

U.S. Army Corps of Engineers

New England District Concord, Massachusetts



U.S. Environmental Protection Agency

New England Region Boston, Massachusetts

ECOLOGICAL RISK ASSESSMENT FOR GENERAL ELECTRIC (GE)/HOUSATONIC RIVER SITE, REST OF RIVER

Volumes 1 and 2 Sections 1-12

DCN: GE-070703-ABRC

July 2003

Environmental Remediation Contract GE/Housatonic River Project Pittsfield, Massachusetts

Contract No. DACW33-00-D-0006

Task Order 0003

03P-0966-1



ECOLOGICAL RISK ASSESSMENT FOR GENERAL ELECTRIC (GE)/HOUSATONIC RIVER SITE REST OF RIVER

VOLUMES 1 AND 2 SECTIONS 1-12

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

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

DCN: GE-070703-ABRC

Prepared for

U.S. ARMY CORPS OF ENGINEERS

New England District Concord, Massachusetts

and

U.S. ENVIRONMENTAL PROTECTION AGENCY

New England Region Boston, Massachusetts

Prepared by

WESTON SOLUTIONS, INC.

West Chester, Pennsylvania

July 2003

Work Order No. 20123.001.096.0733

AUTHORS/CONTRIBUTORS

Tod Delong, Florence Sevold Avatar Environmental, LLC West Chester, Pennsylvania

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

Dwayne Moore, Roger Breton, Drew McDonald, Andrew Pawlicz, Scott Teed, Ryan Thompson The Cadmus Group, Inc. Ottawa, Ontario

> Gary Lawrence, Chessy Langford EVS Environmental Consultants Vancouver, British Columbia

Rich DiNitto Sleeman, Hanley & DiNitto Boston, Massachusetts

> Alice Shelly TerraStat Seattle, Washington

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

Dick McGrath, Scott Campbell **Weston Solutions, Inc.** West Chester, PA; Pittsfield, Massachusetts

John Lortie, Bob Roy, Michael Thompson, Chris Werner Woodlot Alternatives, Inc. Topsham, ME

1		TABLE OF CONTENTS				
2	Sec	Section				
3	ES.	ECO	DLOGICAL RISK ASSESSMENT EXECUTIVE SUMMARY	ES-1		
4		ES.1	OVERVIEW	ES-1		
5		ES.2	SITE DESCRIPTION AND HISTORY	ES-2		
6		ES.3	REGULATORY BACKGROUND	ES-7		
7		ES.4	OVERVIEW OF TECHNICAL APPROACH	ES-8		
8		ES.5	OVERVIEW OF THE ASSESSMENT ENDPOINT CONCLUSIONS	ES-12		
9			ES.5.1 Risks in the Primary Study Area	ES-12		
10			ES.5.2 Risks Downstream of the Primary Study Area	ES-33		
11		ES.6	BROADER IMPLICATIONS	ES-43		
12			ES.6.1 Implications for Other Species in the Primary Study Area	ES-43		
13		ES.7	SOURCES OF UNCERTAINTY	ES-48		
14		ES.8	SUMMARY AND CONCLUSIONS	ES-50		
15	1.	INT	RODUCTION	1-1		
16		1.1	OVERVIEW	1-1		
17		1.2	SITE HISTORY	1-5		
18		1.3	REGULATORY BACKGROUND	1-9		
19		1.4	SITE DESCRIPTION	1-11		
20		1.5	OVERVIEW OF TECHNICAL APPROACH			
21			1.5.1 Problem Formulation	1-23		
22			1.5.2 Assessment of Representative Species			
23		1.6	DATA SOURCES			
24		1.7	QA/QC			
25		1.8	REFERENCES			
26	2.	PRO	DBLEM FORMULATION	2-1		
27		2.1	OVERVIEW	2-1		
28 29		2.2	PHYSICAL AND ECOLOGICAL CHARACTERIZATION OF THE HOUSATONIC RIVER			
30			2.2.1 Physical Characteristics of the Housatonic River Basin			
31			2.2.2 Ecological Characterization of the Study Area			
32		2.3	IDENTIFICATION AND SOURCES OF STRESSORS			
33			2.3.1 Contaminant Stressors			
34			2.3.2 Physical and Biological Stressors			

	Sectio	on		Page
1	2	.4	OVERVIEW OF PRE-ERA	2-14
2			2.4.1 Introduction	2-14
3			2.4.2 Data	2-14
4			2.4.3 Primary Study Area (PSA) Evaluation and Results	2-16
5			2.4.4 PCB Screening Evaluation Downstream of Woods Pond and Results	2-17
6	2	2.5	FATE AND TRANSPORT OF CONTAMINANT STRESSORS	2-23
7			2.5.1 Fate and Transport of PCBs	2-23
8			2.5.2 PCB Distribution by Media	2-25
9 10			2.5.3 Identification of Exposure Pathways	2-39
10			2.5.4 Changes in FCB Congener Fatterns	2-41
12	2	6	EFFECTS ON RECEPTORS	2-45
13	_		2.6.1 Polychlorinated Binhenyls (PCBs)	2-46
14			2.6.2 Dioxins/Furans	2-49
15			2.6.3 2,3,7,8-TCDD Toxic Equivalence (TEQ)	2-51
16	2	.7	CONCEPTUAL MODEL	2-54
17			2.7.1 Exposure Pathways	2-55
18	2	.8	SELECTION OF ASSESSMENT AND MEASUREMENT ENDPOINTS	2-58
19			2.8.1 Assessment Endpoints	2-58
20			2.8.2 Measurement Endpoints	2-60
21	2	.9	WEIGHT-OF-EVIDENCE APPROACH TO ANALYSIS	2-66
22	2	.10	EXTRAPOLATION OF RISK ESTIMATES FOR SELECTED ENDPOINTS	
23			DOWNSTREAM OF WOODS POND	2-72
24	2	.11	REFERENCES	2-73
25	3. A	SSI	ESSMENT ENDPOINT—COMMUNITY STRUCTURE, SURVIVAL,	
26	(GRO	WTH, AND REPRODUCTION OF BENTHIC INVERTEBRATES	3-1
27	3	.1	INTRODUCTION	3-1
28			3.1.1 Conceptual Model	3-7
29	3	.2	EXPOSURE ASSESSMENT	3-11
30			3.2.1 Selection of COCs for Benthic Invertebrates	3-11
31			3.2.2 Types of Exposure Data	3-13
32			3.2.3 Habitat Characterization	3-15
33			3.2.4 Assessment of Sediment Chemistry	3-17
34 25			3.2.5 Lissue Chemistry Assessment	
33 20	2	2	5.2.0 Surface water Unemistry Assessment	3-24
30	3	.3	EFFEUIS ASSESSMENI	3-26

Section

1			3.3.1	Sediment Toxicity	3-26
2			3.3.2	Concentration-Response Analysis – Toxicity Test Endpoints	3-45
3			3.3.3	Toxicity Identification Evaluations	3-52
4			3.3.4	Tissue PCB Effects Thresholds	3-53
5			3.3.5	Sediment Quality Values (SQVs)	3-54
6			3.3.6	Benthic Macroinvertebrate Community Evaluation	3-54
7			3.3.7	Concentration-Response Analysis – Benthic Community Assemblages	3-58
8		3.4	RISK	CHARACTERIZATION	3-61
9			3.4.1	Field Surveys	3-62
10			3.4.2	Comparison of Chemistry Data to Benchmarks	3-63
11			3.4.3	Site-Specific Toxicity Study Results	3-66
12			3.4.4	Integrated Station-by-Station Assessment	3-69
13			3.4.5	Weight-of-Evidence (WOE) Procedure for Assessing Risk from PCBs in	
14				the Housatonic River PSA	3-69
15			3.4.6	Sources of Uncertainty	3-74
16			3.4.7	Extrapolation to Other Species	3-77
17			3.4.8	Downstream Assessment	3-77
18			3.4.9	Conclusions	3-78
19		3.5	REFE	RENCES	3-80
20 21	4.	ASS REF	ESSMI 'RODU	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF	
20 21 22	4.	ASS REF AM	ESSMI PRODU PHIBIA	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF ANS	4-1
20 21 22 23	4.	ASS REF AM	SESSMI PRODU PHIBLA INTRO	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF ANS	 4-1 4-1
20 21 22 23 24	4.	ASS REF AM 4.1 4.2	ESSMI PRODU PHIBLA INTRO CONC	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF ANS ODUCTION CEPTUAL MODEL	 4-1 4-1 4-8
20 21 22 23 24 25	4.	ASS REF AM 4.1 4.2	SESSMI PRODU PHIBLA INTRO CONC 4.2.1	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, OCTION, DEVELOPMENT, AND MATURATION OF ANS ODUCTION CEPTUAL MODEL Amphibian Developmental Studies	 4-1 4-1 4-8 4-13
20 21 22 23 24 25 26	4.	ASS REF AM 4.1 4.2	PRODU PRODU PHIBLA INTRO CONC 4.2.1 4.2.2	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF ANS ODUCTION CEPTUAL MODEL Amphibian Developmental Studies Leopard Frog Study: EPA	 4-1 4-1 4-8 4-13 4-14
20 21 22 23 24 25 26 27	4.	ASS REF AM 4.1 4.2	SESSMI PRODU PHIBL INTRO CONC 4.2.1 4.2.2 4.2.3	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF ANS DDUCTION CEPTUAL MODEL Amphibian Developmental Studies Leopard Frog Study: EPA Wood Frog Study Design (EPA Studies)	 4-1 4-1 4-8 4-13 4-14 4-14
20 21 22 23 24 25 26 27 28	4.	ASS REF AM 4.1 4.2	SESSMI PRODU PHIBL INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, OCTION, DEVELOPMENT, AND MATURATION OF ANS ODUCTION CEPTUAL MODEL Amphibian Developmental Studies Leopard Frog Study: EPA Wood Frog Study Design (EPA Studies) Context-Dependent Wood Frog Study: GE	 4-1 4-1 4-8 4-13 4-14 4-14 4-17
20 21 22 23 24 25 26 27 28 29	4.	ASS REF AM 4.1 4.2 4.3	SESSMI PRODU PHIBL INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4 EXPO	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, OCTION, DEVELOPMENT, AND MATURATION OF ANS ODUCTION CEPTUAL MODEL Amphibian Developmental Studies Leopard Frog Study: EPA Wood Frog Study: EPA Wood Frog Study Design (EPA Studies) Context-Dependent Wood Frog Study: GE SURE ASSESSMENT	 4-1 4-1 4-8 4-13 4-14 4-14 4-17 4-19
20 21 22 23 24 25 26 27 28 29 30	4.	ASS REF AM 4.1 4.2	SESSMI PRODU PHIBLA INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4 EXPO 4.3.1	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF ANS	 4-1 4-1 4-8 4-13 4-14 4-14 4-17 4-19 4-19
20 21 22 23 24 25 26 27 28 29 30 31	4.	ASS REF AM 4.1 4.2 4.3	SESSMI PRODU PHIBL INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4 EXPO 4.3.1 4.3.2	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF ANS	4-1 4-8 4-13 4-14 4-14 4-17 4-19 4-19 4-20
20 21 22 23 24 25 26 27 28 29 30 31 32	4.	ASS REF AM 4.1 4.2 4.3	SESSMI PRODU PHIBLA INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4 EXPO 4.3.1 4.3.2 4.3.3	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, OCTION, DEVELOPMENT, AND MATURATION OF ANS	4-1 4-1 4-8 4-13 4-14 4-14 4-17 4-19 4-19 4-20 4-21
20 21 22 23 24 25 26 27 28 29 30 31 32 33	4.	ASS REF AM 4.1 4.2 4.3	SESSMI PRODU PHIBLA INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4 EXPO 4.3.1 4.3.2 4.3.3 4.3.4	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, OCTION, DEVELOPMENT, AND MATURATION OF ANS	4-1 4-8 4-13 4-13 4-14 4-17 4-17 4-19 4-20 4-21 4-22
20 21 22 23 24 25 26 27 28 29 30 31 32 33 34	4.	ASS REF AM 4.1 4.2 4.3	SESSMI PRODU PHIBLA INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4 EXPO 4.3.1 4.3.2 4.3.3 4.3.4 4.3.5	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, CCTION, DEVELOPMENT, AND MATURATION OF ANS	4-1 4-8 4-13 4-14 4-14 4-17 4-19 4-19 4-20 4-22 4-22 4-25
20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35	4.	ASS REF AM 4.1 4.2 4.3	SESSMI PRODU PHIBLA INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4 EXPO 4.3.1 4.3.2 4.3.3 4.3.4 4.3.5 4.3.6	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, CTION, DEVELOPMENT, AND MATURATION OF ANS	4-1 4-1 4-8 4-13 4-14 4-14 4-17 4-19 4-19 4-20 4-20 4-22 4-25 4-27
20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36	4.	ASS REF AM 4.1 4.2 4.3	SESSMI PRODU PHIBIA INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4 EXPO 4.3.1 4.3.2 4.3.3 4.3.4 4.3.5 4.3.6 EFFEO	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF ANS	4-1 4-1 4-8 4-13 4-14 4-14 4-17 4-19 4-19 4-20 4-21 4-22 4-22 4-27 4-31
20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37	4.	ASS REF AM 4.1 4.2 4.3	SESSMI PRODU PHIBLA INTRO CONC 4.2.1 4.2.2 4.2.3 4.2.4 EXPO 4.3.1 4.3.2 4.3.3 4.3.4 4.3.5 4.3.6 EFFEC 4.4.1	ENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, ICTION, DEVELOPMENT, AND MATURATION OF ANS	4-1 4-1 4-8 4-13 4-14 4-14 4-17 4-19 4-19 4-20 4-21 4-22 4-25 4-27 4-31 4-32

	Sect	ion		Page
1			4.4.2 PCB Effect Thresholds	4-58
2		4.5	RISK CHARACTERIZATION	4-61
3			4.5.1 Concentration-Response Analysis – Toxicity Test Endpoints	4-62
4			4.5.2 Biological Community Endpoints	4-64
5			4.5.3 Comparison of Tissue Chemistry Data to Benchmarks	4-65
6 7			4.5.4 Integrated Station-by-Station Assessment	4-69
8			Housatonic River PSA	4-69
9			4.5.6 Sources of Uncertainty	4-76
10			4.5.7 Conclusions	4-81
11		4.6	REFERENCES	4-85
12	5.	ASS	ESSMENT ENDPOINT – SURVIVAL, GROWTH, AND	F 1
13		KEP	RODUCTION OF FISH	5-1
14		5.1	INTRODUCTION	5-2
15			5.1.1 Conceptual Model	5-3
16		5.2	EXPOSURE ASSESSMENT	5-10
17			5.2.1 Refined Screening of COPCs for Fish	5-10
18			5.2.2 Tissue Chemistry Assessment (Exposure to PCBs and TEQ)	5-11
19			5.2.3 Sediment Chemistry Assessment (Exposure to PAHs)	5-17
20		5.3	EFFECTS ASSESSMENT	5-21
21			5.3.1 Derivation of Literature Tissue Effects Metrics	5-22
22			5.3.2 Site-Specific Toxicity Studies	5-28
23			5.3.3 Concentration-Response Analysis – I oxicity Endpoints	5-39
24			5.5.4 Derivation of Literature-Based Sediment Effects Metrics for PAHs	5-45
25		5.4	RISK CHARACTERIZATION	5-48
26			5.4.1 Introduction	5-48
27			5.4.2 Field Surveys	5-49
20 29			5.4.5 Comparison of Estimated Exposures to Derived Effects Metrics	5-54
30			5.4.5 Weight-of-Evidence Analysis	5-08
31			5.4.6 Sources of Uncertainty	5-71
32			5.4.7 Downstream Extrapolation	5-73
33			5.4.8 Risk Assessment Conclusions	5-74
34		5.5	REFERENCES	5-75
35	6.	WII	DLIFE ASSESSMENT HIGHLIGHTS	6-1
36		6.1	OVERVIEW	6-1

	Section		Page
1	6.2	WILDLIFE EXPOSURE MODEL	6-7
2		6.2.1 Food Intake Rate (FIR)	6-8
3		6.2.2 Body Weight (BW)	6-9
4		6.2.3 Proportions of Dietary Items	6-9
5	6.3	SPATIAL AND TEMPORAL AVERAGING	6-10
6	6.4	TOXIC EQUIVALENCE (TEQ)	6-11
7		6.4.1 Non-Detects	6-15
8		6.4.2 Congener Co-Elution	6-15
9		6.4.3 Summary of Decision Criteria for Estimating Exposure Point	6.16
10		Concentrations	6-16
11	6.5	PROBABILISTIC RISK ASSESSMENT	6-18
12		6.5.1 Distribution Selection	6-18
13		6.5.2 Monte Carlo and Probability Bounds Analysis	6-19
14	6.6	EFFECTS ASSESSMENT	6-21
15		6.6.1 Dose-Response Relationships Using the Generalized Linear Model	()
10 17		6.6.2 Hypothesis Testing to Determine LOAFL and NOAEL	6-22
18		6.6.3 Field-Based Measures of Effect and Threshold Ranges	6-23
19	6.7	RISK CHARACTERIZATION	6-25
20		6.7.1 Risk Categorization	
21		6.7.2 Weight-of-Evidence Assessment	6-28
22	6.8	REFERENCES	6-31
23	7. AS	SESSMENT ENDPOINT – SURVIVAL, GROWTH, AND PRODUCTION OF INSECTIVOPOUS PIPPS	71
24	KE	PRODUCTION OF INSECTIVOROUS BIRDS	
25	7.1	INTRODUCTION	
26	7.2	CONCEPTUAL MODEL	7-8
27	7.3	EXPOSURE ASSESSMENT	7-12
28		7.3.1 Exposure Models for Insectivorous Birds	7-15
29		7.3.2 TDI Model Results	
30		7.3.3 Microexposure Model Results	
22	74	7.5.4 Tree Swallow Tissue Data from Field Study	
32 22	/.4	EFFECTS ASSESSMENT	/-05
55 21		7.4.1 Keview of Effects of tPCBs	
34		7.4.2 2,5,7,6-10DD and Equivalence (TEQ)	/-09 7_70
55		7.1.5 The Swanow Field Study	

	Sect	ion		Page
1			7.4.4 American Robin Field Study (GE)	7 - 71
2			7.4.5 Selection of Effects Metrics for Characterizing Risk	7-72
3		7.5	RISK CHARACTERIZATION	7-75
4			7.5.1 Comparison of Modeled Exposures to Effects	7-75
5			7.5.2 Tree Swallow Field Study	7-78
6			7.5.3 American Robin Field Study (GE)	
7			7.5.4 Weight-of-Evidence Analysis	
8			7.5.5 Sources of Uncertainty	/-86 7 00
9		76	7.5.0 Conclusions and Extrapolation to Other Species	7.02
10		/.0	KEFERENCES	7-92
11 12	8.	ASS REP	ESSMENT ENDPOINT—SURVIVAL, GROWTH, AND PRODUCTION OF PISCINOPOUS BIRDS	8 _1
12		NE 1		••••••••••••••••••••••••••••••••••••••
13		0.1	8.1.1 Overview of Approach	······ 0-1
14		0 1	S.I.I Overview of Approach	
15		8.2		
16		8.3	EXPOSURE ASSESSMENT	8-12
17			8.3.1 Exposure Model	8-12
18			8.3.2 Exposure Model Results	8-19
19		8.4	EFFECTS ASSESSMENT	8-30
20			8.4.1 Total PCBs	8-31
21			8.4.2 TEQ	8-34
22			8.4.3 Effects Metrics	8-34
23		0 5	8.4.4 Belled Kilighsher Fleid Sludy	0.27
24		8.3	RISK CHARACTERIZATION	8-3/
25 26			8.5.1 Comparison of Estimated Exposures to Laboratory-Derived Effect	0 27
20			8 5 2 Belted Kingfisher Field Study	8-37 8-38
28			8 5 3 Weight-of-Evidence Analysis	8-41
29			8.5.4 Sources of Uncertainty	8-46
30			8.5.5 Extrapolation to Other Species	8-48
31			8.5.6 Summary and Conclusions	8-48
32		8.6	REFERENCES	8-49
33	9.	ASS	ESSMENT ENDPOINT – SURVIVAL, GROWTH, AND	
34		REP	PRODUCTION OF PISCIVOROUS MAMMALS	
35		9.1	INTRODUCTION	

	Section		Page
1	9.2	CONCEPTUAL MODEL	9-8
2	9.3	EXPOSURE ASSESSMENT	9-11
3		9.3.1 Exposure Model	9-12
4		9.3.2 Results of Exposure Assessments	9-21
5	9.4	EFFECTS ASSESSMENT	9-36
6		9.4.1 Review of Toxicity from the Literature	9-36
7		9.4.2 Mink Feeding Study	9-40
8		9.4.3 Effects Metrics for Characterizing Risk	9-46
9	9.5	RISK CHARACTERIZATION	9-49
10		9.5.1 Field Surveys	9-49
11		9.5.2 Comparison of Estimated Exposures to Laboratory-Derived Effects	0.50
12			
13		9.5.3 Mink Feeding Study	9-68
14		9.5.5 Sources of Uncertainty	9-09 9_74
16		9.5.6 Comparison to Other Piscivorous Mammals	9-77
17		9.5.7 Risk Downstream of PSA	9-77
18		9.5.8 Conclusions	9-78
19	9.6	REFERENCES	9-80
20 21	10. AS RE	SESSMENT ENDPOINT—SURVIVAL, GROWTH, AND PRODUCTION OF OMNIVOROUS AND CARNIVOROUS MAMMALS	10-1
22	10.1	INTRODUCTION	10-1
23	10.2	2 CONCEPTUAL MODEL	10-8
24	10.3	EXPOSURE ASSESSMENT	10-11
25		10.3.1 Exposure Model	10-13
26		10.3.2 Results of Exposure Assessments	10-22
27	10.4	EFFECTS ASSESSMENT	10-32
28		10.4.1 Review of Effects of tPCBs and TEQ	10-33
29		10.4.2 Effects Metrics for Characterizing Risk	10-36
30	10.5	RISK CHARACTERIZATION	10-41
31		10.5.1 Field Surveys (Performed by EPA)	10-41
32		10.5.2 Comparison of Estimated Exposures to Laboratory-Derived Effects	
33			10-43
54 25		10.5.3 Population Demography Field Study (Performed by GE)	10-53
36		10.5.5 Sources of Uncertainty	10-55
50		10.2.2 Sources of Oncertainty	10-01

viii

	Section					
1		10.5.6 Conclusions	10-63			
2		10.6 REFERENCES	10-68			
3 4	11.	ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF THREATENED AND ENDANGERED SPECIES	11-1			
5		11.1 INTRODUCTION	11-1			
6 7 8		11.1.1 Overview of Approach11.1.2 Conceptual Model11.1.3 Organization	11-2 11-7 11-11			
9		11.2 EXPOSURE ASSESSMENT	11-11			
10		11.2 1 Exposure Model	11 12			
11		11.2.2 Results of Exposure Assessments	11-23			
12		11.3 EFFECTS ASSESSMENT	11-35			
13		11.3.1 Total PCBs	11-36			
14		11.3.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)	11-39			
15		11.3.3 Effects Metrics for Characterizing Risk	11-40			
16		11.4 RISK CHARACTERIZATION	11-45			
17		11.4.1 Field Survey	11-45			
18		11.4.2 Comparison of Estimated Exposures to Laboratory-Derived Effects	11 47			
20		11 4 3 Weight-of-Evidence Analysis	11-58			
21		11.4.4 Sources of Uncertainty	11-63			
22		11.4.5 Conclusions	11-65			
23		11.5 REFERENCES	11 - 69			
24	12.	RISK SUMMARY	12-1			
25		12.1 OVERVIEW	12-1			
26		12.2 SUMMARY OF THE ASSESSMENT ENDPOINT CONCLUSIONS	12-4			
27		12.2.1 Results of Weight-of-Evidence Evaluation	12-4			
28		12.2.2 Hazard Quotient Analyses	12-17			
29		12.2.3 Risk Assessment Downstream of Woods Pond	12-26			
30		12.3 SPECIES SENSITIVITY AND MECHANISMS OF TOXICITY	12-43			
31 32		12.3.1 Mechanism of Action and Sensitivity of Species to tPCBs and TEQ12.3.2 Congener Composition and Toxicity to Biota	12-43 12-46			
33		12.4 BROADER IMPLICATIONS	12-47			
34 35		12.4.1 Implications for Other Species in the Primary Study Area.12.4.2 Ecological Implications and Other Concerns	12-47 12-69			

Section Page 1 2 3 4 5 12.5.4 Risk Characterization 12-78 6 7 8

1		LIST OF TABLES
2	Title	Page
3 4	Table ES-1	Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Benthic CommunityES-14
5 6 7	Table ES-2	Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of Amphibian Populations in the Housatonic River PSA
8 9	Table ES-3	Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Fish CommunityES-17
10 11	Table ES-4	Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to tPCBs in the Housatonic River PSAES-18
12 13	Table ES-5	Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to TEQ in the Housatonic River PSAES-18
14 15	Table ES-6	Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSAES-20
16 17	Table ES-7	Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to TEQ in the Housatonic River PSAES-20
18 19	Table ES-8	Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSAES-22
20 21	Table ES-9	Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSAES-22
22 23	Table ES-10	Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA ES-24
24 25	Table ES-11	Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSAES-24
26 27	Table ES-12	Evidence of Harm and Magnitude of Effects of T&E Species Exposed to tPCBs in the Housatonic River PSAES-26
28 29	Table ES-13	Evidence of Harm and Magnitude of Effects for T&E Species Exposed to TEQ in the Housatonic River PSAES-26
30 31	Table 2.1-1	Surveys Conducted for Ecosystem Characterization and Their Specific Objective(s)
32	Table 2.4-1	Sediment and Surface Water Data Categories
33	Table 2.4-2	COPCs for Sediment Based on Tier III Evaluation
34	Table 2.4-3	COPCs for Surface Water Based on Tier III Evaluation
35	Table 2.4-4	COPCs for Soil Based on Tier III Evaluation
36	Table 2.4-5	COPCs for Fish Based on Tier III Evaluation

Title

1	Table 2.6-1	Common Effects of PCB Exposure Observed in Various Animals2-47
2	Table 2.6-2	TEF Values for Mammals, Fish, and Birds as Predators
3	Table 2.8-1	Ecological Assessment and Measurement Endpoints
4	Table 2.8-2	Summary of GE Ecological Studies
5	Table 2.9-1	Attributes for Judging Measurement Endpoints
6 7 8	Table 3.3-1	Results of Pairwise Statistical Tests Comparing Exposed Stations to Negative Control (T-Ctrl) and Reference (A1, A3) Sediment (Water-Only Exposures Excluded)
9 10	Table 3.3-2	In Situ Evaluation of Toxicity in Housatonic River Sediment (Station-by- Station Assessment)
11 12	Table 3.3-3	Laboratory Evaluation of Toxicity in Housatonic River Sediment (Station- by-Station Assessment)
13 14	Table 3.3-4	Evaluation of Lines of Evidence for Housatonic River Sediment Toxicity, Relative to Reference Responses
15	Table 3.4-1	Weighting of Measurement Endpoints for Weight-of-Evidence Evaluation 3-73
16 17	Table 3.4-2	Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Benthic Community
18 19	Table 3.4-3	Weight-of-Evidence Risk Analysis Summary Indicating Concurrence Among Endpoints for Coarse-Grained and Fine-Grained Sediment
20	Table 4.4-1	Summary of Male Adult Leopard Frog Reproductive Health
21	Table 4.4-2	Summary of Female Adult Leopard Frog Reproductive Health
22 23	Table 4.4-3	Summary of Leopard Frog Larval Development Endpoints Data at End-of- Test
24 25	Table 4.4-4	Summary of Leopard Frog Larval Development Endpoints at Final Test Duration
26 27	Table 4.4-5	Statistical Analysis Results: Wood Frog Reproduction and Development Studies
28 29	Table 4.5-1	Hazard Quotients for Leopard Frog PCB Tissue Residues, Based on Literature-Derived Effects Thresholds
30 31	Table 4.5-2	Integrated Assessment of Potential for Adverse Impacts to Amphibian Populations (Leopard Frog Study)
32 33	Table 4.5-3	Integrated Assessment of Potential for Adverse Impacts to Amphibian Populations (Wood Frog Study)

Title

1 2	Table 4.5-4	Weighting of Measurement Endpoints for Amphibian Weight-of-Evidence Evaluation
3 4 5	Table 4.5-5	Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of Amphibian Populations in the Lower Housatonic River
6 7	Table 4.5-6	Risk Analysis for Amphibians Exposed to tPCBs and Other COCs in the Housatonic River PSA
8 9	Table 5.2-1	tPCB Concentrations in Representative Species Fish Tissue (mg/kg) in the PSA; Data from EPA Tissue Collections (1998-2000)
10 11 12	Table 5.2-2	Total Lipid-Normalized PCB Concentrations (mg/kg lipid) for Representative Species in the PSA; Data from EPA Tissue Collections (1998-2000)
13 14	Table 5.2-3	Summary of tPCB Concentrations (mg/kg) from EPA Samples Collected in Reach 8
15 16 17	Table 5.2-4	TEQ in Representative Species Fish Tissue in the PSA with DL Substitution for NDs (ng/kg); Data from EPA Fish Collections (1998-2000)
18 19	Table 5.2-5	Summary Statistics for Concentrations of PAH COCs in Main Channel Sediment by Reach
20 21	Table 5.3-1	Criteria Used To Screen Available Studies for Determining Threshold Body Burdens
22 23 24	Table 5.3-2	Calculated ED ₅₀ Values (tPCBs and TEQ) for Largemouth Bass, Medaka, and Rainbow Trout Exposed in Ovo to Housatonic River Extracts and PCB-126 and 2,3,7,8-TCDD Standards
25	Table 5.4-1	Probabilities of Exceedances in the PSA for tPCBs
26 27	Table 5.4-2	Probabilities of Exceedances in Reach 8 for tPCBs and TEQ, Based on EPA Sampling
28	Table 5.4-3	Probabilities of Exceedances for TEQ
29 30	Table 5.4-4	Weighting of Measurement Endpoints for Fish Weight-of-Evidence Evaluation
31 32	Table 5.4-5	Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Fish Community
33 34	Table 5.4-6	Risk Analysis for Risk Exposed to tPCBs and TEQ in the Housatonic River PSA

Title

1 2	Table 6.4-1	World Health Organization Toxic Equivalency Factors (TEFs) for TCDD and Equivalents
3 4	Table 6.7-1	Decision Criteria for Converting Risk Category and Range to Evidence of Harm and Magnitude of Effect
5 6	Table 7.5-1	Summary of Qualitative Risk Statements for Insectivorous Birds from the Housatonic River Study Area
7 8	Table 7.5-2	Weighting of Measurement Endpoints for Tree Swallow Weight-of- Evidence Evaluation
9 10	Table 7.5-3	Weighting of Measurement Endpoints for American Robin Weight-of- Evidence Evaluation
11 12	Table 7.5-4	Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to tPCBs in the Housatonic River PSA
13 14	Table 7.5-5	Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to TEQ in the Housatonic River PSA
15 16	Table 7.5-6	Risk Analysis Summary for Insectivorous Birds Exposed to tPCBs in the Housatonic River PSA
17 18	Table 7.5-7	Risk Analysis Summary for Insectivorous Birds Exposed to TEQ in the Housatonic River PSA
19 20	Table 8.5-1	Summary of Qualitative Risk Statements for Piscivorous Birds from the Housatonic River PSA
21 22	Table 8.5-2	Weighting of Measurement Endpoints for Piscivorous Birds Weight-of- Evidence Evaluation
23 24	Table 8.5-3	Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSA
25 26	Table 8.5-4	Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to TEQ in the Housatonic River PSA
27 28	Table 8.5-5	Risk Analysis Summary for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSA
29 30	Table 8.5-6	Risk Analysis Summary for Piscivorous Birds Exposed to TEQ in the Housatonic River PSA
31 32	Table 9.5-1	Results of Snow Tracking and Scent Post Station Surveys in the PSA and Reference Areas
33 34	Table 9.5-2	Summary of Qualitative Risk Statements for Piscivorous Mammals from the Housatonic River Study Area

Title

Table 9.5-3	Weighting of Measurement Endpoints for Piscivorous Mammals Weight- of-Evidence Evaluation
Table 9.5-4	Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSA
Table 9.5-5	Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSA
Table 9.5-6	Risk Analysis Summary for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSA
Table 9.5-7	Risk Analysis Summary for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSA
Table 10.5-1	Summary of Qualitative Risk Statements for Omnivorous and Carnivorous Mammals from the Housatonic River Study Area
Table 10.5-2	Weighting of Measurement Endpoints for Omnivorous and Carnivorous Mammals Weight-of-Evidence Evaluation
Table 10.5-3	Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA 10-58
Table 10.5-4	Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSA 10-58
Table 10.5-5	Risk Analysis Summary for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA
Table 10.5-6	Risk Analysis Summary for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSA
Table 11.4-1	Summary of Qualitative Risk Statements for T&E Species from the Housatonic River Study Area
Table 11.4-2	Weighting of Measurement Endpoints for T&E Species Weight-of- Evidence Evaluation
Table 11.4-3	Evidence of Harm and Magnitude of Effects for T&E Species Exposed to tPCBs in Housatonic River PSA
Table 11.4-4	Evidence of Harm and Magnitude of Effects for T&E Species Exposed to TEQ in the Housatonic River PSA
Table 11.4-5	Risk Analysis Summary for T&E Species Exposed to tPCBs in the Housatonic River PSA
Table 11.4-6	Risk Analysis Summary for T&E Species Exposed to TEQ in the Housatonic River PSA
	Table 9.5-3Table 9.5-4Table 9.5-5Table 9.5-6Table 9.5-7Table 10.5-1Table 10.5-2Table 10.5-3Table 10.5-4Table 10.5-5Table 11.4-1Table 11.4-2Table 11.4-3Table 11.4-3Table 11.4-4Table 11.4-5Table 11.4-5

Title

1 2	Table 12.2-1	Ecological Assessment Endpoints and Conclusions for the Primary Study Area Portion of the Lower Housatonic River
3 4	Table 12.2-2	Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Benthic Community 12-7
5 6 7	Table 12.2-3	Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of Amphibian Populations in the Housatonic River PSA
8 9	Table 12.2-4	Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Fish Community
10 11	Table 12.2-5	Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to tPCBs in the Housatonic River PSA
12 13	Table 12.2-6	Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to TEQ in the Housatonic River PSA
14 15	Table 12.2-7	Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSA
16 17	Table 12.2-8	Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to TEQ in the Housatonic River PSA
18 19	Table 12.2-9	Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSA
20 21	Table 12.2-10	Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSA
22 23	Table 12.2-11	Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA 12-15
24 25	Table 12.2-12	Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSA 12-15
26 27	Table 12.2-13	Evidence of Harm and Magnitude of Effects for T&E Species Exposed to tPCBs in Housatonic River PSA
28 29	Table 12.2-14	Evidence of Harm and Magnitude of Effects for T&E Species Exposed to TEQ in the Housatonic River PSA
30 31	Table 12.2-15	Summary of the Assessment of Risks Conducted for Biota Exposed to tPCBs in the Lower Housatonic River Below Woods Pond
32 33	Table 12.4-1	Comparison of Risks of tPCBs and TEQ to Representative and Other Species in the Housatonic River PSA
34		

1		LIST OF FIGURES		
2	Title	Page		
3	Figure ES-1	Housatonic River		
4	Figure ES-2	Primary Study Area (PSA)ES-5		
5	Figure ES-3	Ecological Risk Assessment RoadmapES-10		
6 7	Figure ES-4	Hazard Quotients for Aquatic Biota Exposed to tPBCs in the Housatonic River PSA		
8 9	Figure ES-5	Hazard Quotients for Fish Exposed to 2,3,7,8-TCDD TEQ in the Housatonic River PSA		
10 11	Figure ES-6	Hazard Quotients for Wildlife Exposed to tPCBs in the Housatonic River PSAES-29		
12 13	Figure ES-7	Hazard Quotients for Wildlife Exposed to 2,3,7,8-TCDD TEQ in the Housatonic River PSA		
14 15	Figure ES-8	Assessment of Risk to Benthic Invertebrates Exposed to tPCBs Downstream of Woods Pond		
16 17	Figure ES-9	Assessment of Risk to Amphibians in Floodplain Exposed to tPCBs Downstream of Woods Pond in Massachusetts		
18 19	Figure ES-10	Assessment of Risk to Amphibians in Sediment Exposed to tPCBs Downstream of Woods Pond in Connecticut		
20 21	Figure ES-11	Assessment of Risk to Warmwater Fish Exposed to tPCBs Downstream of Woods Pond		
22 23	Figure ES-12	Assessment of Risk to Trout Exposed to tPCBs Downstream of Woods Pond		
24 25	Figure ES-13	Assessment of Risk to Mink Exposed to tPCBs Downstream of Woods Pond		
26 27	Figure ES-14	Assessment of Risk to Otter Exposed to tPCBs Downstream of Woods Pond		
28 29	Figure ES-15	Assessment of Risk to Bald Eagle Exposed to tPCBs Downstream of Woods Pond		
30	Figure 1.1-1	Housatonic River		
31	Figure 1.1-2	Primary Study Area (PSA)1-4		
32	Figure 1.1-3	Ecological Risk Assessment Roadmap1-6		
33	Figure 1.2-1	GE Plant Area, Removal Action Areas1-7		
34	Figure 1.4-1	Housatonic River, Reach 5		
35	Figure 1.4-2	Housatonic River, Reaches 5A and 5B1-14		

Title

1	Figure 1.4-3	Housatonic River, Reaches 5C and 5D 1-15
2	Figure 1.4-4	Housatonic River, Reach 6 1-16
3	Figure 1.4-5	Housatonic River, Reaches 7 to 91-18
4	Figure 1.4-6	Housatonic River, Reaches 10 to 13 1-19
5	Figure 1.4-7	Housatonic River, Reaches 14 to 171-21
6	Figure 1.5-1	Eight-Step Ecological Risk Assessment Process for Superfund 1-24
7	Figure 2.2-1	Housatonic River Ecological Characterization2-9
8	Figure 2.5-1	Biphenyl and Representative PCB Congeners
9 10	Figure 2.5-2	Distribution of tPCB Concentrations Detected in Sediment Samples from the GE Facility to Long Island Sound
11	Figure 2.5-3	Mean Total Sediment PCB Concentrations by Reach
12 13	Figure 2.5-4	Mean Total Surficial Soil PCB Concentrations at Floodplain Locations by Reach
14 15	Figure 2.5-5	Spatially Weighted tPCB Concentrations in Floodplain Soil in the Primary Study Area (Tile 1 of 7)
16 17	Figure 2.5-6	Spatially Weighted tPCB Concentrations in Floodplain Soil in the Primary Study Area (Tile 2 of 7)
18 19	Figure 2.5-7	Spatially Weighted tPCB Concentrations in Floodplain Soil in the Primary Study Area (Tile 3 of 7)
20 21	Figure 2.5-8	Spatially Weighted tPCB Concentrations in Floodplain Soil in the Primary Study Area (Tile 4 of 7)
22 23	Figure 2.5-9	Spatially Weighted tPCB Concentrations in Floodplain Soil in the Primary Study Area (Tile 5 of 7)
24 25	Figure 2.5-10	Spatially Weighted tPCB Concentrations in Floodplain Soil in the Primary Study Area (Tile 6 of 7)2-35
26 27	Figure 2.5-11	Spatially Weighted tPCB Concentrations in Floodplain Soil in the Primary Study Area (Tile 7 of 7)
28 29	Figure 2.5-12	Total PCB Concentrations Measured in all Surface Water SamplesCollected from the Housatonic River Since 1980
30 31	Figure 2.5-13	Total Surface Water PCB Concentrations by River Mile (Data Collected Since 1996)
32 33	Figure 2.5-14	Total PCB Concentration (mg/kg wet weight) in Selected Biota (Excluding Fish) for Reaches 5 and 6

Title

1	Figure 2.5-15	Total PCB Concentration (mg/kg wet weight) in Reaches 5 and 6 Fish 2-39
2 3	Figure 2.5-16	EPA Fish Collections (1998-2000) - Median tPCB Concentrations - All Ages by Subreach in the PSA
4 5	Figure 2.5-17	EPA Fish Collections (1998-2000) - Median Lipid Normalized PCB Concentrations - All Ages by Subreach in the PSA
6 7	Figure 2.7-1	Housatonic River Ecological Risk Assessment Conceptual Model: Principal Exposure Pathways for PCBs2-55
8	Figure 2.9-1	Example Endpoint Weighting Sheet
9	Figure 2.9-2	Scoring Sheet for Evidence of Harm and Magnitude
10	Figure 2.9-3	Example of Qualitative Assessment
11 12	Figure 3.1-1	Conceptual Model Diagram: Exposure Pathways for Benthic Invertebrates Exposed to Contaminants of Concern (COCs) in the Housatonic River
13 14	Figure 3.1-2	Overview of Approach Used to Assess Exposure of Benthic Invertebrates to Contaminants of Concern (COCs) in the Housatonic River
15 16	Figure 3.1-3	Overview of Approach Used to Assess the Effects of Contaminants of Concern (COCs) to Benthic Invertebrates in the Housatonic River
17 18	Figure 3.1-4	Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Benthic Invertebrates in the Housatonic River
19 20	Figure 3.1-5	Summary of Studies Conducted in Conjunction with Ecological Risk Assessment for Benthic Invertebrates, and Linkage to ERA
21 22	Figure 3.2-1	Benthic Invertebrate Sampling Locations and Simplified Station Identifiers
23 24	Figure 3.2-2	Median Percent Fines and Percent TOC by Sampling Location, for Benthic Community Grab Samples
25 26 27	Figure 3.2-3	Concentrations of tPCBs in Sediment by Sampling Location for Individual Benthic Community Grab Samples, and Associated Measures of Central Tendency
28 29 30	Figure 3.2-4	Comparison of tPCB Concentrations in Sediment Collected at Benthic Toxicity Sampling Locations, from Various Sampling Efforts Conducted in 1999
31 32	Figure 3.2-5	Medians and Quartiles of PCB and TOC in the Housatonic River PSA, Subdivided by River Reach and 0.25 Mile Subreaches
33 34	Figure 3.2-6	Concentrations of tPCBs in Benthic Invertebrate Tissues by Location and Functional Feeding Group

Title

1 2	Figure 3.3-1	Survival of <i>Hyalella azteca</i> in Chronic Laboratory Toxicity Tests, at Three Time Periods (28 days, 35 days, 42 days)
3 4	Figure 3.3-2	Growth of <i>Hyalella azteca</i> in Chronic Laboratory Toxicity Tests, at Two Time Periods (28 days, 42 days)
5 6	Figure 3.3-3	Reproduction of <i>Hyalella azteca</i> in Chronic Laboratory Toxicity Tests, Based on Mean Number of Young (Days 28-42)
7 8	Figure 3.3-4	Survival and Emergence of <i>Chironomus tentans</i> in Chronic Laboratory Toxicity Tests (43 days)
9 10	Figure 3.3-5	Growth Endpoints for <i>Chironomus tentans</i> in Chronic Laboratory Toxicity Test (20 days)
11 12	Figure 3.3-6	Survival of <i>Hyalella azteca</i> in 48-hour Low Flow In Situ Toxicity Tests Conducted 14-16 June 1999
13 14	Figure 3.3-7	Survival of <i>Hyalella azteca</i> in 10-day Low Flow In Situ Toxicity Tests Conducted 17-27 June 1999
15 16	Figure 3.3-8	Survival of <i>Chironomus tentans</i> in 48-hour Low Flow In Situ Toxicity Tests Conducted 14-16 June 1999
17 18	Figure 3.3-9	Survival of <i>Chironomus tentans</i> in 10-day Low Flow In Situ Toxicity Tests Conducted 17-27 June 1999
19 20	Figure 3.3-10	Survival of <i>Daphnia magna</i> in 48-hour Low Flow In Situ Toxicity Tests Conducted 14-16 June 1999
21 22	Figure 3.3-11	Survival of <i>Lumbriculus variegatus</i> in 48-hour Low Flow In Situ Toxicity Tests Conducted 14-16 June 1999
23 24	Figure 3.3-12	Statistical Endpoints for Toxicity Data, with Comparisons to Station A1 (sorted by LC ₅₀ /EC ₅₀ value)
25 26 27	Figure 3.3-13	Segmented Linear Regression Models Applied to Toxicity Data, Relating Relative Performance Proportion (RPP) to Bulk Sediment tPCB Concentrations (mg/kg)
28 29	Figure 3.3-14	Combined Effects and No-Effects Levels for PCB Concentrations (mg/kg wet) in Benthic Invertebrate Tissue Samples – tPCBs and Aroclor 1254 3-55
30 31	Figure 3.3-15	Average Ranks Analysis for Six Benthic Community Metrics, with Equal Weighting Assigned to Each Metric
32 33	Figure 3.3-16	Multidimensional Scaling for Benthic Community Health Metrics, Showing Metric Medians on MDS Plot

Title

1 2 3	Figure 3.4-1	Hazard Quotients (Median and Range) Based on Median Sediment Chemistry for tPCBs, for Samples Collected in 1999 Close to Sediment Quality Triad Stations
4 5	Figure 3.4-2	Hazard Quotients (Median, Range) Based on Overlying Water PCB Concentrations, Measured Synoptic with In Situ Toxicity Tests
6 7	Figure 3.4-3	Hazard Quotients for tPCB Tissue Residues in Benthic Invertebrates, Relative to Two Effects Thresholds Derived from Literature Studies
8 9	Figure 3.4-4	Weight-of-Evidence Evaluation of Housatonic River Benthic Sampling Locations, with Indications of Alteration/Risk Relative to Background
10 11	Figure 4.1-1	Conceptual Model Diagram: Exposure Pathways for Amphibians Exposed to Contaminants of Concern (COCs) in the Housatonic River PSA
12 13	Figure 4.1-2	Overview of Approach Used to Assess Exposure of Amphibians to Contaminants of Concern (COCs) in the Housatonic River PSA
14 15	Figure 4.1-3	Overview of Approach Used to Assess the Effects of Contaminants of Concern (COCs) to Amphibians in the Housatonic River PSA
16 17	Figure 4.1-4	Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Amphibians in the Housatonic River PSA
18	Figure 4.2-1	Leopard Frog Exposure Pathways
19	Figure 4.2-2	Wood Frog Exposure Pathways
20 21	Figure 4.2-3	General Model of Leopard Frog Vernal Pool (VP) Reproduction and Development Study
22 23	Figure 4.2-4	General Model of Wood Frog Vernal Pool (VP) Reproduction and Development Study
24 25 26	Figure 4.3-1	Frequency Distribution and Cumulative Percentage of Sediment tPCB Exposure Point Concentrations for 66 PSA Temporary and Permanent Pools (Based on EPA Spatially Weighted Data)
27 28	Figure 4.3-2	Total Sediment PCB Concentrations for Wood Frog Vernal Pool Study (mean, n =2) and Leopard Frog Reproduction/Development Study
29 30	Figure 4.3-3	Comparison of Leopard Frog Tissue Samples to Sediment tPCB Concentrations (Reproductive Study Data and Spatially Weighted Data) 4-28
31 32 33	Figure 4.3-4	Comparison of tPCB Concentrations in Tissue (in Various Phases of the Wood Frog Developmental Study) with Mean Vernal Pool and Spatially Weighted Mean tPCB Concentrations in Sediment

Title

1 2 3	Figure 4.4-1	Comparison of Percent Abnormal Sperm Heads (Mean) from Male Adult Chemical Analysis Leopard Frogs, with Mean Sediment tPCB and Spatially Weighted Mean tPCB
4 5 6	Figure 4.4-2	Comparison of Mean Percent of Oocytes at Stage VI (Mature) for Female Leopard Frogs, with Mean Sediment tPCB and Spatially Weighted Mean tPCB
7 8 9	Figure 4.4-3	Days to Gosner Developmental Stage 26 (±1) and Final Developmental Stage Reached at End-of-Test, with Sediment tPCB and Spatially Weighted Mean tPCB: 2000 Leopard Frog Study
10 11 12	Figure 4.4-4	Comparison of Phase I Larval Wood Frog Malformations as Gosner Developmental Stage 20-24 to Mean Sediment tPCB and Spatially Weighted Mean tPCB
13 14	Figure 4.4-5	Incidence of Malformation in Phase I Wood Frog Metamorphs, with Mean Sediment tPCB and Spatially Weighted Mean tPCB
15 16	Figure 4.4-6	Incidence of Malformation in Phase I Wood Frog Metamorphs, Phase I Metamorph Tissue tPCB
17 18	Figure 4.4-7	Ratio of Males to Females in Phase III Wood Frog Metamorphs, with Sediment tPCB and Spatially Weighted Mean tPCBs
19 20	Figure 4.4-8	Ratio of Males to Females in Phase III Wood Frog Metamorphs, with Tissue tPCBs
21 22	Figure 4.4-9	Percent Malformation in Phase III Wood Frog Metamorphs, with Sediment tPCBs
23 24	Figure 4.4-10	Percent Malformation in Phase III Wood Frog Metamorphs, with Tissue tPCBs
25 26	Figure 4.4-11	Summary of Available Literature Effects Data on PCB Tissue Residues in Anuran Amphibians
27 28	Figure 5.1-1	Conceptual Model Diagram: Exposure Pathways for Fish Exposed to COCs in the Housatonic River
29 30	Figure 5.1-2	Overview of Approach Used To Assess Exposure of Fish to COCs in the Housatonic River
31 32	Figure 5.1-3	Overview of Approach Used To Assess the Effects of COCs to Fish in the Housatonic River
33 34	Figure 5.1-4	Overview of Approach Used To Characterize the Risks of COCs to Fish in the Housatonic River

Title

1 2	Figure 5.2-1	Box-and-Whisker Plots of Lipid-Normalized tPCB Concentrations Plotted by Sample Type for Species with Multiple Sample Types
3 4	Figure 5.2-2	Box-and-Whisker Plot of Largemouth Bass tPCB Concentrations (Lipid- Normalized) Versus Age
5	Figure 5.3-1	Literature-Based PCB Fish Tissue Effects Concentrations
6	Figure 5.3-2	Literature-Based TCDD (TEQ) Fish Tissue Effects Concentrations 5-27
7 8	Figure 5.3-3	Effects of in Ovo Exposure to Increasing Doses of Reach 6 Extracts on Largemouth Bass at Swim-Up
9 10	Figure 5.3-4	Effects of in Ovo Exposure to Increasing Doses of Reach 5BC Extracts on Medaka at 5d Post Swim-Up
11 12	Figure 5.3-5	Effects of in Ovo Exposure to Increasing Doses of Reach 6 Extracts on Medaka at 5d Post Swim-Up
13 14	Figure 5.3-6	Effects of in Ovo Exposure to Increasing Doses of Reach 5BC Extracts on Rainbow Trout at 600 DTU
15 16	Figure 5.3-7	Effects of in Ovo Exposure to Increasing Doses of Reach 6 Extracts on Rainbow Trout at 600 DTU
17 18	Figure 5.3-8	TEQ ED ₅₀ Estimates for Fish Exposed to Housatonic River Extracts and PCB-126 and TCDD Standards (Logarithmic Scale)
19 20	Figure 5.3-9	tPCB ED ₅₀ Estimates for Fish Exposed to Housatonic River Extracts and PCB-126 and TCDD Standards (Logarithmic Scale)
21 22 23	Figure 5.4-1	Hazard Quotients for tPCBs in PSA Fish Based on Comparison to the Mean Site-Specific Fish Toxicity Threshold (49 mg/kg tPCB) (Logarithmic Scale)
24 25 26	Figure 5.4-2	Complementary Cumulative Distribution Plot for tPCB Concentrations in Whole Body Tissue Compared to Effects Concentrations – Brown Bullhead
27 28 29	Figure 5.4-3	Complementary Cumulative Distribution Plot for tPCB Concentrations in Whole Body Tissue Compared to Effects Concentrations – Largemouth Bass
30 31	Figure 5.4-4	Complementary Cumulative Distribution Plot for tPCB Concentrations in Whole Body Tissue Compared to Effects Concentrations – Pumpkinseed 5-59
32 33	Figure 5.4-5	Complementary Cumulative Distribution Plot for tPCB Concentrations in Whole Body Tissue Compared to Effects Concentrations – White Sucker 5-60
34 35	Figure 5.4-6	Complementary Cumulative Distribution Plot for tPCB Concentrations in Whole Body Tissue Compared to Effects Concentrations – Yellow Perch 5-61

Title

1 2 3	Figure 5.4-7	Hazard Quotients for TEQ for Fish in Primary Study Area (PSA) Based on Comparison to the Average Site-Specific Tissue Effects Threshold (42 ng/kg TEQ) (Logarithmic Scale)
4 5 6	Figure 5.4-8	Complementary Cumulative Distribution Plot for TEQ Concentrations in Whole Body Tissue Compared to Effects Concentrations for All Species Using DL Substitution for Non-Detects
7 8	Figure 6.1-1	Conceptual Model for the Assessment of Risks from tPCBs and TEQ to Wildlife in the Housatonic River Primary Study Area
9 10	Figure 6.1-2	Framework Used to Model Exposure of Wildlife Species to Contaminants of Concern (COCs) in the Housatonic River PSA
11 12	Figure 6.1-3	Approach Used to Model Effects of Contaminants of Concern (COCs) to Representative Species in the Housatonic River PSA
13 14	Figure 6.1-4	Approach Used to Characterize the Risks from Contaminants of Concern (COCs) to Representative Species in the Housatonic River PSA
15 16	Figure 6.4-1	Molecular Structure of the Planar Chlorinated Hydrocarbon, 2,3,7,8- Tetrachlorodibenzo-p-dioxin
17 18	Figure 6.4-2	Decision Tree for Determining Appropriate Treatment of Data with Non- Detects and Co-Elution
19 20	Figure 6.5-1	Example Exposure Distribution from Monte Carlo and Probability Bounds Analyses (TDI = total daily intake of tPCBs)
21 22	Figure 6.6-1	Decision Criteria Used to Characterize Effects for Each Wildlife Receptor-COC Combination
23 24	Figure 6.7-1	Example Risk Curves Indicating Low, Intermediate, and High Risk Categories
25 26	Figure 7.1-1	Conceptual Model Diagram: Exposure Pathways for Insectivorous Birds Exposed to Contaminants of Concern in the Housatonic River PSA
27 28 29	Figure 7.1-2	Overview of Approach Used to Assess Modeled Exposure of Insectivorous Birds to Contaminants of Concern in the Housatonic River PSA
30 31 32	Figure 7.1-3	Overview of Approach Used to Assess the Modeled Effects of Contaminants of Concern to Insectivorous Birds in the Housatonic River PSA
33 34	Figure 7.1-4	Overview of Approach Used to Characterize the Risks of Contaminants of Concern to Insectivorous Birds in the Housatonic River PSA
35	Figure 7.2-1	Tree Swallow (<i>Tachycineta bicolor</i>)

Title

1	Figure 7.2-2	American Robin (<i>Turdus migratorius</i>)	7-11
2 3	Figure 7.3-1	Tree Swallow Nest Box Locations and Soil Invertebrate Sampling Locations in the Housatonic River PSA	7-14
4	Figure 7.3-2	TDI Exposure Model Input Distributions for Tree Swallows	7-19
5 6	Figure 7.3-3	Microexposure Model Input Distributions for Maternal Transfer for Tree Swallows	7-20
7	Figure 7.3-4	TDI Exposure Model Input Distributions for American Robin	7-21
8	Figure 7.3-5	Concentration of tPCBs in Prey of Tree Swallows	7-27
9	Figure 7.3-6	Concentration of TEQ in Prey of Tree Swallows	7-28
10	Figure 7.3-7	Concentration of tPCBs in Prey of American Robins	7-29
11	Figure 7.3-8	Concentration of TEQ in Prey of American Robins	7-30
12 13	Figure 7.3-9	Tree Swallow TDI Exposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Holmes Road	7-31
14 15	Figure 7.3-10	Tree Swallow TDI Exposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at New Lenox Road	7-32
16 17	Figure 7.3-11	Tree Swallow TDI Exposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Roaring Brook	7-33
18 19	Figure 7.3-12	Tree Swallow TDI Exposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Southwest Branch	7-34
20 21	Figure 7.3-13	Tree Swallow TDI Exposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Threemile Pond	7-35
22 23	Figure 7.3-14	Tree Swallow TDI Exposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Taconic Valley	7-36
24 25	Figure 7.3-15	Tree Swallow TDI Exposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Holmes Road	7-37
26 27	Figure 7.3-16	Tree Swallow TDI Exposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at New Lenox Road	7-38
28 29	Figure 7.3-17	Tree Swallow TDI Exposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Roaring Brook	7-39
30 31	Figure 7.3-18	Tree Swallow TDI Exposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Southwest Branch	7-40
32 33	Figure 7.3-19	Tree Swallow TDI Exposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Threemile Pond	7-41

Title

1 2	Figure 7.3-20	Tree Swallow TDI Exposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Taconic Valley
3 4	Figure 7.3-21	Exposure of American Robins to tPCBs at Site 13 of the Housatonic River PSA
5 6	Figure 7.3-22	Exposure of American Robins to tPCBs at Site 14 of the Housatonic River PSA
7 8	Figure 7.3-23	Exposure of American Robins to tPCBs at Site 15 of the Housatonic River PSA
9 10	Figure 7.3-24	Exposure of American Robins to 2,3,7,8-TCDD TEQ at Site 13 of the Housatonic River PSA
11 12	Figure 7.3-25	Exposure of American Robins to 2,3,7,8-TCDD TEQ at Site 14 of the Housatonic River PSA
13 14	Figure 7.3-26	Exposure of American Robins to 2,3,7,8-TCDD TEQ at Site 15 of the Housatonic River PSA
15 16	Figure 7.3-27	Tree Swallow Microexposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Holmes Road
17 18	Figure 7.3-28	Tree Swallow Microexposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at New Lenox Road
19 20	Figure 7.3-29	Tree Swallow Microexposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Roaring Brook7-52
21 22	Figure 7.3-30	Tree Swallow Microexposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Southwest Branch
23 24	Figure 7.3-31	Tree Swallow Microexposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Threemile Pond
25 26	Figure 7.3-32	Tree Swallow Microexposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Taconic Valley
27 28	Figure 7.3-33	Tree Swallow Microexposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Holmes Road7-56
29 30	Figure 7.3-34	Tree Swallow Microexposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at New Lenox Road
31 32	Figure 7.3-35	Tree Swallow Microexposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Roaring Brook7-58
33 34	Figure 7.3-36	Tree Swallow Microexposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Southwest Branch

Title

1 2	Figure 7.3-37	Tree Swallow Microexposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Threemile Pond
3 4	Figure 7.3-38	Tree Swallow Microexposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Taconic Valley
5	Figure 7.4-1	Effects of Aroclor 1254/1260 on Avian Species (mg/kg bw/d)7-66
6	Figure 7.4-2	Effects of 2,3,7,8-TCDD TEQ on Avian Species (ng TEQ/kg bw/d)7-67
7 8	Figure 8.1-1	Conceptual Model for Exposure of Piscivorous Birds to tPCBs and TEQ in the Housatonic River PSA
9 10	Figure 8.1-2	Overview of Approach Used to Assess Modeled Exposure of Piscivorous Birds to Contaminants of Concern (COCs) in the Housatonic River PSA8-5
11 12 13	Figure 8.1-3	Overview of Approach Used to Assess the Modeled Effects of Contaminants of Concern (COCs) to Piscivorous Birds in the Housatonic River PSA
14 15	Figure 8.1-4	Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Piscivorous Birds in the Housatonic River PSA
16	Figure 8.2-1	Belted Kingfisher (Ceryle alcyon)
17	Figure 8.2-2	Osprey (Pandion haliaetus)
18	Figure 8.3-1	Exposure Model Input Distributions for Belted Kingfisher
19	Figure 8.3-2	Exposure Model Input Distributions for Osprey
20 21	Figure 8.3-3	Concentrations of tPCBs in the Prey of Belted Kingfishers in the Housatonic River PSA and Reference Areas
22 23	Figure 8.3-4	Concentrations of TEQ in the Prey of Belted Kingfishers in the Housatonic River PSA and Reference Areas
24 25	Figure 8.3-5	Concentrations of tPCBs in the Prey of Ospreys in the Housatonic River PSA and Reference Areas
26 27	Figure 8.3-6	Concentrations of TEQ in the Prey of Ospreys in the Housatonic River PSA and Reference Areas
28 29	Figure 8.3-7	Exposure of Belted Kingfishers to tPCBs in Reach 5 of the Housatonic River PSA
30 31	Figure 8.3-8	Exposure of Belted Kingfishers to tPCBs in Reach 6 of the Housatonic River PSA
32 33	Figure 8.3-9	Exposure of Belted Kingfishers to tPCBs in the Housatonic River Upstream Reference Area

Title

1 2	Figure 8.3-10	Exposure of Belted Kingfishers to tPCBs in the Threemile Pond Reference Area
3 4	Figure 8.3-11	Exposure of Belted Kingfishers to TEQ in Reach 5 of the Housatonic River PSA
5 6	Figure 8.3-12	Exposure of Belted Kingfishers to TEQ in Reach 6 of the Housatonic River PSA
7 8	Figure 8.3-13	Exposure of Belted Kingfishers to TEQ in the Housatonic River Upstream Reference Area
9 10	Figure 8.3-14	Exposure of Belted Kingfishers to TEQ in the Threemile Pond Reference Area
11 12	Figure 8.3-15	Exposure of Ospreys to tPCBs in Reaches 5 and 6 of the Housatonic River PSA
13 14	Figure 8.3-16	Exposure of Ospreys to tPCBs in the Housatonic River Upstream Reference Area
15	Figure 8.3-17	Exposure of Ospreys to tPCBs in the Threemile Pond Reference Area
16 17	Figure 8.3-18	Exposure of Ospreys to TEQ in Reaches 5 and 6 of the Housatonic River PSA
18	Figure 8.3-19	Exposure of Ospreys to TEQ in the Upstream Reference Area
19	Figure 8.3-20	Exposure of Ospreys to TEQ in the Threemile Pond Reference Area
20	Figure 8.4-1	Effects of Aroclor 1254/1260 on Avian Species (mg/kg bw/d)
21	Figure 8.4-2	Effects of 2,3,7,8-TCDD TEQ on Avian Species (ng TEQ/kg bw/d)
22 23	Figure 9.1-1	Conceptual Model Diagram: Exposure Pathways for Piscivorous Mammals Exposed to COCs in the Housatonic River PSA
24 25 26	Figure 9.1-2	Overview of Approach Used to Assess Modeled Exposure of Piscivorous Mammals to Contaminants of Concern (COCs) in the Housatonic River PSA
27 28 29	Figure 9.1-3	Overview of Approach Used to Assess the Modeled Effects of Contaminants of Concern (COCs) to Piscivorous Mammals in the Housatonic River PSA
30 31	Figure 9.1-4	Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Piscivorous Mammals in the Housatonic River PSA 9-7
32	Figure 9.2-1	Mink (Mustela vison)
33	Figure 9.2-2	River Otter (Lutra canadensis)
34	Figure 9.3-1	Input Distributions Used in Exposure Modeling for Mink

Title

1	Figure 9.3-2	Input Distributions Used in Exposure Modeling for River Otter	9-16
2	Figure 9.3-3	Concentrations of tPCBs in Prey of Mink	9-19
3	Figure 9.3-4	Concentrations of TEQ in Prey of Mink	. 9-20
4	Figure 9.3-5	Concentrations of tPCBs in Prey of River Otter	. 9-20
5	Figure 9.3-6	Concentrations of TEQ in Prey of River Otter	. 9-21
6	Figure 9.3-7	Exposure of Mink to tPCBs in Reach 5 of the Housatonic River	. 9-22
7	Figure 9.3-8	Exposure of Mink to tPCBs in Reach 6 of the Housatonic River	. 9-23
8 9	Figure 9.3-9	Exposure of Mink to tPCBs in the Housatonic River Upstream Reference Area	. 9-24
10	Figure 9.3-10	Exposure of Mink to tPCBs in the Threemile Pond Reference Area	. 9-25
11 12	Figure 9.3-11	Exposure of Mink to 2,3,7,8-TCDD TEQ in Reach 5 of the Housatonic River	. 9-26
13 14	Figure 9.3-12	Exposure of Mink to 2,3,7,8-TCDD TEQ in Reach 6 of the Housatonic River	. 9-27
15 16	Figure 9.3-13	Exposure of Mink to 2,3,7,8-TCDD TEQ in the Housatonic River Upstream Reference Area	. 9-28
17 18	Figure 9.3-14	Exposure of Mink to 2,3,7,8-TCDD TEQ in the Threemile Pond Reference Area.	. 9-29
19 20	Figure 9.3-15	Exposure of River Otter to tPCBs in Reaches 5 and 6 of the Housatonic River	.9-30
21 22	Figure 9.3-16	Exposure of River Otter to tPCBs in the Housatonic River Upstream Reference Area	. 9-31
23	Figure 9.3-17	Exposure of River Otter to tPCBs in the Threemile Pond Reference Area	. 9-32
24 25	Figure 9.3-18	Exposure of River Otter to 2,3,7,8-TCDD TEQ in Reaches 5 and 6 of the Housatonic River	.9-33
26 27	Figure 9.3-19	Exposure of River Otter to 2,3,7,8-TCDD TEQ in the Upstream Reference Area	. 9-34
28 29	Figure 9.3-20	Exposure of River Otter to 2,3,7,8-TCDD TEQ in the Threemile Pond Reference Area	. 9-35
30	Figure 9.4-1	Dose Response Curve for Effects of tPCBs on Fecundity of Mink	. 9-48
31 32	Figure 9.5-1	Total PCB Risk to Mink Exposed to tPCBs in Reach 5 of the Housatonic River	9-55
			. , 55

Title

1 2	Figure 9.5-3	Total PCB Risk to Mink Exposed to tPCBs in Reach 6 of the Housatonic River
3	Figure 9.5-4	Total PCB Risk to Mink (10% Foraging Time in Reach 6)
4	Figure 9.5-5	Total PCB Risk to Mink Foraging in the Upstream Reference Area
5 6	Figure 9.5-6	Total PCB Risk to Mink (10% Foraging Time in the Upstream Reference Area)
7 8	Figure 9.5-7	Total PCB Risk to Mink Exposed to tPCBs in the Threemile Pond Reference Area
9 10	Figure 9.5-8	Total PCB Risk to Mink (10% Foraging Time in the Threemile Pond Reference Area)
11 12	Figure 9.5-9	Total PCB Risk to River Otter Exposed to tPCBs in Reaches 5 and 6 of the Housatonic River
13	Figure 9.5-10	Total PCB Risk to River Otter (10% Foraging Time in Reaches 5 and 6)9-64
14	Figure 9.5-11	Total PCB Risk to River Otter Foraging in the Upstream Reference Area9-65
15 16	Figure 9.5-12	Total PCB Risk to River Otter (10% Foraging Time in the Upstream Reference Area)
17 18	Figure 9.5-13	Total PCB Risk to River Otter Exposed to tPCBs in the Threemile Pond Reference Area
19 20	Figure 9.5-14	Total PCB Risk to River Otter (10% Foraging Time in the Threemile Pond Reference Area)
21 22 23	Figure 10.1-1	Conceptual Model Diagram: Exposure Pathways for Omnivorous and Carnivorous Mammals Exposed to Contaminants of Concern (COCs) in the Housatonic River PSA
24 25	Figure 10.1-2	Framework Used to Model Exposure of Wildlife Species to Contaminants of Concern (COCs) in the Housatonic River PSA
26 27	Figure 10.1-3	Approach Used to Model Effects of Contaminants of Concern (COCs) to Representative Species in the Housatonic River PSA
28 29 30	Figure 10.1-4	Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Omnivorous and Carnivorous Mammals in the Housatonic River PSA
31	Figure 10.2-1	Red Fox (Vulpes vulpes)
32	Figure 10.2-2	Northern Short-Tailed Shrew (Blarina brevicauda)10-10
33	Figure 10.3-1	Input Distributions for the Exposure Modeling of the Red Fox 10-15
34	Figure 10.3-2	Input Distributions for the Exposure Modeling of Short-Tailed Shrew 10-16

Title

1 2	Figure 10.3-3	Concentrations of tPCBs in Prey of Northern Short-Tailed Shrew (n=1 for invertebrates and earthworms)
3 4	Figure 10.3-4	Concentrations of TEQ in Prey of Northern Short-Tailed Shrew (n=1 for invertebrates and earthworms)
5 6	Figure 10.3-5	Exceedance Probability Distribution for Red Fox Exposed to tPCBs in Reach 5 of the PSA
7 8	Figure 10.3-6	Exceedance Probability Distribution for Red Fox Exposed to TEQ in Reach 5 of the PSA
9 10	Figure 10.3-7	Exceedance Probability Distribution for Short-Tailed Shrew Exposed to tPCBs at Location 13 of the PSA
11 12	Figure 10.3-8	Exceedance Probability Distribution for Short-Tailed Shrew Exposed to tPCBs at Location 14 of the PSA
13 14	Figure 10.3-9	Exceedance Probability Distribution for Short-Tailed Shrew Exposed to tPCBs at Location 15 of the PSA
15 16	Figure 10.3-10	Exceedance Probability Distribution for Short-Tailed Shrew Exposed to TEQ at Location 13 of the PSA
17 18	Figure 10.3-11	Exceedance Probability Distribution for Short-Tailed Shrew Exposed to TEQ at Location 14 of the PSA
19 20	Figure 10.3-12	Exceedance Probability Distribution for Short-Tailed Shrew Exposed to TEQ at Location 15 of the PSA
21	Figure 10.4-1	Dose-Response Curve for Effects of tPCBs on Mortality at Birth of Rats 10-38
22 23	Figure 10.4-2	Dose-Response Curve for Effects of TEQ on Reproductive Fecundity of Rat
24 25	Figure 10.4-3	Dose Response Curve for Effects of TEQ on Reproductive Fecundity of Mouse
26 27	Figure 10.5-1	Risk Function for Red Fox Exposed to tPCBs in Reach 5 of the Housatonic River
28 29	Figure 10.5-2	Risk Function for Red Fox Exposed to TEQ in Reach 5 of the Housatonic River
30 31	Figure 10.5-3	Risk Function for Short-Tailed Shrew Exposed to tPCBs at Location 13 of the PSA
32 33	Figure 10.5-4	Risk Function for Short-Tailed Shrew Exposed to tPCBs at Location 14 of the PSA

Title

1 2	Figure 10.5-5	Risk Function for Short-Tailed Shrew Exposed to tPCBs at Location 15 of the PSA
3 4	Figure 10.5-6	Risk Function for Short-Tailed Shrew Exposed to TEQ at Location 13 of the PSA
5 6	Figure 10.5-7	Risk Function for Short-Tailed Shrew Exposed to TEQ at Location 14 of the PSA
7 8	Figure 10.5-8	Risk Function for Short-Tailed Shrew Exposed to TEQ at Location 15 of the PSA
9 10	Figure 11.1-1	Conceptual Model Diagram: Exposure Pathways for T&E Species Exposed to COCs in the Housatonic PSA
11	Figure 11.1-2	Approach Used to Assess Modeled Exposure of T&E Species to COCs 11-4
12	Figure 11.1-3	Approach Used to Assess the Modeled Effects of COCs to T&E Species 11-5
13	Figure 11.1-4	Approach Used to Characterize Risks of COCs to T&E Species
14	Figure 11.2-1	Input Distributions for the Exposure Modeling of Bald Eagle 11-16
15	Figure 11.2-2	Input Distributions for the Exposure Modeling of American Bittern
16	Figure 11.2-3	Input Distributions for the Exposure Modeling of Small-Footed Myotis 11-17
17	Figure 11.2-4	Median Concentrations of tPCBs in Prey of Bald Eagles
18	Figure 11.2-5	Median Concentrations of TEQ in Prey of Bald Eagles
19	Figure 11.2-6	Median Concentrations of tPCBs in Prey of American Bittern11-22
20	Figure 11.2-7	Median Concentrations of TEQ in Prey of American Bittern 11-22
21 22	Figure 11.2-8	Total Daily Intake (TDI) of tPCBs by Bald Eagles in the Housatonic River Primary Study Area
23 24	Figure 11.2-9	Total Daily Intake (TDI) of TEQ by Bald Eagles in the Housatonic River Primary Study Area
25 26	Figure 11.2-10	Bald Eagle Egg Exposure to PCBs in the Housatonic River Primary Study Area
27 28	Figure 11.2-11	Bald Eagle Egg Exposure to TEQ in the Housatonic River Primary Study Area
29 30	Figure 11.2-12	Total Daily Intake (TDI) of tPCBs by American Bittern in Reach 5A of the Housatonic River Primary Study Area
31 32	Figure 11.2-13	Total Daily Intake (TDI) of tPCBs by American Bittern in Reach 5B of the Housatonic River Primary Study Area

Title

1 2	Figure 11.2-14	Total Daily Intake (TDI) of tPCBs by American Bittern in Reach 5C of the Housatonic River Primary Study Area
3 4	Figure 11.2-15	Total Daily Intake (TDI) of tPCBs by American Bittern in Reaches 5D and 6 of the Housatonic River Primary Study Area
5 6	Figure 11.2-16	Total Daily Intake (TDI) of TEQ by American Bittern in the Housatonic River Primary Study Area
7 8	Figure 11.2-17	American Bittern Egg Exposure to tPCBs in Reach 5A of the Housatonic River Primary Study Area
9 10	Figure 11.2-18	American Bittern Egg Exposure to tPCBs in Reach 5B of the Housatonic River Primary Study Area
11 12	Figure 11.2-19	American Bittern Egg Exposure to tPCBs in Reach 5C of the Housatonic River Primary Study Area
13 14	Figure 11.2-20	American Bittern Egg Exposure to tPCBs in Reaches 5D and 6 of the Housatonic River Primary Study Area
15 16	Figure 11.2-21	American Bittern Egg Exposure to TEQ in Reaches 5 and 6 of the Housatonic River Primary Study Area
17 18	Figure 11.2-22	Total Daily Intake (TDI) of tPCBs by Small-Footed Myotis in Reach 5 of the Housatonic River Primary Study Area
19 20	Figure 11.2-23	Total Daily Intake (TDI) of TEQ by Small-Footed Myotis in Reach 5 of the Housatonic River Primary Study Area
21	Figure 11.3-1	Dose-Response Curve for Effects of tPCBs on Mortality at Birth of Rats 11-43
22	Figure 11.3-2	Dose-Response Curve for Effects of TEQ on Mortality at Birth of Rats 11-44
23 24	Figure 11.4-1	Risk Curves for Bald Eagles Exposed to tPCBs in the Housatonic River Primary Study Area
25 26	Figure 11.4-2	Risk Curves for Bald Eagles Exposed to TEQ in the Housatonic River Primary Study Area11-50
27 28	Figure 11.4-3	Risk for Bald Eagle Eggs Exposed to tPCBs in the Housatonic River Primary Study Area11-51
29 30	Figure 11.4-4	Risk for Bald Eagle Eggs Exposed to TEQ in the Housatonic River Primary Study Area
31 32	Figure 11.4-5	Risk Curves for American Bittern Exposed to tPCBs in Reach 5A of the Housatonic River Primary Study Area
33 34	Figure 11.4-6	Risk Curves for American Bittern Exposed to tPCBs in Reach 5B of the Housatonic River Primary Study Area

Title

1 2	Figure 11.4-7	Risk Curves for American Bittern Exposed to tPCBs in Reach 5C of the Housatonic River Primary Study Area
3 4	Figure 11.4-8	Risk Curves for American Bittern Exposed to tPCBs in Reaches 5D and 6 of the Housatonic River Primary Study Area
5 6	Figure 11.4-9	Risk Curves for American Bittern Exposed to TEQ in the Housatonic River Primary Study Area
7 8	Figure 11.4-10	Risk for American Bittern Eggs Exposed to tPCBs in Reach 5A of the Housatonic River Primary Study Area
9 10	Figure 11.4-11	Risk for American Bittern Eggs Exposed to tPCBs in Reach 5B of the Housatonic River Primary Study Area
11 12	Figure 11.4-12	Risk for American Bittern Eggs Exposed to tPCBs in Reach 5C of the Housatonic River Primary Study Area
13 14	Figure 11.4-13	Risk for American Bittern Eggs Exposed to tPCBs in Reaches 5D and 6 of the Housatonic River Primary Study Area
15 16	Figure 11.4-14	Risk for American Bittern Eggs Exposed to TEQ in the Housatonic River Primary Study Area
17 18	Figure 11.4-15	Risk Curves for Small-Footed Myotis Exposed to tPCBs in Reach 5 of the Housatonic River Primary Study Area
19 20	Figure 11.4-16	Risk Curves for Small-Footed Myotis Exposed to TEQ in Reach 5 of the Housatonic River Primary Study Area
21 22	Figure 12.2-1	Hazard Quotients for Aquatic Biota Exposed to tPCBs in the Housatonic River PSA
23 24	Figure 12.2-2	Hazard Quotients for Fish Exposed to 2,3,7,8-TCDD TEQ in the Housatonic River PSA
25 26	Figure 12.2-3	Hazard Quotients for Wildlife Exposed to tPCBs in the Housatonic River PSA
27 28	Figure 12.2-4	Hazard Quotients for Wildlife Exposed to 2,3,7,8-TCDD TEQ in the Housatonic River PSA
29 30	Figure 12.2-5	Assessment of Risk to Benthic Invertebrates Exposed to tPCBs Downstream of Woods Pond
31 32	Figure 12.2-6	Assessment of Risk to Amphibians in Floodplain Exposed to tPCBs Downstream of Woods Pond in Massachusetts
33 34	Figure 12.2-7	Assessment of Risk to Amphibians in Sediment Exposed to tPCBs Downstream of Woods Pond in Connecticut
LIST OF FIGURES (Continued)

Title

Page

1 2	Figure 12.2-8	Assessment of Risk to Warmwater Fish Exposed to tPCBs Downstream of Woods Pond
3 4	Figure 12.2-9	Assessment of Risk to Trout Exposed to tPCBs Downstream of Woods Pond
5 6	Figure 12.2-10	Assessment of Risk to Mink Exposed to tPCBs Downstream of Woods Pond
7 8	Figure 12.2-11	Assessment of Risk to Otter Exposed to tPCBs Downstream of Woods Pond
9 10	Figure 12.2-12	Assessment of Risk to Bald Eagle Exposed to tPCBs Downstream of Woods Pond
11		

1ES.ECOLOGICAL RISK ASSESSMENT2EXECUTIVE SUMMARY

3			Highlights of the ERA
4 5			 Total PCBs and other COCs in the PSA of the Housatonic River pose unacceptable risks to some assessment endpoints.
6 7 8 9 10			 Risk is high for benthic invertebrates, amphibians, and piscivorous mammals. Confidence in this conclusion is high because (1) multiple lines of evidence with concordant results were available, (2) models used to estimate risk were not conservative, and (3) consideration of uncertainty indicates a high probability of effects.
11 12 13 14 15			 Risk is moderate to high for some piscivorous and carnivorous birds, omnivorous and carnivorous mammals, and high for selected threatened and endangered bird and mammal species. There is uncertainty regarding these conclusions because corroborating lines of evidence were generally not available.
16 17			 Risk is low to moderate for fish and confidence in this conclusion is moderate.
18 19			 Risk is low for insectivorous birds, but confidence in this conclusion is not high.
20 21			 Other species not included in the quantitative risk assessments may also be at risk in the PSA.
22 23 24			 Assessment of risks to the most susceptible endpoints downstream of the PSA indicates that benthic invertebrates, amphibians, warmwater and coldwater fish, mink, river otter, and bald eagles may also be at risk.
25			
26	ES.1	OVE	RVIEW

The purpose of this ecological risk assessment (ERA) is to characterize and quantify the current and potential risks to biota exposed to contaminants of potential concern (COPCs) in the Housatonic River below the confluence of the East and West Branches (known as the "Rest of River"), focusing on polychlorinated biphenyls (PCBs) and other hazardous substances originating from the General Electric Company (GE) facility in Pittsfield, MA.

