offspring, or length of gestation cannot be evaluated. Each of these factors could significantly affect birth weights. HRP 001 1548

Fourth, Barsotti and Van Miller (1984) do not discuss the apparent polybrominated biphenyl (PBB) contamination of monkey chow, which was previously reported elsewhere by Barsotti (1980). During analysis of subcutaneous tissues, PBBs were detected in animals from the 0.025 ppm group. Barsotti (1980) concludes that "the 0.025 ppm Aroclor 1016 group received PBB diets for an undetermined time due to a mix up at the pelleting site." Although Barsotti (1980) does not report PCB feed analysis for the other dose groups, the possibility exists that other feeds were also contaminated.

Finally, in addition to the PBB contamination of the monkey chow, a review of the gas chromatograms suggests that other highly chlorinated compounds were present which were tentatively identified by Barsotti and Von Miller as PCBs, but which probably were not. The presence of these compounds in samples analyzed as part of the study demonstrates another contamination problem that further weakens the validity of the study in linking PCB exposure to effects in the monkeys.

In summary, a number of methodological problems with the Barsotti and Van Miller (1984) study must be evaluated, and important questions should be answered before this study should be considered for use in the establishment of regulatory criteria. These are:

•	Did pre-pregnancy	maternal	body	weights	influence	HR
	birth weights?					q

100

- Did maternal age influence birth weights?
- Did PBB contamination of feed and the presence of other contaminants confound the results?

- Did the ratio of male/female infants impact the results?
- Could length of gestation have affected the outcome?

The Phase 1 Report, therefore, is in error when it used an RfD of 1 x 10^4 mg/kg-day.

3.2.1.2.3 Conclusion

The Phase 1 Report's evaluation of the noncarcinogenic health effects of PCBs on humans is flawed. It does not conform to the procedures set forth in RAGS I; it misrepresents the literature on the subject; and it adopts an unapproved, unsupportable Reference Dose. The Phase 1 Report's risk assessment based on such Reference Dose is therefore scientifically indefensible.

3.2.1.3 References

Allen, J.R., L.A. Carsten and L.J. Abrahamson. 1976. Responses of rats exposed to polychlorinated biphenyls for 52 weeks. I. Comparison of tissue levels of PCB and biological changes. Arch. Environ. Contam. Toxicol. 4:409. (cited in USEPA, 1984)

Alvares, A.P., A. Fischbein, K.E. Anderson A. Kappas. 1977. Alteration in drug metabolism in workers exposed to polychlorinated biphenyls. *Clin Pharmacol. Ther. 22*:140. (cited in ATSDR, 1989).

ATSDR. 1989. Toxicological Profile for Polychlorinated Biphenyls. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry.

Bahn, A.K., I. Rosenwaike, N. Herrmann, P. Grover, J. Stellman, and K. O'Leary. 1976. Melanoma after exposure to PCBs. Letter to the Editor. N. Engl. J. Med. 295:450.

Bahn, A.K., P. Grover, I. Rosenwaike, K. O'Leary, and J. Stellman. 1977. Letter to the Editor. N. Engl. J. Med. 296.108.

Baker, E.L., P.J. Landrigan, and C.J. Glueck. 1980. Metabolic consequences of exposure to polychlorinated biphenyls (PCB) in sewage sludge. Am. J. Epidemiol. 112:553-563.

Balter, N.J., D.J. Eatough, and S.L. Schwartz. 1983. Application of physiological pharmacokinetic modeling to the design of human exposure studies with environmental tobacco smoke. Georgetown University Medical Center, Washington, DC and Brigham Young University, Provo, UT. pp. 179-188.

Barsotti, D.A. 1980. Gross Clinical and Reproductive Effects of Polychlorinated Biphenyls in the Rhesus Monkey. Thesis submitted to University of Wisconsin for the degree of Doctor of Philosophy. August.

Barsotti, D.A. and J.R. Allen. 1975. Effects of polychlorinated biphenyls on reproduction in the primate. Fed. Proc. 34:338.

Barsotti, D.A., R.J. Marlar, and J.R. Allen. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet. Toxicol. 14:99-103.

Barsotti, D.A. and J.P. Van Miller. 1984. Accumulation of a commercial polychlorinated biphenyl mixture (Aroclor 1016) in adult rhesus monkeys and their nursing infants. *Toxicology* 30:31-44.

Bennett, G.A., C.K. Drinker, M.F. Warren. 1938. "Morphological Changes in the Livers of Rats Resulting from Exposure to Certain Chlorinated Hydrocarbons." J. Ind. Hyg. and Toxicol.

Bertazzi, P.A., L. Riboldi, A. Pesatori, L. Radice and C. Zocchetti. 1987. Cancer mortality of capacitor manufacturing workers. Am. J. Ind. Med. 11:165-176.

Birmingham, D.J. 1964. Occupational Dermatology: Current Problems, Skin, <u>38</u>. February.

Brown, D.P. and M. Jones. 1981. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. Arch. Environ. Health 36(3):120-129.

Brown, D.P. 1987. Mortality of workers exposed to polychlorinated biphenyls - An update. Arch. Environ. Health 42(6): 333-339.

Brown, J.F.J., D.L. Bedard, M.J. Brennan, J.C. Carnahan, H. Feng and R.E. Wagner. 1987. Polychlorinated biphenyl dechlorination in aquatic sediments. *Science* 236:709-712.

HRP

40 C.F.R. Section 761.3. Definition of PCBs.

Camanzo, J., C.P. Rice, D.J. Jude, and R. Rossmann. 1987. Organic priority pollutants in nearshore fish from 14 Lake Michigan tributaries and embayments, 1983. J. Great Lakes Res. 13(3):296-309. Drill, V.A., S.L. Freiss, H.W. Hays, T.A. Loomis, and C.B. Shaffer. 1981. Potential Health Effects in the Human from Exposure to Polychlorinated Biphenyls (PCBs) and Related Impurities. (Unpublished report). Arlington, VA: Drill, Freiss, Hays, Loomis and Shaffer, Inc. (cited in ATSDR, 1989).

2

HRP

100

1552

Drinker, C.K. 1939. "Further Observations on the Possible Systemic Toxicity of Certain of the Chlorinated Hydrocarbons with Suggestions for Permissible Concentrations in the Air of Workrooms. J. Ind. Hyg. and Toxicol. 155-159.

Emmett, E.A. 1985. Polychlorinated biphenyl exposure and effects in transformer repair workers. Environ Health Perspect 60:185-192. (cited in ATSDR, 1989).

Emmett, E.A., Maroni, J.M. Smith, B.K. Levin, J. Jeffrys. 1988. Studies of Transformer Repair Workers Exposed to PCBs: 1. Study Design, PCB Concentrations, Questionnaire, and Clinical Examinations, Am. J. Ind. Med., <u>13</u>: 415-427.

Fagan and Singer. 1983. Infant recognition memory as a measure of intelligence In: Advances in Infancy Research. L.P. Lipsitt (ed.) Vol. 2, pp. 31-78. Norwood, NJ. (as cited in Jacobson et al., 1985b)

Fein, G.G., S.W. Jacobson, P.M. Schwartz, and J.L. Jacobson. 1981. Intrauterine exposure to polychlorinated biphenyls: Effects on infants and mothers. University of Michigan, School of Public Health, Ann Arbor, MI. 215 pp. (cited in Swain, 1991).

Fein, G.G., S.W. Jacobson, P.M. Schwartz, and J.L. Jacobson. 1983a. Environmental toxins and behavioral development: A new role for psychological research. Am. Psych. 38(11):1188-1197. (cited in Swain, 1991).

Fein, G.G., S.W. Jacobson, P.M. Schwartz, and J.L. Jacobson. 1983b. Intrauterine exposure of humans to PCBs: Newborn effects. Final report to the U.S. Environmental Protection Agency, Grosse, MI. 54 pp. (cited in Swain, 1991).

Fein, G.G., S.W. Jacobson, P.M. Schwartz, J.L. Jacobson, and J.K. Dowler. 1983c. Prenatal exposure to polychlorinated biphenyls: Effects on birth size and gestational age. *J. Pediatr.* 105:315-320. (cited in Swain, 1991).

Feldman, R.G., N.L. Ricks, and E.L. Baker. 1980. Neuropsychological effects of industrial toxins: A review. Am. J. of Ind. Med. 1::211-227.

Fischbein A. 1985. Liver function tests in workers with occupational exposure to polychlorinated biphenyls (PCB)s:

Comparison with Yusho and Yu-Cheng. Environ Health Perspect 60:145-150. (cited in ATSDR, 1989)

Fischbein A., J.N. Rizzo, S.J. Solomon, and M.S. Wolff. 1982. Dermatological findings in workers with occupational exposure to polychlorinated biphenyls. Br. J. Ind. Med. 42(6):426-430. (cited in ATSDR, 1989)

Fischbein A., M.S. Wolff, Berstein, and I.J. Selikoff. 1982. Dermatological findings in capacitor manufacturing workers exposed to dielectric fluids containing polychlorinated biphenyls. Arch. Environ. Health. 37:69-74. (cited in ATSDR, 1989).

Fischbein A., M.S. Wolff, R. Lilis, J. Thornton, and I.J. Selekoff. 1979. Clinical findings among PCB-exposed capacitor manufacturing workers. Ann NY Acad Sci. 320:703-715.

General Electric Company. 1990. Research Program for the Destruction of PCBs, Ninth Progress Report. General Electric Corporate Research and Development, Schenectady, NY.

Gladen, B.C., W.J. Rogan, P. Hardy, J. Thullen, J. Tingelstad, and M. Tully. 1988. Development after exposure to polychlorinated biphenyls and dichlorodiphenyl dichloroethene transplacentally and through human milk. J. Ped. 113:991-995.

Gladen, B.C. and W.J. Rogan. 1991. Effects of Perinatal Polychlorinated Biphenyls and Dichlorodiphenyl Dichloroethene on later Development. J. Pediatrics 119(1 part 1):58-63.

Gustavsson, P., C. Hogstedt, and C. Rappe. 1986. Short-term mortality and cancer incidence in capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). Am. J. Ind. Med. 10:341-344.

Guzelian, P.S. 1985. Clinical evaluation of liver structure and function in humans exposed to halogenated hydrocarbons. *Environ. Health Persp.* 60:159-164. (cited in ATSDR, 1991)

Jacobson, S.W., J.L. Jacobson, P.M. Schwartz, and G.G. Fein. 1983. Intrauterine exposure of human newborns to PCBs: Measures of exposure In: *PCBs: Human and Environmental Hazards*. F.M. D'Itri an M.A. Kamrin (eds.) Butterworth Publishers, Ann Arbor Science Books, Ann Arbor, MI. pp. 311-343.

Jacobson, J.L., G.G. Fein, S.W. Jacobson, P.M. Schwartz, and J.K. Dowler. 1984a. The transfer of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PCBs) across the human placenta and into maternal milk. AJPH 74(4):378-379.

Jacobson, J.L., S.W. Jacobson, P.M. Schwartz, G.G. Fein, and J.K. Dowler. 1984b. Prenatal exposure to an environmental toxin: A test of the multiple effects model. *Develop. Psychol.* 20(4):523-532.

Jacobson, J.L., S.W. Jacobson, G.G. Fein, and P.M. Schwartz. 1984c. Factors and clusters for the brazelton scale: An investigation of the dimensions of neonatal behavior. Develop. Psych. 20(3):339-353.

Jacobson, J.L., S.W. Jacobson, and G.G. Fein. 1985a. Intrauterine exposure to environmental toxins: The significance of subtle behavioral effects. In: *Environmental Stressors*. Hawthorne Press, Inc. pp. 125-137.

Jacobson, S.W., G.G. Fein, J.L. Jacobson, P.M. Schwartz, and J.K. Dowler. 1985b. The effect of intrauterine PCB exposure on visual recognition memory. *Child Develop.* 56:853-860.

Jacobson, J.L. and S.W. Jacobson. 1988. New methodologies for assessing the effects of prenatal toxic exposure on cognitive functioning in humans In: *Toxic Contaminants and Ecosystem Health: A Great Lakes Focus.* M.S. Evans (ed.) Wiley & Sons Publishing Company, New York. pp. 373-387.

Jacobson, J.L., H.E.B. Humphrey, S.W. Jacobson, S.L. Schantz, M.D. Mullin, and R. Welch. 1989. Determinants of polychlorinated biphenyls (PCBs), polybrominated biphenyls (PBBs), and dichlorodiphenyl trichloroethane (DDT) levels in the sera of young children. AJPH 79(10):1401-1404.

Jacobson, J.L., S.W. Jacobson, and H.E.B. Humphrey. 1990a. Effects of exposure to PCBs and related compounds on growth and activity in children. *Neurotoxicol. Teratol.* 12:319-326.

Jacobson, J.L., S.W. Jacobson, and H.E.B. Humphrey. 1990b. Effects of in utero exposure to polychlorinated biphenyls and related contaminants on cognitive functioning in young children. J. Pediatrics 116(1):38-45.

Jones, J.W. and H.S. Alden. 1936. An acneform dermatergosis. Arch. Dermatol. Syphilol. 33:1022-1034. (cited in Kimbrough, 1987).

Kimbrough, R.D., R.A. Squire, R.E. Linder, J.D. Strandberg, R.J. Montali, and V.W. Burse. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl aroclor 1260. J. Natl. Cancer Inst. 55:1453-1459.

Kimbrough, R.D. 1987. Human health effects of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs). Ann. Rev. Pharmacol. Toxicol. 27:87-111.

Koller, L.D. 1977. Enhanced polychlorinated biphenyl lesions in Moloney leukemia virus-infected mice. *Clin. Toxicol. 11*(1):107-116. (cited in USEPA, 1984)

Kreiss, K. 1985. Studies on populations exposed to polychlorinated biphenyls. *Environ. Health Perspect.* 60:193-199. (cited in ATSDR, 1989)

Lawrence, C. 1977. PCB and melanoma. Letter to the Editor. N. Engl. J. Med. 296:108.

Lawton, R.W., J.F. Brown Jr., M.R. Ross, J. Feingold et al. 1985. Effects of PCB exposure on biochemical and hematological finding in capacitor workers. *Environ. Health Perspect.* 60:165-184. (cited in ATSDR, 1989)

Marconi, N., A. Columbi, G. Arbosti, S. Cantoni, and V. Foa. 1981a. Occupational exposure to polychlorinated biphenyls in electrical workers. II. Health Effects. Br. J. Ind. Med 38:55-60. (cited in ATSDR, 1989)

Maronpot, R.R., C.A. Montgomery, G.A. Boorman, and E.E. McConnell. 1986. National Toxicology Program nomenclature for hepatoproliferative lesions of rats. *Toxicol. Pathol.* 14(2):263-273.

McConnell, E.E., H.A. Solleveld, J.A. Swenberg, and G.A. Boorman. 1988. Guidelines for combining neoplasms for evaluation of rodent carcinogenesis studies. In: Carcinogenicity: The Design, Analysis and Interpretation of Longterm Animal Studies. ILSI Monographs. H.C. Grice and J.L. Cimineri (eds.) Springer-Verlag. New York, NY. pp. 183-196.

Meigs J.W., J.J. Albom, and B.L. Kartin. 1954. Chloracne from an unusual exposure to Arochlor. J. Am. Med. Assoc. 154:1417-1418. (cited in ATSDR, 1989)

Moore, J.A. 1991. Reassessment of liver findings in five PCB studies for rats. Institute of Evaluating Health Risks. July 1, 1991.

National Cancer Institute (NCI). 1978. Bioassay of Aroclor 1254 For Possible Carcinogenicity. <u>NCI Carcinogenesis Technical</u> <u>Report No. 38</u>.

NIOSH. 1977. Criteria for a Recommended Standard. Occupational Exposure to Polychlorinated Biphenyls (PCBs). Rockville, MD. U.S. Department of Health, Education, and Welfare, Public Health Service, Centers for Disease Control. No. 77-225. September. (as cited in ATSDR, 1989)

001 1555

HRP

OSTP. 1985. Chemical Carcinogens: A Review of the Science and its Associated Principles, February 1985. Environmental Protection Agency, Office of Science and Technology Policy, Washington, DC. Federal Reg. 50(50):10380(b). March 14.

Ouw, H.K., G.R. Simpson, and D.S. Siyali. 1976. Use and health effects of Aroclor 1242, a polychlorinated biphenyls, in an electrical industry. Arch. of Environ. Health (July/August):189-194.

Rogan, W.J., B.C. Gladen, K.L. Hung, S.L. Koong, J.S. Taylor, Y.C. Wu, D. Yang, N.B. Ragan, and C.C. Hsu. 1988a. Congenital poisoning by polychlorinated biphenyls and their contaminants in Taiwan. Science 241::334-335.

Rogan, W.J., B.C. Gladen, J.D. McKinney, N. Carreras, P. Haardy, J. Thullen, J. Tinglestad, and M. Tully. 1986b. Polychlorinated biphenyls (PCBs) and dichlorodiphenyl dichloroethene (DDE) in human milk: Effects of maternal factors and previous lactation. Am. J. Public Health. 76:172-177.

Rogan, W.J., B.C. Gladen, J.D. McKinney, N. Carreras, P. Haardy, J. Thullen, J. Tinglestad, and M. Tully. 1986c Neonatal effects of transplacental exposure to PCBs and DDE. J. Pediat. 109:335-341.

Safe, S. 1990. Polychlorinated biphenyls (PCBs), Dibenzo-p-dioxins (PCDDs), Dibenzofurans (PCDFs), and related compounds: Environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). Toxicology 21(1):51-88.

Schaeffer, E., H. Greim, and W. Goessner. 1984 Pathology of Chronic Polychlorinated Biphenyl (PCB) Feeding in Rats. <u>Toxicol.</u> <u>Appl. Pharmacal.</u> 75:276-288.

Schroeder, R.A. and C.R. Barnes. 1983. Polychlorinated Biphenyl Concentrations in Hudson River Water and Treated Drinking Water at Waterford, New York. USGS Water - Resources Investigations Report 83-4188. USGS, Albany, NY.

Schwartz, P.M., S.W. Jacobson, G. Fein, J.L. Jacobson, and H.A. Price. 1983. Lake Michigan fish consumption as a source of polychlorinated biphenyls inhuman cord serum, maternal serum and milk. AJPH 73:293-296.

Smith A.B., J. Schloemer, and L.K. Lowry. 1981a. Cross-Sectional Medical Survey of a Group of Workers Occupationally Exposed to Polychlorinated Biphenyls (PCBs) at an Electrical Equipment Manufacturing Plant. Cincinnati, Ohio: National Institute for Occupational Safety and Health, Division of Surveillance, Hazard Evaluations and Field Studies, and Lipid

Research Center, University of Cincinnati Medical Center (as cited in Drill et al. 1981). (cited in ATSDR, 1989)

Smith A.B., J. Schloemer, and L.K. Lowry. 1981b. Cross-Sectional Medical Survey of Two Groups of Workers Occupationally Exposed to Polychlorinated Biphenyls (PCBs) in the Maintenance, Repair and Overhaul of Electrical Transformers. Cincinnati, Ohio: National Institute for Occupational Safety and Health, Division of Surveillance, Hazard Evaluations and Field Studies, and Lipid Research Center, University of Cincinnati Medical Center (as cited in Drill et al. 1981). (cited in ATSDR, 1989)

Smith A.B., J. Schloemer, and L.K. Lowry. 1981c. Metabolic and Health Consequences of occupational Exposure to Polychlorinated Biphenyls (PCBs). Cincinnati, Ohio: National Institute for Occupational Safety and Health, Division of Surveillance, Hazard Evaluations and Field Studies, and Lipid Research Center, University of Cincinnati Medical Center (as cited in Drill et al. 1981). (cited in ATSDR, 1989)

Smith, A.B., J. Schloemer, L.K. Lowry et al. 1982. Metabolic and health consequences of occupational exposure to polychlorinated biphenyls. <u>Brit. J. Ind. Med. 39:</u> 361-369.

Swain, W.R. 1991. Effects of organochlorine chemicals on the reproductive outcome of humans who consumed contaminated great lakes fish: An epidemiologic consideration. *Toxicol. Environ. Health* 33::587-639.

Taylor, P.R., C.E. Lawrence, H.L. Hwand and A.S. Paulson. 1984 Polychlorinated biphenyls: Influence on birthweight and gestation. AJPH 74:1153-1154.

Taylor, P.R. 1988. The Health Effects of Polychlorinated Biphenyls. ScD Thesis. Harvard School of Public Health. June 1988.

USEPA. 1984. Health Effects Assessment for Polychlorinated Biphenyls (PCBs). U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 540/1-86/004. September.

USEPA. 1986a. Guidelines for Carcinogen Risk Assessment. Fed. Reg. 45(79):347-357.

USEPA. 1986b. Proliferative Hepatocellular Lesions of the Rat: Review and Future use in Risk Assessment. U.S. Environmental Protection Agency, Washington, DC. EPA 625/3-86/011. February.

USEPA. 1988. Ambient Water Quality Criteria for Polychlorinated Biphenyls. U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, DC.
Van Dort, H.M. and D.L. Bedard. 1991. Reductive ortho and meta dechlorination of a polychlorinated biphenyl congener by anaerobic microorganisms. Appl. Environ. Microbiol. 57:1576-1578.

3.2.2 Current Exposures To Hudson River <u>PCBs Do Not Present Unacceptable Risks</u>

The Phase 1 Report's analysis of potential exposure pathways and exposure concentrations are unduly conservative. When combined with EPA's erroneous position on cancer potency, EPA's risk assessment becomes even more unrealistic and inaccurate. When more realistic yet conservative assumptions are employed, the hypothetical risks due to exposure to PCBs in the Hudson River are significantly below those presented in the Phase 1 Report and indeed are in the acceptable risk range.

At the outset, the exposure concentrations employed by the Phase 1 Report include not only exposures from PCBs derived from contaminated sediments, but also exposures from sources above the U.S. Geological Survey monitoring station at State Route 197, such as the remnant deposits. The scope of the Reassessment RI/FS is to:

> "reassess the 1984 no action decision of the U.S. Environmental Protection Agency (USEPA) concerning <u>sediments</u> contaminated with polychlorinated biphenyls (PCBs) in the Upper Hudson River" (p. I-1) (emphasis supplied).

GE has pointed out to EPA in its Phase 1 Work Plan comments and in a letter to the EPA Project Manager dated June 14, 1991, that the existing data on fish and water PCB levels reflects contributions from both sediments and upstream sources. Therefore, it is not possible for EPA to perform a "baseline" risk assessment because an unspecified portion of the risk is due to sources outside the scope of the study. Thus, the preliminary health risk assessment in the Phase 1 Report does not reflect in

a meaningful way the risk associated with the PCBs in the <u>sediments</u> in the Upper Hudson River.

As pointed out in Section 2.0 of these comments, without fully understanding the dynamics of PCBs within this complex physical, biological, and chemical system, it is impossible to prepare any assessment of risks or perform an analysis of remediation benefits. If one does not understand which PCBs are going where and by what means within the system, one cannot know what PCBs result in exposures and what PCBs to control if it is desirable to reduce exposures. Just as the failure to arrive at this understanding has prevented the Phase 1 Report from adequately characterizing the site, it also prevents the Phase 1 Report from validly developing appropriate exposure pathways.

The Phase 1 Report's interim risk assessment determines quantitative estimates for five exposure pathways.

- Fish consumption
- Drinking water
- Dermal contact with river water
- Dermal contact with river sediments
- Accidental ingestion of river sediments

The Phase 1 Report concludes that the most significant source of exposure is fish consumption. The remaining sources of exposure are estimated to result in risk estimates that are 2 to 4 orders of magnitude lower than the risks associated with the fish consumption pathway and thus present negligible risks to human health. The Report discusses but does not develop quantitative estimates for other potential exposure pathways, which were correctly not used as the basis for estimating risks.

101

HRP 001 1560

GE agrees with EPA's conclusions concerning the relative significance of the various exposure pathways. EPA has preliminarily determined that fish consumption is the only potential route of exposure that could conceivably result in intakes that are of toxicological interest. GE has, therefore, focused on this route of exposure and has developed alternative exposure and risk estimates that are scientifically more accurate than those presented in the Phase 1 Report.

3.2.2.1 Fish Consumption

Accurate characterization of the risks associated with human ingestion of fish depends on the use of appropriate, sitespecific fish consumption rates. Most of the fish consumption estimates that are reported in the scientific literature are based on national surveys or are specific to a particular region of the United States (Puffer *et al.*, 1981; Pierce *et al.*, 1981; Humphrey, 1978; Javitz, 1980; Rupp *et al.*, 1980). Many of these surveys have not adequately characterized the types of fish consumed, nor have they distinguished between the consumption of commercially-harvested and recreationally-harvested fish (Javitz, 1980; EPA, 1989a). Thus, these surveys overestimate consumption of sport-caught fish from waterbodies like the Hudson River where fishing is limited to the recreational angler.

In addition, factors such as regional variations in consumption of preferred species, the availability of those species, ease of access to productive fisheries, length of fishing season, and cultural heritage can greatly influence fish ingestion habits. When characterizing potential exposures and

HRP

100

1561

associated human health risks from PCBs found in the Hudson River, the most accurate state- or region-specific data should be used to account for differences in fish consumption (EPA, 1989b).

3.2.2.1.1 EPA's Estimates of Fish Consumption

In developing its Ambient Water Quality Criteria (AWQC), EPA uses a human fish consumption estimate of 6.5 g/day (EPA, 1984). Of this total consumption, 1.7 g/day is attributed to freshwater fish and 4.8 g/day to estuarine fish (EPA, 1989a; Table 2-14). The EPA estimate is based on the national average per capita rate of fish consumption and includes all commercially-harvested and recreationally-caught freshwater and estuarine fish and shellfish (EPA, 1989a). Although the EPA values may be appropriate for estimating an average consumption rate for the U.S. population as a whole, it is inappropriate for estimating actual regional consumption or consumption by recreational anglers or other subpopulations.

EPA has recommended the use of two other fish consumption estimates when site-specific data are unavailable. A value of 20 g/day, which represents the average consumption of marine, estuarine, and freshwater fish (USDA 1984), is recommended as an estimate of consumption of all types of fish by the general population of the United States. A value of 30 g/day is recommended as an average consumption rate for recreational anglers (EPA, 1989b). The latter value is used by the Phase 1 Report and is inappropriate for the reasons discussed below.

HRP 001 1562

3.2.2.1.2 Reported Basis of the 30 g/day Estimate

The EPA consumption rate of 30 g/day is the average of the median values reported for sport anglers by Puffer et al. (1981) and Pierce et al. (1981). Puffer et al. (1981) investigated the fish consumption habits of successful marine fishermen on Los Angeles Harbor. Pierce et al. (1981) interviewed fishermen on Commencement Bay, a marine/estuarine fishery in Puget Sound near Tacoma, Washington. Published studies indicate that the consumption of marine and estuarine fish far exceeds the consumption of freshwater fish (EPA, 1989; Rupp et al., 1980). Therefore, application of marine/estuarine derived estimates of fish consumption is inappropriate.

There are several reasons why consumption rates based on marine or estuarine studies are likely to overstate the amount of fish eaten from the Upper Hudson River or other freshwater bodies. First, both Puffer et al. (1981) and Pierce et al. (1981) investigated consumption of marine and estuarine species by successful fishermen but did not ask anglers to characterize their consumption of freshwater species. While many different species of fish available in the marine waters of the studies cited, only freshwater species are available in the Upper Hudson River. In addition, since marine fish tend to be considerably larger than freshwater species, a single marine fish is likely to provide several meals while a single freshwater fish likely provides only one fish meal. Finally, as marine environments are generally more fertile and productive than riverine environments,

HRP 001 1563

more fish per unit area are expected, increasing the relative ease of catching marine species. Consequently, consumption rates from marine fisheries are considerably higher than rates from freshwater fisheries. For these reasons, marine and estuarine studies are not appropriate for use in approximating fish consumption from the Upper Hudson River.

3.2.2.1.3 Availability of Region-Specific Data

State- and region-specific consumption data are available from an angler survey conducted in New York State (NYSDEC, 1990). Because there are a number of region-specific factors that can affect overall consumption for a specific area, this region-specific data must be used to characterize consumption more accurately. EPA (1991a) has stated that the NYSDEC (1990) data support the estimate of 30 g/day recommended for sport anglers by the EPA (1989b). However, a closer evaluation of the NYSDEC data indicates that this is not completely accurate. The NYSDEC (1990) report estimates that the average New York angler consumes an average of 45.1 meals of fish annually. If the average meal size is 227 g (1/2 pound), the average angler consumes approximately 28 g of fish daily. However, this is an estimate of consumption of all types of fish available to the angler, including market, restaurant, gift, and sport-caught fish. In other words, a variety of fresh, frozen, and canned, marine, estuarine, and freshwater fish obtained from sources both within and outside of New York State are available to and are most likely consumed by New York anglers. The NYSDEC

HRP 001 1564

(1991) value of 45.1 fish meals per year clearly overestimates consumption of fish from the Upper Hudson River, because only sport-caught freshwater species can be obtained there. NYSDEC (1990) did not report on statewide consumption of sport-caught fish alone.

Another factor not detailed in the NYSDEC (1990) fish consumption rate is the fact that sport-caught fish are likely to be taken from several waterbodies in the State, rather than from a single source. Thus, the use of NYSDEC's (1990) value to estimate consumption from a single waterbody such as the Upper Hudson River is unreasonable, because anglers are likely to fish in a number of fishing locations.

This view is supported by an evaluation of the effort reported by anglers in the New York angler survey (NYSDEC, 1990). Table 47 (NYSDEC, 1990) reports fishing efforts by Albany County residents. According to Table 47, Albany County residents spent only 19.9 percent of their total angler-days fishing within Albany County, while 12.5 percent of angler-days were spent in Rensselaer County, 10.6 percent in Saratoga County, and 10.2 percent in Warren County. Thus, 47 percent of the total anglerdays spent by Albany County residents were spent fishing in counties not adjacent to the Upper Hudson. Of the 53 percent of the angler-days spent in counties that are adjacent to the river, it is reasonable to conclude that anglers fished other lakes, ponds, streams, or rivers at least a portion of the time. Therefore, it is unlikely that all of their freshwater fish intake would come from the Upper Hudson.

001 1565

HRP

3.2.2.1.4 Other Studies of Freshwater Fish Consumption

A review of the available fish consumption data from other studies is useful in providing perspective on regional variations in fish consumption (Table 3.2.2-1).

National Studies

Rupp et al. (1980) used data developed by NPD Research to estimate consumption by age group and by region of the country. Rupp et al. (1980) report that in addition to regional variations in fish consumption, there are substantial variations in fish consumption patterns among individuals living in the Middle Atlantic region of the United States (New York, New Jersey, and Pennsylvania). The authors report that only 10.6 percent of the fish consumers surveyed in that region consumed freshwater fish, whereas 92.2 percent of the individuals surveyed consumed saltwater fish (Rupp et al., 1980). These results clearly suggest that most people in that region do not eat freshwater fish.

In estimating a freshwater fish consumption rate, Rupp et al. (1980) report that the average rate of consumption of all adults sampled was 0.35 kg/year (0.96 g/day) and the median rate of consumption was 0 g/day. Based on additional information provided by Rupp et al., the average rate of consumption among those members of the population sampled who indicated that they consumed freshwater fish can be estimated to be 9 g/day. This estimate would include freshwater fish from all commercial and recreational sources in those states.

HRP 001 1566

Using the same data developed by NPD Research for the NMFS survey, SRI International, Inc., (Javitz, 1980) calculated average fish consumption rates among fish consumers in the United States. Unfortunately, the distinction between sport-caught and purchased fish was not maintained in the original compilation of the data (EPA, 1989a). Javitz estimated that the total mean rate of consumption was 14.3 g/day (EPA, 1989a). When Javitz's species-specific consumption rate estimates are separated by marine/estuarine and freshwater species (EPA, 1989a), the estuarine/freshwater fish portion of the total consumption rate can be estimated to average 6.8 g/day. This estimate includes sport-caught and commercially obtained bluegills, crab, herring, lobster, oysters, scallops, shrimp, and other estuarine species that would not be found in the Upper Hudson, where there is no tidal influence.

Regional or Statewide Studies

ChemRisk (1991a) conducted a statewide mail survey of Maine's licensed resident anglers for the 1989/90 ice fishing and 1990 open water fishing season. Anglers were asked to indicate the number, species, and average length of fish caught and consumed from Maine's inland fisheries, and to indicate where the fish were obtained. Analysis of the data indicated that the median rate of consumption from all types of fisheries in the State was 2.0 g/day with a mean consumption (77th percentile) of 6.4 g/day. For river and stream fisheries, median consumption was 0.99 g/day with a mean consumption of 3.7 g/day (81st percentile). The data indicated that only about 44 percent of

001 1567

HRP

the survey respondents who reported that they had caught and consumed fish had obtained a portion of that fish from any of the state's rivers or streams. Results of the survey indicate that participation and effort are much greater on lakes and ponds than they are on rivers and streams (ChemRisk, 1991b).

The Wisconsin Division of Health (WDH, 1987) initiated a study in 1985 to assess the participation and consumption habits of Wisconsin anglers. Based on the data obtained from the survey respondents, WDH estimated that the mean freshwater consumption rate was 12.3 g/day. The median consumption rate was estimated to be 6.2 g/day (MPCA, 1990).

West et al. (1989) estimated an average consumption rate of 18.3 g/day for Michigan anglers. However, this consumption rate was based on consumption of all types of selfcaught, purchased, gift, and restaurant-purchased fish. The data reported in Table 19 of the West et al. (1989) report indicates that only 39 percent of the meals reported were sport-caught, while the remaining fish meals were restaurant-purchased, storebought, or gift fish. If this percentage is applied to the rate of total fish consumption (18.3 g/day) estimated by West et al. (1989), it can be estimated that, on average, Michigan anglers consumed only about 7 g/day of sport-caught fish.

Waterbody-Specific Studies

Honstead et al. (1971) conducted a diet recall survey of 10,900 individuals from households in which there was at least one angler who fished the Columbia River in the Hanford area of Washington. The average size of a fish meal was estimated to be

HRP 001 1568

approximately 200 grams per meal, and individuals reportedly consumed an average of 14 such meals per year. Thus, the annual average rate of consumption was 2.8 kilograms per year, or 7.7g/day.

In a creel survey of recreational anglers who fished in the same area of the Columbia River, the distribution of species reeled and consumed was similar to that reported in the Honstead et al. (1971) diet recall survey. From the data generated from the Soldat (1970) creel survey, an average consumption rate of 1.8 g/day can be estimated.

In a fishery study of the Savannah River, Turcotte (1983) reported that average consumption by anglers on the nontidal portion of the river study area was 11.3 kg/year. This estimate was based on creel survey data for angler days, trips taken, and total fish weight caught. In calculating the consumption rate, it was assumed that 50 percent of the fish was edible. EPA (1989b) suggests that 30 percent is a more reasonable estimate of the edible portion of finfish. If it is assumed that 30 percent of the fish is edible, then maximum consumption for the average angler will be 6.8 kg/year or 18.6 g/day. However, it is likely that the average angler shares most if not all of his or her catch with other fish-consuming family members (Pierce et al., 1981; ChemRisk, 1991c, 1991d). If it is assumed that one or two other family members share the catch, then it can be estimated that the average angler consumes between 6.2 and 9.3 g/day. Thus, the true consumption by average anglers on the Savannah River most likely falls between 6.2 and 18.6 g/day.

HRP

I00

3.2.2.1.5 Estimates of Fish Consumption for the Hudson River

Available studies on fish consumption (see Table 3.2.2-1) indicate that there is considerable variation in the levels of consumption of freshwater fish. This variation is due to differences in species availability, productivity of the waters fished, access to those waters, species preferences, and cultural differences. To characterize Upper Hudson River fish consumption rates based on studies from other regions of the country and different types of waterbodies may result in inappropriate estimates of fish consumption. In addition, the use of estimates from marine or estuarine fishing surveys is clearly inappropriate and will result in an overestimation of freshwater fish consumption rates.

There are issues that need to be addressed in assessing the exposure that could potentially result from eating fish from the Upper Hudson. Currently, recreational fishing on the Upper Hudson River is banned. Therefore, actual consumption by anglers will be significantly depressed in comparison to other rivers. Because the purpose of the assessment is to demonstrate the exposures that could potentially occur in the absence of institutional controls (fishing bans), it is possible to provide a reasonable estimate of what fish consumption might be in the absence of the ban by examining fishing effort on nearby rivers that are not affected by the ban. These estimates do not indicate current risks but rather suggest what risks might be if no ban were in place. Fortunately, the "New York Statewide

HRP 001 1570

Angler Survey" (NYSDEC, 1990) contains information that can be used to make an educated guess as to the levels of consumption from the Upper Hudson River that might exist if no ban were in effect.

To characterize rates of fish consumption that might occur if the fishing ban were removed from Upper Hudson River, it is necessary to choose a surrogate waterbody for which data are available. The Mohawk River joins the Upper Hudson just above Federal Dam. It is reasonable to assume that because of the fishing ban on the Upper Hudson River, anglers in the area would choose to fish from another nearby location on which there is no ban. The Mohawk River is an appropriate substitute. Its proximity to the Hudson, and its status as a river on which there is a ban on only one fish species, make it a good substitute.

Information on angler effort on the Mohawk River is provided in the NYSDEC (1990) report. In Table 29, NYSDEC (1990) reports that the mean number of angler trips to the Mohawk River was 9.8 trips. If it is assumed that the average angler obtains 2 meals per trip (Pierce et al., 1981; Schmitt and Hornsby, 1985), it can be estimated that the average angler harvests 19.6 meals per year from the Mohawk River. This equates to a daily consumption rate of 12.2 g/day. This estimate is plausible and likely to be conservative when one compares it to estimates of mean consumption of sport-caught, freshwater fish reported for other river fisheries (Honstead et al., 1971; Soldat, 1970; ChemRisk, 1991a; Turcotte, 1983).

3.2.2.1.6 Fish Tissue Concentrations

Extensive efforts to sample fish tissues from the Upper Hudson River have been ongoing since PCBs were first discovered there. Early sampling indicated that PCB levels in certain fish were above acceptable regulatory levels (EPA, 1991b). Since that time, however, additional sampling has indicated that PCB levels have decreased over time due to remediation of the site and natural degradation processes.

The EPA Phase 1 risk assessment estimates the potential human exposure associated with the fish consumption pathway from combined fish tissue concentrations of total PCBs for all species collected between River Mile (RM) 153 and RM 195 from 1986 to 1988. EPA uses the 95 percent upper confidence limits on the mean fish tissue concentration as its estimate of the level of PCBs in fish consumed by recreational anglers.

There are several problems with EPA's approach. First, EPA did not carefully select data from relevant sampling locations for its analysis. The purpose of EPA's Phase 1 risk assessment was to assess risks to individuals who would consume fish that were potentially exposed to PCB-contaminated sediments between Fort Edward and the Federal Dam. Fish collected at RM 153 were collected below the Federal Dam. EPA states that it collected those fish because it believed that those fish might be exposed to PCBs that were potentially released from the dam. This assumption is probably invalid (see Section 6.0 of these comments). However, even if the assumption were valid, it is not appropriate to include the tissue concentrations of PCBs in those

HRP 001 1572

fish in estimating exposure resulting from the influence of river sediments above the dam on fish tissue PCB levels. Even if fish below the dam are exposed to some levels of PCBs that have been discharged from the dam, those levels are not likely to be representative of levels present above the dam because the dam acts as a significant barrier. In addition, those fish collected below the dam may potentially be exposed to a number of other sources of PCBs located below the dam. This is particularly true for the striped bass, which are migratory fish that only spend a portion of their lives in the waters below the Federal Dam. Thus, tissue concentrations in fish collected below the dam are not representative of fish tissue concentrations affected by PCBcontaining sediment above the dam and should not be included in the risk assessment.

Second, EPA's analysis includes a number of yearling pumpkinseed sunfish that range in size from 58 to 100 mm (2 to 4 inches). These fish are not likely to be consumed by anglers due to their size. In addition, many of the PCB concentrations measured in pumpkinseed were whole body rather than fillet concentrations. Because human consumers are not likely to consume the entire fish, inclusion of these data points in the analysis is inappropriate and introduces unnecessary uncertainty in the form of overstated exposure estimates into the analysis.

Third, EPA group all species together in its analysis. As indicated in Table B.3-15 of the Phase 1 Report, PCB levels are significantly different in the different species sampled. The assumption that all fish are to be treated the same implies

HRP 001 1573

that the distribution of species sampled is exactly the same as the distribution of species harvested by anglers. This is clearly not the case. PCB levels are highest in the goldfish (carp) which is a relatively undesirable foodfish. By giving carp equal weight with other more desirable species, actual PCB intakes are likely to be overestimated.

Fourth, the statistical approach used by the EPA, the 95 percent upper confidence limit (UCL) on the mean PCB fish concentrations, is inappropriate. This approach inherently assumes that the data follows a normal distribution. EPA has offered no analysis to justify this assumption. A casual review of the PCB data suggests that in nearly all cases, the distributions of fish concentrations from samples taken from the Upper Hudson do not follow a normal distribution. For certain species like American eel, there are too few data points to determine the shape of the distribution. For fish species with more data, the distributions are highly skewed and truncated, making it difficult to determine which indicator of central tendency should be used. In addition, by analyzing all fish species together, the distribution of concentrations derived is likely to be multimodal due to the differences among the individual species. Because of these problems, EPA's attempt to select a single estimate of fish tissue levels by its proposed statistical method is statistically unjustified.

To address the deficiencies in the EPA's approach, GE has reanalyzed the data collected between 1986 and 1988. Only fish from the appropriate reaches were considered, and only

HRP 001 1574

pumpkinseed tissue data from fish that were greater than five inches in length were included. Whole body concentration data for pumpkinseed sunfish were excluded from the analysis. Fish samples were sorted by species so that species-specific distributions of total PCB concentrations could be generated and used as the basis of the risk assessment.

3.2.2.1.7 Estimating PCB Intakes from the Fish Ingestion Pathway

As discussed previously, it can be conservatively estimated that the average Upper Hudson River angler might consume 19.6 fish meals per year (12.2 g/day) from that waterbody if there were no fishing ban. In assessing the potential for exposure via this pathway, it is essential that consideration be given to the species of fish that are actually likely to be consumed. Differences in the numbers of fish meals eaten for each species and the differences among tissue concentrations measured in the various species will have a marked impact on the estimated intake of PCBs by Upper Hudson River anglers.

According to NYSDEC (1990; Table 30), 38 percent of the angler days spent on the Hudson River were spent fishing for bass, 6.5 percent were spent fishing for brown trout, and 55.5 percent were spent fishing for "other" species. For the purpose of estimating species-specific consumption rates from which to estimate potential exposures, it is reasonable to assume that consumption is proportional to angler effort and to adjust the overall waterbody-specific consumption rate accordingly. Thus, it can be estimated that of the 19.6 meals per year consumed,

7.45 meals are bass, 1.27 meals are brown trout, and 10.88 meals are "other" species.

Individual estimates of consumption for each species that contributes to the "other" category can be calculated from the data provided by NYSDEC (1990). In Table 6 of that report, statewide angler effort is reported for 12 target species and one category for "other species" in addition to brown trout and bass. A comparison with Table B.1-3 of the Phase 1 risk assessment indicates that 8 of these 13 other species designations listed in Table 6 (NYSDEC, 1990) are actually found in the Upper Hudson River. If it is assumed that these 8 species groups represent the 55.5 percent of effort (or 10.88 remaining meals) for "other" species on the Upper Hudson indicated in Table 30 of the NYSDEC (1990) report, relative consumption rates by species can be estimated.

The total effort for these 8 species groups as reported in Table 6 (NYSDEC, 1990) was 9,510,820 angler-days. Of the total for the effort for these species, 18 percent of the effort was for yellow perch, 25 percent was for walleye, 12 percent was for northern pike, 12 percent was for bullhead, 15 percent was for brook trout, 9 percent was for sunfish, 3 percent was for chain pickerel, and 6 percent was for "other" species (Table 3.2.2-2). For this analysis, GE has assumed that the "other" category is comprised solely of American eel, white perch, and goldfish, and effort is equally distributed among the three species. Thus, it is assumed that approximately 2 percent of the total effort is for each of these species (Table 3.2.2-2).

117

HRP 001 1576

GE has also made the reasonable assumption that the percentage of total effort directed toward these individual species is proportional to the percentage of the remaining 10.88 meals per year consumption rate estimated (for all species except bass and brown trout), as discussed previously. If these relative percentages are applied to the remaining 10.88 fish meals, an estimated number of meals can be estimated for each species. Table 3.2.2-2 indicates the number of meals attributed to each individual species contributing to the "other" effort on the Upper Hudson River described in Table 30 (NYSDEC, 1990). GE therefore estimates that consumption rates are 1.99 meals per year for yellow perch, 2.73 meals per year for walleye, 1.27 meals/year for northern pike, 1.32 meals per year for bullheads, 1.59 meals per year for brook trout, 0.943 meals per year for sunfish, 0.367 meals per year for chain pickerel, and 0.225 meals per year for each American eel, white perch, and goldfish. Using the estimates for the number of meals by species, plausible estimates of exposure can be made using species-specific fish concentrations.

To avoid having to make assumptions about the distributions of the species-specific fish data, GE chose to estimate exposures through a Monte Carlo simulation using the actual fish data from the Upper Hudson River rather than try to select a single value to represent the body of the data. Each of the distributions of species-specific tissue concentrations were entered into the program. It was assumed that each meal consumed by the hypothetical angler was made up of a single fish. The

001 1577

HRP

appropriate number of fish were selected by species based on the estimated number of meals (Table 3.2.2-2). For example, for bass it has been estimated that 7.45 meals would be consumed annually. Thus, the program randomly selected 8 fish from the distribution. For seven of those fish, it was assumed that a single meal of 227 g (1/2 pound) was consumed and intake for each of those meals was estimated by multiplying 227 g by the tissue concentration in the individual fish. For the partial meal, the same method was used. A single fish was randomly selected from the distribution. Its concentration was then multiplied by 227 g and by 0.45 meals to estimate intake. This method was used to estimate potential intake of each of the individual species according to the number of meals allotted to the species as described in (Table 3.2.2-2). Then, the total intakes for all species were summed to calculate the average daily intake over a lifetime.

The distributions of fish concentrations were entered based on the available data. Separate distributions for American eel, bass (including smallmouth and largemouth), brown bullhead, sunfish (including pumpkinseed and redbreast), goldfish, and white perch were included in the simulation using actual data. For several species for which consumption rate estimates were made, there were no sampling data available for the relevant reaches. For each of these gamefish species, walleye, yellow perch, brown trout, northern pike, brook trout, and chain pickerel, fish tissue concentrations were selected from the bass tissue concentration data. The bass distribution was

HRP 001 1578

conservatively selected because bass are gamefish that are near the top of the aquatic food chain.

Ten thousand iterations of the simulation were run. Results of the simulation are provided in Table 3.2.2-3. The median estimated lifetime average intake level of PCBs resulting from the consumption of Upper Hudson River fish over a 30 year exposure period is estimated to be 0.47 μ g/kg-day, the mean which appears at the 60th percentile of the distribution is 0.55 μ g/kgday, and the 95th percentile is 1.2 μ g/kg-day (Table 3.2.2-3; Figure 3.2.2-1).

3.2.2.1.8 Cooking Loss

Most anglers and their families will cook the fish that they obtain from the Upper Hudson River before they consume it. As discussed previously, PCBs in the fish will be most highly concentrated in the body lipids. Because there is fat lost during cooking, it is likely that some of the PCBs will be removed when the fish are cooked so that tissue concentrations in the cooked fish will be lower than those measured in the raw fish.

Chemical losses have been observed in various methods of cooking of whole fish and fish fillets containing PCBs (Zabik et al., 1979, 1982; Puffer and Gossett, 1983; Smith et al., 1973). The average percentage reductions in the concentrations of PCBs resulting from various cooking methods are presented in Table 3.2.2-4.

Zabik et al. (1979) studied the changes in Aroclor 1254

HRP

baking, and microwaving. Broiling reduced the concentrations by an average of 53 percent, while roasting reduced levels by an average of 34 percent. Cooking fillets by microwave reduced levels by an average of 26 percent.

Zabik et al. (1982) found similar reductions in the concentrations of total PCBs in carp fillets cooked by various methods. Total PCB levels, expressed on the basis of the fat content of the fillet, were reduced by 25 percent by deep-frying, 27 percent by poaching, 25 percent by charbroiling, 33 percent by microwaving, and 20 percent by roasting. However, conflicting information presented in that report results in a level of uncertainty in the experimental results that compromises the reliability of the report's findings and conclusions.

Smith et al. (1973) reported that baking of chinook and coho salmon fillets reduced concentrations of Aroclors 1248 and 1254 by 11 to 16 percent. Poaching resulted in 2 to 6 percent reductions of the two Aroclors (Smith et al., 1973).

Puffer and Gossett (1983) reported cooking losses of Aroclors 1254 and 1242 resulting from pan frying of white croaker, a bottom feeding fish from the southern coast of California. In croaker obtained from Santa Monica Bay, 65 percent of the PCBs were lost during pan frying, while 28 percent of the PCBs were lost from the croaker obtained from Orange County. These differences were assumed to be a function of the differences in the initial levels of PCB contamination in the fish obtained from these two areas. Fish taken from Santa Monica

HRP 001 1580

Bay contained PCB levels four times greater than fish taken from Orange County.

Other studies (cited in Puffer and Gossett, 1983) have reported greater reductions in PCB levels. However, these studies have compared concentrations in whole raw fish to concentrations in cooked fillets and thus are of little use in estimating cooking loss from the fillet portion alone. Based on a review of the PCB cooking losses reported in the scientific literature, it is reasonable to conclude that at least 25 percent of the PCBs found in the fish fillet will be lost as a result of cooking.

In this analysis, a plausible estimate was made that a 25 percent reduction occurs in the concentrations of PCBs in fish fillet as a result of cooking. If estimated exposure levels are reduced by 25 percent due to cooking loss of PCBs, the resulting intake levels are 4.1 x 10^4 mg/kg-day (mean) for EPA's Scenario 1 (1986-1988 upper 95 percent confidence based on mean) and 5.2 x 10^{-5} mg/kg-day (mean) for EPA's Scenario 2 (30 year mean trend).

3.2.2.1.9 Summary of Fish Exposures

The Phase 1 Report uses a very coarse estimate of PCB exposure from the human fish consumption pathway, one that is inaccurate and grossly overstates realistic exposures. GE has performed a more sophisticated analysis that accounts for the way in which anglers in the Upper Hudson area might actually behave in the absence of a fishing ban, the distribution of fish actually likely to be consumed, species-specific PCB levels, and the manner in which PCBs are prepared for human consumption.

GE's analysis shows that the Phase 1 Report's exposure estimate of chronic daily intake (Table B.6-5) is almost an order of magnitude greater than that warranted by the data. GE's calculation does not, of course, account for the effect of the fishing ban. Common sense suggests that the fishing ban provides an additional level of protection and that, with the ban, actual site-specific exposures are virtually non-existent.

3.2.2.2 References

ChemRisk. 1991a. Consumption of Freshwater Fish by Maine Anglers. ChemRisk® - A Division of McLaren/Hart. Portland, ME. July 31.

ChemRisk. 1991b. Consumption of Freshwater Fish from Maine Lakes and Ponds. ChemRisk® - A Division of McLaren/Hart. Portland, ME. September 6.

ChemRisk. 1991c. Saco River Creel Survey. Unpublished data. ChemRisk[®] - A Division of McLaren/Hart. Portland, ME.

ChemRisk. 1991d. Penobscot River Creel Survey. Unpublished data. ChemRisk[®] - A Division of McLaren/Hart. Portland, ME.

Fries, G.F. and D.J. Paustenbach. 1990. Evaluation of potential transmission of 2,3,7,8-tetrachlorodibenzo-p-dioxin contaminated incinerator emissions to humans via foods. J. Toxicol. Environ. Health 29:1-43.

Honstead, J.F., T.M. Beetle, and J.K. Soldat. 1971. A Statistical Study of the Habits of Local Fishermen and Its Application to Evaluation of Environmental Dose, A Report to the Environmental Protection Agency by Battelle Pacific Northwest Laboratories, Richland, WA 99352. (cited in Rupp *et al.*, 1980)

Humphrey, H.E.B. 1978. Personal communication. (cited in Rupp et al., 1980)

Javitz, H. 1980. Seafood Consumption Data Analysis. SRI International. Final report prepared for EPA Office of Water Regulations and Standards. EPA Contract 68-01-3887.

Minnesota Pollution Control Agency. 1990. Appendix E. Fish Consumption. Submission to Administrative Judge Luis re: AWQC for Dioxin.

HRP 001 1582

NYSDEC. 1990. New York Statewide Angler Survey 1988. New York State Department of Environmental Conservation. Albany, N.Y. April.

Pierce, R.S., D.T. Noviello, and S.H. Rogers. 1981. Commencement Bay Seafood Consumption Report. Preliminary Report. Tacoma, WA: Tacoma-Pierce County Health Department.

Puffer, H. 1981. Consumption rates of potentially hazardous marine fish caught in the metropolitan Los Angeles area. EPA Grant #R807 120010.

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

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

SCAQMD. 1988. Multi-Pathway Health Risk Assessment Input Parameters Guidance Document. South Coast Air Quality Management District. June.

Schmitt, D.N. and J.H. Hornsby. 1985. <u>A Fisheries Survey of the</u> <u>Savannah River</u>. Georgia Department of Natural Resources, Game and Fish Division. Atlanta, Georgia. September.

Schroeder, R.A. and C.R. Barnes. 1983. Polychlorinated Biphenyl Concentrations in Hudson River Water and Treated Drinking Water at Waterford, New York. USGS Water - Resources Investigations Report 83-4188. USGS, Albany, NY.

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

Smith, A.H. 1987. Infant exposure assessment for breast milk dioxins and furans derived from waste incineration emissions. *Risk Analysis* 7(3):347-353.

Soldat, J.K. 1970. A statistical study of the habits of fishermen utilizing the Columbia River below Hanford. Chapter 25. In: Environmental Surveillance In the Vicinity of Nuclear Facilities: Proceedings of a Symposium Sponsored by the Health Physics Society. January 24-26, 1968. W.C. Reinig (ed.) Springfield, IL. pp. 302-308.

Stevens, J.B. and E.N. Gerbec. 1988. Dioxin in the agricultural food chain. Risk Analysis 8(3):329-335.

001 1583

HRP

Turcotte, M-D. S. 1983. Georgia Fishery Study: Implications for Dose Calculations. Memorandum to H.P. Olson from M.D.S. Turcotte, Technical Division Savannah River Laboratory. August 5.

USDA. 1984. Consumption and Family Living. Agricultural Statistics, Table 697, p. 506. (cited in EPA, 1989b)

USEPA. 1984. Ambient Water Quality Criteria for 2,3,7,8-Tetrachlorodibenzo-p-dioxin. U.S. Environmental Protection Agency, Office of Water Regulations and Standards, Washington, D.C. February.

USEPA. 1989a. Exposure Factors Handbook. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. EPA/600/8-89/043. July.

USEPA. 1989b. Assessing human health risks from chemically contaminated fish and shellfish - A guidance manual. U.S. Environmental Protection Agency, Office of Water Regulations and Standards. EPA 503/8-89-002. September.

USEPA. 1989c. Risk Assessment Guidance for Superfund: Volume 1 - Human Health Evaluation Manual (Part A). U.S. Environmental Protection Agency, Office of Emergency and Remedial Response. Washington, DC. EPA/540/1-89/002. December.

USEPA. 1991a. Phase 1 Report - Reassessment Remedial Investigation and Feasibility Study: Interim Characterization and Evaluation. Interim Report. U.S. Environmental Protection Agency, Region II, New York. August.

USEPA. 1991b. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual Supplemental Guidance "Standard Default Exposure Factors". U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Toxics Integration Branch, Washington, DC. EPA 540/1-89/002. March 25.

West, P., J.M. Fly, R. Marans and F. Larkin. 1989. Michigan Sport Anglers Fish Consumption Survey. A report to the Michigan Toxic Substance Control Commission. Natural Resource Sociology Research Lab Technical Report #1. May.

WHO. 1987. Environmental Health, Vol.23: PCBs, PCDDs, and PCDFs, Prevention and Control of Accidental and Environmental Exposures. World Health Organization. Copenhagen.

WHO. 1989. Polychlorinated Dibenzo-para-dioxins and Dibenzofurans. United Nations Environment Program, the International Labor Organization, and the World Health Organization. Geneva, Switzerland.

HRP 001 1584

Wisconsin Division of Health. 1987. Study of Sport Fishing and Fish Consumption Habits and Body Burden Levels of PCBs, DDE and mercury of Wisconsin Anglers. Wisconsin Division of Health and State Laboratory of Hygiene. Final report to study participants.

Zabik, M.E., P. Hoojjat, and C.M. Weaver. 1979. Polychlorinated biphenyls, dieldrin, and DDT in lake trout cooked by broiling, roasting, or microwave. *Bull. Environ. Contam. Toxicol. 21*:136-143.

Zabik, M.E., C. Merrill, and M.J. Zabik. 1982. PCBs and other xenobiotics in raw and cooked carp. Bull. Environ. Contam. Toxicol. 28:710-715.

3.2.3 Reassessment Of Risks Associated With <u>PCBs In Hudson River Sediments</u>

3.2.3.1 Carcinogenic Potency Assessment

As discussed in Section 3.2.1.1 above, the recent reevaluation of the rodent PCB bioassays provides an appropriate mechanism for separately assessing the carcinogenic potency of the various Aroclor mixtures containing less than A60 percent chlorine.

The finding that PCBs, other than the highly chlorinated Aroclor 1260 and Clophen A60, have no carcinogenic potential is very significant for the assessment of PCB risks in the Upper Hudson River. PCBs found in the Upper Hudson River do not include highly chlorinated PCBs. Therefore, the most likely estimate of carcinogenic risk is zero.

Another way, which would be contrary to EPA policy in dealing with negative studies (OSTP, 1984) and which GE believes is scientifically invalid but which is sometimes used nevertheless, to perform a human health risk assessment for the lower chlorinated PCBs is to assume some carcinogenic potential based on tumor incidence regardless of statistical significance.

Using the recent reread results (Moore, 1991), and statistically forcing the negative bioassays to produce non-zero estimates of potency, a potency of 0.4 (mg/kg/day)⁻¹ can be estimated for Aroclor 1254 and 0.2 (mg/kg/day)⁻¹ for Aroclor 1242.

A toxicologically equivalent human dose can be estimated by scaling the rodent bioassay results based on body weight. This is consistent with Federal Drug Administration

HRP 001 1586

(FDA) and Center for Disease Control (CDC) methodologies (FDA, 1986; Bayard, 1988). This is the correct scaling methodology for PCBs because the compound itself rather than a metabolized product is the active agent. The EPA policy of extrapolating from rats to humans on the basis of relative surface areas is inappropriate in this context since it is based on a study by Freireich et al. (1966). This study did not consider carcinogenity as the endpoint of concern and thus is inapplicable to extrapolating from rats to humans when deriving cancer potencies. Recent reviews at interspecies scaling factors indicate that all measures of dose, except dose rate per unit of body weight, tend to overestimate human risk (Mordenti, 1986; Brown et al., 1988; Crump et al., 1989).

Thus, using the FDA and CDC scaling methodology and the calculated rat potency based on the tumor incidence data, the resulting cancer slope factor (or q_1 *) is 0.037 (mg/kg-d)⁻¹ for the lower chlorinated PCB mixtures.

3.2.3.2 Consumption of Fish

3.2.3.2.1 PCB Concentrations in Fish Tissue The Phase 1 Report develops two estimates of total PCB intake. The first assumes that levels of PCBs will remain constant at the 1988 levels for the next thirty years. The second assumes that the concentrations of PCB will decline in the future.

As discussed in Section 3.2.2.1, the estimates of fish consumption produced in the Phase 1 Report suffer from a number of technical problems, including improper statistical

HRP

100

1587

assumptions, improper grouping of fish, and overestimates of fish consumption. Revised estimates of fish consumption have been prepared using a Monte Carlo model of PCB levels in fish and species specific consumptions estimates. The results of this model have been applied to both the steady-state and declining estimates of long-term PCB levels.

3.2.3.2.2 Human Exposure Via Fish Ingestion

As discussed above, estimates of species-specific consumption rates were made for the Upper Hudson River based on data from fishing surveys performed in New York State. Table 3.2.2-3 presents estimates of total PCB intake by anglers who might potentially fish the Upper Hudson River if there were no fishing ban there. The estimated lifetime average daily intakes have a mean of 0.41 μ g/kg-day, assuming that 1986-1988 conditions (as hypothesized by the Phase 1 Report) continue for 30 years, and a mean of 0.052 μ g/kg-day, assuming the mean of trends extrapolated for the next 30 years (as hypothesized by the Phase 1 Report).

3.2.3.3 Other Exposures

The other exposure pathways quantitatively investigated by EPA include ingestion and dermal exposure to sediments and surface water. In general, these exposures ranged from 2 to 4 orders of magnitude below fish consumption. Because of the limited potential for exposure from these routes, the Phase 1 Report concludes that estimated upper-bound risks from these sources are within an acceptable risk range. GE agrees but comments that EPA makes a number of unreasonable assumptions both

129

HRP 001 1588

in the extent of exposure and in the level of PCBs to which people were exposed. In particular, EPA failed to consider future declines in environmental concentrations of PCBs when estimating long-term risks from these other pathways.

3.2.3.4 Conclusions

Table 3.2.3-1 and Figure 3.2.3-1 indicate the effects of different assumptions on the estimated upper-bound risks associated with the fish consumption exposure pathway. The risk estimates are divided into the two scenarios postulated in the Phase 1 Report: (1) PCB levels in fish remain steady for 30 years, and (2) PCB levels decline over the next 30 years. The Phase 1 Report concludes that the cancer risk from eating Upper Hudson fish is about 2 in 100 for the first scenario and about 2 in 1000 for the second scenario.

As discussed above, these estimates are based on an outdated and technically incorrect estimate of potency. Using EPA's preferred study, Norback and Weltman (1985) as re-read by Moore (1991), the potency decreases from 7.7 to 5.1 $(mg/kg/day)^{-1}$. Using a geometric average of all positive studies (as advocated by Moore (1991) for PCBs containing 60 percent chlorine) the potency decreases to 1.9 $(mg/kg/day)^{-1}$.

However, PCBs released to the Hudson contained less than 60 percent chlorine. Because there is no evidence that these lightly chlorinated PCBs are carcinogenic, the best estimate for the carcinogenic risks from intake of fish contaminated with these compounds is zero. A highly conservative alternative assumption to this zero estimate can be made by

HRP

100

1589

interpreting the negative bioassays to produce non-zero estimates of potency. Using this approach a potency of 0.2 $(mg/kg/day)^{-1}$ can be derived. Using this potency, the estimated risk range is 5.5 x 10⁻⁵ to 4.4 x 10⁴.

EPA's assumptions of the level of PCB exposure from the consumption of contaminated fish, 0.0022 mg/kg/day, greatly overestimates the actual intake of PCB for fish consumers. Using a site-specific estimate for fish intake based on factors such as species-specific PCB measurements and local fish consumption rates, the lifetime annual daily intake for PCB is estimated to be 0.00041 mg/kg/day. Using this revised estimate of exposure, which does not account for the fishing ban currently in effect, the range of carcinogenic risk (assuming a potency of 0.2 $(mg/kg/day)^{-1}$) is 1 x 10⁻⁵ to 8 x 10⁻⁵.

As discussed in Section 3.2.1.1.7, the use of surface area scaling appears to be unwarranted for PCBs. Use of a body weight scaling factor on the 0.2 $(mg/kg/day)^{-1}$ potency and the revised estimates of PCB exposure from fish consumption results in an estimated risk of 1.9 x 10⁻⁶ to 1.5 x 10⁻⁵.

HRP 001 1590

Table 3.2.3-1

Carcinogenic Risks Associated With Consumption of Fish

	EPA Scenario 1	<u>EPA Scenario 2</u>
Phase 1 Report Estimate	2 x 10 ⁻²	2 x 10 ⁻³
Estimate if Rat Re-read Results Are Used	0	0
Estimate if Rat Re-read Results Are Forced to Produce a Non-Zero Factor	4.4 x 10 ⁻⁴	5.5 x 10 ⁻⁵
And Proper Exposure Estimates Are Used	8 x 10 ⁻⁵	1 x 10 ⁻⁵
And Body Weight Scaling Is Used	1.5×10^{-5}	1.9 x 10 ⁻⁶

Note: EPA Scenarios and Phase 1 Report Estimates are from Phase 1 Report Table B.6-5.

The Phase 1 Report's approach clearly results in a gross overestimate of risk from fish consumption. By contrast, GE estimates that the maximum realistic risk of cancer from fish consumption ranges from zero (assuming the rodent bioassay results are correctly used and that different factors are applied to PCB mixtures depending on the degree of chlorination) to 1.5 x 10^{-5} (assuming negative bioassays are forced to produce non-zero estimates of potency). Even the latter value is an overestimate, if the purpose is to determine the risk from sediments in the Upper Hudson study area, because it includes background levels and contribution from other sources and ignores the declining trend in PCB body burdens in fish.

Given the range of risk estimated by GE using the new science and more site-specific data, the Phase 1 Report incorrectly concludes that there are unacceptable potential HRP 001 1591

cancer risks associated with the ingestion of fish from the Upper Hudson River.

With respect to non-carcinogenic effects of PCBs, the speculation as to chloracne and impaired liver function has been dispelled. Additionally, there has been no validation of the hypothesized relationship between reproductive or neurodevelopment effects in human and low-level PCB exposures. Long-term epidemiological studies have failed to link PCB exposure to excess mortality or to any other significant human health problems. Thus, there is no scientific basis for deriving a Reference Dose based on human data.

Finally, the Phase 1 Report's attempt to derive a PCB Reference Dose based on unexamined, unreviewed, and unvalidated subhuman primate studies is misplaced. The use of this Reference Dose in the Report's preliminary health risk assessment is in error. In the absence of supporting evidence, the Phase 1 Report's conclusion that there are unacceptable non-cancer human health risks associated with the ingestion of Upper Hudson River fish is erroneous.

3.2.3.5 References

Bayard, S.P. 1988. Quantitative implications of the use of different extrapolating procedures for low-dose cancer risk estimates from exposure to 2,3,7,8-TCDD. Review Draft. Appendix A. EPA 600/6-88/007Aa and Ab. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. Washington, DC.

Brown, S.L., S.M. Brett, M. Gough, J.V. Rodericks, R.G. Tardiff and D. Turnbull. 1988. Review of interspecies risk comparisons. *Reg. Tox. Pharm.* 8:191-206.

HRP 001 1592

Crump, K., B. Allen, and A. Shipp. 1989. Choice of dose measure for extrapolating carcinogenic risk from animals to humans: An empirical investigation of 23 chemicals. *Health Phys.* 57:387-393.

FDA. 1986. Biological Basis for Interspecies Extrapolation of Carcinogenicity Data. U.S. Food and Drug Administration. prepared by Life Science Research Office & Federation of American Societies for Experimental Biology and submitted to the Center for Food Safety and Applied Nutrition, Department of Health and Human Services, Washington, DC. July.

Freireich, E.J., E.A. Gehan, D.P. Rall, L.H. Schmidt, and H.E. Skipper. 1966. Quantitative comparison of toxicity of anticancer agents in mouse, rat, hamster, dog, monkey, and man. Cancer Chemotherapy Reports 50(4):219-244.

Mordenti, J. 1986. Man versus beast: Pharmacokinetic scaling in mammals. J. Pharm. Sci. 75(11):1028-1040.
3.3 Ecological Risk Assessment

Section B.7 of the Phase 1 Report is entitled "Interim Ecological Risk Assessment." It concludes:

> "Based on the limited available data, it is premature to conclude whether ecological risks specifically attributable to PCB contamination from the Upper Hudson River exist." (Synopsis to Section B.7.)

This equivocal statement can hardly serve as the basis to conclude that PCBs present any ecological risk to the Upper Hudson River system.

GE's specific comments are:

- 1. The most appropriate way to conduct an ecological assessment of the Upper Hudson River is to examine the biological integrity of its ecosystem, looking at species composition and diversity, nutrient and energy flows and production, consumption and decomposition, and then to determine whether the biological integrity of that system has been impaired by the presence of PCBs.
- 2. The available evidence suggests that the presence of PCBs in the Upper Hudson River ecosystem has not significantly compromised its biological integrity and, whether due to declining PCB loads or otherwise, the trend is toward even more balanced, integrated, adapted communities of organisms with species compositions, diversity, and functional organizations substantially unimpaired by PCBs.
- 3. Even modeling ecological risks at an "interim" level of assessment, however, EPA has made methodological, data use, and analytic errors that may be compounded if not corrected.
- 4. The Phase 1 Report fails to identify the data needed to assess the impact dredging will have on the ecosystem.
- 5. The jump to an ecological risk characterization through the use of PCB criteria and guidelines is premature, theoretical, not site specific, and scientifically invalid.

001 1594

HRP

3.3.1 <u>A Systems Approach Is Most Appropriate</u>

The basic problem with Section B.7 of the Phase 1 Report is that it ritualistically adheres to the reporting format derived from RAGS II (U.S. EPA, 1989a) but pays scant attention to the substantive purposes of the ecological assessment in the RI/FS process: (1) To decide if remedial action is necessary based on ecological considerations, and (2) to compare and evaluate the potential ecological effects of remedial alternatives.

RAGS II makes it clear that these purposes are served only if a systems approach is used in the assessment:

> Because it encompasses all of the relevant physical and biological relationships governing organisms, populations, and communities, <u>the ecosystem is generally</u> <u>considered the fundamental unit of ecology</u>. RAGS II, p. 16 (emphasis supplied).

The systems or holistic approach to ecological assessment is not unique to the RI/FS process, but is the standard scientific method applicable to many other situations in which the goal is to determine the health of an ecosystem or the effect of a perturbation on the system (e.g., USEPA 1990a)).

Under the systems approach, the key factors are the structure and functions of the system, the effect of the presence of a contaminant on the functioning of the system, and impairment (if any) of the biological integrity of the system by the contaminant. Thus, rather than looking at the concentrations of contaminants in specimen organisms and the effect of such concentrations on those organisms, or organisms considered to be

HRP 001 1595

analogous or indicators in other places, the systems approach requires a look at the response of the communities of organisms in that specific ecosystem. Such response is measured in terms of structure and function rather than on an organism-by-organism, or even species-by-species basis.

Unfortunately, the Phase 1 Report presents only "an initial evaluation of potential ecological risks <u>for selected</u> <u>species</u>" (p. B.7-2) (emphasis supplied). The Report mentions the systems approach (p. B.7-7; Subsection B.7.3.1), but this is more a mechanical incantation than a meaningful description of the "functional system of complementary relationships and transfer and circulation of energy and matter" (RAGS II, p.16). To produce a useful product for the RI/FS, upon which meaningful decisions regarding risk and remedial alternatives can be based, a systems approach should be used, and all future ecological assessment work in Phases 2 and 3 should proceed in such manner.

3.3.2 No Impairment to the Ecosystem From the <u>Presence of PCBs</u>

Section B.7 speaks of the "very limited available data" (pp. B.7-2, B.7-8, B.7-9) in the ecosystem description. Nevertheless, the available data, as well as simple observations of the Upper Hudson River corridor, show a river system bounded by abundant riparian wetlands, teeming with fish, and having a large variety of migrant and resident birds, reptiles, and mammals. Diversity, distribution, and abundance of species exist at all trophic levels, and no evidence suggests that the ecosystem is any worse or different below Ft. Edward than it is

HRP

100

1596

above Ft. Edward. Although the portion of the River below Ft. Edward contains greater masses and concentrations of PCBs in certain compartments, this distinction does not appear to affect the ecosystem's structure and function.

Thus, GE does not believe that the ecosystem data is too limited to permit the conclusion that the biological integrity of the Upper Hudson River ecosystem is unaffected by the presence of PCBs. While the data may be too limited to attribute premature or unnatural biological endpoints in individual members of particular species to specific PCB burdens in such species, for the Upper Hudson River site this limitation ' is of no effect. While that limitation might not allow for any meaningful analysis of the ecological risks present at a small site, the Upper Hudson River is itself a large and significant ecosystem that can and should be evaluated in a systematic rather than an compart- mentalized way. Such an evaluation can proceed on the existing database. That database shows a healthy ecosystem, and one that is continuously becoming better balanced, and more diverse.

As an example, because the condition of fish populations in the Upper Hudson River has long been a concern due to the presence of a variety of contaminants, the information on fish populations in the Upper Hudson River is more extensive and covers a wider time frame than information presented for previously discussed communities. A review of this available data shows that there has been a qualitative improvement in the fish population over the past 20 years. Species composition,

138

diversity and abundance show relative well-being of the fish populations in the Upper Hudson River. Studies show a diverse fish community representing a variety of habitats.

Future assessment activities should include an identification of the habitats which support these fish populations so that such habitats are preserved when considering remedial alternatives.

Even if the existing evidence is not conclusive regarding the well-being of the Upper Hudson River ecosystem, it is at least suggestive of such a hypothesis. GE, therefore, believes that if any further ecological assessment work is to be done as part of the RI/FS, it should be planned to test this hypothesis, because no evidence to suggest an alternative hypothesis exists.

However, to do this, EPA must use correct methods, must properly use and analyze data and literature, and must conduct a proper data collection program. Even if EPA were to reject this systematic approach and rely instead on the approach to ecological risk assessment set forth in the Phase 1 Report, EPA must address the deficiencies in its Phase 1 analysis. The next portion of these comments will address these subjects.

3.3.3 Methodological and Analytical Flaws in The Phase 1 Ecological Assessment

The first step in an ecological assessment of the Upper Hudson River ecosystem is to describe the existing setting or baseline conditions in a manner that will allow an evaluation of (a) its existing biological integrity; (b) the effect of the

presence of PCBs on its integrity; and (c) the effect on such integrity of actions to alter the existing PCB condition. The Phase 1 Report's approach to evaluating baseline ecological risk contains a number of major deficiencies including:

- Failure to Address Background Conditions
- Lack of PCB Occurrence Data Reflecting Current Conditions
 - Failure to Specify Endpoints

3.3.3.1 Failure to Address Background Conditions

Since the function of an ecological assessment is in part to demonstrate how PCBs in the Upper Hudson River affect the biological integrity of the ecosystem, it is essential to isolate the effect of PCBs in that site from the effect of other conditions, whether anthropogenic or otherwise. To accomplish this objective, an identification of background conditions is required.

The Phase 1 Report does not adequately address background ecological conditions at the site. For example, populations of aquatic organisms of various trophic levels in an on-site reach of the river should be evaluated for population demographics, density, variation, and general health. This data should then be compared to similar population parameters determined for organisms inhabiting a reference reach. Without an identified background, there is no way to use the description of the on-site ecosystem to accomplish the goals of the RI/FS.

HRP 001 1599

3.3.3.2 Lack of PCB Occurrence Data to Reflect Current Site Conditions

Both historical data and recent monitoring results indicate that levels of PCBs are continuously declining in the river. Therefore, the data used for this baseline evaluation must be current to developing a relevant and accurate representation of existing site conditions. The Phase 1 Report relies on the historical PCB data for water, sediment, and biota.

Based on the the references cited by EPA, data selected in the Phase 1 Report are generally two to five years old. Due to the time lag between report preparation and data collection, these reports probably reflect site conditions no more recent than three to seven years ago. Given the observed natural decreases in PCB levels, use of this data without adjustment for natural attenuation to reflect current time conditions is inappropriate. In addition, to assess the effectiveness of remedial alternatives, the data should be adjusted to reflect conditions in 1993, at which time any remedy would potentially begin. Considering this time factor, the data cited in the Phase 1 Report becomes five to nine years out of date.

3.3.3.3 Failure to Specify Endpoints

RAGS II states that, based on the available information concerning the site, contaminants, and likely exposure pathways, the analyst should identify and select appropriate toxicological endpoints for the assessment. In order to address the uncertainties associated with ecological risk, the level of study must be identified. Endpoints can be evaluated ranging from

141

death to sublethal effects such as altered population dynamics, reproductive potential and fecundity, species diversity, and histopathology. The report discusses a variety of unrelated ecological endpoints. EPA does not identify the overall ecological endpoints and goals for site evaluation.

3.3.4 Insufficient Data is Presented to Allow Evaluation of Ecological Impacts During the Remedial Selection Process

Superfund remedies are to be protective of the environment. To achieve this goal, EPA must evaluate both the benefits to the Hudson River ecosystem that will be achieved by the implementation of the various remedial alternatives <u>and</u> the detrimental impacts to the ecosystem that would result from such implementation. Once baseline conditions are established, the ecological risks and benefits of each remedial alternative must be identified. These risks and benefits must be weighed to select a remedy that is truly protective of the environment.

In other sections of this comment document, GE has voiced its concern that removal or treatment of contaminated sediment in the Upper Hudson River will not achieve any great ecological benefit, due to natural attenuation of PCBs, the location of other sources of PCBs and the lack of any apparent ecological risk attributable to the presence of PCBs in the Upper Hudson. As the ecological benefits to be achieved by remedial action are dubious at best, the detrimental impacts of remedial alternatives must be carefully examained.

The Phase 1 Report fails to address the adequacy of the existing data to allow proper quantification of the damage and

HRP 001 1601

risks to the ecosystem that would result from the implementation of remedial alternatives, particularly dredging. EPA must carefully analyze these potential adverse impacts and collect the data necessary so a proper assessment of the benefits versus damages can be made.

EPA must define the aquatic ecosystem structure and its relationship to key habitats that will be impacted by dredging. This will require that both emergent and riparian habitats be mapped, classified and species dependent on those habitats be identified. Aquatic vegetation is mentioned as being reported in a 1933 survey along a portion of the Upper Hudson River study No recent inventories of aquatic macrophytes have been area. carried out. The Phase 1 Report does not discuss the value of aquatic macrophyte communities as habitat. Nor does the report discuss potential impacts to macrophytes and associated fishery habitats from dredging if that remediation option should be recommended. EPA must document the current site-specific location, composition, and distribution of these important macrophyte communities and associated aquatic and riparian habitats before it can consider the impacts of remedial alternatives.

Additionally, EPA will need to evaluate more thoroughly the data on the benthic invertebrate community to determine if their complete destruction during dredging will irreversibly destroy the current benthic community structure. EPA will also need to determine if any invertebrate species will be adversely impaired due to siltation that will occur during and after dredging.

143

3.3.5 PCB Exposure Assessment

The purpose of an exposure assessment is to estimate the contact a potential receptor may have with a contaminant and the concentration of that contaminant at the point of contact. RAGS II makes clear that before the effects of a contaminant on an organism can be evaluated, it is necessary to know how much of the chemical is actually or potentially reaching the point of exposure. Because this potential for exposure depends on the interplay between the characteristics of the contaminant, the organism and the environment, a valid ecological exposure assessment must rely upon site-specific data. Recognizing the limitations of available site-specific PCB exposure data, the Phase 1 Report states that "the data available specific to PCBs are inadequate to evaluate species, population and community health dynamics which are necessary components of an ecosystem approach" (p. B.7-19). Thus, the PCB exposure assessment in the Phase 1 Report is inadequate due to the limitations of the simplified ecological framework used for evaluation. In future phases of evaluation current site-specific information must be applied to the exposure assessment.

The Phase 1 Report mixes site-specific data with general PCB occurrence and ecology information cited from the literature. Although this approach is not invalid per se, this mixing in the Phase 1 Report has potentially misrepresented and/or obscured pertinent, realistic, site-related exposures. By failing to take into account the limitations of the available data and by neglecting to identify background PCB levels, the

HRP 001 1603

Phase 1 Report overstates the potential for ecological risk attributable to the site-specific presence of PCBs. Further, EPA did not propose activities or approaches which would correct this deficiency in future phases.

In addition to this pervasive problem, there are specific weaknesses relating to the information presented in the Phase 1 Report on exposure pathways, receptors (indicator species), exposure quantification, and toxicity, which will be discussed in detail below.

3.3.5.1 Exposure Pathways

A complete exposure pathway is defined by tracking a contaminant to an exposure point where a receptor may realistically contact the contaminant. The concentration of the contaminant used to estimate exposure must be realistically representative of the media and point of exposure. This matching of exposure point concentration, location, and media with the receptor is critical to evaluating food chain exposures and potential ecological impacts. Such information is relevant both to establishing a baseline and to evaluating any benefits that would be achieved by implementation of various remedial alternatives.

The Phase 1 Report fails to integrate the information presented for pathways, indicator species, exposure quantification and toxicity. The relationships between the fate and transport of the contaminant to the site-specific exposure pathways, exposure routes, potential receptors and habitats should be presented in the Phase 1 Report. Failure to do so

ĨŖ₽

I 0 0

1604

results in confusion in interpreting the information in Section B.7.3 and its relevance to current site-specific exposures.

3.3.5.2 Identification of Indicator Species (B.7.3.2)

For an ecosystem as large and complex as the Upper Hudson River, an exposure assessment can only realistically be performed through the use of indicator species to represent the various trophic levels. Although GE recognizes that the selection of indicator species for the Upper Hudson River ecosystem may need to be driven in part by the availability of data regarding various species, it is critical to the development of a realistic site-specific exposure assessment that the validity of the selection and the applicability of the available data to the site be assessed. The Phase 1 Report fails to undertake such an assessment. Comments regarding particular indicator species selected are presented below.

3.3.5.2.1 Herring Gulls

Birds can function as useful indicator species because of their diet and sensitivity. Indeed, some of the best available PCB toxicity data focuses on Herring Gulls. However, most gulls found in the Upper Hudson River area are migratory, thus data in the literature regarding habitats, feeding and breeding behaviors, and toxicity of PCBs must be adjusted to reflect the conditions present in the study area.

The Herring Gulls is opportunistic in its feeding habits; consequently, any generalization about its diet would be invalid away from the immediate time and place of measurement.

Herring Gulls frequent landfills, where they feed on wasted food. and they have been known to eat such fare as bird's eggs and berries. Thus, assuming a diet of 50 percent fish for Herring Gulls along the Upper Hudson is probably not accurate. If information were available for Hudson River Herring Gull diet and PCB bioaccumulation, it would be difficult to generalize from it, because groups of gulls on different sections of the River probably have widely varying diets, based on location of dams, landfills, towns, etc.

Herring Gulls are not known to breed along the Upper Hudson River, and are migratory. They breed along the Atlantic Coast, in the Adirondacks, and around the Great Lakes, and would occur along the Upper Hudson as winter visitors or sub-adult, non-breeding visitors at other seasons (Andre and Carroll, 1988). Any one individual would probably only spend a portion of its life along the Upper Hudson. Even if a bird spent every winter on the River, it might be there no more than 50 percent of its life.

Even if EPA were to collect Herring Gulls for analysis from within the study area, these data limitations would remain. Birds could theoretically ingest a contaminant in a different system such as the Great Lakes, and be collected on the Hudson River; it may be incorrectly assumed that contaminants were HRP locally ingested. In addition, if EPA were to rely instead on reported data on Herring Gulls populations on the Great Lakes, 100 the difference in composition of the background contaminants would confound direct comparisons of exposure and toxicological

1606

effects between Herring Gulls on the Upper Hudson River with the Great Lakes.

In the Upper Hudson River, few organochlorines other than PCBs are present at potentially environmentally significant levels. In contrast, the Great Lakes are thought to have the highest contamination by chlorinated hydrocarbons in North America (Vermeer and Peakall, 1977). The concurrent presence of a large number of different organochlorine compounds, some of which share structural and toxicological similarities to PCBs, makes interpretation of results and derivations of conclusions very complicated and sometimes impossible. The potential additive, antagonistic, and synergistic relationships between the various chemicals makes it difficult or impossible to determine which are the principal contributors to the observed effects. Recent research innovations and congener-specific analyses are increasing the ability to define effects and derive conclusions.

The important factor is that the chemical exposure and cumulative toxicological circumstances are probably much more complicated in the Great Lakes than on the Hudson, making it difficult to compare, with any degree of certainty, exposure qualifications, bioaccumulation factors (BAFs), and toxicological endpoints from gulls on the Great Lakes to gulls on the Upper Hudson. Because of the simultaneous occurrence of many other toxic chemicals, a no-effect value for a single chemical derived from research on the Great Lakes is probably a conservative one.

HRP 001 1607

3.3.5.2.2 Mink

EPA has selected mink as an indicator mammalian species based not on existence or prevalence in the area, but on the availability in the literature of PCB toxicity data. Although the mink is a piscivorous mammal that inhabits regions of upstate New York, data suggests that populations of mink along the river itself are very small or nonexistent. Thus, the relevance of the reported information to a site-specific ecological risk assessment is dubious. Notwithstanding the questionable relevance, the applicability of the available literature data to whatever minks do inhabit the Upper Hudson River area must be examined.

Much of the information on the toxicity of PCBs in mink has been derived from observations of reproductive failure in ranch mink that were fed Great Lakes fish contaminated with PCBs and other organochlorines in the 1960's (Hartsough, 1965), and from laboratory feeding studies using similar fish stock (Aulerich et al., 1970; 1971; 1973; Ringer et al., 1981). TO date, there are over 30 studies examining chemical toxicity to mink with the majority emphasizing the effects from PCBs (Wren, 1991). Certainly, the accumulating toxicological data base on the effects of PCBs in this species provide an opportunity for species-specific comparisons to modeled or measured exposure HRF values for mink in the Upper Hudson River area. However, it is important to note that the chemical exposures and cumulative 100 toxicological circumstances in the Great Lakes are probably very 1608 different, if not more complicated, than the conditions on the

Upper Hudson. At the least, toxicity values derived from research based on Great Lakes mink population are probably very conservative. Future phases of the ecological risk assessment must take these limitations into account.