32 This information is synthesized, through a weight-of-evidence approach, into a discussion of the

33 nature and magnitude of the risks for the assessment endpoints, and the uncertainties associated

34 with the characterization of these risks. Multiple lines of evidence for each assessment endpoint

35 are evaluated, including where applicable or available:

- Field surveys/studies.
- Toxicity tests.
- 23

Comparison of effects in the literature to a site-specific exposure model.

4

5 ES.2 SITE DESCRIPTION AND HISTORY

6 The Housatonic River flows from east of Pittsfield, MA, to Long Island Sound and drains an area 7 of approximately 1,950 square miles in Massachusetts, New York, and Connecticut (Figure 8 The river is located in a predominantly rural area of western Massachusetts and ES-1). 9 Connecticut, where farming was the main occupation from colonial settlement through the late 10 1800s. The entire site, known as the General Electric/Housatonic River Site, consists of the 254-11 acre (103-hectare) GE manufacturing facility; the Housatonic River and associated riverbanks 12 and floodplain from Pittsfield, MA, to Long Island Sound; as well as other properties or areas 13 that have become contaminated as a result of GE facility operations.

14 Widespread contamination of the river downstream of the GE facility has resulted from the 15 transport of PCB-contaminated river sediment and floodplain soil by river flow, sediment 16 transport, and overbank flooding. Numerous studies conducted since 1988 have documented 17 PCB contamination of soil within the floodplain of the Housatonic River downstream of the GE 18 facility. PCBs have been detected in river sediment in Massachusetts as far downstream as the 19 border with Connecticut (BBL 1995), and in Connecticut as far as the Derby-Shelton Dam and 20 beyond into Long Island Sound (other sources have been identified downstream of this dam). 21 The PCBs detected in Housatonic River floodplain soil and sediment consist of predominantly 22 Aroclor 1260, with a minor contribution of Aroclor 1254.

The GE facility in Pittsfield is the only known source of PCBs found in the Housatonic River sediment and floodplain soil in Massachusetts. GE began operations in its present location in 1903. Three manufacturing divisions have operated at the GE facility (Transformer, Ordnance, and Plastics). Although GE performed many functions at the Pittsfield facility throughout the years, the activities of the Transformer Division, including the construction and repair of



| O:\gepitt\aprs\era_figures2.apr | layout - fig1.1-1 | o:\gepitt\epsfiles\plots\in\fig_es1.eps | 11:25 AM, 7/10/2003 |

- 1 electrical transformers using dielectric fluids, some of which contained PCBs (primarily Aroclor
- 2 1260 and, to a lesser extent, 1254), were one likely significant source of PCB contamination.

3 Because of its size and complexity, the GE/Housatonic River Site has been divided into several 4 areas for investigation and cleanup. The Rest of River is the portion of the river from the 5 confluence of the East and West Branches of the Housatonic River (the confluence) to the 6 Massachusetts border with Connecticut, a distance of approximately 54 miles (87 km), and 7 beyond into Connecticut to Long Island Sound. In addition to the river itself, the Rest of River 8 includes the associated riverbank and floodplain. The lateral extent of the area under 9 investigation includes the floodplain extending to the 1-ppm total PCB (tPCB) isopleth, which is 10 approximately equivalent to the 10-year floodplain. The Rest of River portion of the Housatonic 11 River flows through one of the most biologically diverse regions of Massachusetts and 12 Connecticut. Dams play an integral role in the downstream migration of PCBs and other COPCs 13 from the GE facility.

The ERA focuses on the portion of the river from the confluence, 2 miles (3 km) below the GE facility, to Woods Pond Dam, a distance of approximately 11 river miles (17.7 km). This area is referred to as the Primary Study Area (PSA) (Figure ES-2), and is where much of the PCB contamination was found in previous studies. The ERA also includes an evaluation of the river and floodplain downstream of the PSA to the Derby-Shelton Dam in Connecticut, approximately 13 miles upstream from Long Island Sound.

20 The first 10.5 miles (16.9 km) from the confluence to the headwaters of Woods Pond is referred to as Reach 5. Next to the initial 0.5-mile (0.8-km) reach bordering the GE facility, Reach 5 has 21 22 the highest concentrations and highest frequency of detections of PCBs in sediment. Reach 5 is 23 subdivided further into four segments: Reach 5A, from the confluence to just above the Pittsfield 24 Wastewater Treatment Plant (WWTP); Reach 5B, from the WWTP to Roaring Brook; Reach 5C, 25 from Roaring Brook to the headwaters of Woods Pond; and Reach 5D, the backwaters above Woods Pond. Reach 6 begins 10.5 miles (16.9 km) downstream of the confluence at Woods 26 27 Pond. The pond is approximately 0.2 mile (0.3 km) in length and has an area of 60 acres (24 ha). 28 This reach contains the first impoundment downstream from the GE facility and is a depositional 29 environment.



| O:\gepitt\aprs\era_figures2.apr | layout - fig1.1-2 | o:\gepitt\epsfiles\plots\in\fig_es2.eps | 11:29 AM, 7/8/2003 |

In the PSA, the river channel ranges from 40 to 125 ft (12 to 38 m) in width, is bordered by
extensive floodplain (up to 3,000 ft [900 m] wide), and has a meandering pattern with numerous
oxbows and backwaters. Woods Pond is a shallow 54-acre (22-ha) impoundment that was
formed by the construction of a dam in 1864.

Reach 7, the first reach south (downstream) of the PSA, begins below Woods Pond Dam and
flows for 18.6 miles (29.9 km), ending at the headwaters of Rising Pond, which is Reach 8.
Reach 9 begins downstream of Rising Pond and extends for approximately 24.6 miles (39.6 km)
to the Massachusetts/Connecticut state line. This reach is wide with a flat floodplain and many
oxbows, and agriculture is a predominant land use.

10 Reach 10 begins at the Massachusetts/Connecticut border and extends 7.4 miles (12 km) to the 11 dam at Great Falls Village. Reach 11, which begins on the downstream side of the dam at Great 12 Falls and ends 11.5 miles downstream at Cornwall Bridge, is mostly shallow and fast flowing, 13 and much of the reach is designated as a Trout Management Area. Reach 12 extends from 14 Cornwall Bridge to the dams at Bulls Bridge, a length of 13.1 miles (21.1 km). Reach 13 starts 15 on the downstream side of the dams at Bulls Bridge and runs 10.9 miles (17.5 km) to the now-16 submerged Bleachery Dam at New Milford, CT. Downstream of this point the river is largely 17 confined to a series of large lakes formed by power dams.

Reach 14, from the Bleachery Dam to Shepaug Dam, is known as Lake Lillinonah. Water movement is slow through this reach and the river is deep. Reach 15 encompasses Lake Zoar, from Shepaug Dam to Stevenson Dam. Some homes and boat launches are found on Lake Zoar. Reach 16 is Lake Housatonic, which is formed by the Derby-Shelton Dam. The remaining 13 miles of the river, from Derby-Shelton Dam to Long Island Sound, is tidally influenced and has other industrial sources of PCBs.

The land uses of the floodplain properties in Massachusetts include residential, commercial/industrial, agricultural, recreational (such as canoeing, fishing, and hunting), wildlife management, and parks and a golf course. The Housatonic River floodplain is an attractive area for recreation, including fishing and waterfowl hunting.

The State of Connecticut posted a fish consumption advisory for most of the Connecticut section of the river in 1977 as a result of the PCB contamination in the river sediment and fish tissue. In 1982, the Massachusetts Department of Public Health (MADPH) issued a consumption advisory for fish, frogs, and turtles for the Housatonic River. In addition, in 1999, MADPH issued a waterfowl consumption advisory from Pittsfield to Great Barrington due to PCB concentrations in wood ducks and mallards collected from the river by the U.S. Environmental Protection Agency (EPA).

8 ES.3 REGULATORY BACKGROUND

9 The GE/Housatonic River site has been subject to regulatory investigations dating back to the 10 early 1980s. In 1991, EPA issued a RCRA Corrective Action Permit to the GE Pittsfield facility. 11 Following appeals by GE and others and subsequent modification, the permit became effective in 12 1994. The permit included the 254-acre facility, some filled former oxbows, Silver Lake, the 13 Housatonic River and its floodplains and adjacent wetlands, and all sediment contaminated by 14 PCBs migrating from the GE facility.

EPA proposed the site to the Superfund National Priorities List (NPL) in September 1997. Several federal and state government agencies and GE entered into negotiations late in 1997 with the goal of reaching a comprehensive settlement, which included remediation, redevelopment, and restoration components.

In September 1998, representatives of the federal and state government agencies, GE, the City of Pittsfield, and the Pittsfield Economic Development Authority reached an agreement in principle. This agreement was translated into a Consent Decree that was entered by the court on 27 October 2000. The agreement provides for, in general, the cleanup of the GE plant facility and surrounding areas that have become contaminated as a result of facility operations, including the Housatonic River.

The GE/Housatonic River site is made up of several separate response actions (as described in
 the Consent Decree), including three actions in the river:

- Upper ¹/₂-Mile Reach Housatonic River Removal Action (¹/₂-Mile Reach)
- 1 ½-Mile Housatonic River Removal Action (1 ½-Mile Reach)
- Rest of River

3

4

5

6 7 The primary COPCs are PCBs; other COPCs include volatile organics, dioxins/furans, 8 polycyclic aromatic hydrocarbons (PAHs), semivolatiles, and metals. Certain PCB congeners 9 (known as coplanar or dioxin-like congeners) exhibit a mechanism of toxicity similar to that of 10 the most toxic dioxin congener (2,3,7,8-TCDD). The combined toxicity of these coplanar 11 congeners can be expressed and evaluated as the equivalent toxicity of 2,3,7,8-TCDD using the 12 concept of toxic equivalence (TEQ).

EPA completed an investigation of the Rest of River below the 1½-Mile Reach into Connecticut, which focused on collecting information for and preparing the human health and ecological risk assessments, and modeling PCB fate and transport in the river. The ecological risk assessment, together with the human health risk assessment and the model of PCB fate, transport, and bioaccumulation, will inform EPA's decision on what additional remedial actions, if any, may be required in the future.

19 ES.4 OVERVIEW OF TECHNICAL APPROACH

20 This ERA follows the eight-step technical approach and guidelines detailed in EPA's Ecological 21 Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological 22 Risk Assessments. The first two steps of the ERA process (Screening-Level Problem 23 Formulation and Screening-Level Exposure Estimate and Risk Calculation) were first addressed 24 in the Upper Reach-Housatonic River Ecological Risk Assessment and subsequently refined in 25 Appendix B of this document. Steps 3, 4, and 5 (Baseline Problem Formulation, Study Design 26 and DOO Process, and Verification of Field Data Analysis) are iterative components of the 27 eight-step ERA process. These three steps were initially presented in the Supplemental 28 Investigation Work Plan for the Lower Housatonic River and were modified as necessary during 29 the data collection phase of the project. Steps 6 and 7 (Site Investigation and Data Analysis and 30 Risk Characterization) are presented in detail in this Ecological Risk Assessment report. Step 8

(Risk Management) will be addressed after the ERA has undergone Peer Review. A roadmap for
 the ERA is provided in Figure ES-3. A brief overview of each of the eight steps, particularly as

3 they relate to this document, is presented below.

Problem formulation is an important component of the ERA process that establishes the goals,
objectives, and scope for the ERA. The problem formulation portion of the ERA is discussed in
Section 2 and was outlined in the *Supplemental Investigation Work Plan for the Lower Housatonic River*.

8 An extensive physical and ecological characterization of the Housatonic River is presented in 9 Section 2.2 and Appendix A (Ecological Characterization) of this document. These sections 10 detail the physical setting, habitats, and biotic communities of the river in both the aquatic and 11 terrestrial environments.

12 Investigations of the nature and extent of contaminants in the Housatonic River watershed have 13 previously been conducted by GE, EPA, and others. PCBs have been identified as the main 14 COPC, and other contaminants such as dioxins and furans and PAHs have also been identified at 15 the GE facility. In Section 2.3, the sources, amounts, and patterns of contaminant releases and 16 receiving bodies are presented.

17 The purpose of the Pre-ERA was to identify contaminants that warranted more detailed analyses 18 in the ERA, and those that could be removed from further consideration because they pose 19 minimal risk. A summary of the Pre-ERA is provided in Section 2.4. The complete Pre-ERA is 20 included as Appendix B to this document.

An overview of the environmental behavior of PCBs and other COPCs is presented in Section 2.5. This section includes discussions of the transport of the contaminants from their point(s) of 2.5. release, partitioning behavior in different media, and biotic and abiotic degradation in these 2.4 media.

The effects and mechanisms of toxicity to biota of the contaminants identified as COPCs within the Housatonic River and floodplain are discussed, with an emphasis on PCBs in Section 2.6, and in further detail in the effects assessment portion of each assessment endpoint section and corresponding appendix.



Assessment and measurement endpoints are defined and described in Section 2.8. An assessment endpoint is defined as the "explicit expression of the environmental value that is to be protected." A measurement endpoint is defined as "a measurable ecological characteristic that is related to the valued characteristic chosen as the assessment endpoint," and is a measure of biological effects (e.g., mortality, reproduction, growth).

6	Assessment Endpoint Selected for the Rest of River ERA
7	 Community structure, survival, growth, and reproduction of benthic invertebrates.
8 9	 Community condition, survival, reproduction, development, and maturation of amphibians.
0	 Survival, growth, and reproduction of fish.
1	 Survival, growth, and reproduction of insectivorous birds.
2	 Survival, growth, and reproduction of piscivorous birds.
3	 Survival, growth, and reproduction of piscivorous mammals.
4	 Survival, growth, and reproduction of omnivorous and carnivorous mammals.
5 6	 Survival, growth, and reproduction of Special Status Species (Endangered, Threatened).

17

18 The conceptual model outlined in Section 2.7 describes the relationship between the COPCs and 19 the assessment endpoints. Section 2.9 describes the analytical approach used to estimate risks 20 and the weight-of-evidence approach used to develop the conclusions.

Sections 3 through 11 (and their corresponding appendices) provide an overview of the exposure assessment, the effects assessment, and the risk characterization for each representative species or representative group of species. The exposure assessment sections include a description of the data collection activities and the studies conducted to determine concentrations of COPCs in water, soil, sediment, and biota samples. The list of COPCs was further narrowed with additional screening for the specific endpoint, resulting in the list of contaminants of concern (COCs) retained for that endpoint risk assessment.

The effects assessment sections begin with an overview of the toxicity of PCBs and the other COCs. For each major representative species group and COC, the effects literature was reviewed. The goal of this review was to identify studies that could be used to develop effects metrics for use in risk characterization. The effects metrics developed ranged from

concentration- or dose-response curves to benchmarks depending on the quality and relevance of
 the data available.

The risk characterization sections first provide an overview of site-specific studies, analyses of the results, and conclusions, and then consider the three major lines of evidence (where available) using a weight-of evidence (WOE) approach. There is also a discussion of the uncertainties associated with the assessment for that endpoint, an evaluation of potential risks to receptors other than those chosen as the representative species, and a discussion of potential risks downstream of the PSA for receptors of concern.

9 Section 12 summarizes the conclusions of the ERA, and presents a discussion of these
10 conclusions in the context of the uncertainties and other factors that could not be expressly
11 quantified in the evaluation of the assessment endpoints.

12 ES.5 OVERVIEW OF THE ASSESSMENT ENDPOINT CONCLUSIONS

13 ES.5.1 Risks in the Primary Study Area

14 The assessments in the ERA were conducted using various lines of evidence including, in many 15 cases, different measurement endpoints and effects metrics. These lines of evidence-defined as 16 information derived from different sources that can be used to describe and interpret risk-were 17 integrated into a graphical representation of risk using the weight-of-evidence approach. The 18 WOE provides an objective process by which measurement endpoints are related to an 19 assessment endpoint to evaluate whether significant risk is posed to the environment. A formal 20 WOE can range from a simple qualitative assessment to a highly quantitative evaluation; 21 however, no matter what form the weight-of-evidence takes, it should provide documentation of 22 the thought process used when assessing potential ecological risk.

In the first step of the WOE approach, weights are assigned to measurement endpoints based on 10 attributes related to: (1) strength of association between assessment and measurement endpoints; (2) data and study quality; and (3) study design and execution. The second step of the approach is to evaluate the magnitude of response in the measurement endpoint, considering whether the measurement endpoint indicates the presence or absence of harm, and if the magnitude of response is low, intermediate, or high. The WOE process concludes by plotting the output of the two preceding steps in a matrix for all measurement endpoints associated with a given assessment endpoint. The matrix allows easy visual examination of agreements or divergences among measurement endpoints, facilitating interpretation with respect to the assessment endpoint.

6 The results of the WOE process and the final WOE matrix are summarized below for each of the eight assessment endpoints considered in this ERA. Following that discussion, to facilitate 7 8 comparison of risks among aquatic life and wildlife receptors and to give an overview of the 9 findings of the risk assessment, assessment results are converted to probabilistic hazard quotients 10 (HQs). A HQ is the expected environmental concentration or dose of a contaminant divided by 11 its estimated low or no toxic effect concentration or dose. Higher quotients indicate greater risk. 12 The methods used to calculate the probabilistic HQs and the results of these analyses for each 13 endpoint are summarized in this section.

14 ES.5.1.1 Aquatic Assessment Endpoints

Benthic Invertebrates—The weight-of-evidence results for the benthic invertebrate assessment endpoint are shown in Table ES-1. In this table, the measurement endpoints for the three lines of evidence: water, sediment, and tissue chemistry (C), toxicity tests (T), benthic community measures (B), are listed, as are the weight of the measurement endpoint evidence of harm, and magnitude of response. This table indicates that the majority of endpoints suggest some risk for benthic communities in both coarse- and fine-grained sediment. The conclusion is that there is a moderate to high risk to much of the benthic community indicated by the WOE evaluation.

Amphibians—The results of the weight-of-evidence assessment for amphibians are presented in Table ES-2. In the amphibian weight-of-evidence matrix, the measurement endpoints for the three lines of evidence: the tissue chemistry (C); wood frog toxicity tests (W) and leopard frog toxicity tests (L); and field surveys (B) are listed. As shown on the table, many of the endpoints indicated some degree of risk. The potential for two amphibian studies conducted for GE to determine risk to amphibians was judged to be undetermined due to limitations in the study

4

Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Benthic Community

Table ES-1

	Weighting	Coarse-Gra	nined Sediment	Fine-Grai	ned Sediment
Measurement Endpoints	Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
C. Chemical Measures					
C-1. Concentration of PCB in overlying water in relation to levels reported to be harmful to benthic invertebrates	Low/Moderate	Yes	Intermediate	Yes	Intermediate
C-2. Concentration of PCB in the sediment in relation to levels reported to be harmful to benthic invertebrates	Low/Moderate	Yes	High	Yes	High
C-3. Concentration of PCB in invertebrate tissues in relation to levels reported to be harmful to benthic invertebrates	Moderate	Yes	Intermediate	Yes	Intermediate
T. Toxicological Measures	· · · ·				
T-1. Sediment toxicity to multiple invertebrate species, as measured in laboratory toxicity tests	Moderate/ High	Yes	High	Yes	High
T-2. Sediment toxicity to multiple invertebrate species, as measured in in situ toxicity tests	Moderate/ High	Yes	Intermediate	Yes	High
T-3. Indications of PCB as toxicity driver in toxicity identification evaluations	Moderate	Undetermined		Yes	Intermediate
B. Benthic Community Measures					
B-1. Abundance, richness, and biomass of invertebrates, relative to reference stations of comparable substrate and habitat (ANOVA)	Moderate	Yes	Intermediate	No	_
B-2. Benthic community structure, as assessed using a multivariate assessment of key benthic metrics (rank analysis and MDS)	Moderate	Yes	Intermediate	No	_
B-3. Water quality assessment using modified Hilsenhoff Biotic Index (MHBI) indicator of organic pollution	Moderate	No		No	

Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of Amphibian Populations in the Housatonic River PSA

Table ES-2

	Weighting Value		
Measurement Endpoints	(Hign, Moderate, Low)	(Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
C. Chemical Measures			
C. Concentration of PCB in frog tissues in relation to concentrations reported to be harmful to amphibians	Moderate	Yes	Low
W. Wood Frog Toxicological Measures			
W-1. Sediment toxicity to hatchling/late embryo life stages	Mod/High	No	-
W-2. Sediment toxicity to larval life stages	Mod/High	Yes	Intermediate
W-3. Sediment toxicity to late larval/metamorph life stage	Mod/High	Yes	High
W-4. GE Study (juvenile wood frogs)	Low	Undetermined	-
L. Leopard Frog Toxicological Measures			
L-1. Sediment toxicity to hatchling/late embryo life stages	Mod/High	Yes	Low
L-2. Sediment toxicity to larval life stages	Mod/High	Yes	High
L-3. Sediment toxicity to late larval/metamorph life stage	Mod/High	Yes	High
L-4. Sediment toxicity to adult leopard frogs (reproductive health)	Mod/High	Yes	High
B. Biology			
B-1. Vernal pool community study	Mod/High	Yes	Low
B-2. GE leopard frog egg mass survey	Low/Mod	Undetermined	-
B-3. Anecdotal observations during collections for reproductive study	Moderate	Yes	Low

1 designs. The only endpoint that did not indicate potential risk was the earliest life stage wood 2 frog toxicity endpoint, for which there is mechanistic explanation for the lack of response. The 3 plots also indicate that four endpoints exhibited a high degree of risk combined with a moderate 4 to high confidence rating.

5 In addition, a population model was constructed for wood frogs to determine whether effects 6 from PCBs on individual wood frogs influence the populations within the PSA. A 10-year 7 simulation, both with and without the effects of PCBs, was conducted. The model demonstrated 8 that effects observed in the toxicity studies would result in population-level impacts.

9 The conclusion is that there is a significant risk to amphibians as indicated by the preponderance 10 of the evidence, the relative weights of the measurement endpoints, and the population modeling. 11 The "no harm" value of measurement endpoint W-1 does not diminish the overall conclusion, 12 because the study demonstrated that the embryo/early larval life stages are fairly insensitive to 13 the effects of maternally transferred PCBs relative to later juvenile life stages exposed to 14 contaminated media.

15 Fish—The weight-of-evidence results for fish in the PSA are shown in Table ES-3. In the fish 16 WOE matrix, the measurement endpoints for the three lines of evidence: site-specific toxicity 17 tests (A); fish tissue chemistry (B); and field surveys (C) are listed. This table illustrates that 18 although a high probability of adverse impacts to fish from tPCBs and/or TEQ is predicted 19 throughout the PSA, the impacts predicted are for sensitive sublethal endpoints (reproduction 20 and development); mortality of adults is unlikely. Therefore, the magnitude of impact is not 21 predicted to be catastrophic in any reach; adverse effects, although high in probability, are 22 generally expected to be subtle. The field studies conducted in the PSA (fish community and 23 reproduction studies) support lack of catastrophic effects, but cannot be used to assess lesser 24 impacts.

25 ES.5.1.2 Wildlife Assessment Endpoints

26 **Insectivorous Birds**—The WOE results for exposure of insectivorous birds to tPCBs are 27 presented in Table ES-4, and for exposure to TEQ in Table ES-5. Two lines of evidence are

Table ES-3

Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Fish Community

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
A. Site-Specific Toxicity			
A1. Reproductive success relative to reference	Mod/High	Yes	Low
A2. Reproductive success dose-response	High	Yes	Intermediate
B. Fish Body Burden			
B1. Observed fish tissue/ Literature toxic levels	Mod	Yes	Low
B2. Observed fish tissue/ Phase I toxic levels	Mod/High	Yes	Low
B3. Observed fish tissue/ Phase II toxic levels	Mod/High	Yes	Low
C: Fish Community and Reproduction Studies			
C1: EPA Study and GE Community Study	Low/Mod	Undetermined	-
C2: GE Reproduction Study	Low/Mod	Undetermined	-

- 2
- 3
- 4 5

Table ES-4

Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Study	High (Tree swallow) Moderate/High (American robin)	No (Tree swallow) No (American robin)	Low (Tree swallow) Low (American robin)
Modeled Exposure and Effects	Moderate	Yes	High

6

- 7
- 8
- 9

10

Table ES-5

Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Study	High (Tree swallow) Moderate/High (American robin)	No (Tree Swallow) No (American robin)	Low (Tree Swallow) Low (American robin)
Modeled Exposure and Effects	Moderate	Yes	Intermediate

13

1 presented in the table, the field studies, and modeled exposures and effects. The results from the 2 modeled exposure and effects line of evidence suggest that tPCBs and TEQ pose intermediate to 3 high risks to tree swallows living in the PSA. However, the field study line of evidence suggests 4 that, if effects are occurring, they are minor. The uncertainty concerning the field-based 5 threshold range for tPCBs likely means that risks of this COC are overestimated for the PSA. 6 Even the upper end of the tPCB range is associated with equivocal evidence for adverse effects 7 to tree swallows. For TEQ, the threshold range is quite broad. The available evidence from field 8 studies indicates that tree swallows are tolerant to exposure to persistent organochlorines such as 9 tPCBs and TEQ. If the tree swallow threshold is near the upper end of the threshold range, then 10 the current modeled exposure and effects line of evidence is overestimating risks of TEQ to tree 11 swallows. Thus, the WOE assessment supports a finding of low risk for tree swallows exposed 12 to tPCBs and TEQ in the PSA. This conclusion, however, is uncertain because of the conflicting 13 results in the WOE assessment.

14 The results from the modeled exposure and effects lines of evidence suggest that tPCBs and TEQ 15 pose an intermediate to high risk to American robins inhabiting the PSA of the Housatonic River. 16 The American robin field study, however, suggests that reproductive success is not being 17 impaired by the tPCBs and TEQ in the PSA. The uncertainty in the modeled exposure and 18 effects line of evidence, outlined below, likely means the approach overestimates the risks of 19 tPCBs and TEQ to American robins in the PSA. The WOE assessment, therefore, supports a 20 conclusion of low risk to American robins exposed to tPCBs and TEQ in the PSA. This 21 conclusion, however, is uncertain because of the conflicting results in the weight-of-evidence 22 assessment.

Piscivorous Birds—The WOE analysis indicated that exposure of piscivorous birds, such as the belted kingfisher and osprey (Tables ES-6 and ES-7), to tPCBs and TEQ in the PSA, could lead to adverse reproductive effects in some species. The two lines of evidence used to support this conclusion were the field study of kingfisher productivity and the comparison of modeled exposure with effects to piscivorous birds.

For the assessment of risks to kingfishers, both lines of evidence were employed. The modeled exposure and effects line of evidence indicated that kingfishers in the PSA are likely to receive a

3 4

Table ES-6

Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled Exposure and Effects	М	Yes (Kingfisher) Yes (Osprey)	High (Kingfisher) High (Osprey)
Belted Kingfisher Field Study (Henning 2002)	M/H	No (Kingfisher)	Low (Kingfisher)

5

6

7 8

9

Table ES-7

Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled Exposure and Effects	М	Yes (Kingfisher) Yes (Osprey)	Intermediate (Kingfisher) Intermediate (Osprey)
Belted Kingfisher Field Study (Henning 2002)	M/H	No (Kingfisher)	Low (Kingfisher)

10

1 tPCB dose greater than what the most tolerant species known from the literature can be exposed 2 to without effects. For TEQ, the risk picture is less clear because the threshold range for this 3 COC is very wide and the exposure estimates for kingfishers fell within this range. Thus, without 4 effects data specific to kingfishers, it is difficult to make definitive conclusions about the risks of 5 TEQ to this species. The field study of kingfisher productivity, however, indicated that these 6 birds are able to reproduce in the PSA. This line of evidence was given a higher weighting than 7 the exposure and effects modeling, despite concerns about the field study. Therefore, kingfishers 8 are considered to be at low risk in the PSA as a result of exposure to tPCBs and TEQ. The 9 conclusion of low risk to kingfishers is uncertain because the two lines of evidence did not give 10 concordant results.

11 For ospreys, only the modeled exposure and effects line of evidence was available to assess risk 12 to these birds. As with kingfishers, this line of evidence indicated that ospreys in the PSA are likely to receive a tPCB dose that is greater than what the most tolerant species known from the 13 14 literature can be exposed to without effects. The risks due to exposure to TEQ are unclear, as the 15 estimates for exposure also fell within toxicity threshold range. Ospreys, however, lack a site-16 specific study that investigated the effects of COCs in the PSA. The PSA contains suitable 17 habitat for ospreys, with abundant prey, raising the possibility that they are not resident in the 18 area because of contaminants. Ospreys are therefore considered to be at risk in the PSA as a 19 result of exposure to tPCBs and TEQ.

20 Piscivorous Mammals—The results of the WOE assessment for piscivorous mammals are 21 presented for tPCB and TEQ, respectively, in Tables ES-8 and ES-9. All three lines of evidence 22 -field studies, feeding study, and modeled exposure and effects-indicated that the elevated 23 concentrations of tPCBs and TEQ in the PSA of the Housatonic River are causing adverse 24 effects of high magnitude to mink and river otter. The field surveys indicated that mink and river 25 otter are rarely present in the PSA, except during winter, and likely have not established home 26 territories close to the main channel despite suitable mink and otter habitat. The MSU feeding 27 study indicated that feeding adult female mink with a diet containing as little as 3.51% fish from 28 the PSA caused a statistically significant reduction (46% compared to controls) in kit survival to 29 6 weeks of age. Because mink in the wild typically consume between 20% or more fish in their 30 diet, the associated risk is correspondingly higher. In addition, other components of the mink

2

-3 4

5

Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Table ES-8

Measurement Endpoints		Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Survey	EPA	Moderate/High	Yes	High
Field Sulveys	GE	Moderate	No	Low
Feeding Study		High	Yes	High
Modeled Exposure and Effects		Moderate/High	Yes	High

6

- 7
- 8

8 9

Table ES-9

Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints		Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Surveys	EPA	Moderate/High	Yes	High
r leid Surveys	GE	Moderate	No	Low
Feeding Study		High	Yes	High
Modeled Exposure and Effects		Moderate/High	Yes	High

12

13

diet in the PSA (e.g., crayfish) have high concentrations of tPCBs and TEQ. Further, the jaw lesion study indicated that erosion of the jaw occurs at even lower doses and exhibits a doseresponse relationship. Such effects could eventually lead to starvation. The occurrence of jaw lesions coincides with the induction of Ah-receptor-regulated enzymes (ECOD and EROD) also in a dose-response manner.

6 The high risks evident from the feeding study are further supported by the modeled exposure and 7 effects line of evidence. The estimated potential for exposure is so high that even individual 8 mink and otter that only forage in the PSA for short periods of time (less than or equal to 10% of 9 foraging time) are at an intermediate or higher risk from tPCBs and TEQ.

10 Omnivorous and Carnivorous Mammals-The weight-of-evidence results for omnivorous and 11 carnivorous mammals are shown in Table ES-10 for tPCB and Table ES-11 for TEQ. Data from 12 three lines of evidence were available, including field surveys, a population demography field 13 study of short-tailed shrew, and exposure and effects modeling. The weight-of-evidence analysis 14 indicates an intermediate risk for short-tailed shrews exposed to tPCBs and TEQ in the PSA. 15 This conclusion, however, is uncertain because of the lack of definitive findings as to whether 16 effects are occurring in the field surveys and population demography field study, and the lack of species-specific measures of effects in the literature. The weight-of-evidence also suggests, 17 18 based on one line of evidence for red fox, an intermediate risk to fox exposed to tPCBs and TEQ 19 in the PSA. This finding is also uncertain because a foraging rate of 50% in Reach 5 was used, 20 and species-specific measures of effects were not available.

The field surveys, and conclusions made in a study of short-tailed shrew populations at the site conducted for GE, are not in agreement with the results from the modeling of exposure and effects line of evidence. However, the results of the supplemental analyses of the data from the GE study on survival of short-tailed shrews are in agreement with the modeling results, suggesting that there are intermediate effects from exposure to COCs in the contaminated areas of the PSA.

3 4

Table ES-10

Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Surveys	Moderate/High	Undetermined	Low
Population Demography Field Study	Moderate/High	Undetermined (Shrew)	Intermediate
Modeled Exposure and Effects	Moderate/High	Yes (Shrew) Undetermined (Red Fox)	High Intermediate

5

6

7 8

9

Table ES-11

Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Surveys	Moderate/High	Undetermined	Low
Population Demography Field Study	Moderate/High	Undetermined (Shrew)	Intermediate
Modeled Exposure and Effects	Moderate/High	No (Shrew) Undetermined (Red Fox)	Low Intermediate

10

11

Threatened and Endangered Species—The results of the weight-of-evidence evaluation for threatened and endangered species using a single line of evidence, modeled exposures and effects, are shown in Table ES-12 and Table ES-13 for tPCBs and TEQ, respectively. The results of the risk characterization showed that the highest risk to T&E species is to bald eagles and American bitterns from exposure to tPCBs. The risk for adult bald eagles exposed to TEQ was low; however, risk to bald eagle eggs exposed to TEQ was high. These two risk estimates were combined to yield intermediate risk to bald eagles.

8 The weight-of-evidence analysis indicates that T&E species such as bald eagle and American 9 bittern are at risk in the PSA as a result of exposure to tPCBs. Risks to bald eagles and 10 American bittern exposed to tPCBs are high. There are intermediate risks to bald eagles exposed 11 to TEQ, and risks to American bittern exposed to TEQ are undetermined. Risks to small-footed 12 myotis exposed to tPCBs and TEQ are undetermined.

13 ES.5.1.3 Hazard Quotients

14 To facilitate comparison of risks among aquatic life and wildlife receptors and to give an 15 overview of the findings of the risk assessment, assessment results were converted to 16 probabilistic hazard quotients (HQs). A HQ is the expected environmental concentration or dose 17 of a contaminant divided by its estimated low or no toxic effect concentration or dose, similar to 18 a hazard index that is used to describe noncancer risks to people. Higher quotients indicate 19 greater risk. Figures ES-4 through ES-7 summarize the ranges of hazard quotients for exposure to tPCBs and TEQ determined for each the assessment endpoints in the PSA. The methods used 20 21 to calculate the probabilistic HQs and the results of these analyses for each endpoint are 22 summarized below.

Table ES-12

1 2 3 4

Evidence of Harm and Magnitude of Effects of T&E Species Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled exposure and effects, Bald Eagle	Moderate/High	Yes	High
Modeled exposure and effects, American Bittern	Moderate/High	Yes	High
Modeled exposure and effects, Small-Footed Myotis	Moderate/High	Undetermined	High

5

6

7

8

9 10

Table ES-13

Evidence of Harm and Magnitude of Effects for T&E Species Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled exposure and effects, Bald Eagle	Moderate/High	Yes	Intermediate
Modeled exposure and effects, American Bittern	Moderate/High	Undetermined	High
Modeled exposure and effects, Small-Footed Myotis	Moderate/High	Undetermined	High



WB-R = Whole body,

reconstituted fish tissue. WB = Whole body fish tissue.

Figure ES-4 Hazard Quotients for Aquatic Biota Exposed to tPBCs in the Housatonic River PSA



Figure ES-5 Hazard Quotients for Fish Exposed to 2,3,7,8-TCDD TEQ in the Housatonic River PSA

MK01\O:\20123001.096\ERA_PB\ERA_PB_ES.DOC



Figure ES-6 Hazard Quotients for Wildlife Exposed to tPCBs in the Housatonic River PSA

MK01\O:\20123001.096\ERA_PB\ERA_PB_ES.DOC



Figure ES-7 Hazard Quotients for Wildlife Exposed to 2,3,7,8-TCDD TEQ in the Housatonic River PSA

1

MK01\O:\20123001.096\ERA_PB\ERA_PB_ES.DOC

1 The thresholds used in HQ calculations represent levels at, or close to, levels demonstrated to 2 cause adverse responses to organisms in site-specific studies. Thus, HQ exceedances of 1 are 3 cause for concern.

Benthic Invertebrates—Hazard quotients were calculated by dividing concentrations of COCs in site sediment by 3 mg/kg, the effects benchmark for benthic invertebrates exposed to tPCBs in sediment. These results indicate that significant risk was observed in all reaches of the PSA. Predicted risks were greatest in the upstream (Reach 5A) and Woods Pond (Reach 6) sediment. Because of small-scale variation in sediment tPCB concentrations, although the majority of benthic invertebrates in the PSA are at risk (i.e., HQ > 1), some individuals in less-contaminated areas are not.

Amphibians—For amphibians, HQs were calculated by dividing summary statistics for vernal pool sediment concentrations by the effects benchmark for amphibians exposed to tPCBs in sediment (3 mg/kg tPCBs). These results indicate significant risk in all reaches of the PSA, with HQs above 1. Predicted risks were greatest in the upstream (Reach 5A) vernal pool habitats. These results indicate that the majority of amphibians are at risk (i.e., HQ > 1), with higher levels of risk (i.e., HQ > 5) in a large percentage of vernal pools (about 50% of pools in Reaches 5A and 5B).

Fish—For fish, HQs were calculated separately for the five representative warmwater species by
dividing summary statistics for exposure by the tissue effects benchmark protective of all species
of PSA fish (49 mg/kg tPCB). These results indicate that risk occurs in all reaches of the PSA.

Predicted risks were greatest for predator fish at the top of the food web (e.g., largemouth bass) and when fish reach their maximum adult tPCB concentrations. The ERA indicates that these HQs are indicative of sublethal (e.g., reproductive and developmental) responses to offspring; the pathway for the manifestation of effects is through the maternal transfer of tPCBs to eggs. Acute mortality to adults is not expected for most fish.

26 In addition to tPCBs, fish HQs were derived for TEQ using the average of the effects thresholds

27 relevant to warmwater fish species obtained from site-specific toxicity studies (42 ng/kg TEQ).

28 The magnitudes and probabilities of risk for TEQ are very similar to tPCB risks.

Wildlife Endpoints—For wildlife, the distributions from a Monte Carlo analyses for total daily intake of COCs by representative species were each divided by the corresponding effects metrics used to estimate risks. In the case of a dose-response curve effects metric (e.g., mink exposed to tPCBs), the effects metric was specified as a uniform distribution of dose ranging from 10% to 20% effect. A similar approach was used for NOAEL-LOAEL ranges (e.g., bald eagles exposed to tPCBs), field-based effects metrics (e.g., tree swallows exposed to tPCBs), and threshold ranges (e.g., kingfishers exposed to TEQ).

8 In addition to plots developed for mink and otter exposed to tPCBs and TEQ using the results of 9 literature-based dose-response curve, plots were also developed using the results of the mink 10 feeding study conducted in support of this ERA. In this case, the denominator was the NOAEL 11 to LOAEL range, rather than the 10% and 20% effects doses from the literature-based dose-12 response curve.

Unlike traditional HQs, the probabilistic HQs for wildlife do not include safety factors, i.e., are not conservative. No safety factors were used to estimate the effects metrics (except in the case of the bald eagle), and uncertainties regarding exposure model inputs were explicitly propagated through the probability bounds and Monte Carlo analyses. Thus, HQ exceedances of 1 are cause for concern.

Wildlife risks from tPCBs and TEQ are highest for mink and river otter, with HQs between 100 and 500 for tPCBs, and 5 and 10 for TEQ using the results of the literature-based dose-response curve. The HQs for tPCBs were somewhat lower when the results of the site-specific mink feeding study were used to derive the effects threshold. Wildlife risks from tPCBs are generally higher than risks from TEQ by one to several orders of magnitude.

The risks from tPCBs and TEQ to many of the wildlife species are uncertain to the extent that the range of HQs spans 1. The highest and lowest values depicted for the wildlife HQs are the extreme representations of uncertainty because they are tail outputs from the probability bounds analyses, a technique designed to propagate all forms of uncertainty (e.g., inability to precisely specify distribution type or parameter values for a distribution). Thus, the range of HQs shown in the boxes should be interpreted as representing a reasonable range within which the HQ estimate occurs for the receptor of interest, and the lines should be interpreted as representing the
 extremes within which the HQ could occur.

3 ES.5.2 Risks Downstream of the Primary Study Area

4 Because of the reduced levels of contaminant concentrations downstream of the PSA and 5 significant shifts in aquatic habitat types associated both with river gradient and location of 6 dams, a different approach than that applied in the PSA was followed to assess potential 7 ecological risks of tPCBs to biota in areas downstream of Woods Pond. The assessment of 8 potential ecological risks was conducted using mapping (GIS) techniques and threshold 9 concentrations that indicate potential risk for six taxonomic groups selected based on the 10 outcome of the evaluations performed in the PSA and the habitat characteristics found 11 downstream. These groups or species are benthic invertebrates, amphibians, warmwater fish, 12 trout, mink, otter, and bald eagles.

For each of these groups, a maximum acceptable threshold concentration (MATC) for tPCBs was developed based primarily on the detailed risk assessment performed for the PSA. Each MATC was then compared to medium-specific data for areas downstream of Woods Pond to Long Island Sound. Areas with MATC exceedances, indicating potential risk, were plotted on maps of the river. The methods used for each of the six representative groups and the results of the analyses are discussed in the following sections.

19 Benthic Invertebrates—For benthic invertebrates, the medium of interest was river sediment. 20 An MATC of 3 mg/kg tPCBs was used as a conservative measure of the potential for adverse 21 affects to benthic invertebrates downstream of Woods Pond. The MATC of 3 mg/kg tPCBs was 22 compared to recent surficial sediment data downstream of Woods Pond, and the results were 23 plotted to indicate samples above and below the MATC. Inverse distance weighting was used to 24 interpolate sediment concentrations between discrete sampling points, and the potential for risk 25 to benthic invertebrates was shown by shading the corresponding sections of the river channel 26 Figure ES-8).

In general, potential risks to benthic invertebrates occur in limited areas downstream of WoodsPond to Rising Pond. These areas are depositional and tend to have higher concentrations of



| O:\gepitt\aprs\era_species.apr | layout - benthos d4-7 | o:\gepitt\epsfiles\plots\in\benthos_risk_es8.eps | 11:33 AM, 7/8/2003 |

tPCBs. Below Rising Pond, sediment does not contain concentrations of tPCBs that represent a
 potential risk to benthic invertebrates.

Amphibians—A floodplain soil MATC of 3.0 mg/kg tPCB (dry weight) was derived from the amphibian risk assessment conducted for the PSA. Inverse distance weighting was used to interpolate tPCB concentrations to the limit of the 100-year floodplain (10-year floodplain contours are not available downstream of Woods Pond) using the 0 to 6 inch (0 to 15 cm) depth data from the floodplain downstream of Woods Pond.

8 Several large areas of the floodplain may pose risk to amphibians between Woods Pond and 9 Rising Pond, with only small isolated areas of potential risk downstream of Rising Pond (Figure 10 ES-9). The floodplain risk mapping for amphibians was not conducted downstream of the 11 Massachusetts/Connecticut state line because the extent of the floodplain is more limited in 12 Connecticut and concentrations in Reach 9 floodplain soil were below the MATC.

Potential risks to amphibians exposed to sediments downstream from Woods Pond appear to be
limited to small areas between Woods Pond and Great Barrington (Figure ES-10).

15 Warmwater Fish—As was done for the PSA, risk to fish was evaluated based on concentrations 16 of tPCBs in fish tissue. An MATC of 49 mg/kg tPCB in tissue (whole body, wet weight) 17 developed for the PSA based on site-specific effects to warmwater fish was applied to areas 18 downstream of Woods Pond using the available (e.g., bass, perch, sunfish) tissue data for 19 warmwater species. Each downstream reach (Reaches 7 through 16) was evaluated as a unit, and 20 the mean adult fish tissue concentration in each reach was compared with the MATC to 21 determine potential risk (Figure ES-11). No risks were indicated in any of the reaches below the 22 PSA.

Trout—Trout were evaluated separately from warmwater fish species because of significant differences in habitat requirements and differences in the sensitivity of some trout species to tPCBs documented in the literature. Trout also tend to have higher tPCB concentrations because of their higher lipid content. However, the site-specific studies did not indicate large differences between the effects threshold for rainbow trout and warmwater species, but the strain of rainbow trout used in the site-specific toxicity tests is less sensitive than other strains used widely in


| O:\gepitt\aprs\era_species.apr | layout - amphibs FLD e4-4 | o:\gepitt\epsfiles\plots\in\amphib_fld_risk_es9.eps | 11:34 AM, 7/8/2003 |



| O:\gepitt\aprs\era_species.apr | layout - amphibs SEDS e4-5 | o:\gepitt\epsfiles\plots\in\amphibs_sed_risk_es10.eps | 10:41 AM, 7/10/2003 |





O:\gepitt\aprs\era_species.apr | layout - fish warm f4-10 | o:\gepitt\epsfiles\plots\in\warm_fish_risk_es11.eps | 11:36 AM, 7/8/2003 |

toxicity testing. Furthermore, there are other trout species found downstream of the PSA (e.g., brown trout) for which sensitivity has not been assessed. Given that some trout species have been documented to have greater sensitivity of PCBs and dioxins, relative to the warmwater species considered in the development of the 49 mg/kg tPCB warmwater MATC, a factor of 4 was applied in recognition of these potential interspecies differences. Therefore, a tissue MATC of 12 mg/kg tPCBs (whole body, wet weight) was derived for trout.

7 The results of this evaluation indicate that trout are potentially at risk in Reaches 7 and 9, but not 8 in reaches with suitable habitat further downstream (Figure ES-12). This assessment has high 9 uncertainty due to the number extrapolations required and the low magnitude of exceedance of 10 the MATC value. Potential risk to trout was not evaluated in Reach 8, Reach 10, and 11 downstream of Reach 12 due to lack of suitable trout habitat.

12 Mink—An MATC for mink of 2.65 mg/kg tPCBs in fish (whole body, wet weight) represents 13 the geometric mean of the NOAEL and LOAEL developed in a site-specific study of the toxicity 14 of a diet containing Housatonic River fish to mink. Mean fish concentrations were calculated for 15 each river reach downstream of Woods Pond using available whole body fish tissue data from 16 samples of fish with an overall body length between 7 and 20 cm, corresponding to the size 17 commonly preved on by mink. Potential risk to mink due to consumption of contaminated fish 18 occurs from the Woods Pond Dam downstream to the Great Falls Dam, corresponding to 19 Reaches 7 through 10 (Figure ES-13).

River Otter—The mink MATC of 2.65 mg/kg tPCB in fish (whole body, wet weight) was also used for river otter. Mean fish concentrations were calculated for such areas in river reaches downstream of Woods Pond using available whole body fish tissue data from fish with an overall body length between 5 and 50 cm, corresponding to the size commonly preyed on by otter.

Potential risk to otter due to consumption of contaminated fish occurs from the Woods Pond
Dam downstream to the Bulls Bridge Dam, corresponding to Reaches 7 through 12 (Figure
ES-14).

Bald Eagle—An MATC of 30.4 mg/kg tPCBs (whole body fish tissue, wet weight) was
developed for wintering bald eagles, assuming that the eagle diet was composed of 78% fish, and







| O:\gepitt\aprs\era_species.apr | layout - mink i4-15 | o:\gepitt\epsfiles\plots\in\mink_risk_es13.eps | 11:38 AM, 7/8/2003 |



Riverside NOTES: Risk to otter is based on a maximum acceptable threshold concentration (MATC) of 2.65 mg/kg total CB (tPCB) concentration (wet weight) in (MATC) of 2.65 mg/kg total CB (tPCB) concentration (wet weight) whole body fish tissue 5-50 cm in length. * Only fish collected in 1998 to the present (2000) were included. * Young-of-year bass composites were scaled by a factor of 3.5. * Young-of-year perch composites were scaled by a factor of 2.5. * Trout filet samples were scaled by 1.47. * Brown bullhead filet samples were scaled by 1.5. * Warmuster filet samples were scaled by 2.2 Shelton * Warmwater filet samples were scaled by 2.3. LEGEND: Ecological Risk Assessment GE/Housatonic River Site Town Rest of River Reach Break OTTER RISK FIGURE ES-14 6 8 Miles Roads < 2.65 mg/kg ASSESSMENT OF RISK Housatonic River Basin Hydrology >= 2.65 mg/kg TO OTTER EXPOSED TO tPCBs DOWNSTREAM State Boundary **OF WOODS POND**

| O:\gepitt\aprs\era_species.apr | layout - otter i4-16 | o:\gepitt\epsfiles\plots\in\otter_risk_es14.eps | 11:39 AM, 7/8/2003 |

that the remainder of the diet included non-aquatic species that, for the purpose of this analysis,
 were not contaminated. Potential risk to nesting bald eagles was evaluated using methods
 similar to those discussed above for mink (Figure ES-15).

A more in-depth analysis was performed for Reaches 14 and 15 where bald eagles have nested.
Bald eagles on average consume a summer diet consisting of 78.2% fish, 16.3% birds, and 5%
mammals.

7 The results of the evaluation indicate that potential risks to bald eagles from consuming 8 contaminated fish in areas downstream of Woods Pond are restricted to Reach 8, corresponding 9 to Rising Pond. However, Rising Pond is smaller that the typical eagle foraging area, so this 10 estimate of risk is conservative. In addition, the more in-depth analysis specific to Reaches 14 11 and 15 also did not show risk in the foraging area of the nesting bald eagles.

12 ES.6 BROADER IMPLICATIONS

13 The weight-of-evidence assessments indicate that COCs in the PSA of the Housatonic River, 14 particularly tPCBs, are causing risks to many of the species chosen to represent the assessment 15 endpoints. Risks from COCs, however, may potentially extend beyond adverse effects to 16 survival, growth, and reproduction of representative species. The Housatonic River ERA also 17 explores the broader implications of the risks of COCs to representative species, including 18 extension of the ecological risk assessment to species that occur in the Housatonic River 19 watershed, but that had not been considered explicitly in the quantitative ecological risk 20 assessments, and additional ecological implications.

21 ES.6.1 Implications for Other Species in the Primary Study Area

The major factors that influence exposure to tPCBs and TEQ and that were considered in the analysis include:

- Dietary composition.
- Foraging behavior and home range.
- Size, metabolism, and life history characteristics.
- Sensitivity to COCs.
- 28



EAGLE RISK

< 30.4 mg/kg</td>

> 30.4 mg/kg

> 30.4 mg/kg

> 30.4 mg/kg

> 30.4 mg/kg

State Boundary

Image: Note State Boundary

| O:\gepitt\aprs\era_species.apr | layout - eagle k4-5 | o:\gepitt\epsfiles\plots\in\eagle_risk_es15.eps | 11:40 AM, 7/8/2003 |

1 The ERA compares these factors between the representative species and other species in their 2 foraging groups. The comparison highlights similarities and differences, and their potential to 3 influence exposure and hence risks to tPCBs and TEQ.

Benthic Invertebrates—The benthic invertebrate ERA included the entire benthic community; benthic community composition analysis was a measurement endpoint considered in the weightof-evidence assessment. Both the status of sensitive taxa and community composition are considered indicators of overall health and productivity of the benthic community. Thus, there is no need to extrapolate the findings of the benthic invertebrate assessment described previously to other benthic invertebrate species in the PSA.

10 Amphibians—Certain amphibian species that were not studied may be more susceptible to the 11 effects of tPCBs because of their life history characteristics. For example, blue-spotted 12 (Ambystoma laterale) and spotted salamanders (Ambystoma maculatum) have a lifestage as aquatic carnivorous, bottom-dwelling larvae. Thus, they could potentially bioaccumulate PCBs 13 14 more quickly than herbivorous amphibians. Salamanders appeared in lower numbers in vernal 15 pools with high sediment tPCB concentrations. Several salamander species occur in 16 contaminated habitat in the PSA, including the spotted salamander, the Jefferson salamander 17 (Ambystoma jeffersonianum, formerly considered a variety of blue-spotted salamander), and the 18 four-toed salamander (*Hemidactylium scutatum*), the latter two of which are Species of Special 19 Concern.

20 Fish—There is evidence in the literature that salmonid species may have a higher sensitivity to 21 the effects of PCBs and other dioxin-like COPCs. The use of rainbow trout in the site-specific 22 toxicity testing program, combined with effects data from the literature, provides a high degree 23 of confidence that the ERA included an evaluation of fish species with equal or greater 24 sensitivities than the representative species listed above. However, the procedure used to 25 establish MATCs for fish in the PSA placed a low weight on studies conducted with fish species 26 known to be highly sensitive (e.g., lake trout), to avoid an overly conservative assessment. Risks 27 to coldwater fisheries (e.g., trout) downstream of the PSA were explicitly evaluated using 28 benchmarks developed for salmonids; the uncertainty in these downstream risk estimates is high 29 due to the number of extrapolations required. The risk of COCs to the occasional salmonid

occurring within the PSA is considered to be moderate. The PSA, however, is considered to be a
 warmwater fishery, and thus salmonid abundance is expected to be low in this portion of the
 river, even in the absence of chemical stressors.

Insectivorous Birds—The weight-of-evidence assessment indicated that exposure of insectivorous birds, such as tree swallows and American robins, to tPCBs and TEQ is high but unlikely to lead to adverse reproductive effects. Confidence in this conclusion, however, is not high because the two available lines of evidence for both species did not produce concordant results. There are a number of insectivorous birds with similar feeding habits as tree swallows in the PSA, and these are generally believed to be at the same low to moderate risk as tree swallows, although some species with higher food intake could be at higher risk.

11 Compared to American robins, eastern bluebirds and eastern towhees are expected to experience 12 lower to similar levels of risk from exposure to tPCBs and TEQ. The level of risk for the hermit 13 thrush, northern mockingbird, veery, and wood thrush is expected to be similar to American 14 robins. With the exception of earthworms in the robin diet, the dietary preferences of these birds 15 are similar to the American robin. The absence of earthworms, a major dietary source of 16 contaminants, will decrease their exposure to tPCBs and TEQ. However, their smaller body 17 sizes result in higher food intake rates and hence greater exposure to tPCBs and TEQ through 18 diet compared to American robins.

Piscivorous Birds—The weight-of-evidence assessment indicates that risks of tPCBs and TEQ
to belted kingfisher are low; however, risks of these COCs to osprey are high and could lead to
adverse reproductive effects.

The belted kingfisher and osprey were chosen to represent piscivorous birds inhabiting the Housatonic River area. Belted kingfisher and osprey are common piscivorous birds in the PSA. Great blue herons are also found in the PSA, and are discussed in Appendix K with other piscivorous birds (e.g., American bittern).

Piscivorous Mammals—Mink and river otter, the representative species for piscivorous
 mammals, are the only piscivorous mammals commonly found in the watershed of the
 Housatonic River.

1 Omnivorous and Carnivorous Mammals—The weight-of-evidence assessment indicates that 2 omnivorous and carnivorous mammals, such as red fox and short-tailed shrew, are at risk in the 3 PSA as a result of exposure to tPCBs and TEQ. Masked shrews are expected to experience a 4 level of risk similar to northern short-tailed shrews and smoky shrews are expected to be at 5 higher risk, based on their metabolic rates relative to short-tailed shrews. All three have similar 6 foraging behaviors and ranges.

7 Coyotes have a larger body size and foraging range that decreases their exposure to tPCBs and 8 TEQ. Considering these characteristics, coyotes are expected to experience lower risks from 9 exposure to tPCBs and TEQ than red fox. Gray and red foxes are expected to experience similar 10 risks from exposure to tPCBs and TEQ. Gray fox have a larger foraging range than red fox and 11 that may decrease their exposure to tPCBs and TEQ. Gray fox, however, have a greater reliance 12 on animal matter and therefore greater exposure to tPCBs and TEQ.

Fishers, long-tailed weasels, and short-tailed weasels are expected to experience similar to higher levels of risk from exposure to tPCBs and TEQ compared to the red fox due to greater consumption of animal matter and/or higher metabolic rate.

16 Threatened and Endangered Species—The bald eagle, American bittern, and small-footed 17 myotis were chosen to represent T&E species that are likely to be highly exposed to COCs in the 18 Housatonic River PSA. Other T&E species that occur in the area include one mussel (triangle 19 floater); three dragonflies (riffle snaketail, zebra clubtail, and arrow clubtail); a turtle (wood 20 turtle); three salamanders (Jefferson salamander, four-toed salamander, and northern spring 21 salamander); three hawks (northern harrier, sharp-shinned hawk, and Cooper's hawk); two 22 warblers (northern parula and blackpoll warbler); a wading bird (common moorhen); and a shrew 23 (northern water shrew). Some of these species were qualitatively assessed in other appendices 24 and compared to other, more appropriate, assessment endpoints (e.g., amphibians for 25 salamanders).

The level of risk for soras¹ is expected to be lower than for American bitterns because of greater 1 2 consumption of vegetable matter. Great blue herons and king rails are expected to experience a 3 similar level of risk as American bitterns because of a combination of size differences and some 4 differences in dietary preferences. The least bittern, green heron, Virginia rail, and pied-billed 5 grebe are expected to experience higher levels of risk compared to the American bittern. The 6 foraging and life history characteristics of these birds are similar to the American bittern. 7 However, these birds are much smaller than the American bittern. Their smaller body sizes 8 result in a higher metabolism and greater exposure to tPCBs and TEQ.

9 The Indiana bat, northern myotis, and little brown bat are expected to have similar levels of risk 10 as the small-footed myotis. These species belong to the same genus (*Myotis*) and have similar 11 foraging behaviors and life histories.

12 ES.7 SOURCES OF UNCERTAINTY

The assessment of risks of COCs to aquatic and wildlife species in the Housatonic River contains uncertainties. Each source of uncertainty can influence the estimates of risk; therefore, it is important to describe and, when possible, specify the magnitude and direction of such uncertainties. In this section, the most significant sources of uncertainty commonly encountered throughout the ERA are described. The sources of uncertainty are grouped by phase of the ERA (i.e., problem formulation, exposure assessment, effects assessment, risk assessment).

The problem formulation is intended to define the linkages between stressors, potential exposure, and predicted effects on ecological receptors. As such, the conceptual model provides the scientific basis for selecting assessment and measurement endpoints to support the risk assessment process. Potential uncertainties arise from lack of knowledge regarding ecosystem functions, failure to adequately address spatial and temporal variability in the evaluations of sources, fate and effects, omission of stressors, and overlooking secondary effects (EPA 1998).

¹ Several of the species included in this section (i.e., sora, great blue heron, green heron, Virginia rail, northern myotis, little brown bat) are not threatened and endangered species either federally or in Massachusetts and Connecticut (Appendix A). They are included in the discussion of T&E species because they are taxonomically and ecologically similar to either American bittern or to small-footed myotis.

The types of uncertainties associated with the conceptual model that links contaminant sources to effects include those associated with the identification of COCs, environmental fate and transport of COCs, exposure pathways, receptors at risk, and ecological effects. Of these, the identification of exposure pathways probably represents the primary source of uncertainty in the conceptual model. The detailed ecological characterization performed at this site has greatly reduced the uncertainties associated with problem formulation, yet some remain and are described below.

8 The exposure assessment is intended to describe the actual or potential co-occurrence of stressors 9 with receptors. As such, the exposure assessment identifies the exposure pathways and the 10 intensity and extent of contact with stressors for each receptor or group of receptors at risk. The 11 exposure models for wildlife were energetics-based models requiring information on body 12 weight, free living metabolic rate, proportions of food items in the diets, and the concentrations 13 of COCs in these food items. Each of these variables has associated uncertainties, most of which 14 were propagated through the exposure models. The effects assessment is intended to describe the 15 effects caused by stressors, link them to the assessment endpoints, and evaluate how effects 16 change with fluctuations in the levels (i.e., concentrations or doses) of the various stressors. In 17 this assessment, the effects of tPCBs and other COCs to representative species were assessed. 18 There are several sources of uncertainty in the assessment of effects, including extrapolation 19 errors and a limited number of toxicity studies conducted with the representative species.

20 For benthos and amphibians, the effects benchmarks derived from the literature had a high 21 degree of uncertainty because of the need to extrapolate across sites and species. The site-22 specific fish toxicity studies indicated variations in the concentration-response relationships 23 observed across species, reaches, and treatments, and introduced uncertainty into the 24 development of effects thresholds. The methodology used in site-specific fish studies was 25 developed recently, and there are potential uncertainties inherent to extrapolating these 26 laboratory-based results to Housatonic River fish. Similarly, the extrapolation of concentrations 27 of tPCBs in egg to whole body concentrations has a degree of associated uncertainty.

The greatest potential source of uncertainty for the fish and wildlife effects assessments,
however, was associated with the lack of toxicity studies involving the representative species.

A weight-of-evidence procedure was used to assess risks of tPCBs and TEQ to the assessment
 endpoints in the Housatonic River PSA. The analysis follows the methodology proposed by the
 Massachusetts Weight-of-Evidence Workgroup (Menzie et al. 1996; see Section 2.9 for details).

4 In general, the weight-of-evidence approach is an inclusive process whereby multiple lines of 5 evidence are considered prior to determining risk. For the wildlife risk assessments, these lines 6 of evidence included the exposure and effects modeling results and, in some cases, field survey 7 results, and/or in situ or whole media toxicity test results. For the fish and benthic invertebrate 8 risk assessments, available lines of evidence included field survey results (e.g., community 9 evaluation for benthos), site-specific toxicity tests, and comparison of tissue and sediment 10 concentrations to benchmarks (both from the literature and site-specific benchmarks). The 11 largest source of uncertainty in the weight-of-evidence process was the development of 12 conclusions based upon only one or two lines of evidence.

13 ES.8 SUMMARY AND CONCLUSIONS

Weight-of-evidence assessments indicated that aquatic life and wildlife in the Primary Study Area of the Housatonic River are experiencing unacceptable risks as a result of exposure to tPCBs and other COCs. Confidence in this conclusion is high for benthic invertebrates, amphibians, and piscivorous mammals because multiple lines of evidence gave concordant results.

The risks of tPCBs and other COCs likely extend beyond the representative species considered in the quantitative risk assessments described herein. Qualitative risk assessments indicated that many other species in the PSA are potentially at risk. Further, there are likely indirect effects (e.g., changes in predator-prey relationships, changes in metapopulation dynamics) occurring inside and outside the PSA as a result of the direct impacts caused by tPCBs and other COCs.

An assessment of risk downstream of the PSA indicated that tPCBs could potentially be causing adverse effects to benthic organisms in depositional areas as far as Reach 8, amphibians in floodplain areas as far as Reach 8, trout in Reaches 7 and 9, mink as far as Reach 10, and river otter as far as Reach 12.

1 1. INTRODUCTION

Purposes of ERA 2 3 This ecological risk assessment (ERA) characterizes and quantifies the current and 4 potential risks to biota exposed to contaminants in the Housatonic River below the 5 confluence of the East and West Branches. Specific purposes of the ERA include: 6 1. To consider the fate and transport of PCBs and other contaminants of potential 7 concern (COPCs) to ecological receptors in the river and associated floodplain. 8 2. To identify the potential routes of exposure and toxicological effects of the 9 COPCs for these receptors. 10 3. To identify assessment endpoints representative of species potentially at risk. 11 4. To determine the risk to the assessment endpoints selected.

12 **1.1 OVERVIEW**

13 The purpose of this ecological risk assessment (ERA) is to characterize and quantify the current 14 and potential risks to biota exposed to contaminants of potential concern (COPCs) in the 15 Housatonic River below the confluence of the East and West Branches, focusing on 16 polychlorinated biphenyls (PCBs) and other COPCs originating from the General Electric Company (GE) facility in Pittsfield, MA. This ERA considers the fate and transport of PCBs 17 18 and other COPCs to ecological receptors in the river and associated floodplain, identifies 19 assessment endpoints that are representative of species potentially at risk, and identifies the 20 potential routes of exposure and toxicological effects of the COPCs for these receptors.

This information is synthesized, through a weight-of-evidence approach, into a discussion of the nature and magnitude of the risks for the assessment endpoints, and the uncertainties associated with the characterization of these risks.

24 Multiple lines of evidence for each assessment endpoint are evaluated, including where 25 applicable or available:

26	
27	

- Field surveys/studies.
- Toxicity tests.
 - Comparison of effects in the literature to a site-specific exposure model.

28 29

MK01|O:\20123001.096\ERA_PB\ERA_PB_1.DOC

1 The Housatonic River flows from east of Pittsfield, MA, to Long Island Sound and drains an area 2 of approximately 1,950 square miles (500,000 hectares) in Massachusetts, New York, and 3 Connecticut (Figure 1.1-1). The Housatonic River, its sediment, and associated floodplain have 4 been contaminated with polychlorinated biphenyls (PCBs) and other hazardous substances 5 released from the General Electric Company (GE) facility located in Pittsfield, MA. The entire 6 site, known as the General Electric/Housatonic River Site, consists of the 254-acre (103-hectare) 7 GE manufacturing facility; the Housatonic River and associated riverbanks and floodplain from 8 Pittsfield, MA, to Long Island Sound; former river oxbows that have been filled; neighboring 9 commercial properties; Allendale School; Silver Lake; and other properties or areas that have 10 become contaminated as a result of GE's facility operations.

11 Because of its size and complexity, the GE/Housatonic River Site has been divided into several areas for investigation and cleanup. The "Rest of River" is the portion of the river from the 12 13 confluence of the East and West Branches of the Housatonic River (the confluence) to the 14 Massachusetts border with Connecticut, a distance of approximately 54 miles (87 km), and 15 beyond into Connecticut to Long Island Sound. The total distance from the confluence to Long 16 Island Sound is approximately 139 miles (223 km). In addition to the river itself, the Rest of 17 River includes the associated riverbank and floodplain. The Rest of River is further defined in 18 the Consent Decree entered with the U.S. District Court, Western Region, Massachusetts, in 19 October 2000. The Rest of River includes areas of the Housatonic River and its sediment and 20 floodplain (except Actual/Potential Lawns), in which contaminants originating from the GE 21 facility are located. The lateral extent of the area under investigation includes the floodplain 22 extending to the 1-ppm total PCB (tPCB) isopleth, which is approximately equivalent to the 10-23 year floodplain.

The ERA focuses on the portion of the river from the confluence of the East and West Branches 2 miles (3 km) below the GE facility, to Woods Pond Dam, a distance of approximately 11 river 2 miles (18 km). This area is referred to as the Primary Study Area (PSA) in the Supplemental 2 Investigation (Figure 1.1-2), and is where much of the PCB contamination was found in previous 2 studies. The river (which includes free-flowing and impounded sections) and the floodplain 2 downstream of Woods Pond to the Derby-Shelton Dam in Connecticut are also considered in the



| O:\gepitt\aprs\era_figures2.apr | layout - fig1.1-1 | o:\gepitt\epsfiles\plots\in\fig1-1-1.eps | 11:25 AM, 7/10/2003 |



| O:\gepitt\aprs\era_figures2.apr | layout - fig1.1-2 | o:\gepitt\epsfiles\plots\in\fig1-1-2.eps | 9:50 AM, 3/7/2003 |

ERA. Beyond this dam, the river is subject to tidal influence, as well as COPCs (including
 PCBs) from other hazardous waste sites.

This ERA is structured as an integrated report summarizing information included in the supporting appendices and providing background common to all the assessment endpoints. The potential risk for each assessment endpoint is discussed in detail in Appendices D through K and is summarized in Sections 3 through 11. Other appendices provide additional information such as a comprehensive Ecological Characterization, the identification of COPCs and the extent of contamination for further consideration in the ERA, and other supporting information. Figure 1.1-3 provides a roadmap for the ERA and supporting appendices.

10 1.2 SITE HISTORY

The Housatonic River is located in a predominantly rural area of western Massachusetts and Connecticut, where farming was the main occupation from colonial settlement through the late 1800s. As with most rivers, the onset of the industrial revolution in the late 1800s brought manufacturing to the banks of the Housatonic River in Pittsfield, MA. GE began operations in its present location in 1903. Three manufacturing divisions have operated at the GE facility (Transformer, Ordnance, and Plastics).

17 The 254-acre (103-ha) GE facility in Pittsfield (Figure 1.2-1) has historically been the major 18 handler of PCBs in western Massachusetts, and is the only known source of PCBs found in the 19 Housatonic River sediment and floodplain soil in Massachusetts. Although GE performed many 20 functions at the Pittsfield facility throughout the years, the activities of the Transformer Division, 21 including the construction and repair of electrical transformers using dielectric fluids, some of 22 which contained PCBs (primarily Aroclor 1260 and, to a lesser extent, 1254), were one likely 23 significant source of PCB contamination. According to GE reports, from 1932 through 1977, 24 releases of PCBs reached the wastewater and stormwater systems associated with the facility and 25 were subsequently conveyed to the East Branch of the Housatonic River and to Silver Lake, a 26 25-acre (10-ha) lake adjacent to the GE facility.

During the 1940s, efforts to straighten the Pittsfield reach of the Housatonic River by the City of
Pittsfield and the U.S. Army Corps of Engineers (USACE) resulted in 11 former oxbows being





[|] O:\gepitt\aprs\raas_locs.apr | layout - fig 1.2-1 | o:\gepitt\epsfiles\plots\in\fig1-2-1.eps | 10:31 AM, 7/10/2003 |

isolated from the river channel. The oxbows were filled with material, much of which was later
 discovered to contain PCBs and other hazardous substances.

The State of Connecticut posted a fish consumption advisory for most of the Connecticut section of the river in 1977 as a result of the PCB contamination in the river sediment and fish tissue. In 1982, the Massachusetts Department of Public Health (MADPH) issued a consumption advisory for fish, frogs, and turtles for the Housatonic River. In addition, in 1999, MADPH issued a waterfowl consumption advisory from Pittsfield to Great Barrington due to PCB concentrations in wood ducks and mallards collected from the river by the U.S. Environmental Protection Agency (EPA).

10 Although the first 2 miles (3 km) downstream from the facility have been channelized, the 11 remainder of the river's course is relatively unaffected (with the exception of the numerous dams 12 downstream) in areas south of Pittsfield. The river, from the confluence of the East and West 13 Branches of the Housatonic to Woods Pond Dam in Lenox, is approximately 11 miles (18 km) 14 long. The channel ranges from 40 to 125 ft (12 to 38 m) in width, is bordered by an extensive 15 floodplain (up to 3,000 feet [900 m] wide), and has a meandering pattern with numerous oxbows 16 and backwaters. Woods Pond, the first impoundment below the GE facility, is a shallow 54-acre 17 (22-ha) impoundment that was formed by the construction of a dam in 1864 (Harza, 2001 as 18 cited in BBL and QEA, 2003).

19 The land uses of the floodplain properties in Massachusetts include residential, 20 commercial/industrial, agricultural, recreational (such as canoeing, fishing, and hunting), wildlife 21 management, and parks and a golf course. The Housatonic River floodplain is an attractive area 22 for recreation, including fishing and waterfowl hunting.

Numerous studies conducted since 1988 have documented PCB contamination of soil within the floodplain of the Housatonic River downstream of the GE facility. PCBs have been detected in river sediment in Massachusetts as far downstream as the border with Connecticut (BBL 1995), and in Connecticut as far as the Derby-Shelton Dam and beyond into Long Island Sound (other sources have been identified downstream of this dam). The PCBs detected in Housatonic River floodplain soil and sediment consist of predominantly Aroclor 1260, with a minor contribution of Aroclor 1254. Numerous residential properties have been the focus of efforts by the Massachusetts Department
 of Environmental Protection (MDEP) to coordinate cleanup of residential soil contaminated with
 PCBs that was brought to the properties as fill from GE. GE has cleaned up approximately 170
 properties to date under this program.

5 Other properties or areas in Pittsfield and the surrounding communities have been discovered 6 over the years to have received waste from the GE facility and/or are contaminated with PCBs, 7 including the Pittsfield Landfill, Rose Disposal Site (National Priorities List [NPL] site), and 8 Dorothy Amos Park located on the West Branch of the Housatonic River. Actions to address 9 these properties have been taken or investigation is underway.

The highest concentrations of Aroclors 1254 and 1260 have been detected in the vicinity of the plant and downstream of Building 68 (WESTON 2000; BBL 1994, 1995; O'Brien & Gere Engineers, Inc. 1995). Widespread contamination of the river downstream of the GE facility has resulted from the transport of PCB-contaminated river sediment and floodplain soil by river flow, sediment transport, and overbank flooding (WESTON 2000). Total PCBs have been detected at concentrations of greater than 1 ppm in floodplain soil as far downstream as Bartholomew's Cobble in Massachusetts, close to the Massachusetts-Connecticut state line.

17 1.3 REGULATORY BACKGROUND

18 The GE/Housatonic River site has been subject to regulatory investigations dating back to the 19 early 1980s. For several years, these investigations were consolidated under the following 20 regulatory mechanisms: two Administrative Consent Orders (ACOs) with MDEP and a 21 Corrective Action Permit with EPA under the Hazardous and Solid Waste Amendments to the 22 Resource Conservation and Recovery Act (RCRA).

In 1991, EPA issued a RCRA Corrective Action Permit to the GE Pittsfield facility. Following
appeals by GE and others, and subsequent modification, the permit became effective in 1994.
The permit included the 254-acre facility, some filled former oxbows, Silver Lake, the
Housatonic River and its floodplains and adjacent wetlands, and all sediment contaminated by
PCBs migrating from the GE facility.

1 In addition to the permit, the two ACOs between GE and MDEP became effective in 1990 and 2 included those areas defined in the permit, as well as additional filled former oxbows and 3 Allendale Elementary School. Under the ACOs, GE has performed several investigations and 4 short-term cleanups.

5 EPA proposed the site to the Superfund National Priorities List (NPL) in September 1997. 6 Several federal and state government agencies and GE entered into negotiations late in 1997 with 7 the goal of reaching a comprehensive settlement, which included remediation, redevelopment, 8 and restoration components.

9 In September 1998, representatives of the federal and state government agencies, GE, the City of 10 Pittsfield, and the Pittsfield Economic Development Authority reached an agreement in principle 11 relating to GE's Pittsfield facility, other contaminated areas in Pittsfield, and the Housatonic 12 River. This agreement was translated into a Consent Decree, lodged with the federal court on 7 13 October 1999, and entered by the court on 27 October 2000. The agreement provides for, among 14 other things, the cleanup of the GE plant facility, cleanup and restoration of the former oxbows, 15 cleanup and restoration of Silver Lake, cleanup of Allendale School, environmental restoration 16 projects related to the Housatonic River and floodplains, monetary compensation for natural 17 resource damages, and government recovery of past and future response costs. Entry of the 18 agreement also makes possible large-scale redevelopment of the GE facility.

19 The GE/Housatonic River site is made up of several separate response actions (as described in 20 the Consent Decree), including three actions in the river:

Upper ¹/₂-Mile Reach Housatonic River Removal Action (¹/₂-Mile Reach).

1 ¹/₂-Mile Housatonic River Removal Action (1 ¹/₂-Mile Reach). 23 Rest of River. 24 25 and actions outside the river, including: 26 • GE plant site soil remediation. 27 Unkamet Brook and floodplain remediation. Hill 78/Building 71 consolidation areas. 28 29 Groundwater remediation. • 30 Former oxbow areas. 31 Allendale School.