3.3.5.2.3 Brown Bullhead and Largemouth Bass Brown bullhead and largemouth bass were selected as indicators for fish species based upon data availability, rather than upon the value of such data to an ecological risk assessment. Their appropriateness to an ecological risk assessment is questionable. The selection of indicator species at various trophic levels must take into account the links between such trophic levels. Without such a link, the pathway is incomplete, and the validity of the overall exposure assessment is questionable, at best. The Phase 1 Report fails to show where these species fit into the pathway and how, based on linkage, they are appropriate indicators.

3.3.5.3 Exposure Quantification (B.7.3.3)

Once exposure pathways and receptors have been identified, the next step in the assessment process is a quantification of exposure. At this step, site specific information is critical to a valid assessment. In dealing with a system as dynamic as the Upper Hudson River, changes in PCB concentrations and constituents are expected, and have been found to occur. Without current data on levels of PCBs in both abiotic and biotic components of the Upper Hudson River, exposure quantification errors are greatly magnified.

HRP 001 1609

In addition, current knowledge of PCBs is expanding in the area of toxicity differences of PCB congeners. Saying that a certain amount of PCBs is harmful or fatal to an organism is now considered to be fairly meaningless, because the toxicity of highly chlorinated, coplanar PCB congeners differs dramatically from less-chlorinated ones. The impact of these flaws, and other analytical problems are discussed below for the various media present in the Hudson River.

3.3.5.3.1 Water

The discussion of PCBs in water should provide information concerning solids concentrations in the water. Were the samples filtered and how much variation of PCBs in the water column is related to solids content? This information is important in the assessment of PCBs available to biota in the water column through suspension of contaminated solids.

3.3.5.3.2 Sediments

Information concerning sediment depths used in the PCB exposure analyses should be provided. Surface sediments are normally more available to the biota than deeper sediments. Therefore, if the surface sediments are different than the deeper sediments, then the use of PCB concentrations found in deeper sediments to determine toxicity potential to benthic animals may result in misestimation of the concentrations of PCBs available m

001 1610

3.3.5.3.3 Herring Gull

To quantify the exposure of herring gulls to PCBs, the Phase 1 Reports relies upon available information regarding fish PCB concentration. As a result, the estimated exposure may be incorrect for several reasons:

- Herring gulls are opportunistic feeders, and may not in fact be consuming the estimated levels of fish.
- The relationship to amount of PCBs ingested and the tissue levels in birds is unknown.

The EPA report assumes that 50 percent of the herring gull's diet is comprised of fish and that an adult gull consumes an average of about 20 percent of its body weight each day. The first factor, the percentage of fish in the diet, can vary markedly among individuals and among gull populations. The design of the Phase 2 data collection program should include a component to obtain specific information on this for the "indicator" gulls breeding along the Upper Hudson River. It is probable that this population of gulls secures more of its food resources from upland fields and municipal waste disposal sitesthan do gulls breeding on offshore islands in the Great Lakes. This would reduce the overall proportion of fish in the diet. A more diverse feeding ecology is expected in populations of migrant gulls whose individuals become exposed to and accustomed to feeding in different habitats and on different food types during migratory transit and at their wintering locations.

To estimate the quantity of PCBs consumed by gulls, the assessment uses total PCB concentrations for the three fish

HRP 001 1611

species in the Upper Hudson River for which recent analytical information is available. Two errors appear in this assessment:

- Instead of using mean PCB concentration values, the 95 percent upper confidence bound of the mean (95 percent CB = mean + t (0.975)) \cdot SE) was used for the exposure assumptions.
- The fish species comprising the analyzed data set (Largemouth Bass, Pumpkinseed, Brown Bullhead) are larger in body mass in comparison to the forage fish that gulls typically feed on. Being at a lower trophic level than bass and pumpkinseed, shiners (forage fish) probably have lower body burdens of PCBs. Moreover, the bullhead is a bottom feeder that is not prone to being taken by herring gulls. The range of 95 percent upper confidence bound concentrations in the fish that were assumed in EPA's assessment is 2 to 50 μ g/g. Adjusting this range to meet the assumption that 50 percent of the gull's diet is fish, gives a dietary range of 1 - 25 μ g/g. If values for the bullhead are not considered, the range becomes 3 to 13 μ g/g and the adjusted range 1.5 to 7.5 μ g/g.

In Table B.7-1 of the Phase 1 Report, the daily rate of PCB intake by the herring gull (listed as $0.1 - 5 \ \mu g/g$ body weight/day) is in error. The correct range, using EPA's data and assumptions, is $0.1 - 2.5 \ \mu g/g/day$.

To calculate estimated whole body concentrations of PCBs in herring gulls and their eggs, EPA used empiricallyderived bioaccumulation factors from residue analyses performed on biota inhabiting the Lake Ontario basin. The BAFs are tabulated in Braune and Norstrom (1989); they relate PCB concentrations in the Alewife prey of gulls, to PCB concentrations in gull eggs and to body burdens in adults. These BAFs were used in conjunction with the range of concentrations of 00 PCBs in Upper Hudson River fish to estimate the ranges of body and egg burdens that are listed in Table B.7-1 of the Phase 1 Report. While these BAFs are probably very useful for describing concentration relationships between prey and gulls on the Great Lakes (where herring gulls are year-round residents), they are invalid for use on the Hudson River because these gulls are migratory.

Adult gulls inhabiting the Lake Ontario environs are continuously at steady state with respect to PCBs (except for the temporary dip in female PCB levels associated with translocation of contaminants to the eggs), whereas migrant individuals may never reach steady-state kinetics and are especially unlikely to be in steady-state condition at the time eggs are deposited. Accordingly, applying a BAF determined for birds at steady state to birds (and their eggs) at less than steady-state levels results in overestimation of body and egg concentrations. Depending on the specific accumulation and depuration kinetics, this overestimation could prove to be substantial.

In its Phase 1 assessment, EPA used BAFs that were calculated based on total PCB concentrations. Braune and Norstrom (1989) also tabulated BAF values for all the PCB congeners that were detected in Alewife, Herring Gulls, and gull egg samples. This tabulation demonstrates major congenerspecific differences in bioaccumulation between fish and gulls. The preferential and sometimes dramatic accumulation of non-ortho chlorine substituted PCB congeners in higher animals, relative to the total mix of congeners in the original commercial PCB mixture and the biota lower in food chain, has been quantified recently by several environmental toxicologists (Tanabe *et al.*, 1987,

154

1989; Kubiak et al., 1989; Smith et al., 1990) and certainly occurs in the Upper Hudson River ecosystem.

All these findings strongly suggest the need for the analysis of individual isomers when evaluating the potential toxic effects of PCB mixtures on biota. Tanabe et al. (1989) stated that, "isomer-specific information on both environmental residue levels and their toxic and biological potential are essential for evaluating the toxic significance of man-made chemicals to humans and wildlife."

Little or no data on concentrations of the critical PCB congeners are available for the abiotic and biotic components of the Upper Hudson River. This is a fundamental deficiency in the Phase 1 Report's exposure assessment.

Additionally, the Phase 1 Report describes EPA's method for calculation of PCB levels in Herring Gulls based on previously reported bioaccumulation factors (Braune and Norstrum, 1989). The Phase 1 Report points out that this calculation ignores the mechanisms and rates of PCB transfer from food to body tissues and notes that the resulting PCB estimates in gulls are "very uncertain."

Data from literature reviews of PCBs found in wild waterfowl (including gulls, Osprey, Bald Eagle, herons and loons) reflect a wide range of values depending on whether brain, liver, embryo or fat was analyzed, but generally the concentrations were lower than those estimated by the methodology described in EPA's Phase 1 Report.

3.3.5.3.4 Mink

For mink, the Phase 1 Report discussed possible dietary intake of PCBs based on daily fish consumption and estimated the dose per day. Because of insufficient data, no effort was made to calculate levels of PCBs in mink body tissue. Should EPA choose to expand upon the mink exposure quantification analysis in future phases, it should bear in mind the following comments regarding their quantification assumptions.

In its estimate of the rate of uptake of PCBs in fish from the Upper Hudson River, the Phase 1 Report assumed that 50 percent of the mink's diet is comprised of fish, and that the adult mink consumes approximately 15 percent of its body weight per day. Based on a review of the studies by Linscombe *et al.* (1982) and Aulerich *et al.* (1973), the values used by EPA for the mink's body weight and total food consumption rate seem appropriate. However, a review of these same references used by EPA to develop their estimate of the fish portion of the mink's diet indicates that the 50 percent value used by EPA is exaggerated.

Aulerich et al. (1973) indicated that a 30 percent fish diet was used in their mink feeding studies not because it was typical of mink diets, but because it was the percentage used in mink ranching to yield an optimal product. However, such an optimal portion of fish is not always available to wild populations who feed on a diversified diet of frogs, crayfish, invertebrates, muskrats and any other prey items that they can find and kill (Linscombe et al., 1982). Erlinge (1969) and

Gilbert and Nancekivell (1982) state that small mammals are the predominant food item of mink, followed by fish and perhaps Three studies on the consumption habits of mink crayfish. suggest that the fish portion of the mink diet is well below 50 In a study of mink collected in an Iowa marsh, only percent. 10.5 percent of the minks' diet was comprised of fish (Waller, 1962). In studies of mink in Missouri and Michigan, the occurrence of fish in the mink diet ranged from 11 to 31 percent, and the actual volume of fish measured in mink stomachs ranged from only 6 to 20 percent (Korschgen, 1958; Sealander, 1943). A fourth study from Sweden suggests that fish comprise 60.2 percent of the minks' diet (Erlinge, 1969). None of these authors gave detailed descriptions of the mink habitats encountered in their studies, thus it is somewhat difficult to determine which study reflects conditions most similar to the Upper Hudson River habitat. However, the studies in Missouri and Michigan seem more comprehensive, because volume of fish consumed is considered as well as the occurrence of fish in the diet. In addition, the mink habitats in these states are probably more similar to the Upper Hudson area than is that in Sweden. Because the nearby upland habitats in the Upper Hudson River area support abundant populations of suitable prey, it is most likely that the portion of fish in the diets of mink in the Upper Hudson River area falls at the lower end of the ranges reported in the literature.

In addition, there are a number of problems with the assumptions the Phase 1 Report used in assigning representative $\overset{\circ}{\Box}$ concentrations of PCBs in fish assumed to be ingested by mink.

First, the method used by EPA to estimate typical concentrations in fish consumed by mink greatly overestimates the degree of PCB contamination in most fish from the Upper Hudson River. Second, the fish species used in the EPA analysis are unlikely to be consumed by mink in the Upper Hudson River area. Finally, the fish tissue concentrations were measured in a number of older, larger fish that would not fall prey to mink. Mink are more likely to feed on smaller fish that would have lower body burdens of PCBs. These three factors result in overestimation of PCBs consumed by mink.

The highest fish tissue concentrations measured between, 1986 and 1988 were obtained from Thompson Island Pool. These levels (ranging from 5.9 ppm to 48.7 ppm) were considerably higher than the levels measured in Federal Dam samples (2.3 ppm to 5.8 ppm) and Stillwater samples (3.6 to 13.9 ppm). However, EPA used the highest upper bound concentration (48.7 ppm) from Thompson Island Pool to estimate exposure for mink. This is inappropriate. Because fish tissue levels are substantially higher from Thompson Island Pool than they are from other reaches, or from the combined reaches of the Upper Hudson River, use of these PCB levels will substantially overestimate actual risks to wildlife in this region. For the remaining reaches of the river, the use of Thompson Island Pool levels is inappropriate. Rather, concentration data for all reaches of the river should be used to assess risks to piscivorous mammals on the Upper Hudson River.

HRP 001 1617

An additional concern is that mink do not consume exclusively, or in any significant amount, the types of fish that comprise the fish concentration data set used by EPA. The largemouth bass, pumpkinseed, and brown bullhead are large fish that exist on a fairly high trophic level and thus will have higher concentrations of PCBs than other fish consumed by mink. These fish do not lend themselves to capture by mink in part due to habitat preference. Mink feed in shallow, streamside riparian habitats and are unable to capture these larger fish on a frequent basis due to the deeper water habitats preferred by such species. It is, therefore, not appropriate to model exposures to mink based on the largest fish with the highest concentrations of PCBs of all fish on the Upper Hudson River. Rather, data on concentrations of PCBs in the types of fish consumed by mink should be used in this assessment.

In the Phase 1 assessment of the impacts of PCBs on mink in the Upper Hudson River area, EPA estimated, based on the parameters discussed above, a daily intake or dose of PCBs in mink of 0.15 to 3.8 mg/kg-day. This range correlates with the range of PCB concentrations (2 to 50 mg/kg) assumed by EPA to exist in the fish consumed by mink. Because the majority of studies on the toxicity of PCBs in mink have been based on concentrations of PCBs in the diet, and not on absorbed doses or tissue levels, EPA did not include estimates of potential absorbed doses of these compounds.

If a more appropriate value of 20 percent is used to $\overset{\circ}{\underset{\omega}{\overset{\circ}{\overset{\circ}{1}}}}$ present the portion of the mink's diet that is comprised of fish $\overset{\circ}{\underset{\omega}{\overset{\circ}{1}}}$

HRP

(rather than the 50 percent assumed by EPA), the estimated dietary intakes of PCBs from the Upper Hudson would be reduced to 0.06 to 1.52 mg/kg-day. When this correction is combined with more reasonable estimates of the concentration of PCBs in fish consumed by mink (for example: 1.75 to 20.27 mg/kg), the daily intakes of PCBs by Upper Hudson River mink is estimated to be 0.05 to 0.6 mg/kg-day. This range of fish concentrations represents the range measured in whole body pumpkinseed samples from 1986-1988. Although it is not clear that mink consume pumpkinseed, these smaller fish are more representative of the size of fish normally consumed by these mammals, and thus their PCB concentration range more applicable to a mink exposure assessment.

Additionally, rather than estimate a daily intake based on total concentration of PCBs, it would be best to determine daily intakes for mink on a congener-specific basis. A number of researchers (Bleavins et al., 1980; Hornshaw et al., 1983; Ringer, 1983) have suggested that the bioaccumulation and toxicity of PCBs varies considerably, depending on the degree of chlorination of the particular PCB. It is critical to gain an understanding of the distribution of PCB congeners in the mink diet, the degree of accumulation of these congeners in the mink, and the toxic effect of these various compounds when evaluating the impact of concentrations of PCBs on Upper Hudson River mink. Such considerations are not reflected in the Phase 1 estimate of dietary intake of PCBs in mink.

3.3.5.4 Toxicity Assessment (B.7.4)

The toxicity assessment in the Phase 1 Report opens with an important observation: "The toxicity of PCBs to aquatic and terrestrial organisms can vary considerably depending on congener and Aroclor composition." Yet, this qualification is virtually ignored by EPA in the Phase 1 Report's discussion of Thus, there is nothing in available literature on PCB toxicity. the Report to allow evaluation of the applicability of these studies to site-specific conditions. Without this evaluation an opinion on the relevance and utility of these toxicity studies is not valid.

There is No Valid Scientific Basis for the "Risk 3.3.6 Characterization" Presented in the Phase 1 Ecological Assessment

Although stating at the outset that the ecological data available does not allow for a conclusion that ecological risks specifically attributable to PCBs exist in the Upper Hudson River, the Phase 1 Report ecological assessment nevertheless concludes with a risk characterization. This "risk characterization" is derived from a comparison of estimated PCB exposure levels to published information regarding toxicity and PCB guidelines. Such an exercise provides no defensible result. A risk characterization must be site-specific if it is to provide any guidance in the selection of a remedy. The Phase 1 risk characterization is based upon outdated and limited specific information and inapplicable general information. Table B.7-1 indicates a low level of confidence in data for both the Herring 1620 Gull and mink. In spite of insufficient data, the Phase 1 Report

HRP

provides, in Table B.7-3, proposed ecological guidelines for limits to PCB concentrations in birds and mammals. Although the footnote indicates that the values are not enforceable standards, presentation of this table implies more knowledge than is currently available regarding allowable concentrations of PCBs in wildlife.

Previously, this section of the comment document discussed the problems with the Phase 1 exposure and toxicity information, concluding that no definitive site-specific information had been provided. The Phase 1 Report's identification of proposed criteria and guidance is equally lacking in information relating the criteria and guidance identified to site-specific conditions. If EPA is to adequately characterize ecological risk for use as a basis for determining the risks and benefits of remedial alternatives, it must assess the validity of the "proposed" criteria and guidelines when applied to the Hudson River ecosystem.

Although GE believes that there is insufficient identification of risk to justify proposing guidelines at this time, GE would nevertheless like to take this opportunity to provide comments addressing EPA's proposed guidelines as presented in the text of the Phase 1 Report and at Table B.7-3. It is clear from a review of the various proposed guidelines, and other relevant literature not reviewed by EPA, that the selection process was highly arbitrary and overly conservative.

HRP 001 1621

3.3.6.1 Fish Tissue and Egg Tissue Guideline Values

In evaluating the potential ecological impacts to fish in the Upper Hudson River under the current river conditions (assuming no disruptive remedial action, such as dredging has been implemented), the Phase 1 Report recognizes that PCBs are primarily a chronic toxicant in the environment; *i.e.* ambient PCB concentrations are rarely high enough to pose an acute toxic effect. The Phase 1 Report proposes a maximum PCB fish tissue guideline level of 0.4 μ g/g based on a study of rainbow trout (not a species of concern in the Upper Hudson) which reported embryotoxic effects at tissue levels of 0.39 μ g/g (Eisler, 1986; EPA, 1980). However, the tissue concentration reported in that study is not appropriate for deriving a fish tissue guideline, because the 0.39 μ g/g level was an egg tissue residue concentration and not adult whole-body residue concentration.

Values in the literature for PCB fish tissue levels associated with adverse chronic effects range from 0.6 μ g/g in bluegill to 250 μ g/g in carp (EPA, 1980). The majority of effects measured are non-specific biochemical and physiological responses such as altered enzyme activity and increased thyroid activity. Mayer et al. (1977) observed whole body Aroclor 1254 residue values as low as 0.59 μ g/g associated with increased thyroid activity in coho salmon. In addition, Desaiah et al. Hy (1972) indicated inhibition of ATPase activity at whole body Aroclor 1242 residues of 0.6 g/g in bluegill and Gruger et al. [1977) (1977) reported induction of AHH microsomal enzyme activity at Wole whole body Aroclor 1242 residues of 0.6 g/g in bluegill and Gruger et al. (1977) reported induction of AHH microsomal enzyme activity at whole body Aroclor 1242 residues of 2.0 g/g in coho salmon.

It is important to note that these biochemical responses are not definitive markers for toxicity. There is no positive correlation between these non-specific endpoints and adverse health impacts to fish. In addition, variations in enzyme induction have been demonstrated within a species. For example, species variation in the induction of the hepatic microsomal enzyme aryl hydrocarbon hydroxylase (AHH) has been observed in various inbred strains of laboratory mice (Greig et al. 1984). These authors concluded that AHH induction may be influenced by more than one genetic locus. As the genetic variability of the animal increases, the assortment of gene loci controlling the expression of hepatotoxicity is likely to increase thereby altering responsiveness (Greig et al. 1984). These variations can be further complicated by differences between male and female test organisms as demonstrated by variations in hepatotoxic sensitivity to TCDD for male and female mice (Greig et al., 1984). Although it appears that a conservative maximum permissible PCB tissue level, based on biochemical and physiological endpoints, should be in the range of 0.6 to 1.0 μ g/g, the actual adverse effect level is likely higher. Therefore, the value of 0.4 μ g/g proposed by the Phase 1 Report is inappropriate.

HRP 001 1623

Regarding a fish egg tissue guideline for PCBs, EPA proposes a value of 0.33 μ g/g based on a rainbow trout study reported by Eisler (1986). The actual study (Hogan and Brauhn, 1975) reported a total PCB level of 0.39 μ g/g in the rainbow trout egg associated with 10 to 28 percent mortality. Of this total, 0.33 μ g/g was Aroclor 1254. However, a number of confounding factors do not permit conclusions to be drawn from this study. First, DDT was also detected in these egg tissues at a concentration of 0.15 μ g/g and may have influenced the reported mortality. Second, because no control groups were established it cannot be determined if a portion of the observed mortality may have resulted from the shipping (air-shipped), handling, and laboratory climate controls. A similar study examining PCBs and DDE in lake trout eggs reported by Niimi (1983) reported an average mortality of 22 percent in the control group. Therefore, a mortality rate of between 10 and 28 percent may not be significantly different from that expected for control groups.

A fish egg tissue guideline is difficult to develop from the limited studies available. Snarski (1976) reported favorable hatchability, alevin-juvenile survival and growth resulting from brook trout eggs with mean PCB residues of 1.8 μ g/g. In addition, Zitko and Saunders (1979) reported 80 to 91 percent hatching success in Atlantic salmon eggs containing 1.9 to 6.5 μ g/g Aroclor 1254 per gram lipid. Although it is recognized that there may be species sensitivity differences between rainbow trout and Atlantic salmon, these results do suggest that fish egg tissue concentrations ranging from 1.8 to

165

6.5 μ g/g PCBs (Aroclor 1254) do not impact the hatchability and survival rare of fish eggs.

Clearly, then, a fish egg tissue guideline of 0.33 μ g/l is inappropriate, and insufficient data on relevant species in the Upper Hudson make it inappropriate, to establish such a guideline.

3.3.6.2 Application of EPA's Toxicity Guidelines to Herring Gulls

3.3.6.2.1 Whole Egg Guidelines

The Phase 1 assessment of risk to piscivorous birds uses a value of 0.4 μ g/g as a proposed guideline for protecting avian embryos, and compares this to modeled values for the herring gull egg. This concentration is based on Kubiak's personal interpretation of Britton and Huston (1973), as conveyed to the authors of the risk assessment through personal communication. However, Britton and Huston (1973) concluded that PCB effects are manifested at much higher concentrations than this 0.4 μ g/g level. They fed White Leghorn Chickens diets containing PCBs and demonstrated that a dietary level of 20 μ g/g resulted in a significant reduction in hatchability. Eggs produced by hens fed a diet of 20 μ g/g showed no difference in hatchability from controls in the first 5 weeks of the test period but in the 6th week "a slight reduction which disappeared after one week of feeding the PCB-free diet" occurred. Because this 6th week value was statistically different from that of the control group, 10 μ g/g is likely a conservative LOAEL for PCB embryotoxicity in the White Leghorn. No reduction in

HRP 001 1625

hatchability occurred at the 5 μ g/g dietary concentration. As summarized below, there appears to be no reasonable basis for adjusting the 10 μ g/g guideline used in the risk assessment.

Britton and Huston's (1973) work was performed using domestic chickens as subjects. Several avian toxicologists examining PCB and TCDD fetotoxicity have concluded that the chicken is far more sensitive to these AHH-inducing chemicals than is any of the other species tested. Because of its extreme sensitivity, it is questionable if extrapolations from the chicken should be used for guideline-setting. For example, Brunström (1989) indicated that the several varieties of domestic chicken studied with respect to the embryotoxicity of the very toxic congener 3, 3',4,4'-tetrachlorobiphenyl (TeCB) all proved to be very sensitive. "In contrast, embryos of eight other avian species tested all seem to be considerably less sensitive than chick embryos to TeCB. Only in turkey and pheasant embryos were any adverse effects of TeCB noted, whereas no effects were found in embryos from goldeneyes, mallards, domestic ducks, geese, herring gulls and black-headed gulls at the highest doses administered (1 to 3 orders of magnitude higher doses than the approximate LD_{∞} in chick embryos)." The highest of the doses given to these species were 5,000 ng TeCB/g egg for the domestic duck and 1,000 ng TeCB/g egg for the other duck, goose, and gull species. Brunström (1988) stated, "These doses did not affect the viability of the embryos and caused no gross abnormalities". He concluded that these chemicals are extremely toxic in chick

HRP 001 1626

embryos but appear to be considerably less toxic in embryos of the other avian species testes (Brunström, 1989).

Further demonstration of a substantial difference in sensitivity to toxicity by AHH-active chemicals between chickens and other species comes from Nikolaidis et al. (1988; 1989), who examined the effects of TCDD on lymphoid development in the bursa of Fabricius and the thymus of chickens, turkeys, and ducks. The bursa and the thymus are sites of lymphocyte formation in embryo birds. The toxic PCBs and TCDD act on targets in the immune system, causing a characteristic pattern of effects typified by inhibition of lymphoid development. They concluded that "The chicken embryo thymus was about two orders of magnitude more sensitive than turkey and duck thymus to TCDD in vitro. This finding is in line with a more than 20-fold difference in sensitivity to TeCB in ovo between chicken and turkey embryo thymus reported by Brunström and Lund (1988). Our results strongly suggest that the species differences are inherent to the immune system and not a result of differences in toxicokinetics." (Nikolaidis et al., 1989).

Several other researchers have also concluded that the domestic chicken embryo is far more sensitive than the embryos of other species to 2,3,7,8-TCDD and its congeners (Elliott et al., 1988; 1989; Kenega and Norris, 1983; McConnell, 1985). In fact, Bellward et al. (1990) indicated that because of its ultrasensitivity compared to other species, the chicken embryo may be a poor model for wild avian species. Based upon the

HRP 001 1627

above, EPA's use of a benchmark derived from experiments with chickens is inappropriate.

With respect to the herring gull, egg injection experiments have demonstrated that 142 μ g PCB/g egg is a PCB noeffect level for gull embryos (Gilman et al., 1978). These scientists injected herring gull eggs with known quantities of contaminant mixtures (including PCBs, DDE, mirex and HCB) extracted from Lake Ontario herring gull eggs. It is probable that TCDD and TCDF were also present in these extracts, but analytical techniques of appropriate sensitivity were not available at the time this research was undertaken. After injection, adult herring gulls incubated the eggs, thereby eliminating possible effects of either abnormal or artificial incubation. PCB concentrations in the injected eggs ranged from 51.5 to 142 μ g PCB/g egg. All dose groups showed no difference from the control group in hatching or survival of chicks. Consequently, a whole egg concentration of at least 142 ppm does not affect herring gull embryonic or chick viability. Because the injection included several other toxins, considering a PCB concentration of 142 ppm as a no effect level is very conservative -- antagonism, potentiation, synergism and/or additivity among all chemicals present resulted in no effects on reproduction.

Weseloh et al. (1990) reported PCB concentrations in herring gull eggs collected at 14 breeding colonies in Lake Erie. 100 Egg values at these sites ranged from 35 to 150 ppm. A11

RP

1628

colonies showed normal production of young, indicating that egg concentrations as high as 140 ppm do not result in extrinsically or intrinsically-mediated reproductive dysfunctions in this gull. In view of the availability of these data reported by Gilman et al. (1978) and Weseloh et al. (1990), EPA should compare its modelled egg concentrations for herring gulls on the Upper Hudson River with these empirical results for the same species, rather than compare modelled estimates to a value for the domestic chicken.

With recent advances in analytical techniques that permit congener-specific analysis and with the increasing realization that just a few of the 209 PCB congeners contribute significantly to chronic toxicity at the higher food chain levels, it is probable that criteria and standards for wildlife protection will become modified to specify allowable levels for selected isomers. For subsequent referral when congener-specific data on PCBs in the Upper Hudson River become available to piscivorous birds, the following summarized NOAELs and LOAELs for 3,3',4,4'-TeCB determined by egg injection experiments: (Bronström, 1988; Brunström, 1989; Brunström and Reutergårah, 1986; Brunström and Lund, 1988; Brunström et al., 1990).
	Injected Dose	
	in ng TECB/g	
Species	egg	Versus Control Group
Domestic Chicken	4	Significant effect
Ring-necked Pheasant	100	No effect
	1,000	Significant effect
Domestic Turkey	1,000	Significant effect
Goldeneye	1,000	No effect
Black-headed Gull	1,000	No effect
Herring Gull	1,000	No effect
Domestic Goose	1,000	No effect
Common Eider	1,000	No effect
Mallard	1,000	No effect
Domestic Duck	5,000	No effect

3.3.6.2.2 Herring Gull Dietary Guideline

In its Phase 1 Report, EPA has adopted a PCB dietary guideline of 3 μ g/g. This is a value proposed by Eisler (1986) as being protective of wild birds. It is based on work by McLane and Hughes (1980), who fed Screech Owls a diet containing 3 ppm Aroclor 1248 and monitored reproductive effects. This dietary dose resulted in no detectable effects on Screech Owl reproduction. No differences between experimental and control subjects existed in the quantified parameters: eggshell thickness, clutch size, and hatching and fledgling success. Because only one dose was administered, no conclusions can be derived from this experiment concerning the PCB dietary concentration at which reproductive effects actually become

171

manifested. The dietary level that causes effects could be slightly higher or much higher, but in the absence of a graded dose experimental design it is not possible to determine this threshold. McLane and Hughes (1980) concluded, "The PCB residues in both eggs and carcasses of birds dosed with 3 ppm Aroclor 1248 appear to be in a mid-zone, neither very high nor very low, as compared with residues in tissues of wild birds. Reproduction was not perceptively affected at this dosage level." It makes little sense to use a dietary value as a guidance criterion that results in body and egg burdens that are "in the mid-zone" of typically-occurring concentrations in wild bird populations that are experiencing no adverse health or reproductive effects.

The New York State Department of Environmental Conservation has selected a LOAEL of 0.224 mg/kg body weight/day for fish-eating birds (Newell et al., 1987). After adjusting this LOAEL to a NOAEL and applying a species sensitivity factor, NYDEC calculates a criterion of 0.11 mg/kg dietary PCB as a concentration that is protective of piscivorous birds. This concentration was extrapolated from Britton's and Huston's (1973) feeding studies of chickens. As described above, a body of empirical data is accumulating that demonstrates that domestic chickens are extremely sensitive to PCBs and PCDDs when compared to all other bird species tested. Preferably, guidelines should not be based on results of experimental toxicology work on RP chickens and even if they are, an adjustment factor for 100 interspecific sensitivities should be unnecessary.

172

163]

3.3.6.2.3 Brain Concentration Guideline

The PCB concentration guideline for brain tissue used by EPA in its Phase 1 Report is 54 μ g/g, as taken from Eisler $_{n}$ This guideline value appears to be very conservative. (1986). It comes from work by Stickel et al. (1984), who measured PCB concentrations in brain extracts of several songbird species that had experienced mortality after having been administered a dietary dosage of 1500 ppm Aroclor 1254. PCB brain residuals of the dead birds ranged from 349 to 763 μ g/g, while concentrations in brains of sacrificed birds that had not experienced mortality ranged from 54 (the value adopted by Eisler and used by EPA) to 301 ppm. Stickel et al. (1984) concluded: "An appropriate break point for high probability of PCB-induced mortality would be around 310 ppm (three standard deviations below the mean)." Heinz et al. (1985) found that "laboratory studies demonstrated that 300 ppm or more of PCB residues in brain are needed to cause death." Accordingly, arbitrarily reducing the 310 μ g/g concentrations (Stickel et al., 1984) to the lowest value for all birds not experiencing mortality (54 μ g/g) results in an overly conservative guideline.

3.3.6.3 EPA's Criteria and Guidelines Applied to Mink

3.3.6.3.1 NYSDEC Fish Flesh Criteria for Piscivorous Wildlife

In the Phase 1 Report, EPA adopted the NYSDEC (Newell et al., 1987) fish flesh criteria of 0.13 mg/kg as a dietary guideline for the protection of piscivorous wildlife on the Upper

001 1632

HRP

Hudson River. This dietary guideline is based on a study by Platonow and Karstad (1973) in which the reproductive success of mink fed beef contaminated with 3.57 or 0.64 mg/kg Aroclor 1254 was evaluated. Mink fed diets containing 3.57 mg/kg Aroclor 1254 experienced 100 percent mortality. Decreased reproductive success was observed in mink fed a beef diet containing 0.64 mg/kg Aroclor 1254. NYSDEC (Newell *et al.*, 1987) derived a NOEL of 0.13 mg/kg from this study (Platonow and Karstad, 1973) by applying a factor of 0.2 to the LOEL of 0.64 mg/kg.

There are at least two reasons why this fish flesh value may be inappropriate for use in relating fish levels in the, Upper Hudson River to potential adverse effects in resident mink. First, the 0.13 mg/kg criteria is based on a study that is not conclusive regarding the source of reproductive impairment in the study animals. Unfortunately, the results of the Platonow and Karstad (1973) study are confounded by the fact that the reproductive success in the control group (1.8 kits per female) was also poor when compared to other studies (>6 kits per female) (Wren, 1991). In addition, the beef ration fed to the controls contained very low concentrations of other compounds, including DDE (0.012 ppm), DDD (0.01 ppm), DDT (0.033 ppm) and PCBs (0.3 ppm). As a result, it is not clear whether the reduced reproductive success observed in the 0.64 mg/kg group was directly attributable to the presence of concentrations of PCBs in the mink's diet. In addition, some authors (Ringer, 1983; Hornshaw et al., 1983) have suggested that the effects of

metabolized PCBs (i.e., PCBs that have been fed to and metabolized by cows before being introduced to the mink diet) may be considerably more toxic than those derived directly in the diet. Several toxicity studies suggest that the higher the chlorine content of the Aroclor, the greater the detrimental effect of the particular PCB on reproduction in mink (Aulerick and Ringer, 1977; Goldstein et al., 1985). This may be explained by the fact that higher chlorinated PCBs have a longer half-life iOn mammalian tissues than do the lesser-chlorinated compounds (Curley et al., 1971; Hornshaw et al., 1983). As demonstrated by Ringer (1983), reproductive success was impaired in minks fed 2 mg/kg Aroclor 1254, yet adverse reproductive effects were not observed in mink fed the same concentration (2 mg/kg) of Aroclors 1026, 1221, or 1242. Similarly, as pointed out by Ringer (1983), Bleavins et al. (1980) have shown that the feeding of 5 mg/kg of Aroclor 1242 is detrimental to reproduction, whereas dietary concentrations of Aroclor 1016 as high as 20 mg/kg did not impact the reproductive success of mink.

Results of a recent study conducted by Aulerich and coworkers (1985) demonstrate that certain symmetrical PCBs (pure grade) are more toxic to mink than are some of the Aroclors (1254, 1242, 1016) discussed thus far. Mink fed diets containing 0.1 mg/kg 3,4,5,3,4',5'-hexachlorobiphenyl (345 HCB) exhibited 100 percent mortality within 60 days. Those animals receiving 0.5 mg/kg 345 HCB in the diet showed 50 percent mortality in 3 months. These effects are more severe than the reproductive

HRP 001 1634

effects observed by Platonow and Karstad (1973) in mink fed 0.64 mg/kg in a beef diet. Wren (1991) has suggested that the presence of planar 3-methylcholantrene-type (3MC-type) congeners in technical grade PCBs is associated with adverse responses observed in mink exposed to these compounds. Clearly, it is critical to determine the relative presence of various PCB congeners in the fish of the Upper Hudson River before implementing criteria or guidelines that are based on the extreme toxicity of Aroclor 1254. There is ample data available from the studies described above to develop fish flesh criteria for a variety of Aroclors.

Other researchers studying the effects of PCBcontaminated diets on mink (Aulerich and Ringer, 1977; Bleavins et al., 1980) have observed dietary threshold levels that are 3 to 30 times greater than the 0.64 mg/kg level identified in the beef-diet study (Platonow and Karstad, 1973). In many of these studies, the PCBs being fed to mink were less chlorinated than those used in the Platonow and Karstad (1973) study.

> 3.3.6.3.2 USFWS Recommended Daily Tolerance Level for Mink

For comparison with their estimated intakes of PCBs by mink on the Upper Hudson River, EPA has adopted the USFWS (Eisler, 1986) recommended dietary tolerance level for mink of 1.54 μ g/kg-day. This value, developed by Eisler (1986), is HRP erroneous. It was derived by making several inappropriate 100 adjustments to the Platonow and Karstad (1973) LOEL of 0.64 mg/kg. Eisler (1986) assumed that the mink consume up to twice 1635

the amount of food per day (16.4 to 27.2 percent of their body weight per day) as has been documented in a number of studies (Aulerich et al., 1973; Linscombe et al., 1982; Newell et al., 1987). Eisler also used a safety factor of 100 to adjust the LOEL of 0.64 mg/kg to a NOEL. Use of such a large safety factor is completely inappropriate when establishing criteria levels for a species that has been identified as being most sensitive to the effects of PCBs (Aulerich and Ringer, 1977; Bleavins et al., 1980; Ringer, 1983; Aulerich et al., 1985; Newell et al., 1987).

According to EPA (1988), there is no reason to add a safety factor of 10 for differences in species if it has already been determined that the species under consideration is the most sensitive. Therefore, it is only appropriate to use a safety factor of 10 to adjust a LOEL to a NOEL. If a safety factor of 0.1 were applied to the same LOEL (0.64 mg/kg), and a more representative food consumption rate of 15 percent of the mink body weight were also applied, a daily tolerance level of 9.6 $\mu g/kg$ -day would result.

3.3.6.4 Ambient Water Quality Criteria for PCBs

Under the Clean Water Act, EPA was charged with the development of Ambient Water Quality Criteria (AWQC) for evaluating the hazards to human health and the environment from compounds in surface waters (USEPA, 1980). EPA has established a criterion of 0.0014 mg/l for PCBs in water, based on the protection of the most sensitive mammalian species, the mink.

impacts on mink exposed through ingestion of fish from that water. It was derived using a bioconcentration factor for PCBs in fish of 45,000, and a threshold level for PCBs in the mink diet of 0.64 mg/kg. There are a number of assumptions used in EPA's derivation of the 0.0014 mg/l ambient water quality criteria for PCBs that impact the appropriateness of direct application of this criterion to the Upper Hudson River. These problems are discussed in the following sections.

3.3.6.5 Mink Intake of Fish

The equation used to derive the EPA (1980) AWQC back calculates an acceptable water quality standard by applying an estimated bioaccumulation factor to a PCB level that was shown to cause reproductive failure in mink. There are several factors that affect the appropriateness of direct application of this criterion to the Upper Hudson River.

As previously discussed, there are several confounding factors involved in the Platanow and Karstad (1973) study from which the dietary threshold value of 0.64 mg/kg was derived. In addition, given the wide range of environmental and toxicological behavior of various PCB congeners, it is inappropriate to derive or to apply an AWQC for total PCBs; rather, it is more appropriate to develop guidelines based on specific congeners.

In addition, EPA neglected to account for the fact that the diet of the wild mink is very diverse and would not be comprised totally of fish. Linscomb (1982) estimated that fish comprises between 6 and 20 percent of the mink diet by volume. Given that nearby upland habitats in the Upper River area support

HRP 001 1637

abundant populations of suitable prey, it is likely that the fish in the diet of mink are on the lower end of the range reported to the literature. Also, mink will feed both in the Hudson River and on the river's tributaries. Fish caught in the tributaries are not in equilibrium with PCB levels in the Hudson itself. Thus, it is likely that only a portion of the mink's fish diet will be at concentrations observed in fish tissues from the Upper Hudson.

3.3.6.6 Bioaccumulation in Fish

In developing an ecological guideline for PCBs, an appropriate biological accumulation factor (BAF) is required to predict the levels of PCBs fish will accumulate from their surroundings. The Phase 1 Report uses a fish bioconcentration (BCF) of 45,000 to represent the degree of PCB accumulation in fish in the Upper Hudson River, a value based on the geometric mean of three BCFs from rainbow and brook trout (Eisler, 1986). The BCF approach is not an appropriate model for lipophilic compounds which are primarily bound with the sediment. Additionally, rainbow and brook trout, although present, do not represent the dominant fish species in the Upper Hudson River. Furthermore, the BCF derived from these species were not lipid normalized to reflect the lipid content of the dominant fish species. The ramifications of applying incorrect accumulation factors are significant and will be discussed in detail.

Historically, scientists have used several approaches to predict the uptake and accumulation of chemicals in fish. Two major approaches have been used to estimate the tendency of an

HRP

T00

animal to accumulate environmental contaminants: bioconcentration and bioaccumulation. Methods for estimating bioconcentration factors (BCFs) and bioaccumulation factors (BAFs) include the use of direct measurement in vivo, or the prediction of chemical behavior in a biological system based on physicochemical constants. In order to accurately predict the level of uptake of PCBs by fish it is essential that an appropriate "accumulation factor" be applied. Understanding what constitutes a suitable factor is fundamental to deriving scientifically-based water quality standards and clean-up goals.

The use of a BCF model is an inappropriate measure of accumulation of superhydrophobic compounds such as PCBs. Because PCBs are hydrophobic, they bind primarily to sediment when introduced into an aquatic system (Fox et al., 1983). The BCF model addresses only uptake of the dissolved fraction across the membranous gill surfaces and is calculated by dividing the fish. tissue concentration by the concentration dissolved in the water column (EPA, 1989c). Scientific evidence indicates that, although uptake of lower chlorinated isomers may occur through diffusion across the gill membrane, the body burden of the more highly chlorinated isomers is primarily due to ingestion of food and sediment (Eisler, 1986; Spigarelli et al., 1983; Shaw and Connell, 1982). To more accurately estimate accumulation of PCBs in fish, it is necessary to consider the levels of PCBs in the diet and in the sediment. The bioaccumulation index (BI), which is based on the fish-to-sediment ratio and is a convenient measure of the bioaccumulation of superhydrophobic chemicals

HRP 001 1639

(Cook et al., 1991), would more accurately predict fish tissue accumulation.

In general, chemical accumulation in fish tissues and other aquatic organisms is a net balance between the rate of uptake and the rate of elimination/depuration. The rate of chemical uptake is primarily a function of the exposure concentration and the bioavailability of the compound in the environment. The elimination/depuration rate is primarily a physiological parameter of the fish. The pathways whereby fish or other aquatic organisms accumulate PCBs can be described in the following model:

> sources into the aquatic system \rightarrow partitioning within the aquatic system \rightarrow uptake by an aquatic organism \rightarrow accumulation in the organism \rightarrow elimination by the organism

Fish can assimilate PCBs from three compartments of the aquatic system: through the water column, through incidental ingestion of sediments, and through ingestion of food material containing PCBs (Spigarelli et al., 1983; Shaw and Connell, 1982). The importance of each contributing component is, in part, determined by the physical and chemical properties of the PCB isomer (chlorine content).

Once absorbed by the fish, PCBs will partition to various organs or be eliminated through the feces. Due to their lipophilic nature, PCBs readily partition to those organs with the highest fat or lipid content (Niimi, 1983; Niimi and Oliver, 1983; Shaw and Connell, 1982). Therefore, organs containing a high percentage of lipid (visceral organs, cranial tissue) will

accumulate higher quantities of PCBs (Kuehl et al., 1987; Kleeman et al., 1986a, 1986b). Because the lipid content of a fish can vary with climate and seasonal water temperatures, cold-water fish generally will have a higher percentage of lipid in their tissues than warm-water fish. Seasonal temperature changes can lead to increased metabolism and reduction of lipid stores. When the fat and its associated PCBs are mobilized, PCBs will re-enter the blood stream and eventually may be eliminated through the excretory system.