21

22

- Floodplain current residential and nonresidential properties.
- Silver Lake.

1

2

3

The primary COPCs are PCBs, specifically Aroclor 1260 and, to a lesser extent, 1254. Other
COPCs include volatile organics, dioxins/furans, polycyclic aromatic hydrocarbons (PAHs),
semivolatiles, and metals. These contaminants vary in their distribution in different areas.

7 EPA completed an investigation of the Rest of River below the 11/2-Mile Reach into Connecticut, 8 which focused on collecting information for and preparing the human health and ecological risk 9 assessments, and modeling PCB fate and transport in the river. Under the terms of the Consent 10 Decree, both of the risk assessments and three aspects of the modeling effort are to undergo 11 formal external Peer Review, with the review of the Modeling Framework Design having taken 12 place in April 2001. The ecological risk assessment, together with the human health risk 13 assessment and the model of PCB fate, transport, and bioaccumulation, will inform EPA's decision on what additional remedial actions, if any, may be required in the river and floodplain 14 15 below the confluence.

16 Following the investigations, as required in the Revised RCRA Permit, GE has prepared a 17 Supplemental RCRA Facility Investigation Report (BBL and QEA 2003), will propose cleanup 18 levels (Interim Media Protection Goals), and will analyze cleanup alternatives (Corrective 19 Measures Study) for consideration by EPA. EPA will propose the draft Statement of Basis 20 (cleanup plan, scheduled for 2006) for the Corrective Measure(s) for the Rest of River and, after 21 public comment, will finalize the Statement of Basis. GE and other members of the public may 22 then appeal EPA's decision to the EPA Environmental Appeals Board (EAB) and the First 23 Circuit District Court. GE is then required, under the Consent Decree, to implement and pay for 24 the remedy selected after resolution of any appeals. The Rest of River response action, if any, 25 will be implemented through a modification to the Revised RCRA Permit and an amendment to 26 the CERCLA Consent Decree, and is estimated to begin in 2007.

27 1.4 SITE DESCRIPTION

The Rest of River portion of the Housatonic River flows through one of the most biologically diverse regions of Massachusetts (Barbour et al. 1998) and Connecticut. Dams play an integral role in the downstream migration of PCBs and other COPCs from the GE facility.

1-11

The first 10.5 miles (16.9 km) from the confluence to the headwaters of Woods Pond is referred to as Reach 5 (Figure 1.4-1). Other than the initial 0.5-mile (0.8-km) reach bordering the GE facility, Reach 5 has the highest concentrations and highest frequency of detections of PCBs in sediment. Reach 5 is subdivided further into four parts: Reach 5A, from the confluence to just above the Pittsfield Wastewater Treatment Plant (WWTP); Reach 5B, from the WWTP to Roaring Brook; Reach 5C, from Roaring Brook to the headwaters of Woods Pond; and Reach 5D, the backwaters above Woods Pond (Woods Pond is Reach 6) (Figures 1.4-2 and 1.4-3).

8 The Housatonic River meanders through Reach 5A, with widths between 50 and 120 ft (15 and 9 37 m) and depths up to 11 ft (3.4 m) (HEC 1996). Aquatic habitat includes snags (large woody 10 debris), undercut banks, and rocks. Land use in this section is predominantly forested and 11 cleared, with some residential areas. Reach 5B is similar to Reach 5A from the WWTP to New 12 Lenox Road. The land near New Lenox Road is predominantly agricultural and forested. Below 13 New Lenox Road, the river widens (60 to 160 ft [18 to 48 m]) and becomes shallower (4 to 8 ft 14 [1.2 to 2.4 m]). This portion of Reach 5B is dominated by a broad wetland floodplain, ranging 15 from 800 to 3,000 ft (240 to 910 m) wide (see Appendix A). Reach 5C is similar to Reach 5B, 16 although as the Housatonic River approaches Woods Pond, the velocity decreases and deep pools 17 occur (up to and exceeding 7 ft [2 m]), created by large snags that divert water flow, and the 18 effect of Woods Pond Dam becomes apparent. Dense vegetation lines the banks of the river in 19 the upper portion of this section, while extensive backwaters border the lower section. Reach 5D 20 consists of several upstream backwater areas associated with Woods Pond and covers more than 21 120 acres (49 ha).

Reach 6 begins 10.5 miles (16.9 km) downstream of the confluence at Woods Pond. The pond is approximately 0.2 mile (0.3 km) in length and has an area of 60 acres (24 ha) (Figure 1.4-4).



| O:\gepitt\aprs\era_figures2.apr | layout - fig1.4-1 | o:\gepitt\epsfiles\plots\in\fig1-4-1.eps | 10:13 AM, 3/7/2003 |



| O:\gepitt\aprs\era_figures2.apr | layout - fig1.4-2 | o:\gepitt\epsfiles\plots\in\fig1-4-2.eps | 10:16 AM, 3/7/2003 |



| O:\gepitt\aprs\era_figures2.apr | layout - fig1.4-3 | o:\gepitt\epsfiles\plots\in\fig1-4-3.eps | 10:35 AM, 3/7/2003 |



| O:\gepitt\aprs\era_figures2.apr | layout - fig1.4-4 | o:\gepitt\epsfiles\plots\in\fig1-4-4.eps | 3:36 PM, 6/26/2003 |

1 This reach contains the first impoundment downstream from the GE facility and is a depositional 2 environment (HEC 1996). The water in Woods Pond is relatively slow-moving and contains 3 aquatic habitat characteristic of a standing water environment. The maximum depth is 16 ft (4.9 4 m), but most of the pond is 1 to 3 ft (0.3 to 0.9 m) deep (HEC 1996; Stewart Laboratories, Inc. 1982; CR Environmental 1998). The banks of the pond provide extensive cover, such as 5 6 overhanging vegetation, woody debris, rock piles, and submerged macrophytes. The Woods 7 Pond Dam was built in 1864. In 1989, GE replaced the original dam with a concrete weir dam 8 located 180 ft (55 m) downstream of the original dam site.

9 Reach 7 extends 18.6 miles (29.9 km) from Woods Pond to the upstream end of Rising Pond in
10 Great Barrington (Figure 1.4-5). There are five dams in this reach, and the river has an average
11 gradient of 14.5 ft (4.42 m) per mile, and an average depth of 1 ft (0.3 m) (Stewart Laboratories,
12 Inc. 1982). Agricultural activity becomes more common in this area than in the upstream
13 reaches.

Reach 7 ends above Rising Pond, which is Reach 8 (Figure 1.4-5). This 45-acre (18-ha) pond
was created by the construction of a dam at the Rising Paper Company (WESTON 2000).
Rising Pond has depositional characteristics similar to Woods Pond.

17 Reach 9 begins downstream of Rising Pond and extends for approximately 24.6 miles (39.6 km) 18 to the Massachusetts/Connecticut state line (Figure 1.4-5). It contains low-gradient sections with 19 river habitat, as well as moderate gradient sections with riffle habitat. This reach is wide with 20 flat floodplains and several oxbows, and agriculture is a predominant land use.

21 Reach 10 begins at the Massachusetts/Connecticut border and extends 7.4 miles (12 km) to the 22 dam at Great Falls Village (Figure 1.4-6). The river characteristics are similar to those of Reach 23 9, with a meandering river course. Reach 11 begins on the downstream side of the dam at Great 24 Falls and ends at Cornwall Bridge, where Route 7 crosses the river (Figure 1.4-6). This reach is 25 11.5 miles (18.5 km) long. Reach 11 is mostly shallow and fast flowing, and much of the reach 26 is designated as a Trout Management Area. Reach 12 extends from Cornwall Bridge to the dams 27 at Bulls Bridge (Figure 1.4-6), a length of 13.1 miles (21.1 km). The river is relatively straight 28 through this reach and flows quickly for most of the run. Near the town of Kent, the river slows 29 and deepens as it enters the backwaters from the dams at Bulls Bridge. Reach 13 starts on the



[|] O:\gepitt\aprs\era_figures2.apr | layout - fig1.4-5 | o:\gepitt\epsfiles\plots\in\fig1-4-5.eps | 3:43 PM, 6/26/2003 |



[|] O:\gepitt\aprs\era_figures2.apr | layout - fig1.4-6 | o:\gepitt\epsfiles\plots\in\fig1-4-6.eps | 10:20 AM, 3/7/2003 |

downstream side of the dams at Bulls Bridge and runs 10.9 miles (17.5 km) to the Bleachery
Dam at New Milford, CT (Figure 1.4-6). The Bleachery Dam is virtually submerged as a result
of the backwater created by the Shepaug Dam farther downstream. The river meanders more
than in the previous reach and, as in Reaches 11 and 12, flows quickly.

5 Reach 14, from the Bleachery Dam to Shepaug Dam, is known as Lake Lillinonah (Figure 1.4-6 7). The reach is 11.5 miles (18.5 km) long. The Shepaug Dam is approximately 100 ft (30 m) 7 high. The backwater effect from the Shepaug Dam extends all the way to the upstream 8 Bleachery Dam. The Shepaug Dam is used for power generation and this may affect water 9 levels during the year. Water movement is slow through this reach and the river is deep. Reach 10 15 encompasses Lake Zoar, from Shepaug Dam to Stevenson Dam (Figure 1.4-7). This 11 predominantly slow-moving reach is 10.2 miles (16.4 km) long. The backwater effect of the 12 Stevenson Dam extends upstream for almost the entire reach. The Stevenson Dam is 13 approximately 100 ft (30 m) high and supports power generation. Some homes and boat 14 launches are found on Lake Zoar.

15 Reach 16 is Lake Housatonic and is bounded by the Stevenson Dam and the Derby-Shelton Dam 16 (Figure 1.4-7). The reach is 6.0 miles (9.7 km) long and, like the previous two upstream reaches, 17 is slow moving. The Derby-Shelton Dam (approximately 25 ft [7.6 m] high) is smaller than 18 either the Shepaug or Stevenson Dams. More homes and boat launches occur along this reach. 19 Reach 17, from Derby-Shelton Dam to Long Island Sound, is 13.7 miles (22.0 km) long (Figure 20 1.4-7). This reach is entirely tidally influenced. It is shallow in its upstream portions and 21 deepens downstream. The Naugatuck River enters the Housatonic River approximately 2 miles 22 (3.2 km) from the upstream end of this reach.

23 1.5 OVERVIEW OF TECHNICAL APPROACH

This ERA follows the technical approach and guidelines detailed in EPA's *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments* (EPA, 1997a). Additional documents were also consulted, including, but not limited to, the following:

28



[|] O:\gepitt\aprs\era_figures2.apr | layout - fig1.4-7 | o:\gepitt\epsfiles\plots\in\fig1-4-7.eps | 12:05 PM, 7/1/2003 |
1	•	Framework for Ecological Risk Assessment (EPA/630/R-92/001, 1992a).
2	•	Guidelines for Ecological Risk Assessment (EPA/630/R-95-002F, April 1998).
3 4	•	Ecological Risk Assessment Issue Papers (EPA/630/R-94/009, November 1994, 1994e).
5 6	•	<i>Wildlife Exposure Factors Handbook</i> , Volumes I and II (EPA 600/R-93/187a and 187b, December 1993, 1993a).
7 8	•	Guidance for Disposal Site Risk Characterization: Method 3 Environmental Risk Characterization (MDEP 1996).
9 10	•	The Role of BTAGs in Ecological Assessment, ECO Update, Volume 1, Number 1 (EPA 1991a).
11 12	•	Ecological Assessment of Superfund Sites: An Overview, ECO Update, Volume 1, Number 2 (EPA 1991b).
13 14	•	The Role of Natural Resource Trustees in the Superfund Process, ECO Update, Volume 1, Number 3 (EPA 1992b).
15 16	•	Using Toxicity Tests in Ecological Risk Assessment, ECO Update, Volume 2, Number 1 (EPA 1994a).
17 18	•	Catalogue of Standard Toxicity Tests for Ecological Risk Assessment, ECO Update, Volume 2, Number 2 (EPA 1994b).
19 20	•	Field Studies for Ecological Risk Assessment, ECO Update, Volume 2, Number 3 (EPA 1994c).
21 22	•	Selecting and Using Reference Information in Superfund Ecological Risk Assessments, ECO Update, Volume 2, Number 4 (EPA 1994d).
23	•	Ecotox Thresholds, ECO Update, Volume 3, Number 2 (EPA 1996).
24 25	•	RAGS, Volume 3, Part A: Process for Conducting Probabilistic Risk Assessment (EPA 540-R-02-002, December 2001).
26	•	Guiding Principles for Monte Carlo Analysis (EPA/63C/R-97/001, 1997b).
27 28	•	Ecological Risk Assessment and Risk Management Principles for Superfund Sites (EPA 1999).
29		

1 The Ecological Risk Assessment Guidance for Superfund (EPA 1997a) details an eight-step 2 process for conducting an ERA (Figure 1.5-1). This document provides the user with a basic 3 framework for the ERA process and ensures a consistent approach to conducting ERAs. In 4 addition to these steps, there are several scientific/management decision points (SMDPs). These 5 are opportunities for the risk manager and the risk assessment team to communicate ideas 6 concerning the scope, focus, and direction of the ERA. The first two steps of the ERA process 7 (Screening-Level Problem Formulation and Screening-Level Exposure Estimate and Risk 8 Calculation) were first addressed in the Upper Reach-Housatonic River Ecological Risk 9 Assessment (WESTON 1998) and subsequently refined in Appendix B of this document. Steps 10 3, 4, and 5 (Baseline Problem Formulation, Study Design and DQO Process, and Verification of 11 Field Data Analysis) are iterative components of the eight-step ERA process. Steps 3 through 5 12 were initially presented in the Supplemental Investigation Work Plan for the Lower Housatonic 13 River (SIWP) (WESTON 2000) and were modified as necessary during the data collection phase 14 of the project. Steps 6 and 7 (Site Investigation and Data Analysis and Risk Characterization) 15 are presented in detail in the following sections and appendices. Step 8 (Risk Management) will 16 be addressed after the ERA has undergone Peer Review.

17 **1.5.1 Problem Formulation**

Problem formulation is an important component of the ERA process that establishes the goals, objectives, and scope for the ERA. Products of problem formulation include the identification of assessment endpoints, illustration of exposure pathways (relating fate and transport to ecological effects), a conceptual model depicting the relationships between COPCs and the assessment endpoints, and risk hypotheses and questions that can be drawn from evident or suspected effects. The problem formulation portion of the ERA is discussed in Section 2 and was outlined in the *Supplemental Investigation Work Plan for the Lower Housatonic River* (WESTON 2000).

25 **1.5.1.1** *Physical and Ecological Characterization*

An extensive physical and ecological characterization of the Housatonic River is presented in Section 2.2 and Appendix A (Ecological Characterization) of this document. These sections detail the physical setting, habitats, and biotic communities of the river in both the aquatic and

Ecological Risk Assessment for the Housatonic River



terrestrial environments. The physical and ecological characterizations aid in identifying
 representative species and exposure pathways, provide information for the exposure analyses,
 and can inform risk managers on the potential impacts of future remedial actions.

4 1.5.1.2 Stressors and Their Sources

5 Investigations of the nature and extent of contaminants in the Housatonic River watershed have 6 previously been conducted by GE, EPA, and others. PCBs have been identified as the main 7 COPC, and other contaminants such as dioxins and furans and PAHs have also been identified at 8 the GE facility. In addition, other COPCs, such as pesticides, were detected in the PSA, 9 although at lower concentrations and frequencies of detection. In Section 2.3, the sources, 10 amounts, and patterns of contaminant releases and receiving bodies are presented.

11 **1.5.1.3** Pre-Ecological Risk Assessment

12	COPC vs COC
13 14 15 16 17	In the ERA, contaminants of potential concern (COPC) refer to contaminants considered before, during, and immediately after the Pre-ERA process. A contaminant is considered a contaminant of concern (COC) if it has passed through all screening-level processes and is included as part of the exposure and effects assessment conducted for a specific assessment endpoint.
10	

18

The purpose of the Pre-ERA (Appendix B) was to identify contaminants that warranted more detailed analyses in the ERA, and those that could be removed from further consideration because they pose minimal risk. For those contaminants that screened through to the ERA, the primary media of concern as well as the sections of the study area that are potentially impacted are identified. A summary of the Pre-ERA is provided in Section 2.4. The complete Pre-ERA is included as Appendix B to this document.

25 **1.5.1.4** Fate and Transport of Contaminant Stressors

26 An overview of the environmental behavior of PCBs and other COPCs is presented in Section

27 2.5. This section includes discussions of the transport of the contaminants from their point(s) of

release, partitioning behavior in different media, and biotic and abiotic degradation in these
 media.

3 1.5.1.5 Effects on Representative Species

The effects and mechanisms of toxicity to biota of the contaminants identified as COPCs within the Housatonic River and floodplain are discussed, with an emphasis on PCBs, in Section 2.6, and in further detail in the effects assessment portion of each assessment endpoint section and corresponding appendix.

8 1.5.1.6 Conceptual Model, Selection of Assessment and Measurement 9 Endpoints, and Analysis Plan

10 The conceptual model outlined in Section 2.7 describes the relationship between the COPCs and 11 the biota at the site. Development of a conceptual model includes review of sources of 12 contamination, evaluation of the spatial scale for the assessment, description of the exposure 13 pathways, and formulation of risk questions to be addressed.

14 Assessment and measurement endpoints are defined and described in Section 2.8. An 15 assessment endpoint is defined as the "explicit expression of the environmental value that is to 16 be protected" (EPA 1997a). Because it is often unrealistic to perform an assessment for all 17 species present at a contaminated site, species or populations are often grouped based on their 18 similarities (e.g., exposure pathway, contaminant sensitivity) or societal importance (e.g., 19 threatened and endangered species), and assessment endpoints are established for these groups of 20 similar species. A measurement endpoint is defined as "a measurable ecological characteristic 21 that is related to the valued characteristic chosen as the assessment endpoint," and is a measure 22 of biological effects (e.g., mortality, reproduction, growth) (EPA 1997a). Measurement 23 endpoints are frequently numerical expressions of observations (e.g., toxicity test results, 24 community diversity measures) that can be compared statistically to a reference site to detect 25 adverse responses to a site contaminant (EPA 1997a).

Section 2.9 describes the analytical approach used to estimate risks and the weight-of-evidence
approach used to develop the conclusions.

1-26

1 **1.5.2** Assessment of Representative Species

Sections 3 through 11 (and their corresponding appendices) provide an overview of the exposure
assessment, the effects assessment, and the risk characterization for each representative species
or representative group of species.

5 1.5.2.1 Exposure Assessment

6 The exposure assessment sections include a description of the data collection activities and the 7 studies conducted to determine concentrations of COPCs in water, soil, sediment, and biota 8 samples. Previous sampling and monitoring studies are also described in this section. Variation 9 in PCB and COPC concentrations over space and time in each environmental medium is briefly 10 characterized. For each assessment endpoint, one or more representative species were selected 11 and an appropriate exposure model identified.

12 1.5.2.2 Effects Assessment

The effects assessment sections begin with an overview of the toxicity of PCBs and other COPCs. For each major representative species group and COPC, the effects literature was reviewed. The goal of this review was to identify key studies that could be used to develop effects metrics for use in risk characterization. The effects metrics developed ranged from concentration- or dose-response curves to benchmarks depending on the quality and relevance of the data available.

19 1.5.2.3 Risk Characterization

The risk characterization sections for each assessment endpoint consider three major lines of evidence (where available): (1) comparison of estimated exposures to laboratory-based effects metrics, (2) results of in situ or whole media toxicity tests, and (3) results of field surveys. Probabilistic methods were used to integrate COPC exposure distributions in the study area with laboratory-derived benchmarks and effects curves. The format of the discussion includes an overview of the study, statistical analyses of the results and conclusions stating the observed effects, and likely causal agents. The risk characterization concludes with a weight-of-evidence
 assessment for each assessment endpoint. Primary sources of uncertainty are also identified.

3 1.6 DATA SOURCES

The Housatonic River ERA generally relies on data from studies and research specifically designed for this assessment. Field surveys were conducted to support the ecological characterization and ecological risk assessments for benthic invertebrates, amphibians, fish, birds, and mammals in the Housatonic River floodplain. Prior to the surveys, literature reviews were conducted to establish historic populations and habitats for species within the study area. Surveys were also conducted at several reference sites (i.e., areas of relatively low contamination within the Housatonic River watershed).

- 11 A variety of site-specific studies were conducted, including the following:
- Survival, growth, and reproduction of benthic organisms as part of the Sediment
 Quality Triad (SQT) approach.
- Reproductive success of amphibians in the Housatonic River floodplain and the effects of exposure to PCBs and other COPCs on these species.
- Studies with largemouth bass (*Micropterus salmoides*) to determine if exposure of adults to PCBs and COPCs in river water and sediment adversely affect the survival and development of offspring.
- Investigation of tree swallows (*Tachycineta bicolor*) to determine the extent to which
 PCBs and other COPCs are impairing their reproduction.
- A reproductive toxicology study with farm-raised mink (*Mustela vison*) exposed to PCBs and other COPCs in their diet from fish collected from the Housatonic River.

These and other studies are described in more detail in the *Supplemental Investigation Work Plan for the Lower Housatonic River* (WESTON 2000) and its appendices. Information on study design, methodology, and quality assurance (QA)/quality control (QC) procedures can also be

26 found in the Supplemental Investigation Work Plan for the Lower Housatonic River.

1	In addition, GE conducted the following studies in the PSA (unless otherwise noted):
2	 Productivity of robins.
3	 Productivity and density of belted kingfishers (<i>Ceryle alcyon</i>).
4 5	• Analysis of context-dependent effects on early life stages on wood frogs (<i>Rana sylvatica</i>).
6	 Spatial and demographic effects on tree swallows (performed in Canada).
7	 Demographics of short-tailed shrews (Blarina brevicauda).
8	 Field observations of presence/absence of mink.
9	 Evaluation of largemouth bass habitat, population structure, and reproduction.
10	EPA project data are managed using a relational structure in Microsoft Access. The database
11	contains information on PCBs and other COPCs in soil, sediment, and tissue samples, and other
12	field study data collected by EPA and other parties, constituting more than 2 million records.
13	ArcView (geographic information system [GIS]) was used to illustrate spatial patterns. Data
14	originating from previous or concurrent studies conducted by GE and other sources were used if
15	data quality was acceptable. The procedure followed for evaluating data quality of historical

16 studies is described in Appendix C.

17 **1.7 QA/QC**

QA and QC procedures and techniques are established to guide data collection, analysis, modeling, administration, and auditing. Three documents, the *Quality Assurance Project Plan* (WESTON 2001a), the *Supplemental Investigation Work Plan for the Lower Housatonic River* (WESTON 2000), and the *Field Sampling Plan* (WESTON 2001b), outline the QA/QC procedures and techniques used in the studies conducted by EPA in support of this ecological risk assessment. These documents also provide details on the methods used in the collection and analyses of data.

1 1.8 REFERENCES

Barbour, H., T. Simmons, P. Swain, and H. Woolsey. 1998. *Our Irreplaceable Heritage*, *Protecting Biodiversity in Massachusetts*. Massachusetts Heritage and Endangered Species
Program and The Massachusetts Chapter of the Nature Conservancy. 83 pp.

- 5 BBL (Blasland, Bouck & Lee, Inc.). 1994. *MCP Interim Phase II Report and Current* 6 *Assessment Summary for East Street Area 2/U.S. EPA Area 4*. Volumes I, II, III, IV, V, VI, VII, 7 IV V and VII
- 7 IX, X, and XII.
- 8 BBL (Blasland, Bouck & Lee, Inc.). 1995. *MCP Supplemental Phase II Scope of Work and* 9 *Proposal for RCRA Facility Investigation of Unkamet Brook Area/U.S. EPA Area 1.*
- 10 BBL (Blasland, Bouck & Lee, Inc.) and QEA (Quantitive Environmental Analysis, LLC). 2003.
- 11 Housatonic River- Rest of River RCRA Facility Investigation Report Volume 1. Prepared for 12 General Electric Company, January 2003
- 12 General Electric Company. January 2003.
- 13 CR Environmental. 1998. Housatonic River Supplemental Investigation Sub-bottom Profiling,
- 14 *Woods and Rising Ponds.* Prepared for Roy F. Weston, Inc. December 1998.
- 15 EPA (U.S. Environmental Protection Agency). 1991a. The Role of BTAGs in Ecological
- 16 Assessment. Office of Solid Waste and Emergency Response, Office of Emergency and
- 17 Remedial Response, Hazardous Site Evaluation Division (OS-230). *ECO Update*, Intermittent
- 18 Bulletin, Volume 1, Number 1. Publication 9345.0-05I. September 1991.
- 19 EPA (U.S. Environmental Protection Agency). 1991b. Ecological Assessment of Superfund Sites:
- 20 An Overview. Office of Solid Waste and Emergency Response, Office of Emergency and
- 21 Remedial Response, Hazardous Site Evaluation Division (OS-230). ECO Update, Intermittent
- 22 Bulletin, Volume 1, Number 2. Publication 9345.0-05I. December 1991.
- EPA (U.S. Environmental Protection Agency). 1992a. Framework for Ecological Risk
 Assessment. Risk Assessment Forum, Washington, DC. EPA/630R-92/001.
- EPA (U.S. Environmental Protection Agency). 1992b. *The Role of Natural Resource Trustees in the Superfund Process. ECO Update*, Volume 1, Number 3.
- 27 EPA (U.S. Environmental Protection Agency). 1993a. Wildlife Exposure Factors Handbook.
- 28 Volumes I and II. EPA/600/R-93/187a, EPA/600/R-93/187b. U.S. Environmental Protection
- 29 Agency, Office of Research and Development.
- 30 EPA (U.S. Environmental Protection Agency). 1994a. Using Toxicity Tests in Ecological Risk
- 31 Assessment. Office of Solid Waste and Emergency Response, Office of Emergency and
- 32 Remedial Response, Hazardous Site Evaluation Division (OS-230). ECO Update, Intermittent
- 33 Bulletin, Volume 2, Number 1. Publication 9345.0-05I, EPA 540-F-94-012, PB94-963303.
- 34 September 1994.
- EPA (U.S. Environmental Protection Agency). 1994b. *Catalogue of Standard Toxicity Tests for Ecological Risk Assessment*. Office of Solid Waste and Emergency Response, Office of

- 1 Emergency and Remedial Response, Hazardous Site Evaluation Division (5204G). ECO Update,
- 2 Intermittent Bulletin, Volume 2, Number 2. Publication 9345.0-05I, EPA 540-F-94-013, PB94-
- 3 963304. September 1994.

4 EPA (U.S. Environmental Protection Agency). 1994c. *Field Studies for Ecological Risk*5 Assessment. Office of Solid Waste and Emergency Response, Office of Emergency and
6 Remedial Response, Hazardous Site Evaluation Division (5204G). *ECO Update*, Intermittent
7 Bulletin, Volume 2, Number 3. Publication 9345.0-05I. EPA 540-F-94-014, PB94-963305.
8 September 1994.

- 9 EPA (U.S. Environmental Protection Agency). 1994d. Selecting and Using Reference
 10 Information in Superfund Ecological Risk Assessments. Office of Solid Waste and Emergency
 11 Response, Office of Emergency and Remedial Response, Hazardous Site Evaluation Division
 12 (5204G). ECO Update, Intermittent Bulletin, Volume 2, Number 4. Publication 9345.0-10I, EPA
- 13 540-F-94-050, PB94-963319. September 1994.
- EPA (U.S. Environmental Protection Agency). 1994e. *Ecological Risk Assessment Issue Papers*.
 EPA/630/R-94/009. November 1994.
- EPA (U.S. Environmental Protection Agency). 1996. *Ecotox Thresholds*. Office of Solid Waste
 and Emergency Response, Office of Emergency and Remedial Response. *ECO Update*,
 Intermittent Bulletin, Volume 3, Number 2. Publication 9345.0-12FSI, EPA 540/F-95/038,
 PN95-963324. January 1996.
- EPA (U.S. Environmental Protection Agency). 1997a. Ecological Risk Assessment Guidance for
 Superfund: Process for Designing and Conducting Ecological Risk Assessments. Interim Final.
 EPA 540-R-97-006. U.S. Environmental Protection Agency, Environmental Response Team.
- EPA (U.S. Environmental Protection Agency). 1997b. Guiding Principles for Monte Carlo
 Analysis. EPA/63C/R-97/001. March 1997.
- EPA (U.S. Environmental Protection Agency). 1998. *Guidelines for Ecological Risk Assessment*.
 Risk Assessment Forum, Washington, DC. EPA/630/R-95/002F.
- EPA (U.S. Environmental Protection Agency). 1999. Ecological Risk Assessment and Risk
 Management Principles for Superfund Sites.
- 29 EPA (U.S. Environmental Protection Agency). 2001. Risk Assessment Guidance for Superfund
- 30 (RAGS), Volume 3, Part A Process for Conducting Probabilistic Risk Assessment. EPA 540-R-
- 31 02-002. December 2001.
- Harza. 2001. Woods Pond Dam: Structural Integrity Assessment. Prepared for General Electric
 Company, Pittsfield, MA.
- 34 HEC (Harrington Engineering and Construction, Inc.). 1996. Report on the Preliminary
- 35 Investigation of Corrective Measures for Housatonic River and Silver Lake Sediment. Prepared
- 36 for General Electric Company.

- 1 MDEP (Massachusetts Department of Environmental Protection). 1996. Guidance for Disposal
- 2 Site Characterization in Support of the Massachusetts Contingency Plan. Chapter 9, Method 3,
- 3 "Environmental Risk Characterization," Interim Final Policy. MDEP, Bureau of Waste Site
- 4 Cleanup, and Office of Research and Standards. April 1996.
- O'Brien & Gere Engineers, Inc. 1995. *Phase I Report (MCP)/Current Assessment Summary, Hill*78 Area/Area 2.
- 7 Stewart Laboratories, Inc. 1982. *Housatonic River Study 1980 and 1982*. Volumes I and II.
- 8 TechLaw (TechLaw, Inc.). 1998. Preliminary Report: Wetland Characterization and Function-
- 9 Value Assessment, Housatonic River from Newell Street to Woods Pond. 4 May 1998.
- 10 United States of America, State of Connecticut, and Commonwealth of Massachusetts, Plaintiffs
- 11 vs. General Electric Company, Defendant. 1999. Consent Decree-Main Document and
- 12 Appendices A through W. October 1999.
- WESTON (Roy F. Weston, Inc.). 1998. Upper Reach-Housatonic River Ecological Risk
 Assessment. Prepared for U.S. Environmental Protection Agency.
- WESTON (Roy F. Weston, Inc.). 2000. Supplemental Investigation Work Plan for the Lower
 Housatonic River. Prepared for U.S. Army Corps of Engineers and U.S. Environmental
 Protection Agency. 22 February 2000. DCN GEP2-020900-AAME.
- 18 WESTON (Roy F. Weston, Inc.). 2001a. Final Quality Assurance Project Plan, Vol. I Text,
- 19 Vol. II Appendix A, Vol. IIa Appendix A cont'd., Vol. IV Appendix E & F. Prepared for
- 20 U.S Army Corps of Engineers and U.S. Environmental Protection Agency. DCN GE-021601-
- 21 AAHM.
- WESTON (Roy F. Weston, Inc.). 2001b. *Final Field Sampling Plan*. Prepared for U.S. Army
 Corps of Engineers and U.S. Environmental Protection Agency. DCN GE-053001-AAMA.
- 24 WESTON (Weston Solutions, Inc.). 2002. Rest of River Site Investigation Data Report. Prepared
- 25 for U.S. Army Corps of Engineers and U.S. Environmental Protection Agency. DCN GE-
- 26 080202-ABDK.

1 2. PROBLEM FORMULATION

Problem Formulation Highlights							
The problem formulation establishes the goals, scope, and focus of the baseline ecological risk assessment (ERA). It is a process for generating and evaluating preliminary hypotheses about why ecological effects have occurred, or may occur, a a result of human activity. The problem formulation includes discussions of the ollowing topics:							
 Identification and sources of stressors. 							
 Determination of contaminants of potential concern (COPCs). 							
 Fate and transport of contaminant stressors. 							
 Contaminant effects on receptors. 							
 Site conceptual model. 							
 Assessment and measurement endpoints. 							
 Weight-of-evidence (WOE) approach. 							

16 **2.1 OVERVIEW**

Problem formulation, the planning phase of the ecological risk assessment (ERA), plays an important role in the development and direction of the risk assessment. It builds on and refines the screening-level problem formulation, and with input from stakeholders and other parties, shapes the analysis of ecological issues of concern at a site (EPA 1997). Problem formulation results in three products:

- Conceptual model(s).
 - Assessment and measurement endpoints.
 - Analysis plan.

24 25

22

23

This section describes the process that was followed in developing and refining the problemformulation phase of this ERA.

The screening-level problem formulation provides initial guidance to the risk assessors and managers by providing a preliminary look at potential issues of concern (see Figure 1.5-1). The screening-level problem formulation describes: (1) the environmental setting and contaminants known or suspected at the site; (2) contaminant fate and transport mechanisms; (3) ecotoxicity mechanisms and receptor categories of concern; (4) exposure pathways from contaminant
sources to receptors; and (5) the results of a screening of conservative ecotoxicity values.
Subsequently, the problem formulation is expanded and refined as data collection and analysis
proceed. The ERA performed for the Upper Reach of the Housatonic River (WESTON 1998),
with the ERA Work Plan developed by GE during the previous RCRA process (ChemRisk
1997), together fulfill the functional requirements of the screening problem formulation.

7 A detailed ecological characterization of the site was conducted (and subsequently refined) to 8 expand on the information used in the screening-level problem formulation and to refine the 9 initial conceptual model for the site. The final ecological characterization is summarized in Section 2.2 and presented in its entirety as Appendix A. The objective of the ecological 10 11 characterization was to characterize the ecosystems within the Housatonic River watershed, 12 including both plant and animal communities, with a focus on the Primary Study Area (PSA). 13 Table 2.1-1 summarizes the specific ecological characterization surveys performed to 14 characterize the ecosystems potentially at risk, as well as the specific survey objective(s), and 15 references to the SI Work Plan appendix containing the detailed standard operating procedure 16 (SOP) for each survey. This information was then used as input to the problem formulation.

In Section 2.3, the sources, concentrations, and distribution of contaminants in the study area are discussed. Contaminants of potential concern (COPCs) identified in the screening-level problem formulation were re-examined to determine whether they should be retained in the Pre-ERA screen for the ERA (Section 2.4). The availability of new data, information, or changes in assumptions can alter the results of the preliminary screening. Lack of data was not reason alone to eliminate a potential contaminant, rather best professional judgment was used, and discussion regarding the uncertainty surrounding the decision is presented.

24

1 2 3 4

Table 2.1-1

Surveys Conducted for Ecosystem Characterization and Their Specific Objective(s)

Survey	Specific Objective(s)	SI Work Plan Appendix
Rare Plants and Natural Communities	Determine the potential rare, threatened, or endangered plants or animals occurring within the study area.	A.6
	Determine the presence and areal extent of habitats capable of supporting special status species potentially occurring within the study area.	
	Determine the presence and areal extent of exemplary natural communities within the study area.	
Dragonfly	Determine species of dragonflies present in the study area, with particular attention to rare species.	A.7
Mussel	Determine historical and current distribution within and upstream of the study area.	A.8
	Identify potential mussel hosts.	
	Identify wildlife species that prey upon mussels.	
Reptile and Amphibian Use	Estimate amphibian and reptile species richness in the study area by habitat type.	A.9
	Sample larval amphibians in breeding habitats over a range of PCB concentrations.	
	Determine chemical concentrations in herptiles incidentally succumbing during trapping.	
	Note: The latter two objectives were intended for use in ERA exposure and effects characterization (see Section 7.3 of the SI Work Plan).	
Raptors and Waterfowl	Identify raptors and waterfowl breeding in study area.	A.10
Forest Bird and Marsh and Wading Bird	Identify birds using the study area floodplain forests and scrub-shrub habitats.	A.11
	Identify birds using the study area wetland and aquatic habitats.	
River Otter, Mink, and Bat	Determine if mink and otter are present in the study area and reference areas.	A.12
	Identify bat species present in the study area.	
	Determine habitats bats use for feeding.	

1 In the problem formulation, the fate and transport of contaminants in the ecosystem potentially at 2 risk and the description of exposure pathways were expanded beyond those in the screening-3 level problem formulation. This was generally accomplished through the collection of data or 4 other information (e.g., field studies, modeling results, observations) on the fate and transport of 5 contaminants, the ecological setting and flora and fauna of the site, and the extent of 6 contamination (Section 2.5). In addition, the potential effects and impacts associated with 7 contaminants of potential concern (COPCs) are described in the context of site-specific 8 environmental conditions (Section 2.6).

9 The next step in the problem formulation was to establish the assessment endpoints for the study. 10 Assessment endpoints are an "explicit expression of the environmental value that is to be 11 protected" (EPA 1998). Specific assessment endpoints focus the ERA on the issues that are 12 important at the site and identify the appropriate measurement endpoints required to address 13 these endpoints. Potential adverse effects on local populations and communities, such as 14 reproduction, growth, and survival, or changes in community structure or function, respectively, 15 were identified and described using measurement endpoints to quantify effects for the 16 assessment endpoints (Section 2.8). The identification of assessment and measurement 17 endpoints and the exposure pathway analysis were used to refine the conceptual model for the 18 site (Section 2.7). The intent of the conceptual model was, through the iterative process 19 described above, to develop a thorough understanding and description of the site in a systematic 20 and representative manner, and to identify important data or information gaps.

The problem formulation culminates in a scientific/management decision point (SMDP). A SMDP is an agreement between the risk manager and risk assessor on the assessment endpoints selected, exposure pathways, and questions presented in the conceptual model (EPA 1997).

The initial problem formulation for this ERA focused on the area within the 10-year floodplain of the Housatonic River extending from the confluence of the East and West Branches of the river to and including Woods Pond (Figures 1.4-2 and 1.4-3). This area, referred to as the Primary Study Area (PSA), is downstream from the source of COPCs from the GE facility, as well as the area where cleanup activity (including river sediment, bank soil, upland soil, and groundwater) is currently in progress, and includes the river sediment and floodplain soil where a

2-4

majority of the PCBs are located, as indicated by the historical data and the evaluation of the
recent EPA data, and summarized in the RCRA Facility Investigation Report (RFI) (BBL and
QEA 2003). The RFI states that most of the PCB mass in the Housatonic River and floodplain
downstream of the GE facility is in the PSA.

5 In addition, risks are also evaluated for the portion of the river below Woods Pond, MA, to the 6 point of tidal influence below the Derby-Shelton Dam, approximately 13 miles (21 km) from 7 Long Island Sound and 128 miles downstream from the PSA, but using a less quantitative 8 approach than that used for the PSA and for a focused set of endpoints (see Section 2.4, and 9 Appendix A).

10 2.2 PHYSICAL AND ECOLOGICAL CHARACTERIZATION OF THE HOUSATONIC 11 RIVER

12 **2.2.1** Physical Characteristics of the Housatonic River Basin

The Housatonic River is located in Berkshire County, MA, and western Litchfield, eastern Fairfield, and western New Haven Counties, CT. The river flows approximately 166 miles (240 km) from the headwaters above Dalton, MA, to Long Island Sound, and drains an area of approximately 1,950 square miles in Massachusetts, New York, and Connecticut (BBL and QEA 2003).

For much of its path through Berkshire County, MA, the river lies in a wide alluvial plain called the Central Valley (Weatherbee 1996). The Central Valley is bounded to the east by the Berkshire Plateau, a southern extension of Vermont's Green Mountains, and to the west by the Taconic Range, extending from Vermont to New York. In Connecticut, this same alluvial valley is called the Marble Valley. East of the valley, the Berkshire Plateau from Massachusetts continues southward and is called the Litchfield Hills Plateau.

In general, the plateaus and mountains bounding the river valley are typified by rounded hills and mountains draped with glacial deposits, and relatively narrow, steep-sided valleys cut into the hills by streams and rivers. The principal bedrock underlying much of the river basin is marble formed during the Devonian period, approximately 350 to 400 million years ago. Because of the prevalence of marble, the Housatonic River basin exhibits characteristics different from most other river systems in the northeastern United States. In particular, soil and water pH in the
 valley are high (7.9 to 8.3) and the groundwater contains high concentrations of calcium and
 magnesium (Harris 1997; Olcott 1995).

4 The area has a continental climate, similar to the rest of interior New England, characterized by 5 cold winters and hot summers. In Stockbridge, MA, near the northern end of the study area, average annual temperature was 8 °C, and average daily July and January temperatures were 20 6 7 and -6 °C, respectively, for the period between 1951 and 1974. At Cornwall, CT, at 8 approximately the midpoint of the watershed, average annual temperature was 9 °C, and average 9 daily July and January temperatures were 21 and -4 °C, respectively. At Danbury, CT, nearer the 10 southern end of the study area, the average annual temperature was 10 °C, and average daily July 11 and January temperatures were 22 and -3 °C (SCS 1970, 1981, 1988). The number of frost-free 12 days (growing season) at those locations ranges from 103 to 183 days. Moisture supply usually 13 exceeds evaporation, except during periods of drought. Average total rainfall is 43 inches (110 14 cm) in Berkshire County, increases slightly southward to 45 inches (114 cm) in Litchfield 15 County, and 47 inches (119 cm) in Fairfield and New Haven Counties (SCS 1970, 1979, 1981, 16 1988), and is evenly distributed throughout the year. Conversely, average total snowfall for 17 these counties decreases markedly north to south and is 71, 61, 39, and 32 inches (180, 155, 99, 18 and 81 cm), respectively (SCS 1970, 1979, 1981, 1988).

19 2.2.2 Ecological Characterization of the Study Area

20 For the purposes of the EPA Supplemental Investigation, the Housatonic River was divided into 21 17 reaches from the headwaters to Long Island Sound, with some reaches further subdivided. 22 Reaches 1 to 17 were the focus of earlier ecological characterization studies (Chadwick & 23 Associates, Inc. 1994). Reaches 5 and 6, comprising the PSA, were further investigated in detail 24 by EPA from 1998 to 2000 (see Figure 1.1-2). As a result of that work, an ecological 25 characterization of the PSA was prepared (see Appendix A.1, Ecological Characterization of the 26 Housatonic River). Reaches 7 to 17 were also characterized, using aerial photograph 27 interpretation and data provided by regional references and state natural resource agencies (see Appendix A.2, Ecological Characterization of the Housatonic River Downstream of Woods 28 29 Pond).

1 2.2.2.1 Primary Study Area (PSA) Characteristics

2 Much of the PSA (approximately 770 acres) consists of state lands. Portions of the Housatonic 3 River Valley State Wildlife Management Area, which totals 818 acres including land ranging 4 from the confluence of the East and West Branches of the Housatonic River to Woods Pond 5 (Mass Wildlife 2002), fall within the PSA. Approximately two-thirds of the State Wildlife 6 Management Area is a continuous parcel from just north of New Lenox Road south to Woods Pond. Additional large parcels occur near the confluence (approximately 80 acres) and north of 7 8 the Pittsfield wastewater treatment plant (WWTP) (approximately 120 acres). This area includes 9 most of the forested habitat within the PSA. October Mountain State Forest, which comprises 10 approximately 15,940 acres, occurs immediately adjacent to the eastern side of the lower PSA. 11 This large area consists mainly of mature hardwood, softwood, and mixed forests. The City of 12 Pittsfield owns a 45-acre parcel of land associated with the WWTP. Much of the land associated 13 with the WWTP has been developed and includes buildings, paved parking areas, access roads, 14 and maintained lawns. The remaining WWTP land near the river consists of transitional forests, 15 shrub swamps, and shallow emergent marsh. Canoe Meadows, a Massachusetts Audubon 16 Society property, is located just below Holmes Road. This area contains forests and fields, as 17 well as a large wetland complex.

A total of 18 natural communities occur within the PSA: 1 lacustrine community; 10 palustrine communities primarily associated with the Housatonic River floodplain and shoreline; 3 riverine communities either within the channel itself or draining into it; and 4 upland communities included within the 10-year floodplain¹ (Appendix A).

Portions of the PSA have been cleared for various purposes, primarily agriculture, residences, and various rights-of-way (e.g., roads, railroads, power lines). Agricultural development was the primary source of forest clearing within the floodplain. Several large wet meadows can be found in the PSA in which the species composition is influenced by past farming practices. Shrub swamps are common along pool and river channel borders, but are especially frequent as an

¹ Natural communities have been identified and classified according to Swain and Kearsley (2000). Weatherbee (1996) and Weatherbee and Crow (1992) were used to classify some river and lake systems.

intermediate successional stage in areas where pasture is reverting to forested floodplain. Even within some transitional floodplain forests, it is clear from the subcanopy species present that these areas were farmed in the past century. For example, dotted hawthorn routinely colonizes regenerating pastureland, and survives in the subcanopy of floodplain forests for some time after the tree stratum has returned to the site.

6 Significant portions of the PSA are open wetlands and riverine systems dominated by submersed, 7 floating-leaved, and emergent herbaceous vegetation. Riverine point bars and beaches occur 8 occasionally along the river, primarily near bends in the river channel. Mud flats of limited size 9 begin to appear later in the season as the water level declines and exposes previously inundated 10 sediment. Deep emergent marshes, which are usually inundated through the season and 11 vegetated by robust herbs, are frequent along the river channel and backwater edges (Figure 12 2.2-1). These areas become much more abundant south of New Lenox Road, where backwater 13 sloughs, old oxbows, and cut-off channels are common due to the influence of Woods Pond. 14 Shallow emergent marshes, which are areas with saturated soil or shallow water and lower herbs, 15 are less common in the study area and most frequently observed within more permanent vernal 16 pools.

17 2.2.2.2 Housatonic River Below Woods Pond Dam

18 The Housatonic River below Woods Pond Dam in Lenoxdale, MA, extends downstream to Long 19 Island Sound in Connecticut, encompassing Reaches 7 through 17 (see Figures 1.4-5 to 1.4-7). 20 Reach 8 comprises the next significant impoundment below Woods Pond, formed by Rising 21 Pond Dam. Reach 17 is the tidal portion of the river downstream of the Derby-Shelton Dam and 22 was not included in the Rest of River investigations due to its tidal nature and a number of other 23 sources of COPCs. The river valley in Connecticut becomes narrower with steep uplands 24 flanking both sides, and the free-flowing reaches of the river flow over a harder, coarser 25 substrate of limestone, quartz, and granite (HVA 2001). Because of the constricting valley walls, 26 the floodplain becomes narrower than in much of Massachusetts. However, some localized areas 27 of broader floodplain exist. In these areas, agricultural activities are the dominant land use.



The Housatonic River in Connecticut is affected by six dams, all of which create impoundments; five of the dams are used for electric power generation and all the impoundments are used for recreational purposes. These impoundments are medium to large, deep reservoirs of lacustrine habitat. Between these reservoirs, the free-flowing river is characterized as a medium-gradient stream with moderate to fast currents and pool, riffle, and run habitats.

6 A total of 28 natural communities occur in the Lower Housatonic River study area. Aquatic 7 communities include moderately alkaline lakes and ponds in impounded reaches and low-, 8 medium-, and high-gradient stream communities in free-flowing riverine areas. Palustrine 9 communities include deep emergent marshes, shallow emergent marshes, wet meadows, mud 10 flats, riverside seeps, calcareous sloping fens, shrub swamps, red maple swamps, black ash-red 11 maple-tamarack calcareous seepage swamps, transitional floodplain forests, and high-terrace 12 floodplain forests. Within the terrestrial systems, there are riverine point bars and beaches, high-13 energy riverbanks, riverside rock outcrops, calcareous rock cliff communities, northern 14 hardwoods-hemlock-white pine forests, red oak-sugar maple transition forests, spruce-fir-15 northern hardwood forests, successional northern hardwoods, rich mesic forests, and cultural 16 grasslands. Developed land uses include agricultural, residential, commercial, and public 17 development, along with transportation.

18 2.3 IDENTIFICATION AND SOURCES OF STRESSORS

19 2.3.1 Contaminant Stressors

In this section, the sources, concentrations, and distribution of contaminants in the study area are identified. More detailed discussions of these topics, including information regarding the amounts, form, and conditions of release, are presented in the *Modeling Framework Design* (MFD) (WESTON 2003, in preparation), the *Supplemental Investigation Work Plan for the Lower Housatonic River* (WESTON 2000), and the GE *RCRA Facility Investigation Report* (BBL and QEA 2003).

The GE facility in Pittsfield was the major handler of PCBs in western Massachusetts, and is the only known point source of PCBs in the PSA and downstream to the Derby-Shelton Dam (approximately 13 miles from Long Island Sound). According to previous GE reports, from

1932 through 1977, releases of PCBs reached the wastewater and storm systems associated with 1 2 the facility and were subsequently conveyed to the East Branch of the Housatonic River and to 3 Silver Lake, or were released directly to these waters. In addition to the Housatonic River and 4 Silver Lake, areas of the 254-acre GE facility, filled former river oxbows, neighboring 5 commercial properties, Allendale School, and other properties or areas have become 6 contaminated as a result of the GE operations.

7 Based on historical data and facility operations, the contaminants listed below have been found 8 in the source areas and may have migrated to the Housatonic River:

- 9 PCBs.
- 10 Dioxins/furans.
- 11 Semivolatile organics (e.g., bis(2-ethylhexyl)phthalate, methylphenol, phenol, and polycyclic aromatic hydrocarbon [PAHs]). 12
- 13 Volatile organics (e.g., acetone, benzene, chlorobenzene, tetrachloroethene, trichloroethene, toluene, xylene, and other chlorinated hydrocarbons). 14
- 15 Inorganics (e.g., lead and zinc).

16 According to the Source Area Characterization Report, there are five general categories of 17 contaminant sources potentially impacting the river (WESTON 1998):

- 18 Nonaqueous phase liquid (NAPL) discharge. 19
 - Contaminated groundwater discharge.
 - Riverbank soil/river sediment transport.
 - Desorption/adsorption of residual riverbank and sediment contaminants. •
 - Direct stormwater discharge and surface runoff to the river.
- 22 23

20

21

24 The major areas of contamination designated in the Consent Decree for purposes of investigation 25 and response are shown in Figure 1.2-1.

26 2.3.2 Physical and Biological Stressors

27 In addition to contaminant stressors (e.g., PCBs), physical and biological stressors can alter processes within ecosystems, affect habitat types, and ultimately influence natural communities 28 29 by changing the diversity and abundance of species within habitat types. Physical stressors

include structures and events such as dams, ice scour, floods, and droughts; biological stressors consist of changes in the biological components of a community, such as invasive plants outcompeting native plants within riparian areas. Examples of physical and biological stressors that occur within the PSA and their subsequent effects on the natural communities are presented in the following paragraphs.

6 As mentioned in previous sections, the Housatonic River is a low-gradient river with a complex 7 and diverse array of aquatic, riparian, and terrestrial habitats. These habitat types are largely 8 determined by local conditions such as geology, climate, and soil, but are also influenced by a 9 broad network of watershed processes such as hydrology and sediment transport. These 10 processes can alter habitats by changing river morphology (e.g., eroding banks and creating 11 pools) or by resetting high floodplain forest succession (e.g., uprooting overstory trees during a 12 windstorm). Thus, the variety of habitat types within the PSA varies both spatially and 13 temporally.

14 One of the natural watershed processes of the Housatonic River is for the river to meander 15 laterally within its valley by eroding riverbanks on the outside of bends and depositing sediment 16 on the inside of bends to create point bars. This typically occurs during or after high-flow events 17 and can affect various animals within localized natural communities. For example, high flows 18 undermine riverbanks and cause bank collapse, which may result in nest failures of bank-nesting 19 birds such as the belted kingfisher (Ceryle alcyon). High flows also can flood out animals that 20 den in riverbanks, such as muskrats (Ondatra zibethicus), causing mortality or forced relocation 21 of burrows, or flood nests in the floodplain for species such as waterfowl.

Floods also increase river velocities and shear stresses that can cause the riverbed to scour, transport sediment, and then, as high flows subside, deposit sediment downstream and onto the floodplain. Movement of bed sediment can affect aquatic organisms including benthic macroinvertebrates and macrophytes that depend on specific substrate types. In addition, changes in macroinvertebrate communities can stress localized populations of other aquatic organisms that depend on these animals as food.

High winds, such as those that occur during hurricanes or nor'easters, are another physical
stressor that occurs along the Housatonic River. These winds can cause blowdown of localized

areas of the overstory floodplain vegetation (e.g., silver maple [*Acer saccharinum*] and black
 willow [*Salix nigra*]), which then resets forest succession to pioneer herbaceous species such as
 goldenrod (*Solidago* sp.) that can tolerate increases in light and decreases in soil moisture.

4 Blown-down trees and those undermined by bank failure fall into the river and create complex 5 habitat (large woody debris) for a host of aquatic organisms. Large woody debris alters localized 6 channel processes and influences the development of natural communities. When a tree falls 7 into a riffle, it can cause local bed scour and pool formation that then provides areas of refuge 8 during high flows, hiding cover, rearing areas, and food sources for specific aquatic organisms. 9 For example, pools typically have slower velocities and deeper water depths that are used by fish 10 species such as northern pike (Esox lucius), largemouth bass (Micropterus salmoides), and 11 common carp (Cyprinus carpio). Riffles and runs provide relatively faster moving water for 12 different fish species (e.g., longnose dace [*Rhinichthys cataractae*]).

13 Biological stressors within the PSA influence natural communities by changing the distribution 14 of species, which can then affect other components of the food chain. Such stressors include 15 insect infestations, diseases and pathogens, and exotic or invasive species. Invasive plant species 16 are common in the PSA and include species such as Asiatic bittersweet (Celastrus orbiculata), 17 garlic mustard (Alliaria petiolata), common buckthorn (Rhamnus cathartica), Japanese 18 knotweed (*Polygonum cuspidatum*), and purple loosestrife (*Lythrum salicaria*). These plants can 19 invade natural communities and out-compete native plants, create localized monocultures, 20 provide prolific seed source areas, and reduce species diversity. Such invasions can stress 21 species that depend on specific native plants (e.g., some butterflies require specific plants for 22 food).

Both physical and biological stressors within the PSA are influenced by anthropomorphic changes that have occurred within the watershed. These changes include channelization, riverbank armoring, dams, urban runoff, riparian area management, introduction of non-native and exotic species, invasive plants, business and residential development, watershed restoration projects, stormflow routing, bridge and railroad construction, wastewater transport and treatment facilities, agricultural clearing and ditching, and power lines.

1 2.4 OVERVIEW OF PRE-ERA

2 2.4.1 Introduction

The purpose of the Pre-ERA was to narrow the scope of the ERA by refining the list of contaminants to only those that pose potential risks to biota. The primary objectives of the Pre-ERA are as follows:

- Identify COPCs other than PCBs for the PSA. (Downstream of the PSA, numerous potential sources of COPCs, other than the GE facility, exist along the river.)
- 8 2. Determine the downstream boundary beyond which PCBs from the GE facility pose a negligible risk to aquatic biota and wildlife.

The following discussion provides a brief overview of the steps taken to identify COPCs for the PSA and to determine the downstream extent of the ERA. The COPCs from the Pre-ERA are further refined in the discussion of each individual assessment endpoint, resulting in endpointspecific COCs, as appropriate. A more detailed presentation of the Pre-ERA approach and results is provided in Appendix B.

15 2.4.2 Data

Data sets were developed for the primary media of concern for the PSA, background areas, andfor the area below Woods Pond.

18 2.4.2.1 Primary Study Area

19 Data in the PSA were grouped by media (i.e., sediment, surface water, soil, and fish tissue), 20 subreach, and geomorphological type. The subreaches used for this evaluation were 21 hydraulically similar sections of the Housatonic River, identified by the project modeling team in 22 the Modeling Framework Design (WESTON 2003, in preparation). Geomorphological terrain 23 descriptions (geomorph codes) were assigned to sediment, soil, and surface water samples 24 collected by EPA. Each geomorph code represents a depositional or erosional feature or a terrain 25 type that was formed by a specific geologic process (e.g., main channel, vernal pools, and side 26 channels). The sediment and water data categories used for the Pre-ERA are shown in Table 27 2.4-1.

Floodplain and riverbank soils were evaluated separately for Reaches 5A, 5B, 5C, 6A, and 6B.
 Soil adjacent to Woods Pond (referred to as Reaches 6C and 6D in the Pre-ERA) was also
 evaluated. A more detailed description of the reach designations is provided in Section 1.4.

Fish tissue samples were grouped based on reach (5A, 5B, 5C, 6A, and 6B) and size class. Three
size classes were evaluated, small (< 3 inches [7.6 cm]), medium (≥ 3 inches [7.6 cm] but < 12
inches [30.5 cm]), and large (≥ 12 inches [30.5 cm]).

7 2.4.2.2 Background Data

8 Background data are media-specific (i.e., sediment, surface water, soil, and fish tissue) chemistry 9 data collected within the Housatonic River watershed that were not believed to be influenced by 10 contamination directly originating from the GE Pittsfield facility. The objective of the 11 determination of background concentrations was to identify what the media-specific chemical 12 concentrations would be in the absence of releases from the GE facility, and to use this 13 information in evaluating COPCs for the ERA.

Sediment and Surface Water Data Categories

	Geomorphological Terrain Type									
Medium/Reach	Main Channel/ Aggrading Bars	Side Channels and Oxbows (SCOX)	Vernal Pools	Pond						
Sediment										
5A	\checkmark	\checkmark	\checkmark							
5B	\checkmark									
5C	\checkmark	\checkmark	\checkmark							
6A, 6B	\checkmark	\checkmark								
6C, 6D				\checkmark						
Surface Water										
5A	\checkmark	\checkmark								
5B	\checkmark		\checkmark							
5C										
6C, 6D										

4

5 2.4.3 Primary Study Area (PSA) Evaluation and Results

6 The procedures used to screen potential COPCs were applied to the data groupings summarized

7 above. Three progressive evaluation tiers were used to determine COPCs for the PSA.

8 • Tier I – A three-step process was used to establish the initial COPC list evaluating:

- 9 Frequency of detection.
- 10 Exceedance of benchmarks.
- 11 Comparisons to background concentrations.
- Tier II A more detailed evaluation of frequency of exceedance of benchmarks was performed for contaminants that were not eliminated from further consideration in the Tier I evaluation.
- Tier III The spatial extent of contamination, magnitude of benchmark exceedance,
 presence in other media, and mechanism of toxicity were evaluated subjectively for
 contaminants not removed in Tier I or Tier II.

Using the three-tier approach, a final list of COPCs was developed for each medium within a 1 2 reach/terrain. The final generic COPC lists are presented in Tables 2.4-2 through 2.4-5. A 3 detailed description of this approach is provided in Appendix B (Pre-ERA). Although several 4 pesticides were retained as COPCs from the Pre-ERA process, a subsequent review of pesticide 5 concentrations indicated, in general, relatively few detects and low concentrations. Therefore, it 6 is believed that pesticides are generally not a site-related COPC. The COPCs were then further 7 evaluated for each assessment endpoint; this is discussed in detail in the specific assessment 8 endpoint appendices.

9 2.4.4 PCB Screening Evaluation Downstream of Woods Pond and Results

To determine the downstream limit for the ERA and potential areas of concern, the PCB concentrations measured in sediment at locations downstream of Woods Pond to Derby-Shelton Dam were compared with available benchmarks. PCB concentrations in sediment (rather than another medium) were selected as an indicator of the spatial extent of potential ecological risk, because:

- Sediment serves as a reservoir of PCBs released from the GE facility.
- Sediment concentrations generally reflect the relative concentrations that could be expected in the floodplain.
- Exposure to PCBs in sediment is a major route of exposure for lower trophic levels
 (and subsequently higher trophic levels) in the aquatic food chain.
- 20 Relatively extensive data on PCB concentrations in sediment are available.

The conservative benchmark used for this analysis was a threshold effect concentration (TEC) of
0.0598 mg PCB/kg sediment (MacDonald et al. 2000).

Hazard quotients (HQs) were developed using detected PCB concentrations or sample quantitation limits (SQLs), and the MacDonald TEC benchmark (0.0598 mg PCB/kg). After evaluation of the magnitude by which the benchmark was exceeded and the consistency and frequency of exceedances, the reaches from Woods Pond Dam to Derby-Shelton Dam were retained for quantitative evaluation of risk from exposure to PCBs in the ERA.

COPCs for Sediment Based on Tier III Evaluation

	Reach/Geomorphological Type									
		5A			5B			5C		6AB
Chemical	MC & AB	SCOX	VP	MC & AB	SCOX	VP	MC & AB	SCOX	VP	Pond
Semivolatiles										
Dibenzofuran	X									
PAHs										
Acenaphthene	Х						Х			
Acenaphthylene	X						Х			
Anthracene	X						X			
Benzo(a)anthracene	X		Х	Х	Х	Х	Х		Х	
Benzo(b)fluoranthene	X	Х	Х	Х	Х	Х	Х		Х	Х
Benzo(k)fluoranthene	X		Х				Х		Х	
Benzo(g,h,i)perylene	X	Х	Х	Х	Х	Х	Х		Х	Х
Benzo(a)pyrene	X		Х	Х	Х	Х	Х		Х	
Chrysene	X		Х		Х	Х	Х		Х	
Dibenzo(a,h)anthracene	X	Х	Х			Х	Х		Х	
Fluoranthene	X						Х		Х	
Fluorene	X						X			
Indeno(1,2,3-cd)pyrene	X	Х	Х	Х	Х	Х	Х		Х	Х
Naphthalene	X						Х			
Phenanthrene	X			Х	Х		Х		Х	
Pyrene	X			Х	Х	Х	X		Х	

COPCs for Sediment Based on Tier III Evaluation (Continued)

	Reach/Geomorphological Type										
	5A			5B			5C			6AB	
Chemical	MC & AB	SCOX	VP	MC & AB	SCOX	VP	MC & AB	SCOX	VP	Pond	
Dioxins/Furans	Х	Х	Х	X	Х	Х	Х	Х	X	Х	
PCBs	X	Х	Х	X	Х	Х	Х	Х	Х	Х	
Metals											
Antimony							Х		Х		
Barium							Х		X		
Beryllium								Х	Х		
Cadmium							Х		Х		
Chromium							Х		Х	Х	
Copper			Х			Х	Х		Х	Х	
Lead			Х			Х	Х		Х	Х	
Mercury			Х		Х	Х	Х	Х	Х	Х	
Selenium									Х	Х	
Silver			Х		Х	Х	X		X	Х	
Tin						Х	X		X	Х	

6

7

AB – aggrading bars SCOX – side channels and oxbows

VP – vernal pools

5 Pond – Woods Pond

MC – main channel

Note: Reach designations reflect previous reach boundaries; it is assumed that the revised reach designations do not impact reach-specific COPCs determined in Appendix B.

COPCs for Surface Water Based on Tier III Evaluation

	5A		5B	1	5C	6AB	
Chemical	MC & AB	VP	MC & AB	VP	MC & AB	VP	Pond
Dioxins/Furans	Х	Х	Х	Х	Х	Х	Х
PCBs	Х	Х	Х	Х	Х	Х	Х

MC – main channel

AB – aggrading bars

VP – vernal pools

Pond – Woods Pond

Note: Reach designations reflect previous reach boundaries; it is assumed that the revised reach designations do not impact reach-specific COPCs determined in Appendix B.

MK01|O:\20123001.096\ERA_PB\ERA_PB_2.DOC

COPCs for Soil Based on Tier III Evaluation

	Reach/Geomorphological Type									
	5A		5	В	5	6AB				
Chemical	Floodplain	Riverbank	Floodplain	Riverbank	Floodplain	Riverbank	Pond			
Semivolatiles										
Dibenzofuran	Х	X	Х	Х						
PAHs										
Benzo(a)pyrene		X								
Pyrene		X								
Dioxins/Furans	Х	X	Х	Х	Х	Х	Х			
PCBs	Х	X	Х	Х	Х	Х	Х			
Metals										
Chromium	Х	X	Х	Х	Х	Х	Х			
Lead					Х		Х			
Mercury		X	Х	Х	Х	Х	Х			
Selenium					Х		Х			

Note: Reach designations reflect previous reach boundaries; it is assumed that the revised reach designations do not impact reach-specific COPCs determined in
 Appendix B.

6

7

COPCs for Fish Based on Tier III Evaluation

	Reach/Fish Size									
		5A			5BC			6AB		
Chemical	Small	Medium	Large	Small	Medium	Large	Small	Medium	Large	
Pesticides		·						.		
4,4'-DDE						Х		X	Х	
O,p'-DDT	Х	Х	Х	Х	Х	Х	Х	X	Х	
4,4'-DDT		X			Х	X			Х	
Heptachlor epoxide	Х	Х	Х			Х	Х	Х	Х	
Cis-Nonachlor	X	X	Х	Х	Х	Х	Х	X	Х	
Trans-nonachlor	X	Х	Х	X	Х	Х	Х	Х	Х	
Oxychlordane	Х	Х	Х	Х	Х	Х		Х	Х	
Dioxins/Furans	X	Х	Х	X	Х	Х	Х	X	Х	
PCBs	Х	Х	Х	X	Х	Х	Х	Х	Х	

4 5 Note: Reach designations reflect previous reach boundaries; it is assumed that the revised reach designations do not impact reach-specific COPCs determined in Appendix B.

1 2.5 FATE AND TRANSPORT OF CONTAMINANT STRESSORS

2 Understanding the fate and transport characteristics of COPCs is a major component of the 3 problem formulation phase of an ERA. Although other COPCs are present in the study area, the 4 focus of the Rest of River evaluation is on PCBs, as well as dioxins/furans; therefore, the 5 objectives of this discussion are to:

- 6 Provide a general description of PCB fate and transport mechanisms (Section 2.5.1). 7 Present a summary of PCB distribution within the Housatonic River floodplain • 8 (Section 2.5.2). 9 Identify exposure pathways (Section 2.5.3). 10 Discuss how PCB congener patterns change in environmental media (Section 2.5.4). 11 Present a general overview of the fate and transport mechanisms of dioxins/furans 12 (Section 2.5.5).
- 13 **2.5.1** Fate and Transport of PCBs

14 Polychlorinated biphenyls (PCBs) are formed when hydrogen atoms on a biphenyl molecule are 15 replaced by 1 to 10 chlorine atoms. First manufactured approximately 75 years ago, PCBs are 16 extremely persistent contaminants that are now ubiquitous in the global ecosystem (Eisler 1986). 17 There are 209 possible configurations of PCB molecules, based on the number and position of 18 chlorine substitutions on the biphenyl ring; these individual PCB configurations are known as 19 congeners. Although all possible congeners have been synthesized, only approximately 175 of 20 the 209 congeners were included in the various commercial formulations. Groups of PCB 21 congeners with similar numbers of substituted chlorine atoms are referred to as homologs, 22 including: mono-, di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and decachlorobiphenyl 23 (EPA 1996). Aroclors (Aroclor is a trade name of the Monsanto Company) are commercial 24 mixtures of PCB congeners that were formulated to have specific physical properties, which are 25 based, in general, on the overall amount of chlorine substitution (Figure 2.5-1).

The level of chlorination affects various physicochemical properties of the PCB molecule, such as the octanol/water partition coefficient (K_{ow}), solubility, vapor pressure, and Henry's Law constant. These properties affect processes such as volatilization, and partitioning to water, sediment, and floodplain soil. Similarly, the level of chlorination also controls (in part)
 biologically mediated processes such as biotransformation, uptake, and bioaccumulation
 (WESTON 2003, in preparation). In general, more chlorinated PCBs have greater stability and
 environmental persistence (EPA 1996).

5 PCBs in the environment occur as mixtures of congeners that differ in composition from 6 commercial mixtures because of partitioning, contaminant transformation, and preferential 7 bioaccumulation over time (Aulerich et al. 1986; Hornshaw et al. 1983; EPA 1980). Some 8 congeners are retained in sediment, soil, and biological tissue. Bioaccumulated PCBs appear to 9 be more toxic than commercial PCBs (Aulerich et al. 1986; Hornshaw et al. 1983).

More detailed discussions on the fate and transport of PCBs can be found in the *Modeling Framework Design* (WESTON 2003, in preparation).





14

Source: Adapted from Eisler 1986.

Figure 2.5-1 Biphenyl and Representative PCB Congeners

1 2.5.2 PCB Distribution by Media

2 This section provides an overview of the distribution of PCBs in sediment, soil, surface water, 3 and biota of the PSA. This section also presents a discussion of the sediment grain size analysis 4 and the concentrations of organic carbon in the sediment, soil, and water samples from the PSA 5 and their relationship with PCBs. Sediment and soil data used for this analysis included all 6 samples collected by any organization between 1998 and 2002, a span of 5 years. Earlier data 7 were not included to ensure that any potential temporal trends or the influence of different 8 analytical methods in the data would not potentially mask current spatial patterns. The analysis 9 of surface water included samples collected between 1996 and 2002. This slightly longer span of 10 time was used because of the more robust data set that was available. A full presentation of the 11 spatial and temporal trends is presented in the MFD (WESTON 2003, in preparation) and the 12 RFI report (BBL and OEA 2003).

The term *sediment* is defined for this study as solid material typically inundated under normal hydrologic conditions. Soil samples are defined as those samples collected from areas not typically inundated under normal hydrologic conditions. Sediment and soil samples were collected from across the PSA and classified by the geomorphic terrains (i.e., main river channel, floodplain, riverbanks, etc.) from which they were originally collected. The distribution of PCBs in the PSA and in Reaches 7, 8, and 9 between the PSA and the Massachusetts/Connecticut state line is illustrated in the stack bar figures (see Attachment 2.1).

20 2.5.2.1 Sediment

21 PCBs have been detected in sediment samples collected from all reaches of the Housatonic River 22 from just upstream of the GE facility through the PSA and downstream in Massachusetts and in 23 Connecticut. Figure 2.5-2 presents sediment PCB data for the entire Housatonic River (from the 24 vicinity of the GE facility to the point where the river empties into Long Island Sound). 25 Historically, over 7,500 sediment samples have been collected from the main channel of the river 26 in Massachusetts and Connecticut; almost 5,000 samples since 1998 alone. The highest 27 concentrations of PCBs have been detected in sediment adjacent to the GE facility (river mile 28 137; 9,411 mg/kg in a surficial sample) and continuing downstream to Woods Pond Dam (at 29 river mile 124.37). Within the PSA, the highest PCB concentrations detected by EPA were 614
mg/kg in Reach 5A, 165 mg/kg in Reach 5B, 213 mg/kg in Reach 5C, and 668 mg/kg in Reach 6
(Woods Pond). Sediment samples collected prior to 1998 detected PCBs as high as 2,270 mg/kg
in Reach 5A. PCBs have also been detected as deep as 6 to 8 feet below the riverbed surface
throughout Reaches 5 and 6 (BBL and QEA 2003; WESTON 2003, in preparation).

PCB concentrations in sediment decrease downstream of Woods Pond Dam in Reaches 7, 8, and
9, and decrease further in concentration through most of Connecticut (BBL and QEA 2003). An
increase in PCBs was detected in the most downstream reach (Reach 17 – from the DerbyShelton Dam to Long Island Sound), attributable to other Superfund or designated hazardous
waste sites located within that portion of the river.



10 11 Notes:

12

13

14

15

16

17 18

- 1. All data are plotted at the approximate mid-point of each reach, and represent samples collected from within the top 3 feet of the riverbed.
- 2. Total PCB concentrations above 400 mg/kg were not plotted.
- Symbols represent significant features/names of reach boundaries: GE = General Electric facility; WPD = Woods Pond Dam; RPD = Rising Pond Dam; GFD = Great Falls Dam; CB = Cornwall Bridge; BBD = Bulls Bridge Dam; BD = Bleachery Dam; SD = Shepaug Dam; STD = Stevenson Dam; DSD = Derby-Shelton Dam.

19Figure 2.5-2 Distribution of tPCB Concentrations Detected in Sediment Samples20from the GE Facility to Long Island Sound

The mean tPCB concentrations in sediment samples are plotted by reach in Figure 2.5-3. The data have been presented on a log scale to capture the mean tPCB concentration of 393 mg/kg in Reach 3 (adjacent to the GE facility). For the purposes of calculating statistics, values for nondetects were treated as half the reported detection limit. Likewise, the most commonly reported detection limit of 0.5 mg/kg is shown on the figure for comparison.





6 7 8

9 As shown in Figure 2.5-2, the mean tPCB concentrations are highest adjacent to the GE facility 10 and on through the PSA to Woods Pond Dam (Reaches 3 to 6) and then generally decrease 11 through the remaining reaches in Massachusetts and Connecticut. Many samples from 12 Connecticut were non-detect, resulting in low (<0.5 mg/kg) mean concentrations of PCBs. 13 Reach 11, and approximately half of the length of Reach 10, is shallow and fast-flowing with 14 mostly a gravel to cobble stream bed where PCB-containing solids are not likely to be deposited, 15 resulting in very few samples having PCBs. Reach 16 represents the last impoundment along the Housatonic River, and approximately 99% of the samples collected there were non-detect. 16

1 2.5.2.2 Soil

2 Soil samples were collected from the floodplain and riverbanks along the Housatonic River 3 within Massachusetts) (approximately 4,300 samples were collected by EPA, and 3,300 samples 4 were collected by GE) in the reaches upstream of Woods Pond Dam. Additional samples 5 (approximately 1,600 collected by EPA, and 200 collected by GE) were collected below the 6 PSA, and PCBs have been detected in floodplain soil in all reaches of the Housatonic River from 7 the GE facility to the Massachusetts/Connecticut state line. The highest concentration of tPCBs 8 detected in floodplain soil was 907 mg/kg from soil in Reach 5C above Woods Pond. 9 Conversely, the highest tPCBs detected in riverbank soil were adjacent to the GE facility and just 10 downstream, in Reaches 2 through 4 (between river miles 138 and 135).

11 Figure 2.5-4 presents the spatial distribution of the mean and median tPCB concentrations for all surficial (0 to 6 inches [0 to15 cm]) floodplain soil by reach for the portion of the river upstream 12 13 of the Massachusetts/Connecticut state line, at which point the average PCB concentration in 14 floodplain soil is less than 1 mg/kg. In addition, little floodplain exists within the Connecticut 15 portion of the river; therefore, no samples were collected from those reaches. Mean tPCB concentrations are broadly similar within Reaches 4, 5, and 6, averaging slightly more than 15 16 17 mg/kg, and then on average decreasing by an order of magnitude in Reaches 7, 8, and 9. However, localized areas of higher concentrations are found in Reach 7. 18







Figure 2.5-4 Mean Total Surficial Soil PCB Concentrations at Floodplain Locations by Reach

1Figure 2.5-5 Spatially Weighted tPCB Concentrations in Floodplain Soil in the2Primary Study Area (Tile 1 of 7)



3

1Figure 2.5-6 Spatially Weighted tPCB Concentrations in Floodplain Soil in the2Primary Study Area (Tile 2 of 7)



MK01|O:\20123001.096\ERA_PB\ERA_PB_2.DOC

3

1Figure 2.5-7 Spatially Weighted tPCB Concentrations in Floodplain Soil in the2Primary Study Area (Tile 3 of 7)



3

1Figure 2.5-8Spatially Weighted tPCB Concentrations in Floodplain Soil in the2Primary Study Area (Tile 4 of 7)



3

1Figure 2.5-9Spatially Weighted tPCB Concentrations in Floodplain Soil in the2Primary Study Area (Tile 5 of 7)



3

1Figure 2.5-10Spatially Weighted tPCB Concentrations in Floodplain Soil in the2Primary Study Area (Tile 6 of 7)



Figure 2.5-11 Spatially Weighted tPCB Concentrations in Floodplain Soil in the Primary Study Area (Tile 7 of 7) 3



4

Figures 2.5-5 through 2.5-11 display the spatially weighted floodplain tPCB concentrations in
 the PSA using the inverse distance weighting procedures described in Appendix C.3.

Riverbank soil PCB concentrations are broadly similar in concentration ranges to the floodplain
soil and river sediment samples, being highest adjacent to the GE facility in Reach 3 and
immediately downstream in Reach 4, and decreasing in concentration within Reaches 6 and 7.

6 2.5.2.3 Surface Water

Sampling for PCBs in surface water was conducted during both low flow conditions and during higher or storm flow conditions. During lower flow conditions, PCB-contaminated sediment act as the primary source of PCBs in the water column through the processes of diffusion and groundwater advection through the sediment and associated porewater. During higher flows, the principal source of PCBs in the water column is from resuspended sediment, from both upstream and within the PSA.

Results for all of the surface water samples collected and analyzed for tPCBs since 1980 are presented in Figure 2.5-12 by river mile. In addition, Figures 2.5-12 and 2.5-13 identify the locations of major impoundment structures found along the Housatonic River from the GE facility to Long Island Sound.

- GE = General Electric facility.
- 18 WPD = Woods Pond Dam.
- 19 RPD = Rising Pond Dam.
- GF = Great Falls Dam.
- CB = Cornwall Bridge.
 - BBD = Bulls Bridge Dam.
 - RDD = Bleachery Dam.
 - SD (River Mile 25) = Shepaug Dam.
 - SD (River Mile 15) = Stevenson Dam.
 - DSD = Derby-Shelton Dam.
- 26 27

22

23

24

25

While the analysis of spatial patterns discussed below only used data from 1996 to 2002, all historical data were plotted to show the full set of results, because most of the data downstream of Woods Pond, especially in Connecticut, were collected prior to 1996. Figure 2.5-13 presents



Figure 2.5-12 Total PCB Concentrations Measured in all Surface Water Samples Collected from the Housatonic River Since 1980



1 2

3



F

5 6



MK01|O:\20123001.096\ERA_PB\ERA_PB_2.DOC

only the surface water data collected since 1996. As indicated in this figure, tPCB 1 2 concentrations increase at the GE facility and then decrease downstream through to Rising Pond 3 Dam. Concentrations of tPCBs continue to decrease by an order of magnitude downstream of Rising Pond Dam and into the Connecticut portion of the river. Most (approximately 80%) of 4 5 the samples collected in Connecticut, both before and since 1996, were non-detect. Within 6 Reaches 5 and 6, PCBs were detected at all surface water sampling locations and were fairly 7 constant in concentrations across the study area. More than half of the samples collected from 8 Reaches 5 and 6 contained tPCBs above the chronic ambient water quality criterion (cAWQC) 9 for protection of aquatic life of 0.014 μ g/L.

10 2.5.2.4 Biota

Biological tissue sampling was conducted to support both the human health and ecological risk assessments and modeling study. In general, most tissue samples collected were analyzed for tPCBs and PCB congeners, dioxins/furans, and organic carbon (OC) pesticides. Figures 2.5-14 and 2.5-15 present the distribution of tPCB concentrations for a majority of the biota used to evaluate PCB exposure in the baseline ERA.





Figure 2.5-14 Total PCB Concentration (mg/kg wet weight) in Selected Biota (Excluding Fish) for Reaches 5 and 6



Sediment-associated invertebrates have significant exposures to PCBs because they remain in
 continuous contact with the sediment bed, which contains relatively high PCB concentrations.
 Water column invertebrates also accumulate PCBs, either through respiration of PCBs in the
 water column, or by ingestion of contaminated suspended particulate matter. Overall, food
 ingestion is the dominant pathway of PCB uptake for aquatic organisms in the Housatonic River.

6 Fish species exhibit interspecies variation in PCB concentrations. This partly reflects the 7 differences in PCB concentrations in the abiotic media to which the fish are exposed. For 8 example, forage fish tend to have lower PCB body burdens compared to benthic fish, which are 9 in contact with contaminated sediment and porewater. However, the main reason for the 10 interspecies differences is not direct contact with PCB-contaminated media, but rather 11 differences in the dietary uptake patterns. Biomagnification in the food web also is a major 12 factor controlling the PCB concentrations in fish. Biomagnification represents trophic-level 13 differences in PCB concentrations and is measured as the increase in lipid-based contaminant 14 concentrations in predators over those in prey (Russell et al. 1999). The mean tPCB and lipid-15 normalized tPCB concentrations in whole fish, by reach, are presented in Figures 2.5-16 and 2.5-16 17.



Figure 2.5-16 EPA Fish Collections (1998-2000) - Median tPCB Concentrations All Ages by Subreach in the PSA



Figure 2.5-17 EPA Fish Collections (1998-2000) - Median Lipid Normalized PCB Concentrations - All Ages by Subreach in the PSA

5 Organism foraging behavior plays a substantial role in the bioaccumulation of PCBs. Species 6 that remain in proximity to the areas of higher PCB concentrations (e.g., main channel sediment) 7 have increased exposure relative to those that use habitats such as distal floodplains or 8 woodlands. Some species (e.g., wood frogs) have high exposures during specific life history 9 stages but may migrate to less-contaminated habitats as adults. Other organisms (e.g., ducks, 10 large raptors) may have exposures to highly contaminated prey as both juveniles and adults, but 11 effectively "dilute" their exposures due to their large home ranges and/or seasonal residency in 12 the Housatonic River watershed.

13 2.5.4 Changes in PCB Congener Patterns

Because PCBs constitute a group of contaminants rather than a single contaminant, their fate in the environment is complex. Some congeners are subject to degradation to a greater extent than others, with the transformation of those congeners and the potential creation of, or enhancement of, other congeners. In addition, congeners have different rates and extent of exchange in different media, resulting in differential rates and patterns of transport.

1 2

3

1 In the Housatonic River, the predominant congeners are the highly chlorinated congeners 2 associated with the release of Aroclor 1260, and to a lesser degree, Aroclor 1254. The more 3 highly chlorinated congeners are more resistant to degradation. A number of studies have shown 4 that under laboratory conditions, PCB congeners in sediment samples from the Housatonic River 5 can degrade to varying degrees, with the losses of some congeners and increases in the 6 degradation product congeners (Bedard and May 1996). However, the congener data collected 7 from the river sediment and floodplain soil do not support degradation as a major removal 8 process.

9 During the release and transport of PCBs in the river, the level of chlorination of the congeners 10 controls, in part, the distribution and exchange of the congeners among the solid and liquid 11 phases. Increasing the degree of chlorination decreases the solubility of the congener and 12 increases its tendency to sorb to solid phases. As a result, surface water samples have congener distributions that have a higher percentage of the lower-chlorinated congeners compared to the 13 14 congeners measured on the particulate matter in the same sample. Similarly, the less chlorinated 15 congeners are present at a higher percentage in porewater than those found in the sediment from 16 which the water is extracted. The effect of this partitioning phenomenon is that PCBs tend to fractionate during transport and over time, with the loss of less-chlorinated congeners and the 17 18 retention of more highly chlorinated ones. In the Housatonic River, however, the PCBs 19 discharged from the facility were dominated by the more highly chlorinated congeners, primarily 20 those associated with Aroclor 1260. As a result, only limited changes in the congener 21 distribution are observed from differential congener transport.

In 2001, EPA and GE conducted a joint sampling effort to investigate site-specific PCB partitioning behavior in the Housatonic River. The program entailed synoptic collection of sediment and porewater, and in a complementary effort, synoptically in surface water and suspended solids. The synoptic nature of the collections and analyses allowed a detailed assessment of partitioning behavior and an assessment of shifts in congener distributions among media. Findings from the study include:

28 29

30

• The analyses of approximately 50 paired bulk sediment/porewater samples indicate a shift in the PCB homolog profiles between media. In bulk sediment, the homolog profile averaged 5.9 chlorines per biphenyl (Cl/BP), with hepta-PCBs having the

largest contribution to tPCB mass. In contrast, synoptic porewater samples had an average of 5.3 Cl/BP, with hexa-PCBs having the largest contribution to tPCB mass. This pattern reflects the congener-dependent partitioning behavior described above.

- 4 Spatial trends in chlorination level (which may be used as a surrogate for alterations 5 in congener distributions related to chemical properties) were evaluated in sediment 6 and porewater. No trend with distance downstream was observed in porewater. A 7 modest reduction in chlorination level was observed in bulk sediment, however. 8 Typically, the majority of Cl/BP ranged from 6 to 6.5 for samples collected within 9 Reach 5A of the PSA, whereas downstream samples (Reaches 5B and 5C) usually 10 had 5.5 to 6 Cl/BP. This confirms that changes in the congener distribution with distance from the source are possible, but are limited because of the highly 11 chlorinated nature of PCBs in the source media. 12
- Surface water particulate matter in samples collected from four locations (Pomeroy Avenue, West Branch, New Lenox Road, Woods Pond) exhibited congener/homolog distributions comparable to bulk sediment. The particulate organic matter yielded an average of 6.0 Cl/BP, compared to 5.9 Cl/BP for bulk sediment.
 - Surface water samples from the same four locations yielded dissolved PCB profiles (4.7 Cl/BP) that were slightly "lighter" than porewater samples (5.3 Cl/BP), primarily because of an increased percentage of tri-PCBs.

These findings support the conceptual model that PCB congener distributions will differ in aqueous and particulate media, primarily because of contaminant properties that favor partitioning to solids (and reduced solubility) for higher chlorinated congeners. Some spatial variation in the partitioning behavior for sediment is apparent, but does not dominate the kinetics. Therefore, it appears that physical transport of PCBs (via bedload and suspended particulate matter at higher flows, and diffusive flux at lower flows) is the dominant fate process, with dechlorination of PCB mixtures a relatively minor process.

27 A more extensive evaluation of congener patterns in sediment, soil, and tissue, using multivariate 28 classification analysis (Euclidean distance) and principal components analysis (PCA), was 29 performed. These analyses included a broad range of media, including floodplain soil, bank soil, 30 bed sediment, suspended sediment, and tissue (e.g., bullfrogs, fish, tree swallows, crayfish, and 31 ducks). The analyses were conducted to investigate the similarity of congener patterns within 32 and among groups of samples for the purpose of measuring the differences between groups and 33 the level of consistency within groups. Overall, the congener evaluation (Appendix C.7) 34 indicated that differences in profiles are sometimes evident, but that most media exhibit congener

1

2

3

17

profiles similar to Aroclor 1260 across all reaches. Some changes in congener profiles were
 observed both spatially and across media, with the latter differences larger than the former.

3 2.5.5 Fate and Transport of Dioxins/Furans

4 The following discussion presents an overview of the general fate and transport mechanisms
5 associated with dioxins/furans that were retained as COPCs in all media (see Section 2.4).

6 2.5.5.1 Transport and Partitioning

Dioxins and furans, similar to PCBs, are characterized by low solubility, low vapor pressure, and high affinity for organic carbon (log K_{oc} values as high as 7.39), which suggests that they will strongly adsorb to sediment or soil and that their vertical movement in either medium will be limited. The leaching of dioxins and furans is unlikely if water is the only transporting medium; however, saturation of sorbed sites and the presence of organic solvents or petroleum may result in vertical migration in sediment or soil.

Volatilization from soil during warm months may also be a major partitioning mechanism. In general, the higher the degree of chlorination, the lower the relative degree of volatilization from soil or water.

16 In the atmosphere, dioxins and furans are typically adsorbed to particulates with the vapor-phase 17 tending to be negligible (Paustenbach et al. 1991). Vapor pressure and ambient temperature are 18 the two environmental factors controlling the phase of congeners in the atmosphere. Congeners having a vapor pressure greater than 10^{-4} mm Hg will exist primarily in the vapor phase. Dioxins 19 20 and furans have relatively long residence times in the atmosphere and are removed by wet, dry, 21 and gas-phase (vapor phase onto plant surface) deposition (ATSDR 1998). Contamination of 22 plant foliage via atmospheric deposition is the primary mechanism of accumulation in terrestrial 23 plants.

Dioxin and furan adsorption to particulates in the water column increases with increasing chlorination. Dioxins and furans are removed from the water column primarily by binding with particulates, sediment, or biota and to a lesser extent by volatilization (Paustenbach et al. 1992). Resuspension of sediment-bound dioxins and furans can increase their transport and availability for uptake by aquatic biota. The primary route of exposure to dioxins and furans for lower trophic-level organisms is uptake from water. Bioaccumulation appears to increase with increasing chlorination up to T(tetra)CDDs and TCDFs. For higher trophic-level organisms, the predominant route of exposure is via food chain transfer.

5 2.5.5.2 Transformation and Degradation

6 Photolysis of dioxins and furans in sediment or soil is a relatively slow process when compared 7 with aquatic photolysis rates. However, the addition of organic solvents to contaminated 8 sediment or soil can enhance photolytic transformation. Field and laboratory studies have shown 9 that several microorganisms (e.g., fungi and bacteria) are capable of degrading different 10 congeners. In general, the rate of biodegradation decreases with increasing chlorination.

In the atmosphere, dioxin and furan reactions with hydroxyl radicals appear to be the most significant source of transformation. Vapor-phase dioxins and furans may also undergo photolytic degradation. The estimated half-life for TCDD reactions with hydroxyl radicals is 2 to 8 days, and the estimated photolytic lifetime ranges from 1 to 7 days (ATSDR 1998). OCDDs and OCDFs, with their low vapor pressure, partition to the particulate phase. Atmospheric photodegradation of these highly chlorinated congeners is less likely.

17 Dioxins and furans in aquatic environments are primarily associated with particulate matter. 18 Photodegradation of bound dioxins and furans occurs near the water's surface and decreases with 19 water depth. Biodegradation in the water column does not appear to be a significant 20 transformation mechanism. Limited biodegradation of dioxins and furans has been observed in 21 sediment, with degradation rate decreasing with increasing chlorination.

22 2.6 EFFECTS ON RECEPTORS

There are a number of chemical stressors that may have an adverse impact upon organisms found in the Housatonic River PSA. The preliminary ecological risk assessment (Pre-ERA) identified 24 COPCs that are of interest (Appendix B, Section 2.3). A short review of toxicity 26 mechanisms and the possible effects to aquatic and terrestrial organisms follows for PCBs and dioxins/furans. A more detailed, receptor-specific review of COC toxicity is presented in each of
 the assessment endpoint appendices (Appendices D through K).

3 2.6.1 Polychlorinated Biphenyls (PCBs)

The toxicology of PCBs varies considerably among congeners, depending on the number and location of chlorines on the biphenyl molecule, and also between animal species due to differences in absorption, metabolism, mechanism of action, and potential toxic effects (Eisler and Belisle 1996).

8 PCB congeners vary in toxicity in many ways, including mode of action, potency, and potential 9 for interaction. PCB congeners may interact with each other and with other chemicals when 10 combined in a complex commercial PCB mixture. Lethal and sublethal effects of PCBs on 11 mammals, birds, and aquatic life, are discussed in detail in the appropriate assessment endpoint 12 appendices; a general summary of PCB-associated effects is presented in Table 2.6-1. The 13 following discussion of PCB toxicology focuses primarily on the general mechanisms of PCB 14 toxicity.

15 PCB congeners differ in their biological activities, and different animal species vary in their sensitivity to the individual congeners. Multiple and diverse mechanisms are involved in the 16 17 toxicological responses of animals to PCB exposures. The mechanism of Ah-receptor binding is 18 an initial step in producing toxic effects, and is the basis for the World Health Organization's 19 (WHO) toxic equivalency factors (TEFs) approach for ranking the relative potency of PCBs, 20 PCDDs, and PCDFs (Van den Berg et al. 1998). The WHO TEFs only apply to Ah-receptor-21 mediated biochemical responses and toxic effects. The relationship between PCB molecular 22 structure and the potential for toxic effects independent of Ah-receptor mediation is not clearly 23 understood. Research through the 1990s found increasing evidence for alternative mechanisms 24 for several PCB-induced effects such as neurotoxicity and disruption of neutrofil function 25 independent of Ah-receptor mediation (ATSDR 2000). In addition, there is a third category, 26 where PCB toxicity may be initiated by both Ah-receptor-dependent and independent 27 mechanisms.

2-46

Table 2.6-1

Common Effects of PCB Exposure Observed in Various Animals

System Affected	Specific Effect		
Hepatic effects	 Hepatomegaly, bile duct hyperplasia Widespread (e.g., rabbit) or focal (e.g., mouse) necrosis Lipid accumulation, fatty degeneration Induction of microsomal monooxygenases and other enzymes Decreased activity of membrane ATPases Depletion of fat-soluble vitamins Porphyria 		
Gastrointestinal effects	 Hyperplasia and hypertrophy of gastric mucosa Gastric ulceration and necrosis Proliferation and invasion of intestinal mucosa (monkey) Hyperplasia, hemorrhage, necrosis (hamster, cow) 		
Respiratory system	Chronic bronchitis, chronic cough		
Nervous system	 Alterations in catecholamine levels Impaired behavioral responses Developmental deficits Depressed spontaneous motor activity Numbness in extremities 		
Skin	ChloracneEdema, alopecia		
Immunotoxicity	 Lymphoid involution (spleen, lymph nodes, especially thymus) Subsequent reduction of circulating lymphocytes Suppressed antibody responses Enhanced susceptibility to viruses Suppression of natural killer cells 		
Endocrine system	 Altered levels of circulating steroids Estrogenic, antiestrogenic, antiandrogenic effects Decreased levels of plasma progesterone Adrenocortical hyperplasia Thyroid pathology, changes in circulating thyroid hormones 		
Reproduction	Increased length of estrusDecreased libidoEmbryo and fetal effects following in utero exposure		
Carcinogenesis	PromoterAttenuation of some carcinogens		

1 2

3

Source: Hansen 1994

PCBs are able to induce hepatic Phase I enzymes (CYP oxygenases) and Phase II enzymes (e.g., UDP glucuronyltransferases, epoxide hydrolase, glutathione transferase). Most commercial PCB mixtures induce both 3-methylcholanthrene type (CYP1A1 and 1A2) and phenobarbital-type (CYP2B1, 2B2, and 3A) CYPs. Non-ortho and mono-ortho PCBs can assume a coplanar molecular configuration and bind to the Ah receptor causing CYP1A1/1A2 induction in rodents (Safe 1994). Effects from PCBs involving the Ah-receptor-initiated mechanisms include body weight wasting, thymic atrophy, porphyria, and porphyria cutanea tardea (Safe 1994).

8 There are many examples of the complexity of the relationship between PCB molecular structure 9 and toxic effects independent of Ah-receptor initiation. For example, some PCBs with two ortho 10 chlorines and lateral chlorines induce both types of CYPs but demonstrate little Ah-receptor 11 affinity. Di-ortho PCBs with one or two para chlorines predominantly induce CYP2B1/2B2/3A 12 and have no affinity for the Ah receptor (Connor et al. 1995). The induction of phenobarbital-13 type CYPs by PCBs is independent of the Ah receptor. PCBs with at least two ortho chlorines 14 and one or two para chlorines are the most potent CYP inducers.

Neurological and neurodevelopmental effects involving changes in brain dopamine levels are PCB-induced effects that are Ah-receptor independent. Scientists have hypothesized that the effect on dopamine levels is related to decreased dopamine synthesis by PCB inhibition of certain enzymes or decreased dopamine uptake into vesicles (ATSDR 2000). It is also possible that a connection exists between disruption of Ca^{2+} homeostatic mechanisms and neurological and neurodevelopmental effects. It is clear that Ah-receptor-independent mechanisms are important in the induction of neurotoxic effects by PCBs.

In vitro studies have indicated that PCBs can induce functional changes such as degranulation in neutrofils (ATSDR 2000). These functional changes may be related to PCB toxicity such as immunological effects and tissue damage. Immunological effects that involve neutrofils include defenses against pathogens and inflammatory responses leading to tissue injury.

There are a number of effects that involve both Ah-receptor-dependent and -independent mechanisms. These include liver hypertrophy, neurodevelopmental, or reproduction effects involving changes in steroid hormone homeostasis and/or thyroid hormone disruption, immunological effects, and cancer (Safe 1994; ATSDR 2000). Safe (1994) reviewed numerous studies of PCB-induced hepatoxicity in mammals including rats,
 mice, rabbits, guinea pigs, monkeys, and mink exposed to Aroclors including 1221, 1242, 1248,
 1254, and 1260. From these studies, it appears that PCB-induced liver toxicity is mediated by
 both Ah-receptor-dependent and -independent mechanisms.

5 Reproductive impairment following PCB exposure has been observed in mink, one of the most 6 sensitive mammals to PCB toxicity (Eisler and Belisle 1996; Moore et al. 1999). Although 7 congeners with high Ah-receptor affinity are more potent than congeners with low Ah-receptor 8 affinity, there is evidence that Ah-receptor-independent mechanisms may be involved.

9 Review of the scientific literature indicates that animals exposed to PCBs have an increased risk 10 of cancer. Lifetime oral exposures to a number of commercial PCB mixtures (Aroclors 1016, 1242, 1254, and 1260) have produced liver tumors in female rats and Aroclor 1260 has produced 12 liver tumors in male rats. Mixtures with high chlorine content such as Aroclor 1254 were 13 generally more potent than mixtures with low chlorine content such as Aroclor 1016 (Mayes et 14 al. 1998).

15 **2.6.2 Dioxins/Furans**

16 Many halogenated aromatic compounds have been described as exhibiting dioxin-like behavior, 17 such as polychlorinated dibenzofurans (PCDFs) and some coplanar polychlorinated biphenyls 18 (PCBs), based on similarities in toxicity and mechanism of action. The primary toxic 19 mechanism of action is binding of the PCDD, PCDF, or coplanar PCB compound to the Ah 20 receptor (described in the previous section). Because 2,3,7,8-TCDD binds to the Ah receptor 21 with a high affinity and has a high toxic potency, it has been the focus of experimental toxicity 22 studies. EPA, regulatory agencies in other countries, and international organizations such as 23 WHO use a TEF approach to reflect the varied toxicity of the different PCDDs, PCDFs, and 24 PCBs.

The impact of dioxins in the environment is directly related to their highly lipophilic and hydrophobic nature as well as to the toxic effects of these compounds on plants and animals. These toxic effects have been extensively studied in the laboratory and through evaluation of animals exposed to dioxins in the environment. The toxicology of PCDD/PCDF varies considerably between congeners and between animal species in absorption, metabolism,
 mechanism of action, and potency of toxic and carcinogenic effects. The following discussion of
 PCDD and PCDF toxicology focuses primarily on the general mechanisms of toxicity.

4 2,3,7,8-TCDD and equivalents share a mechanism of toxicity that initially involves binding of 5 the individual congener to the cystolic Ah receptor in all animal species. After initial binding, 6 the ligand-receptor complex is translocated to the nucleus of the cell. It then becomes associated 7 with the DNA and causes transcription of one or more contaminated genes (EPA 1993). The 8 physiological effects that follow are species-specific but there are many similarities, including 9 the induction of enzyme systems such as cytochrome P4501A1, "wasting syndrome," decreased 10 immunocompetence, reproductive effects, edema, and mortality. The Ah receptor is present in 11 all mammalian and bird species that have been tested, as well as in many species of fish. It is 12 unclear whether the Ah receptor is present in amphibians and reptiles.

A protein similar to the Ah receptor has been identified in terrestrial invertebrates, but there is no
evidence to support the existence of an Ah-receptor type protein in aquatic invertebrates (EPA
15 1993).

16 2,3,7,8-TCDD toxicity and the toxicity of the other 74 individual PCDD congeners is mediated 17 by the Ah receptor. Differences between species in sensitivity to 2,3,7,8-TCDD may be related 18 to the size and binding efficiency of the Ah receptor, pharmacokinetic differences between 19 species, and additional contributing factors. Ah-receptor affinity is determined by the chlorine 20 substitution pattern of the individual dioxin congener. 2,3,7,8-TCDD is substituted in all four 21 lateral positions and has the highest affinity for the Ah receptor. Less active congeners have an 22 additional one, two, or four nonlateral chlorine substituents or have lateral chlorine substituents 23 removed. 2,3,7,8-TCDD and structurally related halogenated aromatic compounds induce 24 microsomal hepatic enzymes such as hepatic aryl hydrocarbon hydroxylase (AHH) and 25 ethoxyresorufin-O-deethylase (EROD). Both AHH and EROD are markers of CYP1A1 activity. 26 Increased synthesis of cytochrome P4501A1 (CYP1A1) is induced by an individual dioxin 27 congener binding to the Ah receptor. CYP1A1 functions in the detoxification or activation of 28 endogenous and exogenous chemicals. Cytochrome P4501A2 (CYP1A2) is only induced in 29 hepatic tissue and has a similar function to CYP1A1.

2-50

Effects observed in the offspring of animals exposed to 2,3,7,8-TCDD include fetal/newborn mortality, structural malformations, impaired development of the reproductive system, neurodevelopmental effects, immunotoxicity, and thymic atrophy. Impaired development of the reproductive system and neurobehavioral effects in the developing organism are the most sensitive endpoints of 2,3,7,8-TCDD exposure (ATSDR 1998).

6 2,3,7,8-TCDD is a potent animal carcinogen and has tested positive for carcinogenicity in 19
7 different studies in four animal species: mice, rat, hamsters, and fish (Huff 1992; Johnson et al.
8 1992). EPA classifies 2,3,7,8-TCDD as a B2, probable human carcinogen (EPA 2002).

9 The exact mechanism of how 2,3,7,8-TCDD causes carcinogenicity is not well understood but 10 the evidence indicates that direct DNA damage through formation of DNA adducts is not the 11 mechanism. The carcinogenicity of 2,3,7,8-TCDD is thought to involve the Ah receptor. 12 2,3,7,8-TCDD is considered a nongenotoxic carcinogen and has tested as not mutagenic in the 13 Salmonella/Ames test. 2,3,7,8-TCDD is a potent tumor promoter and is either a weak initiator or 14 a non-initiator. 2,3,7,8-TCDD and the other carcinogenic dioxin congeners are whole and 15 complete carcinogens as tested in mice, rats, hamsters, and fish.

16 PCDD/PCDFs disrupt normal homeostatic processes that regulate cell growth and 17 differentiation. These disruptions produce a wide range of toxic effects and histopathological 18 changes. The PCDD/PCDF congeners vary in many ways including affinity for the Ah receptor, 19 potency, and potential for interaction.

20 2.6.3 2,3,7,8-TCDD Toxic Equivalence (TEQ)

21 The polychlorinated halogenated (PCH) congeners (including both PCBs and dioxins/furans) 22 have different toxicity potencies, and there may be synergistic and/or antagonistic effects among 23 the congeners. To estimate the relative toxicity of mixtures of PCH mixtures, a system of toxic 24 equivalency factors (TEFs) has been developed. This approach is based on in vivo and in vitro 25 toxicity of each of the PCH congeners in relation to 2,3,7,8-TCDD, which is considered to be the 26 most toxic of the PCH class of chemicals (Van den Berg et al. 1998; Birnbaum and DeVito 1995; 27 Safe 1994). There are a number of assumptions made when using the TEF approach. These 28 include: (1) PCH congeners are Ah-receptor antagonists and their toxicological potency is

mediated by their binding affinity; and (2) no interaction occurs between the congeners and thus,
the sum of the individual congener effects accounts for the potency of the PCH mixture. The
overall effect of these assumptions is a potency estimate or toxic equivalency (TEQ) value. To
generate a TEQ, the following equation (equation modified from Van den Berg et al. 1998) is
used:

6
$$TEQ = \sum_{n=1}^{7} [PCDD_n \ x \ TEF_n] + \sum_{p=1}^{10} [PCDF_p \ x \ TEF_p] + \sum_{q=1}^{12} [PCB_q \ x \ TEF_q]$$

7 where:

8 TEQ = Toxic equivalence

9 $PCDD_n = Polychlorinated dibenzo-p-dioxin congener concentration$

10 $PCDF_p$ = Polychlorinated dibenzo-p-furan congener concentration

11 PCB_q = Polychlorinated biphenyl congener concentration

12 $TEF_{n,p,q} = Toxic equivalency factor for appropriate individual PCDD/PCDF and PCB$ 13 congeners, respectively.14

There are a number of TEF approaches available in the scientific literature for PCHs (e.g., Van den Berg et al. 1998; Kennedy et al. 1996; Safe 1994; NATO 1988). For this ERA, the TEFs presented by Van den Berg et al. (1998) were adopted. TEF values were developed for those compounds that: (1) show a structural relationship to PCDDs and PCDFs; (2) bind to the Ah receptor; (3) elicit an Ah-receptor-mediated biochemical and toxic response; and (4) are persistent and accumulate in the food chain (Van den Berg et al. 1998; Birnbaum and DeVito 1995).

The Van den Berg et al. TEFs are the most recently proposed and are based on the best available science in terms of identifying specific endpoints consistent with the mode of action of each of the congeners. They have also been widely accepted and applied in the scientific literature since 1998 (Dyke and Stratford 2002; Lindstrom et al. 2002). Van den Berg et al. (1998) present TEF values for use in deriving TEQ for mammals, fish, and birds as predators (Table 2.6-2).

Table 2.6-2

			TEF		
No.	Congener	Mammals	Fish	Birds	
1	PCB-77	0.0001	0.0001	0.05	
2	PCB-81	0.0001	0.0005	0.1	
3	PCB-126	0.1	0.005	0.1	
4	PCB-169	0.01	0.00005	0.001	
5	PCB-105	0.0001	<0.000005*	0.0001	
6	PCB-114	0.0005	<0.000005*	0.0001	
7	PCB-118	0.0001	<0.000005*	0.00001	
8	PCB-123	0.0001	<0.000005*	0.00001	
9	PCB-156	0.0005	<0.000005*	0.0001	
10	PCB-157	0.0005	<0.000005*	0.0001	
11	PCB-167	0.00001	<0.000005*	0.00001	
12	PCB-189	0.0001	<0.000005*	0.00001	
13	1,2,3,4,6,7,8-HpCDD	0.01	0.001	<0.001*	
14	1,2,3,4,6,7,8-HpCDF	0.01	0.01	0.01	
15	1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.01	
16	1,2,3,4,7,8-HxCDD	0.1	0.5	0.05	
17	1,2,3,4,7,8-HxCDF	0.1	0.1	0.1	
18	1,2,3,6,7,8-HxCDD	0.1	0.01	0.01	
19	1,2,3,6,7,8-HxCDF	0.1	0.1	0.1	
20	1,2,3,7,8,9-HxCDD	0.1	0.01	0.1	
21	1,2,3,7,8,9-HxCDF	0.1	0.1	0.1	
22	1,2,3,7,8-PeCDD	1	1	1	
23	1,2,3,7,8-PeCDF	0.05	0.05	0.1	
24	2,3,4,6,7,8-HxCDF	0.1	0.1	0.1	
25	2,3,4,7,8-PeCDF	0.5	0.5	1	
26	2,3,7,8-TCDF	0.1	0.05	1	
27	2,3,7,8-TCDD	1	1	1	
28	OCDD	0.0001	<0.0001*	0.0001	
29	OCDF	0.0001	<0.001*	0.0001	

TEF Values for Mammals, Fish, and Birds as Predators

1 2 3

*Values that are less than should be considered to be the upper limit for use in any TEQ calculation.

Source: Van den Berg et al. 1998

1 2.7 CONCEPTUAL MODEL

2 A conceptual model is a written description and visual representation of predicted relationships 3 between ecological entities and the stressors to which they may be exposed. In essence, the 4 conceptual model presents a series of working hypotheses regarding how the stressors might 5 affect ecological components at the site. Risk hypotheses are specific assumptions about 6 potential risk to assessment endpoints and may be based on theory and logic, empirical data, and 7 mathematical or probability models. The hypotheses are formulated using professional judgment 8 and available information of the ecosystem at risk, potential stressor sources and characteristics, 9 and observed or predicted effects on assessment endpoints. Conceptual models include 10 ecosystem processes that influence receptor responses, or exposure scenarios that qualitatively link land use activities to stressors and describe primary, secondary, and tertiary exposure 11 12 pathways, ecological effects, and ecological receptors (EPA 1998).

13 The development of the conceptual model is a complex, non-linear process, with many parallel 14 activities that result in modifications to the conceptual model as additional information becomes 15 available. The objectives of the conceptual model presented here are to illustrate the important 16 relationships within the Housatonic study area, and to specify exposure scenarios evaluated in 17 the ERA, as a refinement of the conceptual model outlined in the SI Work Plan. The model was 18 refined based upon physical, chemical, and biological information collected during the 19 investigation, and on the body of scientific knowledge on the COPCs that has also evolved in the 20 interim. The following discussion presents an overview of the primary exposure pathways, risk 21 questions/testable hypotheses, and a visual representation of the predicted relationships between 22 ecological receptors and contaminant stressors (see Figure 2.7-1).



1 2 3 4

5 2.7.1 Exposure Pathways

6 Exposure of receptors to COPCs is possible through various pathways including absorption through gills, dermal contact, ingestion of sediment, ingestion of surface water, ingestion of soil, 7 8 ingestion of contaminated food, and inhalation of volatilized substances. Sediment may become 9 resuspended if hydrodynamics disturb the sediment bed and distribute suspended sediment 10 outside the river when floodplains are inundated. Organisms may also be exposed to chemical 11 contaminants through trophic transfer. Organisms lower in the food chain may ingest and 12 accumulate a contaminant, which is then passed on when they are consumed by higher food 13 chain predators.

Benthic and soil communities are at risk of direct exposure to PCBs and several other COPCs
 (e.g., dioxins, furans, lead, mercury, PAHs). Species in these communities are exposed to
 COPCs through direct contact with interstitial porewater, ingestion of sediment particles, and
 ingestion of organisms that have also been exposed to contaminants.

5 Pelagic organisms in the Housatonic River system are exposed to COPCs through dermal and 6 gill contact with surface water; ingestion of water, suspended sediment, and organic matter; 7 ingestion of sediment for bottom-feeding fish; and ingestion of other benthic and pelagic 8 organisms. Uptake of PCBs by fish occurs mainly through the gills and the gastrointestinal tract 9 (Shaw and Connell 1984). Most PCB accumulation in top fish predators can be attributed to the 10 food pathway (Thomann 1989). Other species, such as amphibians, are also exposed to PCB-11 contaminated surface water. The early life stages of these organisms are entirely aquatic, and 12 because the skin is a respiratory surface during this phase, dermal exposure may be important.

13 Insectivorous, carnivorous, and piscivorous birds and mammals that reside, or partially reside, 14 within the PSA are exposed to PCBs principally through diet and trophic transfer. PCBs are 15 highly bioaccumulative substances that increase in concentration as they are passed up the food 16 chain. For organisms inhabiting the Lake St. Clair ecosystem, Haffner et al. (1994) showed that 17 PCB concentrations increased from 935 μ g/kg in sediment, to 1,360 μ g/kg in bivalves, to 7,240 18 μg/kg in oligochaetes, and to 64,900 μg/kg in predatory gar pike. MacKay (1989) has also noted 19 the food chain biomagnification of PCBs for several piscivorous birds. The avian and 20 mammalian predators of the Housatonic River study area would similarly be expected to 21 accumulate PCBs from the prey they consume. Water, sediment, and soil consumption from 22 foraging activities likely contribute less to PCB exposure.

23 The exposure pathways for other COPCs depend largely on their chemical and physical 24 properties. Highly lipophilic substances, such as dioxins and furans, will behave similarly to 25 PCBs, partitioning to sediment and being upwardly mobile in the food chain.

Figure 2.7-1 characterizes the ecosystem in the Housatonic River PSA, as well as the major exposure pathways for COPCs.

2-56

As a component of the development of the site conceptual model, testable hypotheses or "risk questions" are developed to provide the basis for the study design and selection of measurement endpoints. These hypotheses represent statements regarding anticipated ecological effects and define the focus of the individual studies. In general, the primary question to be asked by the risk hypothesis is "what probabilities are associated with effects of differing magnitudes as a result of exposure of the assessment endpoint to the COPC?" The three major lines of evidence used to answer this question are:

8 9

10

- Comparison of an estimated or measured exposure concentration of a COPC to concentrations known from the literature to be toxic to receptors associated with the assessment endpoint.
- Comparison of laboratory bioassay results using media from the site to the results using media from a reference site, and/or comparing in situ toxicity test results at the site to results at a reference location, or comparisons of results across a concentration gradient.
- Comparison of observed effects in the receptors in the field, with observations in similar receptors at reference locations, or across a concentration gradient (e.g., exposure modeling).

1 2.8 SELECTION OF ASSESSMENT AND MEASUREMENT ENDPOINTS

The selection of endpoints for consideration in an ERA requires identification of ecological characteristics that may be adversely affected by site contaminants. In an ERA, two types of endpoints are required – assessment endpoints and measurement endpoints. Assessment endpoints represent specific ecological values deemed important to protect; measurement endpoints are the tools used to determine the outcome for the assessment endpoints.

7 2.8.1 Assessment Endpoints

8 Assessment endpoints are unambiguous statements or goals concerning specific ecological 9 characteristics (e.g., reproductive effects on aquatic organisms) that are to be evaluated and 10 protected (EPA 1994, 1998). Assessment endpoints determine the foundation for the ERA 11 because they:

12 13 14

15

• Provide guidance for evaluating the site and the extent of contamination.

Establish a basis for assessing the potential risks to identified receptors.

Assist in the identification of the ecological structure and function at the site.

Each site or area evaluated in an ERA has the potential to be biologically unique; therefore, there is no universal list of assessment endpoints (Suter 1993). Because it is not practical or possible to directly evaluate risks to all of the individual components of the ecosystem at a site, assessment endpoints should focus the risk assessment on particular components of the ecosystem that could be adversely affected by contaminants from the site (EPA 1997). According to EPA's *Ecological Risk Assessment Guidance for Superfund* (EPA 1997):

- "Assessment endpoints for the baseline ERA must be selected based on the ecosystems,
 communities, and/or species potentially present at the site. The selection of assessment
 endpoints depends on:
- The contaminants present and their concentration;
- Mechanisms of toxicity of the contaminants to different groups of organisms;
- Ecologically relevant receptor groups that are potentially sensitive or highly exposed to the contaminant and attributes of their natural history; and
- Potentially complete exposure pathways."

To guide this process, EPA (1998) provides further detail on the criteria that assessment
 endpoints should satisfy:

3 Ecological relevance—Assessment endpoints must reflect biologically important 4 characteristics of the ecosystem and should be functionally related to other key 5 components of the system. Ecologically relevant assessment endpoints are particularly 6 valuable for identifying potential cascading adverse effects resulting from the loss or 7 reduction of a species or guild. For example, an alteration of the benthic community is of 8 concern not only to the benthos, but also to higher trophic levels because of disruption at 9 the base of the food web. Alternatively, an alteration at the higher trophic levels may reflect an integration of effects throughout the ecosystem. 10

- *Susceptibility to known or potential stressors*—There should be a cause-effect linkage
 between the assessment endpoint and the magnitude of the contaminant stressor.
- 13 **Relevance to management goals**—The selection of endpoints that reflect societal values 14 and management goals, while not scientifically based, ensures that the risk assessment 15 will have utility for the risk management decisions that must be made. Management goals are desired characteristics of the ecosystem deemed to have value to the public. For 16 17 example, fish abundance and biomass may be used as indicators of whether fisheries are 18 being adequately protected. The status of the benthic invertebrate community in the 19 study area is often a good indicator of the overall productivity of the aquatic ecosystem, 20 making it a relevant endpoint for maintaining a viable fishery in an area.

In addition, specific assessment endpoints should define the ecological value in sufficient detail to identify measures needed to answer specific questions or to test specific hypotheses (EPA 1997). An assessment endpoint must be definable in a practical context, and requires both an entity (that can be clearly defined) and an attribute (that can be measured or assessed). The operational definition ensures that the assessment endpoint can be linked with a measured response.

Ultimately, the value of an ERA depends on whether it can be used to determine if a baseline risk is present and to support appropriate managerial decisions. Therefore, the selection of assessment endpoints is fundamental in determining the utility of the risk assessment process. Once assessment endpoints are selected and the conceptual model of exposure is developed, testable hypotheses and measurement endpoints are developed to determine whether or not a potential threat to the assessment endpoints exists (EPA 1997).

1 2.8.2 Measurement Endpoints

A measurement endpoint is defined as "a measurable ecological characteristic that is related to the valued characteristic chosen as the assessment endpoint." Measurement endpoints link the conditions existing on-site to the goals established by the assessment endpoints through the integration of modeled, literature, field, or laboratory data (Maughan 1993).

6 "Measurement endpoints are frequently numerical expressions of observations (e.g., toxicity test 7 results, community diversity measures) that can be compared statistically to a control or 8 reference site to detect adverse responses to a site contaminant" (EPA 1997). Measurement 9 endpoints can include measures of exposure (e.g., contaminant concentrations in water or 10 tissues) as well as measures of effect.

11 It is desirable to have more than one measurement endpoint for each assessment endpoint, 12 thereby providing multiple lines of evidence for the evaluation. However, the primary 13 consideration for selecting measurement endpoints should always be how many and which lines 14 of evidence are appropriate to support risk management decisions at the site. Once it has been 15 determined which lines of evidence are required to answer questions concerning the assessment 16 endpoint, the measurement endpoints by which the questions or test hypotheses will be examined 17 are selected (EPA 1997).

In selecting an appropriate measurement endpoint to represent an assessment endpoint, thefollowing criteria are considered (Suter 1991):

- 20 Corresponds to or is predictive of an assessment endpoint.
 - Readily measurable.
 - Appropriate to site scale, exposure pathways, and temporal dynamics.
- Diagnostic.
 - Broadly applicable.
 - Standard.
- 25 26

21

22

24

In particular, measurement endpoints that address both sensitivity and likely exposure to
stressors are relevant to management concerns (EPA 1998).

With the selection of measurement endpoints, the conceptual model development is essentially
 completed. The conceptual model, which is discussed in Section 2.7, then is used to guide the
 study design and development of data quality objectives (DQOs).

4 Over a period of several years preceding the Consent Decree, EPA, GE, and other stakeholders 5 discussed available information on contaminants and the Housatonic River ecosystem, and determined the assessment endpoints appropriate for the ERA. Past discussions and written 6 7 comments between these parties demonstrated that while some parties expressed a preference for 8 measurement endpoints using controlled studies, others had a preference for field-based 9 observations and studies. The EPA SIWP addressed both of these preferences by including both 10 a field and a controlled study component for assessment endpoints, where possible and/or 11 appropriate. In addition, GE supplemented the measurement endpoints in the EPA SIWP with 12 studies they conducted independent from agency review, but subject to EPA oversight. GE 13 requested that these studies be incorporated into this ERA, and EPA has done so where the study 14 was determined to be relevant to the assessment endpoint. The assessment and associated 15 measurement endpoints that were used by EPA to evaluate potential ecological risks resulting 16 from PCBs, and possibly other contaminants in the Lower Housatonic River that were 17 established in the EPA SI Work Plan (WESTON 2000), are presented in Table 2.8-1. The 18 independent studies that GE conducted are summarized in Table 2.8-2.

19 The conceptual model for the site demonstrates the complexity of the ecosystem being evaluated. 20 It was necessary to develop assessment endpoints that were representative of the varying habitats 21 and exposure pathways that exist at the site, and for which there is the potential for differing 22 baseline risk to occur (i.e., a deepwater riverine reach versus a forested floodplain). In addition, 23 many studies conducted as part of this investigation included multiple measurement endpoints in 24 the design. Rather than list these individual measurement endpoints separately, the assessment 25 endpoint and principal measurement endpoints are presented in Table 2.8-1. A listing of all the 26 measurement endpoints included in the design is presented in the SOPs for the individual studies 27 (WESTON, 2000b). In some cases, the investigators added additional endpoints during the 28 conduct of the study. These are discussed in the individual investigator reports, and, where 29 relevant to the assessment endpoint, in the appropriate assessment endpoint appendix.
Ecological Assessment and Measurement Endpoints

Receptor	Assessment Endpoint	Measurement Endpoint
Benthic Invertebrates	Community structure, survival, growth, and reproduction	Community composition; species richness, abundance, and biomass and other metrics compared with similar metrics at reference locations.
		Sediment Quality Triad evaluation—Evaluation includes benthic community composition, sediment toxicity testing, and sediment chemistry.
		Sediment macroinvertebrate chronic toxicity testing using <i>Hyalella azteca</i> to determine survival, growth, and reproduction; and <i>Chironomus tentans</i> to determine survival, growth, and emergence.
		In situ toxicity studies using <i>C. tentans, Daphnia magna, H. azteca,</i> and <i>Lumbriculus variegatus</i> to determine survival and growth. (Growth evaluated only in <i>C. tentans.</i>)
		Toxicity Identification Evaluation (TIE) laboratory 24-hour study using <i>Ceriodaphnia dubia</i> to determine survival for different porewater fractions of contaminant classes.
		Comparison of sediment chemistry with sediment quality values (SQVs) and tissue chemistry with tissue effects thresholds.
Amphibians	Community condition, survival, reproduction, development, and maturation	Semiquantitative sampling of larval amphibians in breeding habitats with different sediment concentrations of stressors. Endpoints include species richness per habitat type; species abundance; gross pathology; and body, tail, and total length measurements.
		Surveys of vernal pools to quantitate amphibians entering vernal pools and determine breeding behavior and condition; egg laying, hatching success, and larval growth and development; metamorphosis and emigration.

1 2 3

Ecological Assessment and Measurement Endpoints (Continued)

Receptor	Assessment Endpoint	Measurement Endpoint
Amphibians (cont'd)		Amphibian toxicity tests designed with exposure over a gradient of stressor concentrations in site sediment. Toxicity endpoints include morphology of embryos and juveniles, limb development, skin maturation, and tail resorption of <i>Rana pipiens</i> and <i>R. sylvatica</i> .
		Gravidity of females; egg count; necrotic eggs; oocyte maturity; sperm count, morphology, and viability; fertilization rate; embryo viability; hatching success; mortality; and teratogenesis of <i>Rana pipiens</i> collected from the study area over a contamination gradient and compared with an external control.
		In situ amphibian toxicity study evaluated how multiple stressors (including population density and PCB exposure) affect survival and growth of larval <i>Rana sylvatica</i> .
Fish	Survival, growth, and reproduction	Determine the possible extent of adverse effects by comparing the concentrations of COCs in sediment to the concentrations reported in the literature to cause adverse effects on the survival, growth, or reproduction of fish.
		Compare the concentrations of COCs in fish tissues to the concentrations in fish tissues that may result in adverse effects, based on site-specific fish toxicity studies.
		Compare the concentrations of COCs in fish tissues to concentrations documented in the literature to result in adverse effects.
		Evaluate field survey information (fish biomass study, ecological characterization study, and largemouth bass habitat and reproduction study) to qualitatively assess potential effects.
Insectivorous Birds	Survival, growth and reproduction	Reproductive performance of tree swallows (<i>Tachycineta bicolor</i>) based on the nest box study conducted in areas of varying stressor sediment concentrations. Parameters for evaluation include nest building, egg presence/absence, number of eggs, and hatching success.
		Comparison of site-specific tissue concentrations in tree swallows with reference area concentrations and with residue effects levels from literature.
		Quantitative comparison of daily intakes based on dietary intake of stressors by tree swallows and American robins using site-specific stressor levels in invertebrates and comparison with literature-based effect values.
		American robin productivity within the PSA and reference areas was evaluated and compared to associated PCB exposure. Metrics assessed included clutch size, hatching and fledgling success, and PCB concentrations in robin eggs.

Ecological Assessment and Measurement Endpoints (Continued)

Receptor	Assessment Endpoint	Measurement Endpoint
Piscivorous Birds	Survival, growth, and reproduction	Quantitative comparison of daily intakes based on dietary intake of stressors by belted kingfishers and osprey using site-specific fish tissue concentrations and site-specific stressor levels in other aquatic-related food items (e.g., crayfish and frogs), with literature- based effect values.
		Belted kingfisher nests within and adjacent to the PSA were identified and monitored for productivity (i.e., number of eggs, number of eggs hatched, and fledgling success). Habitat suitability and modeled PCB exposure were also evaluated and related to nest productivity.
Piscivorous Mammals	Survival, growth, and reproduction	Mink toxicity tests using Housatonic River fish in the diet. Toxicity endpoints include body weight, food intake rate, length of gestation, reproductive success (measured by number of females whelping, newborns/female, litter weight, etc.), survival, histopathology, presence/absence of jaw lesions, organ weights, and various biochemical endpoints.
		Quantitative evaluation of mink and otter presence using scent posts and snow tracking. (two separate studies)
		Quantitative comparison of daily intakes based on dietary intake of stressors by mink and river otter using site-specific stressor levels in fish and other aquatic prey with literature-based effect values.
Omnivorous and Carnivorous Mammals	Survival, growth and reproduction	Reproductive evidence in trapped small mammals (e.g., examination of placental scars to determine number of litters, and number/litter).
		Quantitative comparison of daily intakes based on dietary intake of stressors by northern short-tailed shrews and red fox using site- specific stressor levels in soil invertebrates and small mammals with literature-based effect values.
		Demographic characteristics of short-tailed shrew populations were assessed at six locations within the PSA that spanned a range of PCB soil concentrations. Population characteristics measured at each location included survival rate, sex ratio, reproduction and growth rate, and body mass.
Special Status Species (Endangered, Threatened)	Survival, growth, and reproduction	Quantitative comparison of daily intakes based on dietary intake of stressors using site-specific media concentrations and comparison with literature-based effect values.

Summary of GE Ecological Studies

Study	Objectives
Robin Productivity in the Housatonic River Watershed (Henning, Robinson, and Jenkins 2002)	 Document reproductive output of robins in the PSA and reference areas.
	• Evaluate exposure of eggs and young to PCBs.
	 Evaluate relationships between exposure and reproductive output.
Productivity and Density of Belted Kingfishers on the Housatonic River (Henning and Brooks 2002)	 Evaluate kingfisher productivity in situ in a system with known PCB contamination.
	 Determine whether estimated PCB dose, habitat quality, phenology, and/or nest density were significant predictors of reproductive success.
Experimental Analysis of the Context-Dependent Effects of Early Life-Stage PCB Exposure on <i>Rana</i> <i>sylvatica</i> (Resatarits 2002)	 Determine the effects and interactions of PCB exposure and density-dependence on the growth and development of amphibian offspring.
Spatial and Demographic Effects on Tree Swallow Nest Quality and Reproductive Success (Robertson and Jones 2002)	 Determine the effects of: (1) inter-nest spacing, (2) proximity to edge, (3) settlement and nest-building date, (4) availability of nesting material, (5) history of the nest-box and nest-box grid, and (6) female and male age, on both nest quality and reproductive success.
Demography of Short-Tailed Shrew Populations Living on Contaminated Sites (Boonstra 2002)	 Assess whether PCBs adversely affect population demography of short-tailed shrew living in a natural environment.
Evaluation of Mink – Presence/Absence, Distribution, and Abundance in the Housatonic River Floodplain (BBL 2002)	 Qualitatively determine the presence/absence, abundance, and distribution of free-ranging mink in the PSA.
Evaluation of Largemouth Bass Habitat, Population Structure, and Reproduction in the Housatonic	 Determine if largemouth bass (LMB) population in the study reach is self-sustaining.
River, Massachusetts (R2 2002)	 Determine if the LMB population is dependent on tributary recruitment.
	 Identify which attributes of growth, size-class structure, and reproduction of the LMB population are similar to LMB populations in other systems.

Several field surveys were conducted to provide information specifically on species presence.
 Although field surveys can also be used to assess community condition, the majority of the field
 surveys (with limited exceptions) were designed for community characterization and were not
 intended to be used as lines of evidence; therefore, they are not included in Table 2.8-1.

5 Tissue samples were collected for contaminant analyses for a number of species in support of the 6 ecological exposure assessment, human health risk assessment, and PCB fate and effects 7 modeling. Endpoints typically associated with residue effects range from general toxicity to 8 reproductive effects and lethality. Where comparable literature-based residue effects data were 9 identified through various literature and toxicity database searches, these were incorporated to 10 provide a comparison of site-specific tissue data with literature-based effects levels in the risk 11 assessment to provide additional lines of evidence.

Although many of the endpoints presented are linked to organism-level effects (e.g., survival and reproduction), these endpoints are expected to be strong indicators of potential local populationlevel effects (e.g., viability of the benthic community within the Housatonic River study area) (EPA 1992, 1999). Extrapolation from organism-level to population-level effects may be logically achieved based on the predictive nature of the endpoint and/or through the use of process-based models.

18 2.9 WEIGHT-OF-EVIDENCE APPROACH TO ANALYSIS

19 Inferences in ERAs are often made by weight-of-evidence (WOE) rather than traditional 20 scientific standards of proof (EPA 1992). The WOE approach is a process by which 21 measurement endpoints are related to an assessment endpoint to evaluate whether significant risk 22 is posed to the environment (Menzie et al. 1996). A formal WOE can range from a simple 23 qualitative assessment to a highly quantitative evaluation; however, no matter what form the 24 WOE takes, it should provide documentation of the thought process used when assessing 25 potential ecological risk.

The term "line of evidence" as used in this discussion follows the definition of "Information derived from different sources or by different techniques that can be used to describe and interpret risk estimates" provided in the *Guidelines for Ecological Risk Assessment* (EPA 1998). Unlike the term "weight-of-evidence," this definition does not imply assignment of qualitative or
 quantitative weightings to information. The three general lines of evidence under which most
 measurement endpoints fall are (Hull and Suter 1994; Suter et al. 1995):

- 4
- 5 6

7

8

Biological survey data that indicate the state of the receiving environment.

- Media toxicity data that indicate whether the contaminated media are toxic (i.e., laboratory or in situ toxicity testing).

 Single contaminant toxicity data that indicate the toxic effects of the concentration measured in site media (e.g., exposure modeling).

9 Two or three general lines of evidence were considered in evaluating potential risk for each 10 assessment endpoint. A more detailed presentation of the specific lines of evidence used in this 11 risk assessment is provided in the appendix for each assessment endpoint.

The WOE approach used in this ERA for each of the assessment endpoints follows the approach
originally described in the *Massachusetts Weight-of-Evidence Special Report* (Menzie et al.
14 1996).

According to Menzie et al. (1996), WOE is reflected in three characteristics of measurement endpoints: (1) the weight assigned to each measurement endpoint; (2) the magnitude of response observed in the measurement endpoint; and (3) the degree of concurrence among outcomes of multiple measurement endpoints for a given assessment endpoint.

19 First, weights are assigned to measurement endpoints based on ten attributes (summarized in 20 Table 2.9-1) related to: (1) strength of association between assessment and measurement 21 endpoints; (2) data and study quality; and (3) study design and execution. The initial step in this 22 process involves assigning qualitative (low through high) weights to each attribute, which is a 23 subjective process involving professional judgment using criteria outlined in Menzie et al. 24 (1996). This process is described in the appendix for each assessment endpoint. Figure 2.9-1 25 provides a generic example of the measurement endpoint weighting process used to evaluate 26 each assessment endpoint.

Attributes for Judging Measurement Endpoints

1. Strength of Association Between Assessment and Measurement Endpoints

Biological linkage between measurement endpoint and assessment endpoint—This attribute refers to the extent to which the measurement endpoint is representative of, correlated with, or applicable to the assessment endpoint. If there is no biological linkage between a measurement endpoint (e.g., a study that may have been performed for some other purpose) and the assessment endpoint of interest, then that study should not be used to evaluate the stated assessment endpoint. Biological linkage pertains to similarity of effect, target organ, mechanism of action, and level of ecological organization.

Correlation of stressor to response—This attribute relates to the degree to which a correlation is observed between levels of exposure to a stressor and levels of response and the strength of that correlation.

Utility of measure—This attribute relates to the ability to judge results of the study against wellaccepted standards, criteria, or objective measures. As such, the attribute describes the applicability, certainty, and scientific basis of the measure, as well as the sensitivity of a benchmark in detecting environmental harm. Examples of objective standards or measures for judgment might include ambient water quality criteria, sediment quality criteria, biological indices, and toxicity or exposure thresholds recognized by the scientific or regulatory community as measures of environmental harm.

2. Data and Overall Study Quality

Quality of data and overall study—This attribute reflects the degree to which data quality objectives and other recognized characteristics of high quality studies are met. The key factor affecting the quality of the data is the appropriateness of data collection and analysis practices. The key factor of the quality of the study is the appropriateness and implementation of the experimental design and the minimization of confounding factors. If data are judged to be of poor or no quality, the study would be rejected for use in the ERA.

Table 2.9-1

Attributes for Judging Measurement Endpoints (Continued)

3. Design and Execution

Site-specificity—This attribute relates to the extent to which media, species, environmental conditions, and habitat types that are used in the study design reflect the site of interest.

Sensitivity of the measurement endpoint to detecting changes—This attribute relates to the ability to detect a response in the measurement endpoint, expressed as a percentage of the total possible variability that the endpoint is able to detect. Additionally, this attribute reflects the ability of the measurement endpoint to discriminate between responses to a stressor and those resulting from natural or design variability and uncertainty.

Spatial representativeness—This attribute relates to the degree of compatibility or overlap between the study area, locations of measurements or samples, locations of stressors, and locations of ecological receptors and their points of potential exposure.

Temporal representativeness—This attribute relates to the temporal compatibility or overlap between the measurement endpoint (when data were collected or the period for which data are representative) and the period during which effects of concern would be likely to be detected. Also linked to this attribute is the number of measurement or sampling events over time and the expected variability over time.

Quantitativeness—This attribute relates to the degree to which numbers can be used to describe the magnitude of response of the measurement endpoint to the stressor. Some measurement endpoints may yield qualitative or hierarchical results, while others may be more quantitative.

Use of a standard method—The extent to which the study follows specific protocols recommended by a recognized scientific authority for conducting the method correctly. Examples of standard methods are study designs or chemical measures published in the Federal Register or the Code of Federal Regulations, developed by ASTM, or repeatedly published in the peer-reviewed scientific literature, including impact assessments, field surveys, toxicity tests, benchmark approaches, toxicity quotients, and tissue residue analyses. This attribute also reflects the suitability and applicability of the method to the endpoint and the site, as well as the need for modification of the method.

1 Source: Menzie et al. 1996.

Score each measurement endpoint from low to high Assessment Endpoint:

Attribute	Measurement Endpoint A	Measurement Endpoint B	Measurement Endpoint C
I. Relationship between Measurement an	d Assessment Endpo	ints	
 Degree of Association 	Moderate	High	High
 Stressor/Response 	High	Moderate	High
 Utility of Measure 	Moderate	High	High
II. Data Quality			
 Quality of data 	High	High	High
III. Study Design			
 Site-specificity 	High	High	High
 Stressor-specificity 	Moderate	Moderate	Moderate
 Sensitivity 	Moderate	Low	High
 Spatial representativeness 	Moderate	High	Moderate
Temporal representativeness	Low	Low	Moderate
 Quantitativeness 	High	High	High
• Use of a standard method	Moderate	Moderate	Moderate
Total Value	Moderate	Moderate	Moderate-High

4 5

1 2 3

6

Figure 2.9-1 Example Endpoint Weighting Sheet

To ensure that the selected measurement endpoints would support the achievement of the study
objectives, a preliminary WOE was conducted in the SIWP. Therefore, it is expected that low
attribute values will not typically be assigned as total scores for a line of evidence in the final
WOE.

The second step of the Menzie et al. (1996) approach is to evaluate the magnitude of response in
the measurement endpoint, considering two questions:

- Does the measurement endpoint indicate the presence or absence of risk (yes, no, or undetermined)?
- 15 Is the response low or high?

Figure 2.9-2 illustrates a matrix for an assessment endpoint that provides a simple 1 2 communication tool summarizing the conclusions of the WOE evaluation of the magnitude of 3 response.

Measurement Endpoints	Weighting Score (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate Low)
Endpoint A			
Endpoint B			
Endpoint C			

7 8

4 5 6

Figure 2.9-2 Scoring Sheet for Evidence of Harm and Magnitude

9

10 The third step of the WOE process evaluates the degree of concurrence among measurement 11 endpoints by plotting the output of the two preceding steps on a matrix for all measurement 12 endpoints associated with a given assessment endpoint (see Figure 2.9-3). The matrix allows 13 easy visual examination of agreements or divergences among measurement endpoints, 14 facilitating interpretation with respect to the assessment endpoint. Logical connections, 15 interdependence, and correlations among endpoints should also be considered when evaluating 16 concurrence. The generalized matrix shown in Figure 2.9-3 is used for each assessment endpoint 17 to illustrate the results of the WOE assessment of risks of PCBs and other COCs. Completed 18 matrices specific to each assessment endpoint are presented in the respective appendix for each 19 endpoint and each summary section of the report.



1 2

3

Figure 2.9-3 Example of Qualitative Assessment

4 2.10 EXTRAPOLATION OF RISK ESTIMATES FOR SELECTED ENDPOINTS 5 DOWNSTREAM OF WOODS POND

6 Because of the decline in PCB mass and concentrations and the associated decrease in the 7 amount of data collected downstream of the PSA, the detailed approach followed in assessing 8 ecological risks in the PSA was not appropriate or possible. An estimate of potential ecological 9 risks was developed using mapping (GIS) techniques and threshold concentrations, that, if 10 exceeded, would indicate potential risk to six selected target groups: benthic invertebrates, 11 amphibians, warm-water fish, trout, mink, and bald eagles. These target groups were selected 12 based on the risks for these organisms observed in the PSA, and the occurrence of these 13 organisms in the reaches downstream.

For each of these groups, a maximum acceptable threshold concentration (MATC) for total PCBs (tPCBs) in the appropriate medium was developed, based primarily on the detailed risk assessment performed for the PSA. The MATC was then compared to available mediumspecific data for areas downstream of Woods Pond to Long Island Sound. Areas of exceedances

- 1 (HQ > 1), indicating potential risk, were plotted on maps of the river. The specific approaches 2 developed for each of the six target groups are discussed in the appropriate appendices and
- 3 summary sections of this report.

4 2.11 REFERENCES

- ATSDR (Agency for Toxic Substances and Disease Registry). 1995. *Toxicological Profile for Polycyclic Aromatic Hydrocarbons*. Prepared by Research Triangle Institute. August 1995.
- ATSDR (Agency for Toxic Substances and Disease Registry). 1997. *Polychlorinated Biphenyls* (*PCBs*). www.atsdr.cdc.gov/tfacts17.html. June 12, 2001.
- 9 ATSDR (Agency for Toxic Substances and Disease Registry). 1998. *Toxicological Profile* 10 *Chlorinated Dibenzo-p-Dioxins*. Prepared by Research Triangle Institute. December 1998.
- 11 ATSDR (Agency for Toxic Substances and Disease Registry). 2000. *Toxicological Profile for* 12 *Polychlorinated Biphenyls*. November 2000.
- Aulerich, R.J., R.K. Ringer, and J. Safronoff. 1986. Assessment of primary versus secondary
 toxicity of Aroclor 1254 to mink. *Archives of Environmental Contamination and Toxicology* 15:393-399.
- BBL (Blasland, Bouck & Lee, Inc.). 2002. Evaluation of Mink Presence/Absence, Distribution,
 and Abundance in the Housatonic River Floodplain. Report prepared for the General Electric
 Company.
- 19 BBL (Blasland, Bouck & Lee, Inc.) and QEA (Quantitative Environmental Analysis, LLC).
- 20 2003. Housatonic River Rest of River RCRA Facility Investigation Report, Volume 1. Prepared
 21 for General Electric Company. January 2003.
- 22 Bedard, D. and R. May. 1996. Characterization of the polychlorinated biphenyls in the sediments
- of Woods Pond: Evidence for microbial dechlorination of Aroclor 1260 in situ. *Environ. Sci.*
- 24 Technol. 30:237-245.
- Birnbaum, L.S. and M.J. DeVito. 1995. Use of toxic equivalency factors for risk assessment for
 dioxins and related compounds. *Toxicology* 105:391-401.
- Boonstra, R. 2002. Demography of Short-Tailed Shrew Populations Living on PCB
 Contaminated Sites. Report prepared for General Electric Company.
- 29 Callahan, M.A., M.W. Slimak, N.W. Gabel, I.P. May, C.F. Fowler, J.R. Freed, P. Jennings, R.L.
- 30 Durfee, F.C. Whitmore, B. Maestri, W.R. Mabey, B.R. Holt, and C. Gould. 1979. Water-Related
- 31 *Environmental Fate of 129 Priority Pollutants*. Chapter 36. EPA-440/4-79-029a.
- 32 Chadwick & Associates, Inc. 1994. *Aquatic Ecology Assessment of the Housatonic River*, 33 *Massachusetts 1993.* Chadwick & Associates, Inc., Littleton, CO.

- Chem Risk. 1997. Work Plan for the Ecological Risk Assessment of the Housatonic River:
 Volumes I and II. Chem Risk (24 May 1997).
- Connor, K., S. Safe, and C.R. Jefcoate. 1995. Structure-dependent induction of CYP2B by
 polychlorinated biphenyl congeners in female Sprague-Dawley rats. *Biochem. Pharmacol.*50(11):1913-1920.
- 6 Cornelissen, G., H. Rigterink, M.M.A. Ferdinandy, and P. van Noort. 1998. Rapidly desorbing
- 7 fractions of PAHs in contaminated sediments as a predictor of the extent of bioremediation.
- 8 *Environmental Science and Technology* 32:966-970.
- 9 Dyke, P.H. and J. Stratford. 2002. Changes to TEF schemes can have significant impacts on 10 regulation and management of PCDD/F and PCB. *Chemosphere* 47:103-116.
- Eisler, R. 1986. Polychlorinated Biphenyl Hazards to Fish, Wildlife, and Invertebrates: A
 Synoptic Review. Contaminant Hazard Reviews-7. 81 pp. PB86-170057.
- Eisler, R. 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates:
 A Synoptic Review. Contaminant Hazard Reviews, Biological Report 85(1.11). May 1987.
- Eisler, R. and A. Belisle. 1996. Plannar PCB hazards to fish, wildlife, and invertebrates: A
 synoptic review contaminant hazard reviews. Biological Report 31.
- EPA (U.S. Environmental Protection Agency). 1980. *Ambient Water Quality Criteria Document: Polychlorinated Biphenyls*. p. B-2. EPA 440/5-80-068. As cited in ATSDR 2000.
- EPA (U.S. Environmental Protection Agency). 1992. Framework for Ecological Risk
 Assessment. Risk Assessment Forum, Washington, DC. EPA/630/R-92/001.
- EPA (U.S. Environmental Protection Agency). 1993. *Wildlife Exposure Factors Handbook*.
 EPA/600/R-93/187a. Office of Research and Development, Washington, DC.
- EPA (U.S. Environmental Protection Agency). 1994. A Review of Ecological Assessment Case
 Studies from a Risk Assessment Perspective, Volume II. Office of Research and Development,
 Washington, DC. EPA/630/R-94-003.
- 26 EPA (U.S. Environmental Protection Agency). 1996. PCBs: Cancer Dose-Response Assessment
- 27 and Application to Environmental Mixtures. National Center for Environmental Assessment.
- 28 Office of Research and Development, Washington, DC. EPA/600/P-96/001F.
- 29 EPA (U.S. Environmental Protection Agency). 1997. Ecological Risk Assessment Guidance for
- 30 Superfund: Process for Designing and Conducting Ecological Risk Assessments. Interim Final.
- 31 Solid Waste and Emergency Response, U.S. Environmental Protection Agency. EPA-540-R-97-
- 32 006.
- 33 EPA (U.S. Environmental Protection Agency). 1998. Guidelines for Ecological Risk Assessment.
- 34 Risk Assessment Forum, Washington, DC. EPA/630/R-95/002F.

- EPA (U.S. Environmental Protection Agency). 1999. Ecological Risk Assessment and Risk
 Management Principles for Superfund Sites. OSWER Directive 9285.7-28P.
- 3 EPA (U.S. Environmental Protection Agency). 2002. Integrated Risk Information System.
- 4 Haffner, G.D., M. Tomczak, and R. Lazar. 1994. Organic contaminant exposure in the Lake St.
- 5 Clair food web. *Hydrobiologia* 281:19-27.
- Hansen, L.G. 1994. Halogenated Aromatic Compounds. In *Basic Environmental Toxicology*.
 L.G. Cockerham, and B.S. Shane eds. CRC Press. pp. 199-230. ISBN 0-8493-8851-1.
- 8 Harris, S.L. 1997. Inorganic and Organic Constituents and Grain-Size Distribution in Streambed
- 9 Sediment and Ancillary Data for the Connecticut, Housatonic, and Thames River Basins Study
- 10 Unit, 1992-1994. USGS Open-File Report 96-397.
- 11 Henning, M.H. and R.P. Brooks. 2002. Productivity and Density of Belted Kingfishers on the
- 12 *Housatonic River*. Report prepared for General Electric Company.
- Henning, M.H., S. Robinson, and K. Jenkins. 2002. *Robin Productivity in the Housatonic River Watershed*. Report prepared for General Electric Company.
- 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. *Journal of Toxicology and*
- 17 Environmental Health 11:933-946. As cited in EPA 1996.
- Huff, J. 1992. 2,3,7,8-TCDD: A potent and complete carcinogen in experimental animals. *Chemosphere*. 25:173-176.
- Hull, R.N. and G.W. Suter II. 1994. Using the Weight-of-Evidence Approach for Ecological Risk
 Assessment at a DOE Facility. SETAC Denver, CO. November 1, 1994.
- HVA (Housatonic Valley Association). 2001. The Housatonic River Watershed. URL
 http://www.hvathewatershedgroup.org/HousatonicFactSheet.htm.
- Johnson, R., J. Tietge, and S. Botts. 1992. Carcinogenicity of 2,3,7,8-TCDD to medaka.
 Toxicologist. 12(1), 476.
- Kennedy, S.W., A. Lorenzen, and R.J. Norstrom. 1996. Chicken embryo hepatocyte bioassay for
 measuring cytochrome P4501a-based 2,3,7,8-tetrachlorodibenzo-p-dioxin equivalent
 concentrations in environmental samples. *Environmental Science and Technology* 30:706-715.
- Lindstrom, G., L.S. Haug, T. Nicolaysen, and E. Dybing. 2002. Comparability of world-wide analytical data on PCDDs, PCDFs and non-ortho PCBs in samples of chicken, butter and
- 31 salmon. Chemosphere 47:139-146.
- MacDonald, D.D., C.G. Ingersoll, and T.A. Berger. 2000. Development and evaluation of consensus-based sediment quality guidelines for freshwater ecosystems. *Archives of Environmental Contamination and Toxicology* 39:20-31.

- 1 Mackay, D. 1989. Modeling the long-term behavior of an organic contaminant in a large lake
- 2 application of PCBs in Lake Ontario. *Journal of Great Lakes Research* 15:283-297.
- 3 Mass Wildlife. 2002. <u>http://www.state.ma.us/dfwele/dfw/dfw_wd/htm</u>.
- 4 Maughan, J.T. 1993. *Ecological Assessments of Hazardous Waste Sites*. Van Nostrand Reinhold,
- 5 New York, NY.
- Mayes, B.A., E.E. McConnell, and B.H. Nell. 1998. Compenative carcinogenicity in Sprague
 Dawley rets of polychlorinated biphenyl mixtures Arcoclor 1016, 1242, 1254, and 1260. *Toxicol. Sci.* 41(1):62-76.
- 9 Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S.

10 Petron, B. Potocki, S. Svirsky, and P. Tyler. 1996. Special Report of the Massachusetts Weight-

11 of-Evidence Workgroup: A Weight-of-Evidence Approach for Evaluating Ecological Risks.

- 12 Human and Ecological Risk Assessment 2(2):277-304.
- 13 Moore, D.R.J., B.E. Sample, G.W. Suter, B.R. Parkhurts, R.S. Teed. 1999. A Probabilisitic Risk
- 14 Assessment of the Effects of Methylmercury and PCBs on Mink and Kingfishers along East Fork
- 15 Poplar Creek, Oak Ridge, Tennessee, USA. Environmental Toxicology and Chemistry: vol. 18,
- 16 No. 12, pp. 2941-2953.

17 NATO (North Atlantic Treaty Organization). 1988. Pilot study on international information

18 exchange on dioxins and related compounds. International toxicity equivalent factor (I-TEF)

19 method of risk assessment for complex mixtures of dioxins and related compounds. Committee

- 20 on the Challenges of Modern Society, Report 176. August 1988.
- 21 Olcott, P.G. 1995. Ground Water Atlas of the United States: Connecticut, Maine, Massachusetts,
- 22 New Hampshire, New York, Rhode Island, Vermont: Carbonate Rock Aquifers. U.S. Geological
- 23 Survey Publications HA 730-M.

24 Paustenbach, D.J., R.J. Wennig, V. Lau, N.W. Harrington, D.K Rennix, and A.H. Parson. 1992.

25 Recent developments on the hazards posed by 2,3,7,8-tetrachlorodibenzo-p-dioxin in soil:

26 Implication for setting risk-based cleanup levels at residential and industrial sites. *Journal of*

- 27 *Toxicology and Environmental Health* 36(2):103-149. As cited in ATSDR 1998.
- Resetarits, W.J. 2002. Experimental Analysis of the Context-Dependent Effects of Early Life Stage PCB Exposure on Rana sylvatica. Report prepared for General Electric Company.
- Robertson, R.J and J. Jones. 2002. Spatial and Demographic Effects on Tree Swallow Nest
 Quality and Reproductive Success. Report prepared for General Electric Company.
- 32 Russell, R.W., F.A.P.C. Gobas, and G.D. Haffner. 1999. Role of chemical and ecological factors
- 33 in trophic transfer of organic chemicals in aquatic food webs. Environmental Toxicology and
- 34 Chemistry 18:1250-1257.

- 1 R2 (R2 Resource Consultants Inc.). 2002. Evaluation of Largemouth Bass Habitat, Population
- 2 Structure, and Reproduction in the Upper Housatonic River, Massachusetts. Report prepared
- 3 General Electric Company.
- 4 Safe, S.H. 1994. Polychlorinated biphenyls (PCBs): Environmental impact, biochemical and 5 toxic responses, and implications for risk assessment. *Critical Reviews in Toxicology* 24(2):
- 6 87-149.
- SCS (Soil Conservation Service, U.S. Department of Agriculture). 1970. Soil Survey of
 Litchfield County, Connecticut. Storrs, CT, USA.
- 9 SCS (Soil Conservation Service, U.S. Department of Agriculture). 1979. Soil Survey of New
 10 Haven County, Connecticut. Storrs, CT, USA.
- SCS (Soil Conservation Service, U.S. Department of Agriculture). 1981. Soil Survey of Fairfield
 County, Connecticut. Storrs, CT, USA.
- SCS (Soil Conservation Service, U.S. Department of Agriculture). 1988. Soil Survey of
 Berkshire County, Massachusetts, Amherst, MA, USA.
- Shaw, G.R. and D.W. Connell. 1984. Factors controlling bioaccumulation of PCBs. In *PCBs and the Environment, Volume 1*. National Technical Information Service, Springfield, VA. pp. 501 516.
- 18 Suter, G.W., II. 1991. Ecological Endpoints. In Ecological Assessment of Hazardous Waste
- Sites: A Field and Laboratory Reference. U.S. Environmental Protection Agency. EPA/540/R 92/003.
- Suter, G.W, II. 1993. *Ecological Risk Assessment*. Lewis Publishers, Boca Raton, FL. ISBN 0 87371-875-5.
- 23 Suter, G.W., II, B.E. Sample, D.S. Jones, T.L. Ashwood, and J.M. Loar. 1995. Approach and
- 24 Strategy for Performing Ecological Risk Assessments for the U.S. Department of Energy's Oak
- 25 *Ridge Reservation.* Oak Ridge National Laboratory, Oak Ridge, TN. ES/ER/TM-33/R2.
- 26 Swain, P.C. and K.B. Kearsley. 2000. Classification of the Natural Communities of
- 27 Massachusetts. Massachusetts Natural Heritage and Endangered Species Program, Westborough,
- 28 MA, USA.
- Thomann, R.V. 1989. Bioaccumulation model of organic chemical distribution in aquatic food
 chains. *Environmental Science and Technology* 23:699-707.
- 31 Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Freely, J.P. Giesy,
- 32 A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X.R van Leeuwen, A.K.
- 33 Djien Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind,
- 34 M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs) for PCBs,
- 35 PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives* 106(12):775-792.

- Weatherbee, P.B. 1996. Flora of Berkshire County Massachusetts. The Berkshire Museum,
 Pittsfield, MA, USA.
- Weatherbee, P.B. and G.E. Crow. 1992. Natural plant communities of Berkshire County,
 Massachusetts. *Rhodora* 94:171-209.
- WESTON (Roy F. Weston, Inc.). 1998. Source Area Characterization Report. Prepared for U.S.
 Army Corps of Engineers and U.S. Environmental Protection Agency. DCN GEPM-072198 AABA.
- 8 WESTON (Roy F. Weston, Inc.). 2000. Supplemental Investigation Work Plan for the Lower
 9 Housatonic River. Prepared for U.S. Army Corps of Engineers and U.S. Environmental
 10 Protection Agency. 22 February 2000. DCN GEP2-020900-AAME.
- 11 WESTON (Weston Solutions, Inc.). 2003, In Preparation. Modeling Framework Design:
- 12 Modeling Study of PCB Contamination in the Housatonic River. Prepared for U.S. Army Corps
- 13 of Engineers and U.S. Environmental Protection Agency.

ASSESSMENT ENDPOINT—COMMUNITY STRUCTURE, SURVIVAL, GROWTH, AND REPRODUCTION OF BENTHIC INVERTEBRATES

3	Highlights
4	Conceptual Model
5 6	 Sediment and biota tissue are the most relevant exposure media, with the water column of lesser importance.
7	Exposure
8	 PCBs, PAHs, and metals retained in as COCs.
9 10 11 12	 COCs were measured in tissue, sediment, and water at up to 13 sediment quality stations, synoptic with biological effects information. Chemistry data were also collected at numerous other stations throughout the PSA to provide a broader characterization of exposure.
13	Effects
14 15	 Site-specific toxicity tests (laboratory and in situ) indicate adverse responses, relative to both reference stations and negative controls.
16 17	 Benthic community appears altered at multiple stations with elevated PCB concentrations, relative to reference stations.
18	 Toxicological impacts are significantly correlated with PCB exposures.
19	Risk
20 21 22	 Comparison of exposure concentrations to literature effects benchmarks (sediment, tissue, water) indicates high probability of harm, particularly due to PCBs.
23 24	 Toxicity identification evaluation (TIE) implicates non-polar organics (e.g., PCBs) as causal agent in toxicity tests.
25 26	 Contaminants other than PCBs and dioxins/furans do not exhibit concentration gradients consistent with pattern of effects.
27 28 29	 Weight-of-evidence (WOE) approach used to characterize risks, suggesting significant adverse impacts to benthos predicted throughout the PSA; low risks predicted downstream of Woods Pond.
20	

31 3.1 INTRODUCTION

The purpose of this section is to characterize and quantify the current and potential risks posed to benthic invertebrates exposed to contaminants of concern (COCs) in the Housatonic River, focusing on total PCBs (tPCBs) and other COCs originating from the General Electric (GE) facility in Pittsfield, MA. A Pre-ERA was conducted to narrow the scope of the ERA by identifying contaminants, other than tPCBs, that pose potential risks to aquatic biota in the Primary Study Area (PSA) (Appendix B). In the benthic invertebrate ERA, further screening was done to refine the list of contaminants of potential concern (COPCs) to those that were specifically relevant to the invertebrate community in the main channel of the Housatonic River. COCs that screened through to the risk assessment for benthic invertebrates were tPCBs, several metals, several polycyclic aromatic hydrocarbons (PAHs), and dibenzofuran.