Uptake and depuration of hydrophobic compounds, like PCBs and TCDD, can be described by first order kinetics equation (Cook et al., 1990; Opperhuizen et al., 1985):

$$dC_f/dt = k_1 C_w - K_2 C_f$$
⁽¹⁾

The change in the concentration of the chemical in fish over time is a function of the first order rate constant for bioaccumulation (k_1) , first order rate constant for depuration (k_2) , chemical exposure concentration (C_w) , and the fish tissue concentration (C_f) .

Equation (1) can be redefined as:

 $C_f = k_1/k_2 \times C^w \times (1 - e^{-k_1})$

The primary parameter that influences chemical

accumulation (C_f) is the initial exposure concentration (C_w). It is evident from this equation that a decrease in the amount of PCB entering an aquatic system will decrease the amount of PCBs

HRP 001 1641

available to the fish (C_w) . As a result, the balance between uptake and depuration will be shifted and fish tissue PCB levels would be expected to decrease with time.

The period of time that is necessary to detect a measurable reduction in the concentration of PCBs in fish tissues as a result of a reduction in PCB input from a suspected source to a water body is dependent upon several factors. Two of these factors, the biological half-life of PCBs in fish and the amount of PCBs stored in the various compartments of the aquatic system, are most significant.

Empirical data on the behavior of chemicals in the environment indicate that a measurable reduction in the levels of PCBs in sediments may take several months after the input to surface waters have been reduced. Over time, the sediment reservoir will be depleted through biodegradation, physicochemical exchange to the water column or through stochastic events such as spring or storm scouring of the sediments. Natural deposition of new cleaner sediments over older deposits will also reduce the bioavailability of PCBs. Bottom dwelling fish are likely to show the slowest rates of reduction in their tissues due to their relatively high exposure to sediments.

food intake (Spacie and Hamelik, 1982; Spigarelli et al., 1982; Rand and Petrocelli, 1985; Gobas et al., 1987). Consequently, there are several adjustments that must be considered before using laboratory-derived data in environmental modeling. One of the most critical factors in evaluating the bioaccumulation potential of hydrophobic compounds in fish is the lipid content of the species of concern. A correction factor should be used to adjust for the low lipid content of the fillet and the unequal partitioning of hydrophobic compounds between edible and nonedible tissues. The application of an intraspecies correction factor may be necessary if age, sex, and health data indicate differences in lipid content within the same species of laboratory-raised and naturally-occurring fish. An interspecies correction factor may be needed when extrapolating from one species to another.

In addition to choosing an inappropriate bioaccumulation model for determining the degree of uptake of sedimentary PCBs in fish, the Phase 1 Report has used a BCF value that is not applicable to the Upper Hudson River. First, the BCF of 45,000 is based entirely on studies of accumulation of PCBs in brook trout and rainbow trout. EPA has reported that brook trout and rainbow trout are not a significant species in the Upper Hudson. A BCF derived from a dominant species would be more appropriate.

In addition, BCF values were not normalized in terms of the percent lipid. Organisms with higher lipid content have a greater potential to accumulate hydrophobic compounds.

HRP 001 1643

The lipid content of a rainbow trout is estimated to be 13.4 percent (Pennington and Church, 1979) compared to the lipid content of dominant resident fish species which range from 0.9 to 4.2 percent lipid (Pennington and Church, 1979). An average lipid content for the Hudson River fish species (brown bullhead, goldfish and largemouth bass) of 3.1 percent can be calculated based on fish sampling results reported by Sloan et al. (1985). A more appropriate BCF would thus be lipid normalized to 3.1 percent to accurately represent the dominant fish species present in the Upper Hudson.

A summary of BCF studies is presented by Eisler (1986). The BCFs reported for freshwater and marine organisms ranged from 60 to 340,000. However, the highest BCFs were reported for marine invertebrates (51,000 to 340,000). BCFs for marine fish are significantly lower, ranging from 21,800 to 27,800. BCFs for freshwater fish are even lower ranging from 164 to 1,862 (Eisler, 1986). The BCF values reported for freshwater fish by EPA (1980) represent a wider range (5,500 to 120,000). As previously mentioned, the BCF of 45,000 used by the Phase 1 Report is based on the geometric mean of three bioaccumulation studies. The results of the first study (Bills and Marking, 1977) were used to calculate a BCF of 46,000. However, Bills and Marking (1977) do not provide enough information to evaluate the validity of the results. For example, the methodology used in establishing the PCB water concentrations and the protocol used to analyze the fish tissues were not provided, rather, only a brief summary of the results were provided. Until the details of this study are

185

clear the results should not be included in deriving BCF for regulatory or guideline purposes.

The second study (Mauk et al., 1978) reported a BCF range of 40,000 to 47,000 for juvenile brook trout. In this study, brook trout eggs were exposed to PCBs 10 days prior to hatching and the resulting fry were exposed for an additional 118 days. The PCBs levels observed in the juvenile brook trout were a result of PCBs transferred from the egg sack as well as accumulated from the water column. Due to the high lipid content associated with the embryo yolk sack, it is likely that PCBs will concentrate in the egg and the resulting fry. The juvenile brook' trout will have an initial PCB body burden not associated with PCBs accumulated from the water column. This is an inappropriate study to evaluate a fish BCF, which by definition, is the ratio of PCBs in the fish to the PCB concentration in the water column. These results should also be excluded in deriving a BCF for the Upper Hudson River.

The final study used to derive the Phase 1 Report BCF for PCBs was conducted by Snarski and Puglisi (1976). Brook trout were exposed to Aroclor 1254 concentrations of 0.01, 0.03, 0.08, 0.24, and 0.94 μ g/l for up to 71 weeks. Equilibrium was reported to have been reached following 14 weeks of exposure. Although a BCF range of 10,000 to 42,000 was reported by the authors, if all of the equilibrium sample results (n=36) are evaluated, the range of BCFs are from 8,333 to 60,000 with a mean of 20,104. These values are based on a reported lipid content range of 1.3 percent to 12.3 percent and a mean of 6.1 percent. HRP 001 1645

Normalizing the BCF values to 3.1 percent lipid, which is a representative value for fish in the Upper Hudson River, results in mean BCF of 10,600. This value is approximately four times less than the BCF of 45,000 used by EPA in their derivation of an ambient water quality criteria. Even this value most likely overestimates the degree of accumulation of PCBs in fish in the Upper Hudson River.

One useful approach for developing an appropriate "accumulation factor" for PCBs and other hydrophobic compounds is the Bioavailability Index (BI). Coined by Kuehl et al. (1987a, 1987b) and further applied by Goeden and Smith (1989), the BI is defined as the ratio of the concentration of the contaminant in the lipid portion of the fish to the concentration in the organic carbon portion of the sediment (Kuehl et al., 1987a, 1987b; Goeden and Smith, 1989). The use of the BI is more suitable for hydrophobic chemicals like PCBs where the uptake of the dissolved fraction of the chemical is insignificant. In addition, a BI can be derived specifically for each congener, Aroclor or co-planar PCBs. This would allow the accumulation factor to accurately model the fish tissue accumulation of each congener of concern. However, the implementation of the BI approach to derive a water quality standard or guideline will require the development and use of a model to calculate the fate of solids on a site-specific basis (Rifkin and LaKind, 1991). This requirement may prove to be impractical at the present time and, thereby, encouraging the development of alternative accumulation factors.

HRP 001 1646

Finally, the application of a single BCF to estimate PCB accumulation in fish assumes that all PCB isomers accumulate at the same rate. However, the degree of chlorination and the molecular positions of chlorination both affect the rates of \vartheta uptake and depuration. The biological half-life, based on wholebody tissue analyses, for specific PCB isomers range from as low as 5 days for 3,3'-dichlorobiphenyl to 196 days for 2,5,4'trichlorobiphenyl, 890 days for 2,5,3',5'-tetrachlorobiphenyl, and over 1,000 days for many penta-, hexa-, octa-, and decachlorobiphenyls (Niimi and Oliver, 1983). Results from a study conducted by Lech and Peterson (1983) revealed that the higher chlorinated PCBs bioaccumulate to a greater extent than the less chlorinated PCBs. In general, mono-, di-, and trichlorobiphenyl congeners can be metabolized by fish more efficiently than higher chlorinated congeners (Lech and Peterson, 1983). Separate BCFs for the less chlorinated PCB congeners (mon-, di-, and trichlorobiphenyl) and one for the higher chlorinated congeners (penta-, hexa-, octa-, and decachlorobiphenyls) should be developed to accurately estimate the accumulation of total PCBs in fish tissue. Clearly, the application of a single BCF of 45,000 for PCBs is not appropriate for fish on the Upper Hudson River.

The appropriate accumulation factor is required to develop an ecological guideline for PCBs in order to predict the levels of PCBs a fish will accumulate from their surroundings. The BCF approach (water concentration to fish concentration ratio) is not appropriate for hydrophobic compounds like PCBs. A

4RP 001 1647

large portion of PCBs introduced into an aquatic system will bind to the sediments. The application of a BI approach (sediment concentration to fish concentration ratio) takes into account sediment sources of PCBs and more accurately predicts fish levels.

The Phase 1 Report ecological risk assessment is flawed in every way. Not only does it fail to proceed in a holistic way evaluating ecosystem biological integrity, but it also misuses existing data on the effects of PCBs in individual biological compartments. Errors exist in the selection of indicator species, in the development of realistic exposure pathways, in the qualification of exposures and in the assessment of PCB toxicity.

3.3.7 List of References

Andre, R.F., and J.R. Carroll. 1988. The atlas of breeding birds of New York State, Cornell University Press, Ithica, N.Y.

Aulerich, R.J., R.K. Ringer, H.L. Seagren and W.G. Youatt. 1971. Effects of feeding coho salmon and other Great Lakes fish on mink reproduction. <u>Can. J. Zool. 49:611-616</u>.

Aulerich, R.J., R.K. Ringer and S. Iwamoto. 1973. Reproductive failure and mortality in mink fed on Great Lakes fish. J. Reprod. Fertil. Suppl. 19:365-376.

Aulerich, R.J. and R.K. Ringer. 1977. Current status of PCB toxicity to mink, and effect on their reproduction. Arch. Environ. Contam. Toxicol. 6:279-292.

Aulerich, R.J., S.J. Bursian, W.J. Breslin, B.A. Olson and R.K. Ringer. 1985. Toxicological manifestations of 2,4,5,-2',4'5',-,2,3,6,2',3',6',-, and 3,4,5,3',4',5',- hexachlorobiphenyl and aroclor 1254 in mink. J. Toxicol. Environ. Health 15:63-79.

Bache, C.A., J.W. Serum, W.D. Youngs and D.J. Lisk. 1972. Polychlorinated biphenyl residues: Accumulation in Cayuga Lake lake trout with age. <u>Science 177</u>:1991-1192. (cited in Jensen, 1984)

Bellward, G.D., R.J. Norstrom, P.E. Whitehead, J.E. Elliott, S.M. Bandiera, C. Dworschak, T. Chang, S. Forbes and B. Cadario. 1990. Correlation of polychlorinated dibenzodioxin levels with hepatic mixed function oxidase induction in Great Blue Herons. <u>Chemosphere 20</u>:1087-1090.

Bleavins, M.R., R.J. Aulerich and R.K. Ringer. 1980. Polychlorinated biphenyls (Aroclors 1016 and 1242): Effects on survival and reproduction in mink and ferrets. <u>Arch. Environ.</u> <u>Contam. Toxicol. 9</u>:627-635.

Borlakoglu, J.T., J.P.G. Wilkins, C.H. Walker and R.R. Dils. 1990. Polychlorinated biphenyls in extracts of brain from Manx Shearwaters. <u>Bull. Environ. Contam. Toxicol. 45</u>:819-823.

Branson, D.R., I.T. Takahashi, W.M. Parker and G.E. Blau. 1985. Bioconcentration kinetics of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rainbow trout. <u>Environ. Toxicol Chem. 4</u>(6):779-788.

Braune, B.M. and R.J. Norstrom. 1989. Dynamics of organochlorine compounds in Herring Gulls: III. Tissue distribution and bioaccumulation in Lake Ontario gulls. Environ. <u>Toxicol. Chem. 8</u>:957-968.

Britton, W.M. and T.M. Huston. 1973. Influence of polychlorinated biphenyls in the laying hen. <u>Poultry Sci.</u> 52:1620-1624.

Brown, M.P., M.B. Werner, R.J. Sloan and K.W. Simpson. 1985. Polychlorinated biphenyls in the Hudson River, recent trends in the distribution of PCBs in water, sediment and fish. <u>Environ.</u> <u>Sci. Tech. 19</u>(8):656-661.

Brunström B. and L. Reutergardh. 1986. Differences in sensitivity of some avian species to the embryotoxicity of a PCB, 3,3'4,4'-tetrachlorobiphenyl, injected into the eggs. <u>Environ.</u> <u>Pollut. (Series A) 42</u>:37-45.

Brunström, B. and J. Lund. 1988. Differences between chick and turkey embryos in sensitivity to 3,3',4,4'-tetrachlorobiphenyl and in concentration/affinity of the hepatic receptor for 2,3,7,8-tetrachlorodibenzo-p-dioxin. <u>Comp. Biochem. Physiol.</u> <u>67</u>:52-57.

Brunström, B. 1988. Sensitivity of embryos from duck, goose, herring gull, and various chicken breeds to 3,3',4,4'tetrachlorobiphenyl. <u>Poultry Science 67</u>:52-57.

Brunström, B. 1989. Toxicity of coplanar polychlorinated biphenyls in avian embryos. <u>Chemosphere 19</u>:765-768.

Brunström, B. D. Broman and C. Naf. 1990. Embryotoxicity of polycyclic aromatic hydrocarbons (PAHs) in three domestic avian species, and of PAHs and coplanar polychlorinated biphenyls (PCBs) in the Common Eider. <u>Environ. Pollut. 67</u>:133-143.

Buffington, B. 1991. New York State threatened and endangered species list, provided in letter from Burrell Buffington, dated January 18, 1991, of the Wildlife Resources Center, Significant Habitat Unit, NYSDEC, Albany, NY. (cited in EPA, 1991)

Cook, P.M., A.R. Batterman, B.C. Butterworth, K.B. Lodge and S.W. Kohlbry. 1990. <u>Laboratory Study of TCDD Bioaccumulation by Lake</u> <u>Trout from Ontario Sediments, Food Chain and Water: Chapter 6</u>. U.S. Environmental Protection Agency, Environmental Research Laboratory, Duluth, MN and Natural Resources Research Institute, University of Minnesota-Duluth, Dulth, MN.

Cook, P.M., M.K. Walter, D.W. Kuehl and R.E. Peterson. 1991. <u>Bioaccumulation and Toxicity of 2,3,7,8-Tetrachlorodibenzo-p-</u> <u>dioxin and Related Compounds in Aquatic Ecosystems</u>. U.S. EPA, Environmental Research Laboratory, Duluth, MN and School of Pharmacy and Environmental Toxicology Center, University of Wisconsin, Madison, WI.

Curley, A., V.W. Burse, M.E. Grim, R.W. Jennings and R.E. Linder. 1971. Polychlorinated biphenyls: Distribution and storage in body fluids and tissues of Sherman rats. <u>Environ. Res. 4</u>:481. (cited in Aulerich and Ringer, 1977)

Eisler, R. 1986. <u>Dioxin hazards to fish, wildlife, and</u> <u>invertebrates: A synoptic review</u>. U.S. Department of the Interior Fish and Wildlife Service, Laurel, MD. Biological Report 85 (1.8). 37 pp.

Eisler, R. 1986. <u>Polychlorinated biphenyl hazards to fish.</u> <u>wildlife, and invertebrates: A synoptic review</u>. U.S. Fish and Wildlife Service, Laurel, M.D. Biological Report 85 (1.7). April.

Elliot, J.E., R.W. Butler, R.J. Norstrom and P.E. Whitehead. 1988. Levels of polychlorinated dibenzodioxins and polychlorinated dibenzofurans in eggs of Great Blue Herons (<u>Ardea</u> <u>herodias</u>) in British Columbia, 1983-87: Possible impacts on reproductive success. Canadian Wildlife Service Progress Notes

Elliot, J.E., R.W. Butler, R.J. Norstrom and P.E. Whitehead. 1989. Environmental contaminants and reproductive success of Great Blue Herons <u>Ardea herodias</u> in British Columbia, 1986-87. <u>Environ. Pollut. 59</u>:91-114. Foley, R.E., S.J. Jackling, R.J. Sloan and M.K. Brown. 1988. Organochlorine and mercury residues in wild mink and otter: Comparison with fish. <u>Environ. Toxicol. Chem. 7</u>:363-374.

Fox, M.E., J.H. Carey and B.G. Oliver. 1983. Compartmental distribution of organochlorine contaminants in the Niagara River and the western basis of Lake Ontario. <u>J. Great Lakes Res.</u> <u>9</u>:287-294.

Gilman, A.P., D.J. Hallett, G.A. Fox, L.J. Allan, W.J. Learning and D.B. Peakall. 1978. Effects of injected organochlorines on naturally incubated Herring Gull eggs. <u>J. Wildl. Manage. 42</u>:484-493.

Gobas, F.A.P.C., W.Y. Shiu and D. Mackay. 1987. <u>Factors</u> <u>Determining Partitioning of Hydrophobic Organic Chemicals in</u> <u>Aquatic Organisms</u>. D. Reidel Publishing Company. New York, NY.

Gobas, F.A.P.C., D.C.G. Muir and D. Mackay. 1988. Dynamics of dietary bioaccumulation and faecal elimination of hydrophobic organic chemicals in fish. <u>Chemosphere 17(5):943-962</u>.

Gobas, F.A.P.C., K.E. Clark, W.Y. Shiu and D. Mackay. 1989. Bioconcentration of polybrominated benzenes and biphenyls and related superhydrophobic chemicals in fish: Role of bioavailability and elimination into the feces. <u>Environ.</u> <u>Toxicol. Chem. 8</u>:231-245.

Goeden, H.M. and A.H. Smith. 1989. Estimation of human exposure from fish contaminated with dioxins and furans emitted by a resource-recovery facility. <u>Risk Analysis 9</u>(3):377-383.

Goldstein, J.A., P. Hickman, V.W. Burse and H. Bergman. 1985. A comparative study of two polychlorinated biphenyl mixtures (Aroclor 1242 and 1016) containing 42 percent chlorine on induction of hepatic porphyria and drug metabolizing enzymes. <u>Toxicol. App. Pharm. 32</u>:461. (cited in Aulerich and Ringer, 1977).

Green, D. 1985. <u>Initial Report: Hudson River Sampling</u>. (Internal Report to DEC, June 10-12, 1985)

Heinz, G.L., T.C. Erdman, S.D. Haseltine and C. Stafford. 1985. Contaminant levels in colonial waterbirds from Green Bay and Lake Michigan, 1975-80. <u>Environ. Monit. Assess. 5</u>:223-236.

Henney, C.J., L.J. Blus, S.V. Gregory and C.J. Stafford. 1981. PCBs and organochlorine pesticides in wild mink and otters from Oregon In: <u>Worldwide Furbearer Conf. Proc.</u>, J.A. Chapman and D. Pursely (eds.) Frostburg, MD. pp. 1763-1780.

Hoffman, R.D. 1978. The diets of herons and egrets in southwestern Lake Erie. Natl. Aud. Soc. Res. Rep. 7:365-369.

Hogan, J.W. and J.L. Brauhn. 1975. Abnormal rainbow trout fry from eggs containing high residues of a PCB (Aroclor 1242). <u>Prog. Fish Cult. 37</u>(4):230.

Hornshaw, T.C., R.J. Aulerich and H.E. Johnson. 1983. Feeding Great Lakes fish to mink: Effects on mink and accumulation and elimination of PCBs by mink. <u>J. Toxicol. Environ. Health 11</u>:933-946.

Institute for Evaluating Health Risks (IEHR). 1991. Reassessment of Liver Findings in Five PCB Studies in Rats. Washington, D.C.

Jensen, A.L. 1984. PCB uptake and transfer to humans by lake trout. <u>Environ. Poll. 34</u>:73-82.

Jones P.A., R.J. Sloan and M.P. Brown. 1989. PCB congeners to monitor with caged juvenile fish in the upper Hudson River. Environ. Toxicol. Che. 8:793-803.

Keenan, R.E., A.H. Parsons, E.S. Ebert, R.J. Wenning and D.J. Paustenbach. 1990. Setting rational health-based water quality standards for dioxin risk assessment for the Columbia River. Proceedings from 1990 TAPPI Conference.

Kenaga, E.E. 1980a. Correlation of bioconcentration factors of chemicals in aquatic and terrestrial organisms with their physical and chemical properties. <u>Environ. Sci. Tech. 14</u>(5):553-556.

Kenaga, E.E. and C.A.I. Goring. 1980b. Relationship between water solubility, soil sorption, octanol-water partitioning, and concentration of chemicals in biota. In: <u>Aquatic Toxicology</u>. J.G. Eaton, P.R. Parrish and A.C. Hendricks (eds.) American Society for Testing and Materials. pp. 78-115.

Kenaga, E.E. and L.A. Norris. 1983. Environmental toxicity of TCDD. In: <u>Human and Environmental Risks of Chlorinated Dioxins</u> <u>and Related Compounds</u>. R.E. Tucker, A.L. Young and G.P. Grey (eds.) Plenum Publishing Company, New York. pp. 277-300.

Kleeman, J.M., J.R. Olson, S.M. Chen and R.E. Peterson. 1986a. 2,3,7,8-Tetrachlorodibenzo-p-dioxin metabolism and disposition in yellow perch. <u>Toxicol. Appl. Pharm. 83</u>:402-411.

Kleeman, J.M., J.R. Olson, S.M. Chen and R.E. Peterson. 1986b. Metabolism and disposition of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rainbow trout. <u>Toxicol. Appl. Pharm. 83</u>:391-401.

Kubiak, T.J., H.J. Harris, L.M. Smith, T.R. Schwartz, D.L. Stalling, J.A. Trick, L. Sileo, D.E. Docherty and T.C. Erdman. 1989. Microcontaminants and reproductive impairment of the

Forster's Tern on Green Bay, Lake Michigan - 1983. <u>Arch.</u> Environ. Contam. Toxicol. 18:706-727.

Kuehl, D.W. and B.C. Butterworth. 1987a. Environmental contamination by polychlorinated dibenzo-p-dioxins and dibenzofurans associated with pulp and paper mill discharge. Biomed. Environ. Mass Spec. 14:443-447.

Kuehl, D.W., P.M. Cook, A.R. Batterman, D. Lothenback and B.C. Butterworth. 1987b. Bioavailability of polychlorinated dibenzop-dioxins and dibenzofurans from contaminated Wisconsin River sediment to carp. <u>Chemosphere 16(4):667-678.</u>

LaKind, J. and E. Rifkin. 1990. Current method for setting dioxin limits in water requires reexamination. <u>Environ. Sci.</u> <u>Tech. 24(7):963-965</u>.

Lech, J.L. and R.E. Peterson. 1983. Biotransformation and persistence of polychlorinated biphenyls (PCBs) in fish, Ch. 14 In: <u>PCBs: Human and Environmental Hazards</u>. F.M. D'Itri and M.A. Kamrin (eds.) Butterworth Publishers, Ann Arbor Science, Ann' Arbor, MI. pp. 187-194.

Linscombe, G.N. Kinler and R.J. Aulerich. 1982. Mink.:In <u>Wild</u> <u>Mammals in North America</u>. J.A. Chapman and G.A. Feldhammer (eds.) Johns Hopkins University Press, Baltimore, MD. pp. 629-643.

Makarewicz, J.C. 1983. <u>Chaplain Canal Fisheries Survey, New York</u> <u>State Barge Canal</u>. Data Report to Malcolm Pirnie, Inc. 242 pp. (cited in EPA, 1991)

McConnell, E.E. 1985. Comparative toxicity of PCBs and related compounds in various species of animals. <u>Environ. Health Persp.</u> <u>60</u>:29-33.

McKim, J., P. Schmieder and G. Veith. 1985. Absorption dynamics of organic chemical transport across trout gills as related to octanol-water partition coefficient. <u>Toxicol. Appl. Pharmacol</u>. 77:1-10.

McLane, A.R. and D.L. Hughes. 1980. Reproductive success of Screech Owls fed Aroclor R 1248. <u>Environ. Contam. Toxicol</u>. 9:661-665.

Mower, B. 1987. Maine Bioaccumulation Monitoring Program. Maine Department of Environmental Protection, Augusta, ME.

Newell, A.J., D.W. Johnson and L.K. Allen. 1987. <u>Niagara River</u> <u>Biota contamination Project: Fish Flesh Criteria for Piscivorous</u> <u>Wildlife</u>. DEC Technical Report 87-3, Bureau of Environmental Protection, Division of Fish and Wildlife. Albany, NY 155 pp.

Niimi, A.J. 1983. Biological and toxicological effects of environmental contaminants in fish and their eggs. Can J. Fish.

Aquat. Sci. 40:306-312.

Niimi, A.J. and B.G. Oliver. 1983. Biological half-lives of polychlorinated biphenyl (PCB) congeners in whole fish muscle of rainbow trout (<u>Salmo gairdneri</u>). <u>Can. J. Fish. Aquat. Sci.</u> 40:1388-1394.

Niimi, A.J. and B. G. Oliver. 1989. Distribution of polychlorinated biphenyl congeners and other halocarbons in whole fish and muscle among Lake Ontario salmonids. Environ. Sci. Tech. 23:83-88

Nikolaidis, E., B. Brunström and L. Dencker. 1988 Effects of TCDD and its congeners 3,3'4,4'-tetrachoroazoxybenzene and 3,3'4,4'-tetrachlorobiphenyl on lymphoid development in the thymus of avian embryos. <u>Pharmacol. and Toxicol</u>. 63:333-336.

Nikolaidis, E., B. Brunström and L. Dencker. 1989. Effects of TCDD and its congeners 3,3',4,4'-tetrachloroazoxybenzene and 3,3'4,4'-tetrachlorobiphenyl on lymphoid development in the bursa of Fabricius and thymus of the avian embryo. <u>Chemosphere</u> 19:817-822.

Norstrom, R.J., B.M. Braune, C.R. MacDonald, M. Simon and D.V. Weseloh. 1989. Levels and trends of PCDDs and PCDFs in Great Lakes Herring Gull eggs, 1981-1988. Poster SOU27, Dioxin 89, Toronto, Ontario, Sept. 17-33, 1989. 7 pp.

O'Shea, T.J., T.E. Kaiser, G.R. Askins and J.A. Chapman. 1981. Polychlorinated biphenyls in a wild mink population In: <u>Worldwide Furbearer Conf. Proc</u>. J.A. Chapman and D. Pursely (eds.) Frostburg, MD. p. 1746. (cited in Wren, 1991)

Pennington, J.A.T. and H.N. Church. 1980 <u>Bowes and Church's Food</u> <u>Values of Portions Commonly Used</u>. Thirteenth Ed. J.B. Lippincott Company. Philadelphia, PA.

Platonow, N.S. and L.H. Karstad. 1973. Dietary effect of polychlorinated biphenyls on mink. <u>Can. J. Comp. Med</u>. 37:391-400.

Rand, G.M. and S.R. Petrocelli. 1985. <u>Fundamental of Aquatic</u> <u>Toxicology</u>. Hemisphere Publishing Corporation. Washington, D.C.

Rifkin, E. and J. LaKind. 1991. Dioxin bioaccumulation: Key to a sound risk assessment methodology. <u>J. Toxicol. Environ. Health</u> 33:103-112.

HRP

Ringer, R.K. 1983. Toxicology of PCBs in mink and ferrets. In: <u>PCBs: Human and Environmental Hazards</u>. Chapter 17 F. D'Itri and M. Kamrin (eds.) Butterworth Publishing Company. Woburn, MA. pp. 227-241.

Shaw, G.R. and D.W. Connell. 1980. Relationships between steric factors and bioconcentration of polychlorinated biphenyls (PCBs) by the Sea Mullet (<u>Mugil cephalusl linnaeus</u>). <u>Chemosphere</u> 9:731-743.

Shaw, G.R. and D.W. Connell. 1982. Factors influencing concentrations of polychlorinated biphenyls in organisms from an estuarine ecosystem. <u>Aust. J. Mar. Fresh. Res</u>. 33:1057-1070.

Shupp, B.D. 1987. Transcript of Proceedings at the Washington County Office Building, Fort Edward, New York, State of New York Industrial Hazardous Waste Facility Siting Board and the Department of Environmental Conservation, DEC Project No. UPA 50-86-0024, June 30, 1987-July 1, 1987. (cited in EPA, 1991)

Sijm, D.T.H.M. and A. Opperhuizen. 1988. Biotransformation, bioaccumulation and lethality of 2,8-dichlorodibenzo-p-dioxin: A proposal to explain the biotic fate and toxicity of PCDD's and PCDF's. <u>Chemosphere</u> 17(1):83-99.

Sloan, R.J., K.W. Simpson, R.A. Schroeder and C.R. Barnes. 1983. Temporal trends toward stability of Hudson River PCB contamination. <u>Bull. Environ. Contam. Toxicol</u> 31:377-385.

Sloan, R., M. Brown, R. Brandt and C. Barnes. 1984. Hudson River PCB relationships between resident fish, water and sediment. <u>Northeastern Environ. Sci</u>. 3:(3/4):138-152.

Smith, L.M., T.R. Schwartz, K. Feltz and T.J. Kubiak. 1990. Determination and occurrence of AHH-active polychlorinated biphenyls, 2,3,7,8-tetrachlorodibenzo-p-dioxin and 2,3,7,8tetrachlorodibenzofuran in Lake Michigan sediment and biota. The question of their relative toxicological significance. <u>Chemosphere</u> 21:1063-1085.

Spacie, A. and J.L. Hamelink. 1982. Alternative models for describing the bioconcentration of organics in fish. <u>Environ.</u> <u>Toxicol. Chem</u>. 1:309-320.

Spigarelli, S.A., M.M. Thommes and A.L. Jensen. 1982. <u>Prediction of Chemical Accumulation by Fish</u>. Ecological Sciences Section, Radiological and Environmental Research Division, Argonne National Laboratory. PB824-156918. January.

Spigarelli, S.A., M.M. Thommes and W. Prepejchel. 1983. Thermal and metabolic factors affecting PCB uptake by adult brown trout. <u>Environ. Sci. Technol</u>. 17:88-94.

Stickel, W.H., L.F. Stickel, R.A. Dyrland and D.L. Hughes. 1984. Aroclor 1254R residues in birds: Lethal levels and loss rates. Arch. Environ. Contam. Toxicol. 13:7-13.

Tanabe, S., N. Kannan, A.N. Subramonian, S. Watanabe and R. Tatsukawa. 1987. Highly toxic coplanar PCBs: Occurrence, source, persistency and toxic implications to wildlife and humans. <u>Environ. Pollut. 47</u>:147-163.

Tanabe, S., N. Kannan, M. Ono and R. Tatsukawa. 1989. Toxic threat to marine mammals: increasing toxic potential of non-ortho and mono-ortho coplanar PCBs from land to ocean. <u>Chemosphere 18</u>:485-490.

USEPA. 1980. <u>Ambient Water Quality Criteria Document for</u> <u>Polychlorinated Biphenyls</u>. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Cincinnati, OH. EPA 440/5-80-068. May.

USEPA. 1987a. Data Quality Objectives for Remedial Response Activities: Development Process. OSWER Directive 335.0-7B. Washington, D.C. (EPA/540/G-87/003).

USEPA. 1987b. Data Quality Objectives for Remedial Response Activities: Example Scenario: RI/FS Activities at a Site with Contaminated Soils and Ground Water. Washington, D.C. (EPA/540/G-87/004).

USEPA. 1989a. Risk Assessment Guidance for Superfund. Volume II. Environmental evaluation manual. Interim final. Office of Emergency and Remedial Response, Washington, D.C. (EPA/540/1-89/001).

USEPA. 1989b. Ecological Assessment of Hazardous Waste Sites. Environmental Research Laboratory, Corvallis, OR. (EPA/600/3-89/013).

USEPA. 1989c. Interim Procedures for Estimating Risks Associated with Exposure to Mixtures of Chlorinated Dibenzo-p-Dioxins and Dibenzofurans (CDDs and CDFs) - A 1989 Update. Risk Assessment Forum. U.S. Environmental Protection Agency, Washington, D.C. EPA 625/3-89/016.

USEPA. 1989d. <u>Exposure Factors Handbook</u>. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. Washington, D.C. EPA 600/8-89/043. July.

USEPA. 1990a. Biological Criteria: National program guidance for surface waters. Office of Water Regulations and Standards, Washington, D.C. (EPA-440/5-90-004).

USEPA. 1990b. Data Useability in Risk Assessment -- Interim Final. Directive 9285.7-05. (EPA/540/G-90/008). 001 1656

HRP

USEPA. 1991. <u>Phase 1 Report - Reassessment Remedial</u> <u>Investigation and Feasibility Study: Interim Characterization</u> <u>and Evaluation</u>. Interim Report. U.S. Environmental Protection Agency, Region II, New York. August.

USEPA. 1991a. Summary Report on Issues in Ecological Risk Assessment, assembled for the Risk Assessment Forum, Washington, D.C. (EPA/625/3-9./018).

Vermeer, K. and D.B. Peakall. 1977. Toxic chemicals in Canadian fish-eating birds. <u>Mar. Pollut. Bull. 8</u>:205-210.

Weseloh, D., P. Mineau and J. Struger. 1990. Geographical distribution of contaminants and productivity measures of Herring Gulls in the Great Lakes: Lake Erie and connecting channels 1978/79. <u>Sci. Total Environ. 91</u>:141-159.

Wren, C.D., D.B. Hunter, J.F. Leatherland and P.M. Stokes. 1987. The effects of polychlorinated biphenyls and methylmercury, singly and in combination on mink. II: Reproduction and kit development. <u>Arch. Environ. Contam. Toxicol. 16</u>:449-454. (cited in Wrenn, 1991)

Wren, C.D. 1991. Cause-effect linkages between chemicals and populations of mink (<u>Mustela vison</u>) and otter (<u>Lutra canadensis</u>) in the Great Lakes Basin. <u>J. Toxicol. Environ. Health 33</u>:549-585.

4.0

REMOVAL TECHNOLOGIES

Summary: EPA's discussion of removal technologies in the Phase 1 Report is wholly inadequate. Dredging technologies have not significantly advanced since 1984, when EPA concluded that large-scale removal of sediments from the Upper Hudson would be infeasible and unreliable. In addition, the Phase 1 Report contains no discussion of the numerous impediments associated with large-scale dredging in a complex, riverine environment. In particular, EPA fails to document the significant adverse environmental effects of such a dredging project. In light of these problems, there is no basis for concluding that dredging the Hudson River is a feasible remedial action.

4.1 Introduction

In its initial screening of remedial technologies, the Phase 1 Report retains for further consideration the alternative of removal of sediment from the Upper Hudson (p. C.6-1). Yet the Phase 1 Report devotes only one and one-half pages to removal technologies (pp. C.4-7 to C.4-8) and fails to address the effectiveness and feasibility of a large-scale dredging operation at the Upper Hudson River site. Instead, the Report provides only a cursory description of three kinds of dredging technologies: the cutterhead hydraulic pipeline dredge, the clamshell mechanical dredge, and specialty dredges.

Prior to the selection of any remedial alternative that has a dredging element, EPA must consider the complexities of the Upper Hudson River and the difficult problems of removing sediments with existing dredging technology, transporting the sediments to a disposal or treatment site, and ultimately disposing or treating the material. To determine the effectiveness and feasibility of dredging contaminated sediments

1658

HRP

100

at any site, particularly a complex riverine site such as the Upper Hudson River, a detailed study of the parameters of the site is required. These parameters include sediment characteristics, bottom topography, water depth, contaminant depth, distance to the disposal/treatment facility, and necessary infrastructure for offloading and disposal of dredged materials. The Report fails to identify any data available on these important issues.

More important, the Report fails to consider the possible environmental, ecological, and human health effects that will result from dredging, despite the fact that EPA rejected dredging as a remedial alternative in 1984 because of adverse environmental impacts. In fact, the Report makes no mention of EPA's earlier concerns. EPA stated in the 1984 ROD, for example, that "bank to bank dredging could be environmentally devastating to the river ecosystem and cannot be considered to adequately protect the environment" (1984 ROD, p. 6). EPA also acknowledged the inherent problems with dredging as a remedy:

"Dredging activities by their nature tend to result in some degree of disturbance of the highly contaminated sediments, and thus result in some short-term problems, in the form of elevated PCB concentrations in the water and air, as well as increased fish contamination" (1984 ROD, p. 7).

EPA further recognized in 1984 that dredging technology could not control the many problems that must be controlled before dredging would be considered a viable remedial alternative at the Upper Hudson River site:

"Because the technology for reducing the disturbance of the sediment or controlling the spreading of the

HRP 001 1659

suspended materials is unproven in this type of a situation, it is difficult to estimate reliably the amount of the contamination which will be recovered or, on the other hand, the level of short-term damage which may result from releasing the PCB materials into the water column" (1984 ROD, p. 7).

EPA thus concluded in 1984 that the existing technology was "unproven and uncertain" in a riverine environment such as the Upper Hudson River:

"[T]he technology and methodology of [spot] dredging in a dynamic, riverine environment is unproven and uncertain. . . Therefore, it is difficult to conclude at this time that the technology can be considered feasible or reliable" (1984 ROD, p. 7).

Despite EPA's concerns about the feasibility and reliability of dredging technology in 1984 -- concerns that convinced EPA to reject dredging in the 1984 ROD -- the Phase 1 Report does not point to any new or improved dredging technology that will prevent the environmental and human health effects or ease the problems of removing and transporting material. Given EPA's rejection of dredging in 1984, the Agency's cursory discussion of dredging in the Phase 1 Report is as inexplicable as it is inadequate. EPA cannot consider and compare the true risks associated with the Hudson without considering the dramatic negative impacts that remedial dredging would cause.

This section does what EPA did not do in the Phase 1 Report: It reviews dredging technology and the problems associated with dredging contaminated soils in the Upper Hudson River. Although EPA has repeatedly stated that the Phase 1 Report is intended only as a compilation and analysis of existing data, GE believes that comments pertaining to dredging are not

premature for three reasons. First, data that relates to the effectiveness and feasibility of dredging are as much a part of site characterization as the other data collected and reviewed in the Phase 1 Report. Issues relating to dredging and dredging technology are inextricably intertwined with any assessment of the <u>feasibility</u> of dredging and should therefore <u>not</u> be deferred to the remedial design stage. Second, the Reassessment RI/FS is premised on technological advances in techniques for removing contaminated sediment (p. I-2). To the extent the Phase 1 Report has failed to show any such relevant changes, a serious question is raised as to whether continuance of the Reassessment RI/FS is warranted. Finally, absent a detailed analysis of the effectiveness and feasibility of dredging, particularly in light of the concerns expressed by EPA itself in the 1984 ROD, EPA's discussion of treatment technologies in the Phase 1 Report is illogical and premature.

4.2 Site-Specific Impediments to Dredging

The Phase 1 Report fails to address any site-specific problems that affect the feasibility of dredging as a remedial alternative at the Upper Hudson River site. Such a discussion is critical to the evaluation of dredging as a possible remedial alternative and must occur <u>prior</u> to any discussion regarding the treatment of dredged material.

The Upper Hudson River is 40 miles long and up to 2000 feet wide. It is a flowing river and is therefore different in character than most dredging sites such as estuaries and harbors. It is also a long meandering river with many large shallow areas

HRP 001 1661

where contaminated sediments may have been deposited. As discussed in detail below, dredging in shallow waters -- whether by hydraulic or mechanical equipment -- presents many logistical and technical problems. Moreover, most of the shallow water areas in the Hudson River contain submerged aquatic plantlife and would qualify as wetlands under the Clean Water Act. Dredging in this environment would therefore not only destroy this plant life, but would also violate federal law.

A meandering river such as the Hudson is likely to deposit sediments, including contaminated sediments, in shallow areas nearest the shoreline. The problems of dredging near the shoreline compound the problems of shallow dredging. For example, many of the shoreline dredging areas will be inaccessible due to overhanging trees.

The length of the river is also a significant impediment to large-scale dredging. The Upper Hudson is not a small confined area where a simple dredging operation could be planned to dredge the material and to transport it easily by pipeline to an onshore facility. Rather, the section of river at issue is 40 miles long. The problems created by this physical size and shape are complex, and any possible solution would create even more problems. For example, if an onshore facility were to be used to handle the dredged material, it is likely that HRP only some of the dredging locations would be within pipeline reach of that facility, and the rest of the locations would 100 require barge transport of the material to an offloading pier. 1662 As discussed below, barge traffic would be overwhelming, and the

infrastructure associated with barge offloading would be unsightly. Moreover, the waterfront property along the Upper Hudson is privately owned and may not be available for an onshore facility.

In addition to being a long, meandering river with large shallow areas along the shoreline and numerous wetlands, the bottom of Upper Hudson River is composed of many different types of sediments. The sediments range from soft silts to large cobbles and debris. Perhaps the largest problem is the debris, which includes very large items such as logs and tires. Large pieces of debris entrained in a hydraulic dredging system could stop operations completely and cause contaminant spills. Moreover, heavy sediments are difficult to transport by pipeline. The Upper Hudson is also characterized by variable bottom topography, particularly near the shoreline. Such a topography interferes with precision dredging, and overdredging will result. Thus, the nature and topography of the sediments in the river are critical to the feasibility of dredging technology.

The Upper Hudson River is also unique in its system of locks and dams. The locks will be the source of significant navigational problems when there is heavy barge traffic. In addition, the dams create "landlocked" areas in the Upper Hudson River that are inaccessible by boat or barge. For example, the area between Lock 6 and the Thompson Island Pool Dam is a landlocked area.

In sum, the physical characteristics of the Upper Hudson River are unlike any other environment previously studied

HRP 001 1663

or tested with respect to dredging of contaminated sediments on such a large scale. The complexity of the river's ever-changing physical characteristics results in, among other issues:

- Difficulty achieving accuracy and the associated problem of overdredging;
- Problems transporting material long distances, particularly heavy sediments;
- Problems dredging difficult materials, such as large cobbles and debris;
- Problems dredging in shallow water and near the shoreline;
- Problems concerning access to landlocked areas with dredging equipment and transport systems;
- Problems with lack of infrastructure for offloading and disposal or treatment of the dredged material;
- Problems obtaining waterfront property from local landowners; and
- Problems obtaining the necessary local, state, and federal permits (especially given the restrictions under the Clean Water Act in wetland areas).

None of these problems has yet been solved. Nor can they be solved by analogy to other dredging projects that are dissimilar to the Upper Hudson River.

4.3 Dredging Technologies

Conventional dredging is primarily used to maintain or to deepen navigational channels and typically involves handling large volumes of material (Huston, 1970; Turner, 1984). Conventional dredging technology is designed to maximize productivity; in order to accomplish this, dredging systems are designed according to the type of soils to be excavated. Rarely

001 1664

HRP

is there a desire to control turbidity or to minimize the volume of overdredged material.

Dredging contaminated sediments, however, is much different than conventional dredging operations, and many additional requirements must be satisfied in order for the dredging system to be effective. Existing dredging technology is limited in its ability to meet these additional requirements.

Specifically, a feasible dredging operation must (1) minimize resuspension of contaminated sediments into the water column; (2) minimize overdredging and maximize the precision of removing thin layers of contaminated sediments; and (3) maximize productivity to lessen the time during which sediments will be resuspended and lessen the duration of associated exposures to the contaminated sediments (Palermo, 1991). In addition, the dredging system must (1) be available in the United States; (2) be safe and protect workers from construction hazards and exposure to contaminants; (3) be maneuverable within the area being dredged; (4) be flexible to adjust to changes in water depth, sediment type, bottom topography, and disposal conditions; (5) be compatible with disposal options; (6) have the required draft necessary to operate in shallow waters; and (7) must be able to reach inaccessible areas and to transport dredged materials from those areas (Palermo, 1991).

These requirements are difficult to satisfy. No one dredging system can meet all of the above requirements, much less adequately control resuspension and minimize overdredging while maximizing productivity. Moreover, when contaminated sediments

HRP 001 1665
are a concern, the following additional factors must also be considered: characteristics of sediments; quantity of sediments to be removed; degree and concentration of contamination; location of contamination (area and depth); environmental conditions at the site (river flows, etc.); distance to the disposal site; availability of onshore facilities and offloading infrastructure; type of disposal available; and availability of particular equipment.

Perhaps the most important requirement when dredging contaminated sediments is that the sediment must be removed and transported with a minimum of sediment turbidity and associated contaminant release (Palermo, 1991).

To minimize this problem, the dredging operation must be conducted with reasonable speed to shorten the time period during which sediments will be resuspended by the operation, thereby minimizing the duration of associated exposures (Palermo, 1991). The rate at which a given dredge can be operated will depend on the type of sediment, the depth of water and of contaminated sediments, the percent solids in the dredged material, the volume of dredged material, and the need for maintenance and downtime that lessens the amount of time the dredge is operating. It is also a function of the accuracy and control of the vertical and horizontal movement of the dredgehead and overall movement of the equipment, as well as the ability to

In addition, because all contaminated dredged material must be placed in disposal sites with costly treatment and

1666

controls, or treated at large capacity treatment facilities, precision of the dredging process is critical (Palermo, 1991). Any overdredging will result in large quantities of additional unnecessary material for disposal and treatment. Thus, contaminated dredged material must be removed with the objective of leaving little contaminated material behind, while at the same time avoiding the removal of clean underlying material. Unfortunately, no existing dredge technology is capable of dredging a thin surficial layer of contaminated material without leaving behind a portion of that layer or mixing a portion of the surficial layer with underlying clean sediment (Palermo, 1991). Thus, several passes may have to be made, at different levels, to remove all the contaminants. The bottom pass will result in overdredging and the additional passes will, of course, consume time and increase turbidity.

These requirements -- minimal resuspension and maximum precision -- must be considered when evaluating the feasibility of dredging in the Upper Hudson River. It is obvious, however, that the two goals conflict with each other. High productivity and speed, which are required in order to minimize the duration of dredging (and related exposure to contaminants), will result in less accuracy and more overdredging. Precision dredging, on the other hand, which is required to minimize overdredging, will result in slower productivity and longer dredging duration.

EPA identifies three types of dredging systems in the Phase 1 Report: hydraulic, mechanical, and specialty dredges. Each of these dredging systems is designed for particular

HRP 001 1667

applications (p. C.4-7). But none of these dredging systems can reliably remove contaminated sediments from the Upper Hudson River within the constraints previously described. Moreover, each of the dredging systems identified in the Report was available in the United States before EPA issued the 1984 ROD, which rejected dredging as a feasible remedial alternative (1984 ROD, p. 9; Huston, 1970; Turner, 1984).

4.3.1 <u>Hydraulic Dredging</u>

Hydraulic dredging uses a centrifugal water pump to create a vacuum at the dredgehead. Atmospheric pressure acts to force water and sediments through a suction pipe. The dredged materials are usually hydraulically pumped through a pipeline either to the disposal or treatment site or to barges for transportation to the disposal or treatment site (Palermo, 1991). Hydraulic dredges are designed for excavating free-flowing soft material such as silt and clays. The typical hydraulic dredge essentially stirs up the material and then removes the loosened "slurry" material, along with a substantial quantity of water, via a vacuum suction system (Huston, 1970; Turner, 1984).

There are several types of hydraulic dredges. The primary types include plain suction, cutter-head suction, dustpan, sidecast, and trailing hopper dredges. The Phase 1 Report identifies only the cutterhead dredge (p. C.4-7), which is a dredge that is equipped with teeth or blades on a rotating basket. As the cutter rotates, it mechanically loosens the bottom sediment and moves it toward the flow field around the dredge suction to be drawn into the suction pipe.

Among the many limitations of a hydraulic dredging operation in the Upper Hudson River, the primary impediments to such dredging include problems arising from shallow water depths, shoreline dredging, variable soil and bottom conditions, overdredging and accuracy, long pumping distances, turbidity and resuspension, and equipment availability and import restrictions.

4.3.1.1 Shallow Water Depths

Although the exact locations of contaminated sediment are presently unknown, hydraulic dredging of such sediment will be extremely difficult, if not impossible, to the extent that the contaminants are located in shallow areas in the river. These shallow water areas are generally large and broad and range from 2 to 6 feet in depth.

Shallow water depths will require small, shallow draft equipment. The shallowest draft feasible for a barge containing reasonably sized dredging equipment is about 3 feet. In addition, depending on the dredging equipment, tugs may be required to move and to place the dredging equipment barges. Α small tug, such as an interharbor tug, has a 6 foot draft. Such tugs, however, are neither powerful nor efficient. The shallow water will also create problems locating, constructing, and maintaining pipeline routes and booster stations. Dredging of pilot channels to facilitate navigation of the dredging equipment and construction of the pipeline in shallow areas will cause substantial unnecessary overdredging. Thus, hydraulic dredging in the shallow areas would not be feasible.

HRP 001 1669

4.3.1.2 Shoreline Dredging

Many shallow water areas are also located near the shoreline, and several additional problems exist if dredging near the shoreline is required. For example, much of the shoreline along the 40 mile section of the Upper Hudson River at issue is covered with large trees that overhang the river banks by 10 feet These overhanging trees interfere with the vertical or more. clearance necessary for dredging equipment. The cutterhead system, therefore, will not be possible in these areas. And. although a small hydraulic dredge may be able to work perpendicular to the shoreline, such dredges have much slower production rates, resulting in increased duration of resuspension and associated contaminant exposure. Moreover, a small hydraulic dredge would not be feasible for shallow shoreline areas because the rigging for such an operation would require anchoring onto the shore, and shoreline anchoring is not possible along most of the shoreline because of overhanging trees.

Finally, any dredging near the shoreline would undercut the river bank resulting in accelerated erosion and damage to tree roots.

4.3.1.3 Variable Sediments

Hydraulic dredging operations are highly dependent on the nature of the soil. Most hydraulic dredges are designed to handle loose and free flowing soils such as soft silts and sands. They are not well suited to dredging difficult soils such as compact silts and clays, cemented gravels, cobbles, boulders, and debris. The cutterhead dredge can cut and handle compact soils

and cemented gravels. However, the performance and production of the cutterhead system is still highly dependent on the soil conditions, particularly when the materials must be pumped long distances. For example, pipeline head losses for soils containing gravel are much higher than for silts and fine sands and would require additional booster pumps and larger volumes of water for transport.

More important, hydraulic dredges, including the cutterhead dredge, cannot accommodate a wide range of variable materials. The sediments in the Hudson River are highly variable, including compact soils, silts, sands, gravel, cobbles, and debris, such as logs and tires. The cutterhead dredge system is not capable of cutting, entraining, and transporting large material and debris (Palermo, 1991). Even assuming that the cutterhead could handle such materials, the system must be designed to cut and to transport the hardest and heaviest materials that would be encountered. The blades and pumps cannot be changed each time a pocket of different soil is encountered. As a result, the cutterhead dredge will be too powerful, and therefore inefficient, when it encounters the softer sediments and would pump excess quantities of water and sediment into the system, thereby adding to the volume of material requiring treatment and disposal. In addition, extra booster pumps necessary for the heavier materials would be too powerful, and therefore inefficient, when softer sediments are transported via the pipeline.

HRP 001 1671

4.3.1.4 Volume of Dredged Material

A significant problem that must be considered when evaluating hydraulic dredging is the quantity of excess material that is captured in the system and that must be treated and disposed. Conventional hydraulic dredging operations result in a slurried sediment with only 10 percent solids; the other 90 percent is water (Huston, 1970; Turner, 1984). Indeed, when operational controls are in place to control resuspension (e.g., slowing the rotational and swing speed of the dredgehead), the slurried sediment will consist of only about 5 percent solids and 95 percent water. As a result, large volumes of excess water must be treated.

The excess water problem is compounded when the sediments are soft silts, clays, and organic material. As these softer sediments are transported hydraulically to the disposal facility, there is substantial "bulking" of the material. For example, when only one cubic foot of clayey silt is dredged from the bottom, that one cubic foot increases by about 50 percent to create 1.5 cubic feet of material for treatment and disposal. Thus, there will be significantly greater volume of material to be treated and disposed than the volume of sediment originally dredged from the river bottom.

4.3.1.5 Variable Bottom Conditions

Hydraulic dredges excavate most accurately on consistent bottom topography. This is because hydraulic dredges operate in a "sweeping" action along the bottom in order to dredge to an even elevation. A small cutterhead dredge, with a

001 1672

HRP

10 inch discharge line, would sweep approximately 40 feet across the bottom of the river. Therefore, if there are any variations in the area being sweeped (e.g., small hills and valleys), the dredge will be required to overdredge the hills in order to reach the valleys, causing significant overdredging. The dredging operator can skim off a uniform layer of contaminated sediments only to a limited extent. This will be a problem in the Upper Hudson River, which does not have a consistent bottom topography, particularly near the shoreline where the bottom topography changes rapidly.

4.3.1.6 Overdredging and Accuracy

Quite apart from the uneven bottom topography, use of the cutterhead dredge will result in overdredging. Given the nature of river deposition, contaminated sediments may be located at variable depths in the soils. Thus, hills and valleys of contaminants may exist in the cross-section of the sediments, and to remove them, the dredge will need to remove a significant quantity of uncontaminated sediments as well. The 40-foot sweep of the cutterhead dredge will be inaccurate when the contaminants are dispersed unevenly in small localized pockets. Under the best circumstances, with the most accurate operations, overdredging on the order of 6 to 12 inches can be expected.

In addition, overdredging will occur regardless of the greatest possible care and precision during the dredging operation. The inherent inaccuracies involved in locating contaminants which are moving in a dynamic riverine environment will result in inaccuracies in determining where to dredge. Many

HRP 001 1673

uncontaminated areas of the river will likely be unnecessarily dredged in an attempt to locate and remove the contaminated sediments. The extensive overdredging that will be necessary to remove the small, localized areas of contaminants will result in substantial quantities of unnecessary, excess dredged material requiring disposal.

4.3.1.7 Pumping Distances

A hydraulic dredging system will require either a pipeline to an onshore facility or a pipeline to a barge, which will be offloaded at a pier. In general, piping the material directly to the shore, rather than to a barge, is more efficient. Transporting contaminated material by barge is less efficient because additional transportation and handling are required, and because large quantities of water in the slurried material need to be transported. Unlike uncontaminated slurries, excess water cannot be allowed to overflow the barge; instead, the excess water must be transported for treatment and disposal. Typically, when transporting material by barge, the material has a relatively high solids content to minimize the number of required barge loads. Pumping slurried material via pipeline to a barge for subsequent transport is therefore very inefficient and will result in excessive river traffic.

The length of the pipeline will depend on where the onshore facility or barge is located and on the hydrostatic pressure provided by the pump, unless extra pumping power from booster pumps is provided. A small dredge pipeline can carry material only about half a mile without a booster pump. Even

with a booster pump located every half-mile, the maximum distance feasible would be about 5 miles. The heavier sediments in the Hudson River, however, will be more difficult to transport and may require additional booster pumps spaced closer together. Additional booster pumps, and longer pipelines, will need maintenance and repair and, as a result, will increase dredging downtime. This in turn increases the duration (and cost) of the operation and therefore should be avoided (Palermo, 1991).

Because the practical pipeline distance is limited to a maximum of 5 miles, and the section of river at issue is 40 miles long, it would be impossible to transport all dredged material by a pipeline directly to a treatment and disposal facility. Hydraulic dredging via a pipeline to shore would therefore not be feasible for a substantial part of the 40 mile stretch of river.

In addition, pipelines of any length generally must be flexible to follow the dredge and to deflect wave action without building up excessive stresses. Pipelines must also be rotated approximately once a month during operation for maintenance purposes to prevent wear and tear on their bottoms (Turner, 1984). In typical hydraulic dredging operations, pipeline joints are not rigid or leakproof because of the higher maintenance requirements and need for flexibility. Obviously, when contaminated sediments are transported via pipeline, the joints and connectors must be leakproof, requiring additional maintenance. When pipelines are maintained, the dredging must cease, and the pipelines must be flushed thoroughly with clean

HRP 001 1675

water to avoid leakage of contaminants when the pipelines are disconnected.

Finally, the location of the pipelines and the booster pump stations must account for easement requirements and navigational interferences. Land ownership along the river may create problems for access for construction and location of the pipeline and booster pump stations. In addition, floating and submerged pipelines, floating booster pump barges, and underwater crossings can cause significant navigational problems during installation, operation, and relocation. Likewise, increased barge traffic resulting from transporting slurried material pumped from the hydraulic dredges to barges will tie up the locks in the river and seriously disrupt the navigation of commercial and recreational boats in the river. If hydraulically dredged material is transported by barge, the barge traffic will increase ten-fold because of the greater volume of material, most of which is water.

4.3.1.8 Turbidity and Resuspension

Turbidity is generated by the dredging operation itself when the cutterhead cuts the material to be entrained in the suction system. With hydraulic dredging, excavation-related turbidity can be reduced to some extent by operational controls, but it cannot be eliminated.

In addition, secondary sources, such as tugs, anchorage, and work boats, are a major source of resuspension and turbidity. For example, when the dredge system is placed in a particular location for operation or moved to another dredging

location, the dredge itself will create some turbidity, the swing anchors and spuds being set and removed to hold the dredge in place will create turbidity, and the tug moving the dredge will cause a significant amount of resuspension and turbidity due to the propeller wash (Palermo, 1991). Maneuvering in any part of the river will generate resuspension. Turbidity will also result from leaks in the pipeline and pipeline joints, or when the pipeline is removed for maintenance or relocated.

Finally, if barge transport is used instead of or in addition to pipelines, the barges themselves will create resuspension and turbidity; the tugs moving the barges will create turbidity from the propeller wash; any spillage from the barges during handling, transport, and offloading will create turbidity; and the construction of piers and facilities for offloading will cause some turbidity. Thus, even if the cutterhead dredge is better than other systems at reducing turbidity at the dredgehead by controlling the dredging operation, it cannot control turbidity from secondary sources.

4.3.1.9 Equipment Availability

Conventional hydraulic dredging equipment, including the cutterhead, is generally available in the United States. The small cutterhead, however, may be located far from the Upper Hudson River site (Palermo, 1991). Also, special modified equipment may not be available in the United States due to Jones Act restrictions. It is imperative to consider availability and distance from the Upper Hudson River before deciding that hydraulic dredging is a feasible remedial alternative.

4.3.1.10 Not New Technology

Hydraulic dredging is not new technology. The cutterhead system, which EPA mentioned in its Phase 1 Report, has been available for many years and well before the 1984 ROD (Huston, 1970; Turner, 1984). No new improvements or modifications to the cutterhead system or any other hydraulic dredging system have been developed to control the problems associated with dredging contaminated sediments in a complex and dynamic riverine environment like the Upper Hudson River. Moreover, no existing hydraulic dredge, including the cutterhead, has yet been proven in a dynamic riverine environment when large volumes of dispersed contaminated sediments are involved.

4.3.1.11 Conclusion

Based on any single factor and on the cumulative effect of all of the problems enumerated above, hydraulic dredging would be neither feasible nor effective for the Upper Hudson River site.

4.3.2 <u>Mechanical Dredging</u>

Mechanical dredging is very similar to conventional land-based excavation techniques. It involves excavation of sediment using such devices as clamshell dredges, dipper dredges, draglines, grab buckets, and backhoes.

There are many kinds of mechanical dredges. The Phase 1 Report, however, discusses only the clamshell mechanical dredge $\frac{H}{Z_{P}}$ (p. C.4-7). The clamshell dredge consists of a crane, fitted with a clamshell type bucket, and mounted to a barge, which is anchored in position. The clamshell bucket is dropped to the

bottom, where, because of the impact of the bucket on the bottom, the bottom material is penetrated and then scooped into the bucket. The dredged material is typically loaded onto a barge and hauled to the disposal site (Huston, 1970; Palermo, 1991).

The Phase 1 Report observes (p. C.4-7) that "[i]n the case of the Hudson River project, the barge contents would probably be slurried for removal by a hydraulic pump-out system located on shore." But the benefits of using mechanical dredging, namely the higher solids content of the dredged material, will be lost if the material is subsequently slurried and pumped to shore. The original dredged volume of material will increase ten-fold when slurried, creating substantially more material for treatment and disposal. The only possible benefit realized would be decreased barge traffic. Even this benefit is illusory, however, because of the long distance to an onshore facility that will prevent piping from a barge at most of the dredging locations in the 40-mile stretch of river.

As discussed above, only those dredging locations within about half a mile from an onshore facility (without booster pumps) or a maximum of about 5 miles (with many booster pumps) will be within pipeline reach. The remote locations will require barge traffic to transport the dredged materials from the dredging location to the pipeline within reach of the onshore facility, or to the shore itself. Therefore, any plan to excavate sediments mechanically, place the sediments on a barge, and then pump those sediments (along with a substantial quantity

HRP 001 1679

of water) from the barge to the onshore facility is simply not logical, let alone feasible.

The Phase 1 Report also states (p. C.4-7) that "where circumstances permit, bottom dump scows can be used in concert with a mechanical system and would discharge dredged material at sub-aqueous disposal sites." This proposal, in essence, requires the removal of contaminated sediment from one place where they are buried in the river and reburial of those sediments in another place in the river. It is impossible to comment on the feasibility of this option because EPA has provided no information regarding the possible sites for sub-aqueous disposal, the requirements for disposal (i.e., lining and capping the site), or the volume of material that would be disposed in the sub-aqueous site.

Preliminarily, however, it is clear that the dumping of the material from the bottom dump scows into the sub-aqueous disposal site will create a substantial amount of turbidity from resuspension, as well as turbidity from spillage from the dumping action itself. Moreover, the difficulties of dredging operations in the Upper Hudson River and the environmental impacts of dredging, apart from the serious treatment and disposal issues associated with subaqueous disposal, will still apply whether or not EPA decides to dispose of the dredged contaminated sediments in a sub-aqueous site. Thus, these comments will focus on the HRP problems with mechanical dredging operations in the Upper Hudson 100 River, apart from the problems associated with subaqueous disposal.

Limitations to mechanical dredging operations in the Upper Hudson River make such operations infeasible regardless of the method of disposal. These limitations include: shallow water depths, shoreline dredging, accessibility, overdredging and accuracy, barge traffic, and turbidity and resuspension. If the material is transported to an onshore facility for disposal or treatment, there will be additional problems associated with barge traffic and infrastructure impacts.

4.3.2.1 Shallow Water Depths

A fundamental problem in dredging with mechanical dredging equipment such as the clamshell dredge is the draft limitation when working in the extensive shallow water areas. The problem concerns not only the floating dredging equipment, but, more importantly, the barges used for hauling the dredged material and the tugs needed to move the floating dredging equipment and the hauling barges. The available depth of water in many areas of the river where contaminants may be located is 2 to 6 feet, whereas the shallowest equipment barge would have a draft of about 3 feet and must be accompanied by a materials barge with a draft of about 8 feet, and a tug with a draft of about 6 to 12 feet (depending on the weight and size of the load being moved).

In small, confined areas, a crane boom may be attached to a crane floating in deeper water within a maximum of 100 feet from the dredging site, in order to reach into shallow water. However, the limited length of a crane boom will not be capable of reaching into all shallow areas. In addition, there will be

shallow areas near the shoreline that cannot be reached by a crane boom because of interference from overhanging trees.

4.3.2.2 Shoreline Dredging

In addition to the many problems associated with operating in shallow water, a clamshell dredging operation will not be possible near the shoreline where there are overhanging trees interfering with the vertical clearance necessary for the dredging equipment. Much of the 40 mile shoreline is covered with large trees overhanging the river banks by ten feet or more. Thus, mechanical dredging is not possible up to the shoreline. In addition, any work near the shoreline may undercut the river bank resulting in damage to the tree roots and loss of privately owned waterfront property.

4.3.2.3 Accessibility

In order to dredge mechanically, it must be possible to get to and from the dredging location with barges for loading and transporting the dredged material. If an area is "landlocked," meaning there is no means of getting a barge to the area or out of the area, then mechanical dredging is not feasible. In fact, there is a two mile landlocked section of the Upper Hudson River between Lock 6 and the Thompson Island Dam. If an onshore disposal area is located more than about 5 miles from the landlocked dredging site, then the dredged material cannot be pumped via pipeline to the onshore disposal site; it will have to ^P transported from the landlocked dredging location via pipeline to

223

a barge in a non-landlocked area for its final transportation to the onshore facility.

4.3.2.4 Overdredging and Accuracy

Accuracy is difficult, if not impossible to achieve, with the clamshell dredge. Conventional clamshell dredging involves either excavating deep holes at selected locations and letting the side slopes slough in to level out the bottom or excavating a cratered surface and leveling the bottom surface between the craters. Neither method would be appropriate in the Upper Hudson River.

A small size bucket may be necessary in order to make a' shallow cut in the surface layer of the contaminated sediment. Even with a shallow bucket, however, several passes may be necessary to dredge to an even design depth (Palermo, 1991). Areas with a hard bottom will require a heavier, larger bucket, and overdredging in such areas will be more difficult to control.

Uneven bottom topography and inconsistent contaminated layer thickness will increase the difficulty in an attempted "precision" cut. Unfortunately, no matter how careful and precise, significant overdredging will result in order to attain a design dredge depth. Thus, overdredging is inherent in mechanical dredging operations because of the limited ability to make precise cuts.

The inherent difficulty involved in locating contaminants that have been constantly moving in a dynamic river environment will result in additional inaccuracies in determining where to dredge. Locating the contaminants with any precision

HRP 001 1683

will be impossible because of the highly variable and localized sediment and bottom conditions in the vast section of the river at issue. The exact location of the contaminated sediments are unknown. Many "clean" areas of the river will therefore be unnecessarily dredged in an attempt to locate and remove the small amount of contaminated sediments that may remain in the vast 40 mile section of the river. The extensive overdredging that will be necessary to remove the small localized areas of contaminants will result in substantial quantities of unnecessary, excess dredged material requiring disposal.

4.3.2.5 Barge Traffic and Infrastructure Needs In general, mechanically dredged material is loaded onto barges and hauled by tug to a disposal site. Assuming the material is transported to an onshore disposal facility, waterfront structures and material handling facilities will be necessary for offloading of the barges. These structures and facilities do not yet exist anywhere on the 40 mile stretch of the Upper Hudson River. To handle the quantity of material and the associated barge traffic, a two-berth pier would be necessary, plus additional waiting berths.

A threshold problem with constructing a pier is the availability of waterfront property and riparian rights in the vicinity of the chosen disposal site. Unless a long stretch of waterfront property can be acquired, construction of the required $\frac{14}{50}$ offloading facilities will not be feasible. In addition, the construction and presence of such facilities would have significant environmental impacts. For example, construction of $\frac{16}{50}$

a large pier facility will require additional dredging for the berths and for a turning basin at the pier.

Even apart from the problems associated with the pier and offloading facility, the barge traffic associated with mechanical dredging will also cause significant problems. In addition to the increased turbidity resulting from the barge draft and tug boat propeller wash, a substantial increase in marine traffic on the river will result from the barges and tugs. The locks in the Upper Hudson River are only large enough to hold one barge plus one tug. Multiple barges could occupy the locks for long periods and preclude commercial and recreational boats from passing. If the barge traffic continues for several years, as it most likely would in conjunction with the dredging program, the adverse public impact will be significant.

4.3.2.6 Turbidity and Resuspension

Turbidity will result from sediment resuspension caused as the bucket strikes, bites, and is pulled off the bottom, and as water and sediment spills from the bucket when it is pulled up through the water and loaded into the barge. Turbidity caused by the dredging operation itself can be reduced to some extent with a water-tight bucket (p. C.4-8) and with carefully controlled operation of the equipment. A water-tight bucket has jaws that seal when the bucket is closed; the top is also covered so that the dredged material cannot escape once the bucket is closed (Palermo, 1991). Although the use of such a bucket reduces turbidity, the water volume of the dredged material increases as a result of the water trapped in the bucket. This extra water

226

increases the volume of material that must be transported, treated, and disposed.

The most significant source of resuspension is the impact of the bucket hitting the bottom. This source cannot be eliminated because of the nature of the digging operation, i.e., breaking the bottom is necessary in order to entrain the material in the bucket. The turbidity associated with a clamshell dredging operation is unavoidable.

In addition, each step of the clamshell dredging operation -- including initial dredging, placement of the material into the barge, transport of the material, and final offloading of the material for disposal -- is subject to spillage, which allows contaminated sediment to return to the water. Although spillage can be controlled to some extent with careful operation and overflow prevention equipment, it cannot be eliminated completely. In addition, considerable uncontrollable sediment resuspension is caused by barges used to transport the sediment and propeller wash from tugs that move the barges (Palermo, 1991).

4.3.2.7 Not New Technology

Mechanical clamshell dredging is not new technology. Clamshell dredging, which EPA mentioned in its Phase 1 Report, has been available for many years and well before the 1984 ROD (Huston, 1970; Turner, 1984). There are no significant

227.

in the Phase 1 Report, *i.e.* overflow prevention and the use of an enclosed bucket, were available before 1984. In fact, the watertight clamshell dredge was evaluated by the U.S. Army Corps of Engineer Waterways Experiment Station in 1982.

4.3.2.8 Conclusion

Given any one of the above problems, and certainly considering the totality of circumstances associated with clamshell dredging, such dredging would not be feasible at the Upper Hudson River.

4.3.3 Specialty Dredging

The Phase 1 Report mentions (p. C.4-7), but does not discuss in detail, a third category of dredging systems, namely, specialty dredging. Specialty dredges include pneumatic dredges, and modified hydraulic dredges (Palermo, 1991).

The Phase 1 Report notes (p. C.4-8), however, that specialty dredges would not be appropriate for the Upper Hudson River because "no reduction in sediment resuspension was found" during field tests conducted by the U.S. Army Corps of Engineers. Thus, as the Phase 1 Report itself recognizes, none of the existing specialty dredges would be viable for a dredging operation at the Upper Hudson River because of their limited application and the additional problems they create. GE agrees with EPA that specialty dredges are not a viable option at the Upper Hudson River. Specialty dredging systems should therefore be eliminated from further consideration in the RI/FS.

HRP 001 1687

4.3.4 Field Studies

As discussed above, currently available dredging technology cannot feasibly control all of the problems at the Upper Hudson River site to dredge safely the contaminated sediments.

The Phase 1 Report refers to recent field studies conducted by the U.S. Army Corps of Engineers Waterways Experiment Station as evidence of the cutterhead's ability to minimize resuspension. According to the Phase 1 Report, these field studies demonstrated that the cutterhead dredge is "the most successful in limiting sediment resuspension into the water column." In that study, however, the cutterhead was being compared to other dredging systems, such as the mechanical clamshell dredge. In such a limited comparison, absent other factors restricting its use, the cutterhead dredge may be the best for limiting resuspension. By the same token, the cutterhead dredge may not be appropriate in certain other circumstances. In fact, the cutterhead dredge has not been proven to control resuspension in a dynamic riverine environment similar to that of the Hudson River. The complexities of the Upper Hudson River that were discussed in Section 4.2 simply were not and could not have been adequately simulated in the U.S. Army Corps of Engineers Waterways Experiment Station field studies.

Hudson River. In 1984, EPA asked the Army Corps of Engineers to conduct an Engineering Feasibility Study of dredging and disposal alternatives at the Acushnet River Estuary site adjacent to New Bedford, Massachusetts. The Army Corps of Engineers studied the site and, based on site specific parameters, selected several possible dredging technologies for an experimental study at a small portion of the New Bedford Harbor site (Palermo, 1991).

Prior to the pilot dredging program, the Army Corps evaluated alternative dredging techniques and concluded that three types of dredges generally appeared to be suited for the New Bedford Harbor site, at least for the small scale pilot dredging study. The Army Corps then dredged a small pilot area of the harbor with these three types of dredges. The Army Corps found that the conventional cutterhead suction dredge gave the best performance as compared to the other two types of dredges, given the particular site specific characteristics at the pilot study area of the New Bedford Harbor site.

The cutterhead dredge was not, however, problem-free. One operational problem observed with the cutterhead dredge was caused by the movement of swing cable anchors in the softer sediments. The anchor movement, plus the workboats used to set and move the anchors, caused substantial resuspension of sediment that could not be controlled. The Army Corps recommended locating the swing anchors on land to prevent these turbidity problems. This option would not be possible at the Upper Hudson River site. Onshore placement of anchors would be precluded by the overhanging trees and private ownership of waterfront

230

property, and it would not be feasible where open water dredging is required. Silt curtains were attempted, but it was found that the movement of the silt curtains was a difficult operation that in turn generated considerable turbidity.

Even apart from these acknowledged limitations of the cutterhead dredge, the New Bedford Harbor pilot study has limited relevance to the Upper Hudson River. Whereas the Upper Hudson River site consists of 40 miles of a meandering river up to 2000 feet wide, the entire New Bedford site is less than 2 miles long and is only 0.2 miles wide, and the pilot study was conducted on an even smaller, more confined area than the total site (Palermo, 1991). Moreover, the New Bedford Harbor does not have a steady and strong one-directional current like that of the Hudson River.

In addition, the New Bedford Harbor is composed of consistent soft sediments that are easily dredged with a hydraulic dredging system, unlike the Hudson River, which has variable sediments (including large debris such as logs and tires) that are a problem in hydraulic dredging operations (Palermo, 1991). Finally, the New Bedford Harbor pilot study area did not include shallow areas combined with difficult shoreline access.

In sum, the test results at New Bedford are inapplicable to the Upper Hudson River because of site differences such as bottom conditions, currents, coverage area, water depth variations, soil conditions, navigation, distances to treatment and disposal facilities, and quantity of dredged material to be treated and disposed. The fact is that

231

the ool 1691

conventional dredging remains an "uncertain and unproven" remedy "for dredging of contaminated sediment from an environment such as this one" -- the Upper Hudson River (1984 ROD, pp. 7, 9).

4.3.5 Insignificant Changes in Technology Since 1984

No significant changes in dredging technology have been developed. EPA's cursory and vague discussion of dredging technology in the Phase 1 Report (pp. C.4-7 to C.4-8) illustrates this fact. The Phase 1 Report mentions (p. C-4-7) only two conventional dredging systems: the cutterhead hydraulic dredge and the mechanical clamshell dredge. Both of these dredging systems were available prior to the 1984 ROD, which rejected dredging as a remedial alternative.

GE has conducted a comprehensive literature and data base search using the resources of the Information Research Division of Engineering Societies Library in New York and the Center for Dredging Studies at Texas A & M University, which is the world's leading center for dredging research. No major developments in dredging technology, applicable to the Upper Hudson River site were found. As discussed above, the minor modifications made to the conventional dredging systems did not address, let alone solve, the complex problems associated with dredging contaminated sediments in a dynamic riverine environment. Thus, these modifications cannot be considered significant improvements in dredging technology.

The Phase 1 Report alludes (p. C.4-7) to two modifications to the conventional clamshell mechanical dredge: overflow prevention and enclosed buckets. These modifications

HRP 001 1691

were also available prior to 1984. More important, as discussed above, these modifications do not solve the serious environmental problems caused by dredging contaminated sediments in a riverine environment.

In addition, the 1989 pilot study conducted in New Bedford Harbor, Mass., did not demonstrate any development of new technology. On the contrary, the EPA pilot study selected conventional dredging technology -- the cutterhead hydraulic dredge (without modification) -- a technology that was available prior to 1984.

4.3.6 <u>Conclusion</u>

Existing dredging technology is neither proven nor reliable in controlling the many problems associated with dredging contaminated sediments in a dynamic and complex riverine environment like that of the Upper Hudson River. Moreover, there have been no new developments in technology or information since EPA issued its initial decision rejecting dredging in 1984. Therefore, dredging in the Upper Hudson remains an infeasible and unproven remedial action.

4.4 Adverse Environmental Effects of Dredging

Superfund remedies must be protective of the environment (CERCLA § 121(b)(1), 42 U.S.C. § 9621(b)(1), 40 C.F.R. § 300.430(e)(9)(iii)(A)). To achieve this goal, EPA must evaluate both the benefits to the Hudson River ecosystem that can be achieved by the implementation of various remedial alternatives and the detrimental impacts to the ecosystem that

above, the existing evidence does not show ecological risks attributable to the presence of PCBs. For that reason, it is crucial to consider the detrimental impact to the ecosystem that would result from the implementation of remedial alternatives. In this regard, it is clear that dredging would have a substantial adverse effect on the Hudson River ecosystem. As EPA itself recognized in 1984, large-scale dredging of PCBcontaminated sediment from the Upper Hudson River would be "environmentally devastating" (1984 ROD, p. 6). This conclusion remains true today. This section provides an overview of dredging-related impacts on the environment. These impacts make dredging in the Upper Hudson River unreasonable and illogical.

Major environmental impacts due to dredging in the Upper Hudson will result from: (1) turbidity and resuspension generated by dredging and related activities; (2) changes in river conditions due to erosion and deposition; (3) destruction of habitats such as wetlands and benthic communities; (4) decreases in air and water quality; and (5) navigational and infrastructure interferences due to barge traffic and dredging activities. These environmental impacts are discussed in turn below.

4.4.1 Effects of Turbidity and Resuspension

Turbidity is the release of sediment into the water column due to resuspension of bottom sediments or spillage after the material has been dredged. Increased suspension of sediment particles in the water column as a result of dredging is HRP 001 1693

virtually unavoidable. Such turbidity will have several adverse effects on aquatic life.

For example, several studies have confirmed that turbidity and resuspension caused by dredging increase the concentration of PCBs in the water column and, as a result, increase the uptake of PCBs by fish and other organisms in the river (Rice and White, 1987; Tofflemire et al., 197 $\frac{1}{2}$; Hafferty et al., 1977). In addition, exposure to high concentrations of suspended solids can reduce filter-feeding activities of invertebrates. Suspended solids will also clog gills of larvae and young fish, smother eggs, and interfere with photosynthesis by submerged aquatic vegetation.

Resuspension of sediment particles also results in an increase in suspended organic content. Increased suspended organics will increase the biological oxygen demand, thereby reducing the oxygen content of water available to aquatic organisms. This decrease in dissolved oxygen can have debilitating or even lethal effects on aquatic life.

Related to turbidity is the release of contaminated pore waters, particularly from softer soil where the in situ water content is quite high. Even if the released sediments could be controlled by careful operating techniques, contaminants in pore water released into the water column in the dissolved phase will be impossible to contain or to control. HRP 00/ 169.

Substantial turbidity (including both resuspended sediments and the release of pore water) is generated by all types of dredging operations. It is a problem that cannot be eliminated. The excavation process itself is a major source of turbidity. In addition, major secondary sources include: resuspension of sediments caused by tug propeller wash (tugs that move the dredging equipment and tugs that haul dredging spoils up and down the river); dragging of swing anchors and setting and removal of spuds; spillage from loading of material onto barges and double handling of material during loading and unloading; spillage from barges when transporting the material and spillage from leaking pipelines; and spillage from offloading of the dredged material from barges and from pipelines.

The controls currently available to reduce turbidity are not effective in the Hudson River situation. Operational controls -- e.g., slowing the speed of cutting in order to prevent unnecessary movement in the water -- will substantially prolong the duration of dredging and will increase the volumes of contaminated water to be treated. Increased dredging duration translates into increased duration of the turbidity being created. Because the operational controls cannot eliminate turbidity completely, there will necessarily be a turbidity problem for the entire duration of the dredging operation.

The use of silt curtains can be effective in some circumstances, but their use is not feasible in a rapidly flowing river like the Hudson River. A silt curtain acts as a barrier to the normal river flow and in effect becomes a partial dam. A silt curtain would therefore be difficult to deploy and hold in place in a large and dynamic river like the Hudson. In fact, the New York State Department of Transportation has attempted to use

HRP 001 1695

silt curtains with its maintenance dredging operations on the Hudson River in the past, but found them to be generally ineffective due to such operating difficulties. In addition, anchorages and work boats required for the silt curtain operation would themselves become a significant source of turbidity, as was discovered in the pilot study at New Bedford Harbor.

Thus, turbidity cannot be adequately controlled and, for this reason alone, dredging in the Upper Hudson River is not a feasible option.

4.4.2 Increased Bioavailability of PCBs

Of particular concern is the potential increase in uptake of PCBs in fish and other aquatic organisms during and after the dredging operations. Several studies have concluded that there is a definite increase in water-borne concentrations of PCBs during and after dredging (Rice and White, 1987; Tofflemire *et al.*, 1979; Hafferty *et al.*, 1977). These studies found that dredging of contaminated sediments increased the bioavailability of PCBs to river organisms during the dredging operations and for at least six months following dredging (Rice and White, 1987). As Rice and White aptly noted in their study, "[t]he obvious conclusion is that dredging appears to have worsened the problem of contamination at least over the short term" (Rice and White, 1987).

Thus, fish and other river organisms and biota will be H_{V} adversely affected due to increased bioavailability of PCBs for at least the duration of the dredging activities and perhaps for Ω_{V}^{O} more than six months after the dredging is completed. If

dredging is selected as a remedy at the Hudson River, it will likely require several years to implement. The longer the duration of the dredging, the longer the duration of bioavailability of PCBs in the river for uptake by fish and other river organisms. Indeed, a massive dredging operation that spans over several years will have a substantial adverse impact on the organisms in the Hudson River.

In addition to the increased bioavailability of pollutants due to the resuspension of sediment, the eventual deposition of resuspended contaminants on the river bed (siltation) may also heighten bioavailabilty.

4.4.3 Destruction of River Habitats and Benthic Communities

One of the most significant ecological impacts that would be wrought by dredging is the destruction of valuable and significant habitats, including wetlands and benthic communities. Shallow and shoreline areas support the most diverse and significant habitats in the Upper Hudson River ecosystem. The riverbanks of the Upper Hudson are home to macrophytic communities (also known as wetlands) that are invaluable habitats to species of all trophic levels. Submerged aquatic macrophytes provide dissolved oxygen for the water, feeding and shelter areas for fish and macroinvertebrates, as well as spawning and nursery areas for various species. Additionally, these wetlands serve the valuable functions of substrate stabilization and water quality improvement.

Emergent macrophytic communities are also present along the river banks which, in addition to sharing the features of their submerged counterparts, provide shallow water areas that function as feeding and nesting habitats for wading birds and waterfowl, as well as shelter, feeding and breeding habitats for mammals, amphibians, reptiles and mollusks. Pockets of submerged aquatic macrophytes can also be found in deeper water areas. Dredging of sediments from these areas would necessarily destroy these significant habitats. As a consequence, the species that are dependent upon their existence would suffer significant impairment.

In addition, dredging threatens the viability of benthic communities in the Hudson River. Surface sediments support many communities of benthic organisms. These organisms include worms, fresh-water mussels and aquatic insects that provide an important food source for large aquatic animals. Dredging would result in the loss of these benthic communities in two ways. First, communities residing in the sediments in areas proposed for dredging would be removed along with those sediments. Additionally, neighboring communities would be buried as sediments are disturbed and redeposited, resulting in further loss of benthic life through suffocation.

Although recolonization will occur once dredging activities cease, the recolonizing organisms will consist of opportunistic species whose environmental requirements are flexible enough to allow them to occupy the disturbed areas. This recolonization by different species will in turn further

239

disrupt larger aquatic organisms. Existing populations at higher trophic levels may suddenly find their food source gone or substantially diminished. Re-establishment of the pre-dredging aquatic community may take years to occur once dredging activities finally cease.

Adverse ecological impacts to wetlands and benthic communities would result from the dredging process itself and from secondary activities such as channel dredging to navigate the equipment barges and workboats, trench dredging to place pipelines, and turbidity associated with work boats and barges. In addition, ecological damage would be caused by increased sedimentation and siltation resulting from accelerated upstream erosion of the river banks and river beds and from resuspension due to dredging operations. In short, damage to wetlands and emergent wetlands would be unavoidable.

4.4.4 Long-Term Ecological Effects

Dredging all contaminated sediment from the Upper Hudson River would be an undertaking of unprecedented proportion, both in terms of the extensive quantity of sediments to be removed and the time it would take to remove them. The scope of such an undertaking greatly multiplies the adverse ecological effects of dredging.

The environmental impacts of maintenance dredging of the Upper Hudson River have long been a concern to regulatory agencies. Limitations and conditions on maintenance dredging activities to minimize these impacts have, however, been imposed. Moreover, maintenance dredging can be a short term operation,

240

which reduces the extent of the damage caused by resuspension of sediment and allows time for benthic communities to recolonize. Additionally, maintenance dredging can be restricted in location and season of occurrence (avoidance of spring/summer months of greatest egg/larvae levels), to minimize adverse affects to aquatic biota. Such protective restrictions cannot be applied to an area as expansive as the Upper Hudson River PCB sediments.

Beyond the immediate ecological impacts of dredging, long term effects are also clear. Alterations in the food chain as a result of the extinction of lower trophic components will have a long-lasting effect on the diversity and abundance of species of upper trophic levels. Destabilized river banks and beds will cause accelerated erosion, resulting in siltation of downstream wetland areas. Other fundamental changes in river hydraulics and sediment transport processes would result in further ecological impacts in the future.

Additionally, the dredged contaminated sediments must be disposed of, a process which may involve dewatering and a consequent return of potentially contaminated water to the Hudson River. Ultimate spoil disposal may also result in the destruction of further aquatic or terrestrial habitats.