- 8 A step-wise approach was used to assess the risks of these COCs to benthic invertebrates in the
- 9 Housatonic River watershed. The four main steps in this process included:
- 10

11

12 13

- 1. Development of a conceptual model (Figure 3.1-1).
- 2. Assessment of exposure of benthic invertebrates to COCs (Figure 3.1-2).
- 3. Assessment of the effects of COCs on benthic invertebrates (Figure 3.1-3).
 - 4. Characterization of risks to benthic invertebrates (Figure 3.1-4).
- 14 This section is organized as follows.
- Section 3.1 (Introduction and Conceptual Model)—Describes the conceptual model for benthic invertebrates, including selection of representative taxa and establishment of measurement and assessment endpoints.
- Section 3.2 (Exposure Assessment)—Describes the quantification of exposures, both specific to stations for which linked biological effects information was collected (n=13) as well as for the broader study area.
- Section 3.3 (Effects Assessment)—Describes the potential effects to benthic
 invertebrates exposed to site COCs, as indicated by the toxicological and biological
 investigations conducted in the PSA. This section also summarizes the ranges of
 benchmarks (toxicity thresholds) derived from the literature.
- Section 3.4 (Risk Characterization)—Integrates the exposure and effects assessments, and makes conclusions regarding risk for benthic invertebrates in the Housatonic River using three main lines of evidence. A discussion of the sources of uncertainty regarding risk estimates follows. Section 3.4 also presents an extrapolation of risks beyond the PSA to areas downstream of Woods Pond.
- 30 31

This section provides a summary of the ERA for benthic invertebrates, which is presented in detail in Appendix D.



Figure 3.1-1 Conceptual Model Diagram: Exposure Pathways for Benthic Invertebrates Exposed to Contaminants of Concern (COCs) in the Housatonic River



Figure 3.1-2 Overview of Approach Used to Assess Exposure of Benthic Invertebrates to Contaminants of Concern (COCs) in the Housatonic River

Effects



Figure 3.1-3 Overview of Approach Used to Assess the Effects of Contaminants of Concern (COCs) to Benthic Invertebrates in the Housatonic River





1 3.1.1 Conceptual Model

Total PCBs, dioxins, and furans are persistent and hydrophobic and lipophilic. Therefore, organic carbon pools (both living and non-living) are the primary uptake vectors for benthic invertebrates. Less hydrophobic COCs, such as low molecular weight PAHs and metals, are not as strongly associated with organic pools, and exhibit more complex partitioning behavior. The COCs identified for benthic invertebrates exhibit both direct (i.e., contact with contaminated source media) and indirect (i.e., food web bioaccumulation) pathways.

8 The conceptual model presented in Figure 3.1-1 illustrates the exposure pathways for benthic 9 invertebrates in the PSA. The benthic invertebrate ERA considered organisms that reside in, or 10 are in direct contact with, Housatonic River sediment. For sediment invertebrates, the dominant 11 abiotic exposure media were sediment (solid phase and/or porewater) and surface water. 12 Concentrations of COCs in tissues of benthic invertebrates were also considered. Tissue data 13 provide an organism-based measure of bioavailability, and provide an additional line of evidence 14 to consider along with the conventional Sediment Quality Triad approach (synoptic measurement 15 of sediment chemistry, toxicity, and invertebrate communities).

16 The problem formulation (Section 2) identified species used in toxicity tests as surrogates for the 17 Housatonic River freshwater benthic community (i.e., *Chironomus tentans*, *Hyalella azteca*, 18 *Lumbriculus variegatus*, *Daphnia magna*, *Ceriodaphnia dubia*). Both the status of sensitive 19 indicator taxa and the overall community composition are considered indicative of the condition 20 and productivity of the benthic community.

The assessment endpoints that are the subject of this section are benthic invertebrate community structure, survival, growth, and reproduction. The measurement endpoints used to evaluate the assessment endpoint are presented below.

1	Measurement Endpoints for Benthic Invertebrates
2 3 4 5 6	 Determine, based on field studies, the extent to which reductions in benthic community abundance, biomass, species richness, and other community metrics have occurred, including species-specific indications of adverse effects. Determine if these changes can be related to exposure to PCBs or other COCs in the sediment of the river.
7 8 9 10	 Determine, based on in situ and laboratory toxicity studies performed for this ERA, the extent to which the exposure to PCBs and other COCs in the river sediment may result in adverse impacts to survival, growth, and/or reproduction of representative benthic taxa.
11 12 13	 Determine, based on effects information from the literature, the extent to which the concentrations of PCBs and other COCs in Housatonic River sediment and/or water may cause adverse impacts to the benthic community.
14 15 16 17	 Determine, based on a combination of in situ tissue measurements and literature effects values, the extent to which the concentrations of PCBs bioaccumulated in the tissues of the benthic organisms will cause effects to survival, growth, or reproduction.

The approach used to characterize risks to benthic invertebrates was based upon evaluation of numerous data sources, many of which support the Sediment Quality Triad approach. The Sediment Quality Triad approach to assessment of sediment quality is based on synoptic measurement of sediment chemistry, site-specific sediment toxicity, and benthic invertebrate community structure (Long and Chapman 1985; Chapman 1996).

23	Sediment Quality Triad Components Investigated in this Study
24	Standard Triad Components:
25 26 27	 Site-specific toxicity studies (laboratory and in situ); multiple species (<i>Hyalella magna</i>, <i>Chironomus tentans</i>, <i>Daphnia magna</i>, <i>Lumbriculus variegatus</i>), multiple test durations (48-hour, 7-day, 10-day, 42-day).
28	 Benthic macroinvertebrate community structure.
29	 Abiotic media chemistry (sediment, overlying water, and porewater).
30	Additional Components:
31 32 33	 Bioaccumulation assessment (chemistry in resident invertebrates [predators and shredders]; 7-day bioaccumulation assessment to deposit-feeding invertebrates in laboratory [oligochaete]).
34	 Toxicity Identification Evaluations (TIEs).
35	

36 A summary of the studies conducted and their linkage to the ERA is provided in Figure 3.1-5. In

37 addition to the targeted Sediment Quality Triad studies summarized above, the ERA considered

- 1 broader site characterization information, such as PCB concentrations in surface sediment (0 to
- 2 6 inches [15 cm]), ecological characterization studies (Appendix A), and literature information
- 3 on the potential toxicity of COCs.



Figure 3.1-5 Summary of Studies Conducted in Conjunction with Ecological Risk
 Assessment for Benthic Invertebrates, and Linkage to ERA

1 3.2 EXPOSURE ASSESSMENT

2 The exposure assessment estimates the exposure of benthic invertebrates to tPCBs and other 3 COCs in the Housatonic River PSA (Figure 3.1-2). The exposure assessment for benthic 4 invertebrates also considered the influences of habitat and sediment substrate, and assessed the 5 degree to which exposure data can be appropriately linked to biological effects studies. Unlike 6 higher trophic level receptors (Sections 6 - 11), a complex exposure model was not required. 7 Instead, exposures were assessed as either the COC concentrations in abiotic site media (i.e., 8 sediment, water), or as the tissue body burdens that represent integrated exposure from all 9 sources.

To match exposure data with effects-based measures, many of the data considered were derived from sampling conducted in association with the 13 benthic community sampling locations and/or the 7 sediment toxicity locations (Figure 3.2-1). For the purposes of this report, the 13 benthic macroinvertebrate sampling stations are referenced using the "simplified IDs" presented below, rather than the more complex field sampling IDs.

15 16

17

18

Summary of IDs for 13 Benthic Invertebrate Sampling Locations

- Upstream Reference Locations: A1, A2, A3 (arranged north to south).
- Exposed Locations on Housatonic River: 1 to 9 (arranged north to south).
- Downstream (watershed) Reference Location: R4 (Threemile Pond).
- 19

Exposure assessments were also undertaken for abiotic media at a broader scale than the stations shown in Figure 3.2-1, such that findings from the Sediment Quality Triad study could be extrapolated to the larger PSA and Rest of River areas. These extrapolations relied on the development of exposure-response relationships from the Sediment Quality Triad stations.

24 **3.2.1** Selection of COCs for Benthic Invertebrates

The contaminants initially considered in the benthic invertebrate exposure assessment (COPCs) were identified in the Pre-ERA (Appendix B). The invertebrate Pre-ERA included screening on a reach-by-reach basis and subdivision of COPCs by major hydrological/geomorphological category.



| O:\gepitt\aprs\macro_int.apr | layout - mac locs 3.2-1 | o:\gepitt\epsfiles\plots\in\mac_int_3-2-1.eps | 11:42 AM, 7/8/2003 |

The sediment COCs identified in "main channel and aggrading bar" sediment are presented below. PCBs were identified as sediment COCs in all PSA reaches. PAHs were retained throughout the PSA, although the number of individual PAH compounds screened was greater for Reaches 5A and 5C, relative to the other reaches. Dibenzofuran was retained only for Reach 5A. Metals were retained in Reaches 5C and 6 only.

6 Contaminants of Concern for Benthic Invertebrates 7 Chlorinated organic compounds – tPCBs, dioxins/furans. 8 Metals - antimony, barium, cadmium, chromium, copper, lead, mercury, silver, 9 and tin. 10 Semivolatile organic compounds (SVOCs) - dibenzofuran. 11 PAHs - numerous individual PAH compounds, including low- and high-molecular . 12 weight PAHs.

13

Surface water COPCs identified in the pre-ERA (Appendix B) included dioxins/furans, PCBs, and silver. Therefore, the water chemistry screening did not result in any additional contaminants that were not already considered as sediment COCs for invertebrates.

17 Several additional contaminants (mainly pesticides) were determined in the pre-ERA to be below 18 detection limits in sediment, but had detection limits that exceeded screening benchmarks. An 19 examination of the detection limits for invertebrate tissues indicated that concentrations for most 20 of the pesticides of concern were below detection limits. Tissue effects concentration data were 21 available for many pesticides, and screening of maximum observed tissue concentrations showed 22 that even the contaminants detected in invertebrate tissues were below concentrations shown to 23 be of ecological concern. On this basis, and considering that some pesticide detections may be 24 attributable to laboratory interference artifacts, the entire suite of organochlorine pesticides was 25 eliminated from further consideration in the invertebrate portion of the ERA.

26 **3.2.2 Types of Exposure Data**

The approach used to characterize exposure to benthic invertebrates was based upon evaluation of numerous data sources, including both sediment and water column COC concentrations and invertebrate tissue COC concentrations (Figure 3.1-2). Many of the data applicable to benthic
 endpoints relate to the Sediment Quality Triad.

3 Use of the Sediment Quality Triad approach requires selection of exposure data to achieve 4 maximum correspondence between exposure and effects endpoints, while also recognizing the 5 need to address spatial and temporal variability in the data, and inherent limitations in field 6 sampling. Concentration-response relationships were investigated using both: (a) a combined 7 data set, screened using the criteria listed below; and (b) the single "most synoptic" chemistry 8 value paired with each toxicity endpoint. Section 3 and Appendix D present the results of the 9 first method, and Attachment D.5 presents an analysis using the latter method, for comparison. 10 In most cases, the two approaches yielded similar results, demonstrating that the interpretations 11 in the risk assessment were not an artifact of the data processing methods.

12	Criteria for Selection of Exposure Data Linked to Toxicity Endpoints
13	 Sediment data collected within a radius of 5 m from the benthic biota sampling
14	location.
15	 Exposure data collected between March and October 1999, the period over
16	which all site-specific effects measurements were performed.
17	 Sediment samples collected from within the top 6 inches (15 cm) of sediment,
18	and included at least the top 2 inches (5 cm).
19	 In merging data sets from multiple studies, exposure data were combined (using
20	a median of individual data) for all points collected on the same calendar day at
21	the same location. This was done to avoid bias resulting from a higher number
22	of samples or replicates on one day, which would have potentially obscured
23	temporal variability.

24

Because the sediment PCB concentration data were lognormally distributed, the median was chosen as the measure of central tendency for use in concentration-response assessments. Unlike wildlife ERA components, the benthos were assumed to be relatively sessile, and therefore, integrating exposures over a home range (i.e., using arithmetic mean to integrate concentrations as they vary over space) was not appropriate.

1 3.2.3 Habitat Characterization

To provide the foundation for the risk characterization, the preliminary results of physical and ecological investigations were used to define appropriate "clustering" of benthic stations for the exposure assessment. In this case, clustering refers to the grouping of stations of similar properties (e.g., sediment types), to discriminate between substrate-related responses and those attributable to contaminants.

Rationale for "Clustering" of Benthic Sampling Stations in ERA
Clustering was relevant for the exposure assessment for three primary reasons:
 To determine the appropriateness of reference stations for making statistical comparisons to exposed stations.
 To provide a means of separating physical and ecological "regimes" in a manner consistent with both the exposure and effects assessment.
 To provide a tool to assess if there are other influences that were not well characterized.

16 Two approaches were used to evaluate the benthic sampling stations in terms of their gross 17 habitat characteristics:

- Physical substrate variables were evaluated within and among benthic sampling stations downstream of identified contaminant sources (Figure 3.2-2) to identify significant break points in physical habitat features (e.g., substrate type, organic carbon content, sediment particle size distribution).
- Aquatic habitat was evaluated to identify broad biological regimes within the benthic
 sampling locations. Factors such as surrounding vegetation, macrophyte coverage,
 and cluster analysis of benthic invertebrate assemblages were used to identify
 changes in macro-habitats.
- Considering the above, a clear change in substrate and habitat type was observed between Stations 5 and 6, which is coincident with the transition in river regime from Reach 5A to Reach 5B, and the location of the Pittsfield wastewater treatment plant (WWTP) outfall. This shift in habitat (e.g., particle size distributions and organic carbon content of sediment) was used to identify appropriate statistical contrasts in the ERA.



1 2 3	Grouping of Benthic Sampling Stations Based on Habitat Benthic sampling stations in Figure 3.2-1 were assigned to one of the following categories:
4	 "Coarser" Reference Locations (C/R) – Low total organic carbon (TOC) (typically
5	less than 1%), sandy sediment found either upstream of influence from the GE
6	facility or on the West Branch. Three locations (A1, A2, A3).
7	 "Coarser" Contaminated Locations (C/C) – Low TOC (typically less than 1%),
8	sandy sediment found between the confluence and the Pittsfield WWTP. Five
9	locations (1, 2, 3, 4, 5).
10	 "Finer" Reference Locations (F/R) – High TOC, silty sediment found outside the
11	PSA at Threemile Pond (Location R4).
12	 "Finer" Contaminated Locations (F/C) – High TOC (typically a few percent or
13	greater), silty sediment found downstream of the Pittsfield WWTP. Four
14	locations (6, 7, 8, 9).

15 **3.2.4 Assessment of Sediment Chemistry**

16 3.2.4.1 Sources of Sediment Data

17 There are multiple sources of sediment data, each with a varying degree of correspondence to 18 various effects metrics. The use of specific data sets depended on the ERA goal; for example, 19 "discrete sampling" data not associated with benthic sampling stations were used to extrapolate 20 risk estimates, but were not used for development of concentration-response relationships.



1 3.2.4.2 Distribution and Concentrations of PCBs

2 **3.2.4.2.1** Benthic Community Grabs

Individual replicate concentrations of tPCBs for each benthic sampling station are presented on a logarithmic scale in Figure 3.2-3. The data indicate highly elevated tPCB concentrations in the C/C sites, with median values of approximately 5 to 25 mg/kg. In the F/C sites, median PCB concentrations were significantly lower (pooled variance t-test; p < 0.001). There was considerable variability in tPCB concentrations between replicates at most stations (Figure 3.2-3), indicative of small-scale variability in PCB concentrations.

9 **3.2.4.2.2** Toxicity Test Samples

10 Concentrations of tPCBs were measured in Housatonic River sediment in conjunction with 11 laboratory and in situ toxicity and bioaccumulation tests conducted between May and July 1999 12 (EVS 2003). As with the benthic community grab samples, tPCB concentrations were quite 13 variable within stations across the four toxicity sampling events (Figure 3.2-4).

These results suggest that PCB exposure data from the toxicity testing data sets should not be extrapolated to benthic community composition endpoints, and also indicate that the variability in the chemistry data associated with the toxicity program must be considered when deriving concentration-response relationships. Because sediment samples were not replicated in individual toxicity sampling events, data from all relevant sampling events (as defined in Section 3.2.2) were included in the development of concentration-response relationships for toxicity endpoints.

21 **3.2.4.2.3** Broad Scale Sediment Characterization

Figure 3.2-5 depicts the spatial distribution of tPCB concentrations within the PSA. The data indicate that median PCB concentrations are highest in the upstream reaches of the PSA, and decrease with distance from the GE facility. The median concentrations are lowest just downstream of the WWTP, but increase moving farther downstream to Woods Pond. For areas



- Mean of 12 replicates
- Replicate concentration, assuming non-detected values equal to half MDL

Figure 3.2-3 Concentrations of tPCBs in Sediment by Sampling Location for Individual Benthic Community Grab Samples, and Associated Measures of Central Tendency

5




Figure 3.2-5 Medians and Quartiles of PCB and TOC in the Housatonic River PSA, Subdivided by River Reach and 0.25 Mile Subreaches

downstream of Woods Pond, the tPCB concentrations are lower than in the PSA (by
 approximately an order of magnitude). Sediment tPCB concentrations downstream of the
 Connecticut border were generally below 1 mg/kg, reflecting the general trend of decreasing
 concentration with distance downstream.

5 **3.2.4.3** Distribution and Concentration of Other COCs

Fewer sediment data are available for other COCs; however, there were sufficient data to
characterize patterns of concentrations throughout the PSA (Appendix D; Figures D.2-16
through D.2-28). A brief summary of spatial trends for these COCs is provided below.

9	Summary of Trends in Other COCs in Sediment Data
10	 Dioxin/furan concentrations are elevated at downstream fine-grained locations
11	relative to upstream and/or reference locations, and are positively correlated with
12	tPCB concentrations.
13	 Dibenzofuran concentrations do not occur in a pronounced spatial pattern
14	throughout the PSA; most concentrations were in the 0.1 to 1.0 mg/kg range.
15	This COC was eliminated from the ERA (rationale provided in Appendix D).
16	 Total PAH concentrations were highly variable, both spatially and between
17	sampling events. The median concentrations were greatest near the urbanized
18	areas of the Housatonic River watershed, and were lowest at Station A1 and at
19	the Woods Pond headwaters. The broad spatial pattern of PAH concentrations
20	in the toxicity locations was opposite to that observed for PCBs.
21	 Metals concentrations were typically lower at upstream sites relative to the fine-
22	grained sediment found downstream. Different metals had generally similar
23	concentration patterns. Metals concentrations were significantly correlated
24	(p <0.05) with TOC concentrations.

25

26 **3.2.5 Tissue Chemistry Assessment**

Benthic tissue data are less abundant than data for abiotic media, and generally did not include
replication, due to limited volumes of tissue available for chemical analysis. Nevertheless, the
available data provide a measure of the site-specific bioavailability of the COCs.

30 Figure 3.2-6 presents the distribution of tPCB concentrations by sampling location and tissue

- 31 type, for samples collected at the benthic sampling stations. Most reference samples had tPCB
- 32



tissue concentrations below 1.0 mg/kg. In contrast, contaminated locations had elevated
 concentrations ranging from 2 to 48 mg/kg.

Sources of Benthic Invertebrate Tissue Chemistry Data
 Analysis of composite samples of "predators" and "shredders," respectively, conducted in 1999 by EPA. Each tissue sample was analyzed for lipids and a number of organic contaminants, including PCBs (as congeners, as Aroclors, and as tPCBs), dioxins/furans, and pesticides.

- Data from the 7-day in situ bioaccumulation study (EVS 2003) conducted with the oligochaete worm *Lumbriculus variegatus* at 6 sampling stations.
- Academy of Natural Sciences of Philadelphia long-term historical tissue PCB monitoring of dobsonfly, caddisfly, and stonefly nymphs/larvae collected downstream of the PSA near Cornwall, CT (BBL & QEA 2003).
- 13

3

4

5

6

7

8

9

10

11 12

14 Concentrations of other COCs, such as Appendix IX pesticides, are also available in the benthic 15 tissue chemistry data set. However, because pesticides were screened out of the benthic ERA 16 (based on detection limit considerations, conservative tissue concentration screening, and 17 potential for artificially high laboratory values due to PCB interference), these contaminants 18 were not considered further. Although PAHs and metals were retained as COCs, the tissue 19 analyses did not include these parameters due to the lack the sufficient sample volume for 20 analysis.

21 **3.2.6 Surface Water Chemistry Assessment**

Surface water chemistry data have limited application to the benthic ERA due to the uncertainty in extrapolating from water chemistry to effects in sediment-dwelling biota. Because of this uncertainty, the only data considered relevant were those collected synoptic with effects measurements. Unfiltered overlying site water was collected in conjunction with the 7-day in situ toxicity testing (EVS 2003) and evaluated for tPCBs, PCB congeners, PAHs, pesticides, and metals. Only tPCBs were measured for the 48-hour and 10-day exposures.

10

Summary of Water Chemistry COC Concentrations

- Concentrations of tPCBs at upstream reference stations were less than 10 nanograms per liter (ng/L); concentrations at contaminated stations ranged from approximately 100 ng/L to 300 ng/L.
- Concentrations of furans matched the spatial patterns in tPCBs, with total detected furans well below 10 picograms per liter (pg/L) at upstream reference stations, and concentrations of 60 to 120 pg/L at contaminated stations.
- No dibenzo-p-dioxins or silver were detected in the samples collected in conjunction with the 7-day in situ tests.

MK01|O:\20123001.096\ERA_PB\ERA_PB_3.DOC

1 3.3 EFFECTS ASSESSMENT

The effects assessment for benthic invertebrates (Figure 3.1-3) emphasizes the site-specific biological investigations performed at the 13 benthic sampling stations because these studies provided direct indications of the bioavailability, toxicity, and effects of site COCs. Both toxicity assessments (i.e., laboratory toxicity, in situ toxicity, and TIE) and community evaluations (i.e., benthic macroinvertebrate community composition) were compared to appropriate field references to determine whether the exposed sites on the Housatonic River exhibited biological impairment.

9 The effects assessment also provides an overview of the literature on the effects of tPCBs and 10 other COCs to survival, growth, and reproduction of benthic invertebrates. Studies were 11 screened and used to derive the most appropriate effects metrics for tissue, sediment, and water. 12 In recognition of the uncertainty inherent in threshold effects concentrations for these media, 13 ranges of benchmarks were derived instead of relying on single effects thresholds.

Detailed evaluations of concentration-response relationships are discussed immediately following the broad discussion of inter-station differences (i.e., differences between exposure location responses and control and/or reference location responses). This corresponds to Sections 3.3.2 and 3.3.7 for sediment toxicity and benthic community structure, respectively. The effects thresholds derived therein are carried forward into the Risk Characterization, and are used as maximum acceptable threshold concentrations (MATCs) for extrapolation to areas downstream of the PSA.

21 3.3.1 Sediment Toxicity

22 3.3.1.1 Methods

Wright State University (WSU) conducted site-specific toxicity testing of Housatonic River sediment (EVS 2003). Test protocols, study methods, and other detailed documentation for the Housatonic River sediment toxicity testing program are presented in EVS (2003) and in the SIWP (WESTON 2000). Seven stations were sampled for in situ and laboratory toxicity analyses. Of these, six were within the group of 13 locations where the benthic
 macroinvertebrate sampling was conducted (Figure 3.2-1).

3	Toxicity Test Methods
4	Laboratory testing (EPA 2000 protocols)
5 6 7	 Chronic 42-day bulk sediment test using a freshwater amphipod (<i>Hyalella azteca</i>). Duration and endpoints were 28 days, 35 days, and 42 days for survival; 28 days and 42 days for growth; 35 days and 42 days for reproduction.
8 9 10	 Chronic 43-day sediment test using a freshwater midge (<i>Chironomus tentans</i>). Duration and endpoints were 20 days for survival and growth, and 23 days to 43 days for mortality and emergence.
11	In situ testing (including both sediment and water-only exposures):
12 13	 48-hour toxicity test using a freshwater cladoceran (<i>Daphnia magna</i>, 48 hours old). Endpoint: survival.
14 15	 48-hour and 10-day toxicity test using a freshwater midge (<i>Chironomus tentans</i>, 8 to 12 days post-hatch). Endpoint: survival.
16 17	 48-hour and 7-day toxicity test using a freshwater oligochaete worm (<i>Lumbriculus variegatus</i>, multiple ages). Endpoint: survival and tissue bioaccumulation.
18 19	 48-hour and 10-day toxicity test using a freshwater amphipod (<i>Hyalella azteca</i>, 7 to 14 days old). Endpoint: survival.

20

The organisms selected are both environmentally relevant to the Housatonic River (e.g., chironomid and oligochaete species were present in high numbers in PSA sediment) and have a large toxicological database demonstrating their relative sensitivity to the COCs. The selected species inhabit sediment during the life stages tested, remain relatively immobile, and have a high potential for exposure. The test organisms selected are tolerant to a broad range of sediment physicochemical characteristics (EPA 2000).

Sediment grain size is a significant variable that can affect benthic organisms, both in the field and in some laboratory toxicity studies. Because the field reference sediment (Stations A1 and A3) both represented coarse-grained sediment, it was important to assess the potential for confounding effects of particle sizes in toxicity test treatments with fine-grained sediment. To this end, a literature review was conducted to document the sensitivity of the test organisms to changes in particle size distributions (Attachment D.4). The review indicated that the indicator species chosen for toxicity testing are quite tolerant of a broad range of particle sizes (hence their selection as measurement endpoints). In addition, the in situ tests were conducted using sediment exposure chambers placed immediately on top of the sediment. The organisms were separated from the sediment by a fine-mesh screen and therefore not affected by the sediment grain size distribution. Therefore, comparison of all contaminated sediment treatments to the reference sediment A1 and A3 was appropriate.

6 3.3.1.2 Results

Table 3.3-1 presents the results of statistical tests of significance (comparisons to negative controls and reference stations) for most toxicity test endpoints. Toxicological responses for each test type and treatment are also presented graphically (Figure 3.3-1 to Figure 3.3-11). PCB concentrations are presented in two ways, based on the two data processing approaches discussed in the exposure assessment:

- 12 13
- The values in bold represent the median of all spatially and temporally relevant PCB concentrations at each station; and
- 14 15
- The values in italics represent the single PCB concentration measurement taken closest to the effects endpoint (i.e., most synoptic concentration).

The station-by-station summary assessment is presented in Tables 3.3-2 and 3.3-3 (in situ and laboratory endpoints, respectively). Each endpoint was rated as exhibiting negligible, moderate, strong, or very strong evidence for toxicological effects to freshwater organisms, based on the effect magnitude observed relative to the negative control(s). The degree of confidence in the assessment of potential for ecological risk is in large part a function of the degree of concordance observed among endpoints.

1	Summary of Site-Specific Toxicity Outcomes
2	In situ exposures (acute mortality endpoints):
3	 Negligible toxicity at both reference locations (Stations A1, A3) and the most
4	upstream "contaminated" location with the lowest PCB concentration (Station 4).
5	 Toxicity was evident in multiple tests for the remaining three contaminated
6	locations (Stations 5, 7, 8), with the magnitude of response generally greatest at
7	the two locations with the highest PCB concentrations.
8	 Modest effects were observed in some water-column exposures, but most
9	pronounced effects were observed in the sediment-exposure treatments.
10	Laboratory exposures (chronic lethal and sublethal endpoints):
11	 Overall frequency of toxic responses greater than for acute in situ exposures.
12	 Minor indications of reduced endpoint performance (relative to negative control)
13	in both reference locations (Stations A1, A3). These responses consisted mainly
14	of marginal reductions in <i>Hyalella</i> reproduction and <i>Chironomus</i> endpoints.
15	 Large magnitude adverse responses were much greater at all four contaminated
16	locations (e.g., <i>Chironomus</i> toxicity, <i>Hyalella</i> mortality, and reproductive effects).
17	 Conclusion of a "high" toxicity rating for all four contaminated locations.

Table 3.3-1

1
2
3
4

Results of Pairwise Statistical Tests Comparing Exposed Stations to Negative Control (T-Ctrl) and Reference (A1, A3) Sediment (Water-Only Exposures Excluded)

Station		4			5			7			8A			8	
Pairwise Comparison	T-Ctrl	A1	A3												
28-d Hyalella survival	Yes	Yes	Yes	N/A	N/A	N/A	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
35-d <i>Hyalella</i> survival	Yes	Yes	Yes	N/A	N/A	N/A	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
42-d <i>Hyalella</i> survival	Yes	No	Yes	N/A	N/A	N/A	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
28-d Hyalella dry weight	No	No	No	N/A	N/A	N/A	NC	NC	NC	No	No	No	No	No	No
42-d Hyalella dry weight	No	No	No	N/A	N/A	N/A	NC	NC	NC	No	No	No	No	No	No
42-d <i>Hyalella</i> young per female	Yes	No	No	N/A	N/A	N/A	NC	NC	NC	Yes	No	Yes	Yes	Yes	Yes
42-d Hyalella mean young	Yes	No	Yes	N/A	N/A	N/A	NC	NC	NC	Yes	Yes	Yes	Yes	Yes	Yes
20-d Chironomus survival	Yes	Yes	Yes	N/A	N/A	N/A	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
43-d <i>Chironomus</i> emergence	Yes	Yes	Yes	N/A	N/A	N/A	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
20-d Chironomus dry weight	Yes	Yes	Yes	N/A	N/A	N/A	NC	NC	NC	NC	NC	NC	Yes	Yes	Yes
20-d <i>Chironomus</i> ash-free dry weight	Yes	Yes	Yes	N/A	N/A	N/A	NC	NC	NC	NC	NC	NC	Yes	Yes	Yes
48-h <i>Hyalella</i> survival (sediment)	No	No	No	No	No	No	No	Yes	No	N/A	N/A	N/A	No	No	No
10-d <i>Hyalella</i> survival (sediment)	No	No	No	No	No	No	No	Yes	Yes	N/A	N/A	N/A	No	Yes	Yes
48-h <i>Chironomus</i> survival (sediment)	No	No	No	No	No	No	No	No	No	N/A	N/A	N/A	No	No	No

Table 3.3-1

Results of Pairwise Statistical Tests Comparing Exposed Stations to Negative Control (T-Ctrl) and Reference (A1, A3) Sediment (Water-Only Exposures Excluded) (Continued)

Station	4		5		7			8A			8				
Pairwise Comparison	T-Ctrl	A1	A3	T-Ctrl	A1	A3	T-Ctrl	A1	A3	T-Ctrl	A1	A3	T-Ctrl	A1	A3
10-d <i>Chironomus</i> survival (sediment)	No	No	No	No	No	No	Yes	Yes	Yes	N/A	N/A	N/A	Yes	Yes	Yes
48-h <i>Daphnia</i> survival (sediment)	No	Yes	No	No	No	No	Yes	Yes	Yes	N/A	N/A	N/A	Yes	Yes	Yes
48-h <i>Lumbriculus</i> survival (sediment)	No	No	No	No	No	No	No	No	No	N/A	N/A	N/A	No	No	No

Yes = Statistically different at alpha = 0.05

2 No = Not statistically different at alpha = 0.05

3 NC = Sublethal endpoint not calculable (due to zero survival in treatment)

N/A = Not applicable; not tested for endpoint/station combination

4 5





3	Notes:	Labels represent tPCB concentration (mg/kg) in sediment.
4 5		Value in bold represents median tPCB concentration (from all measurements made within 5 meters of station in 1999; see Appendix D).
6 7		Value in italics represents "most synoptic" tPCB concentration; single concentration measured closest to toxicity test in space/time.
8 9	Figure	3.3-2 Growth of <i>Hyalella azteca</i> in Chronic Laboratory Toxicity Tests, at Two Time Periods (28 days, 42 days)
10		



■ 28-42 day mean young per female □ 28-42 day mean young (unstandardized)

3	Notes:	Labels represent tPCB concentration (mg/kg) in sediment.
4 5		Value in bold represents median tPCB concentration (from all measurements made within 5 meters of station in 1999; see Appendix D).
6 7		Value in italics represents "most synoptic" tPCB concentration; single concentration measured closest to toxicity test in space/time.
8		
9 10	Figur	e 3.3-3 Reproduction of <i>Hyalella azteca</i> in Chronic Laboratory Toxicity Tests, Based on Mean Number of Young (Days 28-42)

2





3

1

Notes:	Labels represent tPCB concentration (mg/kg) in sediment.

- 4 Value in bold represents median tPCB concentration (from all measurements made within 5 meters 5 of station in 1999; see Appendix D).
- 6 Value in italics represents "most synoptic" tPCB concentration; single concentration measured 7 closest to toxicity test in space/time.
- 8 T-Control, C-Control, and F-Control are negative laboratory controls ("Trout Farm", "Cellulose", 9 and "Florissant", respectively).

10

Figure 3.3-5 Growth Endpoints for *Chironomus tentans* in Chronic Laboratory Toxicity Test (20 days)





3 Notes: Labels represent tPCB concentration (mg/kg) in sediment.

4	Value in bold represents median tPCB concentration (from all measurements made within 5 meters
5	of station in 1999; see Appendix D).

Value in italics represents "most synoptic" tPCB concentration; single concentration measured closest to toxicity test in space/time.

Figure 3.3-7 Survival of *Hyalella azteca* in 10-day Low Flow In Situ Toxicity Tests Conducted 17-27 June 1999

10

6 7

2





Value in italics represents "most synoptic" tPCB concentration; single concentration measured closest to toxicity test in space/time.

9 Figure 3.3-9 Survival of *Chironomus tentans* in 10-day Low Flow In Situ
 10 Toxicity Tests Conducted 17-27 June 1999







Table 3.3-2

L. Varie-C. tentans Median H. azteca D. magna H. azteca C. tentans L. variegatus Bulk 48-hour 48-hour 48-hour 10-dav 10-dav gatus **Sampling Station** 48-hour Overall Sediment Survival Survival Survival Survival Survival Residue (ID, Location, WESTON ID) Survival Assessment (mg/kg [PCB] (Water -(Water – (Water -(Water -(Water -(Water -(mg/kg) Sediment) Sediment) Sediment) Sediment) Sediment) lipid) Sediment) Dalton Reference 011 0.018 0-0 0-0 0-0 0-0 0-0 0-0 5.3 Ο A1 Lower West A3 398 0-0 0-0 0-0 0-0 0-0 0-0 Ο 0.28 20.7 **Branch Reference** 1.5 miles below 019 0-0 0-0 0-0 0-0 0-0 0-0 Ο 4 5.9 232.6 Holmes Road Near WWTP 0-0 5 428 O - O 0-0 0-0 O - O 0-0 0 7.3 380.6 Discharge 2 miles below O - ●[‡] 7 0 - • 0-0 0-0 0 - • O - ●‡ 389 54 128.3 • New Lenox Road $\frac{1}{2}$ mile above 8 O - •‡ O - •‡ O - •‡ 0-0 0-0 0-0 031 77 314.5 Woods Pond

In Situ Evaluation of Toxicity in Housatonic River Sediment (Station-by-Station Assessment)

O = Negligible to low toxicity: less than 20% effect size relative to negative control. Overall assessment – negligible indication of ecological risk.

• = Moderate toxicity: 20 to 50% effect size relative to negative control. Overall assessment – ecological effects possible, but not conclusive.

• = High toxicity; greater than 50% effect size relative to negative control. Overall assessment – strong indication of potential ecological effects.

• \ddagger = Very strong toxic response for individual endpoint; greater than 90% effect size relative to negative control.

7 8

4

5

6

Table 3.3-3

1 2 3

Laboratory Evaluation of Toxicity in Housatonic River Sediment (Station-by-Station Assessment)

Sampling Station (ID, Location, WESTON ID)			Median Bulk Sediment [PCB] (mg/kg)	H. azteca Survival (28 day – 35 day – 42 day)	H. azteca 28-42 day Reproduction (young/female)	H. azteca Dry weight (28-day – 42 day)	<i>C. tentans</i> Survival	<i>C. tentans</i> Growth (Total Wt – Ash Free)	<i>C. tentans</i> Emergence	Overall Assessment
A1	Dalton Reference	011	0.018	0-0-0	0	0 - 0	0	0 - •	0	0
A3	Lower West Branch Reference	398	0.28	0-0-0	ο	0 - 0	0	0-0	0	0
4	1.5 miles Below Holmes Road	019	5.9	0 - 0 - 0	•	0 - 0	•‡	●‡ - ●‡	•‡	•
7	2 miles Below New Lenox Road	389	54	● [‡] - N/A - N/A	N/A	N/A - N/A	•‡	N/A	•‡	•
8	¹ / ₂ mile above Woods Pond	031	77	• - • - •	•	0 - 0	•‡	●‡ - ●‡	•‡	•
8 A	¹ / ₂ mile above Woods Pond	023	4.6	• - • - •	•‡	0 - 0	•‡	N/A	•‡	•

4 O = Negligible to low toxicity: less than 20% effect size relative to negative controls. Overall assessment – negligible indication of ecological risk.

5 **O** = Moderate toxicity: 20 to 50% effect size relative to negative controls. Overall assessment – ecological effects possible, but not conclusive.

6 • = High toxicity; greater than 50% effect size relative to negative controls. Overall assessment – strong indication of potential ecological effects.

7 \bullet^{\ddagger} = Very strong toxic response for individual endpoint; greater than 90% effect size relative to negative control.

8 N/A = Sublethal endpoint not measured due to complete mortality in treatment.

1 A comparative approach (i.e., relative to reference) was also applied to help distinguish 2 background field reference responses from those observed at contaminated locations. In this 3 assessment, comparisons were made not to the negative control, but to the two upstream 4 reference locations (Stations A1, A3). Despite the modest toxicity observed in the laboratory 5 toxicity endpoints for Stations A1 and A3, the comparative assessment (Table 3.3-4) still 6 indicated a moderate to strong incremental toxicity associated with contaminated PSA sediment.

7 The three most downstream stations (7, 8, and 8A) had "high" ratings due to the consistency and 8 severity of toxicity observed for numerous endpoints.

9 3.3.2 Concentration-Response Analysis – Toxicity Test Endpoints

10 A statistical assessment was conducted to quantify the observed relationship between toxicity 11 test endpoints and COC concentrations measured concurrent with the biological tests. The 12 assessment focused on the relationship between PCBs and toxicity endpoints because other lines 13 of evidence indicated a high probability that PCBs were a causal agent for toxicity to benthic 14 invertebrates within the Housatonic River PSA.

15 This section emphasizes concentration-response using the "median" sediment PCB exposure 16 concentration at each station. An alternative analysis, using only the "most synoptic" exposure 17 concentration is presented in Attachment D.5. Generally, the two approaches yield comparable 18 results (i.e., most endpoints within a factor of 2); the "median" analysis yielded effects 19 thresholds that were slightly lower than the other method.

26

Methods for Evaluating C	concentration-Response for Toxicity Data
la dividual Ea da sint An shusis	E a de taxis ita an de sint constituente d

- Individual Endpoint Analysis Each toxicity endpoint was investigated individually using conventional descriptive statistics that related degree of effect to PCB concentrations (e.g., LC₅₀, IC₂₀, NOAEL, LOAEL).
- Combined Endpoint Analysis The toxicological endpoints were integrated using a general linear modeling approach to identify similarities and differences in concentration-response relationships across species and endpoints.

Table 3.3-4

Evaluation of Lines of Evidence for Housatonic River Sediment Toxicity, Relative to Reference Responses

		Chronic Laboratory Endpoints (20 day, 42 day)		Acute In Situ Endpoints (48 hours, 10 days)					
Sampling Station (ID, Location, WESTON ID)		<i>H. azteca</i> Laboratory (Survival, Growth, Reproduction)	<i>C. tentans</i> Laboratory (Survival, Emergence, Growth)	<i>H. azteca</i> In situ Survival (Water, Sediment)	<i>C. tentans</i> In situ Survival (Water, Sediment)	D. magna In situ Survival (Water, Sediment)	<i>L. variegatus</i> In situ Survival (Water, Sediment)	Overall Assessment	
4	1.5 miles below Holmes Road	019	0 - 0 - 0	• - • - •	0 - 0	0 - 0	0 - 0	0 - 0	0
5	Near WWTP Discharge	428	NA - NA - NA	NA - NA - NA	0 - 0	0 - 0	0 - 0	0 - 0	0
7	2 miles below New Lenox Road	389	• - NA - NA	•-•-	0 - •	○ - ●	0 - ●	0 - 0	•
8A	¹ / ₂ mile above Woods Pond	023	•-0-•	• - • - •	NA - NA	NA - NA	NA - NA	NA - NA	•
8	¹ / ₂ mile above Woods Pond	031	•-0-•	• - • - •	0 - •	0 - ●	0 - •	0 - 0	•

O = Negligible to low toxicity: less than 20% effect size relative to upstream background (A1, A3). Overall assessment – negligible indication of ecological risk.

 \mathbf{O} = Moderate toxicity: 20 to 50% effect size relative to upstream background (A1, A3). Overall assessment – ecological effects possible, but not conclusive.

• = High toxicity; greater than 50% effect size relative to upstream background (A1, A3). Overall assessment – strong indication of ecological effects.

NA = Endpoint not measured due to complete mortality in treatment (Location 7), or samples not collected at station (Locations 5, 8A).

1 3.3.2.1 Approach 1: Calculation of Individual Toxicity Test Endpoints

Point estimates were calculated for each toxicity test, including LC_{20} and LC_{50} values for survival endpoints, and IC_{20} and IC_{50} values for sublethal response endpoints (e.g., growth, reproductive success). For each data set, test endpoints were calculated based on comparison to both negative controls and reference sediment (Stations A1, A3). Full statistics are presented in Appendix D; a graphical presentation of toxicity thresholds relative to reference Station A1 is depicted in Figure 3.3-12.

8 Although there are small differences in the toxicity threshold values calculated using different 9 statistical methods (i.e., choice of extrapolation model or choice of reference sediment), the data 10 indicate an increase in the frequency and magnitude of adverse biological responses with 11 increasing sediment tPCB concentration. The following ranges of tPCB concentrations and 12 associated responses were developed, based on comparisons of contaminated station responses to 13 reference stations:

- <3 mg/kg Some sensitive endpoints exhibited apparent responses, but the magnitude of responses was not large. These subtle responses were difficult to evaluate precisely due to statistical power limitations, caused in part by the high variability in some treatments.
- 3 to 10 mg/kg Numerous endpoints indicated ecologically significant responses, with many LC₅₀/EC₅₀ values falling in this range. Statistically significant responses were observed in most *Hyalella* and *Chironomus* life-cycle endpoints at 4.56 mg/kg.
- 10 to 30 mg/kg Nearly all toxicity endpoints indicated large (>50%) responses relative to reference stations. The only endpoints that did not exhibit large responses in this concentration range were either growth endpoints or were short-term (48-hour) tests and/or with tolerant species.
- >30 mg/kg The concentration-response analyses indicated that most survival and
 reproduction endpoints exhibited very large reductions at these concentrations, with
 complete mortality of some species.

27 Dose/response modeling was also conducted using individual chemistry values considered to be 28 "most synoptic" with the toxicity tests (Attachment D.5). These tests had results similar to those 29 presented above (i.e., most LC_{50}/EC_{50} values were in the 3 to 30 mg/kg tPCBs range).





1 Threshold effects concentrations were calculated using the individual endpoint data, in order to 2 allow derivation of site-specific hazard quotients in the Risk Characterization, and to serve as 3 MATCs for downstream risk extrapolations. To calculate threshold effects concentrations, the 4 average of values from the six most sensitive endpoints was calculated for both 50% effects and 5 20% effects levels. This approach was based on the rationale that thresholds should consider 6 multiple sensitive endpoints, but should not be based on the single most sensitive endpoint. The 7 50% effects level corresponds to major impacts, for which there is a high degree of confidence in 8 a significant biological impact. The 20% effects levels correspond to lower but potentially 9 biologically significant effects.

10 Calculations were performed for comparisons to negative control sediment and also to field 11 reference sediment. In general, comparisons to field references were preferred for derivation of 12 sediment MATC values, since field references account for physicochemical factors that may 13 mediate sediment toxicity.

14 Summary of 50% and 20% Effects Levels 15 Comparison to Negative Control - The mean of the lowest six 50% effects levels 16 was 1.3 mg/kg tPCB. The mean of the lowest six 20% effects levels was 0.1 mg/kg tPCB. 17 18 Comparison to Reference A1 – The mean of the lowest six 50% effects levels 19 was 3.5 mg/kg tPCB. The mean of the lowest six 20% effects levels was 0.9 20 mg/kg tPCB. 21 Comparison to Reference A3 – The mean of the lowest six 50% effects levels . 22 was 3.3 mg/kg tPCB. The mean of the lowest six 20% effects levels was 0.9 23 mg/kg tPCB. 24

25

Using the "median" exposure data, the 50% effect level for sensitive toxicity endpoints is approximately 3 mg/kg tPCB. The analysis conducted using "most synoptic" exposure data only (Attachment D.5) indicated that the 50% effect level for sensitive toxicity endpoints is approximately 6-7 mg/kg tPCB, and that the 20% effect level for sensitive toxicity endpoints is approximately 3 mg/kg tPCB. Based on this information, 3 mg/kg tPCB was selected as the sitespecific threshold for sediment tPCB.

1 3.3.2.2 Approach 2: General Linear Model of Concentration-Response

2 The assessment of individual endpoints is sensitive to test variability, which can mask broader 3 trends in toxicity of PCBs. Therefore, a supplemental approach was applied that combined the 4 toxicity results from various endpoints to identify the overall trend(s) in concentration-response 5 observed. The endpoints for all toxicity tests were standardized so that the response variables 6 were equivalent (i.e., responses represented the proportion of their control mean response). This 7 transformation of all endpoints to the relative performance proportion (RPP) values standardized 8 results from different toxicological endpoints to similar ranges and facilitated the search for a 9 single unified model among all endpoints. The results of the general linear modeling are 10 depicted in Figure 3.3-13. Overall, the linear modeling indicated that seven of eight toxicity 11 endpoints evaluated were significantly correlated with log-transformed PCB concentration. 12 Differences between acute endpoints and chronic endpoints were observed; these are likely 13 related to the greater sensitivity of chronic endpoints in toxicity tests. The modeling procedure 14 enabled the identification of threshold tPCB concentrations in sediment. These results are in 15 agreement with the summary of individual test endpoints provided in Section 3.3.2.1. In 16 summary, sediment tPCB concentrations above 3 mg/kg indicate significant adverse effects for 17 sensitive (chronic) endpoints, and tPCB concentrations in the 10 to 30 mg/kg tPCBs range may 18 result in acute mortality to multiple organisms.

Attachment D.5 presents the results of the linear modeling using only the "most synoptic" exposure data. Results were qualitatively similar to those described above; the analysis indicated that the threshold for manifestation of tPCB effects is likely greater than 1 mg/kg and less than 10 mg/kg. There is some uncertainty within this concentration range due to variations in exposure and effects data, with the frequency of adverse effects increasing toward the upper end of this range.

25 **3.3.2.3** Relationships of Effects with Other COCs

The data for other COCs were also evaluated qualitatively to assess whether the concentrations of these contaminants were likely to have confounded the results of the PCB concentrationresponse presented above. The spatial patterns in COC concentrations were compared against the pronounced gradient in toxicity.



Figure 3.3-13 Segmented Linear Regression Models Applied to Toxicity Data, Relating Relative Performance Proportion (RPP) to Bulk Sediment tPCB Concentrations (mg/kg)

1	Comparison of Other COC Trends to Toxicity Trends
2	 PAHs – Most PAH data show a trend of reduced PAH concentrations with
3	distance downstream that is the reverse of the observed trend for toxic
4	responses. Therefore, with the possible exception of Station 7, there is no
5	evidence that PAHs were a major contributor to the observed pattern of
6	sediment toxicity.
7	 Dioxins/Furans – These analytes exhibited a spatial trend similar to the trend in
8	toxicity. This is likely due to co-occurrence between PCBs and dioxins/furans in
9	environmental samples.
10	 Metals – Metals generally exhibited a pattern of increasing concentration with
11	distance downstream, which matched the pattern of toxic responses. However,
12	the trends in metals concentrations also followed the sediment TOC and particle
13	size distributions. Once metals concentrations are normalized to the substrate
14	differences (thus accounting for lower bioavailability in downstream areas), there
15	was no indication that metals concentrations were responsible for observed
16	effects. This was confirmed by the low hazard quotients (HQs) for these metals,
17	and the results of the TIE.

19 On the basis of the information presented above, other COCs (other than tPCBs) do not explain 20 the patterns of toxicity observed in the in situ toxicity tests. One possible exception is for 21 dioxins and furans, which correlate strongly with tPCBs (i.e., co-contaminants in PCB mixtures).

22 **3.3.3 Toxicity Identification Evaluations**

23 EVS (2003) conducted TIEs to broadly define the physical/chemical characteristics of the 24 contaminants causing observed toxic responses. TIEs were conducted using porewater from 25 Housatonic River sediment to which the daphnid (Ceriodaphnia dubia) was exposed for 48 26 hours. Several TIE treatments were initiated in late summer 1999, including baseline tests, 27 oxidant reduction addition tests, EDTA chelation addition, pH-adjusted filtration, pH-adjusted 28 aeration, and pH-adjusted C₁₈ solid phase extraction (SPE). None of the individual treatments 29 provided a definitive identification of toxic agent; however, integration of the results of various 30 treatments provides strong indications of the class of toxic agents.

31 Based on the TIE study, EVS (2003) concluded that non-polar organic compounds (most likely

32 PCBs) were responsible for the observed pattern of toxicity responses.

1 A more comprehensive Phase II or Phase III TIE would be required to make a definitive 2 conclusion. However, the indications of PCB toxicity in the Phase I TIE are consistent with the 3 large exceedances of sediment quality values (SQVs) and water quality guidelines for PCBs 4 observed in site media.

5	Rationale for Implication of Non-Polar Organic Compounds as Active			
6	Toxicants in TIE Treatments			
7	 Significant reduction in toxicity in the pH-adjusted/filtration treatments – Higher			
8	survival in the filtration test was attributed to organic colloids in the samples			
9	being filtered out and/or pH-mediated toxicity alteration of organic compounds			
10	(EVS 2003). Filterable compounds can include non-polar organics, such as			
11	PAHs, PCBs, and some metals.			
12	 Significant reduction in toxicity in the pH-adjusted C₁₈ SPE treatments – The			
13	results of these manipulations indicated that the filtration reduced the toxicity of			
14	the original samples. Therefore, this test implicated non-polar organics,			
15	pesticides, and/or some metals.			
16	 EDTA treatments – These treatments did not result in a reduction of toxicity.			
17	This provides evidence against metals as the dominant causal agent.			
18	 Sediment and porewater chemistry – PCB concentrations in TIE treatments were			
19	observed to be well above upper-bound sediment quality guidelines and water			
20	quality criteria applied to porewater. Conversely, PAH concentrations in these			
21	TIE treatments (EVS 2003) were below applicable criteria (i.e., Swartz [1999]			
22	sediment quality guidelines for total PAHs). Furthermore, the two samples			
23	demonstrated to be the most toxic in the initial 24-hour screening toxicity test had			
24	the highest sediment tPCB concentrations.			

25

26 **3.3.4** Tissue PCB Effects Thresholds

Data were compiled on PCB tissue concentrations associated with lethal or sublethal effects in aquatic invertebrates. The purpose was to estimate threshold tissue concentrations beyond which adverse effects might occur in Housatonic River benthos. The review focused on data for Aroclors 1260 and 1254, in addition to tPCBs. No studies conducted specifically for Aroclor 1260 were identified. Because tissue effects data were limited for these PCB metrics, both freshwater and marine/estuarine invertebrate species were considered in the review. To estimate PCB effects thresholds, the tissue effects data were ranked in order of increasing tissue concentration to illustrate studies where effects did and did not occur (Figure 3.3-14). The figure includes only the subset of studies deemed appropriate for threshold derivation (screening rationale provided in Appendix D). Ten studies of a total of 11 freshwater species and seven estuarine/marine species were deemed applicable. The majority of data applied to effects on mortality; however, there were also data for growth, development, behavior, physiological, and cellular effects endpoints.

8 Figure 3.3-14 shows the distribution of no effect and effect tissue concentrations. Based on this 9 distribution, it appears that adverse effects are unlikely to occur at tissue concentrations at or 10 below 3 mg/kg, that they are likely to occur to sensitive organisms above 10 mg/kg, and that 11 there is some uncertainty about whether they will occur at concentrations between these points.

12 **3.3.5 Sediment Quality Values (SQVs)**

Numerous sediment quality benchmarks have been developed, using various derivation procedures. The limitations associated with the derivation of the SQVs must be considered in their application. In recognition of these limitations, SQVs were used in the benthic invertebrate ERA as an additional line of evidence, rather than as a conclusive statement, regarding the toxicity of COCs in Housatonic River sediment. A summary of the values used in the ERA is provided in Appendix D (Table D.3-10).

19 3.3.6 Benthic Macroinvertebrate Community Evaluation

20 **3.3.6.1** *Methods*

21 Multiple lines of evidence were considered in the evaluation of benthic community data.



Figure 3.3-14 Combined Effects and No-Effects Levels for PCB Concentrations (mg/kg wet) in Benthic Invertebrate Tissue Samples – tPCBs and Aroclor 1254
1 Statistical Approaches Applied in Benthic Macroinvertebrate Community Assessment 2 3 Comparison of benthic assemblages between contaminated locations and reference 4 locations. Tools used to make these comparisons included: 5 Average rank plots, combining relevant summary metrics in a non-parametric multivariate approach. 6 7 • Multidimensional scaling (MDS) plots, using the same summary metrics in a 8 parametric multivariate approach. 9 Univariate tests using key summary metrics. 10 Analysis of the relationship between sediment COC concentrations and benthic community structure indices, using a regression/correlation approach. This 11 12 required partitioning of the data set into broad habitat types to help reduce the confounding effect of habitat type on benthic assemblages. 13 14

15 Because the regression/correlation approach represents an integration of exposure and effects

16 assessments, these analyses were deferred to the risk characterization (Section 3.4). The

17 comparisons of exposed locations to reference locations are discussed here.

18 Using the screening rationale provided in Appendix D (Attachment D.3), six benthic community

19 metrics were included in multivariate statistical analyses.

20	Benthic Community Metrics Evaluated
21	Multivariate Assessment:
22 23 24 25 26 27	 Organism abundance (number of animals per replicate or station). Taxonomic richness (number of unique taxa per replicate or station). "EPT" relative abundance (mayflies, stoneflies, caddisflies). Relative abundance of tolerant dipterans Relative abundance of tolerant oligochaetes. Relative abundance of tolerant gastropods.
28	Univariate Assessment:
29 30 31	 Organism abundance Taxonomic richness Modified Hilsenhoff Biotic Index (MHBI)
32	

1 **3.3.6.2** *Results*

2 Detailed results are presented in Appendix D; a summary is provided below. Overall, the benthic 3 macroinvertebrate community evaluation indicated a high degree of variability, both within and 4 among locations. Despite within-station variability, some significant locational differences were 5 observed that were consistent across the metrics considered. Specifically, for most metrics, the 6 coarse-grained contaminated stations exhibited impaired benthic communities relative to the 7 three coarse-grained reference stations; impairment was most pronounced at Stations 3 through 8 No habitat differences were identified that would explain the differences in benthic 5. 9 assemblages observed among coarse-grained locations. No strong or consistent differences in 10 benthic assemblages were observed among the fine-grained stations.

11 The results of the benthic community evaluation are summarized as follows:

- Average Rank Plots (Figure 3.3-15) Median ranks at Stations 3, 4, and 5 were significantly higher than all reference stations, indicating degraded conditions for the six metrics evaluated. Although the median ranks for Stations 1 and 2 were higher than at the coarse-grained reference sites, these differences were not statistically significant. Fine-grained stations did not differ significantly from reference locations.
- Multidimensional Scaling (Figure 3.3-16) In the MDS plot, all coarse-grained contaminated (C/C) stations (Stations 1 through 5) were set apart from the remaining stations, suggesting community alteration. Stations 1 and 2 indicated benthic communities that were different but not consistently degraded relative to the coarse-grained reference sites. The MDS analysis did not indicate benthic community alteration at the fine-grained stations.
- ANOVA (Total Abundance) The analysis indicated that all five coarse-grained contaminated stations had significantly lower total abundances than coarse-grained reference stations. There was no indication of impairment in fine-grained sediment relative to reference, however.
- ANOVA (Taxa Richness) In coarse-grained sediment, all five contaminated stations yielded significantly lower taxa richness relative to references; differences were somewhat more pronounced for Stations 3 through 5, compared to Stations 1 and 2. No evidence of ecological disruption in the fine-grained sediment was seen.
- MHBI Metric There was no compelling evidence of incremental habitat degradation due to PCBs at any of the contaminated stations using the MHBI metric. However, the appropriateness of this metric was questionable for the study area because the MHBI was not developed to address effects of PCBs, and because the reference locations indicated a high "background" proportion of pollution-tolerant taxa.

 Biomass Assessment – Overall, the biomass assessment yielded similar findings to the abundance assessment, in that lower biomass was evident in coarse-grained contaminated stations compared to reference stations. No impairment was evident in fine-grained sediment; however, the very high variability in taxonomic distributions among fine-grained locations suggests that habitat variations may limit the ability to detect perturbations.

7 3.3.7 Concentration-Response Analysis – Benthic Community Assemblages

8 The concentration-response assessment for benthic community assemblages was conducted 9 using only the PCB data collected synoptic with the benthic community grab sampling. The 10 replication at each station (i.e., characterization of micro-variation by using 12 replicates), 11 combined with the highly synoptic nature of these data, justified this approach.

Although the comparison-to-reference approach yielded significant differences for coarsegrained contaminated stations, these differences were not supported by a linear relationship with PCB concentrations over a wide range of PCB concentrations in coarse-grained sediment. It is possible that the micro-scale variation in PCB sediment chemistry confounded the determination of a relationship between PCB chemistry and benthic abundance and/or richness.

17 18

19

No pattern in other COC concentrations (or habitat variables) was observed that would explain the impaired benthic communities observed in coarse-grained contaminated sediment.



7/10/2003





4

2

1

5 Figure 3.3-16 Multidimensional Scaling for Benthic Community Health Metrics, 6 Showing Metric Medians on MDS Plot

1 3.4 RISK CHARACTERIZATION

2		Purpose of Benthic Invertebrate Risk Characterization
3		 Integrate exposure and effects assessments.
4 5		 Summarize three major lines of evidence and conduct WOE for adverse effects to benthic invertebrates.
6		 Discuss sources of uncertainty.
7 8		 Extrapolate risk findings to other species and portions of the Housatonic River downstream of the PSA.
9		
10	The	risk characterization evaluates the likelihood that adverse effects may occur as a result o
11	inver	rtebrate exposure to COCs. Three lines of evidence were used in the risk characterization
12	for b	benthic invertebrates (Figure 3.1-4):
13 14 15	•	Field surveys (i.e., benthic community structure) – For these endpoints, care was exercised to discriminate, to the extent possible, between responses related to COCs and those related to other factors such as substrate or habitat type.
16 17 18 19	•	Comparison of field-measured exposures to effects levels or benchmarks – For these endpoints, the risk characterization integrated exposure and effects by relating the two terms quantitatively (e.g., hazard quotient [HQ] method for chemistry data compared to SQVs from the literature and/or site-specific effects thresholds).
20 21	•	Site-specific toxicity study results – These endpoints (e.g., in situ and laboratory toxicity tests, TIEs) directly evaluated biological responses to COCs.
22	These	e three lines of evidence were independent, allowing for a robust weight-of-evidence
23	(WOI	E) assessment of the potential for risk using the approach of Menzie et al. (1996). All line
24	of evi	idence suggested some degree of harm to benthic invertebrates in the Housatonic River. In
25	additi	ion, for each category of measurement endpoint, there were indications that PCBs are
26	respo	onsible for the observed patterns of responses.
27	A W	OE assessment was conducted to combine the results from each line of evidence. This
28	inclu	ided a station-by-station assessment of each benthic sampling location, as well as an overal
29	WOI	E assessment for the assessment endpoint. The section concludes with a discussion o
30	sourc	ces of uncertainty in the assessment of risks of COCs to invertebrates, and the conclusion
31	of the	e risk characterization.

1 3.4.1 Field Surveys

The benthic invertebrate community study (Section 3.3.6 and 3.3.7) directly assessed the assemblages of organisms found throughout the PSA, and related these assemblages to concentrations of COCs and other stressors. After controlling for broad habitat factors (sediment particle size distributions and organic carbon content), significant differences between coarsegrained contaminated sites and coarse-grained references were observed. These differences were not observed in fine-grained sediment, however.

8 There are several possible explanations for the lack of community responses observed in the 9 downstream fine-grained sediment within the PSA, including:

 Microhabitat variation – Unlike the coarse-grained sediment, the fine-grained portions of the PSA exhibited considerable inter-station differences in invertebrate communities.
 These variations may have masked any subtle impacts due to PCBs.

13 Lower sediment chemistry – The concentrations of tPCB in the benthic community 14 sampling program were lower than for other sampling efforts associated with effects 15 endpoints (e.g., toxicity studies). As shown in Figure 3.2.3, the median sediment tPCB concentration was generally in the 1-10 mg/kg range in the fine-grained sediment 16 17 collected synoptic with the benthic community grabs. Because these concentrations are close to the site-specific toxicity threshold of 3 mg/kg derived from sediment toxicity 18 19 endpoints, large alterations in community structure would not necessarily be observed at 20 these levels. Although some biological alteration may be occurring at this concentration 21 range, the statistical power for detecting these differences is very low given the other 22 sources of variability in the study.

Reduced bioavailability of tPCB – As shown in Figure 3.2-2, some of the downstream stations exhibited high organic carbon content, which may act to sequester PCBs.
 Although the fine-grained sediment were clearly toxic at the higher exposure concentrations in the sediment toxicity tests, the high TOC may have been sufficient to suppress effects in the benthic community grab samples that had tPCB concentrations close to the 3 mg/kg threshold.

Overall, due to a relatively narrow range of exposure concentrations and high natural variability, the benthic community study was not suited to the identification of low-level environmental perturbations in fine-grained sediment. Responses in coarse-grained sediment were evident, and consistent across a number of biologically relevant effects metrics (e.g., abundance, taxonomic richness, multivariate community structure).

3.4.2 Comparison of Chemistry Data to Benchmarks

2 For chemistry data (water, sediment, and invertebrate tissue), HQs were used to quantify the 3 degree to which chemistry measurements exceeded environmental benchmarks considered 4 protective of the assessment endpoint. To address the uncertainty in generic benchmarks, the 5 HQs assessment used in the benthic ERA considered multiple benchmarks from different jurisdictions, and calculated a range of HQs. For each contaminant and medium, the full range 6 7 of HOs was considered. Furthermore, to depict the "central tendency" of the benchmarks, HOs 8 were also calculated using the median value of all applicable benchmarks. The extremes of the 9 HQ distribution are called "upper-bound" and "lower-bound" HQs in the following discussion.

10 SQVs derived from the literature are generally conservative and have high associated 11 uncertainty; hazard quotients greater than one based on literature SQVs must be interpreted in 12 this context. However, HQs based on site-specific effects thresholds are more reliable indicators 13 of potential effects. This section discusses both types of HQs; however, only literature-based 14 HQs were derived for COCs other than PCBs.

15 3.4.2.1 Sediment Chemistry

Figure 3.4-1 shows the ranges of HQs for the PCB measurements made at the seven toxicity testing stations in 1999. Within the time period (March to October 1999) 11 sampling events were conducted that were relevant to the effects data. The bars for each station indicate that the range of benchmarks derived from the literature (and thus HQs) is more than two orders of magnitude. The median SQV-based HQs for the contaminated stations are all greater than one, usually by a large amount.

Figure 3.4-1 also depicts HQs derived using the site-specific effects threshold (MATC) of 3 mg/kg. From a comparison of the two types of HQs, it is apparent that site-specific thresholds for toxicity observed in the Housatonic River fall within the range of values found in the literature, but are toward the higher end of SQVs (and therefore the lower end of HQs). All contaminated stations yielded HQ values greater than one.



- 1 HQs were also determined for other COCs that have SQVs. HQs were calculated for Sediment
- 2 Quality Triad station concentration data, and also for broader reach-wide data.

3		Sediment Hazard Quotients for Other COCs
4 5	•	Median antimony HQs were below 1, and maximum antimony HQs barely exceeded 1 at downstream stations.
6	-	Barium HQs barely exceeded 1, and only at downstream stations.
7 8	•	Median cadmium HQs exceeded 1 only at Station 7, and maximum HQs were 10 or less even at the most contaminated sites.
9 10	•	Median chromium concentrations barely exceeded 1 at downstream locations, and maximum HQs were below 10.
11 12	•	Maximum copper and lead HQs were 10 or less, even at the most contaminated stations.
13 14	•	Mercury and silver exhibited median HQs between 1 and 10 at most downstream locations.
15 16 17 18	•	The HQs for total PAHs also indicated low risk at stations from these compounds, with median HQs below 3 at all stations. However, the wide range of PAH SQVs resulted in higher HQs (i.e., greater than 10) if lower-bound SQVs are applied.
19		

The broader PSA data indicated HQs that were equal to or lower than those described above for the sampling locations. For example, the PAH HQs were much lower using the broader PSA data, with median HQs below 1 for all reaches and both substrate types.

Overall, the HQ assessment for sediment indicated that the chemical hazard for tPCBs was much higher than for other COCs. The median HQ for tPCBs was often 100 to 1,000, compared to other COCs that rarely exceeded an HQ of 10. This finding is in agreement with the TIE conclusions, which implicated PCBs and/or other non-polar organics as the dominant causative agents in toxicity tests. When HQs based on site-specific tPCB effects information are considered, risks are moderate to high for most sediment found within the PSA.

29 3.4.2.2 Water Chemistry

HQs for PCBs (Figure 3.4-2) were calculated by comparing the PCB water column data derived
from the toxicity study (EVS 2003) to water quality criteria for PCBs. The median HQs for both

reference stations (A1, A3) were less than 1.0 in all three sampling events. In contrast, the PCB concentrations at contaminated locations exhibited median HQs that were elevated and fairly consistent among stations and across monitoring events (i.e., median HQ of approximately 10). The maximum HQs, using worst-case PCB benchmarks, were approximately 100. Overall, the results indicate a moderately high hazard based on PCB chemistry in the water column, with negligible risk from other water column contaminants.

7 3.4.2.3 Tissue Chemistry

8 HQs were derived for tissue PCB burdens in benthic invertebrates sampled near the Sediment 9 Ouality Triad stations. Two sets of HQs were derived representing different levels of 10 conservatism (Figure 3.4-3). One set of HQs was based on comparison of observed tissue 11 concentrations to an effects benchmark of 3 mg/kg tPCBs, which represents the lowest 12 concentration at which significant adverse effects were found in the literature. Nearly all HQs 13 derived in this manner were greater than 1.0, and three HQs were greater than 10. The second 14 method compared observed tissue concentrations to 10 mg/kg tPCBs, a concentration that the 15 literature review suggested would cause impacts to numerous species. Even with this relaxed 16 benchmark, most HQs still exceeded 1.0.

17 **3.4.3 Site-Specific Toxicity Study Results**

Both the in situ and laboratory toxicity tests (Section 3.3.1) exhibited significant adverse effects 18 19 in both coarse- and fine-grained sediment, relative to both negative controls and field reference 20 sediment. The only toxicity test endpoints that did not yield significant adverse responses at the 21 highest tPCB concentrations were: (a) limited exposure pathways, such as water-only in situ 22 exposures; (b) short test durations; and/or (c) tolerant test species, such as freshwater 23 oligochaetes used for bioaccumulation. The large number of endpoints indicating significant 24 toxicity (even for some acute lethal endpoints), and the high magnitude of response at the highest 25 PCB concentrations (100% mortality in some treatments), indicates a significant potential for 26 environmental harm. The evaluation of concentration-response (Section 3.3.2) and the TIE study 27 (Section 3.3.3) both indicated that non-polar organics (principally PCBs) were likely the 28 dominant toxic agents in the toxicity tests.





(b) 7-day in situ



(c) 10-day in situ



Figure 3.4-2 Hazard Quotients (Median, Range) Based on Overlying Water PCB Concentrations, Measured Synoptic with In Situ Toxicity Tests

(a) Relative to lower-bound effects threshold of 3 mg/kg (HQ > 1 in black)



Predators

Shredders



(b) Relative to upper-bound effects threshold of 10 mg/kg (HQ > 1 in black)



Figure 3.4-3 Hazard Quotients for tPCB Tissue Residues in Benthic Invertebrates, Relative to Two Effects Thresholds Derived from Literature Studies

3.4.4 Integrated Station-by-Station Assessment

Potential impacts of contaminated sediment to local ecological resources at each contaminated station were assessed using a graphical approach that considered multiple lines of evidence (Figure 3.4-4). Multiple measurement endpoints were used, and the results of each were integrated into a single conclusion regarding potential ecological impacts. For the purposes of evaluating each measurement endpoint, results were categorized/simplified based on ecologically based decision criteria. The categorizations facilitated the interpretation of the results for each leg of the Sediment Quality Triad, on a station-by-station basis.

9 Each measurement endpoint was assigned a rating of high, medium, or low impact. Where 10 applicable, indications of potential for harm were standardized to appropriate background 11 conditions (e.g., toxicity endpoints were compared to reference Stations A1 and A3 rather than 12 negative control sediment). The decision criteria used to make the evaluations are summarized 13 in Appendix D.

14 The ratings in Figure 3.4-4 indicate evidence for ecological disruption for all three components 15 of the Sediment Quality Triad. For each component, there are multiple indications of "major" 16 risk, and at multiple stations. The overall assessment yielded a rating of "high" overall risk for 17 all stations except Stations 6 and 9, for which no toxicity testing was conducted. Although there 18 was a high degree of overall concordance, one area of discrepancy was in the benthic community 19 endpoints for fine-grained stations. The strong toxicological responses at these stations were not 20 associated with strong indications of benthic community alterations. The differences in PCB 21 chemistry associated with these endpoints (i.e., higher tPCBs concentrations observed in the 22 toxicity samples relative to toxicity test samples) may explain this apparent difference.

3.4.5 Weight-of-Evidence (WOE) Procedure for Assessing Risk from PCBs in the Housatonic River PSA

A formal WOE process was applied to determine whether PCBs pose a significant risk to the Housatonic River benthos. The three-phase approach of Menzie et al. (1996) and the Massachusetts Weight-of-Evidence Workgroup was applied for this purpose, in which WOE was

	Stations										
Endp	Endpoint			3	4	5	6	7	8A	8	9
1. Sediment Toxicity		1	1			1				1	
42-d <i>Hyalella</i> (lab)	Survival	-	-	-	0	-	-	•	•	•	-
42-d <i>Hyalella</i> (lab)	Growth	-	-	-	0	-	-	-	0	0	-
42-d <i>Hyalella</i> (lab)	Reproduction	-	-	-	0	-	-	-	•	•	-
43-d Chironomus (lab)	Survival	-	-	-	•	-	-	•	•	•	-
43-d Chironomus (lab)	Emergence	-	-	-	•	-	-	•	•	•	-
43-d Chironomus (lab)	Growth	-	-	-	•	-	-	•	•	•	-
10-d Hyalella (in situ)	Survival	-	-	-	0	0	-	•	-	•	-
10-d Chironomus (in situ)	Survival	-	-	-	0	0	-	•	-	•	-
48-h Daphnia (in situ)	Survival	-	-	-	0	0	-	•	-	•	-
48-h Lumbriculus (in situ)	Survival	-	-	-	0	0	-	0	-	0	-
TIE Treatments with Ceriodaphnia	Survival effect linked to PCBs	-	-	-	-	-	-	•	-	•	-
2. Benthic Community											
Multivariate – Average Rank Plots	Equal Endpoint Weighting	0	0	•	•	•	0	0	-	0	0
Multivariate – MDS	Separation in 2-dimensional plot	0	0	0	0	•	0	0	-	0	0
Modified Hilsenhoff Biotic Index	MHBI scores (ANOVA)	0	0	0	0	0	0	0	-	0	0
Taxa Richness	ANOVA versus references	0	0	•	•	•	0	0	-	0	0
Total Abundance	ANOVA versus references	•	•	•	•	•	0	0	-	0	0

1

Figure 3.4-4 Weight-of-Evidence Evaluation of Housatonic River Benthic Sampling Locations, with Indications of

Alteration/Risk Relative to Background

						Stat	tions				
Endj	Endpoint			3	4	5	6	7	8A	8	9
3. Chemistry											
Toxicity - Sediment tPCB	Synoptic with sediment toxicity tests	-	-	-	•	•	-	•	•	•	-
Benthos - Sediment tPCB	Synoptic with benthos collection	•	•	•	•	•	•	•	-	•	0
PSA Data - Sediment tPCB	Reach-wide sampling (median)	•	•	•	•	•	•	•	•	•	•
Water column tPCB	Synoptic with toxicity tests	-	-	-	0	0	-	•	-	0	-
Tissue tPCB in predators	Relative to literature benchmark	0	-	0	•	•	-	•	-	0	•
Tissue tPCB in shredders	Relative to literature benchmark	•	0	-	•	•	•	0	-	•	0
Tissue tPCB in oligochaetes (lab)	Relative to literature benchmark	-	-	-	0	0	-	0	-	0	-
4. Integrated Assessment	·										
Toxicity Endpoints	Combined Assessment	-	-	-	0	0	-	•	•	•	-
Benthic Endpoints	Combined Assessment	0	0	•	•	•	0	0	-	0	0
Chemistry Endpoints	Combined Assessment	●	•	•	•	•	•	•	•	•	0
OVERALL		●	•	•	•	•	0	•	•	•	0

2 Notes:

 \bullet = major impact.

4 \mathbf{O} = moderate impact.

5 O = negligible impact.

6

3

1

7

8 Figure 3.4-4 Weight-of-Evidence Evaluation of Housatonic River Benthic Sampling Locations, with Indications of 9 Alteration/Risk Relative to Background (Continued)

reflected in the following three characteristics: (1) the weight assigned to each measurement endpoint; (2) the magnitude of response observed in the measurement endpoint; and (3) the concurrence among outcomes of the multiple measurement endpoints.

4 A discussion of attributes considered in the WOE is provided in Section 2, and the rationales for 5 weighting of measurement endpoints are provided in Appendix D. A summary of the derived weightings for each attribute is provided in Table 3.4-1. The chemistry endpoints yielded the 6 7 lowest overall values because of lower site-specificity and some uncertainties in the biological 8 association between the measurement endpoints and the assessment endpoint(s). The toxicity 9 testing endpoints yielded the highest overall values, because of the high degree of biological 10 relevance of the tests. The benthic community structure endpoints had intermediate values. 11 Although these endpoints were site-specific, collected at a time when effects would be expected, 12 and were measures of the community structure component of the assessment endpoint, the 13 potential for the confounding effects of other factors in the direct attribution of the response to 14 the stressor reduced the utility of these endpoints to some degree.

The magnitude of the response in the measurement endpoint is considered together with the measurement endpoint weight in judging the overall WOE (Menzie et al. 1996). This requires assessing the strength of evidence that ecological harm has occurred, as well as an indication of the magnitude of response, if present. The weighting scores, evidence of harm, and magnitudes of responses were combined in a matrix format and are presented in Table 3.4-2.

20 A graphical method was used for displaying concurrence among measurement endpoints (Table 21 3.4-3). The method entailed plotting the nine symbols representing the toxicity (T), benthic 22 community (B), and chemistry (C) endpoints in a matrix, with the weight of the measurement 23 endpoint and the degree of response as axes. These graphics indicate that the majority of 24 endpoints suggest some risk for benthic communities in both coarse- and fine-grained sediment. 25 The plots also indicate that several of the endpoints suggest a high degree of risk with a 26 relatively high weight (e.g., toxicity endpoints). The conclusion from interpretation of Table 27 3.4-3 is that there is a moderate to high risk to much of the benthic community indicated by the 28 WOE evaluation.

Table 3.4-1

Weighting of Measurement Endpoints for Weight-of-Evidence Evaluation

	Endpoint Group C: Chemistry		Endpoi	nt Group T: '	Toxicity	Endpoint Group B: Benthic Community			
Attributes	C-1 (Water)	C-2 (Sediment)	C-3 (Tissue)	T-1 (Lab)	T-2 (in situ)	T-3 (TIE)	B-1 (Metrics)	B-2 (Multivar)	B-3 (MHBI)
I. Relationship Between Measurement and Assessment Endpoints									
1. Degree of Biological Association	Low	Low	Mod	Mod/High	Mod/High	Mod	Low/Mod	Low/Mod	Low/Mod
2. Stressor/Response	Low	Low/Mod	Mod	Mod/High	Mod/High	Mod/High	Low/Mod	Low/Mod	Low/Mod
3. Utility of Measure for Judging Risk	Low	Mod	Mod	High	High	Mod/High	Low/Mod	Low/Mod	Low/Mod
II. Data Quality									
4. Data Quality	High	High	High	High	High	High	High	High	High
III. Study Design									
5. Site Specificity	Low/Mod	Low/Mod	Low/Mod	Mod/High	Mod/High	Mod/High	High	High	High
6. Sensitivity to Detecting Changes	Low/Mod	Low/Mod	Low/Mod	High	Mod/High	High	Low/Mod	Low/Mod	Low/Mod
7. Spatial Representativeness	Mod/High	High	Mod	Mod	Mod/High	Low	Mod/High	Mod/High	Mod/High
8. Temporal Representativeness	High	High	Mod/High	Mod	Mod	Mod	Mod	Mod	Mod
9. Quantitativeness	Mod/High	Mod/High	Mod/High	High	High	Mod/High	Mod/High	Mod/High	Mod
10. Standard Method	Mod	High	High	High	Mod/High	Mod/High	High	High	High
Overall Endpoint Value	Low/Mod	Low/Mod	Mod	Mod/High	Mod/High	Mod	Mod	Mod	Mod

C. Chemical Measures

C-1. Concentration of PCB in overlying water in relation to concentrations reported to be harmful to benthic invertebrates.

C-2. Concentration of PCB in the sediment in relation to concentrations reported to be harmful to benthic invertebrates.

C-3. Concentration of PCB in invertebrate tissues in relation to concentrations reported to be harmful to benthic invertebrates.

T. Toxicological Measures

T-1. Sediment toxicity to multiple invertebrate species, as measured in laboratory toxicity tests.

T-2. Sediment toxicity to multiple invertebrate species, as measured in the in situ toxicity tests.

T-3. Indications of PCB as toxicity driver in TIE investigations.

B. Benthic Community Measures

B-1. Abundance, richness, and biomass of invertebrates, relative to reference stations of comparable substrate and habitat (ANOVA analysis).

B-2. Benthic community structure, as assessed using multivariate assessment of key benthic metrics (rank analysis and multidimensional scaling).

B-3. Water quality assessment using modified Hilsenhoff Biotic Index (MHBI) indicator of organic pollution.

1 **3.4.6** Sources of Uncertainty

The assessment of risks to benthic invertebrates contains uncertainties. Each source of uncertainty can influence the estimates of risk, therefore, it is important to describe and, when possible, specify the magnitude and direction of such uncertainties. Appendix D contains a more complete list of uncertainties; some of the most significant uncertainties are summarized below.

- Small-scale variability in COC exposure concentrations, which complicated the development of concentration-response relationships The variability in exposure concentrations within and among studies, and the differences in spatial trends across some studies required careful characterization of exposures that are appropriately matched to effects data, particularly for sediment concentrations. Analytical variability (Appendix C.11) and field variability both contribute to uncertainty in exposure data.
- 12 Inconsistencies in exposure concentrations across studies – The patterns of PCB concentrations observed in the benthic community study, sediment toxicity study, and 13 14 benthic invertebrate tissue sampling study were not always consistent. For example, the 15 PCB concentrations measured at Stations 7 and 8 during the benthic community sampling 16 (n=12), were lower than most other PCB measurements made at those locations. This 17 complicated the integrated station-by-station assessment presented in Section 3.4.4, since 18 the magnitudes of exposure at a given station were not always equal across all effects 19 endpoints.
- 20 Calculations of site-specific effects thresholds (e.g., sediment MATC of 3 mg/kg) had the 21 following uncertainties: (a) uncertainty due to application of dose-response models 22 required for interpolation; (b) uncertainty regarding the choice of exposure data that is 23 considered synoptic to effects information; (c) uncertainty due to natural variability in 24 exposure data and effects data. These uncertainties were addressed in the ERA by 25 conducting multiple assessments (e.g., applying different statistical models and exposure 26 The general concordance of the findings using many different data assumptions). 27 processing assumptions provides confidence that the derived thresholds are not based on 28 spurious statistical outcomes.
- Occurrence of elevated PCB concentrations (tissue and sediment) in samples collected from the West Branch of the Housatonic River near the confluence – These elevated concentrations may be due to localized PCB inputs from contamination in the vicinity of Dorothy Amos Park; this uncertainty cast some doubt on the appropriateness of Station A3 as a reference.
- The effects benchmarks derived from the literature carry a high degree of uncertainty, due to the need to extrapolate across sites, species, and PCB mixtures. The site-specific Sediment Quality Triad studies indicated that the lower-bound (most conservative) benchmarks are over-protective for PCB and other COCs, and that upper-bound benchmarks are more indicative of Housatonic River effects thresholds.

Table 3.4-2

Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Benthic Community

	Weighting	Coarse-Gra	ained Sediment	Fine-Grained Sediment		
Measurement Endpoints	Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	
C. Chemical Measures						
C-1. Concentration of PCB in overlying water in relation to levels reported to be harmful to benthic invertebrates	Low/Moderate	Yes	Intermediate	Yes	Intermediate	
C-2. Concentration of PCB in the sediment in relation to levels reported to be harmful to benthic invertebrates	Low/Moderate	Yes	High	Yes	High	
C-3. Concentration of PCB in invertebrate tissues in relation to levels reported to be harmful to benthic invertebrates	Moderate	Yes	Intermediate	Yes	Intermediate	
T. Toxicological Measures						
T-1. Sediment toxicity to multiple invertebrate species, as measured in laboratory toxicity tests	Moderate/ High	Yes	High	Yes	High	
T-2. Sediment toxicity to multiple invertebrate species, as measured in in situ toxicity tests	Moderate/ High	Yes	Intermediate	Yes	High	
T-3. Indications of PCB as toxicity driver in toxicity identification evaluations	Moderate	Undetermined	_	Yes	Intermediate	
B. Benthic Community Measures						
B-1. Abundance, richness, and biomass of invertebrates, relative to reference stations of comparable substrate and habitat (ANOVA)	Moderate	Yes	Intermediate	No	_	
B-2. Benthic community structure, as assessed using a multivariate assessment of key benthic metrics (rank analysis and MDS)	Moderate	Yes	Intermediate	No	_	
B-3. Water quality assessment using modified Hilsenhoff Biotic Index (MHBI) indicator of organic pollution	Moderate	No	_	No	—	

5

Table 3.4-3

Weight-of-Evidence Risk Analysis Summary Indicating Concurrence Among Endpoints for Coarse-Grained and Fine-Grained Sediment

Assessment Endpoint: Community, structure, survival, growth, and reproduction of benthic invertebrates

6 (a) Coarse-grained contaminated (C/C) sediment

Weighting Factors (increasing confidence or weight)							
Low	Low/Moderate	Intermediate	Moderate/High	High			
	C-2		T-1				
	C-1	B-1, B-2, C-3	T-2				
					l		
	1	Т-3	[Т		
	1	В-3			T		
	Low	Low Low/Moderate C-2 C-1	Weighting Factors (increasing confid Low Low/Moderate Intermediate C-2 C-1 B-1, B-2, C-3 T-3 B-3	Weighting Factors (increasing confidence or weight) Low Low/Moderate Intermediate Moderate/High C-2 T-1 C-1 B-1, B-2, C-3 T-2 T-1 T-3 T-3 B-3 B-3	Weighting Factors (increasing confidence or weight) Low Low/Moderate Intermediate Moderate/High High C-2 T-1 C-2 T-1 C-1 B-1, B-2, C-3 T-2 C-1 T-3 T-3 T-1 T-1 B-3 B-3 T-1 T-1		

7 8

(b) Fine-grained contaminated sediment (F/C)

		Weighting Factors (increasing confidence or weight)								
Harm/Magnitude	Low	Low/Moderate	Intermediate	Moderate/High	High					
Yes/High		C-2		T-1, T-2						
Yes/Intermediate			T-3, C-3			14				
Yes/Low										
Undetermined]	T			Т				
No Harm]	B-1, B-2, B-3							
				•						

9 10

Note: See Tables 3.4-1 and 3.4-2 for definitions of endpoint codes.

- There is uncertainty with respect to the confounding role of micro-habitat for benthic communities. Although the study design controlled for habitat (physical and biological) to the extent possible, variations in micro-habitat factors may have obscured alterations due to chemical stressors.
- Individual toxicity effects endpoints carry some uncertainty because individual taxa have
 specific tolerances to both chemical and background environmental factors. The strength
 of the Sediment Quality Triad approach comes from the multiple lines of evidence (lethal
 and sublethal test endpoints with different exposure durations) from multiple test species.
 The concurrence of findings from different taxa substantially reduced this uncertainty.

10 **3.4.7 Extrapolation to Other Species**

11 The benthic invertebrate ERA included the entire benthic community; benthic community 12 composition analysis was a measurement endpoint considered in the weight-of-evidence 13 assessment. Individual species were also used in toxicity tests as surrogates for the Housatonic 14 River freshwater benthic community. Both the status of sensitive taxa and community 15 composition are considered indicators of overall health and productivity of the benthic 16 community. As a result, no explicit extrapolation to other species was required. The toxicity test 17 species and endpoints encompass a range of toxicological sensitivities, ranging from sensitive 18 (e.g., *Hyalella* chronic reproduction) to tolerant (e.g., *Lumbriculus* survival); similar variation in 19 sensitivity can be expected in the field.

20 3.4.8 Downstream Assessment

Because of the more limited amount and spatial coverage of data on contaminant concentrations downstream of the PSA, the more rigorous approach followed in assessing ecological risks in the PSA was not appropriate or possible. Risk estimates for downstream of Woods Pond were derived by comparing observed sediment and tissue concentrations with maximum acceptable threshold concentrations (MATCs) for tPCBs developed from the Sediment Quality Triad.

8 9

10

11

12

13

14

15

16

1

MATCs for PCBs Used to Assess Risks Below Woods Pond

- The sediment MATC of 3 mg/kg tPCB was used as a conservative measure of the potential for adverse effects on benthic invertebrates downstream of Woods Pond. This concentration was developed in the risk assessment for the PSA using multiple lines of evidence (e.g., benthic community studies, in situ and laboratory toxicity testing, bioaccumulation testing, Sediment Quality Triad) and was selected as the concentration at which some sensitive endpoints exhibited apparent responses, but the magnitude of responses was not large. Above a concentration of 3 mg/kg tPCB, numerous endpoints indicated ecologically significant responses, with many LC₅₀/EC₅₀ values falling in this range.
- The tissue MATC of 3 mg/kg tPCB was used as a conservative measure of the potential for adverse effects on benthic invertebrates downstream of Woods Pond. This concentration was developed considering the frequency of adverse effects observed in the literature studies; none of the available studies yielded toxic responses below 3 mg/kg tPCB, but numerous studies above 3 mg/kg yielded significant adverse responses.

17

18 Using the MATC values, potential risks to benthic invertebrates are predicted to occur in limited 19 areas downstream of Woods Pond to Rising Pond, where pockets of sediment contaminated with 20 higher concentrations of PCBs appear to have accumulated. Below Rising Pond through the 21 remainder of Massachusetts and Connecticut, sediment does not contain concentrations of PCBs 22 that are sufficiently elevated to represent a potential risk to benthic invertebrates. Tissue 23 concentration data for caddisflies, dobsonflies, and stoneflies (BBL and OEA 2003) collected 24 from Cornwall CT indicate tPCB concentrations at or below the toxicity threshold of 3 mg/kg, 25 and therefore support the conclusions based on sediment.

26 **3.4.9 Conclusions**

Overall, the benthic ERA indicates significant risk to aquatic invertebrates based on a WOE evaluation of multiple Sediment Quality Triad endpoints. Furthermore, the available data suggest that PCBs are the primary chemical stressor responsible for such impairment. The confidence in the conclusion is moderate to high, based upon the concordance in predictions of risk from multiple measurement endpoints.

Compelling evidence for ecological risk comes from the sediment toxicity tests, which not only
 indicated significant toxicological effects in multiple appropriate indicator species and endpoints,
 but also indicated a correlation between the level of effect and sediment PCB concentration.

This correlation was consistent with the TIE results, which implicated non-polar organics as the dominant toxicants in Housatonic River sediment. The evidence of effects to benthic community structure was not as compelling, because significant alteration relative to reference conditions was not observed in the fine-grained sediment downstream of the WWTP.

5 The magnitude of risk to benthic invertebrates in the Housatonic River varies spatially, primarily 6 as a function of sediment PCB concentration and also in relation to sediment characteristics, 7 primarily organic carbon content. The WOE assessment of benthic invertebrate endpoints 8 indicates a high risk of ecologically significant effects at the PCB concentrations observed at the 9 Sediment Quality Triad stations. The toxicity studies within the PSA indicated that ecologically significant effects were observed at sediment tPCB concentrations of 3 mg/kg or higher, and that 10 11 effects were large in magnitude (i.e., 50% responses in most test species) at 10 mg/kg tPCBs. 12 These concentrations are in general agreement with a threshold identified from benthic 13 community studies (5 mg/kg tPCB) and are in concordance with the higher end of the SQVs for 14 tPCBs identified in a literature review (i.e., 1 to 10 mg/kg). The spatial distribution of tPCB 15 concentrations in the PSA (Figure 3.2-5) indicates that most of the sediment in the PSA exceeds 16 these threshold effects levels. Unacceptable risks are predicted for the majority of sediment 17 sampled within Reach 5A. In the downstream reaches of the PSA, risks were lower; however, 18 the tPCB data indicate that a high percentage of samples still exceed the site-specific thresholds 19 described above. Downstream of Woods Pond, risks are reduced relative to the PSA, and are 20 negligible to low downstream of Rising Pond.

1 3.5 REFERENCES

- 2 Academy of Natural Sciences of Philadelphia, Patrick Center for Environmental Research. 1999.
- 3 PCB Concentrations in Fishes and Benthic Insects from the Housatonic River, Connecticut, in
 4 1984-1998. November 15, 1999. Report No. 99-10F.
- 5 BBL (Blasland, Bouck & Lee, Inc.) and QEA (Quantitative Environmental Analysis, LLC). 6 2003. *Housatonic River – Rest of River RCRA Facility Investigation Report*. Prepared for
- 7 General Electric Company. January 2003.
- 8 Chapman, P.M. 1996. Presentation and interpretation of Sediment Quality Triad data.
 9 *Ecotoxicology* 5:327-339.
- 10 EPA (U.S. Environmental Protection Agency). 2000. Methods for Measuring the Toxicity and
- 11 Bioaccumulation of Sediment-Associated Contaminants with Freshwater Invertebrates. EPA
- 12 600/R-99/064. 192 pp.
- 13 EVS Environment Consultants. 2003. Assessment of in situ Stressors and Sediment Toxicity in
- 14 *the Lower Housatonic River*. Final Report, adapted from a study by G.A. Burton, 2001, Institute
- 15 for Environmental Quality, Wright State University, Dayton Ohio.
- Long, E.R., and P.M. Chapman. 1985. A Sediment Quality Triad: measures of sediment
 contamination, toxicity and infaunal community composition in Puget Sound. *Marine Pollution Bulletin* 16:405-415.
- Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S.
 Petron, B. Potocki, S. Svirsky, and P. Tyler. 1996. Special report of the Massachusetts Weightof-Evidence Workgroup: A weight of evidence approach for evaluating ecological risks. *Human*
- 22 and Ecological Risk Assessment 2:277-304.
- Swartz, R.C. 1999. Consensus sediment quality guidelines for polycyclic aromatic hydrocarbon
 mixtures. *Environ. Tox. Chem.* 18(4):780-787.
- 25 WESTON (Roy F. Weston, Inc.). 2000. Supplemental Investigation Work Plan for the Lower
- 26 *Housatonic River*. Prepared for U.S. Army Corps of Engineers and U.S. Environmental 27 Protection Agency, DCN: GEP2 020000 AAME
- 27 Protection Agency. DCN: GEP2-020900-AAME.

4. ASSESSMENT ENDPOINT—COMMUNITY CONDITION, SURVIVAL, REPRODUCTION, DEVELOPMENT, AND MATURATION OF AMPHIBIANS

4	Highlights of the Amphibian ERA
5	Conceptual Model
6 7 8 9	The assessment endpoint is the survival, development, and reproduction of amphibians in the PSA. Amphibians, including leopard frogs and wood frogs, selected as representative species for the ERA, are exposed to contaminants of concern (COCs) via diet and possibly dermal absorption.
10	Exposure
11 12 13 14	Exposure of the representative species to tPCBs, dioxins and furans, metals, and PAHs was determined through three site-specific studies that evaluated reproductive performance and developmental effects. Routes of exposure and rates of bioaccumulation were also assessed.
15	Effects
16 17 18 19 20 21	Reproductive performance and early developmental effects were assessed using a number of measurement endpoints in frogs from contaminated areas in the PSA and frogs from reference areas from the Housatonic River watershed and external reference sources. These effects were compared to those reported in the literature to identify similarity of responses for COCs, types of effects, and mechanisms of effects.
22	Risk
23 24 25 26 27 28 29 30 31 32	There is a high probability of risk of ecologically significant effects at PCB concentrations observed in the PSA. There were significant correlations between adverse effects in late larval-stage wood frogs and PCB concentrations in sediment and tissue. Leopard frogs appear more acutely sensitive than wood frogs, with strong indications of toxicity observed through the range of tPCB concentrations tested in the PSA. These findings suggest that amphibian populations are impacted throughout much of the PSA. The indications of community responses from the population studies (i.e., localized depressions of richness and abundance near high tPCB vernal pools, and high incidence of malformations observed) substantiate these findings.

33 4.1 INTRODUCTION

The purpose of this section of the ecological risk assessment (ERA) is to characterize and quantify the current and potential risks posed to amphibians exposed to contaminants of potential concern (COPCs) in the Housatonic River, focusing on total PCBs (tPCBs) and other COPCs originating from the General Electric Company (GE) facility in Pittsfield, MA. The watershed is located in western Massachusetts and Connecticut, discharging to Long Island Sound, with the GE facility located near the headwaters of the watershed. The Primary Study Area (PSA)
includes the river and 10-year floodplain from the confluence of the East and West Branches of
the Housatonic River downstream of the GE facility to Woods Pond (Figure 1.1-2).

A Pre-ERA was conducted to narrow the scope of the ERA by identifying COPCs, other than tPCBs, posing potential risks to aquatic biota in the PSA (Appendix B). The amphibian ERA further screened COPCs for specific relevance to the amphibian community occupying the vernal pool, floodplain, and backwater habitats of the Housatonic River. The contaminants of concern (COCs) that were retained for the detailed risk assessment for amphibians were tPCBs, six metals, several polycyclic aromatic hydrocarbons (PAHs), and dibenzofurans.

A step-wise approach was used to assess the risks of these COCs to amphibians in the
Housatonic River watershed. The four main steps in this process included:

- 12 1. Derivation of a conceptual model (Figure 4.1-1).
- 13 2. Assessment of exposure of amphibians to COCs (Figure 4.1-2).
- 14 3. Assessment of the effects of COCs on amphibians (Figure 4.1-3).
- 15 4. Characterization of risks to amphibians (Figure 4.1-4).

16The detailed ecological risk assessment for amphibians is provided in17Appendix E.

18

- 19 This section is organized as follows:
- Section 4.2 (Conceptual Model) describes the conceptual model for amphibians,
 including selection of representative taxa and establishment of measurement and
 assessment endpoints.

Section 4.3 (Exposure Assessment) describes the quantification of exposures, both specific to the amphibian study's effects stations and for the broader study area.

Section 4.4 (Effects Assessment) describes the potential effects to amphibians
 exposed to site COCs, as indicated by the toxicological and field investigations
 conducted in the PSA. Section 4.4 also summarizes the ranges of tissue benchmarks
 (toxicity thresholds) derived from the literature.



Figure 4.1-1 Conceptual Model Diagram: Exposure Pathways for Amphibians Exposed to Contaminants of Concern (COCs) in the Housatonic River PSA

Exposure



Figure 4.1-2 Overview of Approach Used to Assess Exposure of Amphibians to Contaminants of Concern (COCs) in the Housatonic River PSA

Effects



Figure 4.1-3 Overview of Approach Used to Assess the Effects of Contaminants of Concern (COCs) to Amphibians in the Housatonic River PSA

Risk Characterization



Figure 4.1-4 Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Amphibians in the Housatonic River PSA Section 4.5 (Risk Characterization) integrates the exposure and effects assessments and makes conclusions regarding risk for amphibians in the Housatonic River and floodplain/backwater habitats using three lines of evidence. A discussion of the sources of uncertainty regarding risk estimates follows. Section 4.5 also presents an extrapolation of risks beyond the PSA to areas downstream of Woods Pond.

6 7

1

2

3

4

1 4.2 CONCEPTUAL MODEL

Total PCBs, dioxins, and furans are persistent and hydrophobic and lipophilic. Therefore, organic carbon pools (both living and non-living) are the primary uptake vectors for juvenile amphibians, with aquatic and terrestrial invertebrates an important uptake pathway for adults. Less lipophilic COCs, such as low molecular weight PAHs and metals, are less associated with organic pools, and exhibit more complex partitioning behavior. The COCs identified for amphibians exhibit both direct (i.e., contact with contaminated source media) and indirect (i.e., food web bioaccumulation, maternal transfer) exposure pathways.

9 The conceptual models presented in Figures 4.2-1 and 4.2-2 illustrate the exposure pathways for 10 amphibians in the PSA. The amphibian assessment focused on life stages that are in direct 11 contact with Housatonic River sediment. For amphibian larvae, the dominant abiotic exposure 12 media were sediment (solid phase and/or porewater) and surface water. Concentrations of COCs 13 in tissues of amphibians were also considered. Tissue data provide an organism-based measure 14 of bioavailability, and provide an additional line of evidence to consider along with the effects 15 data gathered in the two frog developmental studies (FEL 2002a, 2002b).

Section 2, Problem Formulation, identified two indigenous species to be used in toxicity tests representative of the Housatonic River amphibian community: leopard frogs (*Rana pipiens*) and wood frogs (*Rana sylvatica*). Summary life history profiles for both species are found in the following text boxes, and detailed profiles are provided in Appendices A and E.



Figure 4.2-1 Leopard Frog Exposure Pathways







Life History of Northern Leopard Frog

The northern leopard frog is a slender, medium-sized frog, sometimes referred to as the "meadow frog" because of its preference for grassy habitats. It is not considered an obligate vernal pool species in New England, primarily because it prefers lakes, ponds, and slow-moving streams for breeding. Its life cycle includes an aquatic larval stage and semi-terrestrial juvenile and adult stages.

- Habitat—Considered semi-terrestrial. Breed and overwinter in water bodies, adults spend the entire post-breeding summer period in grassy meadows, open shrub areas, or damp woods, often far from any water. In southern New England, appear to be restricted to floodplains along large streams and rivers, wetlands along lake margins, and meadows adjacent to freshwater and brackish tidal wetlands. Often inhabit cattle pastures and hay fields, otherwise seem to avoid severely disturbed or sites with poor water quality. In the spring, attracted to vegetated shorelines by a greater abundance of food, moderated temperatures, and protective cover.
- Home Range and Territoriality—Adults show marked fidelity to home areas, with individuals remaining in a relatively confined area for most of the summer, returning to that area after nighttime excursions and the following year after hibernation and breeding. Especially active during rainy nights, when they will often move to warm road surfaces. Temperature (air and water) may play a major role in the timing of their movements between wintering and breeding areas and between summering and wintering areas. Have shown excellent homing ability when displaced moderate distances (i.e., <1 km) from home area.</p>
- Food Habits and Diet—Foods of adults and juveniles include insects, as well as spiders, snails, and frogs. Availability rather than preference likely determines food types; beetles are a staple in the diet of adults and juveniles. Moth and butterfly larvae; grasshoppers and crickets; bees, wasps, and ants; and bugs are also common. Vegetation can also make up a significant volume (10 to 20%) of adult and juvenile food. Diet of adults more diverse than that of juveniles. Tadpoles are primarily herbivorous, consuming algae, plankton, and small plant materials (detritus) from the substrate and the undersides of aquatic vegetation within the natal pond.


Life History of Wood Frog

The wood frog is one of the smaller frogs inhabiting the Northeast. Like the spotted and Jefferson salamanders, it is considered an obligate vernal pool amphibian species because it requires (or, more accurately, prefers) vernal pools for breeding. Its life cycle includes an aquatic larval stage and terrestrial juvenile and adult stages.

- Habitat—Entirely terrestrial except during the brief breeding season, when they move to vernal pools and other aquatic habitats to mate and lay eggs. Preferred terrestrial habitats are cool, moist upland woods, often far from water, but also found in wooded swamps and bogs. In summer, are active day and night. Use brush piles and other terrestrial features for cover, rather than seeking aquatic escape like some other frogs. During winter, hibernate in upland areas under rotting wood, moss, stones, or decaying leaf litter, never in water. Preferred breeding habitat are vernal pools, however, will also utilize ditches, cattail swamps, gravel pits, slow-moving streams, and other ephemeral habitats that lack fish.
- Home Range and Territoriality—Summer home range estimated for adults was 77.2 square yards (695 sq ft), with a range of 3.5 to 440 sq yd, not significantly different between males and females. Suggested that many remain in a "home area," at least during the summer. Availability of food was likely one of the principal factors affecting home range size. Adults exhibit high degree of fidelity to their breeding ponds each year; some juveniles may disperse to breeding ponds other than the ones in which they were born. No information was found in the literature regarding the territoriality of adult in terrestrial habitats. Frogs in general may defend their shelters against other amphibians. Males are only somewhat territorial in the breeding pools during the brief mating period.
- **Food Habits and Diet**—Food includes insects, especially beetles, flies, slugs, snails, spiders, bugs, moth larvae, and earthworms. Tadpoles thought to be mostly herbivorous feeders, consuming algae, decaying plants (detritus), and various microorganisms scraped from aquatic plants present in the breeding pools. Tadpoles found to be extremely effective predators of American toad eggs and hatchlings inhabiting the same pool, despite the fact that American toad eggs and larvae are thought to be toxic or distasteful to other organisms.

The assessment endpoint that is the subject of this section is the maintenance of local populations of amphibians by ensuring the survival, reproduction, and development of local species. The measurement endpoints used to evaluate the assessment endpoint are presented below.

4	Measurement Endpoints for Amphibians
5	 Semiquantitative sampling of larval amphibians in breeding habitats with different
6	sediment concentrations of stressors. Endpoints include species richness per
7	habitat type; species abundance; gross pathology; and body, tail, and total length
8	measurements.
9	 Surveys of vernal pools to quantify amphibians entering vernal pools and
10	determine breeding behavior and condition; egg laying, hatching success, and
11	larval growth and development; metamorphosis and emigration.
12	 Amphibian toxicity tests designed with exposure over a gradient of stressor
13	concentrations in site sediment. Toxicity endpoints include morphology of
14	embryos and juveniles, limb development, skin maturation, and tail resorption of
15	<i>Rana pipiens</i> .
16	 Gravidity of females; egg count; necrotic eggs; oocyte maturity; sperm count,
17	morphology, and viability; fertilization rate; embryo viability; hatching success;
18	mortality; and teratogenesis of <i>Rana pipiens</i> collected from the study area
19	compared with a reference area.

20 4.2.1 Amphibian Developmental Studies

21 Three separate site-specific studies were conducted to evaluate reproductive performance and 22 developmental effects in frogs exposed to PCBs and other COCs (two studies were conducted by 23 EPA, and one was conducted by GE). The studies focused on reproduction, early development, 24 and maturation (metamorphosis) in northern leopard frogs, and development and maturation in 25 wood frogs. These represent critical stages in amphibian life cycles and provide information on 26 the capacity of PCB and other COCs to disrupt the life-cycle processes (Sparling et al. 2000). 27 Various reproductive and developmental endpoints were assessed, such as gravidity of female 28 frogs, egg mass fertilization and hatching success, larval and metamorph mortality, growth, and 29 incidence of larval and metamorph malformation. Bioaccumulation of COCs in amphibian 30 tissue was also evaluated. The selection of individual test endpoints was made a priori and based 31 on previous investigations into the sensitivity of various life stages to organic contaminants.

1 4.2.2 Leopard Frog Study: EPA

2 This study was designed to evaluate both the reproductive fitness of adult leopard frogs in the 3 PSA, as well as monitor development of hatchlings through the metamorphosis stage. Adult frogs (male and female) were collected from nine sampling areas within the PSA; these animals 4 5 were to be fertilized in the laboratory, with the resulting larvae to serve as the test organisms in 6 the developmental portion of the study. The reproductive condition of the adults was examined, 7 including body weight, sperm count, sperm morphology, ovary weight, egg count, and egg 8 maturity. However, the field-collected females possessed virtually no mature oocytes, so 9 fertilization was unsuccessful. In addition, the males exhibited a high proportion of malformed 10 sperm heads. The study design was modified to include the field collection of egg 11 masses/hatchlings at five of the nine original sampling stations; these stations were the only ones 12 that contained live leopard frogs for sampling. The egg masses/hatchlings were returned to the 13 laboratory for developmental evaluation. Larval mortality, malformation, growth, and incidence 14 of metamorphosis were recorded. Figure 4.2-3 illustrates the general study design.

15 Cross-over treatments were also included, wherein control egg masses were cultured in 16 contaminated sediment; the resulting larvae remained in the test media and were observed 17 through metamorphosis. A sediment spiking treatment was conducted to further investigate the 18 relationship between vernal pool media and larval development (effectively removing any 19 influence of COC transfer via the maternal pathway). The treatment involved culture of control egg masses in reference site sediment that had been spiked with 30 mg/kg Aroclor 1260. COC 20 21 tissue concentrations were measured in samples representing various leopard frog life stages 22 (adult whole body, egg mass/ovary, and larvae whole body).

23 4.2.3 Wood Frog Study Design (EPA Studies)

The wood frog study was initiated in April 2000 and evaluated the growth, development, and maturation of wood frogs. The study design combined laboratory exposures to vernal pool water and sediment and assessment of field-collected animals, and consisted of three separate phases. Figure 4.2-4 shows a simplified model of the study design, showing the life stage, exposure scenario, and endpoints evaluated for each phase of the study.



Figure 4.2-3 General Model of Leopard Frog Vernal Pool (VP) Reproduction and Development Study



Figure 4.2-4 General Model of Wood Frog Vernal Pool (VP) Reproduction and Development Study

1		Design of Wood Frog Vernal Pool Study
2 3 4 5 6 7 8 9	•	Phase I: Egg masses collected from 11 vernal pools – 8 contaminated vernal pools with low, medium, and high PCB concentrations (the ninth pool, with the second-highest tPCB concentration, did not have any egg masses); and 3 reference pools. Egg masses were cultured in the laboratory in sediment and water collected from the associated contaminated and reference pool. Egg mass fertilization, egg counts, egg weight, hatching success, larval growth, percent metamorphosis, and malformations were the endpoints for this phase. Egg mass tissue and metamorph samples were analyzed for tPCBs.
10 11 12 13 14	-	Phase II: Following natural hatching of egg masses in the pools, tadpoles were collected during 4 sampling events, each about 2 weeks apart to assess in situ development. Endpoints for this phase included larval growth and malformations. Larval tissue samples from the first and third collection event were analyzed for tPCBs.
15 16 17 18	-	Phase III: 50 wood frog metamorphs were collected from the 11 pools, and individual weight, gender, and malformations were recorded. Tissue samples (one composite per station) were analyzed for tPCBs and other COCs (PAHs, OC-pesticides, metals, and dioxins/furans).
19		

20 4.2.4 Context-Dependent Wood Frog Study: GE

The objectives of this study were to address whether larval density in the natal pool and PCB exposure affects the survival and growth of wood frog larvae and whether the two factors interact to influence larval developmental success.

24 The study was initiated in April 2001 and began with the collection of egg masses from five 25 vernal pools within the PSA. The pools included 8-VP-4, 8-VP-5, 23b-VP-2, 40-VP-1, and 40-26 VP-3 (see Figure E.2-1). Approximately 21 egg masses were collected from each pond; the eggs 27 were then transported to a building at the GE facility in Pittsfield, MA, and maintained until they 28 hatched. Composite tissue samples were collected from each pool (approximately 200 29 hatchlings per sample) and analyzed for whole body PCB content. Although there were no 30 associated egg mass tissue samples, the study assumed that these hatchling (i.e., larvae) samples 31 served as an indicator of the transfer of maternal contaminants from parent to offspring.

Hatchling tissue tPCB concentrations were the basis for the selection of experimental treatment groups. The study design specified three concentrations for evaluation in the field exposures: a "low" (3.3 mg/kg) hatchling tissue tPCB concentration bounded by a "very low" (0.89 mg/kg) and a "high" (11.2 mg/kg) concentration. The experimental design utilized these three levels of hatchling tissue PCB concentration, (i.e., very low, low, and high), and three levels of initial larval density (i.e., 200, 400, and 800). Each of these combinations was exposed in two vernal pools (23b-VP-1 and 23b-VP-2). These two vernal pools were chosen because they supported natural populations of wood frogs, had very low concentrations of tPCBs, and were believed to be deep enough to hold water longer than most of the other floodplains in the PSA. A total of 18 experimental treatments were established:

Density	Hatchling PCB = 0.89 mg/kg (40-VP-3)	Hatchling PCB = 3.3 mg/kg (23b-VP-2)	Hatchling PCB = 11.2 mg/kg (8-VP-5)		
200 larvae	N=2	N=2	N=2		
400 larvae	N=2	N=2	N=2		
800 larvae	N=2	N=2	N=2		

8

9 Approximately 200, 400, or 800 larvae were selected at random from each of the three vernal 10 pool's hatchling crop and placed in the in situ experimental enclosures (depending on each 11 treatment's assigned initial larval density). Concurrent to this field selection, three additional 12 sets of larvae were selected at random for tissue PCB analysis (composited samples).

The survival and growth of both tadpoles and metamorphs were assessed in the study. An analysis of variance (ANOVA) approach was used to test whether any combination of initial larval density, hatchling tissue tPCB concentration, or vernal pool sediment tPCB concentration affected the number and weight of juveniles of each life stage at the end of the test.

17

1 4.3 EXPOSURE ASSESSMENT

The exposure assessment estimates the exposure of amphibians to tPCBs and other COCs in the Housatonic River PSA (Figure 4.1-1). Exposures were assessed as either the COC concentrations in sediment or water, or as the tissue body burdens that represent integrated exposure from all sources. Routes of exposure were assessed to determine the contribution of maternal transfer and the extent of bioaccumulation during various stages of development.

To match exposure data with effects-based measures, many of the sediment tPCB data considered were from sampling conducted as part of the EPA studies that evaluated reproductive performance and developmental effects in frogs exposed to tPCBs and other COCs. These synoptic sediment samples are referred to as discrete sample data. Additional exposure data included spatially weighted sediment tPCB concentrations. These were calculated using all available sediment data, to develop average concentrations based on habitat types preferred by wood frogs and leopard frogs during the reproductive period.

Sediment data were included in the GE wood frog study for the five pools of original egg mass selection and for the two experimental ponds used for the larval development portion of the study. No source or methods of sample collection was given for the sediment data; however, concentrations were fairly similar to the spatially weighted tPCB values for a given vernal pool.

18 **4.3.1** Selection of COCs for Amphibians

19 The contaminants initially considered in the amphibian exposure assessment were identified in 20 the Pre-ERA (Appendix B). The Pre-ERA included screening on a reach-by-reach basis and 21 subdivision of COPCs by major hydrological/geomorphological category.

A refined screening was conducted on the sediment data collected in support of the two EPA amphibian developmental studies. The intent of this exercise was to further identify the sediment COCs that were most relevant to local amphibian populations. The sediment COCs retained for the amphibian assessment are presented below. Total PCBs were identified as sediment COCs in all reaches of the PSA. A number of PAHs were retained throughout Reach 5, and three PAH compounds were retained for Reach 6 (Woods Pond). Dioxins/furans were
retained in the PSA, as well as some metals.

3 4

5

6

7

Contaminants of Concern for Amphibians

- Chlorinated organic compounds tPCBs, dioxins/furans.
- Metals Cadmium, chromium, copper, lead, mercury, silver.
 - PAHs Some individual PAH compounds, including low and high molecular weight PAHs.

8

9 Several additional contaminants (mainly pesticides) were determined in the Pre-ERA to be below 10 detection limits in sediment, but had detection limits that exceeded screening benchmarks. An 11 examination of the amphibian tissue data indicated that concentrations for most of the pesticides 12 of concern were below detection limits or below background. On this basis, and considering that 13 some pesticide detections may be attributable to laboratory interference artifacts, the entire suite 14 of organochlorine pesticides listed above was eliminated from further consideration in the 15 amphibian portion of the ERA.

16 4.3.2 Exposure Data

17 The approach used to characterize exposure to amphibians was based upon evaluation of 18 numerous data sources, including both sediment and water column COC concentrations and 19 amphibian tissue COC concentrations (Figure 4.1-2). Exposure data were evaluated to achieve 20 acceptable synopticity between exposure and effects endpoints, while also recognizing the need 21 to address spatial and temporal variability in the data, and inherent limitations in field sampling. 22 Concentration-response relationships were investigated using both (1) the single "most synoptic" 23 chemistry value paired with each toxicity endpoint (i.e., the average of the April and May 2000 24 sediment sampling events); and (2) a combined data set, based on all available relevant sediment 25 data and used to generate a spatially weighted average exposure concentration for vernal pools 26 and backwaters within the PSA. In most cases, the two approaches yielded similar results and 27 helped to reduce the uncertainty associated with the use of either particular source of data.

A community-wide assessment of amphibians in the PSA was conducted from 1998 to 2000 using visual and audio surveys, dip-netting, funnel-trapping, and pit-trapping techniques. A total of 13 species were observed, including 4 salamanders, 8 frogs, and 1 newt. Amphibian
 observation and capture data were combined with detailed habitat-type maps to predict likely
 occurrences per species and to identify breeding, post-breeding, and wintering habitat.

4 4.3.3 Habitat Characterization

5 To provide the foundation for the problem formulation, the preliminary results of physical and 6 ecological investigations were used to evaluate potential habitat influences on amphibian 7 reproduction and development. Detailed characterization of the sediment type provides an 8 indication of the potential contaminant bioavailability, and the characterization of habitat allows 9 for the identification of physical and ecological characteristics that could affect the amphibian 10 endpoints.

Sediment characterization was based on examination of gross physical parameters (such as total organic carbon [TOC] and grain size distributions) known to affect contaminant partitioning, and therefore, bioavailability. In addition, potential effects of the Pittsfield Wastewater Treatment Plant (WWTP) discharge were also examined with respect to the backwater habitats (to evaluate potential impacts of the WWTP). Evaluation of effects from the WWTP was based on the assumption that backwater habitats receive a portion of their sediment load from the river main stem; therefore, influence from the WWTP may be carried into the backwaters.

18	
19 20 21 22	
23 24 25 26 27	
28 29 30	

Vernal Pool/Backwater Substrate Evaluation

- Vernal pool and backwater sediment is much richer in organic matter than main channel sediment, particularly in the upper Reach 5 area. The range of TOC for the amphibian sampling areas was 1.7 to 59.1%, with a median TOC concentration of 7% in the wood frog vernal pools, and 6% in the leopard frog sampling areas.
- Grain size distributions for the wood frog sampling areas in the PSA were fairly homogeneous and similar to reference sites. However, comparison of backwater sediment characteristics at locations in the leopard frog study indicated that the reference station had more coarse-grained sediment than did the sampling areas in the PSA.
- The relationship between tPCB and TOC appeared qualitatively similar upstream and downstream of the WWTP (no excess organic enrichment attributed to the WWTP, thus, no confounding influence).

31

1 4.3.4 Assessment of Sediment Chemistry

2 4.3.4.1 Sources of Sediment Data

3 Sediment data were collected for different programs, each with varying degrees of synopticity to 4 amphibian effects metrics. The sediment data that were used included all floodplain samples 5 collected from depths of 0 to 6 inches; all floodplain vernal pool samples collected in 1998 and 6 1999 to characterize floodplain sediment tPCB contamination; and all leopard frog and wood 7 frog samples collected as part of the EPA amphibian developmental studies. Use of all relevant 8 data for the purposes of the risk assessment helped to reduce the uncertainty associated with any 9 of the individual data sets (i.e., small-scale spatial variability, analytical variability, limited 10 spatial coverage).



32 4.3.4.2 Distribution and Concentrations of PCBs

33 4.3.4.2.1 Vernal Pool Characterization Data

The 1998 to 1999 ecological characterization sampling resulted in more than 500 samples analyzed for tPCBs. Many of these samples were collected from vernal pools, with less than 10% collected from pool perimeters. Perimeter samples were collected in areas that, in wetter
years, would be submerged during the breeding period. A few of these samples were analyzed
for other COCs: six for PCB congeners, seven for PAHs, eight for metals, and eight for
dioxins/furans.

5 4.3.4.2.2 Spatially Weighted Average Exposure Concentrations

6 Many floodplain soil and sediment samples were collected over a several-year period in the PSA 7 (a subset of which are described above). All surficial (0-15 cm) data collected in the PSA, 8 combined with detailed habitat type maps and an understanding of site-specific hydrodynamics, 9 were used to estimate spatially weighted surficial PCB concentrations. Data from similar habitat 10 types were used in conducting the spatial weighting exercise, and were grouped into six similar 11 habitat types; sampling area boundaries were then incorporated as an overlay. The approach is 12 summarized in Appendix C.3, and the results are shown on Figures 2.5.5 through 2.5.11 of the 13 ERA.

14 For application in the amphibian risk assessment, an exposure point concentration (EPC) was 15 computed as the spatially weighted average (arithmetic mean) of the cells contained within each leopard or wood frog sampling area boundary. The EPC represents the estimated average 16 17 concentration for a juvenile frog during development or an adult during breeding and foraging. 18 Juvenile frogs were assumed to move at random within their natal ponds or pools, and to be 19 equally exposed to every point within these areas; thus, the spatially weighted average served as 20 an appropriate representation of exposure. The individual amphibian endpoints (from the EPA 21 studies) for a given sampling station were then evaluated with respect to both the discrete 22 developmental study data and the spatially weighted EPCs.

Spatially weighted EPCs for the leopard frog sampling areas ranged from 0.4 to 44 mg/kg, with six of the nine areas greater than 20 mg/kg tPCB. Spatially weighted EPCs for the wood frog vernal pools ranged from 0.2 to 99.5 mg/kg; approximately two-thirds were greater than 10 mg/kg tPCB (see Figure 4.3-1).



2 Figure 4.3-1 Frequency Distribution and Cumulative Percentage of 3 Sediment tPCB Exposure Point Concentrations for 66 PSA 4 **Temporary and Permanent Pools (Based on EPA Spatially** 5 Weighted Data)

6

1

7 4.3.4.2.3 Data Collected in Support of the EPA Amphibian Developmental Studies

8 The sediment chemistry data with greatest synopticity with the effects endpoints in the 9 amphibian ERA were data collected in conjunction with the collection of amphibian tissues (egg 10 masses and larvae) during the EPA developmental studies. These sampling efforts included:

- 11 Nine samples from the PSA and one reference site sediment sample for the 2000 12 leopard frog study. These samples were analyzed for PCBs (total and Aroclors) and 13 all other COCs.
- 14 A total of 23 sediment samples collected for the 2000 wood frog developmental study 15 (collected in April and May). Samples were analyzed for PCBs (total and Aroclors), 16 dioxins/furans, and inorganics (cyanide and sulfide). The May 2000 samples were 17 also analyzed for other COCs (metals, PAHs, herbicides, and organophosphate [OP] 18 pesticides). Samples collected during the April event were later analyzed for 19 congeners.

Figure 4.3-2 shows the sediment tPCB concentrations for both amphibian developmental studies.
 The pools used as experimental ponds in the GE study also are shown on Figure 4.3-2; the pools
 are 23b-VP-1 and 23b-VP-2.

4 5

6 7

8

9

10

11 12 Summary of Occurrence of Other COCs in Sediment Data

- Metals concentrations at leopard frog sites were similar to the Muddy Pond reference station; only Station E-1 had elevated metals concentrations. Metals in the wood frog vernal pools were similar to the reference stations.
 - Total PAH concentrations were elevated at most leopard frog stations, relative to the reference site. PAH concentrations in the wood frog vernal pools were elevated at three stations. Elevated PAH compounds included acenaphthylene, benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, and pyrene.

13 4.3.5 Surface Water Chemistry Assessment

14 Surface water chemistry data is applicable to the amphibian ERA because the early life stages of 15 both frog species are entirely aquatic (water and sediment exposure). However, there is 16 uncertainty associated with extrapolating water chemistry effects in biota when the animals also 17 have the potential for exposure to sediment. Variability in water column concentrations over 18 time adds additional uncertainty. For these reasons, bulk sediment and porewater contaminant 19 concentrations are more commonly used as exposure metrics for toxicity testing. Because of the 20 uncertainty in relating water chemistry to effects on amphibians, the most relevant water data 21 were those collected in conjunction with effects measurements (from EPA studies). These data 22 were collected in conjunction with sediment sampling conducted for the two EPA amphibian 23 developmental studies (10 water samples for the leopard frog study, 22 water samples for the 24 wood frog study).

Total PCBs in water samples collected from both amphibian studies were reported as either Aroclors 1254 and 1260, or both, with tPCBs used as the single PCB metric in data evaluation. Sediment and water tPCB concentrations were correlated in both amphibian studies. Surfacwater tPCB concentrations were lowest in the reference stations and the target stations with low sediment tPCB concentrations (0.01 to 0.03 μ g/L for the wood frog study, 0.013 μ g/L for the leopard frog study). Elevated water tPCB concentrations corresponded to amphibian 31



1

2

3 4

Figure 4.3-2 Total Sediment PCB Concentrations for Wood Frog Vernal Pool Study (mean, n =2) and Leopard Frog Reproduction/Development Study

1 sampling areas with elevated tPCB concentrations (0.1 to 0.47 µg/L for the wood frog study,

2 0.03 to 0.41 μ g/L for the leopard frog study).

For the most part, all other COCs measured in the water samples were screened out of the amphibian risk assessment and, therefore, out of further data analysis. No water data were reported in the GE wood frog study report.

6

7

.

8 9 10

11

12

All metals screened in the sediment assessment were measured as non-detects in the two water samples except for zinc. The measured value for zinc (17 μ g/L) is below both the EPA federal and British Columbia provincial criteria for protection of aquatic life. Therefore it was not retained as a COC.

Elimination of Other COCs in Surface Water Data

- All PAHs were screened out of the water assessment because they were not detected.
- 13

14 **4.3.6 Tissue Chemistry Assessment**

There was less tissue chemistry data from the two EPA studies than abiotic data, and these data generally did not include replication due to limited volumes of tissue available for chemical analysis. Nevertheless, the available data provide indications of the site-specific bioavailability of the COCs.

19 Figure 4.3-3 presents the distribution of tPCB concentrations by sampling location and tissue 20 type for adult samples collected at the leopard frog stations. Adult whole body tissue 21 concentrations ranged from 0.15 to 5.4 mg/kg at PSA sites; Figure 4.3-3 shows whole body 22 samples only from sites with associated offal and egg mass/ovary samples. PSA site offal 23 (whole body minus ovary/egg mass) tPCB ranged from 0.02 to 2.6 mg/kg; egg mass/ovary 24 samples ranged from 0.24 to 45.1 mg/kg. Larval tissue concentrations ranged from 0.05 to 1.4 25 mg/kg tPCB. For all tissue sample types, there was a direct relationship between sediment tPCB 26 concentration and tissue tPCB concentration.

Tissue samples from the cross-over and Aroclor 1260-spiked treatments confirmed the importance of the sediment uptake pathway; control animals raised in PSA site media had a tissue concentration of 0.37 mg/kg, while the control animals raised in reference media had a



Note: Data include adult whole body and adult offal (with associated egg mass); male frogs were included in adult whole body tissue sample and data are shown for informational purposes only. N=2 for offal/egg mass samples (except for station W-6; n=1). N=1 for adult whole body samples.

Figure 4.3-3 Comparison of Leopard Frog Tissue Samples to Sediment tPCB Concentrations (Reproductive Study Data and Spatially Weighted Data)

mean body burden of 0.056 mg/kg (based on two treatments). Animals in the spiking study had
a body burden of 0.55 mg/kg (spiked treatment) and 0.007 mg/kg (control treatment).

3		Sources of Amphibian Tissue Chemistry Data
4 5 6 7 8 9 10		 Leopard frog study (EPA): Nine composite whole body samples were analyzed for tPCBs. Nine individual offal samples with associated egg mass/ovary tissue removed were analyzed for tPCBs (Figure 4.3-3). Five offal samples were analyzed for other COCs. Five composite larval samples were analyzed for tPCBs. Six composite larval samples were analyzed for tPCBs from the cross-over and Aroclor 1260 spike treatments. All tissue samples confirmed contaminant bioavailability from sediment.
11 12 13 14 15 16 17 18 19		Wood frog study (EPA): 15 egg mass samples, 13 Phase I metamorph samples, 20 Phase II larval samples (from two discrete sampling events), 10 Phase III metamorph samples (five of these samples analyzed for other COCs) were analyzed for tPCBs. Four larval tissue samples from cross-over treatments and one larval tissue sample from the Aroclor 1260 spike treatment were also analyzed for tPCBs. Results of analysis showed that PCBs are bioavailable at all life stages, but that egg mass tissue concentration is not related to sediment tPCB. However, all other tissue samples showed a trend of increasing contaminant uptake with increasing exposure concentration and duration in the vernal pools.
20 21 22 23 24		 Wood frog study (GE): Composite tissue samples were collected at two events during the juvenile period: at the hatchling stage (1 – 2 days post-hatch) and at the early larval stage (approximately 11 – 12 days post-hatch), one from each of the five vernal pool stations during the first event, and 4 composites during the second event (Pool 10.9 [EPA 40-VP-1] was not sampled).
25 26	Wood	t frog tissue concentrations (FPA study) across various life stages are included in F

Wood frog tissue concentrations (EPA study) across various life stages are included in Figure 26 27 4.3-4. PSA site egg mass tissue concentrations ranged from 0.01 to 2.1 mg/kg, but were 28 unrelated to sediment tPCB concentrations. This was not surprising, given that the egg mass 29 concentration would be more representative of the female's exposure prior to moving into the 30 pool to breed. Phase I metamorph (laboratory-cultured) tPCB concentrations ranged from 0.06 31 to 5.83 mg/kg in the PSA animals and were related to sediment tPCB concentration. Phase II 32 larval tissue samples (from collection event 1) ranged from 0.28 to 3.44 mg/kg and were not 33 related to sediment tPCB concentration. However, later-stage larval samples (from Phase II, 34 collection event 3) were related to sediment tPCB concentrations (tissue tPCBs ranged from 0.09 35 to 10.4 mg/kg). Phase III metamorph (field-collected) samples ranged from 0.13- to 15-mg/kg 36 tPCB and also were related to sediment tPCB concentrations.

In the cross-over and spiking studies, tissue tPCB concentrations were elevated in all samples
 exposed to PCBs and were much lower in the reference exposures.



Vernal Pool ID

Note: No tissue samples for 39-VP-1; no Phase II, event 3 tissue data for WML-2; Phase I egg mass tissue values for WML-1, WML-2, and WML-3 were non-detect (ND) – values shown reflect detection limits (0.007, 0.004, and 0.008, respectively). * WML-1 Phase III metamorph sample is anomalous.

Figure 4.3-4 Comparison of tPCB Concentrations in Tissue (in Various Phases of the Wood Frog Developmental Study) with Mean Vernal Pool and Spatially Weighted Mean tPCB Concentrations in Sediment

Hatchling tissue concentrations in the GE study ranged from 0.26 mg/kg to 11.2 mg/kg; no other COC tissue data were included in the report (n = 5 composites). The hatchlings from three of the five pools were then selected for placement in the in situ vernal pool enclosures. Just prior to placing the test organisms in the enclosures, larval tissue samples representing animals from the three hatchling test concentrations were analyzed for PCB body burden. Larval tissue concentrations ranged from 1.4 to 7.2 mg/kg tPCB. A fourth larval tissue sample was also analyzed and contained 6.1 mg/kg tPCBs, although hatchlings from this location were not evaluated in the experimental treatments.

4.4 EFFECTS ASSESSMENT

The effects assessment for amphibians (Figure 4.1-3) emphasizes the site-specific field investigations; because these studies provided direct indications on the bioavailability, toxicity, and effects of site-specific COCs. Both toxicity assessments (i.e., laboratory toxicity, in situ toxicity) and the community evaluations (i.e., amphibian community composition) were compared to appropriately matched field references to determine whether the exposed sites in the Housatonic River vernal pool/backwater habitats exhibited biological impairment.

The effects assessment also provides an overview of the literature on the effects of tPCBs and other COCs to survival, growth, and reproduction of amphibians. At the time of the literature review, there were virtually no amphibian studies available that contained paired sediment or water exposure data with effects data. However, there were sufficient studies available that evaluated tissue PCB concentrations with response data. A total of five different frog species were used in the studies, including both leopard frogs and wood frogs. There were a total of 18 "no effect" measurements (ranging from 0.02 to 11.2 mg/kg tPCB wet weight) and 11 "effect" measurements (ranging from 0.96 to 128 mg/kg tPCB wet weight).

Detailed evaluation of concentration-response relationships, for both toxicity assessments and amphibian community structure assessments, are not included in this section. These are presented in the risk characterization section (Section 4.5). Accordingly, this section is limited to a discussion of differences between effects at the exposure locations and control and/or reference locations.

4.4.1 Sediment Toxicity

4.4.1.1 Data Evaluation

The two EPA amphibian studies contained over 200 individual metrics that were collected to track reproduction and development through multiple life stages; however, because some of these metrics were redundant, they were not used in the ERA. Metrics that were the most biologically relevant and that provided independent measurements of an effect were selected to assess the overall degree of effects to amphibian communities. The majority of endpoints selected for detailed discussion in the ERA were determined on an a priori basis based on hypotheses formulated during the study design; others were selected based on the patterns of effects that emerged during initial exploration of the data. Approximately 50 discrete endpoints, representing each major life stage of leopard and wood frogs, were initially evaluated for the ERA data analysis. The approach used to evaluate these endpoints was based on two objectives:

- Determination of relative sensitivity of various life stages.
- Evaluation of COC concentration-response relationships.

The wood frog data were fairly well suited to the use of inferential statistics in the evaluation of relationships. The leopard frog data, however, required a more qualitative approach.

4.4.1.1.1 Leopard Frog Data

Inferential statistics were not deemed appropriate for analysis of the leopard frog effects data, for three reasons:

- The magnitude of the effects observed made the use of statistical analyses less appropriate than for the wood frog data. The leopard frogs exhibited much more of a threshold effect response for various endpoints; fairly low responses in the reference/control treatments, and very high responses in the exposure treatments. Such data distributions are better evaluated through visual interpretation (figures) and examination of average response data (for a given endpoint) at each station.
- Sample sizes were small, primarily because of limited availability of test animals in the field (often, n = 5 or n = 6), which makes the application of many standard statistical tests inappropriate.
- Because fertilization of the field-collected females was unsuccessful due to the lack of mature oocytes, there was no biological relationship between female PCB body burdens or reproductive tissue endpoints (i.e., percent Stage VI oocytes, percent

malformed sperm cells) and larval/developmental effects endpoints (i.e., incidence of metamorphosis).

In summary, the evaluation of leopard frog concentration and response data was limited to a more qualitative presentation. Non-parametric Spearman's correlation tests were conducted on adult tissue samples and sediment tPCB concentrations (sufficient paired sediment/tissue data), to determine the potential influence of environmental exposure on contaminant uptake into animal tissue. Data from the cross-over and spiked studies were evaluated via hypothesis testing for differences among groups (always a two-sample comparison of a control treatment and a target treatment of interest).

4.4.1.1.2 Wood Frog Data: EPA Study

Tests for correlation were used to determine relationships between variables of interest. Evaluation of the distribution of much of the exposure/response data revealed that the distributions were not normal, thus precluding the use of parametric statistics and reducing the confidence that could be placed in such approaches as simple linear regression.

The non-parametric Spearman's rank-order correlation coefficient, r_s (Spearman's correlation), was selected. The Spearman's correlation is less sensitive to data "outliers," thereby reducing their influence when evaluating a relationship. The choice of calculating a Spearman's correlation provided a conservative and robust approach to the data analysis.

The overall consistent pattern of significant relationships between biological effects and PCB concentration at sensitive life stages, combined with corroborating literature-based effects data, provided a check against spurious correlations.

As with the leopard frog data, the cross-over and spiked study data were evaluated via hypothesis testing for differences among groups (a two-sample comparison of a control treatment and a target treatment of interest).

4.4.1.1.3 Wood Frog Data: GE Study

Multivariate and univariate analysis of variance (MANOVA and ANOVA) were used to evaluate the interaction of vernal pool, hatchling tissue tPCB concentration, and initial larval density on the survival and growth of the test organisms. In addition, correlation analyses were used to determine whether there was a relationship between hatchling and larval tissue concentration and the sediment tPCB concentration of the vernal pool from which the egg masses were collected.

4.4.1.2 Results

4.4.1.2.1 Leopard Frog Study

Reproductive Fitness: Adult male and female leopard frogs (and some juveniles of both sexes) were collected from the nine contaminated sampling areas in the PSA and transported to Fort Environmental Laboratories, Inc. (FEL). No leopard frogs were collected at the three reference areas; therefore, control animals purchased from a commercial supplier (Carolina Biological Supply, CBS) were used. These frogs were collected in Vermont directly upon order, shipped to CBS, and then forwarded to FEL (formerly part of The Stover Group).

It is not known why leopard frogs were not available in the reference areas or why no eggs were found during the year this study took place. They had been observed in the prior year, and it was assumed that there was a large enough population present for sampling. This assumption may have been incorrect. Because of the timing of collection and the limited number of reference areas with suitable potential habitat, and because soil and sediment samples confirmed only background concentrations of tPCBs and COCs, it was necessary to obtain outside control frogs.

The timing of collection of adult leopard frogs from the target stations coincided with the normal onset of reproductive receptiveness and initiation of breeding activity. Adult specimens were collected between March 25 and April 22, 2000. Surface water temperatures in the PSA were approximately 8 to 10° C at this time (WESTON 1998 – 1999). These temperatures represent the ideal environmental "triggers" for the frogs to emerge in the early spring and gather in breeding areas. Typically, males begin chorusing when water temperatures reach approximately 8° C, with oviposition peaking when water temperatures reach 10° C (Gilbert et al. 1994). Hine et al. (1981) reported the occurrence of breeding when water temperatures reached or slightly exceeded 10° C in Wisconsin ponds.

After collection and transport to the laboratory and acclimatization for 24 hours, female frog gravidity was recorded, and mature (gravid) females were hormonally induced to super-ovulate egg masses; fertilization was then attempted on these egg masses using sperm collected from

male frogs from the same sampling area. The number of eggs produced per female, rates of necrosis, and oocyte developmental stage distribution were determined. Sperm count, morphology, and overall viability were also assessed. The eggs were monitored for fertilization, morphology, and coloration.

Male body weight and sperm count did not appear to be related to exposure media tPCB concentrations; however, there was a strong inverse relationship between incidence of sperm head abnormalities and sediment tPCB concentrations (Table 4.4-1 and Figure 4.4-1). Sperm head abnormalities may have contributed to the low fertilization success of the eggs from the field-collected females. Tissue data were not collected on the male frogs, so a comparison of body burden to sediment tPCB concentration or percent abnormal sperm heads could not be conducted.

Data on female frog reproductive fitness were limited because of small sample sizes (see Table 4.4-2). Findings regarding reproductive fitness of the female leopard frogs include:

- Adult leopard frog specimens collected from contaminated sampling areas in the PSA showed marked signs of reproductive stress.
- None of the females collected from Sites E-5, W-9a, W-8, and E-1 (37.0, 4.3, 120.0, and 160.0 mg/kg sediment tPCBs, respectively) were found to be gravid (eggs mature enough for successful fertilization).
- Few of the PSA sites produced female specimens that possessed any biologically significant quantity of Stage VI oocytes (mature eggs capable of fertilization), with the exception of Station W-7a (Figure 4.4-2). Immature oocytes (< Stage III) were observed in mature female specimens collected from all PSA sampling areas, however developing oocytes were found in specimens from Sites W-7a, W-4, EW-3, and W-1 (18.0, 0.5, 30.0, and 0.2 mg/kg sediment tPCBs, respectively). Therefore, the lack of success in artificially fertilizing oocytes from contaminated site specimens was not surprising, and appeared to be the primary limiting factor in the reproductive dysfunction observed in the contaminated site specimens evaluated from the PSA.
- Even though more advanced oocytes were found in specimens containing greater concentrations of ovary tPCBs, only a few Stage VI oocytes were found, indicating that the final stage of maturation that involved hormonal induction of the final preparatory event known as germinal vesicle breakdown (GVBD) may have been inhibited. Further, since oogenesis and, to a greater extent, egg maturation, were inhibited in ovaries with tissue residues of as low as 0.3 mg/kg, the threshold for inhibition appeared to be below this residue level.



Figure 4.4-1 Comparison of Percent Abnormal Sperm Heads (Mean) from Male Adult Chemical Analysis Leopard Frogs, with Mean Sediment tPCB and Spatially Weighted Mean tPCB



Figure 4.4-2 Comparison of Mean Percent of Oocytes at Stage VI (Mature) for Female Leopard Frogs, with Mean Sediment tPCB and Spatially Weighted Mean tPCB

Table 4.4-1

Summary of Male Adult Leopard Frog Reproductive Health

Sampling	Sediment tPCB (mg/kg)*		Maan Total	Moon Molo	Mean Testes Weight (% of	Mean %	Mean Sperm	
Area ID	tPCB	Sp. Wt. PCB	Water PCB (µg/L) (SD)		Total Body Weight) (SD)	Sperm Heads (SD)	Gonad Tissue (SD)	
R1	-	-	-	40.6 (6.29)	0.176 (0.016)	0.42 (0.32)	5.38 (1.25)	
R2	-	-	-	40.8 (4.75)	0.126 (0.083)	0.89 (0.51)	7.88 (5.94)	
R3	-	-	-	35.9 (1.48)	0.179 (0.039)	2.36 (0.49)	3.61 (0.56)	
pooled R1, R2, R3	-	-	-	39.2 (4.91)	0.162 (0.051)	1.14 (0.95)	5.60 (3.40)	
W-1	0.15	0.4	0.013	36.5 (4.08)	0.163 (0.044)	4.33 (0.76)	5.06 (2.86)	
W-4	0.46	0.4	0.013	37.2 (NA)	0.140 (NA)	3.15 (NA)	4.48 (NA)	
W-9a	4.3	7.5	0.013	40.7 (4.82)	0.147 (0.023)	8.26 (2.39)	2.42 (0.87)	
W-7a	18	27.6	0.03	34.2 (4.93)	0.106 (0.040)	12.0 (4.72)	3.52 (1.67)	
EW-3	30	23.8	0.41	31.4 (4.28)	0.112 (0.026)	49.5 (10.8)	7.87 (0.28)	
W-6	42	21	0.22	39.6 (5.28)	0.138 (0.058)	37.3 (6.26)	2.01 (1.30)	
W-8	120	43.5	0.14	34.8 (11.8)	0.089 (0.010)	42.7 (2.39)	6.08 (0.20)	
E-1	160	26.6	0.24	41.4 (7.39)	0.113 (0.075)	14.3 (4.43)	3.83 (3.26)	

* tPCB = Value from amphibian developmental studies; Sp. Wt. PCB = mean tPCB for each sampling area based on spatial weighting of sediment data.

SD = Standard deviation.

Table 4.4-2

Summary of Female Adult Leopard Frog Reproductive Health

Sampling Area	Sediment tPCB (mg/kg)*		Total Water	Mean Female	Mean Egg Mass/Ovary	Mean Female	Proportion	Mean Ovary Weight (% of	Mean Total	Mean % of < Stage III	Mean % of Store VI
ID	tPCB	Sp. Wt. PCB	PCB (µg/L)	(mg/kg) (SD)	PCB (mg/kg) (SD)	(g) (SD)	Gravid	Body Weight) (SD)	(SD)	< Stage III Oocytes (SD)	Oocytes (SD)
R1	-	-	-	0.012 (0.014)	0.036 (0.007)	77.41 (5.55)	4/4	30.78 (3.87)	1264 (908)	23.01 (32.75)	56.75 (25.20)
R2	-	-	-	0.011 (0.013)	0.012 (0.005)	79.32 (15.26)	4/4	23.32 (5.71)	2811 (1342)	27.82 (8.93)	65.25 (7.22)
R3	-	-	-	0.017 (0.010)	0.024 (0.003)	76.10 (12.33)	5/5	32.14 (5.44)	119 (49)	0	89.46 (6.16)
Pooled R1, R2, R3	-	-	-	0.015 (0.010)	0.024 (0.011)	77.61 (11.14)	13/13	29.01 (6.14)	1174 (1339)	14.62 (21.85)	72.50 (20.78)
W-1	0.15	0.4	0.013	0.022 (NA)	0.240 (NA)	48.83(18.28)	1/5	4.56 (4.59)	1008 (961)	84.86 (30.29)	0.44 (0.89)
W-4	0.46	0.4	0.013	-		43.26 (10.69)	2/2	6.23 (0.36)	1038 (110)	28.38 (7.90)	2.22 (0.60)
W-9a	4.3	7.5	0.013	1.260 (NA)	45.086 (NA)	51.59 (12.04)	0/3	3.88 (1.28)	1238 (799)	99.70 (0.52)	0
W-7a	18	27.6	0.03	1.407 (1.636)	14.219 (17.801)	52.43 (12.87)	5/5	21.35 (2.88)	2918 (1663)	20.00 (44.72)	4.88 (3.04)
EW-3	30	23.8	0.41	-		55.73 (4.97)	2/3	13.04 (5.67)	419 (436)	67.93 (29.50)	1.02 (1.37)
E-5	37	19.6	0.043	-		50.33 (NA)	0/1	1.25 (NA)	177 (NA)	100	0
W-6	42	21	0.22	0.386 (NA)	9.477 (NA)	57.35 (12.66)	2/5	4.65 (1.99)	2401 (841)	97.52 (3.39)	0.06 (0.13)
W-8	120	43.5	0.14	-		52.66 (11.66)	0/1	5.03 (NA)	307 (NA)	100	0
E-1	160	26.6	0.24	-		37.91 (9.73)	0/4	3.17 (0.97)	1168 (733)	99.26 (1.55)	0

* tPCB = Value from amphibian developmental studies; Sp. Wt. PCB = mean tPCB for each sampling area based on spatial weighting of sediment data.

SD = Standard deviation.

NA = Not applicable; only 1 replicate.

1 Only two PSA sampling areas had frogs in the same body size range as the control 2 frogs. Purchased frogs (collected in Vermont) were larger than most contaminated 3 site frogs, and had relatively larger ovaries. Vermont is approximately 45 miles north 4 of the PSA, and the local climatic conditions are similar to those found in western 5 Massachusetts. Because the control frogs were collected in a similar climate, region, and timeframe, differences in body sizes were not expected. 6 7 Juvenile frogs collected for this portion of the study were not included in the 8 assessment of overall female reproductive fitness. 9 In summary, the evidence supporting impairment of reproductive fitness related to PCB exposure 10 includes: 11 Low rates of egg maturation. 12 Poor egg mass fertilization from field-collected female frogs.

 High incidence of sperm head abnormalities in males from vernal pools with high sediment PCB concentrations.

Few contaminated site female oocytes reached Stage VI (only 381 of 10,611 eggs from all contaminated sites; these eggs came from only 4 frogs), whereas control eggs totaled 5,785, and more than half reached Stage VI (3,653 eggs). Rosenshield et al. (1999) found a significant negative correlation between sediment PCB concentration and hatching success of green frog (*Rana clamitans*) and leopard frog embryos exposed along a PCB gradient.

20 **Developmental Endpoints:** Because of poor egg fertilization success, where most of the females 21 from the PSA sampling areas were reproductively unfit, the study design was modified to 22 include the field collection of egg masses from the leopard frog sampling areas, and to raise them 23 in the laboratory, as was done for wood frogs. Each contaminated area and reference area was 24 surveyed for egg masses and hatchlings, which were then collected at five of the nine 25 contaminated sampling areas (EW-3, W-6, W-1, W-7a, and W-4). No egg masses were found in 26 the two locations with the highest sediment PCB concentrations (Stations W-8 and E-1), or at the 27 reference areas. Therefore, control egg masses were obtained from the females leopard frogs 28 from CBS fertilized in the laboratory.

The four larval endpoints measured in the study (mortality, metamorphosis, malformation, and growth) were not evaluated for the same amount of time; larval malformation and growth endpoints had shorter test durations than larval mortality and metamorphosis endpoints.

13

14

Differences in test duration for the four endpoints were normalized to a common test duration to
make comparisons among the endpoints. To perform comparisons among the four endpoints,
data for the mortality and metamorphosis endpoints were taken from the last day that
malformation and growth were measured. Additional endpoints included:

5 6

7

8

- Mean percent metamorphosis at end-of-test (EOT).
- Mean percent larval mortality at EOT.
- Days to reach Gosner developmental Stage 26 (±1).
 - Developmental stage reached at EOT.

9 Gosner Stage 26 is the point in development when tadpoles go from a relatively immobile 10 embryo to an active, feeding tadpole. EOT was used to designate the shorter test duration (the 11 last day that larval growth and malformation were measured). Final test duration refers to the 12 last day that larval mortality and metamorph data were recorded.

Table 4.4-3 and Table 4.4-4 show the responses of the four developmental endpoints from the main study, as well as the results of the cross-over and Aroclor 1260-spiked treatments. Findings with respect to the leopard frog developmental endpoints are presented in the following paragraphs.

Mortality was high (85 to 100%) for larvae raised in contaminated sediment regardless of PCB concentration, when compared to R3 control larvae (44%) raised in Muddy Pond sediment and water. Effects, in the form of high larval mortality, occurred at all sampling sites in the PSA.

The incidence of larval malformations was low (0 to 3.4%) in sampling areas with tPCB concentrations below 1 mg/kg, and higher (46 to 54%) in sampling areas with tPCBs greater than 20 mg/kg. Malformations were similar to those observed in studies of exposure of other frog species, including other ranids and the South African clawed frog (*Xenopus laevis*) to PCBs and similar contaminants (Birge et al. 1978; Eisler and Belisle 1996; Gutleb et al. 1999, 2000). Thus, the effects observed in the leopard frog study appeared to be characteristic of exposure to these PCB or PCB-like contaminants.

27 Larval developmental delay was observed in leopard frogs raised in contaminated sediment. 28 There was an obvious relationship between sediment tPCBs and the amount of time for the 29 larvae to reach Stage 26 (\pm 1) (Figure 4.4-3). Control larvae reached this stage in 13 days,

Table 4.4-3

Summary of Leopard Frog Larval Development Endpoints Data at End-of-Test

				Sedimer (mg.	nt tPCB /kg) ^c		Living	Maan %		Mean % Larval Malformed	Days to		Mean
Sampli	ng Area ID ^a	Test Duration ^b	Initial Larval Count	tPCB	Sp. Wt. PCB	Water tPCB (µg/L)	at End of Test (EOT)	Larval Mortality at EOT	Mean % Metamorph at EOT	(Based on Surviving Larvae)	Reach Stage 25 (±2)	Develop. Stage at EOT	Growth: Length at EOT (cm)
EW-3		22	13	30	23.8	0.41	NA	100	NA	NA	NA	22	NA
W-6		91	98	42	21	0.22	11	88.8	0	54.5	91	25-27	4.48
W-1		105	105	0.15	0.4	0.013	14	86.7	0	0	49	37-40	3.88
W-7a		105	105	18	27.6	0.03	16	84.8	0	45.8	105	25-27	4.47
W-4		111	210	0.46	0.4	0.013	15	92.8	0.83	0	55	36-37	4.55
MP Ref.		69	160	0.04	-	0.013	125	21.8	0	3.4	13	38	4.30
Cross	R1 Target	91	40	120	-	0.14	19	52.5	2.5	25.9	91	26	4.06
over	R3 Target ^d	69	80	120	-	0.14	70	12.5	0	26.1	69	32	4.39
Study	Reference ^d	69	160	0.04	-	0.013	125	21.8	0	3.4	69	38	4.3
Aroclor	Spiked	23	80	30	-	-	57	28.7	NA	29.8	NA	NA	NA
Spike Study	Control	23	80	0.04	-	-	59	26.2	NA	0	NA	NA	NA

^aSampling areas arranged in order of increasing test duration.

^bDurations vary for endpoints; larval malformation and growth had shorter test durations than larval mortality/metamorphosis. Test durations shown here are for the malformation/growth endpoints. Last day of test duration shown here is used as end-of-test (EOT) for a given sampling area.

^ctPCB = Value from amphibian developmental studies; Sp. Wt. PCB = mean tPCB for each sampling area based on spatial weighting of sediment data.

^dTreatments used in hypothesis testing.

NA = Not applicable.

Table 4.4-4

Summary of Leopard Frog Larval Development Endpoints at Final Test Duration

					Sedime (mg	nt tPCB /kg) ^c	Living Larvae and			
Sampling Area ID ^a		FEL Site ID	Final Test Duration ^b	Initial Larval Count	tPCB	Sp. Wt. PCB	Metamorphs at End of Test (EOT)	Final Mean % Larval Mortality	Metamorphs (EOT)	Mean % Metamorph (at Final Test Duration)
EW-3		37	28	13	30	23.8	NA	100	0	0
W-6		35	128	98	42	21	8	91.8	3	3.1
W-1		39	142	105	0.15	0.4	9	91.4	0	0
W-7a		34	142	105	18	27.6	13	87.6	1	0.95
W-4		36	148	210	0.46	0.4	9	95.7	2	0.95
MP Ref.		40	106	160	0.04	-	90	43.8	9	5.6
Cross- over Study	R1 Target	-	128	40	120	-	19	60.0	2	5.0
	R3 Target ^d	-	106	80	120	-	70	32.5	7	8.8
	Reference ^d	-	106	160	0.04	-	125	43.8	6	5.6
Aroclor 1260 Spike Study	Spiked	-	23	80	30	-	57	28.7	NA	NA
		-	23	80	0.04	-	59	26.2	NA	NA

^aSampling areas arranged in order of increasing test duration.

^bTest durations for the larval mortality/metamorphosis endpoints were longer than for the larval malformation/growth endpoints. Endpoint measures in this table correspond to final test durations.

^ctPCB = Value from amphibian developmental studies; Sp. Wt. PCB = mean tPCB for each sampling area based on spatial weighting of sediment data.

^dTreatments used in hypothesis testing.

NA = Not applicable.



Test duration	ons:	
Control	69 days	
W-1		105 days
W-4		111 days
W-7a		105 days
EW-3		22 days
W-6		91 days
		•

Figure 4.4-3 Days to Gosner Developmental Stage 26 (±1) and Final Developmental Stage Reached at End-of-Test, with Sediment tPCB and Spatially Weighted Mean tPCB (FEL 2002b): 2000 Leopard Frog Study whereas larvae from sampling area W-7a (18 mg/kg sediment tPCB) took 105 days, and larvae from sampling area W-6 (42 mg/kg sediment tPCB) took 91 days. Even given the uncertainty that comes with comparing the control animals to the contaminated larvae, this difference appears too large to be attributable to genetics alone. In addition, larvae from the two most contaminated stations (W-7a and W-6) never developed beyond the Stage 26 (±1) endpoint.

Extended time to metamorphosis, and a low incidence of metamorphosis, was observed in juveniles from the PSA sites. Leopard frog larvae normally spend 63 to 90 days as tadpoles (DeGraaf and Rudis 1983; Taylor and Kollros 1946; Gosner 1960) before metamorphosis. The test durations for the four treatments with larvae surviving beyond day 28 exceeded the time period of normal development, and metamorphosis was expected before the end of the test durations. Few larvae reached metamorphosis.

12 The study results indicate that some endpoints demonstrate a very strong toxic response to PCBs,13 even at low concentrations.

14	Summary of Northern Leopard Frog Toxicity Study
15	Adult Reproductive Fitness Endpoints:
16 17 18 19 20 21	 Both male and female adult frogs showed signs of reproductive stress, with the females showing more severe effects. Males exhibited a high incidence of malformed sperm in the higher-sediment tPCB sites (up to 50%). Females had virtually no mature eggs (Stage VI, which the eggs must reach in order for fertilization to occur). Incidences of immature oocytes (Stage III or smaller) were high in the sites with high concentrations of sediment tPCB (up to 99% Stage III).
22	Developmental Endpoints:
23 24 25 26	 High sensitivity to acute endpoints: larval mortality very high (88 to 100% in the PSA treatments, 44% in the control treatment); low incidence of metamorphosis (0 to 6% in the PSA treatments, 6% in the control treatment, but 63% in the water-only control treatment).
27	 Minor indications of reduced endpoint performance for larval malformations.
28 29 30	 High incidence of larval developmental delay, such that subsequent environmental changes (i.e., decreased water temperature) may prohibit animals developing in situ from reaching metamorphosis.
31	

1 4.4.1.2.2 Wood Frog Study: EPA

2 Table 4.4-5 presents the results of statistical tests of significance (comparisons to reference 3 stations) for wood frog toxicity test endpoints. Toxicological responses for impacted endpoints 4 that were related to exposure media concentrations are also presented graphically in detail in 5 Appendix E. Sediment tPCB concentrations are presented in two ways based on the two data 6 processing approaches discussed in the exposure assessment: (1) the concentrations measured in 7 the sediment samples collected with the amphibian samples, which represent the single PCB 8 concentration measurement taken closest to the effects endpoint (i.e., most synoptic 9 concentration); and (2) spatially weighted exposure point concentrations (EPCs) for each vernal 10 pool as discussed above.