4.4.5 River Erosion and Deposition

Rivers are not static. They are in a dynamic changing state, even over a short period of time. Dredging the Upper Hudson River would produce significant changes in the composition of bottom sediments, which would result in accelerated river bed erosion in some areas and increased deposition of sediment in

241

other areas. In addition to direct impacts caused by excavation, the dredging could trigger fundamental changes in the local river hydraulics and sediment transport processes. These changes are likely to include, for example, accelerated river bed erosion caused by removing the natural protective armor layer on the river bed; undermining of river banks; siltation of the main channel; and modifications or accelerations of the natural river meandering processes resulting in further bank erosion and siltation as the river migrates laterally. River mechanics are complex and beyond the scope of these comments. The discussion below simply presents the adverse impacts to the natural state of the river that will result from dredging, keeping in mind that the artificially induced changes in the river will also cause additional future environmental impacts unrelated to the dredging itself.

4.4.5.1 River Bed Erosion

A common feature of rivers that have mixed silt, sand, and gravel beds is the formation of a coarse surface layer, known as an "armor layer." The armor layer is formed during periods of high river flow by erosion of the river bed, with the progressive removal of lighter/finer grained material, leaving behind an immobile surface layer of coarse material. This selective transport and sorting of finer grained sediments is a natural process. The resulting armor layer is essentially a natural protective shell that shields the underlying finer grained material from erosion. The gradation and thickness of the armor layer depends on a number of factors, including the local river

HRP 001 1701
hydraulics (characterized by the bed shear stress), the grain size distribution of the original in-situ "parent" sediment that forms the river bed, and the sediment transport characteristics of the river (in particular, the net bedload transport rate for the area).

Artificial removal of the armor layer by dredging would expose the underlying finer sediments to direct rivar currents, resulting in accelerated erosion of the underlying finer sediments. Localized erosion of the river bed would result in various adverse impacts, such as deposition of the eroded finer grained sediments within the deeper waters of the main channel. Such deposition in turn causes (1) navigational problems for commercial barge traffic, requiring costly maintenance dredging; (2) excessively deep erosion of areas along the shoreline, resulting in undercutting and subsequent erosion of river banks; (3) resuspension of previously buried and protected sediments into the water column as suspended load sediment transport, thereby releasing contaminants such as heavy metals and PCBs into the water column; and (4) modification of the natural hydraulic and sediment regimes of the river, leading to modifications of channel and river meandering patterns and scouring or silting over of marine and plant life. Clearly, the potential for adverse environmental impact on the entire river is substantial. In order to fully understand this problem, hydraulic and sediment transport modeling (as discussed in Section 2.0) is necessary.

HRP 001 1702

4.4.5.2 River Bank Erosion

In addition to the river bed erosion, dredging would cause river bank erosion that could be disastrous at certain locations on the Hudson River. Dredging "up to the shoreline," which may be necessary if the contaminants are located in shallow waters near the shoreline, would result in undermining of the river banks and would cause localized slope stability failure. River bank stability is a particular concern where land is high and slopes are steep.

Additionally, nearshore dredging would adversely effect the stability of existing waterfront structures such as bulkheads: and cribwalls. Roads and utilities along the shoreline would also be damaged or destroyed as a result of slope stability failure in the river banks. It is noted that roads in the Upper Hudson area are frequently located along the riverfront, with guard rails or shoulders that sit right at the top of the river bank slope.

Maintaining the slope conditions will also require significant overdredging, which, as discussed above, will increase the amount of dredged material for disposal. In addition, as a result of the removal of additional material to maintain the slope, the armor layer and natural vegetation of the slope will be removed. This will result in additional accelerated erosion of the slope and substantial undermining of the river bank. Thus, in addition to losses from dredging, the river bank stability problems can be considerably aggravated if

HRP 001 1703

accelerated river bed erosion develops after the dredging is completed.

4.4.5.3 Downstream Deposition

Erosion losses described above are caused by river bed scour due to the loss of the armor layer and river bank sloughing due to slope stability failure. Such erosion losses result in suspended sediment, which is eventually deposited at a downstream location. The resulting downstream deposition can have additional adverse impacts, including deposition in main navigational channels (necessitating additional maintenance dredging) and deposition in shallow wetland areas (adversely affecting aquatic biota and vegetation).

4.4.6 Navigational Impacts

Many possible dredging areas would be far from a central onshore treatment and disposal facility. Barge transport of the dredged material would therefore be required because the distances are excessive for hydraulic pipeline transport. Barge transport, of course, would generate significant volumes of river traffic that would adversely affect other navigational uses of the river. Hydraulic dredging pipelines would also block navigation.

Because the Upper Hudson River consists of a series of dams and pools, barge transport would require transfer through a series of locks. It is conceivable that barges would have to transfer through 6 or more locks, for example to travel from lock 2 to lock 7. Only one barge at a time can transfer through each lock; and therefore, the other commercial and recreational boats

HRP 001 1704

would be forced to wait long periods of time while the barges pass. Because a large number of barges would be required in support of any dredging operation in remote areas, such barge traffic would significantly affect other navigational uses of the Hudson for the duration of the dredging project.

Any dredging in the landlocked portion of the river between Thompson Island Dam and Lock 6 raises a particularly difficult problem. The barging requirements for this area would be quite severe if the area is dredged hydraulically and then material is transferred to material hauling barges in the adjacent non-landlocked area. Such an operation would require that barges transport primarily water, rather than soil, thereby requiring many additional barge trips and a short turnaround time. Major bottlenecks at Lock 6 would develop, interfering with recreational and commercial traffic.

In addition to a pier, an offloading facility would require an access road and a trestle to the pier, an additional pier with a waiting berth located downstream of the offloading pier, a dredged turning basin with a minimum 10 foot depth, full time tug assistance at the pier to aid in barge berthing and deberthing and turnaround operations, and navigation aids and lighting.

Construction and operation of the piers and offloading facilities will of course cause unavoidable adverse impacts to the environment of the Hudson River. In addition to noise, debris, oil slicks, and destruction of vegetation on the banks and riverbottom, there will be significant unavoidable

HRP 001 1705

environmental impacts caused by the dredging necessary to construct and maintain the facilities. Most likely, the full width of the river in the area of the piers would have to be deepened to provide sufficient depth for barge and tug maneuvering and turnaround. Such dredging would adversely affect the aquatic life and shallow water plants, may disrupt wetlands, and will result in additional unnecessary overdredged material for disposal.

Hydraulic pipelines would also cause navigational problems in the Hudson River. Routing of hydraulic pipelines would need to be carefully planned to minimize interferences with normal river traffic and to prevent closing off river access to property owners who own boats. Underwater crossings of the pipelines would be necessary when the dredging operations are located across the river from the onshore facility. The pipelines crossing the river would have to be buried in dredged trenches to provide sufficient clearance for river traffic and for dredging barges. Dredging trenches for pipelines would be yet another source of uncontrollable turbidity, unnecessary overdredged material, and adverse environmental impacts.

4.4.7 <u>Aesthetic Impacts</u>

If barge transport is required, the associated offloading and materials handling facilities would be highly obtrusive on what is now a natural river reach. The waiting berth, offloading berth, slewing unloaders, pipelines, access roads, barges, tugs, etc. would be an eyesore. This portion of the Upper Hudson River would take on an "industrial look."

HRP 001 1706

Additionally, the private homes in the immediate vicinity would lose their view and market value and would be blocked off from direct access to the water.

4.4.8 Health and Safety Risks

There are many risks to human health and safety that may be caused by dredging operations. Briefly, such risks may include air quality impacts from volatilization of PCBs, water quality impacts and fish intake of PCBs released due to resuspension of sediments, possible spillage of contaminated material during offloading of the material to the onshore facility and during transport via trucks through residential areas to a disposal site, possible exposure to the contaminants at the disposal and treatment facilities, and construction safety risks.

For example, air quality impacts may result from volatization of PCB's from contaminated sediments exposed to the atmosphere at the disposal facility and during barge transport. EPA recognized in 1984 that "any large-scale excavation action will result in an increase in a PCB release to the air," as documented by past dredging operations (1984 ROD, p. 11).

In addition, heavy construction in general, and dredging in particular, is a hazardous activity. A study by the U.S. Bureau of Mines (Swan, 1984) revealed that over a 10 year period there were 63 deaths recorded in dredging accidents in the mining industry alone, which represents only a fraction of the total dredging industry. These safety risks, as well as other health risks caused by dredging, cannot be justified in view of

HRP 001 1707

the lack of benefits achieved by dredging. These risks further demonstrate that dredging is not feasible.

In conclusion, there are serious environmental impacts associated with dredging in general and even more devastating impacts associated with dredging contaminated sediments in a dynamic river environment like that of the Upper Hudson.

4.5 Other Concerns

In addition to the issues identified in the previous sections, there are other problems such as land ownership and permit issues that must be carefully analyzed before any further consideration can even be given to dredging. These issues include easements and permits required for installation of pipelines and booster stations in the water and on the land; easements and property condemnations needed for construction of a large pier and shoreline facility for offloading and materials handling; impacts on privately owned riverfront properties, including land losses due to bank erosion; damage from dredging related activities such as grubbing of trees to dredge shoreline areas; and access to privately owned properties for installation of range lines, survey equipment, and anchorages.

A related consideration is the need for construction and environmental permits from federal, state, and local agencies. A fundamental practical issue is the time, cost, and legal obstacles that must be overcome to obtain the necessary permits. Difficulties in obtaining these permits may make dredging in the Hudson River impossible. Moreover, even if the permits can be obtained, by the time the permits are actually

HRP 001 1708

obtained and the dredging is ready to start, the PCB contaminants and the river conditions are likely to have changed considerably due to the dynamic nature of the river.

4.6 <u>Conclusion</u>

The effectiveness and feasibility of dredging in the Hudson River must be considered in the larger context of the costs and environmental impacts of the overall project. Problems associated with treatment and disposal, and environmental impacts due to resuspension of contaminated sediments into the water column are aggravated by unavoidable overdredging due to the limited accuracy of any dredging operation; reduced production rate as a result of careful dredging operations; which increases the duration of dredging and thereby increases the duration of exposure and quantity of resuspension; the volume of excess water included with the dredged material in a hydraulic dredging operation; and the physical restrictions of the Hudson River.

EPA did not consider any of these difficult issues in its one and a half page discussion of dredging in the Phase 1 Report. Had it considered, as it did in 1984, the complexity of the Upper Hudson River and the many insolvable problems associated with dredging contaminated sediments in a dynamic and complex environment like the Upper Hudson, EPA could have reached only one conclusion, the same conclusion it reached in 1984: dredging in the Upper Hudson River is not feasible.

The facts relating to dredging have not changed since 1984. The same problems that persuaded EPA to reject dredging as a remedial alternative then, still exist now, and no new

HRP 001 1709

applicable technology has been developed to solve these problems. Indeed, the various problems associated with dredging on the Upper Hudson are of such magnitude that the selection of any remedial alternative incorporating dredging would be found arbitrary and capricious. Therefore, because nothing has changed since 1984 to make dredging a feasible option at the Upper Hudson, dredging should be removed from further consideration in the Reassessment RI/FS.

4.7 List of References

Borah. 1988. Scour-Depth Prediction Under Armoring Conditions.

Hafferty, A. J., S.P. Pavlou, and W. Hom. 1977. Release of Polychlorinated Biphenyls in a Salt-Wedge Estuary as Induced by Dredging of Contaminated Sediments, Sci. Total Env. 8:229-239.

Huston. 1970. Hydraulic Dredging.

Palermo, M.R. 1991. <u>Equipment Choices for Dredging Contaminated</u> <u>Sediments</u>, Remediation J.

Rice, C.P. and D.S. White. 1987. PCB Availability Assessment of River Dredging Using Caged Clams and Fish, Env. Tox. 6:259-274.

Swan, S.A. 1984. <u>Analysis of Dredge Safety Hazards</u>, United States Department of the Interior, Bureau of Mines, Information Circular 9008.

Tofflemire, T.J., L.J. Hetling, and S.O. Quinn. 1979. PCB in the Upper Hudson River: Sediment Distributions, Water Interactions, and Dredging, Technical Paper No. 55, NYSDEC, Bureau of Water Research, Albany, N.Y.

Turner. 1984. Fundamentals of Hydraulic Dredging.

HRP 001

5.0

IN SITU BIOREMEDIATION

Summary: Recent scientific evidence demonstrates that natural processes are continuously and significantly reducing the impact of PCBs in the Hudson River. Laboratory and field studies show that Hudson River sediments have undergone widespread anaerobic dechlorination, which reduces the toxicity of the PCBs and reduces the accumulation of such PCBs in fish. In addition, the lower chlorinated PCBs that result from anaerobic dechlorination are further degraded by the natural process of aerobic dechlorination. EPA should therefore give proper consideration to this naturally occurring process of biological degradation of PCBs in Hudson River sediments. Biodegradation represents a solution to the problem of PCBs in Hudson River sediments without the devastating ecological impacts of dredging.

5.1 Introduction

In the Phase 1 Report, EPA inexplicably rejects the importance of the naturally occurring biological dechlorination of PCBs that is taking place in the river. Moreover, EPA's discussion of dechlorination demonstrates a number of fundamental scientific misunderstandings on the part of the Agency. GE, an acknowledged leader in PCB biodegradation research, believes EPA must fully evaluate the ongoing natural biodegradation that is occurring in the river. Biodegradation is a fundamental process affecting PCB concentrations and movement that must be incorporated into a comprehensive quantitative approach toward assessing the PCB fate and transport (see Section 2.0 above), in evaluating both human health and ecosystem risks (see Section 3.0 above), and in comparing remedial alternatives. To assist the Agency in this effort, GE is willing to continue to provide the results of its ongoing work to answer any questions EPA may have on the occurrence of biodegradation in the Hudson River sediments.

HRP 001 1711

There are two major scientific findings that warrant EPA's attention. First, PCBs in the sediments in the Upper Hudson River have already undergone extensive anaerobic dechlorination. EPA does not seem to be convinced of this indisputable fact. The consequences of this development are critical to the issue being examined by EPA because lower chlorinated PCBs are not carcinogenic, have significantly lower toxicity, tend to be less persistent in animals and therefore possess a significantly lower tendency to bioconcentrate.

Second, aerobic degradation is known to occur readily on PCBs with lower levels of chlorination. The combined anaerobic and aerobic destruction of PCBs is an important dynamic in the environment. Since the sediments in the river have undergone extensive anaerobic dechlorination, they mainly contain PCBs with lower levels of chlorine. The application of aerobic biodegradation to these sediments will lead to the complete destruction of PCBs.

On this topic of biodegradation of PCBs, GE has included in Appendix H a publication by Dr. Daniel A. Abramowicz, a publication by Donna L. Bedard, and a recent report on GE's research and development program for the destruction of PCBs. GE urges EPA to consider each of these carefully in order that further work is not characterized by the deficiencies of the Phase 1 Report.

HRP 001 1712

5.2 Anaerobic Dechlorination

5.2.1 Introduction

The reductive dechlorination of PCBs has been observed in the laboratory and the environment (Brown et al., 1984; 1987a; 1987b; Brown and Wagner, 1990; Quensen et al., reviewed in Abramowicz, 1990; and Bedard, 1990). Its occurrence in the environment has been confirmed by the altered distribution of residual PCB congeners in aquatic sediments at several locations. In general, this microbial reductive dechlorination affects the preferential removal of meta and para-chlorines, resulting in a depletion of highly chlorinated PCB congeners with corresponding increases in lower chlorinated, ortho substituted PCB congeners. Recent findings demonstrate widespread and progressive PCB dechlorination in the Upper Hudson River (mile point 195 to 156).

This ubiquitous environmental transformation directly results in gradual losses of PCB congeners that are readily bio-accumulated in higher animals and in more rapid losses of the potentially toxic PCB congeners (Safe et al. 1985a; 1985b) (e.g., non-ortho and mono-ortho congeners containing at least four metaand para-chlorines). Therefore, these widespread microbial dechlorination processes have resulted in significant reductions of the theoretical health risks associated with the PCB residues in the upper Hudson River.

What this means in lay terms is that naturally occurring bacteria present generally in river and lake sediments, and definitely present in the Hudson River, are degrading PCBs by removing chlorine atoms. The compounds resulting from this

001 1713

dechlorination process are far less toxic than their origins. Nature is thereby solving the problem at the same time that EPA is spending millions of dollars on yet another Hudson River study.

5.2.2 <u>Dechlorination Status of Upper Hudson</u> <u>PCBs in 1984</u>

The first report of anaerobic PCB dechlorination was made by observing unusual PCB congener distributions in Hudson River sediments (Brown et al., 1984). These initial observations were based upon the limited number of sediment samples available at the time. Confirmation of these environmental changes was obtained by Bopp et al. (1984), who noted that "in every core from the upper Hudson examined thus far, a significant shift toward relative higher abundances of peaks with retention times corresponding to lower chlorinated PCB congeners has been observed. Further evidence of anaerobic dechlorination of PCB congeners in sediments of natural systems should be sought."

An extensive survey of the Thompson Island Pool, a six mile stretch of the Upper Hudson, was performed by the NYSDEC in 1983-1984 (Brown et al., 1988). This survey resulted in 2,073 packed column PCB analyses of sediments collected from approximately 1000 sampling locations (mile point 194.5 to 188.5). In Figure 5.2.2-1, the source locations of samples containing at least 10 pm PCB at the time of sampling (545 of the 2,073 samples) were mapped. The stored database contains the peak areas from the original packed GC analyses, in addition to total PCB concentrations. Ratios of peak areas were used to

HRP 001 1714

estimate the extent and breadth of PCB dechlorination in the sediments. One measure of dechlorination can be obtained by determining the ratio of mono-ortho tetra-chlorinated PCB's (peak 70) to mono-ortho tri-chlorinated PCBs (peak 47). For example, in pure Aroclor 1242, the principal material used by the GE plants, this ratio is approximately 1.8. Any sample with a ratio of less than or equal to unity is considered "significantly dechlorinated" and is circled in Figure 5.2.2-1.

Quantitative determination of the average chlorine level is not possible because the response factors for the individual peaks were not determined in the original analysis. Over 70 percent of the 1984 samples displayed significant dechlorination, with the ratio of peak 70 to peak 47 less than or equal to unity. The average peak ratio for all PCB concentrations surveyed was less than 0.5, as shown in Table 5.2.2-1. Therefore, the peak ratio decreased nearly four-fold on the average for all samples containing greater than 5 ppm PCB, and decreased five-fold on the average for samples containing greater than 100 ppm PCB (Table 5.2.2-1). The proportions of samples within the different PCB concentration ranges showing significant dechlorination are given in Table 5.2.2-2.

Table 5.2.2-2 shows that the prevalence of significant dechlorination increased from 63 percent of the samples in the lowest PCB concentration range (5-10 ppm) to 93 percent in the highest range (greater than 100 ppm). Both the Table 5.2.2-1 and Table 5.2.2-2 results indicate that environmental PCB dechlorination may proceed faster and more extensively in

HRP

100

1715

sediments containing higher concentrations of PCBs, in agreement with previous laboratory studies (Quensen, 1988).

The peak ratio discussed above (peak 70/peak 47) is an indicator of overall dechlorination from packed column PCB analyses. Similar results supporting widespread PCB dechlorination in the Upper Hudson River were obtained with other peak ratios. Examples include peak 37/peak 21 (indicator of pattern B and B' dechlorination), and peaks 37+40/peak 11 (indicator of mono-CB formation). Pattern designations are defined in Brown *et al.* 1987a.

These results demonstrate that microbial PCB dechlorination was already widespread throughout Upper Hudson River sediments in 1984. Extensive changes had occurred in sediments exhibiting a broad range of PCB concentrations, even as low as 5 ppm. More refined quantitative comparisons would require high resolution PCB analysis (capillary GC), as shown in the following section.

5.2.3 <u>Dechlorination Status of Upper Hudson</u> <u>PCBs in 1990</u>

Extensive high resolution PCB analysis of Upper Hudson River sediments has been performed at the H7 site (mile point 193.5). PCB chromatograms displaying typical PCB distributions from 1982-1990 are shown in Figure 5.2.3-1. Even the earliest sample (Figure 5.2.3-1A) displays a considerable level of ortho substituted products (2- and 2-2/26-CB), compared to Aroclor 1242, indicating that dechlorination was already well-advanced.

HRP 001 1716

However, a significant amount of the more highly chlorinated PCB congeners still remained at the time of the earliest sample.

Over the next eight years (Figure 5.2.3-1B and C), dechlorination continued until over 80 percent of the total PCBs in the sediment samples consisted of 2-CB and 2-2/26-CB. The chromatogram displayed in Figure 5.2.3-1C represents an extensively dechlorinated environmental sample, and is similar to dechlorination Pattern C previously described in published papers (Brown et al., 1987a). The average chlorine level decreased from 3.6 to approximately 2.0 in this sample, indicating the removal of most of the meta and para chlorines.

It is difficult, however, to determine accurate environmental dechlorination rates from these few samples because significant spatial variations may exist at the different timepoints.

Therefore, in order to determine the spatial variation in this dechlorination activity, a dense grid of core samples was obtained from the H7 site in the summer of 1990 (68 sampling sites on 12 foot centers with 151 high resolution PCB analyses). The results of the capillary PCB analyses are shown in Figure 5.2.3-2. The results show extensive variations in PCB concentrations even in adjacent samples and core sections. The corresponding dechlorination levels, expressed as average chlorine content per biphenyl, are shown in Figure 5.2.3-3. HRP Sediments obtained before the onset of dechlorination contained **L**00 approximately 3.6 C1/BP. The mean of these average C1/BP ratios from the H7 site is 2.3 (n = 62, σ = 0.3), similar to the 1717

distribution represented in Figure 5.2.3-1A. Extensive anaerobic dechlorination had occurred uniformly throughout the entire site, at both low and high PCB concentrations.

In hopes of identifying minimally dechlorinated sites that could be used for future field tests of techniques for accelerating PCB dechlorination rates, additional sampling was performed in 1990. Eighteen locations (ranging between mile points 163 and 195) were selected as the <u>least</u> dechlorinated areas based upon the 1984 NYSDEC survey results and other sampling. The results of the 1990 survey and high resolution capillary PCB analyses are shown in Table 5.2.3-1. Site 11 (mile point 169, 3.4 C1/BP) appeared to be least active, but further sampling revealed that significant dechlorination had occurred even at this site (elevated levels of 2- and 2-2.26-CB). The high average chlorine level was found to originate from additional contamination from a more highly chlorinated PCB mixture. This was most probably the result of a small, localized spill of Aroclor 1254, because subsequent sampling nearby yielded only 2.7 and 2.6 C1/BP (dechlorinated Aroclor 1242 only).

Likewise, additional sampling at site 18 yielded 2.3 and 2.3 C1/BP. The mean of these average C1/BP ratios from the survey was 2.5 (n = 32, σ = 0.3) when the uncontaminated samples from sites 11 and 18 were used.

Therefore, even at sites selected for minimal dechlorination in 1984, significant changes from the original 3.6 C1/BP had occurred by 1990. The chromatographic changes observed in the environmental samples demonstrated the selective loss of

meta and para chlorines, which is characteristic of natural microbial dechlorination.

5.2.4 <u>Summary and Conclusions</u>

Several different indicators (decreasing PCB levels in sediments, fish, and the water column) have established that PCB levels in the Upper Hudson River are declining. It is now also established from 1984 and 1990 sediment survey data that anaerobic PCB dechlorination has occurred on a wide scale throughout the upper Hudson. It is also known that aerobic microorganisms capable of degrading the lightly-chlorinated, ortho-substituted products of anaerobic activity are widespread and common in Upper Hudson sediments. Therefore, sequential anaerobic dechlorination/aerobic biodegradation is eliminating PCBs from the Upper Hudson River.

In addition, the pervasive dechlorination process already completed has resulted in reduced concentrations of highly chlorinated PCB congeners in sediments, including those congeners that can bioaccumulate in fish and those that are potentially toxic. Therefore, microbial dechlorination is a significant ongoing process that must be taken into account in characterizing the site, modeling the mechanisms that affect PCB fate and transport, evaluating human health and ecological risks, and comparing remedial alternatives.

Furthermore, anaerobic biodegradation is not limited to the Hudson River. PCB-containing sediments from Escambia Bay, FL; Hoosic River, MA; Hudson River, NY; Kalamazoo, MI; Massena, NY; New Bedford Harbor, MA; Sheboygan River, WI; Silver Lake, MA;

001 1719

HRP

Waukegan Harbor, IL; and Woods Pond, MA all undergo environmental PCB dechlorination (Brown et al., 1987b; Abramowicz, 1990). Recent reports on New Bedford Harbor (Brown and Wagner, 1990; Lake et al., 1991) indicate that the observed activity at that site is also not localized. Even uncontaminated sediments (from an Adirondack marsh near Stony Creek, NY; Center Pond, MA; Red Cedar River, MI; Saline River, MI; and the Hudson River at Spier Falls, NY) contain microorganisms capable of catalyzing the reductive dechlorination of PCBs (GE Report, 1990). This evidence suggests that the metabolic capability utilized in this process is common and widespread among many different anaerobic microorganisms.

5.3 Sequential Microbial PCB Degradation

As discussed above, the PCBs remaining in the Upper Hudson River have undergone extensive reductive dechlorination. Not only are the remaining lower chlorinated PCBs less toxic and less prone to bioconcentration (Bopp, 1989), they are also amiable to complete destruction by naturally occurring aerobic bacteria. It has been demonstrated that organisms found in the Hudson River sediment are capable of destroying the anaerobically altered PCBs found in the sediments of the Upper Hudson River (Abramowicz et al., 1990). To better understand the process of aerobic degradation of PCBs in the Hudson River sediments, GE is currently performing a field experiment, under EPA permit. This experiment will help resolve many issues related to factors controlling the occurrence and rate of aerobic PCB destruction in the river. HRP 001 1720

With respect to the ongoing aerobic destruction of PCBs that have already been dechlorinated, there are two processes that must be understood. The first is that the lower anaerobic zone contains the sediments that have already been significantly dechlorinated. These sediments with altered PCBs (most likely isolated from the active biological layer of the sediment), may slowly supply PCBs to the overlying sediment and water column by chemical diffusion. The altered PCBs may then be exposed to aerobic conditions, and aerobic degradation will occur. Additionally, the PCB homologs (mono- and di-) that dominate the PCB mixtures are more volatile, less readily adsorbed by particulate matter, and bioaccumulated to a lesser extent than the homologs with higher amounts of chlorine.

Diffusion is not the only process that may expose isolated (below the top few centimeters of sediment) PCBs in the Upper Hudson River sediment to more active biological zones. EPA and the NYSDEC have considered the potential for a major flood event to "stir up" the buried sediments, with the fear that the PCB levels in the fish and water column would increase due to this enhanced availability. This fear, however, may be unfounded due to the potential aerobic degradation that may then occur. It is likely that the buried PCBs that have undergone anaerobic dechlorination would undergo complete destruction when placed into an aerobic environment due to such a scouring event. Additionally, even if these scoured PCBs were redeposited after a $_{_{\rm III}}$ RP flood in the portion of the sediment layer that is biologically 100 available, these lower chlorinated PCBs have less

bioconcentration potential (and lower toxicity) than the more highly chlorinated PCBs that may currently reside in the upper portion of the sediment. Thus, to determine what impact a major scouring event might have, the effect of both anaerobic and aerobic biodegradation needs to be considered.

Based on the work done by GE to date, the naturally occurring sequential anaerobic-aerobic PCB destruction is expected to be very effective. Moreover, the fact that the sediments in the Upper Hudson have already experienced significant reduction in chlorine content makes the results already beneficial in view of the reduced toxicity of the lower chlorinated compounds. The advantages of permitting such a natural process to continue as compared to initiating an invasive remediation, such as dredging and removal include:

- Biodegradation is a permanent solution that completely destroys PCBs as opposed to only relocating contaminated sediments, and in this respect it is the type of process favored by Congress in SARA;
- 2. No landfills are required for biodegradation, and therefore there is no land destruction and community disruption, which would be attendant to relocating contaminated sediments;
- 3. Biodegradation does not disrupt wetlands and aquatic habitats or have the other devastating by ecological effects of dredging.

1722

In addition to performing field tests relating to the conditions for aerobic biodegradation, GE is evaluating the extent to which aerobic destruction of PCBs is already occurring naturally. GE has been able to demonstrate the widespread natural occurrence of anaerobic PCB dechlorination in the Upper Hudson River sediments based on a distinctive shift in the amount of chlorine in the PCBs. On the other hand, until recently, such a measurable indicator for the occurrence of aerobic degradation was not present. Based on recent research, however, GE believes that if the buried sediments are supplying lower chlorinated PCBs to the water column (via diffusion), then they will be broken down aerobically yielding a chlorobenzoate intermediate. Next year, actual Hudson River samples will be tested to determine if this is an indicator of aerobic PCB destruction and, if so, to what extent complete destruction of PCBs is naturally occurring in the river system.

5.4. Specific Comments on EPA Review

5.4.1 PCB Biodegradation

Technically, Phase 1 Report subsections C.4.2 and C.4.4.3 are inadequate and misleading. Poor, discredited studies are given equal weight with well-designed, confirmed results. In addition, there is no mention of the widespread, pervasive dechlorination that is known to exist throughout the Upper Hudson River. EPA acknowledges in the Phase 1 Report that dechlorination is a possible, and even likely, explanation for the congener redistribution in the Upper Hudson (p. C.4-6), but the extent of the transformation is not documented in the Report.

001 1723

HRP

Moreover, the data provided by GE to EPA, demonstrating widespread dechlorination (reanalysis of the NYS DEC 1984 data; GE 1990 survey of less dechlorinated sites; GE 1990 survey of H7 site), were not even evaluated in the Report.

In section C.4.2, on natural biodegradation in sediments, the data GE provided to the EPA on widespread dechlorination in the Upper Hudson River were noticeably absent. The discussion of microbial anaerobic dechlorination underway in the Upper Hudson River occupies only one page (C.4-2) in a document of several hundred pages. Such a superficial treatment of a topic with potentially critical consequences to the Hudson River RI/FS process is unjustified scientifically. In addition, anaerobic dechlorination is mentioned as only one possible explanation for the unusual Aroclor patterns in the Upper Hudson River (section C.4.2.1).

The section on natural PCB biodegradation in sediments also fails to mention the two preeminent publications concerning anaerobic dechlorination in Hudson River sediments (Brown et al., 1987a; Quensen et al., 1988). These publications in a leading scientific journal proposed that the specific removal of <u>meta</u> and <u>para</u> chlorines observed in environmental samples (e.g., Hudson River and Silver Lake) was the result of microbial reductive dechlorination (Brown et al., 1987a) and demonstrated that similar biologically-meditated transformations occurred with Hudson River sediments in the laboratory confirming that $\frac{m}{2}$ hypothesis (Quensen et al., 1988).

265

In section C.4.2.3, the discussion of dechlorination with Hudson River sediments first observed in Tiedje's lab fails to mention several recent publications in the field (Abramowicz and Brennan, 1991; Brown and Wagner, 1990; Lake et al., 1991; and Van Dort and Bedard, 1991). These papers discuss the dechlorination of endogenous PCB contamination in Hudson River sediments and Drag Strip soils (Abramowicz and Brennan, 1991), document natural dechlorination in New Bedford Harbor sediments (Brown and Wagner, 1990; Lake et al., 1991), and demonstrate the potential to remove microbially the previously inert ortho chlorines with river sediments. These references represent significant recent advancements with particular bearing on the Hudson River project.

The Report also fails to mention other results that support Brown's hypothesis of natural dechlorination in aquatic sediments, including Bopp's sampling of the Upper Hudson, which found dechlorination in every sample (mentioned on page B.3-12), documented environmental dechlorination observed at nearly a dozen sites around the country, and research efforts by Woods (Oregon State University), Reeves (Oak Ridge National Laboratory), and Celgene (Warren, NJ).

In addition, EPA's own research laboratory in Gulf Breeze has begun dechlorination research. The GE data demonstrating widespread dechlorination in the Upper Hudson River provided to the EPA, which add additional support to this claim, was ignored in the report.

266

Such widespread confirmation of microbial anaerobic dechlorination with aquatic sediments in both laboratory and environmental settings requires that the changes observed in the Upper Hudson River be unequivocally attributed to this natural microbial process.

PCBs are not the only chlorinated organic anthropogenic chemicals that can undergo microbial reductive dechlorination naturally in the environment. The widespread environmental dechlorination of chlorinated organics is also not mentioned in the Phase 1 Report. Examples of this common phenomenon include: chlorinated dioxins and dibenzofurans (Parson, University of Amsterdam), pentachlorophenol and chlorinated benzenes (Beurskens, Institute of Inland Water Management), pesticides (Suflita, University of Oklahoma), chlorinated phenols in Baltic sediments (Neilson, Swedish Environmental Research Institute), and PCBs in marine sediments (Lake, EPA-Narraganset). This worldwide evidence of reductive dechlorination is further confirmation of this natural microbial process. An international conference sponsored by the American Society of Microbiology on "Anaerobic Dehalogenation and its Environmental Implications" will be held April 12-17, 1992 in Helen, Georgia (Co-chairs Daniel Abramowicz from GE/CRD and John Rogers from EPA/Athens).

In section C.4.4.3, the Phase 1 Report incorrectly asserts that PCBs pose greater challenges to bioremediation than other contaminants (e.g., petroleum products). In fact, PCBs and petroleum products are very similar in terms of their biodegradation potential: both are complex mixtures of

hydrophobic compounds; both can be degraded by organisms found commonly in the environment; in each case the higher molecular weight material is of relatively greater risk and more difficult to degrade; and widespread environmental degradation of petroleum products and PCBs are documented. In spite of these similarities, the Phase 1 Report states that oils are easy to bioremediate, while PCBs pose "greater challenges". In addition, the Report incorrectly states that successful PCB bioremediation requires the identification of a microbial population capable of degrading a large number of different PCB congeners. In the Upper Hudson River, widespread natural anaerobic dechlorination to a few lightly chlorinated PCBs has removed this prerequisite.

In section C.4.4.3 of the Phase 1 Report (p. C.4-28), it is mentioned that in <u>situ</u> anaerobic dechlorination easily could be accomplished, but that it would not reduce the total molar PCB concentrations. No mention is given to the promising ortho dechlorination recently discovered that may overcome this limitation (Van Dort and Bedard, 1991). The Phase 1 Report also fails to mention the significant detoxification demonstrated by meta and para removal alone (Quensen et al., GE Report, 1990).

Moreover, the Phase 1 Report omits mention of the dramatic effect this widespread dechlorination would have on the bioaccumulation of PCBs. The less chlorinated PCBs are significantly less hydrophobic and are metabolized and/or cleared RP from fish and humans much more readily than the more highly 001 chlorinated congeners (Brown et al., 1989). The human clearance rates for the lightly chlorinated products of anaerobic 1727 dechlorination are quite rapid. For example, nearly 80 percent and more than 90 percent of the PCBs present in sediments of the Upper Hudson River displaying pattern C dechlorination are cleared by humans with half-lives of <0.01 yr. and <0.1 yr., respectively. Nearly 80 percent of the PCBs present in sediments of the Upper Hudson River displaying pattern B dechlorination are cleared by humans with half-lives of <0.1 yr. These facts should have important implications on the human risk assessment of PCBs for the Upper Hudson; however, these facts are not acknowledged in the Phase 1 Report.

Finally, the Phase 1 Report fails to mention the rapid progress of bioremediation, as evidenced by Ecova's recent completion of the largest bioremediation cleanup to date (<u>Genetic</u> <u>Engineering News</u>, 1991). Other examples of notable bioremediation efforts include the clean-up of the Valdez beaches (Pritchard and Costa, 1991) and a discussion of over 140 bioremediation projects being considered, planned, or implemented at various sites (EPA, 1991). In the 1991 EPA report on bioremediation, over a dozen sediment applications are identified.

5.5 <u>Summary</u>

The existing information on PCB biodegradation is very important and persuasive. It has been demonstrated that the PCBs in the sediments in the Upper Hudson River have already had a significant amount of chlorine removal due to anaerobic microbial dechlorination, thereby reducing them to less toxic compounds. Additionally, the process of sequential anaerobic-aerobic

269

biodegradation of PCBs has been proven to occur in naturally occurring sediments in the laboratory under conditions similar to those of Hudson River sediments.

EPA must carefully consider the fate and transport process that will affect these altered sediments in the future, as well as the effect of other remedies on this naturally occurring remedial process. It is imperative that EPA fully understand this important ongoing process and the beneficial consequences it has for the long term recovery of the river.

5.6 List of References

Abramowicz, D.A. and M.J. Brennan. 1991. In: Biological remediation of contaminated sediments with special emphasis on the Great Lakes. Aerobic and anaerobic biodegradation of endogenous PCBs. C.T. Jafvert and J.E. Rodgers (eds), EPA/600/9-91/001, pp. 78-86.

Bedard, D.L. and M.L. Haberl. 1990. Influence of chlorine substitution pattern on the degradation of polychlorinated biphenyls by eight bacterial strains. <u>Microb. Ecol., 20:</u> 87-102/

Brown, J.F., Jr., D.L. Bedard, M.J. Brennan, J.C. Carnahan, H. Feng and R.E. Wagner. 1987a. Polychlorinated biphenyl dechlorination in aquatic sediments. <u>Science 236:</u> 709-712.

Brown, J.F., Jr., R.E. Wagner, H. Feng, D.L. Bedard, M.J. Brennan, J.C. Carnahan and R.J. May. 1987b. Environmental dechlorination of PCBs. <u>Environ. Toxicol. Chem. 6:</u> 579-593.

Brown, J.F., Jr., R.W. Lawton, M.R. Ross, J. Feingold, R.E. Wagner and S.B. Hamilton. 1989. Persistence of PCB congeners in capacitor workers and yusho patients. <u>Chemosphere 19:</u> 829-834.

Brown, J.F., and R.E. Wagner. 1990. PCB movement, dechlorination, and detoxication in the Acushnet estuary. <u>Environ. Toxicol. Chem., 9:</u> 1215-1233.

Environmental Protection Agency. 1991. Bioremediation in the field. EPA/540/2-91/007, no. 2, March 1991.

General Electric Company Research and Development Program for the Bestruction of PCBs, Eighth Progress Report. 1989. General Electric Corporate Research and Development, Schenectady, NY.

HRP

General Electric Company Research and Development Program for the Destruction of PCBs, Ninth Progress Report. 1990. General Electric Corporate Research and Development, Schenectady, NY.

General Electric Company Research and Development Program for the Destruction of PCBs, Tenth Progress Report. 1991. General Electric Corporate Research and Development, Schenectady, NY.

Genetic Engineering News. 1991. Ecova Corp., General Electric Move Bioremediation Technology Forward. September 1991, pp. 20-21.

Lake, J.L., R.J. Pruell, and F.A. Osterman. 1991. In: Organic substances and sediments in water, Lewis publishers. Dechlorinations of PCBs in sediments of New Bedford harbor, (in press).

Pritchard, P.H. and C.F. Costa. 1991. EPA's Alaska oil spill bioremediation project. <u>Environ. Sci. Technol. 25:</u> 372-379.

Quensen, J.F., III, S.A. Boyd and J.M. Tiedje. 1990. Dechlorination of four commercial polychlorinated biphenyl mixtures (Aroclors) by anaerobic microorganisms for sediments. <u>Appl. Environ. Microbiol. 56:</u> 2360-2369.

Quensen, J.F. III, J.M. Tiedje and S.A. Boyd. 1988. Reductive dechlorination of polychlorinated biphenyls by anaerobic microorganisms from sediments. <u>Science 242:</u> 752-754.

Van Dort, H.M. and D.L. Bedard. 1991. Reductive <u>ortho</u> and <u>meta</u> dechlorination of polychlorinated biphenyl congener by anaerobic microorganisms. <u>Appl. Environ, Microbiol. 57:</u> 1576-1578.

OTHER PCB SOURCES

Summary: The effectiveness of potential remedies cannot properly be assessed until sources of PCBs to the relevant media have been adequately characterized. The Phase 1 Report fails to address this fundamental issue. EPA's basic assumption of massive movement of PCBs from the Upper River is flawed. Radionuclide dating shows that the PCB peak in the Lower River occurred before the 1973 dam removal. This pre-1973 peak has been observed at other sites, and all other categories of Hudson sediment data point to local PCB sources. Furthermore, analysis of striped bass data shows that the Upper River accounts at most for only a small fraction of PCBs accumulated by the fish. EPA's approach to long distance transport from the Upper River and the effects of future floods must be reevaluated in light of this evidence. EPA must recognize that multiple PCB sources exist and that these sources, and not the load from the Upper River, are the primary sources of PCBs in the River.

6.1 The Benefits of Potential Remedies Cannot Be Assessed Without Adequate Characterization of Sources

It is fundamental to the Superfund RI/FS process that before any potential remedies are assessed, the site must be adequately characterized, and in particular, the sources of contamination must be defined. Logic dictates that this step be taken early, and indeed, EPA's guidance on RI/FS procedure mandates it (40 CFR § 300.430(d)(2)(iv)). The reason for early identification of sources is obvious. Without source identification, it is impossible to predict what impact, if any, potential remedies will have on reducing exposure to the contamination.

Against this backdrop of a fundamental first step, $\frac{72}{9}$ EPA's definition of PCB sources in the Phase 1 Report can be $\frac{9}{10}$ summarized as follows:

173]

- EPA accepts, without question, the commonly-held assumption that historical PCB contamination of both the Upper and Lower Hudson is dominated by massive movement of PCBs from the two GE facilities after the 1973 dam removal (pp. E-5, A.3-2).
- EPA also assumes that the only current significant sources of PCB contamination in the Upper River are deposits from GE's historical discharges, and that transport from those deposits continues to be a major source for the Lower River (pp. B.2-1, A.2-2).
- EPA acknowledges that there are other current sources of PCBs to the Lower Hudson that are of similar magnitude to PCB transport from the Upper River (p. A.2-3). The Agency attempts to estimate the quantity of PCBs from some of those other current sources. There has been no examination of historical sources in the Lower River.
- EPA also acknowledges that further investigation of other Lower River sources may be necessary to assess potential effects of remedial efforts (p. E-6). There is no indication what the Agency will do to correct the deficiency.

A thorough review of existing data shows that the PCB source analysis EPA has conducted is fundamentally flawed. The analysis is incomplete, and its underlying assumptions are incorrect. As further amplified in this Section, proper conclusions from the data are as follows:

- Based on radionuclide dating of sediment, the peak PCB concentration in Lower River sediment occurred prior to, rather than after, the 1973 dam removal in the Upper River. A pre-1973 peak in PCB concentration has been observed at other sites and is consistent with the 1971 peak in nationwide use of PCBs.
- All other categories of sediment data further support the conclusion that PCB contamination in the entire River has not been caused by massive movement from a primary, single source in the Upper River, but rather by minimal movement of PCBs from multiple sources.
- Analysis of fish data shows that fish accumulate PCBs from local sources. In the case of striped bass, which spend as little as two months in the Hudson, those local sources are primarily outside of the Hudson.