Phase I: Egg mass viability for each vernal pool was evaluated relative to PCB concentrations in sediment, water, and tissue. In general, there were no significant relationships found between egg mass tissue concentration and any of the egg mass endpoints (such as hatching success or percent fertilization). Egg mass tissue concentrations or any egg mass endpoints were not significantly related to sediment tPCB concentration.

The Phase I egg mass viability studies indicate that these early life stage endpoints do not exhibit consistent adverse effects that can be linked to PCB concentrations at the concentrations measured in this portion of the study. Egg mass tPCB concentrations were not related to any relevant Phase I endpoints, such as larval or metamorph mortality or the incidence of larval/metamorph malformations. These results (relative to other life stages) suggest that maternal transfer in wood frogs is not the dominant exposure pathway through which toxicity was manifested in this study.

Both the magnitude and duration of exposure were shown to be important factors in the
manifestation of adverse effects in developing wood frogs. Larval development, metamorphosis,
and mortality assessment endpoints included:

- 26 27
- Evaluating larval mortality and metamorphosis at day 95, the longest uniform exposure duration that could be applied to all treatments.
- Evaluating larval mortality and incidence of malformation at the end of each test treatment.

Table 4.4-5

Statistical Analysis Results: Wood Frog Reproduction and Development Studies

Variable (shading indicates sig	/Endpoint gnificant relationship)	Sample Size (n)	Statistical Test	Results
Phase I Egg Mass	tPCB sediment (discrete) and tPCB egg mass tissue	11	Spearman's	r = 0.36, p > 0.05
	tPCB sediment (SW) and egg mass tissue	8	Spearman's	r = -0.19, p > 0.05
	tPCB water and tPCB egg mass tissue	11	Spearman's	r = 0.31, p > 0.05
Egg Mass Viability	tPCB sediment (discrete) and mean egg weight	12	Spearman's	r = -0.014, p > 0.05
	tPCB sediment (SW) and mean egg weight	9	Spearman's	r = 0.067, p > 0.05
	tPCB sediment (discrete) and % fertilized eggs	12	Spearman's	r = -0.29, p > 0.05
	tPCB sediment (SW) and % fertilized eggs	9	Spearman's	r = -0.28, p > 0.05
	tPCB water and % fertilized eggs	12	Spearman's	r = -0.10, p > 0.05
	tPCB egg mass tissue and % fertilized eggs	11	Spearman's	r = -0.27, p > 0.05
	tPCB sediment (discrete) and hatching success	12	Spearman's	r = 0.18, p > 0.05
	tPCB sediment (SW) and hatching success	9	Spearman's	r = -0.067, p > 0.05
	tPCB water and hatching success	12	Spearman's	r = -0.26, p > 0.05
	tPCB egg mass tissue and hatching success	11	Spearman's	r = -0.08, p > 0.05
Phase II Larvae, Event 1	tPCB sediment (discrete)and tPCB Event 1 larvae tissue	10	Spearman's	r = 0.29, p > 0.05
	tPCB sediment (SW) and tPCB Event 1 larvae tissue	8	Spearman's	r = -0.26, p > 0.05
	tPCB water and tPCB Event 1 larvae	10	Spearman's	r = 0.098, p > 0.05
Phase II Larvae, Event 3	tPCB sediment (discrete)and tPCB Event 3 larvae tissue	11	Spearman's	r = 0.89, p < 0.002
	tPCB sediment (SW) and tPCB Event 3 larvae tissue	8	Spearman's	r = 0.74, p = 0.05
	tPCB water and tPCB Event 3 larvae tissue	10	Spearman's	r = 0.69, p < 0.05
Phase II Larval Growth and Development (Field- Collected Animals), Event 4	tPCB sediment (SW) and Event 4 larval malformations	8	Spearman's	r = 0.83, p = 0.02

Table 4.4-5

Statistical Analysis Results: Wood Frog Reproduction and Development Studies (Continued)

Variable/Endpoint (shading indicates significant relationship)		Sample Size (n)	Statistical Test	Results
Larval Development, Metamorphosis, and Mortality	tPCB sediment (discrete) and Phase I larval mortality (day 95)	11	Spearman's	r = -0.41, p > 0.05
	tPCB sediment (SW) and Phase I larval mortality (day 95)	8	Spearman's	r = -0.88, p < 0.02
	tPCB water and Phase I larval mortality (day 95)	11	Spearman's	r = -0.65, p < 0.05
	tPCB egg mass tissue and Phase I larval mortality (day 95)	11	Spearman's	r = 0.19, p > 0.05
	tPCB sediment (discrete) and Phase I larval mortality (EOT)	11	Spearman's	r = -0.41, p > 0.05
	tPCB sediment (SW) and Phase I larval mortality (EOT)	8	Spearman's	r = -0.57, p > 0.05
	tPCB water and Phase I larval mortality (EOT)	11	Spearman's	r = -0.43, p > 0.05
	tPCB egg mass tissue and Phase I larval mortality (EOT)	11	Spearman's	r = 0.21, p > 0.05
	tPCB sediment (discrete) and Phase I larval metamorphosis (day 95)	11	Spearman's	r = 0.43, p > 0.05
	tPCB sediment (SW) and Phase I larval metamorphosis (day 95)	8	Spearman's	r = 0.57, p > 0.05
	tPCB water and Phase I larval metamorphosis (day 95)	11	Spearman's	r = 0.42, p > 0.05
	tPCB egg mass tissue and Phase I larval metamorphosis (day 95)	11	Spearman's	r = -0.13, p > 0.05
	tPCB sediment (discrete) and Phase I larval metamorphosis (EOT)	11	Spearman's	r = 0.41, p > 0.05
	tPCB sediment (SW) and Phase I larval metamorphosis (EOT)	8	Spearman's	r = 0.57, p > 0.05
	tPCB water and Phase I larval metamorphosis (EOT)	11	Spearman's	r = 0.43, p > 0.05
	tPCB egg mass tissue and Phase I larval metamorphosis (EOT)	11	Spearman's	r = -0.21, p > 0.05
	tPCB sediment (discrete) and no. of days to 50% mortality Phase I larvae	11	Spearman's	r = 0.53, p > 0.05
	tPCB sediment (SW) and no. of days to 50% mortality Phase I larvae	8	Spearman's	r = 0.81, p < 0.05
	tPCB water and no. of days to 50% mortality Phase I larvae	11	Spearman's	r = 0.71, p < 0.02
	tPCB egg mass tissue and no. of days to 50% mortality Phase I larvae	11	Spearman's	r = -0.30, p > 0.05
	tPCB sediment (discrete) and % Phase I larval malformation Gosner stage 20-24	11	Spearman's	r = 0.80, p = 0.005
	tPCB sediment (SW) and % Phase I larval malformation Gosner stage 20- 24	8	Spearman's	r = 0.74, p = 0.05
Table 4.4-5

Statistical Analysis Results: Wood Frog Reproduction and Development Studies (Continued)

Variabl (shading indicates s	le/Endpoint significant relationship)	Sample Size (n)	Statistical Test	Results
Larval Development, Metamorphosis and	tPCB water and % Phase I larval malformation Gosner stage 20-24	11	Spearman's	r = 0.77, p < 0.01
Mortality (Cont'd)	tPCB tissue and % Phase I larval malformation Gosner stage 20-24	11	Spearman's	r = 0.53 p > 0.05
	tPCB sediment (discrete) and Phase 1 mean metamorph weight	11	Spearman's	r = 0.56, p > 0.05
	tPCB sediment (SW) and Phase 1 mean metamorph weight	8	Spearman's	r = 0.26, p > 0.05
	tPCB water and Phase 1 mean metamorph weight	11	Spearman's	r = 0.67, p < 0.05
	tPCB egg mass tissue and Phase 1 mean metamorph weight	11	Spearman's	r = 0.66, p < 0.05
	tPCB sediment (discrete) and Phase 1 metamorph malformations	11	Spearman's	r = 0.84, p < 0.005
	tPCB sediment (SW) and Phase 1 metamorph malformations	8	Spearman's	r = 0.81, p < 0.05
	tPCB water and Phase 1 metamorph malformations	11	Spearman's	r = 0.73, p < 0.02
	tPCB egg mass tissue and Phase 1 metamorph malformations	11	Spearman's	r = 0.49, p > 0.05
	tPCB metamorph tissue and Phase 1 metamorph malformations	11	Spearman's	r = 0.54, p > 0.05
Phase I Metamorphs	tPCB sediment and tPCB metamorph tissue	11	Spearman's	r = 0.55, p > 0.05
	tPCB sediment (SW) and tPCB metamorph tissue	8	Spearman's	r = 0.76, p < 0.05
	tPCB water and tPCB metamorph tissue	11	Spearman's	r = 0.67, p > 0.05
	tPCB egg mass tissue and tPCB metamorph tissue	11	Spearman's	r = 0.16, p > 0.05
Phase III Metamorphs	tPCB sediment (discrete) and tPCB metamorphs (all)	10	Spearman's	r = 0.43, p > 0.05
	tPCB sediment (discrete) and tPCB metamorphs (exclude WML-1)	9	Spearman's	r = 0.70, p = 0.05
	tPCB sediment (SW) and tPCB metamorphs (exclude WML-1)	8	Spearman's	r = 0.76, p < 0.05
	tPCB water and tPCB metamorphs (all)	10	Spearman's	r = 0.74, p < 0.05
	tPCB water and tPCB metamorphs (exclude WML-1)	9	Spearman's	r = 0.81, p < 0.02

Table 4.4-5

Statistical Analysis Results: Wood Frog Reproduction and Development Studies (Continued)

Varia (shading indicate	Sample Size (n)	Statistical Test	Results	
Phase III Metamorph Development (Field-	tPCB sediment and Phase III metamorph mean weight	10	Spearman's	r = 0.25, p > 0.05
Collected Animals)	tPCB metamorph tissue (excluding reference) and Phase III metamorph mean weight	9	Spearman's	r = 0.37, p > 0.05
	tPCB sediment (discrete) and sex ratios	10	Spearman's	r = -0.77, p < 0.02
	tPCB sediment (SW) and sex ratios	8	Spearman's	r = -0.91, p = 0.005
	tPCB metamorph tissue (excluding WML-1) and sex ratios	9	Spearman's	r = -0.91, p < 0.005
	tPCB sediment (discrete) and % malformation Phase III metamorphs	10	Spearman's	r = 0.93, p < 0.001
	tPCB sediment (SW) and % malformation Phase III metamorphs	8	Spearman's	r = 0.93, p < 0.005
	tPCB metamorph tissue (excluding WML-1) and % malformation Phase III metamorphs	9	Spearman's	r = 0.85, p < 0.01
	tPCB sediment (discrete) and % female gonadal malformation,* Phase III metamorphs	10	Spearman's	r = 0.95, p < 0.002
	tPCB sediment (SW) and % female gonadal malformation,* Phase III metamorphs	8	Spearman's	r = 0.95, p < 0.005
	tPCB metamorph tissue (excluding WML-1) and % female gonadal malformation,* Phase III metamorphs	9	Spearman's	r = 0.88, p < 0.005
	tPCB metamorph tissue and % female gonadal malformation,* Phase III metamorphs	10	Spearman's	r = 0.72, p < 0.05
	Metamorph sex ratio and % female gonadal malformation*	10	Spearman's	r = -0.93, p< 0.002

* Although the relationship between female gonadal malformation and total incidence of malformation is arguably correlated, these comparisons are still of interest. There are many types of malformations that a juvenile could show; however, the malformed females had a high incidence of gonadal aberrations that increased in relation to increasing sediment and tissue tPCB concentration. Gonadal malformations can lead to sterility of the females.

5

• Evaluating larval malformations at the first observation period, which occurred at approximately Gosner developmental Stages 20 to 24. These are pre-feeding stages characterized by full development of external gills (Gosner 1960). The transition from embryo to a free-swimming, feeding tadpole (Gosner Stage 25/26) occurs in these stages.

6 The larval development and metamorphosis component of the Phase I wood frog study produced 7 mixed indications of toxicity to individuals. There were virtually no adverse effects related to 8 PCB concentrations for mortality, time to metamorphosis, incidence of metamorphosis, or 9 growth endpoints. The pattern of responses appears to be related to the exposure duration and/or 10 the organism life stage. Hatchling stages indicated no concentration-response relationships, 11 whereas larval malformations at Gosner developmental Stages 20 to 24, and metamorphs 12 exhibited indications of toxicity (Figure 4.4-4). The malformation endpoint appeared sensitive 13 and was significantly correlated with sediment, water, and tissue tPCB concentrations (see Figure 4.4-5 and Figure 4.4-6). The highest sediment tPCB concentrations caused an order of 14 15 magnitude increase in the incidence of malformed metamorphs.

16 Cross-Over and Aroclor 1260-Spiked Treatments: These treatments confirmed the
 17 importance of vernal pool media as an exposure pathway:

- Reference site larvae raised in sediment and water from their native reference site locations (i.e., Sites WML-1 and WML-2) had low tissue PCB concentrations of 0.340 and 0.242 mg/kg, respectively, while larvae from the same reference sites raised in PSA vernal pool media (i.e., Sites 38-VP-1 and 38-VP-2) had tissue PCB concentrations of 6.61 and 7.82 mg/kg, respectively.
- PSA pool larvae raised in native media had tissue PCB concentrations of 4.66 and 6.61 mg/kg, respectively, while larvae from the same pools raised in reference site media had tissue PCB concentrations of 0.109 and 0.053 mg/kg, respectively. This indicates that uptake from sediment is more important than maternal transfer.

The cross-over study confirmed the overall findings of the Phase I main study, and indicated that mortality and metamorphosis endpoints were not significantly affected by PCB exposures, while moderate toxicity was observed for the malformation endpoint. The study indicated that tissue burdens in later larval and metamorph stages were more directly linked to contaminated exposure media (sediment, water) than to maternal transfer to the eggs. This finding has implications for the interpretation of other wood frog toxicity endpoints (particularly Phase III metamorphs).

1

2

3

4

5



Figure 4.4-4 Comparison of Phase I Larval Wood Frog Malformations as Gosner Developmental Stage 20-24 to Mean Sediment tPCB and Spatially Weighted Mean tPCB (FEL 2002a)



Figure 4.4-5 Incidence of Malformation in Phase I Wood Frog Metamorphs, with Mean Sediment tPCB and Spatially Weighted Mean tPCB (FEL 2002a)



Note: No tissue sample for 39-VP-1; sediment tPCB analyses for WML-1, WML-2, and WML-3 were non-detect (ND) – numbers shown represent detection limits (0.069, 0.13, and 0.11, respectively).

Figure 4.4-6 Incidence of Malformation in Phase I Wood Frog Metamorphs, Phase I Metamorph Tissue tPCB (FEL 2002a) Tissue PCB concentrations in the spiked and unspiked reference site treatment were 0.526 and 0.138 mg/kg PCB, respectively. The percentage of malformed metamorphs was higher in the spiked treatment than in the control treatment (11% vs. 0.5%), indicating that exposure to the spiked sediment had an adverse effect on metamorph malformation; however, larval mortality and the incidence of larval metamorphosis appeared unaffected.

6 Phase II: The Phase II larvae showed a similar pattern of responses to that of the Phase I 7 animals. While only growth and malformation were evaluated in Phase II, the effects for the two 8 endpoints were similar in the two study phases. Mean larval length at each station increased 9 between sampling events 1 and 3 (as the larvae grew); and mean length was similar between 10 PSA and reference pools, showing no apparent relationship to tissue PCB concentration. The 11 larval growth endpoint has not proven to be a strong indicator of adverse biological effects in 12 other studies; Berven (1990) found no significant relationship between juvenile size and adult 13 survival. However, as the exposure duration increased for the Phase II animals, the incidence of 14 malformation increased. The event 4 animals had the highest incidence of malformation, with 15 response showing a significant relationship to sediment tPCB concentration.

16 The findings of the Phase II study show that PCB accumulation and the incidence of 17 malformations increase with exposure duration.

18 Phase III: This phase of the study represented an in situ exposure, with the same pools visited 19 at larval metamorphosis as those where egg masses for Phase I were collected. Endpoints 20 included incidence of malformation, growth, and sex ratio (number of males to females). As 21 with the first two study phases, there was no significant relationship between metamorph weight 22 and sediment or tissue PCB concentration. However, the sex ratios changed with increasing 23 sediment concentration. The metamorph sex ratio (males:females) ranged from 0 (Site 8-VP-1, 24 24.6 mg/kg) to 1.0 (Site 23b-VP-1, 0.2 mg/kg) for the PSA pools, and from 0.62 to 1.0 for the 25 reference sites (0.07 and 0.11 mg/kg). In general, as the PCB concentration in sediment or tissue 26 increased, the proportion of males to females decreased. There was a significant correlation 27 between skewed sex ratios and sediment and tissue tPCB concentrations (Figure 4.4-7 and Figure 4.4-8). Berven (1990) found a juvenile sex ratio of 1:1 in a Maryland study of population 28 29 fluctuations in larval and adult wood frogs, suggesting that this ratio is biologically "normal."

1 The incidence of juvenile malformations was highest in this phase (both internal and external 2 malformations were assessed). Incidence of malformation was correlated with sediment and 3 metamorph tissue tPCB concentration. The percentage of malformed metamorphs ranged from 4 4.9% to 66.7% for the contaminated sites, and from 0 to 2.9% for the two reference sites (0.07 5 and 0.11 mg/kg). There was a significant relationship between metamorph malformation and 6 sediment and tissue tPCB concentrations (Figure 4.4-9 and Figure 4.4-10). Except for site 7 WML-1 (no malformed metamorphs, but with an anomalous tissue tPCB concentration), 8 treatments with the highest sediment or tissue tPCB concentrations also had the highest 9 percentages of malformed metamorphs.

The incidence of metamorph malformation is expected to be significant at the population level, as a high degree of malformations could lead to reduced population recruitment at local and regional scales (Ouellet 2000). The types of malformations observed in the metamorphs may affect survivorship by interfering with swimming, hopping, foraging, and predator avoidance (see following photos).

15	Summary of Wood Frog Toxicity
16	 No observed toxicity in egg mass viability; egg fertilization, hatching success, and
17	egg counts were unaffected by vernal pool tPCBs, or egg mass tissue tPCB
18	concentrations.
19	 Contaminant effects were not observed in early-stage juveniles, although high
20	mortality in the reference animals makes it difficult to assess the acute sensitivity of
21	the wood frogs. Incidence of metamorphosis appeared unaffected.
22	 Manifestation of effects increased with time spent in the vernal pools. Late-stage
23	larvae/metamorphs (laboratory-cultured and field-collected) had elevated levels of
24	both internal and external malformations, with magnitude of response related to
25	sediment and tissue tPCB concentrations.
26	 Metamorphs collected after in situ exposure in natal pools showed alteration in sex
27	ratio in relation to sediment and tissue tPCB concentrations.
28	



Figure 4.4-7 Ratio of Males to Females in Phase III Wood Frog Metamorphs, with Sediment tPCB and Spatially Weighted Mean tPCBs (FEL 2002a)



Figure 4.4-8 Ratio of Males to Females in Phase III Wood Frog Metamorphs, with Tissue tPCBs (FEL 2002a)



Figure 4.4-9 Percent Malformation in Phase III Wood Frog Metamorphs, with Sediment tPCBs (FEL 2002a)



Figure 4.4-10 Percent Malformation in Phase III Wood Frog Metamorphs, with Tissue tPCBs (FEL 2002a)



Example of Axial Flexure and Notochord Lesions



Example of Normal Tail

4.4.1.2.3 Wood Frog Study: GE

The study reported that there was not a statistically significant relationship between vernal sediment tPCB concentrations and juvenile tissue concentrations. This finding concurs with that

of the EPA study, wherein natal pool sediment tPCB was shown to be unrelated to egg mass tissue concentration.

MANOVA conducted on the grouped response variables of interest (juvenile survival and growth) showed a significant difference between the two ponds in which the experiment was conducted. However, this conclusion is difficult to interpret, as the sediment tPCB concentrations were so similar between ponds 23b-VP-1 and 23b-VP-2 (both < 1 mg/kg). Differences are more likely due to environmental conditions, as the pools varied both according to habitat type and size. Overall, there was a high degree of uncertainty associated with this study, due mostly to an inadequate evaluation of relevant exposure pathways and study duration.

4.4.2 PCB Effect Thresholds

Data were compiled on sediment and tissue PCB concentrations associated with lethal or sublethal effects in representative amphibians from the literature. The purpose was to estimate threshold tissue concentrations where adverse effects might occur in Housatonic River amphibians. The review focused on data for Aroclor 1254 and PCB-126 (considered one of the more toxic congeners) in addition to tPCBs. Studies evaluating Aroclor 1260 were not found in the literature, and studies with soil and sediment effects data were limited.

A total of five different species of frogs were used in the studies, including the leopard frog and wood frog. There were a total of 18 no-effect measurements (ranging from 0.02 to 11.2 mg/kg ww) and 11 effect measurements (ranging from 0.96 to 128 mg/kg ww). The majority of data applied to effects on growth, development, behavior, physiological, and cellular effects. Seven studies also evaluated mortality.

Figure 4.4-11 shows the distribution of no effect and effect tissue concentrations. No adverse effects were observed at tissue concentrations of 0.1 mg/kg ww, whereas above 1 mg/kg ww, the frequency of occurrence of adverse effects was more than 40%. There were six instances of adverse effects occurring between 1 and 10 mg/kg. Based on this distribution, it is unlikely that adverse effects will occur at tissue concentrations below 1 mg/kg, and it is likely that they will occur above 10 mg/kg.

Overall, the upper and lower bounds of the effect concentration ranges from the EPA wood frog study closely match those derived from the literature. Three endpoints indicated a consistent relationship between tissue tPCB concentrations and adverse effects: metamorph malformations in Phase I and Phase III and skewed sex ratios in the Phase III metamorphs. Specifically, 1 mg/kg was the approximate tissue concentration where ecologically significant adverse effects began to occur, and responses became frequent and more severe at approximately 10 mg/kg.

No tissue effects threshold could be established for the leopard frogs, due to the difficulty in establishing relevant biological linkages between tissue data and effects endpoints. However, the leopard frogs appeared more acutely sensitive than the wood frogs.

In addition, protection of urodels (salamanders) also was considered in the derivation of a sitespecific tissue effects threshold. These animals sometimes spend almost their entire lives in the vernal pools if they are a facultative neotonic species (i.e., fails to complete metamorphosis). Given the increased sensitivity of the leopard frogs relative to the wood frogs, and the possibility of neotony in the salamanders (and thus a much longer exposure period than would be typical for the ranids), some conservatism was applied in the derivation of the 1 mg/kg tPCB tissue effects threshold concentration.



Figure 4.4-11 Summary of Available Literature Effects Data on PCB Tissue Residues in Anuran Amphibians

1 4.5 RISK CHARACTERIZATION

The risk characterization evaluates the likelihood that adverse effects may occur as a result of amphibian exposure to tPCBs and/or other COCs. Three broad categories of measurement endpoints in the Housatonic River amphibian risk assessment were used to develop the risk characterization:

- Endpoints based on field surveys (i.e., amphibian community structure) For these
 endpoints, care was exercised to discriminate, to the extent possible, between
 responses related to COCs and those related to other factors such as substrate or
 habitat type.
- Endpoints based on site-specific toxicity study results These endpoints (e.g., toxicity tests involving both in situ and laboratory exposures) directly evaluated biological responses to COCs.
- Endpoints that compared field-measured exposures to effects levels or benchmarks –
 For these endpoints, the risk characterization integrated exposure and effects by
 relating the two terms quantitatively (e.g., hazard quotient [HQ] method for tissue
 chemistry data and derivation of concentration-response relationships for toxicity
 data).

18 These three categories of endpoints were independent, allowing for a robust weight-of-evidence

19 (WOE) assessment of the potential for risk using the approach of Menzie et al. (1996).

All three lines of evidence suggested some degree of harm to amphibians in the Housatonic River. In addition, for each line of evidence, there were indications that PCBs are primarily responsible for the observed patterns of responses.

A WOE assessment was conducted to combine the results from each line of evidence. This included a station-by-station assessment of each amphibian sampling location, as well as an overall WOE assessment for the assessment endpoint. The section concludes with a discussion of sources of uncertainty in the assessment of risks of COCs to amphibians and the conclusions of the risk characterization.

Much of the risk characterization that follows is devoted to quantifying the relationship between tPCB exposure concentrations and corresponding effects to amphibians. The formal concentration-response analyses (for toxicity endpoints) strengthen the findings of the exposureresponse relationships identified in Section 4.5. Various statistical methods were applied, with a
 resulting level of concordance supporting the risk conclusions.

3 4.5.1 Concentration-Response Analysis – Toxicity Test Endpoints

A statistical assessment was conducted to quantify the relationship between toxicity test endpoints and COC concentrations measured concurrent with the wood frog study. The assessment focused on the relationship between PCBs and toxicity endpoints, since other lines of evidence indicated a high probability that PCBs (as opposed to other COCs) were a causal agent for toxicity to amphibians within the Housatonic River PSA.

9 Endpoints Selected for Concentration-Response Analysis 10 Regardless of study phase, the late larval/metamorph endpoints were consistently 11 correlated with contaminant media concentrations. Therefore, the following endpoints 12 were selected for the more detailed statistical assessment: 13 Phase I metamorph percent malformed larvae (compared to sediment and Phase I 14 metamorph tissue tPCB concentrations). 15 Phase III percent malformed metamorphs (compared to Phase III metamorph tissue 16 and sediment tPCB concentrations). 17 Phase III metamorph sex ratio (proportion of females) (compared to sediment tPCBs 18 and Phase III metamorph tissue tPCB concentrations).

19

20 **4.5.1.1** Calculation of Individual Toxicity Test Endpoints

21 Comparisons based on magnitude of effects for various endpoints deemed biologically relevant 22 were considered. Effects observed at frequencies of 20% and 50% were selected as indicators of 23 moderate and major toxic effects, respectively.

Three sets of exposure data (two sediment, one tissue) were used to evaluate tPCBs concentration-response relationships. Summary metrics (e.g., EC_{20} , EC_{50}) were calculated for each endpoint based on sediment tPCB concentrations measured concurrent with the tests, and also with spatially weighted sediment tPCB concentrations. In addition, tissue tPCB concentrations were compared to effects.

Calculation of EC_{50} and EC_{20} values (with their corresponding 95% confidence limits) was conducted using a linear probit method. If the probit model was not appropriate for the data 1 (based on a goodness-of-fit test), the EC_{50} value was estimated by the nonparametric statistical

2 procedure, the Trimmed Spearman-Karber method.

3 **4.5.1.2** Results of Concentration-Response Analysis

4 **4.5.1.2.1 Sediment**

Ecologically significant adverse effects in late stage juvenile wood frogs occurred in the
sediment tPCB concentration range of 9.54 to 59.3 mg/kg, although responses of lesser
magnitude, yet statistically significant, were observed at 0.52 mg/kg tPCBs and lower. MATC
of 3 mg/kg was established for sediment.

Estimated Toxicity Threshold Values Most endpoints followed a fairly smooth (typically sigmoidal) concentration-response, which could be fit using the probit model.

- Concordance was observed among endpoints for sediment concentrations causing significant effects (i.e., 50% responses occurred at sediment tPCB concentrations of 9.54 to 59.3 mg/kg).
- Concordance was observed among endpoints for tissue residues causing significant effects (50% responses occurred at tissue tPCB concentrations of 3.09 to 6.54 mg/kg).

18 **4.5.1.2.2** Tissue

.

19 The threshold concentration range for amphibian tissues was 0.60 mg/kg to 6.54 mg/kg tPCB, 20 and was based on the sex ratio endpoint (both an EC_{20} and EC_{50}) and the Phase III metamorph 21 malformation endpoint (an EC_{50} point estimate). As there was not a 20% effect size for 22 malformations in the Phase III metamorphs, a tissue EC_{20} could not be calculated. Tissue 23 concentrations below 1 mg/kg are not expected to cause biologically significant adverse 24 responses in the wood frogs. The tissue concentration-response modeling predicted significant 25 risk in the range of 1 to 10 mg/kg. At tissue concentrations >10 mg/kg, adverse ecological 26 effects are expected to occur with certainty.

1 4.5.2 Biological Community Endpoints

2 4.5.2.1 Amphibian Community Evaluation: EPA

3 Population responses of amphibians were measured in field studies of amphibian communities 4 conducted in 1999 and 2000 (Woodlot Alternatives, Inc. 2003). Detailed data were collected for 5 wood frogs (e.g., numbers of frogs entering and leaving pools, numbers of metamorphs captured leaving the pools). In addition, species abundance, richness, and presence of malformations 6 7 were assessed for multiple species in selected vernal pools. Data describing the dominant plant 8 communities, dominant plants per community, soil, and general site hydrology were collected for 9 the entire PSA, as well as each amphibian sampling area (Appendix A). Descriptive information 10 on more than 60 amphibian breeding sites (e.g., size of vernal pools, average depth, percent 11 shading, and amphibian species observed breeding), and species observed at each site, is included in the ecological characterization. Although most of these data were not collected 12 13 directly in conjunction with effects data, they were used in design of the subsequent amphibian 14 developmental studies, and were used to characterize the relative abundance of physically 15 suitable breeding sites for both leopard and wood frogs.

16 The findings included:

- Species richness was lower in the vernal pools with higher average sediment tPCB concentrations; 6 in 8-VP-2 (55 mg/kg tPCBs), 8 in 38-VP-2 (32.3 mg/kg tPCBs), 8 in 8-VP-1 (24.6 mg/kg tPCBs), and 11 in 46-VP-5 (0.72 mg/kg tPCBs). Overall, density and biomass (on a per m² basis) were lower in the more contaminated vernal pools; 0.5 g/m² wood frogs in 8-VP-2 versus 10.7 g/m² wood frogs in 46-VP-5.
- Salamanders appeared to be sensitive to tPCBs, appearing in lower numbers in vernal pools with high sediment tPCB concentrations. Salamander species observed included the Jefferson salamander (*Ambystoma jeffersonianum*) and the four-toed salamander (*Hemidactylium scutatum*), both of which are Species of Special Concern in Massachusetts.

Gross malformation rates in adults (wood frogs and spotted salamanders) and metamorphs (wood frogs) were low. However, malformation rates in larval wood frogs were high in all pools. The malformation rates in the pools were 46% in 8-VP-1 (24.6 mg/kg tPCBs), 35% in 38-VP-2 (32.3 mg/kg tPCBs), and 30% in 46-VP-5 (0.72 mg/kg tPCBs).

1 4.5.2.2 Leopard Frog Egg Mass Survey: GE

In the spring of 2003, ARCADIS G&M, Inc. (ARCADIS) conducted a survey of leopard frog egg masses occurring in the vernal pool and backwater habitats of the PSA. The primary objective of the survey was to determine whether adult leopard frogs are reproducing successfully in the PSA; the metric chosen for evaluation of leopard frog reproductive health was the presence/absence of egg masses within breeding habitats.

The investigators examined 44 ponds within the PSA for the presence of leopard frog eggs and found egg masses in 17 ponds (a total of 216 egg masses). The study concluded that there was no relationship between vernal pool sediment tPCB concentration and the presence/absence of egg masses. In addition, the investigators concluded that there was no evidence of reproductive impairment in leopard frogs within the PSA.

4.5.2.3 Amphibian Community Measures Observed During Developmental Study Field Sampling: EPA

14 Additional evidence for population responses of amphibians was derived from anecdotal 15 information from field studies collected in support of the FEL developmental studies. No egg 16 masses were found at three of the leopard frog sampling areas and one of the wood frog vernal 17 Sediment tPCB concentrations at these areas were among the highest of the pools. 18 concentrations measured for the two studies: between 50 and 160 mg/kg. In addition, female 19 leopard frogs were not found at three contaminated sampling areas. Sediment PCB 20 concentrations at two of these areas were over 100 mg/kg tPCBs.

21 **4.5.3** Comparison of Tissue Chemistry Data to Benchmarks

As an additional line of evidence, hazard quotients (HQs) were used to quantify the degree to which amphibian tissue COC concentrations exceeded the literature-based and site-specific thresholds deemed protective of assessment endpoints. In theory, adverse ecological responses are possible if any HQ exceeds 1.0 (i.e., if exposure exceeds the lower threshold level). Separate HQs were calculated for each tissue type and species. Tissue HQs were based on comparison of observed tissue residues to an effects threshold of 1 mg/kg tPCBs, which represents a conservative interpretation of the LOAELs at which significant adverse effects were found in the
 literature.

3 4.5.3.1 Leopard Frog HQs

4 HQs for leopard frogs could not be derived using site-specific leopard frog effects data due to the 5 modification to the study design based on the field conditions (i.e., leopard frog tissue burdens 6 and effects data were not synoptic or biologically related). However, HQs were calculated based 7 on comparison to literature-derived effects thresholds, which in turn are supported by the wood 8 frog study effects threshold. For the purposes of this exercise, it was assumed that there was a 9 similar sensitivity of the two representative species (although it is likely that the leopard frog is 10 more sensitive, as discussed in Section 4.4.1.2). The LOAEL of 1 mg/kg in tissue was 11 compared to tissue concentrations. Table 4.5-1 presents the range of HQs for leopard frog 12 tissues, using the literature-derived LOAEL. The tissue types evaluated included adults, 13 metamorphs, and egg mass/ovaries.

Based on the comparison to the LOAEL and the 10 mg/kg effects threshold, the abundance of tissue HQs between 1 and 10 indicate a strong likelihood for adverse effects. Site-specific reproductive and developmental effects clearly support this LOAEL for tissue tPCBs.

17 4.5.3.2 Wood Frog HQs: EPA Study

Wood frog HQs based on concentrations of tPCBs measured in the egg mass were relatively low. Only two stations (18-VP-2 and 23b-VP-2) had a HQ greater than 1.0, and not by a large amount. These HQs reflect a PCB exposure attributable to maternal transfer of PCBs. The lowto-marginal HQs indicate that the chemical hazard for this life stage is fairly low. This finding is consistent with the lack of significant toxicity observed in the egg mass toxicity endpoints, such as hatching success, percent fertilization, and percent necrotic eggs.

Tissue HQs based on Phase II wood frog tadpoles (event 3; approximately 9 to 12 weeks old)
were variable, ranging between <0.1 and 10. The three stations with the highest sediment tPCB
concentrations (14.5 to 62 mg/kg) had HQs greater than 1.0 (8-VP-1, 38-VP-1, and 38-VP-2).

Hazard Quotients for Leopard Frog PCB Tissue Residues, Based on Literature-Derived Effects Thresholds

Sampling Area ID	Life Stage or Tissue Type	HQ				
	Adult chemical analysis (whole body)	0.03				
Muddy Pond Reference	Adult experimental (female whole body minus ovaries/egg masses)	0.012				
	Ovary/egg mass (from adult experimental female) ^a	0.024				
	Larvae-to-metamorphs ^b					
	Adult chemical analysis (whole body)	0.15				
W-1	Adult experimental (female whole body minus ovaries/egg masses)	0.023				
	Ovary/egg mass (from adult experimental female) ^a	0.26				
	Larvae-to-metamorphs	NA				
	Adult chemical analysis (whole body)	0.34				
W-4	Adult experimental (female whole body minus ovaries/egg masses)	NA				
	Ovary/egg mass (from adult experimental female) ^a	NA				
	Larvae-to-metamorphs ^b	1.4				
	Adult chemical analysis (whole body)	3.59				
W-9a	Adult experimental (female whole body minus ovaries/egg masses)	1.24				
	Ovary/egg mass (from adult experimental female) ^a	5.05				
	Larvae-to-metamorphs ^b	NA				
W-7a	Adult chemical analysis (whole body)	2.11				
	Adult experimental (female whole body minus ovaries/egg masses)	1.4				
W-7a	Ovary/egg mass (from adult experimental female) ^a	6.61				
	Larvae-to-metamorphs ^b	1.11				
	Adult chemical analysis (whole body)	4.26				
EW-3	Adult experimental (female whole body minus ovaries/egg masses)	1.23				
	Ovary/egg mass (from adult experimental female) ^a	1.52				
	Larvae-to-metamorphs ^b	0.96				

Hazard Quotients for Leopard Frog PCB Tissue Residues, Based on Literature-Derived Effects Thresholds (Continued)

Sampling Area ID	Life Stage or Tissue Type	HQ
	Adult chemical analysis (whole body)	1.31
E-5	Adult experimental (female whole body minus ovaries/egg masses)	NA
	Ovary/egg mass (from adult experimental female) ^a	NA
	Larvae-to-metamorphs ^b	NA
	Adult chemical analysis (whole body)	1.78
W-6	Adult experimental (female whole body minus ovaries/egg masses)	0.386
	Ovary/egg mass (from adult experimental female) ^a	9.45
	Larvae-to-metamorphs ^b	0.67
	Adult chemical analysis (whole body)	5.39
W-8	Adult experimental (female whole body minus ovaries/egg masses)	NA
	Ovary/egg mass (from adult experimental female) ^a	NA
	Larvae-to-metamorphs	NA
	Adult chemical analysis (whole body)	3.10
E-1	Adult experimental (female whole body minus ovaries/egg masses)	NA
	Ovary/egg mass (from adult experimental female) ^a	NA
	Larvae-to-metamorphs	NA

Sampling areas arranged in order of increasing sediment PCB concentration.

NA = No sample available because specimens were not found.

^aEgg mass/ovary HQs based on a geometric mean of the two tissue concentrations per station. This was done because of the large difference between the two concentrations for a given station.

^bHQs for larvae-to-metamorph samples cannot all be compared to one another, as the specimens were not all the same age when the samples were collected. Animals from sampling areas W-6, W-4, and EW-3 are comparable; animals from sampling areas W-7a and W-1 are comparable.

Phase III wood frog metamorphs (12 to 15 weeks old) had tissue HQs exceeding 1.0 in several samples, with maximum HQs above 10. This phase represented sediment exposure over the entire juvenile period, and exhibited the most pronounced toxicological responses. The reference tissue concentration for station WML-1 was not included in the HQ calculations.

5 4.5.4 Integrated Station-by-Station Assessment

6 Potential impacts of contaminated sediment to local amphibians at each location were assessed 7 using a graphical approach that considered multiple lines of evidence (Table 4.5-2, leopard frogs; 8 Table 4.5-3, wood frogs). Multiple measurement endpoints were included, and the results of 9 each were integrated into a single conclusion regarding potential ecological impacts. For the 10 purposes of evaluating each measurement endpoint, results were categorized and simplified 11 based on ecologically-based decision criteria. The decision criteria used to make the evaluations 12 are summarized in Appendix E.

In summary, there was evidence for ecological effects for both acute and chronic developmental endpoints in the leopard frog study and for several important developmental endpoints in the wood frog study. For both amphibian developmental studies, there are multiple indications of significant risk at multiple stations. There was a high degree of overall concordance among the late-larval/pre-metamorph stage endpoints.

4.5.5 Weight-of-Evidence Procedure for Assessing Risk from PCBs in the Housatonic River PSA

A formal WOE process was applied to determine whether PCBs pose a significant risk to the Housatonic River benthos. The three-phase approach of Menzie et al. (1996) and the Massachusetts WOE Workgroup was applied for this purpose, in which WOE was developed using the following three characteristics: (1) the weight assigned to each measurement endpoint; (2) the magnitude of response observed in the measurement endpoint; and (3) the concurrence among outcomes of the multiple measurement endpoints.

Integrated Assessment of Potential for Adverse Impacts to Amphibian Populations (Leopard Frog Study)

	Sediment tPCB (mg/kg)					
Sampling Area	Sampling Area tPCB	Spatially Weighted tPCB	Adult Reproductive Health ^a	Larval Development ^b	Tissue Concentration	Overall Rating
W-1	0.15	0.4	0	•	\odot	0
W-4	0.46	0.4	•	•	0	•
W-9a	4.3	7.5	•	NA	•	•
W-6	42	21	•	•	0	•
EW-3	30	23.8	•	×	0	•
E-1	160	26.6	•	NA	0	•
W-7a	18	27.6	0	•	•	•
W-8	120	43.5	•	NA	0	•

Sampling areas sorted by spatially weighted tPCB concentration.

• Negligible-to-low toxicity: negligible indication of ecological risk. No exceedances of tissue benchmark (1 mg/kg tPCB).

• = Moderate toxicity; ecological effects possible, but not conclusive. At least 1 exceedance of tissue benchmark.

• = High toxicity; strong indication of potential ecological effects. At least 1 tissue concentration is $\geq 10x$ the tissue benchmark.

 \bigstar = Very strong toxic response.

^aIncludes 6 endpoints: Adult body weight (male and female), sperm count, % abnormal sperm heads, egg count, % mature oocytes. ^bIncludes 5 endpoints: Larval growth, % metamorphosis, % malformation, growth, and days to reach Gosner Stage 26±1.

Integrated Assessment of Potential for Adverse Impacts to Amphibian Populations (Wood Frog Study)

	Sediment tPCB (mg/kg)							
Sampling Area ID	VP PCB	Spatially Weighted tPCB	Egg Mass ^a	Early larvae (up to Gosner Stage 20-24) ^b	Mid- to Late-stage larvae (after Gosner Stage 24) ^c	Metamorph ^d	Tissue tPCB	Overall Rating
23b-VP-1	0.19	0.2	\odot	\odot	0	\odot	0	\odot
23b-VP-2	0.11	0.3	0	\odot	Θ	Θ	0	\odot
46-VP-5	2.18	0.7	0	\odot	Θ	Θ	0	\odot
46-VP-1	0.5	0.8	\odot	\odot	0	Ο	0	\odot
18-VP-2	6.05	4.9	0	Θ	Θ	•	0	0
8-VP-1	14.5	24.6	0	0	0	•	•	•
38-VP-1	28	28.5	\odot	0	Θ	•	•	•
38-VP-2	62	32.3	0	0	Θ	•	0	0
39-VP-1 ^e	52	43	NA	NA	NA	NA	NA	NA

Sampling area sorted by spatially weighted tPCB concentration.

• Negligible-to-low toxicity: negligible indication of ecological risk. No exceedances of lower or upper tissue benchmarks (1 and 10 mg/kg tPCB).

• = Moderate toxicity; ecological effects possible, but not conclusive. At least 1 exceedance of lower tissue benchmark.

• = High toxicity; strong indication of potential ecological effects. At least 1 exceedance of upper tissue benchmark.

 \mathbf{x} = Very strong toxic response.

^aIncludes 4 endpoints: Phase I egg mass weight (total), % fertilized, %viable, and % hatching success.

^bIncludes 4 endpoints: Phase I early larval malformation (Gosner Stage 20-24); Phase II, Event 1 and 2 larval abundance, % malformed and growth.

^cIncludes 4 endpoints: Phase I larval mortality at day 95; Phase II, Event 3 and 4 larval abundance, % malformed and growth.

^dIncludes 3 endpoints: Phase I malformed metamorphs, Phase III malformed metamorphs, Phase III sex ratio.

^eNo frogs were found in this pool for collection and subsequent study.

1 The rationale for weighting of measurement endpoints is provided in Appendix E, along with a 2 discussion of attributes considered. A summary of the weighting for each attribute is provided in 3 Table 4.5-4. The chemistry endpoints yielded the lower overall values due to low-to-moderate 4 site specificity and some uncertainty with the linkage between the measurement endpoints and 5 the assessment endpoint(s). There is a stronger biological linkage to effects expected when 6 exposures are considered at the organism tissue level (i.e., incorporation of bioavailability). The 7 toxicity testing endpoints yielded the highest overall weighting, due to the site specificity and 8 high degree of biological relevance in the reproductive endpoints. The three field studies of 9 biological community endpoints had intermediate values. Although these endpoints were highly 10 site-specific and were direct measures of the assessment endpoint(s), confounding effects of 11 other environmental factors and lack of a quantitative assessment method reduced the utility of 12 these endpoints.

The magnitude of the response in the measurement endpoint is considered together with the measurement endpoint weight in judging the overall WOE (Menzie et al. 1996). This requires assessing the strength of evidence that ecological harm has occurred, as well as an indication of the magnitude of response, if present. The weighting values, evidence of harm, and magnitude of response were combined in a matrix format and are presented in Table 4.5-5.

A graphical method was used for displaying the degree of concurrence among measurement endpoints (Table 4.5-6). The 12 symbols representing the chemistry (C), wood frog toxicity (W), leopard frog toxicity (L), field biology (B), and (P) population model endpoints were displayed in a matrix, with the weight of the measurement endpoint and the degree of response as the axes.

The resulting plots show that 9 out of the 12 endpoints indicated some degree of risk. The potential for the two GE studies to determine risk to amphibians was judged to be undetermined due to limitations in the study designs. The only endpoint that did not indicate potential risk was the earliest life stage wood frog toxicity endpoint, for which there is mechanistic explanation for the lack of response. The plots also indicate that four endpoints exhibited a high degree of risk combined with a moderate to high confidence rating.

Weighting of Measurement Endpoints for Amphibian Weight-of-Evidence Evaluation

Measurement Endpoints	Endpoint Group: Chemistry	En	dpoint Group: Wo	od Frog Toxicology	· (W)	En	dpoint Group: Leop	pard Frog Toxicolog	gy (L)	E	ndpoint Group: Bi	blogy
Attributes	C (tissue)	W-1 (hatchling)	W-2 (larvae)	W-3 (metamorph)	W-4 (GE juveniles)	L-1 (hatchling)	L-2 (larvae)	L-3 (metamorph)	L-4 (adult)	B-1 (community)	B-2 (GE egg mass survey)	B-3 (anecdotal)
I. Relationship between Measurement and Asse	ssment Endpoints											
1. Degree of Association	Mod	Mod/High	Mod/High	Mod/High	Low	Mod/High	Mod/High	Mod/High	Mod/High	Mod/High	Mod	Mod/High
2. Stressor/Response	Mod	Mod/High	Mod/High	Mod/High	Low	Mod/High	Mod/High	Mod/High	Mod/High	Mod	Low	Low/Mod
3. Utility of Measure	Mod	Mod	Mod/High	Mod/High	Mod	Mod	Mod/High	Mod/High	Mod	Low/Mod	Low	Low/Mod
II. Data Quality												
4. Data Quality	Mod/High	Mod/High	Mod/High	Mod/High	Low	Mod/High	Mod/High	Mod/High	Mod/High	Mod/High	Mod	Mod/High
III. Study Design												
5. Site Specificity	Low/Mod	Mod/High	Mod/High	Mod/High	Low/Mod	Mod	Mod	Mod	Mod	High	Mod/High	Mod/High
6. Sensitivity	Mod	Mod	Mod/High	Mod/High	Low	Mod	Mod	Mod	Mod	Mod	Low	Low/Mod
7. Spatial Representativeness	Mod	High	High	High	Low	Mod/High	Mod/High	Mod/High	Mod/High	Low	Mod/High	Mod
8. Temporal Representativeness	Mod	Mod/High	Mod/High	Mod/High	Mod	Mod/High	Mod/High	Mod/High	Mod/High	Mod/High	Mod	Mod/High
9. Quantitative Measure	Mod	High	High	High	Low	Mod/High	Mod/High	Mod/High	Mod	High	Low	Low
10. Standard Method	Mod/High	Mod/High	Mod/High	Mod/High	Low	Mod/High	Mod/High	Mod/High	Mod/High	Mod/High	Mod/High	Mod/High
Overall Endpoint Value	Mod	Mod/High	Mod/High	Mod/High	Low	Mod/High	Mod/High	Mod/High	Mod/High	Mod/High	Low/Mod	Mod

C. Chemical Measures

C. Concentration of PCB in frog tissues in relation to levels reported to be harmful to amphibians

W. Wood Frog Toxicological Measures

W-1. Sediment toxicity to hatchling/late embryo life stages

W-2. Sediment toxicity to larval life stages

W-3. Sediment toxicity to late larval/metamorph life stage

W-4. GE Context-Dependent Wood Frog Study (hatchlings, tadpoles, and metamorphs evaluated)

L. Leopard Frog Toxicological Measures

L-1. Sediment toxicity to hatchling/late embryo life stages

L-2. Sediment toxicity to larval life stages

L-3. Sediment toxicity to late larval/metamorph life stage

L-4. Sediment toxicity to adult leopard frogs (reproductive health)

B. Biology

B-1. Vernal pool community study

B-2. GE Leopard frog egg mass survey

B-3. Anecdotal observations during collections for reproductive stud

Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of Amphibian Populations in the Lower Housatonic River

Measurement Endpoints	Weighting	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Moderate, Low)
C. Chemical Measures			
C. Concentration of PCB in frog tissues in relation to concentrations reported to be harmful to amphibians.		Yes	Low
W. Wood Frog Toxicological Measures			
W-1. Sediment toxicity to hatchling/late embryo life stages.	Mod/High	No	-
W-2. Sediment toxicity to larval life stages.	Mod/High	Yes	Moderate
W-3. Sediment toxicity to late larval/metamorph life stage.	Mod/High	Yes	High
W-4. GE Study (juvenile wood frogs)		Undetermined	-
L. Leopard Frog Toxicological Measures			
L-1. Sediment toxicity to hatchling/late embryo life stages.	Mod/High	Yes	Low
L-2. Sediment toxicity to larval life stages.		Yes	High
L-3. Sediment toxicity to late larval/metamorph life stage.		Yes	High
L-4. Sediment toxicity to adult leopard frogs (reproductive health).		Yes	High
B. Biology			
B-1. Vernal pool community study.	Mod/High	Yes	Low
B-2. GE leopard frog egg mass survey	Low	Undetermined	-
B-3. Anecdotal observations during collections for reproductive study.	Moderate	Yes	Low

Risk Analysis for Amphibians Exposed to tPCBs and Other COCs in the Housatonic River PSA

Assessment Endpoint: Community condition, survival, reproduction, development, and maturation of amphibians

	Weighting Factors (increasing confidence or weight)							
Low	Low/Moderate	Moderate	Moderate/High	High				
			L-2, L-3, L-4, W-3					
			W-2					
		C, B-3	L-1, B-1					
W-4, B-2								
			W-1					
	Low	Low Low/Moderate Weighting Factors Weighting Factors Work Weighting Factors Weighting Factors	Weighting Factors (increasing confidence Low Low/Moderate Moderate	Weighting Factors (increasing confidence or weight) Low Low/Moderate Moderate Moderate/High Low Low/Moderate Moderate Moderate/High Low V L-2, L-3, L-4, W-3 W-4, B-2 C, B-3 L-1, B-1 W-4, B-2 W-4 W-1				

C = Chemistry (tissue).

W = Wood frog study (1 = hatchling, 2 = larvae, 3 = metamorphs, 4 = GE Study).

L = Leopard frog study (1 = hatchling, 2 = larvae, 3 = metamorphs, 4 = adult).

B = Field study (1 = community, 2 = GE egg mass survey, 3 = anecdotal).

The conclusion from interpretation of Table 4.5-6 is that there is a significant risk to amphibians as indicated by the preponderance of the evidence and the relative weights of the measurement endpoints. The "no harm" value of measurement endpoint W-1 does not diminish the overall conclusion, because the study demonstrated that the embryo/early larval life stages are fairly insensitive to the effects of maternally transferred PCBs relative to later juvenile life stages exposed to contaminated media.

7 4.5.6 Sources of Uncertainty

8 **4.5.6.1 EPA Studies**

9 The assessment of risks to amphibians in the PSA contains uncertainties, which can influence 10 overall conclusions of risk. Uncertainty associated with the assessment of risk of PCBs and 11 other COCs to amphibian receptors are described below.

- 12 The greatest uncertainties in the exposure assessment in the amphibian ERA are (1) 13 the mobility of the animals and their exposure to concentrations that are not known, 14 and (2) the potential for small-scale variability (as was observed in PCB concentrations in the river main channel sediment) in exposure concentrations in the 15 sampling areas. These two factors can confound the extrapolation of the quantitative 16 17 concentration-response relationships to the spatial scale of the PSA and its associated 18 backwater habitats. To overcome these uncertainties, spatially-weighted data were 19 used to determine an overall average exposure concentration for the leopard frog and 20 wood frog sampling areas, and for many of the vernal pools within the PSA.
- There is some uncertainty associated with the range of effects thresholds in the literature and, therefore, with the selection of the tissue effects threshold. However, studies within the literature do not exist from which definitive generalizations regarding amphibian sensitivity to chlorinated organic contaminants can be made, either for the class itself or for an individual genera or species. Therefore, the literature review served as a supplement to the site-specific developmental studies, but not to supplant the study results.
- The tissue effect threshold was calculated to provide a general indication of risk . The possible increased sensitivity of the leopard frogs and salamanders (see Section E.3.6.4) relative to the wood frogs outweighs the uncertainty associated with the derivation of the literature effects threshold. Overall, added conservatism in development of a tissue effects threshold for amphibians within the PSA is warranted.
- There were some unusual data observations that were evaluated in the exposure assessment:

1 2 3 4 5 6	- The observation of elevated PCB concentrations in a wood frog Phase III metamorph sample (WML-1; 4.3 mg/kg) collected at a reference location is anomalous. All other reference sample concentrations were ≤0.4 mg/kg, and sediment PCB concentrations from this area were non-detect. Statistical analyses were conducted both with and without this anomalous value to evaluate the potential effect of this value.
7 8 9	- The lipid content of the wood frog Phase I egg mass samples were variable. A number of lipid measurements were 0.1%, while other samples collected from the same location yielded lipid contents as high as 1.5% (i.e., a 15-fold difference).
10 11 12 13 14 15 16 17	- Several of the higher biota-sediment accumulation factor (BSAF) values in the frog studies appear to be driven by the low lipid contents (0.1%) in the samples, resulting in higher BSAFs than would be predicted on the basis of theoretical equilibrium partitioning or observed at other PCB-contaminated sites. When central tendency values (i.e., 1.0%; comparable to the lipid contents observed in many of the tissue samples) are substituted, the BSAFs are more plausible. This suggests that the low lipid content values may be underestimates, although the variability in sediment PCB concentrations must also be considered.
18 19	- Detection limits for PAH compounds in the wood frog sediment data were high, particularly for reference locations.
20 21	- Detection limits for metals (vanadium and nickel) in the wood frog tissue data were also high.
22 • 23 24 25 26	There is some uncertainty associated with the wood frog mortality endpoint. The poor performance of the reference animals could be masking a natural sensitivity in the wood frogs with respect to mortality because mortality in PSA animals was high (greater than 50% at five stations). Thus, the sediment threshold effect concentrations may be overly conservative with respect to the acute sensitivity of wood frogs.
27 28 29 30 31 32 33 34 35	In addition, there is some uncertainty associated with the lack of replication in the leopard frog data. As the study progressed, there were simply not enough test organisms available for adequate replication (due to failed fertilization of the field-collected adult females and limited availability of field-collected egg masses for laboratory culture). Lack of replication prohibited the use of quantitative inferential statistics to determine relationships between contaminant exposure concentrations and response variables of interest. However, the magnitude of impacts observed in the target animals (high mortality, low incidence of metamorphosis) helps to reduce the uncertainty associated with limited replication.
36 • 37 38 39 40	Based on the above-noted uncertainty associated with the three site-specific amphibian developmental studies, extrapolation to the level of population is uncertain. The wood frog Phase I metamorph malformation endpoint is suspected of contributing to reduced young-of-year recruitment. However, relationships between juvenile malformations and mortality need to be better defined because the mortality

data revealed little difference between the reference and PSA animals. Still, the high incidence of both internal and external malformations in the Phase III metamorphs indicates that metamorph recruitment to the population may be affected by exposure to COCs.

- 5 Skewed sex ratios observed in the wood frog study could be affecting the frog . population. Berven (1990) conducted a large-scale study to examine the factors 6 7 affecting population fluctuations in larval and adult stages of the wood frog in 8 Maryland. The study determined the sex ratio of recently metamorphosed juveniles 9 to be approximately 1:1 (male:female). The ratio of breeding adults, however, 10 averaged 3.1:1 (male: female. This skewed sex ratio was attributed to the majority of female frogs breeding at age two, while male frogs started breeding at age one. The 11 females breeding in their second year were exposed to an additional year of mortality 12 13 than males, resulting in 2.3 times as many males as females from a given clutch 14 surviving to breed.
- 15 The Phase III wood frog metamorph results for the Housatonic sampling exhibited a range of sex ratios. Sediment in the two most contaminated vernal 16 pools (38-VP-1 and 38-VP-2) contained PCB concentrations of 28.5 and 32.3 17 PCBs based on spatial weighting of sediment data. 18 These two pools also 19 exhibited the lowest metamorph sex ratios at 0.25 and 0.24 (male:female). 20 Another pool with sediment PCB concentrations of 24.6 mg/kg (8-VP-1) had 21 entirely females, although there were only three individual metamorphs captured. 22 The two vernal pools (23b-VP-1 and 23b-VP-2) with the lowest PCB concentrations (0.2 and 0.3 mg/kg tPCB, respectively) had sex ratios of 1.00 and 23 24 0.65 (male:female). The two reference sites (WML-1 and WML-3) had 25 metamorph sex ratios of 0.62 and 1.00 (male:female), respectively.
- 26 Sex ratios were also determined for breeding adult wood frogs in four pools 27 within the Housatonic River study area (Woodlot Alternatives, Inc. 2003). The 28 two vernal pools with the highest contaminations in this study (38-VP-2 and 8-29 VP-2) contained soil tPCB concentrations of 32.3 and 54.9 mg/kg, respectively. 30 The corresponding sex ratio of breeding wood frogs from these pools was 1.5:1 and 0.9:1 (male:female). Vernal pool 8-VP-1 had tPCB concentrations of 24.6 31 mg/kg and a corresponding sex ratio of breeding wood frogs of 0.8:1 32 (male:female). In the pool with the lowest contamination (46-VP-5, 0.59 mg/kg 33 34 tPCB), breeding wood frogs had a sex ratio of 1.3:1 (male:female).
- 35 The Housatonic River vernal pool sex ratio data for wood frog metamorphs and 36 breeding adults exhibit strong differences from Berven's data at a non-37 contaminated site. The general trend for the wood frogs examined near the 38 Housatonic River PSA is a marked decrease in the male to female ratio in both 39 metamorphs and breeding adults. This feminization of the wood frogs in this 40 study may be adversely impacting the local population. Hayes (2000) reports that 41 alterations of sex ratios in amphibians may result in decreased recruitment and 42 population declines in what otherwise appear to be normal healthy adults. 43 Studies have also found that breeding between normal and sex-reversed adults

1

2

3

4

can lead to even more skewed sex ratios (Mikamo and Witschi 1964; Richards and Nace 1978, in Hayes 2000).

- Reeder et al. (1998) examined intersexuality and the effects of environmental contaminants on the cricket frog in Illinois. Their study determined that sex ratios in cricket frog metamorphs also varied significantly between PCB/PCDF contaminated and control sites. In nature the sex ratio in cricket frog metamorphs favors females (Burkett 1984, in Reeder et al. 1998).

8 **4.5.6.2** GE Studies

1

2

3

4

5

6

7

9 **4.5.6.2.1 Wood Frog Study**

The GE study did not examine the exposure pathway of contaminated media (i.e., sediment and water), as the vernal pool exposure scenario did not represent the range of sediment PCB concentrations within the PSA; the two pools used in the development portion of the study had concentrations near detection limits.

14 Because wood frog larvae were placed in relatively clean sediment throughout the experimental 15 period, exposures to developing larvae were underestimated, relative to exposure during in situ development in much of the PSA. Vernal pool sediment tPCB concentration was shown to be a 16 17 significant factor in the tissue uptake of PCBs and the subsequent manifestation of effects as the 18 frogs matured in the pools (FEL 2002a, 2002b). Therefore, the GE study exposed the developing 19 larvae to an atypically low range of sediment tPCB concentrations that were not characteristic of 20 the floodplain/backwater habitats above Woods Pond. Exposure to the full range of sediment 21 tPCB concentrations is necessary throughout the developmental period to understand 22 contaminant fate and effects in wood frogs because it is the later larval stages that were shown to 23 be the most vulnerable to contaminant-induced effects. The failure to expose developing larvae 24 to representative sediment tPCB concentrations is the primary source of uncertainty associated 25 with the study.

While the explicit consideration of density dependence is a valid consideration in the examination of COC effects on amphibians, the limitation of inadequate contaminant exposures outweighs the explicit consideration of density-dependence (detailed in Section 2.8 of the ERA). In addition, some wood frogs enclosures had predators, which were unevenly distributed, and not documented in the GE study report. Differing levels of predation were not factored into the
 study and could affect density-dependence.

The GE study report did not provide a rationale for the selection of the three levels of initial larval density evaluated, and a work plan was not provided. The relevance of the three densities to wood frog ecology was not demonstrated or discussed, nor did the report indicate what change in density is required to affect populations. The potential confounding effects of larval density in the vernal pools remains a source of uncertainty.

8 The GE report states that it has evaluated the contaminant/effects question at the population 9 level. However, given the two significant sources of uncertainty discussed above, the question 10 of the effect of PSA contaminants on amphibian populations remains outstanding based on the 11 findings from this study.

12 4.5.6.2.2 Leopard Frog Egg Mass Survey

13 The principal area of uncertainty associated with this study is the conclusion that the egg masses 14 found are indicative of the reproductive health of the leopard frogs. While 216 egg masses were 15 found, the study did not show how the total number of egg masses could be extrapolated to the 16 level of overall species reproductive health. While the study provided qualitative verification 17 that some leopard frog egg masses were fertilized, quantitative data on fertilization, embryo 18 development, hatching success, and larval growth and development were not collected. The egg 19 mass measurement endpoint alone is not adequate for an assessment of adult frog reproductive 20 health or fitness. There is much more information that is necessary to adequately determine the 21 reproductive health of the leopard frog population.

Finally, the study states that sediment tPCB concentration is not related to the presence/absence of egg masses or to egg mass density. Given the mobility and life history of the leopard frog adults, this statement is likely correct. The sediment tPCB concentration of a breeding area is not likely to be representative of a female's exposure to PCBs throughout the PSA. A female's selection of an egg deposition site will be dependent upon habitat suitability and the presence of males; sediment tPCB concentration in the breeding area is not likely the most critical metric for determining trends in the presence or abundance of leopard frog egg masses within the PSA. There is no known mechanism by which leopard frogs can discern the toxic concentrations of
 chlorinated organic contaminants and thereby avoid exposure to the contaminant.

3 4.5.7 Conclusions

4 Overall, the amphibian ERA indicates significant risk to frog species in the Housatonic River 5 PSA based on a WOE evaluation of multiple effects endpoints and their associated contaminant 6 media. Furthermore, the available data implicate tPCBs as the stressor responsible for such 7 impairment. The confidence in the conclusion is moderate to high, based on the concordance in 8 predictions of risk from multiple measurement endpoints. The most compelling evidence for 9 ecological degradation comes from the two frog toxicity studies, which not only exhibited 10 significant toxicological effects in both frog species and endpoints, but which also indicated a 11 correlation between level of effect and sediment tPCB concentration.

12 4.5.7.1 Population Modeling

A stochastic population model was developed to determine whether effects from tPCBs on individual wood frogs influences wood frog populations within the Housatonic River PSA (Appendix E, Attachment E.3). The population model projected wood frog population trends 10 years into the future and computed the risk of population decline (Ginzburg et al. 1982) using vital rate information from the literature (Berven 1990) and initial abundances derived from studies of vernal pools in the PSA (Woodlot Alternatives, Inc. 2002, 2003).

The impact of tPCBs on the wood frog population was assessed by comparing population projections from a base population model (i.e., a wood frog population in the absence of tPCBs), with projections from population models that included the effect of tPCBs on population vital rates (see FEL 2002b). Two projection comparisons were performed based on simulations of (1) a non-declining base population, and (2) a declining base population. All models were constructed using RAMAS Metapop (Akçakaya 2002).

25 Findings from the population modeling were:

- 26
- tPCBs have an impact on wood frog population growth and abundance.

- tPCBs hasten population decline, reduce population numbers, and increase the likelihood of extinction.
 - Data collected in the PSA provide field evidence supporting the population-level effects of tPCBs seen in the simulations.
- 5 6

7

1

2

3

4

 The relationship between sediment tPCB concentrations and adult male and female density indicate that increased tPCB concentration leads to decreased density particularly for adult females.

8 4.5.7.2 Risks Within the PSA

9 The WOE assessment of amphibian endpoints indicated a high probability of risk of ecologically 10 significant effects at PCB concentrations observed in the PSA vernal pools included in the 11 Extrapolation to other areas of the PSA required use of concentration-response studies. relationships derived from the site-specific studies. The ERA findings suggest that amphibian 12 13 populations are impacted throughout much of the PSA, with leopard frogs impacted at a wide 14 range of sediment concentrations (likely due to the life history of contact with sediment PCB 15 concentrations, which were not measured in the study), and with stronger responses from wood 16 frogs expected in the more highly contaminated vernal pools. The indications of community 17 responses from the population studies (i.e., localized depressions of richness and abundance near 18 high tPCB vernal pools, and high incidence of malformations observed) substantiate these 19 findings.

20 **4.5.7.3** Extrapolation of Risk Estimates Downstream of Woods Pond

21 Amphibians are primarily exposed to PCBs in floodplain soil, particularly vernal pools and other 22 wet, low-lying areas. The risk assessment focused on vernal pools, but such detailed data were 23 not available below Woods Pond, so the parameter of interest was tPCBs in floodplain soil and 24 sediment in general. Sediment was included to account for more aquatic amphibians such as 25 bullfrogs, and to account for the aquatic life phases of leopard frogs. IDW was used to 26 interpolate PCB concentrations to the limit of the 100-year floodplain (10-year floodplain 27 contours are not available downstream of Woods Pond) using the 0- to 6-inch-depth data from 28 the floodplain downstream of Woods Pond.

1 4.5.7.3.1 Sediment

Ecologically significant adverse effects in late stage juvenile wood frogs occurred in the
sediment tPCB concentration range of 9.54 to 59.3 mg/kg, although responses of lesser, yet
statistically significant, magnitude were observed at 0.52 mg/kg tPCBs and lower.

5 A sediment MATC of 3 mg/kg tPCB was determined based on the results of the point estimate 6 calculations presented in section E.4.3.3. This concentration was just below the EC_{20} values for 7 the Phase III metamorph malformation endpoint (based on both measured and spatially weighted 8 tPCB concentrations), and well within the range of the 95% confidence limit (based on the probit 9 statistical analysis) for the point estimate. The MATC was rounded to 3 mg/kg to account for 10 uncertainty. The EC₂₀ for the sex ratio endpoint was 0.52 and 0.61 mg/kg tPCB (based on 11 measured and spatially weighted sediment tPCB concentrations, respectively). However, as 12 noted in Section E.4.3.3, the 20% effect size is likely not of biological relevance, and therefore a 13 sediment MATC based on the sex ratio EC_{20} may be overly conservative. Selecting an MATC of 3 mg/kg for this endpoint, just outside the 95% confidence limits for the EC₂₀, is 14 15 believed to provide adequate protection for other amphibian species.

16 **4.5.7.3.2 Tissue**

The threshold concentration range for amphibian tissues was 0.60 mg/kg to 6.54 mg/kg tPCB, and was based on the sex ratio endpoint (both an EC_{20} and EC_{50}) and the Phase III metamorph malformation endpoint (an EC_{50} point estimate). As there was not a 20% effect response for malformations in the Phase III metamorphs, a tissue EC_{20} for malformations could not be calculated.

22 Again, the EC_{20} of approximately 0.60 mg/kg for the sex ratio endpoint was considered to be 23 more conservative than necessary from the standpoint of biological relevance for wood frogs. 24 However, use of the tissue EC_{50} s for the two endpoints was considered under-protective, 25 particularly given the likely increased sensitivity of the leopard frogs and salamanders relative to 26 the wood frogs. A tissue MATC of 1 mg/kg was therefore believed to provide a suitable balance 27 between the protection of other amphibian receptors and the lower tissue MATC 28 of approximately 0.65 mg/kg tPCB.

4-83
In summary, tissue concentrations below 1 mg/kg are not expected to cause biologically
 significant adverse responses in the wood frogs. The tissue concentration-response modeling
 predicted significant risk in the range of 1 to 10 mg/kg. At tissue concentrations >10 mg/kg,
 adverse ecological effects are expected to occur.

MATCs for PCBs Used to Assess Risks Below Woods Pond
The soil and sediment MATC of 3 ma/kg tPCR was used as a measure of the

- The soil and sediment MATC of 3 mg/kg tPCB was used as a measure of the potential for adverse effects to amphibians downstream of Woods Pond (Figures F.4-10 and F.4-11). This concentration was developed in the risk assessment for the PSA using multiple lines of evidence (e.g., amphibian community studies, in situ and laboratory-exposure toxicity testing) and was selected as the concentration at which some sensitive endpoints exhibited apparent responses, but the magnitude of responses was not large. Above a concentration of 3 mg/kg tPCB, numerous endpoints indicated ecologically significant responses.
- The tissue MATC of 1 mg/kg tPCB was used as a conservative measure of the
 potential for adverse effects to amphibians downstream of Woods Pond. This
 concentration was developed considering the frequency of adverse effects observed
 in the literature studies, in the site-specific studies, and in an effort to compensate for
 the uncertainty associated with the sensitivity of salamander species.

1 4.6 REFERENCES

- Akçakaya, H.R. 2002. *RAMAS Metapop: Viability Analysis for Stage-structured Metapopulations* (Version 4.0). Applied Biomathematics, Setauket, New York.
- 4 Berven, K.A. 1990. Factors affecting population fluctuations in larval and adult stages of the 5 wood frog (*Rana sylvatica*). *Ecology* 71:1599-1608.
- Birge, W.J., J.A. Black, and A.G. Westerman. 1978. Effects of polychlorinated biphenyl
 compounds and proposed PCB-replacement products on embryo-larval stages of fish and
 amphibians. University of Kentucky. Lexington, KY. pp. 1-33.
- 9 Burkett, R.D. 1984. An ecological study of the cricket frog, *Acris crepitans. Vert. Ecol. Syst.*10:89-103.
- 11 DeGraaf, R.M. and D.D. Rudis. 1983. *Amphibians and Reptiles of New England: Habitats and* 12 *Natural History*. University of Massachusetts Press, MA.
- Eisler, R. and A.A Belisle. 1996. Planar PCB Hazards to Fish, Wildlife, and Invertebrates: A
 Synoptic Review. National Biological Service Report 31, Washington, DC.
- 15 FEL (Fort Environmental Laboratories, Inc.). 2002a. Final Report Frog Reproduction and
- 16 Development Study. 2000 Rana sylvatica Vernal Pool Study. Study protocol no.: WESR01-
- 17 RSTS03–1. Prepared by Fort Environmental Laboratories Inc., Stillwater, OK.
- 18 FEL (Fort Environmental Laboratories, Inc.). 2002b. Final Report Frog Reproduction and
- 19 Development Study. 2000 Rana pipiens Reproduction and Development Study. Study protocol
- 20 no.: WESR01–RSTS03–1. Prepared by Fort Environmental Laboratories Inc., Stillwater, OK.
- Fontenot, L.W., G.P. Noblet, J.M. Akins, M.D. Stephens, and G.P. Cobb. 2000. Bioaccumulation of polychlorinated biphenyls in ranid frogs and northern water snakes from a hazardous waste site and a contaminated watershed. *Chemosphere* 40:803-809.
- Gilbert, M., R. Leclair, Jr., and R. Fortin. 1994. Reproduction of the northern leopard frog (*Rana pipiens*) in floodplain habitat in the Richelieu River, P. Quebec, Canada. *Journal of Herpetology* 28(4):465-470.
- Ginzburg, L.R., L.B. Slobodkin, K. Johnson, and A.G. Bindman. 1982. Quasiextinction probabilities as a measure of impact on population growth. *Risk Analysis* 2:171-181.
- Gosner, K. 1960. A simplified table for staging anuran embryos and larvae with notes on identification. *Herpetologica* 16(2):183-190.
- 31 Gutleb, A.C., J. Appelman, M.C. Bronkhorst, J.H.J. van den Berg, A. Spenkelink, A. Brouwer,
- 32 and A.J. Murk. 1999. Delayed effects of pre- and early-life time exposure to polychlorinated
- 33 biphenyls on tadpoles of two amphibian species (Xenopus laevis and Rana temporaria).
- 34 Environ. Toxicol. Pharm. 8:1-14.

- 1 Gutleb, A.C., J. Appelman, M. Bronkhorst, J.H.J. van den Berg, and A.J. Murk. 2000. Effects of
- 2 oral exposure to polychlorinated biphenyls (PCBs) on the development and metamorphosis of
- 3 two amphibian species (*Xenopus laevis* and *Rana temporaria*). Sci. Tot. Environ. 262:147-157.
- Hayes, T.B. 2000. Endocrine Disruption in Amphibians. In Chapter 10A, *Ecotoxicology of Amphibians and Reptiles*. D.W. Sparling et al., editors. SETAC Press, Pensacola, FL.
- 6 Hine, R.L., B.L. Les, and B.F. Hellmich. 1981. Leopard frog populations and mortality in 7 Wisconsin, 1974-76. Wis. Dep. Nat. Resour., Tech. Bull. No. 122.
- 8 Huang, Y.W., M.J. Melancon, R.E. Jung, and W.H. Karasov. 1998. Induction of cytrochrome
- 9 P450-associated monooxygenases in northern leopard frogs, *Rana pipiens*, by 3,3'4,4'5-
- 10 pentachlorobiphenyl. *Environ. Tox. Chem.* 17(8):1564-1569.
- 11 Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S.
- 12 Petron, B. Potocki, S. Svirsky, and P. Tyler. 1996. Special report of the Massachusetts Weight-
- 13 of-Evidence Workgroup: A weight of evidence approach for evaluating ecological risks. *Human*
- 14 and Ecological Risk Assessment 2:277-304.
- Mikamo, K. and Witschi, E. 1964. Masculinization and breeding of the WW *Xenopus*.
 Experentia 20:622-623.
- Ouellet, M. 2000. Chapter 11, Amphibian Deformities: Current State of Knowledge. In
 Ecotoxicology of Amphibians and Reptiles. Society of Environmental Toxicology and Chemistry
 (SETAC), Pensacola, FL. 904 p.
- Reeder, A.L., G.L. Foley, D.K. Nichols, L.G. Hansen, B. Wikoff, S. Faeh, J. Eisold, M.B.
 Wheeler, R. Warner, J.E. Murphy, and V.R. Beasley. 1998. Forms and prevalence of
 intersexuality and effects of environmental contaminants on sexuality in cricket frogs (*Acris crepitans*). *Environ. Health Persp.* 106:261-266.
- Richards, C.M. and Nace, G.W. 1978. Gynogenetic and hormonal sex reversal used in tests of the XX-XY hypothesis of sex determination in *Rana pipiens*. *Growth* 42:319-331.
- Rosenshield, M.L., M.B. Jofre, and W.H. Karasoy. 1999. Effects of polychlorinated biphenyl
 126 on green frog (*Rana clamitans*) and leopard frog (*Rana pipiens*) hatching success,
 development, and metamorphosis. *Environmental Toxicology and Chemistry* 18(11):2478-2486.
- Savage, W.K., F.W. Quimby, and A.P. DeCaprio. 2002. Lethal and sublethal effects of
 polychlorinated biphenyls on *Rana sylvatica* tadpoles. *Environmental Toxicology and Chemistry* 21(1):168-174.
- Sparling, D.W., G. Linder, and C.A. Bishop. Editors. 2000. *Ecotoxicology of Amphibians and Reptiles*. Society of Environmental Toxicology and Chemistry (SETAC), Pensacola, FL. 904 p.
- 34 Taylor A.C. and J.J. Kollros. 1946. Stages in the normal development of *Rana pipiens* larvae.
- 35 *The Anatomical Record* 94:7-23.

- 1 Woodlot Alternatives, Inc. 2002. Ecological Characterization of the Housatonic River. Prepared
- 2 for U.S. Environmental Protection Agency.
- 3 Woodlot Alternatives, Inc. 2003. Amphibian Reproductive Success within Vernal Pools
- 4 Associated with the Housatonic River, Pittsfield to Lenoxdale, Massachusetts. Report prepared
- 5 for Weston Solutions, Inc. DCN: 99-1275.