273

- EPA's estimate of PCB discharges from identified current sources is low. The Upper River contributes, at most, only a small fraction of the Lower River PCB loadings. Of critical importance, the evidence demonstrates that the Upper River will play an even smaller role -- in both absolute and relative terms -in the future.
- EPA has ignored significant evidence in its files and those of other regulatory agencies regarding numerous other PCB sources.

The importance of the above conclusions, particularly those regarding the fundamental assumption about massive movement of PCBs in the Hudson, cannot be overstated. If no massive movement of PCBs occurred historically, EPA must seriously evaluate: (1) whether GE could be significant source of Lower Hudson PCBs; and (2) whether a significant quantity of PCBs could possibly be transported today, even under flood conditions, over long distances from the Thompson Island Pool to other parts of the River. Furthermore, if PCB impacts within the River are primarily the consequence of local sources, EPA must seriously investigate those sources so that truly effective remedies can be assessed.

6.2 Sediment Data Demonstrates That The Origin And Movement Of Hudson River PCBs Is, And Has Historically Been, Dominated By Multiple Sources

As previously noted, a fundamental premise accepted without question in the Phase 1 Report is that virtually all of the PCBs in the Hudson River originated from two Upper River GE plants. This premise may be termed the "single source/massive movement" model for Hudson River PCB contamination. This hypothesis was originally proposed by investigators who were then

at the Lamont-Doherty Geological Observatory of Columbia University (Bopp, 1979; Bopp et al., 1981, 1982).

An alternative hypothesis is that the PCBs now detectable in Hudson River sediments came from multiple sources that were generally located no more than a few miles away. This view may be termed the "multiple source/minimal moviment" model. The concept of multiple sources was originally proposed by investigators at EPA Region II (USEPA, 1977). Subsequently, investigators at the New York University (NYU) Institute of Environmental Medicine Laboratory for Environmental Studies also observed multiple sources with minimal transport (O'Connor et al., 1982). This alternative hypothesis recently inspired extensive examination of PCBs in the sediments and biota of the Lower Hudson River, New York Harbor, and Long Island Sound by the Harza Engineering Co. with funding from GE (Shephard et al., 1990). Data from Harza's investigation are presented in Tables 6.2-1 and 6.2-2.

The table below summarizes the various PCB sediment surveys that have been conducted in the Lower River:

YEAR	SPONSOR	NO. OF SA LOCATIO	MPLE <u>NS</u>	REFERENCE
1976	EPA	28	1	USEPA, 1977
1977	Columbia (Lamon Doherty)	it- 24	1	Bopp, 1979, 1981, 1982
1981	EPA	12	· · · · · ·	USEPA, 1982
1988-90	Harza, GE	114		Shephard, 1990
	Interestingly,	the 1977	EPA report	and the 1979-81 Bop
et al. re	ports drew their	sharply	divergent c	onclusions from

virtually identical survey data. Each reported PCB levels in approximately 25 sediment cores taken in 1976-77 from very similar sets of sites along the tidal (i.e., Lower) Hudson between New York City and Albany, with the Bopp 1977 sampling plan being clearly guided by that used by EPA in December 1976. Each study showed considerable point-to-point variation in PCB levels between sites and some variation in PCB composition (Aroclor 1254/1242 ratio). Each study found that the average level of PCBs in the surficial sediments of the Lower Hudson was 6 to 8 ppm in 1977, as contrasted to overall average levels in the 15 to 25 ppm range that were indicated by the 1977-78 sampling of the Upper Hudson study site by NYSDEC (Tofflemire and Quinn, 1979). Furthermore, follow-up studies by both groups (USEPA, 1982; Bopp and Simpson, 1989) indicated that the PCB levels in surficial sediments of the Lower Hudson were declining, and at quite similar rates (half-lives of about 4.5 and 3.5 yrs, respectively). Evidently, the divergent conclusions reached by the original (and subsequent) investigators have resulted not from differences in their collected data, but in the ways in which the data were compared and interpreted.

To determine which of two alternative hypotheses provides the better interpretation of a body of data, it is standard scientific practice to set forth the predictions made by each, and then to determine which of the hypotheses should be rejected on the basis of the incompatibility of its predictions with the available observations. The remainder of this section (Section 6.2) does exactly that for each of the seven categories $\frac{7}{2}$

of data now available for testing the validity of the predictions of the single source/massive movement and multiple source/minimal movement models for Hudson River PCB contamination.

6.2.1 Radionuclide Dating Of Sediment Cores -- The <u>PCB Peak Occurred Before The 1973 Dam Removal</u>

The most important and most frequently cited support for the single source/massive movement hypothesis is a series of reports on radionuclide dating of sediment cores in the Lower Hudson and New York Harbor performed by the Lamont-Doherty Observatory (Bopp, 1979; Bopp et al., 1981; Bopp et al., 1982; Bopp and Simpson, 1989). EPA relies upon these reports in the Phase 1 Report (p. A.3-2).

In the Lower Hudson, the Lamont-Doherty reports conclude that PCB concentrations in sediment cores are highest during 1973 and that these peaks in concentration were caused by the transport of sediment from the Upper River when the Fort Edward Dam was removed in October 1973. A careful review of the actual radionuclide and PCB data in Bopp (1979) and Bopp et al. (1982) does not support that conclusion. Instead, the data show that the PCB concentration maxima south of Albany pre-date 1973 and thus cannot be attributed to the 1973 dam removal.

Sediment core data reported in Bopp et al. (1982) show that, for cores collected at Foundry Cove, Indian Point, and New York City, the PCB concentration maxima are found either in the same strata or deeper in the core than the Cs-137 maxima attributed to radionuclide releases at the Indian Point Nuclear Generating Station in 1971. Portions of the relevant core data

HRP 001 1736

are reproduced and highlighted in Table 6.2.1-1. Although as previously noted in Section 2.4.1, radionuclide dating can lead to erroneous inferences about broad-scale PCB deposition patterns, the core data relevant to the PCB peak here is very close in time and space to the Indian Point release and therefore gives a reliable indication of the time of deposition.

Also significant in the Lamont-Doherty data is the absence of PCB deposition maxima in Lower Hudson sediments dated from either 1974 or 1976. Known flood events took place during those two years, with the 1974 flood being the first probable event to cause any transport after the 1973 dam removal. If transport of Upper Hudson sediments to the Lower Hudson were responsible for PCB contamination of the Lower Hudson, PCB concentration maxima should be observed in sediments of the Lower Hudson for these two years. However, no such maxima are observed.

The correct interpretation of the data in Bopp *et al.* (1982) is that the PCB maxima occurred in 1971 or earlier, which corresponds with the peak in maximum production and use of PCBs nationally. The peak in national production and use is shown in Figure 6.2.1-1 (Monsanto sales) and Table 6.2.1-2 (PCB environmental load), both compiled by Versar for EPA in 1976. The rates of increase and decrease in PCB concentrations in Lower Hudson sediments, as shown by the Lamont- Deherty data, track the same rates reported by Versar for PCB use and PCB releases into the environment.

HRP 001 1737
The 1971 PCB peak observed in Lower Hudson sediments is not an isolated observation. The same pattern corresponding to national PCB use has been seen in numerous other bodes of water. The Canada Centre for Inland Waters observed for Lake Ontario that "peak concentrations for . . . PCBs occurred in the mid-1960s (up to 1971), and there is good agreement between the core record and the production or usage history" (Oliver et al., 1989, p. 204; Figure 6.2.1-2). The same pattern was seen in Lake Erie (Mackey et al., 1983, p. 257). Furthermore, the U.S. Fish and Wildlife Service, as part of its National Pesticide Monitoring Program, found that the peak concentrations of Aroclor 1254 in all fish sampled nationwide occurred in 1971 (Schmitt, 1981, p. 282).

The facts show that PCB concentrations were already declining by 1973 in the lower reaches of the Hudson estuary. The cores provide no evidence of a 1973 PCB maximum and, therefore, no evidence of extensive downstream transport of PCBs into the Lower Hudson due to removal of the Fort Edward Dam.

6.2.2 Local Variability in PCB Levels

The second category of data that can be used to test the two alternative hypotheses of sources and transport in the Hudson consists of sediment data on PCB levels within a local area. As long suspected (Bopp, 1979) and as now amply confirmed by observations in both the Upper and Lower Hudson, PCB levels in riverbottom sediments can vary widely over scales of just a few yards because of local variations in the ability of the riverbottom to accumulate either the fine or coarse organic

HRP 001 1738

particles that carry PCBs. Since this local variability arises from local hydrodynamic variations, it cannot be used to discriminate between the alternative hypotheses regarding distant PCB sources. Moreover, this local variability in PCB levels means that conclusions about regional PCB levels cannot be drawn from isolated individual samples.

6.2.3 Local Variability in PCB Composition

Although the single source/massive movement and multiple source/minimal movement models make identical predications as to local variability in PCB levels, they make quite different predictions as to local variability in PCB composition. The single-source model predicts that PCB deposits within a given area in the river should have nearly the same composition (i.e., that of the release from the singular source), whereas the multiple source/minimal movement model predicts that at least part of any differences in source PCB composition should be reflected in those of nearby sediment deposits.

The 1977 Lamont-Doherty survey of the Lower Hudson (Bopp, 1979; Bopp et al., 1981, 1982) and the 1976 and 1981 EPA surveys (USEPA, 1977, 1982) each involved collections of individual cores at widely spaced sites. Thus, small-scale horizontal variations in sediment composition could not be evaluated.

The Harza survey (1988-1990) did, however, collect multiple samples from each study area so that the local heterogeneities in PCB composition in the horizontal dimension could be evaluated. Close examination of these variations

indicated that they were of two types. First, in some samples the compositions were well outside the normal range of variation for the region, and the capillary gas chromatogram indicated a composition dominated by homologs indicative of Aroclors more chlorinated than those used by GE in the Upper Hudson. These compositions were attributed to local releases of bulk, undispersed PCBs, which produced limited and sharply defined areas of sediment contamination, like those that have been noted by many investigators of PCB distributions in the Acushnet Estuary (Brown and Wagner, 1990).

The other samples did not show such highly deviant compositions. Instead, the ratios of higher PCB homologs (e.g., penta-, hexa-, hepta-, and octachlorobiphenyls) to the lower ones varied at those sites by factors of two to five (Table 6.2-1). This type of blurred local variability in PCB composition is what would be expected for multiple sources of PCBs that were sufficiently well-dispersed to remain suspended for at least a few hours before settling into the sediments, and hence contaminating an area that was at least as large as the amplitude of tidal motion in the estuary. As a result, overlapping of the zones of contamination produced by nearby sources would occur, and the differences in source composition would be blurred, but quite variably.

Thus, local variability in PCB composition supports the multiple source/minimal movement model.

HRP 001 1740

6.2.4 <u>Regional Trends in PCB Levels</u>

Since PCBs are water-insoluble materials that bind strongly to sedimentable organic particles, their release into a river should result in the formation of a deposition wedge, i.e., a contaminant distribution with the heaviest deposition near the source, and progressively lighter deposition downstream.

The number and shape of such deposition wedges are quite different for the two PCB contamination models under consideration. The single source/massive movement hypothesis predicts the presence of only a single, very gently tapering, deposition wedge. The multiple source/minimal movement model predicts a multiplicity of more sharply tapering deposition wedges, one for each source, and with the possibility of overlap between the wedges produced by sources that are near to each other. In addition, within estuarine portions of the Hudson, the deposition pattern should taper off upstream as well as downstream because of tidal movements.

The 1977-78 NYSDEC survey of the Upper Hudson (Tofflemire and Quinn, 1979) showed two deposition maxima (Figure 6.2.4-1). The first was located in River Reaches 6 to 8, and can be attributed to redeposition of Reach 9 sediments during the 1974-77 scouring events that followed the October 1973, removal of the old Fort Edward Dam. The second was located downstream in River Reaches 3 to 4 and can be attributed to contributions from some additional source because of the paucity of the wood chips and sawdust that were so characteristic of the redeposited Reach of 9 sediments.

282

The authors of the report on the 1977-78 NYSDEC survey at first attributed the drop in PCB concentration between the two deposition wedges to the nature of the samples taken in Reach a (the center of the channel). They later state, however, that "A recent tabulation for Reach 5, employing 79 grabs with good distribution across the river, confirmed that the PCB in Reach 5 was significantly lower than for Reaches 6, 7 and 8" (Tofflemire and Quinn, 1979, p. 4).

The 1976 USEPA survey of the Lower Hudson recorded elevated PCB levels for its samples taken near Albany, Saugerties, Foundry Cove, Peekskill, and Piermont. The 1977 Lamont-Doherty survey recorded elevated PCB levels for the samples taken near Albany (same site as EPA's), Germantown, Kingston, Poughkeepsie, Foundry Cove, Peekskill, and New York. Neither the 1977 Lamont-Doherty survey nor the 1981 EPA follow-up survey, however, could confirm the "hot spot" found by EPA near Piermont in 1976, which presumably represented a highly localized PCB release. Neither study, however, can now be considered as providing definitive indications of either the presence or absence of local PCB sources because of the paucity of sampling points.

Less ambiguous was a 1978-81 NYU survey, where resident biota (Gammarus) were collected monthly for four non-winter seasons from fifteen Lower Hudson locations (O'Connor et al., 1982). The results showed consistent elevations in total PCBs for specimens collected near Albany, Kingston, Poughkeepsie, Foundry Cove (Cold Spring), Peekskill (Jones Pt. and Indian Pt.),

283 HRP 001 1742

the Tappan Zee, and New York City Lower Bays as well as considerable variations in Aroclor 1242/1254 ratios (Figures 6.2.4-2 and 6.2.4-3).

The 1988-90 Harza survey sought to avoid the local variation problem by collecting 5 to 8 well-spaced surficial sediment samples near each target site. The resulting average values show a continuation of the temporal decline already noted by USEPA (1982) and Bopp and Simpson (1989), but relative to 1988-89 average values there are still elevated PCB levels in the Troy-Albany, Kingston-Poughkeepsie, Foundry Cove-Peekskill, Stony Point-Haverstraw, and Tappan Zee-NYC stretches of the River (Table 6.2-1).

The results of the four Lower Hudson sediment surveys and the 1977-78 NYSDEC Upper Hudson survey are depicted in Figure 6.2.4-4. Together, they indicate that there were originally several sizeable PCB deposition maxima in the Lower Hudson. The first occurred in the Troy-Albany area and may have been produced by discharges from local sources (see Section 6.4).

Below Albany, between Castleton and Hudson, there is a long, largely rural, stretch of the Hudson River where none of the previously cited investigators, nor the U.S. Army Corps of Engineers (USACOE, 1985), has been able to detect more than minimal PCB deposition, despite the presence of numerous sediment deposition areas. Further south, there appear to have been significant PCB sources near Kingston, Poughkeepsie, Foundry Cove, and Peekskill, and along Haverstraw Bay and the Tappan Zee, all probably associated with diverse industrial activities.

HRP 001 1743

Finally, there was -- and is -- PCB contamination throughout the New York metropolitan area (Fava et al., 1985; Mueller et al., 1982; MacLeod et al., 1981; Strainken and Rollwegen, 1979) and northern Long Island Sound (Turgeon et al., 1989; NOAA, 1988; Rogerson et al., 1985), where PCB use has historically been extensive (see Section 6.4). This probably resulted from earlier concerns over putative fire hazards associated with industrial, utility and railroad installations in urban areas. The fire concerns lead to extensive local preferences for the use of the lower chlorinated PCBs in industrial heat exchangers, hydraulic/lubrication systems, and plasticizers, and of higher chlorinated PCBs in network (i.e., sidewalk vault), substation, and railroad/transit car transformers. All such uses would have resulted in direct or indirect releases to the Hudson River, New York Harbor, or Long Island Sound.

It might be argued that even the NYU and Harza surveys still involved observations that integrated PCB levels over limited areas, and hence that the apparent local maxima seen near various Lower Hudson cities represent local sediment deposition areas rather than local or regional PCB sources. If this were true, however, there would still remain one valid test of the single source/massive movement model -- there should be an overall statistically significant decline in mean PCB level between Troy and Yonkers. As discussed below, there is no such decline.

HRP 001 1744

Bopp et al. (1981) compares sediment PCB levels at selected points (Table III, p. 213) as a basis for an argument that there should be an overall decline in mean PCB levels with river mile between Troy and New York City. Although that study asserts that PCB "concentrations in sediments of the Lower Hudson decrease with distance downstream from the [Troy] dam," it provides no statistical analysis to support this assertion.

When a linear regression of PCB concentrations in the top stratum of the sediment cores collected by Lamont-Doherty (1977) is performed, a statistically significant decline in concentration between Troy and New York City is in fact observed. There are, however, relatively few data points in this analysis.

In contrast to the findings of Bopp et al. (1981), regression analyses of data from the two EPA studies (1976, 1981) and from the Shephard et al. (1990) study all show no statistically significant downstream decline in sediment PCB concentrations between Troy and New York City. The results are shown in Table 6.2.4-1. The absence of a statistically significant decline in sediment PCB concentrations provides powerful evidence that the hypothesized large-scale transport of PCBs from the Fort Edward area downstream throughout the Lower Hudson in fact never occurred.

This conclusion is further supported by the fact that the EPA (1976) survey pre-dates the Lamont-Doherty (1977) survey, and is therefore the survey closest in time to the 1973 breaching of the Fort Edward Dam. If the large-scale downstream PCB transport from Fort Edward to the Hudson estuary actually

HRP 001 1745

occurred, the 1976 EPA survey would be the most likely study to have detected a declining downstream concentration gradient. Moreover, Shephard's (1990) finding of no downstream trend is based on over 100 samples collected from the Lower Hudson, far more than the two EPA (1976, 1981) and Lamont-Doherty (1977) studies combined, resulting in a statistically more powerful test of trends.

Plots of all samples from all four extensive sediment surveys performed in the Lower Hudson (Figure 6.2.4-4) reveal a number of locations with elevated PCB concentrations (Albany, Kingston, Poughkeepsie, Foundry Cove, and several locations in or just upstream of Haverstraw Bay) separated by reaches of comparatively low PCB concentrations. This pattern is indicative of multiple PCB sources in the Hudson estuary, and not of a single upstream PCB source responsible for the majority of contamination of the entire Hudson estuary.

6.2.5 <u>Regional Trends in PCB Composition</u>

The single source/massive movement and multiple source/minimal movement models both make at least partially quantifiable predictions as to both the original composition of the PCB source and the type of changes that would be expected as the PCBs moved downstream. Specifically, the single source model predicts (1) an original composition like that of the material which was translocated from Reach 9 to Reach 8 in 1974-77, and (2) progressive losses of lower congeners due to elutriation and evaporation as the PCBs made their long journey downstream. The multiple-source model predicts that the original composition of

HRP 001 1746

PCBs in the Fort Edward and Mechanicville deposition wedges should resemble those used in capacitor manufacturing during the 1950s and 1960s, while those of the Lower Hudson, where there appears to have been a diversity of mainly industrial uses (see later discussion), should correspond to the national pattern of PCB use. Since only minimal PCB movement along with sediment is hypothesized by this model, only minimal losses of lower congeners (e.g., those observed in the Acushnet Estuary PCB deposits (Brown and Wagner, 1990)) would be predicted.

To put these predictions on a quantitative basis, GE endeavored, first, to estimate the original composition of the PCB mixtures that were redeposited in Reach 8, using as a data source congener-specific PCB analyses of various samples, including all 1" sections of the four "hot spot" cores collected by GE with NYSDEC (Tofflemire's) assistance in 1984 and one archived core that Tofflemire had collected during the original NYSDEC survey in 1977. It was possible to estimate the original composition of the most recent (1977+) deposition (unfortunately, only 1-5 percent of the total) from highly concordant analyses of near-surface samples that had not undergone subsequent dechlorination, and that of the 1976 deposition (about 20 percent of the total) from other highly concordant analyses of the top four 1" sections of the archived (January 1977) core (Brown et al., 1984). To estimate the extent of any compositional differences in PCBs deposited in 1974-75 (about 75 percent of the total), all of which deposits had subsequently undergone extensive dechlorination (Brown et al., 1984, 1987a, 1987b), GE

quantified the sums of two dechlorination reactant-product pairs (i.e., 2356-245 and 2356-25-CB and 2356-2345 and 2356-235-CB) that were selected on the basis of the previously observed resistance of the product congeners to further dechlorination at that site (Brown *et al.*, 1987b). These determinations permitted calculations of the original levels of Aroclor 1254 and 1260 in the redeposited PCBs. This procedure indicated that the original composition of the PCBs scoured from Reach 9 during 1974-77 had been about 95 percent Aroclors 1242 and 1016 (probably including only about 3 percent of the latter), 4.5 percent 1254, and 0.3 percent Aroclors 1260 and 1268 (mostly the former). The results of these analyses are presented as homologs in Table 6.2.5-1.

To predict the original composition of a collection of sources that were sufficiently diverse to reflect the national use pattern, GE added the published Monsanto data on Aroclor sales by year for the 1957-77 period. Table 6.2.5-1 indicates this distribution and the PCB homolog distributions calculated for both the average U.S. 1957-77 PCB usage and the original compositions of the PCBs released into and redeposited from Reach 9. Comparison of the national average versus the Reach 8-9 distributions indicates that major differences occur only among the higher homologs.

In order to estimate the compositional changes that would result from elutriative/evaporative losses during transit, GE used previously reported (Brown and Wagner, 1990) experimental data on the relative rates of PCB congener loss during Aroclor 1242 evaporation. These showed that such losses are very

289

HRP 001 1748

sensitive to degree of chlorination, so that elutriative/ evaporative losses of 16.7 percent and 31.4 percent would result in considerable losses in lower congeners, with concomitant decreases in the dichlorobiphenyl to trichlorobiphenyl ratios from 0.31 to 0.23 and 0.12, respectively, with only minor losses of tetra- or higher chlorobiphenyls.

Comparisons of Tables 6.2-1 and 6.2.5-1 show that the higher homolog levels in the Lower Hudson are, on average, very close to those predicted by the 1957-77 national average PCB usage and considerably above those determined for the Reach 9 releases, even before dechlorination. This increase cannot be attributed to elutriative or evaporative losses of lower congeners, because there is no general decrease in dichlorobiphenyl/trichlorobiphenyl ratios beyond those that might be expected from sediments in place, as was seen in the Acushnet sediments which did not undergo significant transport (see Section 6.2.7). If PCBs were transported in the water column, either on particulate matter or in dissolved phase, over the great distance from the Upper River (Reach 8 or 9) to the Lower River, dichlorobiphenyls would have been nearly eliminated. The dichlorobiphenyls in the Lower River must therefore be from local sources. Moreover, the higher chlorinated homologs are also from local sources, because the Upper River source cannot account for the distribution of higher homologs.

The increase in higher homolog levels in the Lower Hudson could arguably be explained by a combination of the single source/massive movement and multiple source/minimal movement

001 1749

HRP

models; i.e., by postulating that most of the PCBs still came from Fort Edward, but with some additions of Aroclors 1254 and 1260 in the Troy-Albany area or below. This hypothesis, however, would require the identification of upper estuary sources that had much higher averages than the national average in these higher Aroclors and would result in predicted dichlcrobiphenyl levels even lower than those of the original single source/ massive movement model, thus making the data presented in Table 6.2.1 even harder to understand.

Thus, the regional trends in PCB composition support the multiple source/minimal movement model.

6.2.6 <u>Regional Differences in Total PCB Loading</u>

The single source/massive movement model hypothesizes that the Hudson River contains a single PCB deposition wedge, whose heavy end, and hence the bulk of the total PCB loading, is located in the Upper River. Currently, there appears to be no reliable way of estimating the total PCB loadings in upper and lower sections of the Hudson River at a common date; however, there is enough data for an estimate of the ratio between the two loadings.

From the Tofflemire and Quinn (1979) report, one can calculate that in 1977-78 the geometric mean PCB loading in the surficial (*i.e.*, grab-sampled) sediments of Upper Hudson reaches 1 to 9 was about 15 ppm, with an arithmetic mean of about 25 ppm. The arithmetic mean PCB level for the Lower Hudson upper core sections collected by EPA in December 1976 (USEPA, 1977) was 6.34 ppm, and for the upper core sections collected by Lamont-Doherty

291

HRP 001 1750

(mostly in July 1977; Bopp, 1979) was about 8.09 ppm. In all three studies, the sampling was concentrated on deposition areas, but the levels determined must be similarly related to those of the river bottom as a whole. However, the total area of contaminated Upper Hudson riverbottom is 5.6 square miles, as contrasted to 129 square miles for the Lower Hudson. Thus, for argument's sake, even taking the higher of the Upper Hudson averages, *i.e.*, the arithmetic mean, and the lower average for the Lower Hudson, a comparison of 25 x 5.6 (or 140) vs. 6.3 x 129 (or 812.7) indicates that in 1977 there must have been 5.8 times as much PCB in the Lower River as in the Upper. Even if the average concentration used for the Lower Hudson is higher, it is clear that the load in the Lower River in 1977 was already much greater than in the Upper River.

It could also be argued in opposition to the above analysis of relative PCB loads that the average deposition depth in the Lower Hudson was less than in the Upper, and that the sediment depositional "hot spots" were proportionately more extensive in the Upper Hudson than in the Lower. However, the former alternative would appear contradicted by Lamont-Doherty's 1977 Lower River coring data (Bopp et al., 1979), which showed PCB penetrations at least as great as those of the Upper River. The latter argument conflicts with the long-standing NYSDEC conclusion that most of the Upper River PCBs were concentrated in "hot spots" covering only a small fraction of the total riverbottom (Tofflemire and Quinn, 1979). Thus, the available data would indeed seem to indicate that in 1977 there was already

292 HRP 001 1751

several times as much PCB in the Lower River as in the Upper. This too supports the multiple source/minimal movement model.

6.2.7 <u>PCB Movements in Other Estuaries</u>

A key feature of the single-source model is the assumption of highly effective PCB transport processes (e.g., cycles of PCB-bearing particle suspension, downstream movement, redeposition or PCB desorption, downstream movement, and reabsorption) to account for the postulated massive long-distance movement of PCB (Bopp, 1979). The presence of such transport processes has been questioned. Independent researchers have observed that any downstream sediment movement in the Hudson is modest and most movement is oscillatory because of the tidal nature of the river (Bakunowicz, 1980).

If such processes were operating in the Hudson estuary, they should have been operating in other estuaries as well. The heavily studied PCB-contaminated Acushnet River, at New Bedford, Massachusetts, is an excellent site to examine on this point. Within the Acushnet Estuary, PCB movements are much more easily defined than in the Hudson, because (1) there was only one major and one minor PCB source involved, rather than a multiplicity; (2) the PCBs were released from the major source in undispersed form, thereby giving sharply defined and compositionally distinguishable areas of primary deposition; and (3) the upper estuary PCBs exhibit extensive dechlorination through the sediment surface and into the water column, hence permitting a tracking of their downstream movements past the undechlorinated lower estuary PCBs.

HRP 001 1752

Review of these compositional differences shows that in the Acushnet Estuary, despite the presence of much sharper PCB concentration gradients than in the Hudson and much higher PCB levels in the water column, no significant PCB movements have occurred either between upper estuary sediment patches or between upper and lower estuary sediments (Brown and Wagner, 1990). This means that in the Acushnet there was neither significant downstream movement of sediments from the heavily contaminated upper estuary to the lightly contaminated lower estuary nor significant adsorption of upper estuary PCBs from the water column by the lower estuary sediments.

If none of the frequently modeled transport processes was occurring under the seemingly favorable conditions presented by the Acushnet Estuary, it is difficult to see how any could be operating on the hypothesized massive scale in the Hudson.

* * *

In summary, GE has examined seven categories of data for compatibility with the predictions of the two previously proposed models for Hudson River PCB contamination. One of these data sets, namely that related to local variabilities in sediment PCB levels, appears equally compatible with the predictions of either model. The other six data sets are all compatible with the predictions of the multiple source/minimal movement model but show various degrees of incompatibility with the single source/massive movement model. Particularly severe problems for the latter model are presented by the data on the relationships

294

between sediment PCB levels and river miles and on the dating of the lower Hudson PCB deposits.

The alternative multiple source/minimal movement model, which was tentatively proposed by two of the three original groups of investigators (USEPA, 1977; O'Connor et al., 1982), is the only one that is compatible with the entirety of the available data. This model recognizes that: (1) PCBs were widely used materials during the 1950s and 1960s, particularly in the Hudson Valley and New York metropolitan area (at the 1971 peak, Monsanto had some 3,000 customers, including distributors, for its Aroclor product line); (2) many of these uses led to environmental releases; and (3) such releases led to contamination of nearby riverbottom sediments.

The contamination of Upper Hudson Reaches 5 to 8 that occurred in 1974-77 as a result of the Fort Edward Dam removal has attracted much attention as the largest documented PCB contamination event. However, based on available sediment data it is possible that the dam removal resulted in contamination extending no more than 25 to 30 miles downstream. Further downstream, the Hoosic/Mechanicville and Troy/Albany PCB sources produced much shorter deposition wedges. PCBs leached from any of these deposition wedges into the water column could, of course, have continued to move with the water to the Atlantic. However, in neither the Hudson nor the Acushnet Estuaries is there any evidence that PCBs once extracted into the water column can return to the sediments. Instead, the downstream sediment

HRP 001 1754

deposits, like those of the Upper River, must be attributed to local sources.

EPA appears ready in its Phase 1 Report to adopt the conclusions of the single source/massive movement model without question. Careful analysis of sediment data shows the error in that approach.

6.3 PCBs From The Upper River Account, At Most, For Only A Small Fraction Of The PCBs Accumulated By Lower River Fish

Proponents of the single-source model also offer data on PCB contamination in fish, particularly striped bass, to support that model. A thorough analysis of these data, however, also supports the existence of multiple PCB sources and local impact.

The data show either no gradient in PCB concentrations in striped bass moving downriver or, if there is a gradient, it is due to residence time of the striped bass in the River and not higher PCB concentrations in the surrounding environment, *i.e.* sediment and water. Furthermore, the data in the Phase 1 Report clearly demonstrate that there is no such gradient for resident fish species. In fact, some species have higher PCB levels further downriver than they have in the Albany-Troy area.

6.3.1 <u>PCB Concentrations in Lower River Fish</u>

HRP

001

1755

In the Phase 1 Report, EPA implies the existence of a gradient in striped bass PCB concentrations by contrasting averages for Upper versus Lower Estuary fish (p. A.3-10). Recently, NYSDEC expressly advocated the existence of the gradient when it released a report entitled <u>PCB in Striped Bass</u>

from New York Marine Waters (September 1991). NYSDEC's press release accompanying that report attributes the gradient to the massive movement of PCBs from a single source in the Upper River.

Analysis of the gradient argument requires a review of the available data to determine whether a significant gradient in fact exists and, if so, the causes for the gradient. The more recent striped bass data available for analysis is contained in Table 20 of NYSDEC's 1991 report (reproduced as Table 6.3.1-1). The State bases its gradient hypothesis on the average concentrations calculated at six locations. The two northernmost locations (Albany/Troy and Catskills) cannot be used as comparable to the other four, however, because the samples were collected at later times in the year than at the four locations lower on the River. The date of collection is extremely material because, as the NYSDEC report acknowledges, PCB levels in fish increase during the summer months when compared to the spring. For the four remaining locations, there appears to be a slight increase in PCB levels as fish move north, but the data is unclear because of the wide range of concentrations at any one site. For example, some fish caught at the Tappan Zee Bridge have higher levels than those caught at Croton Point or Poughkeepsie.

To the extent a gradient in striped bass PCB levels exists, NYSDEC's acknowledgement of seasonal differences points at a cause of the gradient that is probably more significant than differences in PCB concentrations in sediments. The simple fact is that the Hudson, like many bodies of water, contains PCBs

HRP 001 1756

throughout the system. Logically, therefore, a migratory fish such as striped bass that spends more time in the River will have accumulated more PCBs. It is also logical that striped bass that are farther north in the River will have had longer residence times. The consequence is that migratory fish further north in the River will in general have higher PCB levels as a function of residence time rather than as a function of higher ambient PCB concentrations as they move north.

A much clearer test of whether there is a gradient in Hudson River fish due to changes in sediment concentrations would be to examine PCB concentrations in resident species where migration is not a confounding factor. Unfortunately, the Phase 1 Report provides little data on these fish. The data that are provided, however, clearly demonstrates that there is no gradient. Table B.3-16 of the Report shows that largemouth bass at River mile 153 to 155 had average PCB concentrations of 2.0 and 3.6 ppm in 1987 and 1988 respectively. By contrast, Table A.3-7 shows that for the same resident species at River mile 112, the average concentrations were 11.1 and 5.9 ppm for 1986 and 1988. Simply stated, fish that reside more than forty miles farther downstream had PCB concentrations that are 2 to 5 times higher than those upstream. Resident fish data provided in the Phase 1 Report contradict the single source model and support the model that there are multiple PCB sources in the Lower River with HRP local impacts on fish.

100

6.3.2 <u>Migration Patterns of Striped Bass</u>

To assess and quantify contamination sources of PCBs to a migratory species such as striped bass, knowledge of the species life history, including its migration and feeding habits, is essential. This fact is implicitly recognized in the Phase 1 Report (p. A.3-9), which states that "[b]ecause striped bass were caught during spring migration, the location at which they were caught probably bears little or no relationship to the PCBs in the sediment and water at that location." Further, the Report cites with apparent approval Thomann's estimate that "the Upper Hudson load contributes only 10 percent to PCB levels in striped bass" (p. A.4-9). For the reasons set forth below, GE believes the correct percentage is far lower. Unfortunately, the Phase 1 Report provides few details of either striped bass life history or of PCB concentrations and composition in the striped bass outside of the Marine District. These omissions prevent EPA from making a complete assessment of the sources of PCBs in striped bass and other Lower River fish. The following comments provide some of that missing but necessary information.

Migratory patterns indicate that once Hudson River striped bass reach sexual maturity, the large majority of their life (8 to 10 months each year) is spent outside of the Hudson estuary where they cannot accumulate PCBs from the Upper Hudson.

Numerous studies (Merriman 1941, Raney et al. 1954, Clark 1968, McLaren et al. 1981, Waldman et al. 1990) have recognized that the Hudson River stocks of striped bass are migratory. This point is indirectly referred to at several

HRP 001 1758

locations in the Phase 1 Report (e.g., pp. A.3-9, A.3-11, A.4-9), but the migratory patterns of striped bass are not described in detail anywhere in the Report. Information on striped bass movements has been determined largely through mark-recapture studies. Although the individual studies vary in the number of fish studied and level of detail in their description of migratory patterns, the general migratory pattern has been consistently demonstrated by the various studies.

The most recent study of striped bass migration (Waldman et al., 1990) has expanded the known coastal range of migrating Hudson River striped bass previously identified by Clark (1968) and McLaren et al. (1981). The known range now extends between Nova Scotia and North Carolina. This more expansive range is due not to an actual expansion of the range, but rather to the availability of a vastly greater amount of information derived from large numbers of fish tagged compared to earlier studies such as Clark (1968), with a concomitant increase in recaptures, providing more detailed information about migrations. Waldman et al. (1990) also concluded that striped bass migrate farther and farther from the Hudson as the fish grow older and larger.

Hudson River striped bass stocks spawn in the middle reaches of the Hudson estuary, upstream of the salt wedge. Spawning activity ranges from Croton Point to Coxsackie (Hoff et H al., 1988) but appears to be concentrated in the West Point to Newburgh reach of the River (McLaren et al., 1981). Peak spawning usually occurs in mid-May when the water temperature is 175

14°C (Klauda et al., 1980), but can occur anywhere from late April to early June.

Hudson River striped bass spend their first two years of life in the lower reaches of the Lower Hudson or New York Harbor, with the young-of-year generally heading downstream after hatch. Boreman and Klauda (1988) observed that juvenile striped bass approximately two months old were most commonly found in the upper half of Haverstraw Bay, considerably downstream from the peak spawning grounds. Young-of-year striped bass overwinter in the New York metropolitan area. McLaren et al. (1981) observed that immature striped bass in the Hudson estuary moved downstream starting in April, at the same time that mature striped bass are moving upstream to spawn. Table 3 (p. 914) of Waldman et al. (1990) states that 65 percent of all recaptures of fish larger than 200 mm (Age I+ and older, McLaren et al., 1981) were from outside the Hudson, despite the fact that all were tagged in the Hudson. This finding is contrary to the assertion by Thomann et al. (1989) that Age I-II striped bass remain in the Hudson estuary year-round.

After spawning, the vast majority of the spawning striped bass spend 8 to 10 months outside of the Hudson River estuary before reentering to spawn again. Beginning in their third year, Hudson River striped bass leave the Hudson River and generally move northeasterly into Long Island Sound. Some move south into the New York Bight and along the New Jersey shoreline. Striped bass that have moved into Long Island Sound have been found as far east as Rhode Island, Cape Cod, Massachusetts and

HRP 001 1760

Nova Scotia, where they have been captured as late as November (Waldman et al., 1990). Some adult striped bass are known to remain throughout the entire length of the Hudson estuary (New York City to Troy) during the summer. However, by December, the majority of the Hudson River striped bass stock can be found in the New York City metropolitan area. The fish reside in the New York metropolitan area through March, after which they begin their migration upstream to the mid-Hudson spawning grounds and repeat the cycle.

Waldman et al. (1990) is based on a review of data collected in a study by Normandeau Associates, Inc., together with the Hudson River Foundation and others. Since the publication of Waldman et al. (1990) describing that study, additional data has been gathered raising the number of tagged fish considered to over 93,000. Although analysis of this data is preliminary and has not been published, it provides important details regarding the migrations of Hudson River striped bass.

The analysis reveals large-scale migrations between New Jersey and Massachusetts by fish 450 mm (about 17-3/4 inches) or longer. In April and May, the period during which spawning occurs in the Hudson River, the striped bass population is concentrated along the New Jersey coast (52 percent of the total population) and in the Hudson River (38 percent of the total population). Small portions of the population are also located off the coasts of Connecticut (6 percent) and Massachusetts (3 percent).

HRP 001 1761

In June and July, striped bass have migrated from the Hudson and the New Jersey coast and are concentrated in New York Harbor (39 percent) and along the Connecticut coast in Long Island Sound (42 percent). Most of the remaining population is split, located off the coast of Massachusetts (9 percent) and off Long Island (9 percent).

As the summer continues, the striped bass population is concentrated further north and east along the New England coastline. In August and September, over half of the population is off Massachusetts (56 percent), while much of the remainder is either off the coast of Connecticut (22 percent) or off the shores of Long Island (8 percent). The Hudson River contains 11 percent of the population during this time.

In October and November, striped bass populations are widely dispersed. The largest proportion of the population is located off the shores of Long Island (43 percent). An additional 29 percent of the population is found off Massachusetts, while 21 percent are found further south off of New Jersey.

Prior to the spawning run, in the winter months of December through March, the striped bass population is concentrated in New York Harbor (69 percent) and off the New Jersey coast (29 percent).

In summary, the latest data and analysis show that only a fraction of the striped population spawn in the Hudson and that for adult fish the residence time is usually less than 2 months.

HRP 001 1762

6.3.3 Feeding Habits of Striped Bass

The Phase 1 Report correctly recognizes that "[t]he main avenue of PCB accumulation in fish is via consumption of food containing PCBs" (p. A.4-7). Laboratory studies (Pizza and O'Connor, 1983; O'Connor, 1984) as well as modelling studies (Thomann, 1989) confirm that most striped bass PCB bioaccumulation is through their diet, as opposed to direct bioconcentration from the water column. Critical to determining how Hudson striped bass accumulate their PCBs, therefore, is an understanding of their feeding habits.

As poikilotherms (cold-blooded animals), striped bass have metabolism and growth rates that are greatest during the warmest periods of the year (late spring, summer and early autumn), correlating almost precisely with the months spent outside of the Hudson by members of the spawning population. During this season of maximum metabolic rate and growth, striped bass feeding can also be assumed to be at a maximum. By contrast, Clark (1968) has confirmed that Hudson River striped bass are relatively inactive during their overwintering in New York Harbor. And although striped bass do feed during their spring spawning run in the Hudson River, they feed very little. Gardinier and Hoff (1982) provided evidence that only 17 percent of the fish captured immediately prior to or during the spawning run actually had food in their stomachs. This feeding is consistent with the feeding behavior of Chesapeake Bay striped $\underline{\tt H}$ bass stocks, which cease feeding for a short period before, as well as during, spawning (Trent and Hassler, 1966).

To confirm that striped bass are getting large quantities of more heavily chlorinated PCBs such as Aroclor 1254 from their food, several pieces of information are required, including the diet of striped bass and the PCB concentrations and composition of striped bass food items. Several studies (Merriman, 1941; Schaefer, 1970; Gardinier and Hoff, 1982; Hjorth, 1988) have reviewed the feeding habits of striped bass. Larval fish and young-of-year fish generally feed on zooplankton such as copepods, cladocerans and gammarus (Hjorth, 1988; Gardinier and Hoff, 1982). Age I and II fish become increasingly piscivorous, feeding on a wide variety of species. Large adult fish can be described as generalist feeders, feeding on species as diverse as Gammarus, shrimp, lady crabs in the soft shell stage of development, small forage fish such as silversides, mummichogs and anchovies, and larger fish. Gardinier and Hoff (1982) indicate that adult striped bass prefer to feed on soft-rayed species of fish, a conclusion that can also be reached by examination of data tables in Merriman (1941) and Schaefer (1970).

A group of fish identified as preferred prey items for large striped bass in three studies (Merriman, 1941; Schaefer, 1970; Gardinier and Hoff, 1982) are members of the family <u>Clupeidae</u> (herrings), including the Atlantic menhaden, the dominant prey item found by Merriman (1941) and blueback herring, one of the dominant prey items identified by Gardinier and Hoff (1982). These findings are consistent with the conclusion of Gardinier and Hoff (1982) that adult striped bass prefer to feed

305

HRP 001 1764

on soft-rayed fish species. Data collected by Spagnoli and Skinner (1977) indicated that the PCB burden of Atlantic menhaden and blueback herring collected during the early and mid-1970s was predominantly Aroclor 1254.

Both Atlantic menhaden and blueback herring are found throughout the migratory range of striped bass (Smith, 1985). Blueback herring are anadromous, spawning in fresh water streams while spending the rest of the year either in estuaries or offshore. Atlantic menhaden are coastal marine fish, spawning offshore, then moving into estuaries to feed during summer months (Smith, 1985). The fact that these two species are migratory, moving between offshore areas and PCB contaminated rivers and estuaries, combined with their importance to the striped bass diet makes them a vector of PCB contamination to the striped bass. Forage fish such as Atlantic menhaden could pick up their body burdens in coastal areas or rivers contaminated with Aroclor 1254, then pass on their body burden to striped bass feeding on them.

Evidence indicates that coastal areas along the entire migration path of Hudson River striped bass are contaminated with Aroclor 1254. Brown and Wagner (1990) have documented the massive Aroclor 1254 and Aroclor 1242 contamination of the Acushnet estuary (New Bedford, Massachusetts). Battelle Ocean Sciences (1990) has observed that the principal PCB congener in $_{
m in}$ F mussels collected during the mid-to late-1980s throughout Long [00] Island Sound as part of the Administration's (NOAA) National Status and Trends program is a

hexachlorobiphenyl (IUPAC No. 153, 2,2',4,4',5,5'), which is indicative of Aroclor 1254 and/or Aroclor 1260 (Schulz et al., 1989). This particular hexachlorobiphenyl is absent from Aroclor 1016, and, depending on the standard analyzed, is either only a minor component (0.68 percent by weight, Schulz et al., 1989) or is entirely absent (Erickson, 1986) from Aroclor 1242. This information lends further credence to the belief that one reason for the preponderance of Aroclor 1254 in Hudson River striped bass (p. A.3-11) is PCB bioaccumulation outside of the Hudson River estuary.

The most recent study of the PCB composition of striped bass food organisms by Shephard et al. (1990) confirms that for food organisms such as Gammarus, mummichogs, Atlantic silversides and Atlantic menhaden captured from New York Harbor and Long Island Sound Aroclor 1254 is the predominant Aroclor. This finding is consistent with the belief that striped bass bioaccumulate most of their PCB body burden from their food, most of which is ingested outside of the Hudson River estuary.

6.3.4 Composition of PCBs and Other Contaminants in Striped Bass

The locations where striped bass bioaccumulate PCBs can also be inferred simply by looking at the composition of the PCBs -- as well as other contaminants -- in striped bass tissue. This method is tacitly approved in the Phase 1 Report, which states that the domination of highly chlorinated PCBs in Lower River striped bass is "of significant interest, because sediment data for the Lower Hudson suggest that there are sources of highly

HRP 001 1766

chlorinated PCB mixtures from the New York City metropolitan area" (p. E-6).

PCBs found in Hudson River striped bass do not resemble those found in Hudson River sediments. Relative to the composition of Hudson River sediments, Hudson River striped bass have a high ratio of Aroclor 1254 to Aroclors 1242 and 1016; moreover, that ratio is continuing to increase (p. A.3-11; Sloan, 1988). The presence of Aroclor 1254 in Hudson River striped bass at concentrations in excess of the U.S. Food and Drug Administration action level (then 5.0 ppm) was found in fish collected as early as 1970 (Spagnoli and Skinner, 1977). As cited with apparent approval in the Phase 1 Report (p. A.3-11), Aroclor 1254 is now the determinant for the fate of PCB in Hudson River striped bass. Further, the Phase 1 Report cites several studies indicating the increasing proportion of Aroclor 1254 in the lower portions of the Hudson estuary and New York Harbor.

No indication is given in the Phase 1 Report of the Aroclor concentrations or composition of biota or the environment in Long Island Sound or the other locations where migratory Hudson River striped bass stocks are found during much of the year. However, the recent study by Shephard *et al.* (1990) provides additional insight regarding the source of PCB uptake by Hudson River striped bass. That study collected sediment, benthic invertebrates, mussels, forage fish and predatory fish species, including striped bass, from 96 locations in the Lower Hy Hudson, New York Harbor and western Long Island Sound. The results of this study confirmed the findings of Sloan (1988)

308

regarding the predominance of Aroclor 1254 in striped bass. Just as significant, the Shephard study also found an increasing proportion of Aroclor 1254 relative to Aroclor 1242 in both sediments and biota with distance downstream from the Troy Dam. Samples from New York Harbor contained a greater proportion of Aroclor 1254 than samples from the Lower Hudson, while samples from Long Island Sound, in turn, contained a still greater proportion of Aroclor 1254 than did samples from either the Lower Hudson or New York Harbor.

The origin of Aroclor 1254 in the striped bass can also be determined by looking at other contaminants with similar solubilities, sorption tendencies, and stabilities. The chlorinated pesticides chlordane and DDD/DDE are present in Hudson River striped bass, as well as in the sediments of a number of locations in Long Island Sound and New York Harbor. These pesticides are present only at very low concentrations in Hudson River estuary biota and sediments relative to their concentration in the Sound and Harbor (Shephard et al., 1990).

In sum, the foregoing discussion of fish data shows:

- Because they migrate, striped bass are not an appropriate species to demonstrate the distribution of PCBs in the Lower River sediments;
- The habits of striped bass and the type of PCBs found in them prove that Hudson River sources are not even a main contributor to striped bass PCB body burdens;
- Resident fish data disprove the existence of an Upper to Lower River PCB concentration gradient; and
- The only relevant fish data support the multiple source/minimal movement model.

9 HRP 001 1768

6.4 EPA's Investigation And Estimation Of The Contribution Of Other PCB Sources Has Been Grossly Insufficient

Presumably because of its early acceptance of the single source/massive movement theory to explain Lower River PCB loadings, EPA has thus far neglected to look for other sources. The Agency acknowledges that contributions of Lower River sources are presently "poorly identified and quantified," and that what discussion the Phase 1 Report does contain regarding these sources is based on minimal data (pp. E-5, A.2-3 to A.2-6). However, EPA's efforts to identify other sources, ordinarily a detailed process, have been superficial at best.

The comments in Sections 6.2 and 6.3 demonstrate the very real and extensive existence of these sources. The categories of sources discussed in this subsection track those contained in the Phase 1 Report at Section A.2. The locations of many facilities and other sources are specifically described. Where they are not, publicly available documents that identify hundreds of actual and potential discharges are cited.

6.4.1 <u>Industrial Discharges</u>

In searching for industrial facilities that are potential Lower River PCB sources, EPA looks only at New York facilities that currently hold discharge permits under the State Pollutant Discharge Elimination System (SPDES) (p. A.2-6). The Agency identifies only five such facilities and makes no estimates of the volume of PCBs that is, or may in the past have m been, discharged from them. The Report further gives no indication that, other than reviewing the State of New York's

310

list of permittees, EPA investigated those dischargers in any way to determine the volume or nature of their PCB discharges, or that it intends to do any investigation in the future. The Phase 1 Report also acknowledges that, in addition to discharges identified in SPDES permits, there may have been accidental spills or illegal dumping that contributed to Lower River PCB loadings, but simply says "the extent and total PCB loadings of these releases . . . remain unknown" (p. A.2-6). Again, investigative steps necessary to assess the significance of these discharges are not mentioned.

As noted in a 1976 report prepared for EPA by Versar, Inc., although PCBs have been used primarily in electrical applications, which are "closed," a rapid growth in "open-end" and "nominally closed" applications occurred during the 1950s and 1960s (Versar, 1976). In 1971, Monsanto Industrial Chemicals Co., the supplier of approximately 99 percent of PCBs in the United States, voluntarily restricted its sales to closed applications because, with other applications, "entries of PCBs to the environment are more probable and PCB emissions are uncontrollable" (Versar, 1976, p. 204). Prior to this restriction, however, as much as 26 percent of PCBs in the United States were used in "open-end" applications, with an additional 13 percent used in "nominally closed" applications, for a total of 39 percent. (Versar, 1976, p. 204; Table 6.4.1-1). Versar estimated that over 172 million pounds of PCBs were released into the environment through 1974. In 1970 alone, the year prior to Monsanto's restriction of sales, over 15 million pounds are

HRP 001 1770

estimated to have been released. By contrast, in the three years following the restriction, average annual releases dropped by almost 90 percent to less than 1.7 million pounds (Table 6.2.1-2).

Examples of open-end and nominally closed PCB applications include heat transfer fluids, hydraulics/lubricants (e.g., hydraulic fluids, vacuum pumps, and gas-transmission turbines), plasticizers (e.g., rubbers, synthetic resins, carbonless paper), miscellaneous industrial uses (e.g., surface coating, adhesives, wax extenders, dedusting agents, inks, cutting oils, and pesticide extenders), and even petroleum additives (Versar, 1976, p. 204; Table 6.4.1-1). Minimum, average, and maximum concentrations of PCBs in the water effluent of twelve industries within just one industrial category addressed in the Versar report, "Machinery & Mechanical Products Manufacturing," are provided in Table 6.4.1-2. As shown by this table, average concentrations within a particular industry's effluent could be as high as 28 ppm, with maximum concentrations up to 225 ppm.

Less obvious sources of PCBs also play a significant role in environmental contamination. For example, PCBs originally in carbonless carbon paper are believed to be a major source of contamination of effluents from the secondary fiber recovery (i.e., paper recycling) industry (Versar, 1976, pp. 19-20). Paper mills that used recycled paper as a source of fiber are yet other potential sources (NYSDEC, 1976).

HRP 001 1771

Moreover, PCBs are inadvertently produced. A common form of this production results from chlorination of biphenyl in wastewater during treatment. At the time of Versar's 1976 report, U.S. industry used approximately 50 million pounds of biphenyl each year. At least half of this was used in the dyeing of synthetic fibers, where much of the biphenyl leaves the process as waste (Versar, 1976, p. 20). Accordingly, the report states specifically that "[f]urther investigation of biphenyl chlorination as a possible source of PCBs is recommended" (Versar, 1976, pp. 20-21).

Literally hundreds of facilities in the Upper and Lower Hudson watershed now conduct, or in the past conducted, the very operations identified above as likely sources of PCB contamination (P. Moskowitz et al. (1977) (listing approximately 220 industrial direct dischargers and over 200 indirect dischargers in the Lower Hudson Drainage Basin)). Most, in fact, employed open-end or nominally closed applications, where releases to the environment were far more prevalent.

Perhaps even more telling, Monsanto sales data reveal that, in 1971 and 1972 alone, over 3 million pounds of PCBs were sold to users on or near the Lower Hudson. Extrapolating these numbers to all years in which PCBs have been used in the United States -- during which over 1.5 billion pounds were sold -indicates that tens of millions of pounds were likely used on or near the Lower Hudson or its major tributaries. The fact that these facilities may not, today, have SPDES permits for discharges is virtually meaningless; most of these facilities

HRP 001 1772

ceased using PCBs in or around 1971 (Versar, 1976), before the SPDES permitting system came into existence. The absence of a SPDES permit, therefore, is certainly a poor reason to overlook these facilities as potential sources.

Moreover, particularly through the 1960s, many users of PCBs followed accepted disposal practices and simply landfilled their PCBs on or near their facilities, where PCBs may continue to leach into nearby waterways for many years (Versar, 1976). Even as late as 1976, approximately 12 million pounds of PCBs were landfilled (Versar, 1976, p. 8). Although not mentioned in the Phase 1 Report, the federal government itself appears to have followed such practices along the Hudson at its arsenal in Watervliet. That site is a well-known past as well as present source of PCB releases (NYSDEC, 1991, Site No. 401034).

Despite this knowledge, EPA has ignored its own guidance documents and failed to take virtually all of the many actions called for in investigating potential sources in the Lower Hudson (e.g., Potentially Responsible Party [PRP] Search Manual, Final Report, OSWER Dir. 9834.3-01a (Aug. 1987); PRP Search Supplemental Guidance for Sites in the Superfund Remedial Program, OSWER Dir. 9834.3-2a (June 1989)). Indeed, the Agency has apparently ignored readily available studies done by other entities identifying numerous additional sources and potential sources (e.g., NYSDEC (1991); P. Moskowitz et al. (1977); S. Rohmann et al. (1977)). PRP search procedures are required not simply to find parties able to conduct or pay for response of measures, but also because they are essential in understanding ...

314
the site contamination and the best way to clean it up. (USEPA, 1987; 42 U.S.C. § 9604(e)(2)(A), (B)). In fact, EPA has instructed that PRP searches should be started immediately after a release or threat of release is detected, and should be <u>completed</u> "well before" the RI/FS is even begun. (USEPA, 1987, p. 3). Accordingly, EPA should immediately search for other Upper and Lower River PCB sources.

6.4.2 <u>Sewage discharges</u>

The Phase 1 Report implies that the upper range of Lower River PCBs from sewage effluent discharges sources is 4.6 lb/day (pp. A.2-3 to 2-4). However, this estimate is derived solely from loadings from the New York City metropolitan area. Again, EPA has ignored information currently available to it and has failed to take steps necessary to gather evidence that certainly exists concerning other sources.

The Lower Hudson and its major tributaries receive direct discharges from over twenty municipal treatment systems with multiple on-line industrial dischargers. Although these facilities are not the only treatment plants that are potential PCBs sources, they certainly are a critical starting point in understanding past and present PCB loadings. In Albany County, for example, two treatment plants not even mentioned by EPA are known to have discharged Aroclor 1254 at a rate of 1.37 lb/day (NYSDEC, 1976). These samples were taken in late September 1975, when flow conditions would be expected to be low, and after most "open-end" and "nominally closed" uses of PCBs in the area had presumably ceased and the plants had likely taken steps to remove

HRP 001 1774

before discharging any PCBs that it did receive. Thus, these samples almost certainly underrepresent earlier discharges from these plants.

The Albany treatment plants alone had twenty on-line industrial dischargers, twelve of which are believed to have had no industrial pretreatment whatsoever (Moskowitz et al., 1977). Other municipal treatment facilities had even more on-line dischargers -- e.g., Newburgh (63) and Poughkeepsie (67) -- with no required pretreatment.

In short, because (1) literally millions of pounds of PCBs were used by industries on or near the Lower Hudson in just the two-year period for which Monsanto sales records are available; (2) these industries generally employed open-end and nominally closed applications of the PCBs; and (3) these users, for the most part, were able to discharge to treatment plants with no industrial pretreatment, the conclusion that such discharges are critical to a full understanding of Lower River contamination is inescapable.

6.4.3 <u>Tributaries</u>

The Phase 1 Report acknowledges that "[e]stimates of PCB loadings from tributaries to the Lower Hudson can all be characterized as poor" (p. A.2-4). Indeed, there are "essentially no measurements of PCB concentrations in the tributary flow" (p. A.2-4). The published estimates that do exist are based on measurements of flow and suspended matter, not H PCBs. Although there are "essentially no measurements" of PCBs in the tributary flow, some do exist, and more should be made.

As noted in Section 6.2.4, sediment samples taken near the mouth of the Hoosic River strongly indicate that the Hoosic River in Reach and contains a significant PCB source. Sampling from the Hoosic River shows PCB concentrations as high as 70 ppm (S. Rohmann et al., 1987). Those PCBs can be attributed to any of several known activities -- including capacitor manufacturing -- along the banks of the Hoosic and its tributaries, where over a million pounds of PCBs were purchased in 1971 alone.

The Phase 1 Report itself identifies present dischargers into the Mohawk and Kinderhook Rivers (p. A.2-6). The many sewage treatment plants and industrial sources along the Mohawk River (P. Moskowitz et al., 1977), as well as samples taken from those waters as late as 1983 (S. Rohmann et al., 1987) establish the Mohawk as an almost certain major past and continuing source of Lower River PCBs. Sediment samples of 4,350 ppm at a Chatham, New York gas pipeline station adjacent to the Kinderhook further implicate that tributary as a potential PCB source (NYSDEC, 1991, Site No. 411006).

Lagoon sludge samples and ground water samples of 225 ppm and 1.4 ppb, respectively, taken near the Kromma Kill, and sludge and surface water samples of 1,016 ppm and 0.103 ppb, respectively, taken in or near the Rondout Creek, indicate those tributaries as likely sources (NYSDEC, 1991, Site Nos. 401003, 356014).

Finally, 1975 sampling by NYSDEC also shows PCBs within the Roeliff, the Jansen Kill, and other tributaries (NYSDEC, 1976).

The above handful of sample results are certainly not sufficient to form a reasonable estimate of the volume or nature of Lower River PCBs coming from tributaries. However, they do provide sufficient information to show there is a potential for these sources to contribute significantly to the current or historic PCB load. As EPA implicitly acknowledges, the PCB measurements have been too few to form any conclusions regarding the magnitude of the tributaries as a source of PCBs. The above sample results and references do, however, establish that further study is required before EPA's conclusion that tributaries together currently contribute in the range of 0.2 to 2.3 lb/day can be accepted as a basis for selecting a remedy in this case.

6.4.4 Landfill leachates

The Phase 1 Report again notes with candor that its present estimate of Lower River PCB loadings from leachate is "based on a minimal number or measurements and on a simple model of leachate transport" (p. A.2-5). However, despite this lack of data and the enormity of the area from which landfill leachate might flow to the Lower Hudson (estimated by EPA to be between 2,000 and 3,000 acres), EPA is apparently ready to conclude that less than 0.3 lb/day (and possibly as little as 0 lb/day) of PCBs flow from these sources to the Lower Hudson.

Documents available from NYSDEC and other entities show that EPA's estimate is entirely premature. Numerous landfills that are immediately adjacent to the Lower Hudson and its tributaries will, until remediated, continue to release and threaten to release PCBs directly or indirectly into the Lower

318

Hudson. NYSDEC documents identify three separate facilities in Watervliet, New York with significant surface contamination (NYSDEC, 1991, Site Nos. 401003, 401032, 401034). Another such landfill is located on land currently used by a Poughkeepsie medical facility and possibly owned and operated in part by the State of New York. It reportedly has Aroclor 1260 contamination as high as 1,700 ppm. This landfill, possibly operated as an uncontrolled disposed facility in the 1960s, is reportedly in a low, wet area with a stream running directly to the Hudson (NYSDEC, 1991; NYSDEC Phase 2 Investigation, Site No. 314063). Another site on the Lower River is Harbor at Hastings described by NYSDEC as having soil contamination up to 100 ppm and "fill material extending into the Hudson River" (NYSDEC, 1991, Site No. 360022).

Finally, highly likely sources of past and ongoing PCB contamination to the Lower Hudson through landfills and other rural runoff are rural roads (upon which PCBs have historically been used for dust control) (Versar, 1976), railroad tracks, and gasoline pipeline gate stations. An example of railroad runoff is the Harmon Railroad Yard (NYSDEC, 1991, Site Nos. 360010, 360019).

6.4.5 Storm Water and Combined Sewer/Storm Water Outfalls

With regard to storm water and combined sewer/storm water outfalls, the Phase 1 Report again implicitly acknowledges that meaningful data do not yet exist. The Report recites the 2-3 lb/day estimates by Thomann (1989) and Mueller (1982), but

HRP 001 1778

also notes that these estimates "are based on modeling efforts with relatively little field data" (p. A.2-4).

EPA's apparent response to this lack of data is not to gather it through the many tools at its disposal, but instead to select uncorroborated and conservative numbers to support the "single source/massive movement" model set forth above.

6.4.6 <u>Atmospheric Deposition</u>

Atmospheric deposition is an important land-to-water pathway. EPA's Phase 1 Report, however, analyzes this pathway in a very superficial manner.

Air deposition processes consist of wet, particle-dry, and vapor-dry deposition. Wet deposition flux is a function of the amount of precipitation, the particle-raindrop collision efficiency, rain-cloud height, and raindrop radius (Andren, 1983). For PCBs, the most significant input pathway to large bodies of water that are far from major sources is wet deposition (Eisenreich, 1987). Wet deposition flux is sometimes approximated using measurement of the total PCB concentration in rainwater (dissolved and particle-bound) and the average yearly rainfall over the area of interest (Mueller et al., 1982).

Dry deposition flux of particulate PCB is a function of particle size, wind velocity, type of receptor surface, and PCB concentration in air (Doskey and Andren, 1981). Estimates of dry deposition to aqueous surfaces are uncertain due to the lack of acceptable methods of measuring these fluxes (Andren, 1983). Dry deposition flux of vapor-phase PCB is governed by molecular diffusion and depends on the concentration gradient between the

equilibrium PCB concentrations of the air and water and the Henry's law constant (Eisenreich, 1987).

The Phase 1 Report assesses the significance of atmospheric deposition of PCB to the Lower Hudson River based on two studies -- Mueller et al. (1982) and Thomann et al. (1989). Mueller et al. (1982) base their estimate of PCB wet deposition flux on an empirical relationship between PCB concentration in rainwater and annual precipitation over an area of 711 km². A range of dry deposition flux was obtained using dry deposition velocities of 0.1 and 1.0 cm/s (Galloway et al., 1980), resulting in a total (wet and dry) mean PCB flux of 1.8 μ g/m²-d. Thomann et al. (198) use an estimated atmospheric precipitation concentration of 0.1 μ g/l to estimate the total downstream atmospheric PCB load without accounting for dry deposition. Α flux of 0.30 μ g/m²-d was calculated based on the total mass loading rate of 0.23 kg/d (0.5 lb/d) and a river surface area of 760 km² (Thomann et al., 1989).

Recent data on dry deposition of PCB in the Chicago and Los Angeles area (Holsen *et al.*, 1991) suggests that the New York metropolitan area may be a major atmospheric source of PCB for the Lower Hudson river. Although PCBs are most likely associated with submicrometer-size particles with low deposition velocities (Doskey and Andren, 1981), Holsen *et al.* (1991) have shown that urban atmospheres contain a significant amount of PCB associated with coarse (>25µm) particles. The dry deposition flux measured in Chicago between May and November of 1989 varied between 2.8 and 9.7 μ g/m²-d and averaged 4.5 μ g/m²-d (Holsen *et al.*, 1991).

321

Using this value, the total PCB load falling on an area the size of the Lower Hudson River (760 km^2) would be 3.4 kg/d.

The table below summarizes the estimates of PCB flux $(\mu g/m^2-d)$ and mass loading (kg/d) derived from Mueller et al. (1982), Thomann et al. (1989), and Holsen et al. (1991).

Source	Type of Deposition	Min. Flux (µg/m²-d)	Max. Flux (µg/m²-d)	Mean Flux (µg/m²-d)	Misan Load* (kg/d)	Percent of Upper Hudson River Load**
Mueller et al.	wet and dry	0.33	3.3	1.8	1,4	230 percent
Thomann et al.	wet only	-	•	0.3	0.23	38 percent
Holsen et al.	dry only	2.8	9.7	4.5	3.4	566 percent

based on river surface area of 760 km² (Thomann et al., 1989).

based on estimated current Upper Hudson River load of 0.6 kg/d (EPA Phase 1 Report, Table B.4-4)

As the table above indicates, the atmospheric PCB load of 0.1-0.5 kg/d presented in the Phase 1 Report (Table A.2-2) may not only grossly underestimate atmospheric loads, but may mischaracterize the current and future significance of this loading.

6.4.7 Total Lower River Sources Relative To Upper River Transport

Against the Lower River PCB sources discussed above, the Phase 1 Report estimates that, in 1980, approximately 4.4 pounds per day (2.0 kg/day) passed over the Federal Dam from the Upper to the Lower River (p. B.4-28). Further, the Report notes that this load decreases exponentially, with a half-life of approximately three years, resulting in present Upper River contributions of approximately 0.3 lb/day (pp. A.4-2, B.4-27). These numbers, of course, are dwarfed in comparison with those

derived from other Lower River sources which, as admitted by EPA, are falling less rapidly than the Upper Hudson River contributions (p. A.4-2). Thus, the Upper Hudson River will play an increasingly smaller role both in absolute and relative terms in coming years.

6.4.8 <u>Upper River Sources</u>

The Phase 1 Report also discusses, to some extent, Upper River PCB sources. As with the Lower River, the EPA has done very little investigation of Upper River sources. For example, in determining industrial dischargers, EPA again looks only to current SPDES permit holders (p. B.2.2) and ignored the many industries along the Upper Hudson that employed open-end and nominally closed applications of PCBs.

Indeed, even as to the few current SPDES permit holders that EPA discusses, EPA ignores past discharges, even major discharges, that were not permitted. NYSDEC documents reveal, for example, that as late as 1979 one current SPDES permit holder located on the banks of the river just west of Rogers Island released PCB-contaminated paper sludge several hundred feet in length and as much as 21 inches thick. PCBs in the sludge from that facility have been measured at levels as high as 224 ppm. Unpermitted discharges in the tributaries are also discussed only very superficially. For example, EPA notes that one discharger on the Hoosic River is permitted to discharge PCBs at .01 ppm, but fails to mention records from the State of Massachusetts indicating that substantial unpermitted releases have occurred from that discharger.

HRP 001 1782

Similarly, Table B.2-2 in the Report notes an inactive waste disposal site upstream of the GE facilities with contaminated soil that is eroding into the Hudson River. Soil contamination at the site is as high as 37,737 ppm of PCBs; river bottom concentrations are 86.5 ppm. Yet the text of the Report makes no attempt to quantify these releases.

* * *

The foregoing discussion demonstrates that the multiple-source model is not an abstract theory constructed on statistics. It is supported by abundant data that multiple sources of PCBs existed and continue to exist along the length of the River. While it might be convenient to assume that all or almost all of the PCBs in the River had a single source, that assumption is contrary to the evidence. Persisting in that incorrect assumption will result in an incorrect understanding of PCB fate and transport, an erroneous identification of the source of the risk (if any) from PCBs in the River, and a remedy selection that will fail to address those sources.

6.5 <u>Recommendations</u>

The Phase 1 Report states that Lower Hudson River sources of PCBs are important to consider but also acknowledges that data and other information concerning these sources are deficient (Section A, Synopsis, pp. A.2-2 to A.2-6). Further, the Report states only generally that "field sampling and additional data evaluation are necessary in Phase 2 to provide improved understanding of PCB levels and transfer mechanisms among sediments, water, air and biota" (p. E-13). The Report

HRF 001 1783

does not take the next logical step of determining what steps should be taken to understand other PCB sources. The data presently available show that these sources are extensive. The data are not, however, presently sufficient to estimate with the required confidence the volume and nature of discharges from these sources. To obtain the required information, additional investigation is necessary. GE recommends that, at a minimum, the following steps be taken:

- 1. EPA should critically evaluate the scientific bases for the tacitly assumed single source/massive movement model for Hudson River PCB contamination.
- 2. This critical evaluation should include references to and descriptions of the various Lower Hudson PCB surveys that produced contradictory information or conclusions, including the EPA's own 1976 and 1981 sediment PCB surveys, NYU's 1978-81 Gammarus PCB survey, the 1988-90 Harza surveys of sediment and biota PCBs and pesticides, the NYSDEC reports on pre-1974 PCB levels in Hudson River fish, and the various studies by the U.S. Army Corps of Engineers indicating very low sediment PCB levels in the stretch of the River between Castleton and Hudson.
- 3. Any high-resolution studies of PCB distributions in Upper Hudson sediments should include determinations of specific PCB congeners or congener combinations that indicate original PCB composition or dechlorination status, as a basis for establishing stratigraphic relationships in areas where the radionuclide profile reflects redepositional fractionation, as well as for assessing the progress of the ongoing local anaerobic microbial dechlorination/detoxication processes.
- 4. EPA must also recognize that high-resolution sediment testing techniques have limited utility in establishing absolute PCB loadings to the Lower River. At best this data will yield information on relative changes in PCB concentration and composition over a relative period of time, at a point in the river. Interpretations of this data are based on numerous assumptions that are difficult or impossible to validate.
- 5. EPA should implement the investigative steps called for in its 1987 and 1989 Potentially Responsible Party

Search Guidances. In particular, EPA should, at a minimum:

- (a) Determine likely discharges from Monsanto's PCB customers for all years. As noted, the only such data presently available to GE covers 1971 and 1972. The Phase 1 Report (p. B.2-1) indicates EPA has Monsanto sales data for all years of U.S. production. This data should be used.
- (b) Identify and investigate facilities in the Hudson River Basin that now fall or formerly fell in the PCB-use categories identified by the 1976 Versar report.
- (c) Interview federal, state, and local agency officials.
- (d) Review documents from federal, state, and local agencies, including site inspection, assessment, and investigation reports, spill reports, remedial investigations, consent orders, and similar documents.
- 6. EPA should include data on PCB homolog and pesticide distributions in migratory fish to permit identification of the areas where they actually acquire their PCB burdens.

6.6 List of References

Battelle Ocean Sciences. 1990. Mussel Watch Phase 4 Final Report, National Status and Trends Mussel Watch Program. Prepared for the National Oceanic and Atmospheric Administration by Battelle Ocean Sciences, Duxbury, MA.

Bopp, R.F. 1979. The Geochemistry of DS3 1618 polychlorinated biphenyls in the Hudson River. Ph.D. Dissertation, Columbia University, New York, NY.

Bopp, R.F., H.J. Simpson, C.R. Olsen, R.M. Trier, and N. Kostyk. 1981. Polychlorinated biphenyls in the sediments of the tidal Hudson River, New York. *Environ. Sci. Technol.* 15:210-216.

Bopp, R.F., H.J. Simpson, C.R. Olsen, R.M. Trier, and N. Kostyk. 1982. Chlorinated hydrocarbons and radionuclide chronologies in sediments in the Hudson River and Estuary. *Environ. Sci. Technol.* 16:666-672.

Bopp, R.F., and H.J. Simpson. 1989. Contamination of the Hudson River, the sediment record. In Contaminated Marine Sediments -- O Assessment and Remediation, pp. 401-416, National Academy Press, Washington, DC.

IRP

Boreman, J. and R.J. Klauda. 1988. Distributions of Early Life Stages of Striped Bass in the Hudson River Estuary, 1974-1979. American Fisheries Society Monograph 4:53-58.

Brown Jr., J.F., R.E. Wagner, D.L. Bedard, M.J. Brennan, J.C. Carnahan, and R.J. May. 1984. PCB transformations in upper Hudson sediments. Northeast. Environ. Sci. 3:166-178.

Brown Jr., J.F., D.L. Bedard, M.J. Brennan, J.C. Carnahan, H. Feng, and R.E. Wagner. 1987a. Polychlorinated biphenyl dechlorination in aquatic sediments. *Science* 236:709-712.

Brown Jr., J.F., R.E. Wagner, H. Feng, D.L. Bedard, M.J. Brennan, J.C. Carnahan, and R.J. May. 1987b. Environmental dechlorination of PCBs. Environ. Toxicol. Chem. 6:579-593.

Brown Jr., J.F., and R.E. Wagner. 1990. PCB movement, dechlorination and detoxication in the Acushnet Estuary. *Environ. Toxicol. Chem.* 9:1215-1233.

Brown Jr., J.F., and R.E. Wagner. 1990. PCB Movement, Dechlorination, and Detoxication in the Acushnet Estuary. Environmental Toxicology and Chemistry 9:1215-1233.

Brown Jr., J.F., G.M. Frame II, R.J. May, and R.E. Wagner. 1990. Origins of PCBs and pesticides in Hudson River striped bass. Abstracts of Papers Presented to the Society of Environmental Toxicology and Chemistry, November 15-15, 1990, No. 138.

Brown, M.P., M.B. Werner, R.J. Sloan and K.W. Simpson. 1985. Polychlorinated Biphenyls in the Hudson River. Environmental Science and Technology 19:656-661.

Bush, B., R.W. Street, and R.J. Sloan. 1990. Polychlorobiphenyl (PCB) congeners in striped bass (Morone saxatilis) from maine and estuarine waters of New York State determined by capillary gas chromatography. Arch. Environ. Contam. Toxicol. 19:49-61.

Clark, J. 1968. Seasonal Movements of Striped Bass Contingents of Long Island Sound and the New York Bight. Transactions of the American Fisheries Society 97:320-343.

Erickson, M.D. 1986. Analytical Chemistry of PCBs. Butterworth Publishers, Stoneham, MA. 508 pp.

Gardinier, M.N. and T.B. Hoff. 1982. Diet of Striped Bass in the Hudson River Estuary. New York Fish and Game Journal 29:152-165.

Hjorth, D.A. 1988. Feeding Selection of Larval Striped Bass and White Perch in the Peekskill Region of the Hudson River. p. 134-147 in Smith, C.L., editor. Fisheries Research in the Hudson River. SUNY Press, Albany, NY. 407 pp.

Hoff, T.B., J.B. McLaren and J.C. Cooper. 1988. Stock Characteristics of Hudson River Striped Bass. American Fisheries Society Monograph 4:59-68.

Klauda, R.J., W.P. Dey, T.B. Hoff, J.B. McLaren and Q.T. Ross. 1980. Biology of Hudson River Juvenile Striped Bass. Marine Recreational Fisheries 5:101-124.

McLaren, J.B., J.C. Cooper, T.B. Hoff and V. Lander. 1981. Movements of Hudson River Striped Bass. Transactions of the American Fisheries Society 110:158-167.

Merriman, D. 1941. Studies on the Striped Bass (Roccus saxitilis) of the Atlantic Coast. U.S. Fish and Wildlife Service, Fish Bulletin 50 (35):1-77.

Moskowitz, P. et al. (1977). Troubled Waters: Toxic Chemicals in the Hudson River (1977).

NYSDEC (New York State Department of Environmental Conservation). 1975. Monitoring of PCB's in fish taken from the Hudson River. Albany, NY.

NYSDEC (New York State Department of Environmental Conservation). 1976. PCB Data in Hudson River Fish, Sediments, Water and Wastewater.

NYDEC (New York State Department of Environmental Conservation). 1991. Inactive Waste Disposal Sites in New York State.

NYSDEC (New York State Department of Environmental Conservation). Various Engineering Investigations at Inactive Hazardous Waste Sites.

Niimi, A.J., and B.G. Oliver. 1983. Biological Half-Lives of Polychlorinated Biphenyl (PCB) Congeners in Whole Fish and Muscle of Rainbow Trout (Salmo Gairdner). Canadian Jnl. of Fisheries and Aquatic Sciences, Vol. 40, pp. 1388-1394.

O'Connor, J.M., R.J. Califano, J.C. Pizza, C.C. Lee, and L.S. Peters. 1982. PCBs in microzooplankton, macrozooplankton, and selected benthos from the lower Hudson River. In Final Report, The Biology of PCBs in Hudson River Zooplankton, Including: Environmental Distribution, Dynamics and Kinetics of Bio-accumulation, and Environmental Impact. Submitted by NYS Dept. of Environmental Conservation, Bureau of Water Research, by New York University Medical Center, Institute of Environmental Studies, 550 First Avenue, New York,NY.

O'Connor, J.M. 1984. PCBs: Dietary Dose and Burdens in Striped Bass from the Hudson River. Northeastern Environmental Science

Pizza, J.C. and J.M. O'Connor. 1983. PCB Dynamics in Hudson River Striped Bass. II. Accumulation from Dietary Sources. Aquatic Toxicology 3:313-327.

Raney, E.C., W.S. Woolcott and A.G. Mehring. 1954. Migratory Patterns and Racial Structure of Atlantic Coast Striped Bass. Transactions of the North American Wildlife Conference 19:376-396.

Rohmann, S. et al., 1985 (Phase 1) and 1987 (Phase 2). Tracing A River's Toxic Pollution: A Case Study of the Hudson (1985).

Sanders, J.E. 1989. PCB-pollution in the Hudson River: From environmental disaster to environmental grid lock. Northeast. Environ. Sci. 8:1-86.

Schaefer, R.H. 1970. Feeding Habits of Striped Bass from the Surf Waters of Long Island. New York Fish and Game Journal 17:1-17.

Schulz, D.E., G. Petrick and J.C. Duinker. 1989. Complete Characterization of Polychlorinated Biphenyl Congeners in Commercial Aroclor and Clophen Mixtures by Multidimensional Gas Chromatography-Electron Capture Detection. Environmental Science and Technology 23:852-859.

Shephard, B.K., J.W. Meldrim, and J.F. Brown, Jr. 1990. PCB and pesticide residues in sediments and biota of the tidal Hudson River, New York Harbor, and Long Island Sound. Abstracts of Papers Presented to the Society of Environmental Toxicology and Chemistry, November 11-15, 1990, No. 137.

Sloan, R.J. 1988. Results of 1988 Hudson River Fish Sampling for PCB Analyses. New York State Department of Environmental Conservation, Albany, NY. 23 pp.

Smith, C.L. 1985. The Inland Fishes of New York State. New York State Department of Environmental Conservation, Albany, NY. 522 pp.

Thomann, R.V. 1981. Equilibrium Model of Fate of Microcontaminants in Diverse Aquatic Food Chains. Canadian Journal of Fisheries and Aquatic Sciences 38:280-296.

Tofflemire, T.J., and S.O. Quinn. 1979. PCB in the upper Hudson River: Mapping and sediment relationships. NYSDEC Technical Paper No. 56, Albany, NY, 140 pages.

Trent, L. and W.W. Hassler, 1966. Feeding behavior of adult striped bass, Roccus saxitilis, in relation to stages of sexual maturity. Chesapeake Science 7:189-192.

USEPA (United States Army Corps of Engineers). 1985. Public Notice No. 12270-FP (regarding contaminant surveys and plans for maintenance dreading of the Castleton, Stuyvesant and North Germantown sections of the Hudson River). New York District, New York, NY, March 19.

USEPA (United States Environmental Protection Agency). 1977. PCB's in lower Hudson River sediments, a preliminary survey 12/11/76-12/15/76. Surveillance and Analysis Division, Region II, US Environmental Protection Agency, Edison, NJ 08817, Feb. 23.

USEPA (United States Environmental Protection Agency). 1982. PCB's in Hudson River sediments 10/7/81-10/22/81. Report prepared by Billie Jo Johnson for Surveillance and Analysis Division, Region II, US Environmental Protection Agency, Edison, NJ 08817.

USEPA (United States Environmental Protection Agency). 1987. Potentially Responsible Party Search Manual, Final Report. OSWER Dir. 9834.3-ola (formerly 9834.6).

USEPA (United States Environmental Protection Agency). 1989. PRP Search Supplemental Guidance for Sites in the Superfund Remedial Program. OSWER Dir. 9834.3-2a.

Versar, Inc. 1976. PCBs in the United States: Industrial Use and Environmental Distribution.

Waldman, J.R., D.J. Dunning, H.E. Ross, and M.T. Mattson. 1990. Range dynamics of Hudson River striped bass along the Atlantic coast. Transactions of the American Fisheries Society 119:910-919.

Waldman, J.R., D.J. Dunning, Q.E. Ross and M.T. Mattson. 1990. Range Dynamics of Hudson River Striped Bass along the Atlantic Coast. Transactions of the American Fisheries Society 119:910-919.

7.0 <u>CONCLUSIONS</u>

EPA's Phase 1 Report fails to demonstrate that the conclusions of the 1984 ROD were wrong or that there have been any changes in circumstances that warrant a modification in such conclusions. EPA must recognize that the existing data demonstrate that PCBs in the Hudson River do not present an unacceptable risk to human health or the ecosystem.

If EPA intends to proceed with the Reassessment, it must correct three fundamental problems with the Phase 1 Report: (1) the absence of critical data; (2) the reliance on old, faulty assumptions; and (3) the use of an inadequate, qualitative method^{*} of analyzing the complex Hudson River system.

Correction of these flaws requires the collection and consideration of, among other information, additional data pertaining to PCB interactions in Hudson River sediment, water, and biota; site-specific data pertaining to exposure to PCBs from the Upper Hudson; current data relating to natural bioremediation in the Hudson River sediment; data pertaining to the impediments to and adverse environmental effects of massive dredging in the Upper Hudson; and information regarding sources of PCBs in the Hudson River other than GE. The analysis of this data requires the use of an integrated, quantitative model.

In these comments, GE has attempted to correct some of the Phase 1 deficiencies. Consideration of the information provided by GE with a more integrated mode of analysis produces conclusions different from those in the Phase 1 Report.

PCBs in the Upper Hudson present no unacceptable risk to human health.

The Phase 1 Report properly recognizes that PCB levels in water, sediment, and biota have significantly declined sinde EPA's 1984 no-action decision. It is undeniable that the Hudson River is cleansing itself. EPA's preliminary "baseline" risk assessment, however, significantly overestimates current risks. First, EPA's assessment does not accept the important new scientific information which establishes the different toxicities of differently chlorinated PCBs. Second, EPA's exposure assumptions are, contrary to EPA guidance, unrealistic and not site-specific. Third, EPA's "baseline" assessment fails to consider the effect of other sources of PCBs in Upper Hudson fish. Finally, current evidence shows that the presence of PCBs in the Upper Hudson ecosystem has not significantly impaired its biological integrity.

• Dredging technologies have not significantly advanced since 1984, and all of the adverse consequences of dredging that were decisive in 1984 are equally applicable today.

In 1984, EPA concluded that dredging to remove sediments from the Upper Hudson was unproven and unreliable. Nothing in the Phase 1 Report supports a contrary conclusion today. In addition, the Phase 1 Report contains no discussion of the numerous practical impediments that would make large-scale dredging, of the type involved here, infeasible in the Hudson River. In particular, EPA fails to recognize the ecologically destructive impact that such a dredging project would have in the Hudson River.

S. W.

с. й У

Natural processes are continuously and significantly reducing any impact of PCBs in the Hudson River, and these natural processes should be permitted to solve this problem.

Laboratory and field studies show that Hudson River sediments have undergone widespread anaerobic dechlorination, which produces PCBs that are not carcinogenic, are far less toxic, and accumulate less readily in fish. In addition, the lower chlorinated PCBs that result from anaerobic dechlorination are further degraded by the natural process of aerobic dechlorination. EPA must therefore give proper consideration to the importance of biological dechlorination of PCBs in Hudson River sediments, and the Agency must recognize that this natural bioremediation is by far the best solution to these problems. It is far better than moving PCBs from the River to the land, in violation of Federal and State policies and disrupting local communities in the process.

• <u>PCB contamination of the Hudson River did not result</u> from the massive movement of PCBs from a single Upper River source, but rather resulted from minimal movement from local sources.

Proper analysis of radionuclide, sediment, and fish data reveals that PCBs in the Hudson are more likely to have resulted from the minimal movement of multiple sources. In addition, as the Phase 1 Report recognizes, the Upper River contributes at most a small fraction of Lower River PCB loadings. Full consideration of this important evidence is essential to a proper characterization of the Hudson River site.

- 24 Clark Republic

بت في

ln i

A simple, gualitative model of PCB fate and transport is inadequate for a proper characterization of the site and for a meaningful assessment of remedial alternatives.

Proper characterization of the site requires an integrated understanding of the numerous complexities of PCB interactions in Hudson River sediment, water, and biota. A meaningful assessment of remedial alternatives requires a quantitative tool for analyzing the existing data, in order that predictions of future PCB concentrations under various assumptions may reliably be made. Absent such an integrated understanding and quantitative tool, EPA's qualitative analysis of the existing data is likely to lead to a faulty understanding of the site and to an erroneous assessment of remedial alternatives.

* * *

In sum, although the Phase 1 Report is intended only as an <u>interim</u> characterization and evaluation of the Hudson River site, it creates a foundation for the remainder of the Reassessment that is flawed and inadequate. The enormity of EPA's responsibility, the complexity of the Hudson River site, and the potentially devastating impact that the selection of an improper remedy will have, demand that EPA correct these deficiencies.

In the final analysis, the focal point of these comments is neither law nor policy. It is science. EPA, an agency whose very existence is predicated upon scientific data

HRP 001 1793

and conclusions, must be prepared to evaluate the available data in a scientifically responsible manner.

80 F1 I 335 -

DF:

HRP