5. ASSESSMENT ENDPOINT – SURVIVAL, GROWTH, AND REPRODUCTION OF FISH

3	Highlights
4	Conceptual Model
5	 Conceptual model for fish indicates that the most important exposure pathways
6	are diet and contaminated sediment.
7	Exposure
8	 COCs evaluated were total PCBs (tPCBs), 2,3,7,8-TCDD TEQ, and polycyclic
9	aromatic hydrocarbons (PAHs).
10	 Whole body fish concentrations in five representative fish species were used to
11	evaluate exposures to both tPCBs and TEQ.
12	 Sediment concentrations were used to evaluate risks to fish from PAHs.
13	Effects
14	 Site-specific toxicity tests (Phase I and Phase II) indicate adverse effects at
15	contaminated locations relative to reference areas, and although variable,
16	general PCB dose-dependency. Effects observed are indicative of a dioxin-like
17	etiology.
18	 Literature review indicates that PCB and TEQ threshold tissue concentrations
19	identified in the literature are in the same range as those from site-specific
20	toxicity tests.
21	Risk Characterization
22	 A weight-of-evidence approach was used to characterize risks. A high probability
23	of adverse impacts to fish from tPCBs and/or TEQ is predicted throughout the
24	PSA. Impacts are likely for sensitive sublethal endpoints (reproduction and
25	development), but mortality of adults is unlikely.
26	 Risks attributable to PAHs are negligible to low.
27	 Impacts downstream of Woods Pond are limited to marginal risks for coldwater
28	fish (trout).
29	 Magnitude of impact is not predicted to be catastrophic in any reach; adverse
30	effects, although high in probability, are generally expected to be subtle.
31	 Field surveys (fish community and reproduction studies) support lack of
32	catastrophic effects, but cannot be used to assess lesser impacts.

1 5.1 INTRODUCTION

The purpose of this section is to characterize and quantify the current and potential risks posed to
fish exposed to contaminants of concern (COCs) in the Housatonic River, focusing on total
PCBs (tPCBs) and other COCs originating from the General Electric (GE) facility in Pittsfield,
MA.

A Pre-ERA was conducted to narrow the scope of the ecological risk assessment by identifying contaminants, other than tPCBs, that posed potential risks to aquatic biota in the PSA (Appendix B). The ERA further screened the above contaminants of potential concern (COPCs) for specific relevance to fish inhabiting the main channel of the Housatonic River, and identified COCs to be retained in the detailed assessment. COC groups that were retained were tPCBs, dioxin-like TEQ, and polycyclic aromatic hydrocarbons (PAHs).

12 A stepwise approach was used to assess the risks of COCs to fish in the Housatonic River. The

- 13 four main steps in this process included the following:
- 14 1. Development of a conceptual model (Figure 5.1-1).
 - 2. Assessment of exposure of fish to COCs (Figure 5.1-2).
 - 3. Assessment of the effects of COCs to fish (Figure 5.1-3).
- 17 4. Characterization of risks to fish (Figure 5.1-4).
- 18 This section is organized as follows:

15

- Section 5.1 (Introduction and Conceptual Model)—Describes the conceptual model for fish, including selection of representative species and establishment of measurement and assessment endpoints.
- Section 5.2 (Exposure Assessment)—Describes the quantification of exposures,
 both within the Primary Study Area (PSA) and downstream of Woods Pond.
- Section 5.3 (Effects Assessment)—Describes the potential effects to fish exposed to site COCs, as indicated by the toxicological and field investigations conducted in the PSA. Assesses the concentration-response relationships from site-specific studies and identifies corresponding effects thresholds. This section also summarizes the ranges of relevant tissue and sediment effects thresholds (toxicity thresholds) derived from the literature.
- Section 5.4 (Risk Characterization)—Integrates the exposure and effects
 assessments, summarizes field surveys, and makes conclusions regarding risk for fish

4

5

6

in the Housatonic River using three main lines of evidence. A discussion of the sources of uncertainty regarding risk estimates follows. Section 5.4 also presents an extrapolation of risks beyond the PSA to areas downstream of Woods Pond, for both coldwater and warmwater species.

This section provides a summary of the ERA for fish, which is presented in detail in Appendix F.

7

8 5.1.1 Conceptual Model

9 Total PCBs, dioxins, and furans are persistent and hydrophobic and lipophilic. Food web 10 bioaccumulation and biomagnification represent the most important pathways for PCBs and 11 TEQ (Oliver and Niimi 1985). Direct uptake pathways, through respiration of dissolved PCBs or 12 through incidental ingestion of sediment, are less important pathways for most fish species. 13 PAHs are metabolically transformed in most teleosts, and because of their poor water solubility 14 are more strongly associated with sediment. In summary, the COCs identified for fish exhibit 15 both direct (i.e., contact with contaminated media) and indirect (i.e., food web bioaccumulation) 16 pathways, with emphasis on the latter pathway for PCBs.

17 The conceptual model presented in Figure 5.1-1 illustrates the exposure pathways for fish in the 18 PSA. For strongly hydrophobic COCs (PCB, dioxins/furans), the dominant exposure media 19 were COC tissue concentrations, with uptake into tissues occurring mainly via ingestion of 20 contaminated prey. Tissue concentrations reflect the net COC uptake from food, sediment, 21 overlying water, and porewater, and therefore integrate all primary exposure pathways of 22 interest. For PAHs, sediment were considered the most relevant exposure media. Because 23 PAHs are rapidly degraded to daughter products (some toxic), tissue PAH concentrations cannot 24 be used as exposure metrics for linkage to effects (Johnson et al. 2002, Malins et al. 1985).

Five fish species were selected as the representative species for the ERA. The selected fish species include representatives of the different trophic levels and exposure routes for fish in the PSA.





Figure 5.1-1 Conceptual Model Diagram: Exposure Pathways for Fish Exposed to COCs in the Housatonic River



Figure 5.1-2
 Figure 5.1-2
 Overview of Approach Used To Assess Exposure of Fish to COCs in the Housatonic River



Figure 5.1-3 Overview of Approach Used To Assess the Effects of COCs to Fish in the Housatonic River



 Figure 5.1-4
 Overview of Approach Used To Characterize the Risks of COCs to Fish in the Housatonic River

1	Representative Species
2	 Largemouth bass (<i>Micropterus salmoides</i>) – predator fish.
3	 Yellow perch (<i>Perca flavescens</i>) – predator fish.
4	 Brown bullhead (Ameiurus nebulosis) – bottom feeder.
5	 White sucker (Catostomus commersoni) – bottom feeder.
6	 Pumpkinseed (<i>Lepomis gibbosus</i>) – forage fish.
7	

8 Criteria considered in selecting representative fish species for the ERA included trophic level 9 and feeding preferences, abundance and biomass in the study area, availability of site-specific 10 data, and availability and appropriateness of toxicological data. Because trout have greater 11 importance downstream of the PSA (due to the presence of suitable habitat for a coldwater 12 fishery), a separate analysis using information on trout toxicity from general literature and site-13 specific studies was also conducted as a part of the downstream risk prediction section of the 14 ERA.

15 The assessment endpoint that is the subject of this section is the survival, growth, and 16 reproduction of fish. Measurement endpoints were selected to assess risks of PCBs and other 17 COCs to the representative fish species:

18 Measurement Endpoints for Fish 19 Determine the possible extent of adverse effects by comparing the 20 concentrations of COCs in sediment to the concentrations reported in the 21 literature to cause adverse effects on the survival, growth, or reproduction of fish. 22 Compare the concentrations of COCs in fish tissues to the concentrations in fish 23 tissues that may result in adverse effects, based on site-specific fish toxicity 24 studies. 25 Compare the concentrations of COCs in fish tissues to concentrations . 26 documented in the literature to result in adverse effects. 27 Evaluate field survey information (fish biomass study, ecological characterization . 28 study, and largemouth bass habitat and reproduction study) to gualitatively 29 assess potential effects. 30

The approach used to characterize risks to fish was based upon evaluation of numerous data sources. These included site-specific toxicity investigations, chemical measurements of fish tissue and sediment, biological/community assessments, and literature reviews.

1	Summary of Studies Used To Characterize Risks to Fish
2	 Phase I site-specific studies – Investigated contaminant accumulation and effects
3	in largemouth bass from the PSA, Rising Pond, and a reference area and in their
4	offspring. Adults were evaluated for contaminant-related biochemical, cellular,
5	and organism level effects. Offspring were monitored for survival, development,
6	gross abnormalities, and biochemical alterations.
7	 Phase II site-specific studies – Simulated the maternal transfer of contaminants
8	to developing oocytes. Investigated the effects of injected organic contaminants
9	(extracted from largemouth bass tissue collected in the Phase I studies) on the
10	development of largemouth bass, medaka (<i>Orizias latipes</i>), and rainbow trout
11	eggs.
12	 Sampling and analysis of fish tissue.
13	 Sampling and analysis of sediment.
14	 Field studies – EPA Fish Community and Ecological Characterization Study
15	(Appendix A) and GE Largemouth Bass Community, Reproduction, and Habitat
16	Study.
17	 Literature review – Evaluated the range of PCB, TEQ, and PAH concentrations
18	observed to elicit adverse effects to fish.
19 20	

1 5.2 EXPOSURE ASSESSMENT

The exposure assessment estimates the exposure of fish to tPCBs and other COCs in the Housatonic River PSA (Figure 5.1-2). The COPCs that were retained in the Pre-ERA (Appendix B) were screened specifically for relevance to fish, resulting in the COCs that were retained in the exposure assessment.

6 The vast majority of relevant exposure data were those collected within the PSA. More limited 7 tissue data were also available for areas downstream of the PSA. Extrapolations of risk to most 8 downstream areas relied on the development of exposure-response relationships developed from 9 the site-specific studies and the literature. In some cases, upstream data were used to standardize 10 downstream data for use in the ERA (e.g., lipid-based conversions of fillet PCB concentrations 11 to whole-body concentrations).

12 **5.2.1 Refined Screening of COPCs for Fish**

The Pre-ERA (Appendix B) developed separate lists of COPCs for fish tissue, water, and sediment. Water and sediment screening included comparisons to thresholds considered protective of aquatic life, including invertebrates and fish. Because fish tissue COPC concentrations in the Pre-ERA were not screened against fish tissue effects thresholds, a step was conducted to ensure that no bioaccumulative COPCs were eliminated prematurely that could be of concern to fish.

PCBs – PCBs were identified as COPCs for sediment and water in all PSA reaches. The ERA
 for fish considered tPCB risks as well as dioxin-like toxicity (2,3,7,8-TCDD TEQ) from coplanar
 PCBs, dioxins, and furans.

Dioxins and furans – The ERA considered dioxin and furan risks in the context of their
 contribution to TEQ. The semivolatile contaminant dibenzofuran was eliminated on the basis of
 the very few isolated exceedances of screening criteria (Reach 5A only).

PAHs – Of the principal PAH compounds detected in sediment, only pyrene and fluoranthene
 had significantly elevated concentrations relative to those observed in reference areas. Although
 the conservative screening using sediment quality values suggested a potential for aquatic risk,

concentrations of most PAHs appear to be similar across contaminated and reference sites in the
 PSA. Using a conservative approach, total PAHs plus eight individual PAHs were retained in
 the fish risk assessment.

Pesticides – 4,4'-DDE and 4,4'-DDT were identified as COPCs for sediment in the Pre-ERA,
but were identified only in isolated vernal pools and side channels. Furthermore, some pesticide
detections may be attributable to laboratory interference artifacts. Pesticides were therefore not
considered further in the fish assessment.

8 Metals – Eleven metals were identified as COPCs in sediment based on thresholds developed for

9 the protection of benthic invertebrates. However, all metals were eliminated from the fish ERA,

10 based on an ecological relevance screen (Appendix F; Attachment F.1).

11 **Summary** – The list of COCs retained in the risk assessment for fish is provided below.

12	COCs for Fish
13 14	 Chlorinated organic compounds – PCBs as tPCBs and TEQ, dioxins/furans expressed as TEQ equivalents.
15 16	 PAHs – Total PAH, benzo(g,h,i)perylene, indeno(1,2,3-cd)pyrene, phenanthrene, anthracene, benzo(a)anthracene, pyrene, fluorene, fluoranthene.
17	

17

18 5.2.2 Tissue Chemistry Assessment (Exposure to PCBs and TEQ)

19 The most robust data set for fish tissue concentrations of tPCBs and TEQ was collected by EPA 20 from September 1998 through October 2000. Other fish tissue tPCB data collected within the 21 PSA by GE and others from 1977 to 2002 are also available. These additional data sets were 22 evaluated, and either the inclusion or exclusion of these data would result in very similar risk 23 conclusions. For example, BBL and QEA (2003) state that "PCB concentrations in largemouth 24 bass collected in 2002 from Reach 5B and Reach 6 were similar to each other and, on a whole-25 body basis, to the 1998 EPA data." Therefore, the ERA conclusions are not sensitive to the 26 inclusion or exclusion of data from this recent GE sampling effort.

1	Fish Chemistry Types Considered in the ERA
2	 CM – Composite samples – represent the combination of multiple fish, typically
3	young-of-year or other small fish.
4	 WB – Whole body samples – represent the analysis of single larger fish, often for
5	species that were not considered in the human health risk assessment (e.g.,
6	white sucker).
7	 WB-R – Whole body reconstituted samples – represent individual fish
8	concentrations that were calculated/estimated using separate fillet and offal
9	measurements.

In general, game fish of edible and/or legal size were analyzed separately as skin-off fillet and offal for use in the human health risk assessment and the ERA. The whole-body tissue concentrations for these fish (WB-R) were calculated for this assessment as the weighted average concentration of the individual tissues, using calculation methods described in Appendix F.

14 5.2.2.1 Total PCBs

Table 5.2-1 presents summary statistics for fish tissue tPCB concentrations in the PSA by sample type and species, for all representative species. The table combines samples across all PSA reaches. The reach-by-reach distributions are evaluated in Appendix F; in general there are relatively consistent fish tissue concentrations across the entire PSA. The composite fish tissue samples had lower PCB concentrations than other tissue types (Figure 5.2-1); the small fish in the composite samples were typically representative of younger fish or of species that are small even as adults (i.e., dace).

22 Table 5.2-2 presents summary statistics for lipid-normalized tPCB concentrations by sample 23 type, species, and reach. The mean and median concentrations in Table 5.2-2 indicate that the 24 highest PCB concentrations (i.e., median \geq 2,000 mg/kg lipid) are observed in adult (WB-R) 25 predator fish, due to biomagnification in the food web. Age can be a factor even after lipid 26 normalization because older fish have accumulated PCBs over a longer period of time. This is 27 evident in Table 5.2-2, which shows that the lowest mean and median fish concentrations were 28 observed for composites (CM) of smaller younger fish. Figure 5.2-2 shows the relationship 29 between age and PCB body burden in largemouth bass.

Table 5.2-1

tPCB Concentrations in Representative Species Fish Tissue (mg/kg) in the PSA; Data from EPA Tissue Collections (1998-2000)

Sample Type	Species	Sample count	Min	25 th Percentile	Median	Mean	75 th Percentile	Max
	BB	43	7.19	25.3	32.3	37.6	45.6	103
	LB	38	10.9	42.3	67.8	97.1	125	424
WD-R	PS	51	7.82	23.2	34.6	36.7	44.7	82.1
	YP	75	6.11	61.3	76.1	87.3	104	329
	BB	2	20.9	21.3	21.7	21.7	22.0	22.4
WD	GF*	42	10.8	95.5	143	163.6	215	447
WD	LB	26	3.03	22.7	36.5	57.1	78.4	220
	WS	57	7.96	36.2	56.6	70.6	86.5	216
	LB	12	9.03	19.9	26.1	27.9	36.3	51.2
СМ	PS	9	8.8	26.4	27.5	26.2	27.9	35.1
	YP	15	16.5	27.4	31.0	31.4	35.7	46.9

5 6 7

1

2

3

4

*Goldfish (GF) were not selected as a representative species, but were included because of large sample size and high tPCB concentrations.

8

9 BB Brown Bullhead

- 10 LB Largemouth Bass
- 11 PS Pumpkinseed
- 12 WS White Sucker
- 13 YP Yellow Perch
- 14 CM Composite
- 15 WB Whole Body
- 16 WB-R Reconstituted Whole Body
- 17



- 2 The shaded box represents the interquartile range, the white bar represents the median, and the whiskers extend to the full range of the data.
- 4 CM = composite samples
- 5 WB = whole body samples
- 6 WB-R = whole body reconstituted samples

1

8 Figure 5.2-1 Box-and-Whisker Plots of Lipid-Normalized tPCB Concentrations 9 Plotted by Sample Type for Species with Multiple Sample Types

Table 5.2-2

Total Lipid-Normalized PCB Concentrations (mg/kg lipid) for Representative Species in the PSA; Data from EPA Tissue Collections (1998-2000)

Sample Type	Species	Sample count	Min	25 th Percentile	Median	Mean	75 th Percentile	Max
	BB	43	224	1010	1520	2160	1870	14700
WD D	LB	38	591	1600	2490	2960	3720	8280
WD-K	PS	51	210	692	1270	1370	1960	3600
	YP	75	154	1410	2060	2510	2920	9990
	BB	2	2030	NA	2060	2060	NA	2090
WD	GF*	42	578	1050	1480	1550	1770	4710
WD	LB	26	168	837	1420	1980	2270	8150
	WS	57	252	927	1480	2780	2900	44700
	LB	12	636	936	1080	1440	1490	3580
СМ	PS	9	664	758	854	998	1070	1760
	YP	15	681	990	1210	1340	1410	3350

Soldfish (GF) were not selected as a representative species, but were included because of large sample size and high tPCB concentrations.

7	BB	Brown Bullhead
8	LB	Largemouth Bass
9	PS	Pumpkinseed
10	WS	White Sucker
11	YP	Yellow Perch
12	СМ	Composite
13	WB	Whole Body
14	WB-R	Reconstituted Whole Body
15		



2 The shaded box represents the interquartile range, the white bar represents the median, and the whiskers extend to3 the full range of the data.

4

5Figure 5.2-2Box-and-Whisker Plot of Largemouth Bass tPCB Concentrations6(Lipid-Normalized) Versus Age

Fish tissue PCB data for samples collected downstream of Woods Pond were also evaluated to determine trends in concentrations for the Rest of River area. Overall, fish tissue tPCB concentrations were significantly lower below Woods Pond (Table 5.2-3) relative to those measured in the PSA. Details on the various sources of downstream fish tissue data, and on the non-EPA data sets considered within the PSA, are provided in Appendix F.

6 5.2.2.2 2,3,7,8-TCDD TEQ

7 Fish tissue TEQ were calculated using the approach outlined in Appendix C.10 (Van den Berg et 8 al. 1998). Some individual congener and dioxin/furan concentrations were below the method 9 detection limit (DL). Following the standard approach to non-detects adopted for this ERA (see 10 Appendix C.2), results were compared using 0 and DL substitution for non-detects. A bounding 11 analysis with the two methods indicated median TEQ concentrations that were within a factor of 12 two for nearly all cases. Table 5.2-4 presents the summary statistics for TEQ concentrations (representative species only) in PSA fish tissue by sample type and species, with DL substitution 13 14 for non-detects. As with tPCBs, there are trends of increasing TEQ concentrations with age and 15 fish size.

16 **5.2.3** Sediment Chemistry Assessment (Exposure to PAHs)

There were no data on fish tissue concentrations in the PSA for the eight individual PAHs retained as COCs for fish, or for total PAHs because PAHs are readily metabolized by most aquatic biota, including fish (Johnson 2000). Exposure for these contaminants was therefore assessed based on sediment concentrations only. Table 5.2-5 displays the mean, minimum, median, and maximum main channel sediment concentrations for the eight individual PAH COCs and total PAHs. These sediment concentrations are compared to threshold sediment concentrations in the risk characterization section.

Table 5.2-3

Summary of tPCB Concentrations (mg/kg) from EPA Samples Collected in Reach 8

Sample Type	Species	Sample Count	Min	25 th Percentile	Median	Mean	75 th Percentile	Max
WB-R	BB	7	3.46	3.58	3.83	4.97	3.93	12.5
	LB	17	1.30	23.0	28.8	28.8 38.2		145
	PS	13	5.87	12.7	13.7	14.6	14.9	26.0
	YP	6	13.3	23.5	31.8	50.0	41.5	158
WB	LB	14	12.8	18.1	22.4	24.2	29.3	41.5
СМ	LB	5	9.98	10.5	10.6	11.9	13.0	15.3
	PS	5	9.74	9.98	10.4	10.5	10.7	11.8
	YP	5	8.08	8.70	8.91	9.62	11.2	11.2

- 5 BB Brown Bullhead
- 6 LB Largemouth Bass
- 7 PS Pumpkinseed
- 8 YP Yellow Perch
- 9 CM Composite
- 10 WB Whole Body
- 11 WB-R Reconstituted Whole Body

Table 5.2-4

1 2 3

3 4

TEQ in Representative Species Fish Tissue in the PSA with DL Substitution for NDs (ng/kg); Data from EPA Fish Collections (1998-2000)

Sample Type	Species	Sample Count	Min	25 th Percentile	Median	Mean	75 th Percentile	Max
	BB	31	22.9	48.8	62.2	70.3	86.2	152
WED	LB	29	20.3	48.0	58.5	78.2	100	196
WD-R	PS	31	24.3	33.6	41.3	44.5	51.3	108
	YP	45	17.8	74.2	91.7	102	122	246
	BB	1	43.3	NA	NA	NA	NA	43.3
WB	GF*	29	37.8	69.0	104	118	142	288
	LB	15	12.6	23.6	37.5	43.0	54.1	86.9
	LB	12	29.2	36.8	41.9	46.1	57.2	63.1
СМ	PS	9	27.7	31.7	32.7	36.1	42.0	47.1
	YP	15	34.2	41.3	42.9	45.8	52.3	63.1

5 DL=detection limit

6 ND=non-detect

⁷ *Goldfish (GF) were not selected as a representative species, but were included because of large sample size and

8 high tPCB concentrations.

9

10 BB Brown Bullhead

- 11 LB Largemouth Bass
- 12 PS Pumpkinseed
- 13 YP Yellow Perch
- 14 CM Composite
- 15 WB Whole Body
- 16 WB-R Reconstituted Whole Body

Table 5.2-5

Summary Statistics for Concentrations of PAH COCs in Main Channel Sediment by Reach

5A			5B			5C			6			
	n = 20			n = 6			n = 11			n = 3		
	Mean	Mean Median Range		Mean	Median	Range	Mean	Median	Range	Mean	Median	Range
Contaminant	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)	(mg/kg)
Total PAHs	21.8	7.72	0.37 - 159	3.13	3.65	0.056 - 5.48	26.8	3.62	0.289 - 255	3.13	3.28	1.9 - 4.20
Anthracene	0.982	0.18	0.023 - 11	0.220	0.12	0.076 - 0.48	1.59	0.46	0.024 - 14	0.435	0.078	0.026 - 1.2
Benzo(a)anthracene	1.96	0.635	0.03 - 15	0.337	0.375	0.072 - 0.48	2.07	0.26	0.025 - 20	0.213	0.19	0.15 - 0.3
Benzo(g,h,i)perylene	0.706	0.34	0.022 - 3.8	0.247	0.21	0.076 - 0.48	0.628	0.22	0.03 - 4.9	0.217	0.25	0.12 - 0.28
Fluoranthene	3.15	1.1	0.058 - 20	0.445	0.475	0.027 - 0.84	4.13	0.42	0.037 - 40	0.413	0.46	0.26 - 0.52
Fluorene	0.469	0.125	0.031 - 4	0.203	0.112	0.032 - 0.48	1.32	0.57	0.048 - 10	0.601	0.56	0.043 - 1.2
Indeno(1,2,3-cd)pyrene	0.748	0.345	0.021 - 4.4	0.232	0.215	0.066 - 0.48	0.623	0.2	0.026 - 5	0.197	0.22	0.1 - 0.27
Phenanthrene	3.16	0.73	0.037 - 29	0.423	0.405	0.08 - 0.84	5.20	0.24	0.096 - 54	0.256	0.33	0.079 - 0.36
Pyrene	3.26	1.1	0.058 - 22	0.440	0.495	0.029 - 0.75	3.98	0.61	0.054 - 36	0.490	0.54	0.34 - 0.59

1 5.3 EFFECTS ASSESSMENT

This section describes the literature and site-specific studies used to characterize the effects of PCBs and other COCs to fish in the Housatonic River PSA. Results of site-specific fish toxicity studies and literature effects levels were synthesized to develop tissue concentration ranges at which adverse developmental effects can be expected in the representative fish species in the Housatonic River.

7 Results of field surveys are described in Section 5.4 (Risk Characterization) because they are

8 largely qualitative in nature and could not be used to derive effects metrics in the fish ERA.

9 Three sources of data were considered in the development of tissue effects thresholds for PCBs

- 10 and TEQ (Figure 5.1-3). These include the following:
- 11 • General Literature—The literature review evaluated the range of PCB and TEO 12 concentrations observed to cause adverse effects to ecologically relevant endpoints in 13 fish, such as reproduction and development. The types of responses observed in PCB 14 and dioxin spiking studies were also evaluated, to assess whether the effects observed in the Phase I and Phase II studies were consistent with a PCB and/or dioxin-based 15 mechanism of action. The literature review emphasized studies conducted in the 16 17 laboratory using freshwater fish species, and for which egg or whole body tissue 18 concentration data were reported. In addition, a review was conducted to assess field 19 studies, marine fish species, and thresholds based on liver concentrations (Attachment 20 F.6).
- Phase I Site-Specific Studies—These studies investigated contaminant accumulation and effects in largemouth bass from the PSA and in their offspring. Adult largemouth bass from the PSA, Rising Pond, and a reference area (Threemile Pond) were evaluated for contaminant-related biochemical, cellular, and organism-level effects. Adult fish were spawned and the development of their offspring was monitored for survival, development, gross abnormalities, and biochemical alterations.
- Phase II Site-Specific Studies—These studies investigated the effects of organic contaminants extracted from largemouth bass tissue collected in the Phase I studies on the development of uncontaminated largemouth bass, medaka, and rainbow trout eggs exposed via injection. This study design simulated the maternal transfer of contaminants to developing oocytes.

32 In this ERA, separate effects thresholds were derived for each of the sources of data described 33 above. Concordance between these separate effect levels was observed not only in the 34 magnitude of the thresholds derived, but also in the type of effects observed.

Effects of PCBs that have been documented on fish include mortality, growth-related effects, 1 2 behavioral responses, biochemical alterations, and adverse reproductive effects. Of particular 3 concern are the effects of dioxin-like PCB congeners that have the same toxic mechanism as 4 2,3,7,8-TCDD (Walker and Peterson 1991; Zabel et al. 1995a). Reproductive and developmental 5 effects have also been observed in fish exposed to 2,3,7,8-TCDD or other dioxin-like substances. 6 The early life stages of offspring from exposed adults are more sensitive to TCDD toxicity than 7 are adults (Walker et al. 1994). The effects of TCDD in sac fry include yolk sac edema, 8 pericardial edema, multifocal hemorrhages, craniofacial malformations, and mortality (Zabel et 9 al. 1995b).

10 **5.3.1 Derivation of Literature Tissue Effects Metrics**

A literature review was conducted to develop threshold effects concentrations for species that
 occur in the Housatonic River.

13 **5.3.1.1 Total PCBs**

14 A total of 39 scientific papers were reviewed to identify the range of tPCB concentrations 15 associated with adverse effects on survival, growth, and reproductive success in fish. The papers 16 were screened using the criteria summarized in Table 5.3-1. Of the papers reviewed for the 17 effects of PCBs on fish, 6 met the screening criteria outlined above. The effects and no-effects 18 levels from these studies are shown in Figure 5.3-1. None of these articles described controlled 19 toxicity studies performed directly on the representative fish species selected for the Housatonic 20 River. Reported tissue concentration LOAELs ranged from 1.53 mg/kg for increased mortality 21 in lake trout (Salvelinus namaycush) sac fry (Berlin et al. 1981) to 125 mg/kg for fry mortality in 22 brook trout (Salvelinus fontinalis) that were exposed to Aroclor 1254 in water pre- and post-23 hatch (Mauck et al. 1978).

A number of different methods have been used to select tissue effects concentrations, based on approaches used in deriving sediment and water quality criteria and guidelines. Given the uncertainties in relying on one method only, the following potential effects thresholds were calculated, following the lines of evidence approach by EPA (1999):

Table 5.3-1

Criteria Used To Screen Available Studies for Determining Threshold Body Burdens

	Decision			
Criterion	Accept	Reject		
PCBs and TCDD				
Body burden data	Reported (whole body preferred)	Not reported or reported fillet concentrations only		
Endpoints	Population-level reproductive, development and survival effects	Chemical level (i.e., enzyme induction) effects		
Exposure route	Studies mimicking maternal transfer, exposure of eggs, juveniles and adults via diet, water and/or sediment	Intraperitoneal injection of adults		
Statistics	Study included a control	No control		
PCBs only				
PCB type	Aroclor 1254, Aroclor 1260, Clophen A50, tPCBs	Other PCB mixtures or individual congeners, or when co-occurring contaminants present		



2 Figure 5.3-1 Literature-Based PCB Fish Tissue Effects Concentrations

3

- Highest no observed adverse effect levels (NOAEL) and lowest observed adverse 1 2 effect levels (LOAEL) reported in the literature for fish. Selected percentiles for various effects concentrations and tissue types - 10th and 50th 3 4 percentiles calculated for all effects data and for groups of effects data (i.e., all 5 LOAELs; all effects concentrations; egg tissue effects concentrations; juvenile tissue effects concentrations; and adult tissue effects concentrations where possible), where 6 7 sufficient data-points exited. 8 Geometric means of paired NOAELs and LOAELs. 9 • The effects data and the potential effects thresholds for PCBs are shown in Figure 10 5.3-1. There are two groups of results, one in the range of 1-11 mg/kg ww and the other in the range of 61-90 mg/kg ww. The lower results (i.e., 1-11 mg/kg) 11 correspond to the lowest LOAEL selected from the literature, the 10th and 50th 12 percentiles for sac-fry/juvenile effects, and, the 10th and 50th percentiles of the 13 concentrations in all life stages for which data were available (i.e., egg, sac-frv, 14 15 juvenile). The higher group of results (i.e., 61-90 mg/kg) corresponds to the highest NOAEL reported, the paired NOAEL/LOAEL geometric means, and the mean 16 concentration for effects observed in all tissues (i.e., egg, sac-fry, juvenile). 17 18 Based on the lines of evidence approach, a threshold effects concentration of 61 mg/kg ww tPCB 19 for egg/sac-fry tissue was chosen. This value corresponds to the following:
- The average concentration for all effects reported in the studies used (61 mg/kg).
- 21

The highest NOAEL reported in the studies used (71 mg/kg ww).

22 23 The geometric mean of the paired NOAEL/LOAELs reported in the studies used (92.9 mg/kg ww).

To scale the selected egg/sac-fry tissue concentration to a whole body concentration for warmwater fish, a factor of 0.5 was applied based on site-specific and literature information indicating that egg PCB concentrations are higher than the maternal whole body PCB concentrations in PSA fish species (Section F.3.4.2.2). As a result, a whole body tissue concentration of 31 mg/kg ww is recommended and is expected to be protective of reproductive and developmental endpoints for PSA fish species.

In summary, adult fish with tissue concentrations greater than 31 mg/kg ww may have reduced reproductive success and/or their offspring may experience adverse early life stage developmental effects. Attachment F.4 provides additional details for the derivation of this threshold. This effects threshold is consistent with a recent review of PCB toxicity conducted by the National Oceanographic and Atmospheric Administration (Monosson 1999). The NOAA study concludes that Aroclor 1254 at concentrations ranging from 25 mg/kg to 70 mg/kg in the liver of adult fish "interferes with the proper functioning of the reproductive system." Because the report also concludes that the "liver is estimated to have similar concentrations as the whole body or eggs," the NOAA effects range is consistent with the 31 mg/kg threshold derived above. Additional information on liver effects thresholds and field-based assessments of PCB toxicity are provided in Attachment F.6.

8 5.3.1.2 2,3,7,8-TCDD TEQ

9 Nineteen papers were reviewed to identify the range of TEQ concentrations associated with 10 adverse effects on survival, growth, and reproductive success in fish. The same screening 11 criteria applied to tPCBs were used to select the relevant papers (Table 5.3-1). The 11 studies 12 that met the screening criteria involved maternal transfer to eggs from adults fed TEQ-13 contaminated diets, waterborne exposure of juveniles, or injection of eggs. Most studies 14 provided egg concentrations, with only one of the papers reporting adult female whole body 15 contaminant concentrations.

Nine of the 11 studies included effects for mortality of sac fry, a reproductive effect. The species included in these studies were primarily trout (lake trout, rainbow trout, and brook trout), but the Elonen et al. (1998) study included assessment of several warmwater species, including white sucker (a representative species in the PSA). Warmwater species were less sensitive than most trout species, with increases in early-lifestage mortality observed in the 100 to 1,000 ng/kg TCDD range (Elonen et al. 1998).

22 These data are summarized in Figure 5.3-2. There appear to be two general groups of results—a 23 lower group at approximately 50 ng/kg ww, and a higher group between 400 and 1200 ng/kg 24 ww. Basing a threshold concentration for adult whole bodies on the lower group (which corresponds to the lowest LOAEL observed, and the 10th percentile of egg concentrations at 25 26 which effects were observed) may be overly conservative due to the known sensitivity of the 27 trout species (e.g., lake trout) used in those studies. Conversely, basing the threshold 28 concentration on the higher group may not be protective against adverse effects for all the 29 species of concern in the Housatonic River.



2 Figure 5.3-2 Literature-Based TCDD (TEQ) Fish Tissue Effects Concentrations

An egg effects threshold of 100 ng/kg TEQ, which is intermediate between the high and low groups discussed above, was selected for the Housatonic River PSA fish. This concentration represents the level at which early lifestage mortality starts to increase in several species, including warmwater fish species. Although the mortality rates at 100 ng/kg are not statistically significant (i.e., NOAELs in Elonen et al. 1998 are 270 ng/kg and higher), the gradient in toxicity is fairly steep above 100 ng/kg, such that approximately 50% mortality to several species is observed at 1,000 ng/kg TEQ (see Attachment F.4).

8 As with the tPCB threshold derivation, the threshold egg concentration was scaled to an adult 9 whole body concentration based on the site-specific data and literature information (Niimi 1983; 10 Monosson 1999) indicating that the concentration of PCBs in eggs will be higher than the 11 maternal whole body. Using a conversion factor of 0.5 (Section F.3.4.2.2), a whole body tissue 12 concentration of 50 ng/kg ww TEQ was derived and is expected to be protective of sensitive 13 reproductive and developmental endpoints in PSA fish. Adult fish with tissue concentrations 14 greater than 50 ng/kg ww may have reduced reproductive success and/or their offspring may 15 experience adverse early life stage developmental effects.

16 5.3.2 Site-Specific Toxicity Studies

17 5.3.2.1 Housatonic River Fish Toxicity Study - Phase I

18 **5.3.2.1.1** Methods

The Phase I study for the Housatonic River fish reproductive health assessment was conducted by the USGS Columbia Environmental Research Center (CERC) (Tillitt et al. 2003a). The study investigated contaminant-associated effects in fish collected from the PSA and spawned under controlled conditions. The main objective of the study was to characterize differences that may be related to PCB toxicity between Housatonic River fish and those at a reference area (Threemile Pond).

Adult largemouth bass were collected from two Housatonic River locations within the PSA (Reach 5C and Reach 6), from Rising Pond (Reach 8) and from the Threemile Pond reference location. Adult fish (both pre- and post-spawning) were analyzed for elevated liver enzymes (EROD), abnormal gonadal and liver histology, elevated occurrences and rates of macrophage
 aggregates, body and organ sizes and weights, and steroid hormone concentrations.

Adult largemouth bass from the PSA and the reference area were spawned, and their lab-reared offspring were monitored for survival, developmental delays and deformities, growth, and cytochrome P450 induction.

6 **5.3.2.1.2** Results

Adult largemouth bass from the Housatonic River sites exhibited multiple sublethal effects at
frequencies of occurrence that were statistically different from those observed in bass from the
reference location.

10	Phase I Adult Largemouth Bass – Effects Observed
11	 Elevated EROD levels In livers.
12	 Thickened lobule wall in testes.
13	 Elevated occurrence of macrophage aggregates.
14	 Reduced growth in females.
15	 Reduced estrogen levels.

16

17 The enzymatic responses, histopathologies, macrophage aggregates, and estrogen responses 18 listed above are biomarkers for the induction of organic contaminant-derived responses. 19 Although not necessarily indicative of ecologically significant impairment to the adults 20 themselves, they are indications of biological and chemical alterations that may lead to 21 reproductive effects. These effects are also consistent with the summary of Monosson (1999) in 22 which Aroclor 1254 was found to interfere with the reproductive system (altered steroid 23 hormone metabolism, altered testes and ovarian development, and altered concentrations of 24 neurotransmitters and gonadotrophins) at concentrations of approximately 25 to 70 mg/kg tPCB 25 in liver tissue.

Effects were also observed in offspring from the contaminated areas that were statistically different from those observed in offspring from the reference site. At 15 days post swim-up, deformities were observed in fry from all three reaches on the Housatonic River, while none were observed in fry from the reference location. Partially external swim bladders, an extremely unusual deformity that has also been observed in dioxin dosing studies, were observed at all three Housatonic River reaches. In addition, shortened opercula were observed in 22% of fry from Reach 5BC and tail deformities were observed in 2% of fry from Reach 6 at 15 days post swim-up; no opercular or tail deformities were observed in fry from the other Housatonic River locations or the reference location.

7		Phase I Largemouth Bass Offspring – Effects Observed
8 9	•	Survival – Reduced survival from hatch to swim-up, or reduced survival post swim-up.
0	•	Development – Developmental delays (increased days to swim-up).
1	•	Growth – Reduced growth from swim-up to 15 days post swim-up.
23	•	Deformities – Increase in eye deformities from hatch to swim-up; shortened opercula; tail deformities; external swim bladders.

14

15 In summary, the effects observed in the Phase I study were suggestive of PCB-related toxicosis.

Although responses were not all consistent across all exposed reaches, the adults and offspring
both exhibited a suite of symptoms that was consistent with PCB-related toxicity.

18 **5.3.2.2** Housatonic River Fish Toxicity Studies – Phase II

19 **5.3.2.2.1** Methods

In Phase II of the fish toxicity studies conducted by USGS CERC (Tillitt et al. 2003b), organic contaminants present in the largemouth bass from the Phase I studies were extracted and injected into cultured eggs of largemouth bass, medaka, and rainbow trout. This study provided a simulation of maternal transfer of PCB contamination to offspring.

24 Egg Production and Injection

Largemouth bass and medaka eggs were produced by brood stocks held in experimental ponds and rainbow trout eggs were produced in the laboratory using unfertilized rainbow trout eggs and milt obtained from a hatchery. Eggs were randomly distributed to each treatment. The number of trials for each species/life stage/treatment combination varied with egg availability and quality (i.e., control survival). Each trial had three replicates, with 10 eggs per replicate for largemouth bass and medaka, and approximately 24 eggs per replicate for rainbow trout. Trials for each
 species/life stage/treatment combination were conducted at different times.

3 Extracts were produced from tissue homogenates of largemouth bass collected from Reaches 4 5BC (i.e., Reaches 5B and 5C combined), Reach 6, and Reach 8 and the Three-mile Pond 5 reference location during the Phase I study. Extracts for each site were diluted into five different 6 concentrations and injected into clean largemouth bass, rainbow trout, and medaka eggs; 7 concentrations of tPCBs (mg tPCB/kg egg) and TEQ (ng TEQ/kg egg) for each dose group were 8 calculated. For comparative purposes, PCB-126 and 2,3,7,8-TCDD (TCDD) standards diluted 9 into six different concentrations were injected into the eggs. Uninjected eggs and eggs injected 10 with triolein were used as negative controls.

11 Assessment of Lethal and Sublethal Effects

The assessment of lethal and sublethal effects in fish was focused on the later (i.e., swim-up and post swim-up) stages of development. At early stages of development (i.e., hatch), effects are less pronounced or likely to occur because chemicals present in the egg yolk have not yet been fully absorbed by the developing fish (Papoulias, personal communication 2003a). At later stages of development, after yolk absorption is complete, effects are more apparent. Accordingly, data collection, analyses, and interpretation were focused on effects on swim-up and post swim-up fish.

19 The percent survival was determined for each treatment group and compared to controls. To 20 ensure data quality, trials in which largemouth bass survival was \leq 50% or medaka or rainbow 21 trout survival were \leq 70% were excluded from statistical analyses, in accordance with standard 22 toxicity testing data quality objectives (ASTM 2002). LD₅₀ (or lethal dose 50) values were also 23 calculated to determine the concentration at which mortality is observed in 50% of the 24 population, relative to the negative controls.

Lengths and weights of largemouth bass and medaka were measured at the end of the experiment (i.e., 15 days post swim-up) and compared to controls. Lengths and weights of rainbow trout were not measured.

Each fish was examined for deformities at important life stages. For each deformity, all trials for 1 2 each species/life stage/treatment combination were combined and normalized for sample size 3 (per 1,000 fish) to allow for comparisons across treatment groups with different sample sizes. ED₅₀ values (or 50% effective doses for sublethal and lethal effects), which represent the 4 5 concentration at which mortality or one or more abnormalities were observed in 50% of the 6 population, were calculated relative to the negative controls. Trials were included in statistical 7 evaluations where effects (mortality or one or more pathologies were observed) were \leq 50% in 8 negative controls and \geq 50% in the high dose treatment groups (ASTM 2002).

9 **5.3.2.2.2** Results

The following effects were observed in offspring of largemouth bass, medaka, and rainbow trout
 exposed to extracts from the Housatonic River and PCB-126 and TCDD standards.

12 Survival and Growth

Survival of largemouth bass, medaka, and rainbow trout was assessed at swim-up, swim-up and
3 and 15 days post swim-up, and 600 daily temperature units (DTU; approximately 3 days post
swim-up), respectively.

16 Statistically significant reductions in survival were most evident in fish exposed to PCB and 17 TCDD standards. Reduced survival was also observed in medaka exposed to Housatonic River 18 extracts. Between 3 and 15 days post swim-up, medaka exposed to extracts from Reach 5BC 19 and Reach 6 exhibited statistically significant reductions in survival relative to control fry. 20 Survival was not affected in largemouth bass and rainbow trout exposed to Housatonic River 21 extracts.

High mortality was observed in largemouth bass control fish between 3 and 15 days post swimup. Largemouth bass, which are not typically used in toxicity testing, encountered difficulties during the transition from endogenous to exogenous feeding and starved to death. Other sources of variability in survival data (for all fish) were the occurrence of fungal infections, as well as differences in egg quality and unknown sources of variation associated with temporally distinct trials.
1 LD₅₀s were calculated for select trials for largemouth bass (swim-up), medaka (3 and 15 days 2 post swim-up), and rainbow trout (600 DTU) for Reaches 5BC and 6, PCB-126, and/or TCDD. 3 LD_{50} s estimates could not be calculated for trials for all species and life stage combinations 4 because the data did not pass the acceptance criterion and/or the magnitude of the response 5 observed was not sufficient. LD₅₀s (confidence intervals) for Reach 5BC extracts ranged from 6 16 (-7 to 40) to 95 (75 to 116) TEQ/kg egg. LD₅₀s for Reach 6 ranged from 7 (-18 to 31) ng 7 TEQ/kg egg to 178 (68-287) ng TEQ/kg egg. LD₅₀s for PCB-126 ranged from 580 (379 to 782) 8 ng TEQ/kg egg to 5,217 (3,610 to 6,824) ng TEQ/kg egg. TCDD LD₅₀s ranged from 889 (673-9 760) ng TEQ/kg egg to 5,481 (3,950 to 7,013) ng TEQ/kg egg.

10 Overall, medaka at 15 days post swim-up exhibited the lowest LD₅₀s, relative to other species, 11 for all extracts and standards, with the exception of TCDD. The overall results (i.e., order of 12 magnitude difference in TEQ-based LD_{50} s between site extracts and standards) appears to 13 indicate that the Housatonic River extracts are more toxic than would be predicted on the basis of 14 an additive model of dioxin-like toxicity alone. The increased toxicity observed with the 15 Housatonic River extracts could be attributed to synergistic effects of PCB mixtures and effects 16 of other PCBs in the mixture that are not considered using the TEQ approach (Tillitt, personal 17 communication 2003).

18 Largemouth bass and medaka length and weight (growth endpoints) were not affected by 19 exposure to extracts or standards.

20 Individual Deformities

At certain stages of development, largemouth bass, medaka, and rainbow trout were examined for a variety of abnormalities. Several abnormalities exhibited an apparent dose-related or threshold response to high doses of Housatonic River extracts or standards, relative to control fish.

Overall, increased rates of deformities were most evident in fish at swim-up and post swim-up following in ovo exposure to PCB and TCDD standards. Fish exposed in ovo to high doses of these standards exhibited a variety of gross pathologies that are characteristic of PCB and dioxin exposure (craniofacial deformities, spinal deformities, swim bladder deformities, hemorrhage, pericardial edema, peritoneal edema, yolk sac edemas, and delayed development). Generally, rainbow trout appeared to be the most sensitive of the three species evaluated and medaka
 appeared to be the least sensitive.

3 Fish exposed in ovo to high doses of Housatonic River extracts exhibited similar types of gross 4 pathologies as the dioxin-like standards, including craniofacial deformities, fin deformities, 5 spinal deformities, swim bladder deformities, hemorrhage, pericardial edema, peritoneal edema, 6 yolk sac edemas, and larval weakness and delayed development, along with a group of "other" 7 abnormalities. Rainbow trout exhibited the largest magnitude dose-related response to 8 Housatonic River extracts; delayed development was observed in 62% of trout exposed to Reach 9 6 extract containing 83 mg tPCBs/kg egg. Some of the pathologies were only observed in one of 10 the three species. For example, rainbow trout were the only species that exhibited opercular 11 deformities.

Some of the deformities observed in fish were only weakly related to tPCB or TEQ concentrations for one species/life stage/treatment combination. The lack of a dose-response in fish injected with Housatonic River extracts and/or PCB and TCDD standards and the occurrence of these deformities in fish injected with control and reference site extracts indicates that these abnormalities are not the most reliable markers of PCB exposure.

17 Total Abnormalities

18 To provide an overall picture of relationship between PCB exposure and the occurrence of 19 abnormalities, the proportion of fish exhibiting one or more abnormalities was determined and 20 compared to the control fish (at swim-up and post swim-up). In some cases, the highest 21 frequency of abnormalities was observed in the second highest dose group; the reason for this 22 was not apparent. Fish exposed to PCB and TCDD standards exhibited significantly higher 23 percentages of abnormalities, relative to control fish. Rainbow trout were the most sensitive of 24 the three species. Similar, but more variable, dose-response relationships were observed in fish 25 exposed to Housatonic River extracts. Figures 5.3-3 to 5.3-7 summarize the concentration-26 response relationships for the PSA reaches. Again, rainbow trout appeared to be the most 27 sensitive of the three species.



1

Notes: Bar height indicates percentage of fish affected with one or more pathologies.
 Asterisks indicate significant differences from negative controls (uninjected and triolein).
 Doses are 1, 2, 19, 93, and 185 mg/kg tPCB and 0.6, 1, 12, 59, and 118 ng/kg TEQ for Dose IDs 1-5.

5 Source: Adapted from Tillitt et al. 2003b.

Figure 5.3-3 Effects of in Ovo Exposure to Increasing Doses of Reach 6 Extracts on Largemouth Bass at Swim-Up

- 8
- 9



- Notes: Bar height indicates percentage of fish affected with one or more pathologies. Asterisks indicate significant differences from negative controls (uninjected and triolein). Doses are 2, 3, 31, 155, and 310 mg/kg tPCB and 0.6, 1, 13, 64, and 128 ng/kg TEQ for Dose IDs 1-5.
- 5 Source: Adapted from Tillitt et al. 2003b.

6 Figure 5.3-4 Effects of in Ovo Exposure to Increasing Doses of Reach 5BC Extracts on Medaka at 5d Post Swim-Up 7



Dose Identification Number

- 8 9 Notes: Bar height indicates percentage of fish affected with one or more pathologies. 10 Asterisks indicate significant differences from negative controls (uninjected and triolein). 11 Doses are 1, 2, 19, 93, and 185 mg/kg tPCB and 0.6, 1, 12, 59, and 118 ng/kg TEQ for Dose IDs 1-5.
- 12 Source: Adapted from Tillitt et al. 2003b.

13 Figure 5.3-5 Effects of in Ovo Exposure to Increasing Doses of Reach 6 Extracts on Medaka at 5d Post Swim-Up 14

15

1 2 3



Dose Identification Number

- Bar height indicates percentage of fish affected with one or more pathologies. Notes: Asterisks indicate significant differences from negative controls (uninjected and triolein). Doses are 1.4, 2.8, 28, 70, and 139 mg/kg tPCB and 0.6, 1, 11, 29, and 57 ng/kg TEQ for Dose IDs 1-5.
- 5 Source: Adapted from Tillitt et al. 2003b.

Effects of in Ovo Exposure to Increasing Doses of Reach 5BC 6 Figure 5.3-6 Extracts on Rainbow Trout at 600 DTU 7



89

- Notes: Bar height indicates percentage of fish affected with one or more pathologies. 10 Asterisks indicate significant differences from negative controls (uninjected and triolein). 11 Doses are 0.8, 1.7, 17, 42, and 83 mg/kg tPCB and 0.5, 1, 11, 27, and 53 ng/kg TEQ for Dose IDs 1-5.
- 12 Source: Adapted from Tillitt et al. 2003b.

13 Figure 5.3-7 Effects of in Ovo Exposure to Increasing Doses of Reach 6 14 **Extracts on Rainbow Trout at 600 DTU**

1 Cytochrome P450

2 Cytochrome P450 induction was evaluated qualitatively in largemouth bass, medaka, and 3 rainbow trout tissues using immunochemical histological techniques. Cytochrome P450 4 induction was observed in fish exposed to both standards and Housatonic River extracts. 5 Rainbow trout was the most sensitive test species, exhibiting apparent dose-related increases in 6 cytochrome P450 induction. The strongest response (i.e., highest induction) was observed in 7 trout exposed to Reach 5BC extracts. Low and moderate level cytochrome P450 induction was 8 observed in bass exposed to 6 µg TCDD/kg egg and medaka exposed to 2 to 6 µg TCDD/kg egg. 9 Medaka also exhibited moderate dose-related cytochrome P450 induction following exposure to 10 reference site extracts containing 0.15 mg tPCBs/kg egg. Largemouth bass did not appear to 11 exhibit a dose-related induction of cytochrome P450 following exposure to Housatonic River 12 extracts.

13 5.3.2.2.3 Study Conclusions

14 A high degree of variability was observed in many of the parameters evaluated in the Phase II 15 study. However, despite this variability, an overall pattern of PCB-related toxicity was apparent. 16 The types of abnormalities observed in fish exposed to Housatonic River extracts in the Phase II 17 study corresponded with abnormalities reported in the Phase I study, as well as with dioxin-like 18 effects documented in the literature. As expected, effects were most pronounced at later stages 19 of development (i.e., swim-up and post swim-up), after maximum contaminant exposure (i.e., 20 completion of yolk absorption) occurred. Rainbow trout and medaka appeared to be more 21 sensitive to the Housatonic River extracts and PCB-126 and TCDD standards than largemouth 22 bass.

Given the range of tPCB and TEQ concentrations used in the Phase II study, it was not unexpected that the magnitude of effects observed with the standards (2,3,7,8-TCDD) and PCB-126) would be greater than those observed with the Housatonic River extracts. However, when ED₅₀ concentrations were normalized using TEQ, the Housatonic River extracts were more toxic than the standards. The increased toxicity associated with the Housatonic River extracts could be attributed to synergistic toxicity of the PCB mixtures, as well as the effects of PCBs that are not incorporated into the TEQ model (Tillitt, personal communication 2003).

5.3.3 Concentration-Response Analysis – Toxicity Endpoints

2 5.3.3.1 Phase I Study Threshold Effects Concentration Derivation

The Phase I fish toxicity study identified reproductive effects including reduced survival and
growth, as well as developmental delays and deformities, in Housatonic River offspring.

5 Specific abnormalities were observed in Housatonic River fish that were not observed in the fish
6 from the Threemile Pond reference location.

Both tPCB and TEQ tissue effects thresholds were derived from the Phase I study results. These thresholds are not bounded because adverse effects were observed in spawn of bass from all three Housatonic River sites, at the lowest tissue concentration measured, 45 mg/kg tPCBs (or 38 ng/kg TEQ); consequently, adverse effects may also occur at lower tissue concentrations. These tissue effects thresholds are similar to those derived from a literature review of PCB and dioxin effects (Section 5.3.1) and the Phase II studies (Section 5.3.2.2).

13 **5.3.3.2** Phase II Study Threshold Effects Concentration Derivation

The primary objective of the Phase II study was to determine the toxic effects of in ovo exposure 14 15 of fish to extracts containing organic contaminants from adult Housatonic River fish. Although 16 the Phase I study identified a suite of effects that were consistent with PCB-related toxicity, the 17 Phase II study evaluated the cause-effect linkage more directly. The results of the Phase II study 18 indicated that fish exposed to Housatonic River extracts exhibited decreased survival and 19 increased abnormalities and biochemical alterations, in response to high doses of tPCBs and 20 TEQ. The patterns of responses observed were not always consistent across species and 21 treatments; however, gross pathologies observed were characteristic of PCB-related effects 22 reported in the literature and corresponded with a number of the effects observed in the Phase I 23 study.

24 **5.3.3.2.1** ED₅₀ Estimates

ED₅₀s derived from the Phase II study data were used to develop thresholds for Housatonic River extracts and PCB-126 and TCDD standards. An ED₅₀ value represents the concentration at

1 which sublethal or lethal effects (i.e., either mortality or one or more abnormalities) was 2 observed in 50% of the population, relative to the negative controls. This combined toxicity endpoint provides an indication of the concentration threshold for sublethal and lethal effects in 3 4 early life stages of fish. The ED_{50} endpoint represents a large effect size and indicates an 5 unacceptable level of biological harm. However, given the extent to which early lifestage fish 6 may be impacted by multiple natural and chemical stressors, it was considered desirable to select 7 a large effect size, with high statistical power for detecting a response. Because of the large 8 effect size, the use of an ED_{50} endpoint requires some conservatism in the processing of ED_{50} 9 values from multiple trials and treatments; this process is described below.

10 ED₅₀ values were calculated using raw data from the Phase II Studies (Attachment F.7), using 11 methods described in Appendix F (Section F.3.4.2.1). ED₅₀ values calculated for largemouth 12 bass (swim-up), medaka (swim-up and 15 days post swim-up), and rainbow trout (600 DTU or 13 approximately 3 days post swim-up) exposed to Housatonic River extracts and PCB-126 and TCDD standards are presented in Table 5.3.2 and Figures 5.3-8 and 5.3-9. Because Tillitt et al 14 15 (2003b) emphasize TEQ as exposure measures, TEQ were converted to tPCB concentrations 16 using linear regression (i.e., TEQ versus tPCB doses); regression equations used for the conversions are provided in Appendix F (Table F.3-9). For many of the trials, an ED_{50} value 17 18 could not be calculated because the data did not meet the criteria specified above, the magnitude 19 of the effect was too small to calculate a toxicity threshold, and/or there was no dose-related 20 response.

21 **5.3.3.2.2** Threshold Derivation

22 Rather than selecting an individual ED_{50} concentration as a threshold value, the entire 23 distribution of ED_{50} values was considered. The following procedures were applied in the 24 derivation of an egg-based maximum acceptable tissue concentration (MATC):

- 25 26 27
- Selection of Controls—Where ED₅₀ values were calculated separately for the two controls (due to statistically significant differences between controls), the arithmetic mean of the two ED₅₀ values was used. This prevented bias in the MATC derivation from double counting the results of a single trial.
- 29

30

Table 5.3-2

Calculated ED₅₀ Values (tPCBs and TEQ) for Largemouth Bass, Medaka, and Rainbow Trout Exposed in Ovo to Housatonic River Extracts and PCB-126 and 2,3,7,8-TCDD Standards

Endpoint ^a	Species	Life Stage	Extract	Concentration ^b	TEQ (pg/g)
	Largemouth bass	Swim-up	Reach 6 (Trial 1)	136.58 µg tPCBs/g egg	87
			PCB-126 (Trial 2)	657.2 ng PCB-126/g egg	3286
		Swim-up	Reach 5BC (Trial 1) Reach 5BC (Trial 1) 82.49 µg tPCBs/g eq (uninjected) 34.11 µg tP egg (triolein)		34 (uninjected) 14 (triolein)
			Reach 5BC (Trial 3)	43.78 μg tPCBs/g egg	18
ED ₅₀	Medaka		PCB-126 (Trial 2)	46.6 ng PCB-126/g egg (uninjected) 64.4 ng PCB-126/g egg (triolein)	233(uninjected) 322 (triolein)
			PCB-126 (Trial 3)	44.8 ng PCB-126/g egg	224
			2,3,7,8-TCDD (Trial 3)	2667 ng TCDD/g egg	2667
		15 days post swim-up	Reach 5BC (Trial 1)	48.62 μg tPCBs/g egg	20
			Reach 5BC (Trial 3)	9.91 μg tPCBs/g egg (uninjected) 22.25 μg tPCBs/g egg (triolein)	4.0 (uninjected) 9.1 (triolein)
			PCB 126 (Trial 2)	29 ng PCB-126/g egg	145
			PCB 126 (Trial 3)	31 ng PCB-126/g egg	155
		600 DTU	Reach 6 (Trial 1)	11.85 μg tPCBs/g egg	7.6
			Reach 5BC (Trial 4)	107.21µg tPCBs/g egg	44
	Rainbow trout		PCB-126 (Trial 1) 24.2 ng PCB-126/g egg		121
			PCB-126 (Trial 2) 84.6 ng PCB-126/g egg		423
			2,3,7,8-TCDD (Trial 1) 294 pg TCDD/g egg		294
			2,3,7,8-TCDD (Trial 2)	152 pg TCDD/g egg	152

^aED₅₀ endpoints were based on the combined effects observed per fish (pathology and mortality combined) for each species/life stage/treatment/dose combination.

^bTotal PCB concentrations were interpolated from non-standardized concentrations through linear regression. PCB 126 and 2,3,7,8-TCDD concentrations were converted using toxicity equivalent factors for fish provided in Van

10 den Berg et al. (1998)



Figure 5.3-8 TEQ ED₅₀ Estimates for Fish Exposed to Housatonic River Extracts and PCB-126 and TCDD Standards (Logarithmic Scale)



Figure 5.3-9 tPCB ED₅₀ Estimates for Fish Exposed to Housatonic River Extracts and PCB-126 and TCDD Standards (Logarithmic Scale)

- Exclusion of Reference Station—Threemile pond extracts were excluded from the MATC derivation; the maximum PCB concentrations tested for these reference fish (0.15 mg/kg tPCB and 6 pg/g TEQ) were insufficient to yield large toxic responses or provide meaningful information on the magnitude of the ED₅₀ value. The concentrations in these extracts were well below the levels causing effects in the contaminated site extracts.
- 7 Treatment of Unbounded ED_{50} —Where no ED_{50} value could be determined, a 8 value was set equal to the highest concentration tested. This approach is 9 conservative, since effects in these trials would theoretically only have occurred at 10 concentrations higher than the highest concentration tested. However, it was 11 considered necessary to include results from all trials (including those that did not 12 yield a 50% response), and some conservatism was warranted due to the large effect 13 size under consideration.
- Statistical Measure The arithmetic mean of the relevant ED₅₀ values was chosen as the MATC. The analysis was also conducted using median values, and the results were very similar. Use of a central tendency value ensures that the thresholds derived are not based on results from a single trial, and balances sensitive trials with those that did not yield large effect sizes.
- Species Included —As indicated above, the ED₅₀ values were variable but did not differ substantially among the three test species. The mean rainbow trout ED₅₀ values were only slightly lower than the combined medaka and largemouth bass values (i.e., within a factor of two). Therefore, the ED₅₀ values from all three species were combined to yield an integrated ED₅₀ value for all species that is deemed protective of all PSA fish species.
- Based on the above criteria, ED₅₀ values in eggs were calculated as 104 mg/kg tPCB and 89 pg/g
 TEQ.
- Before the threshold values could be applied in risk characterization, the egg concentrations first
 required conversion to whole body concentrations. For this purpose, three independent
 evaluations of egg versus whole weight concentrations were considered:
- Site-Specific Conversion—PCB chemistry data were available in whole bodies and ovaries of largemouth bass. These data have the advantage of site-specificity, but are limited to a single species, and require the assumption that ovaries and eggs are in chemical equilibrium during the reproductive phase.
- Direct Comparison of Eggs to Whole Bodies (from Literature)—Niimi (1983)
 measured the concentrations in whole body and egg tissue concurrently for several
 freshwater species. These data have the advantage of multiple species, some of
 which are found in the PSA, but are not site-specific.

• Direct Comparison of Eggs to Muscle (from Literature)—Monosson (1999) compiled information on ratios of ovary or egg concentrations relative to muscle tissue (fillet). Results of eleven studies are presented; however the approach requires estimation of the relationship between muscle and whole body concentrations.

5 The greatest weight was placed on the first method, due to site-specificity, with moderate weight 6 placed on the Niimi (1983) information. The lowest weight was placed on the Monosson (1999)

7 data due to the extrapolations required and lack of site- and species-specificity. Details on each

8 method are provided in Appendix F (Section F.3.4.2.2).

1

2 3

4

9 Considering the three methods for extrapolation of concentrations from egg to adult whole body,

10 a value of 0.5 was selected, and was applied for both tPCB and TEQ. This value is slightly

11 higher than the site-specific ratios based on regression of site-specific ovary and whole body

12 data, but is lower than the values indicated by the literature reviews.



25 **5.3.4** Derivation of Literature-Based Sediment Effects Metrics for PAHs

PAHs tend to be associated with bottom sediment because of the chemical and physical properties that affect their environmental fate. As a result, fish that are closely associated with sediment (i.e., benthic species) are most affected through direct contact or incidental ingestion of PAH-contaminated sediment. Exposure to PAHs in sediment can result in reproductive, developmental, and carcinogenic effects in fish.

MK01|O:\20123001.096\ERA_PB\ERA_PB_5.DOC

2	
3	
4	

5

6

7

8

9

1

Potential Effects of PAHs to Fish

- Reproductive and Developmental Effects PAHs have been shown to be immunotoxic and to have adverse effects on reproduction (reduced egg fertility, increased fry mortality) and development, with egg and larval stages the most vulnerable.
- Carcinogenic Effects A number of studies of bottom-dwelling fish, including tomcod, English sole, Pacific staghorn sculpin, rock sole, brown bullhead, and winter flounder indicate a link between sediment exposures and hepatic neoplasms.

10

The assessment of fish toxicity of PAHs is complicated by the fact that PAHs are readily metabolized by most aquatic animals, including teleost fish (Johnson et al. 2002). Because tissue concentrations are not a reliable predictor of adverse effects in fish, the relationship between exposure to contaminated sediment and effects can be used to derive an effects threshold (described below).

16 Development of the sediment PAH effects metrics were based on two major groups of studies of 17 field exposures of benthic fish, conducted in Black River, Ohio, and in the Puget Sound, 18 Washington (detailed in Appendix F; Attachment F.5). Because these studies investigated 19 effects in benthic fish with high sediment exposures, the thresholds derived from these studies 20 represent conservative estimates, which are expected to be protective of fish species present in 21 the Housatonic River. Lack of biomagnification of PAHs (due to metabolism) means that fish 22 feeding directly on bottom sediment and associated prey are at the greatest risk from PAH 23 contamination.

24

25

26 27

Sediment Threshold for Total PAH

 Most relevant threshold (10 mg/kg PAH) – Based on brown bullhead effects observed over changing sediment PAH levels at an industrial site on the Black River, Ohio.

28

The 10 mg/kg threshold was considered the most appropriate value for application to the Housatonic ERA because of common environments (i.e., freshwater), species (i.e., brown bullhead), and endpoints of interest. The worst-case threshold of 1 mg/kg provides a level at which there is very high confidence in a conclusion of no effect, but it is likely overprotective,
 especially for non-benthic Housatonic River species.

3 In addition to the threshold for total PAHs, thresholds of 0.92, 0.68, and 0.64 mg/kg were

4 identified for individual PAHs, phenanthrene, benzo(a)anthracene, and indeno(1,2,3-cd)pyrene,

5 respectively; these values were based on the no observed effect concentrations (NOECs) from

6 EPA (2000). Refer to Attachment F.5 for additional information on the derivation procedure.

1 5.4 RISK CHARACTERIZATION

2 **5.4.1** Introduction

3 The risk characterization for fish integrates the exposure assessment (Section 5.2) and effects 4 assessment (Section 5.3) to evaluate the assessment endpoint of survival, growth, and 5 reproduction of fish in the Housatonic River.

6 The following three lines of evidence were used to develop the risk characterization in the

- 7 Housatonic River fish risk assessment (Figure 5.1-4):
- Field surveys Two field surveys were conducted in the study area. EPA evaluated fish abundance/biomass and conducted an ecological characterization of the site. GE independently evaluated largemouth bass reproduction, community, and habitat data. Interpretation of these studies was constrained by the data limitations; therefore, only a qualitative analysis was performed.
- 13 • Comparison of field-measured exposures to effects levels or thresholds – For 14 these endpoints, the risk characterization integrated exposure and effects by relating the two terms quantitatively. This method consisted of a comparison of tissue 15 16 chemistry (PCBs and TEQ) to tissue effects thresholds, and sediment chemistry 17 (PAHs) to literature-based sediment effects thresholds. Hazard quotients were calculated for PCBs by comparing observed tissue concentration data to site-specific 18 19 MATCs. Probabilities of exceeding various effects threshold levels were also 20 calculated.
- Site-specific toxicity study results These endpoints (e.g., Phase I and Phase II toxicity tests) directly evaluated biological responses to COCs.

23 These lines of evidence allowed for a robust weight-of evidence assessment of the potential for

risk using the approach of Menzie et al. (1996).

Two of the three lines of evidence listed above suggest some degree of harm to fish in the Housatonic River. Although the field surveys suggest that PCBs and/or other COCs are not causing catastrophic effects to fish reproduction and community structure, they were inconclusive with respect to evaluating more subtle potential alterations to fish community health and reproductive capacity.

1 5.4.2 Field Surveys

2 5.4.2.1 EPA Fish Community Study

A survey of fish biomass in the mainstem and Woods Pond (Reaches 5 and 6) was conducted by
EPA in fall 2001 to generate information for use in the modeling study and risk assessments
(Woodlot 2002).

Biomass estimates were developed for each species for both prey-sized fish (fish <10 cm) and all
other size classes pooled (fish >10 cm). Separate estimates were developed for each species/size
class group in each of five reaches (i.e., Reaches 5A, 5B, 5C, 5D, and 6). Separate age-class
biomass estimates, however, were generated for largemouth bass in each reach. Survey methods
were based on existing population estimation protocols (Zippin 1958, Ricker 1975, Mitro and
Zale 2000).

Electrofishing sampling was conducted during 21 to 25 August 2000 and 23 to 25 October 2000. Eleven sample locations were randomly chosen and surveyed within each reach. One location per reach was surveyed using multi-pass, or depletion, sampling; and the remaining 10 sites were surveyed using single-pass sampling (i.e., one pass of the electrofishing boat). Survey areas included the entire channel width and were generally about 200 m in length. Based on previous electrofishing surveys in each reach, this length was expected to provide a suitable number of fish for the study (Woodlot 2002).

19 An ecological characterization of the Housatonic River PSA (Appendix A.1) was also conducted 20 to describe the aquatic and ecological habitats throughout the study area. Four separate fish 21 collection events occurred within the PSA during 1998-2000. The principal method employed to 22 collect fish was electro-shocking fish (electrofishing) from one or two boats operated by the 23 United States Fish and Wildlife Service (USFWS 1999). In September and October 1998, 24 electrofishing was conducted to collect fish community characterization data and fish tissue. 25 Timed (30-minute) surveys to collect community composition data were conducted between 26 river miles 3 and 4 and between river miles 8 and 11. During each timed event, the total number 27 of all fish per species observed was estimated and recorded. In addition, target species within 28 different taxonomic fish groups (e.g., largemouth bass, yellow perch, brown bullhead, common carp) were collected for tissue analysis. Collections occurred along river miles 3 and 7 to 11,
 and at Woods Pond. Each fish was weighed and measured prior to processing for analysis. A
 sample of otoliths and scales was collected from largemouth bass to estimate ages of specimens
 (USFWS 1999).

Results of the EPA biomass study are presented in Woodlot (2002) and summarized in Table
F.4-1. Chadwick (1993, 1994) also conducted an earlier biomass study that generally yielded
lower biomass estimates (Table F.4-2).

8 Fish captures in the Woodlot (2002) study totaled 7,064 individuals and included 17 species, 2 9 hybrids (bluegill-pumpkinseed and chain-redfin pickerel), and 1 group of taxa (cyprinids). The 10 most common predators were largemouth bass, yellow perch, and northern pike. Common 11 forage fish were bluegill, rock bass, pumpkinseed, and cyprinids, whereas the most abundant 12 bottom feeders were white sucker, common carp, brown bullhead, and goldfish. Other species 13 captured included smallmouth bass, chain pickerel, redfin pickerel, brown trout, rainbow trout, 14 black crappie, yellow bullhead (a single individual), and the hybrids mentioned above. Woodlot (2002; Attachment C) provides the capture data in a standardized biomass (g/m^2) format. 15

16 Results of the fish collection events associated with the Ecological Characterization Study are 17 provided in Appendix A.1. White suckers were clearly the dominant fish species in Reaches 5A 18 and 5B. They still represented the greatest component of the sample biomass in Reach 5C, but 19 they declined to a smaller component of the fish community in the backwaters and Woods Pond. 20 In Reach 5C, as well as in the backwaters and Woods Pond, carp become a common member of 21 the bottom-feeding guild. Goldfish and brown bullhead also represent significant portions of the 22 bottom-feeding guild in the backwaters and Woods Pond. Bluegills, pumpkinseed, cyprinids, 23 and rock bass share dominance of the forage fish group, which comprised 11 to 24% of the 24 overall fish community (based on biomass), in Reaches 5A–5C and the backwaters. Bluegills, 25 however, were abundant in Woods Pond, where they represented 30% of the total biomass 26 sample and forage fish, as a group, comprised 40% of the overall fish community. Largemouth 27 bass and yellow perch were the predominant predators in all reaches.

Based on these and other biological surveys, it is clear that the five representative fish species
chosen for this assessment (i.e., largemouth bass, pumpkinseed, yellow perch, white sucker,

brown bullhead) are found in suitable habitats of the PSA. There is also evidence that these 1 2 species are self-sustaining; therefore, total reproductive failure or catastrophic effects to the fish 3 populations are not apparent. A pattern of fish assemblages was apparent from the biological 4 surveys; this pattern appears related to major changes in habitat across the PSA. Specifically, the 5 downstream areas of the PSA (Reach 5C, 5D, and Woods Pond) contained a greater proportion 6 of fish that (a) have a soft-sediment-based feeding strategy, or (b) exploit epifauna on the 7 macrophyte beds, which are more abundant downstream. Within the forage fish category, the 8 upstream coarser-grained areas had a higher biomass of cyprinids (e.g., fallfish, shiners), 9 whereas the downstream areas had a higher biomass of centrarchid sunfish (e.g., bluegill, 10 pumpkinseed). These differences in species assemblages appear to be mainly related to habitat 11 differences among reaches.

12 Chadwick (1993, 1994) also conducted an earlier biomass study that generally yielded lower 13 biomass estimates. Based on these and other field surveys, it is clear that the five representative 14 fish species chosen for this assessment (i.e., largemouth bass, pumpkinseed, yellow perch, white 15 sucker, and brown bullhead) are found within suitable habitats of the PSA. There is also 16 evidence that these species are self-sustaining; therefore, total reproductive failure or 17 catastrophic effects to the fish populations are not apparent.

Because of the variability in habitat across the PSA, small-scale variability in PCB concentrations, and the small gradient in PCB tissue concentrations across the PSA, it is difficult to discriminate habitat influences from potential contaminant influences. Therefore, meaningful statistical assessment of PCB or TEQ relationship to fish community parameters was not feasible. In summary, no quantitative conclusions can be drawn regarding the health of the Housatonic River fish community based on the field surveys conducted to date.

24 **5.4.2.2** GE Largemouth Bass Community and Reproduction Study

A field study was conducted in the summer and early fall of 2000 and 2001 to assess largemouth bass habitat, community structure, and reproduction in the Housatonic River (R2 Resource Consultants Inc. 2002). The assessment of these metrics was made independent of contaminant concentrations in the environment and fish tissue; therefore no assessment of concentrationresponse was attempted. The study area extended from the confluence of the East and West branches downstream to the Woods Pond Dam, and included major stream branches and
 tributaries.

3 The methods applied in the GE study included the following:

Nest surveys

4

5

6

7

- Habitat surveys
- Community estimates

8 The study confirmed that there is a population of largemouth bass in the Housatonic River. 9 However, the extent of tributary recruitment remains unknown, and more important, the question 10 of whether PCBs are causing or may cause effects to population strength or viability is largely 11 unaddressed.

Reproduction was confirmed to occur in the study area, although the data suggest that reproductive success is lower than in other systems. Growth also appears lower than in other systems. The current population (biomass), which is not exploited due to a consumption advisory that in effect creates a catch-and-release fishery, is dominated by older, larger fish in good condition. Although the population appears stable, this is likely due to the lack of typical harvesting pressure, which places very little demand on recruitment in order to sustain overall numbers.

Summaries of the three major components of the GE study are provided below. Additional
details are provided in Appendix F (Section F.4.1.2) and in R2 Resource Consultants Inc. (2002).

21 **5.4.2.2.1** Nest Surveys

In the spring and summer of 2000 and 2001, nest surveys were conducted to determine if largemouth bass in the Housatonic River were successfully reproducing and to assess the condition of young-of-year (YOY) bass. Data collected included nest condition, egg presence and condition, and identification/enumeration of fry.

The nest survey data do not provide strong evidence of a high degree of reproductive success. Of the 77 nests observed, only 13 (16.9%) contained eggs described as being in "good" condition; furthermore, only 17 (22%) produced sac fry or swim-up fry. Comparisons to literature information suggest relatively poor spawning success at the sites examined in the Housatonic. Length data for YOY suggest that YOY from the Housatonic are somewhat smaller than YOY largemouth bass sampled at other locations at similar latitudes. Overall, although the data provide an indication that reproduction is occurring, the data do not provide a measure of reproductive rates. Comparisons of nesting success and growth for YOY suggest that reproduction may not be similar to other systems. Ultimately, the apparent self-sustaining nature of this population may be more a function of the low mortality rate of the adults rather than high reproductive output.

8 5.4.2.2.2 Habitat Surveys

9 In the spring of 2000, habitat surveys were conducted at a total of 13 locations in the main river 10 channel, backwater areas, three major branches, and six tributaries. In 2001, surveys were 11 conducted at 15 locations in the backwater areas. During the surveys, habitat characteristics 12 (e.g., stream gradient, substrate), water chemistry (pH, DO, conductivity, temperature), stream 13 velocity, and other physical attributes were measured.

The results indicate that in the main channel largemouth bass habitat is good and that the tributaries generally have poor habitat, with the exception of Moorewood and Yokun Brooks. The study concludes that the fish community is not controlled by fluctuations in temperatures and water levels, although nesting and spawning success may be sensitive to these factors. The incidence of dead or fungus-affected eggs in the nests was also attributed to conventional water quality variables. The investigators did not speculate about the influence of other potential factors, including contaminants.

21 **5.4.2.2.3** Community Estimates

In June or late July/early August 2000, electrofishing surveys were conducted in the main channel, backwater areas, and the East and West Branches. In 2001, sampling was conducted only in main channel and backwater sites located between the confluence of the East and West Branches. Data collected included lengths/weights, fish aging, and select community metrics including the proportional stock density (PSD; a measure of the dominance of large fish in the population) and relative weight (a measure of condition of the fish). Generally, largemouth bass were found throughout the sites sampled, except for selected tributaries. No sampling was done on Moorehead and Yokun Brooks, even though they had been identified as the two tributaries that offered suitable habitat. Weight and length data were used to assess the condition of largemouth bass; these data demonstrated that bass were robust and in good condition, relative to other systems.

6 The age analyses indicated that the bass population consists primarily of large older fish. These 7 analyses also indicated that largemouth bass in the Housatonic River grow at a slower rate as 8 they age. The high proportion of older fish is a function of the lack of harvesting pressure on the 9 community. Comparison of the age structure of the largemouth bass community and/or 10 condition of individual fish to other sites is not a reliable measure of effects.

11 5.4.3 Comparison of Estimated Exposures to Derived Effects Metrics

For the representative fish species (largemouth bass, pumpkinseed, yellow perch, white sucker,
brown bullhead), risks were assessed separately for three different river segments (PSA, Reaches
7 and 8, and Reach 9 and below).

15 **5.4.3.1 Total PCBs**

16	Summary of Tissue Effects Thresholds for tPCB
17	 31 mg/kg tPCB – based on literature review (all species).
18	<45 mg/kg tPCB – based on Phase I study (largemouth bass).
19 20	 52 mg/kg tPCB – based on Phase II study (warmwater species and rainbow trout).
21 22	 12 mg/kg tPCB – based on Phase I/Phase II studies, and literature information on trout sensitivity (coldwater species downstream of the PSA).

23 **5.4.3.1.1 PSA (Reaches 5 and 6)**

Overall, the independent lines of evidence exhibit strong concordance in the concentrations of PCBs expected to cause adverse responses to PSA fish. Although there is some variation in the responses across reaches and across species, there is sufficient concordance for the development of threshold levels applicable to all species and reaches. In the risk characterization, exposure concentrations were displayed in relation to multiple effects thresholds (i.e., literature-based and site-specific reproductive study based). These multiple thresholds also depict some of the
 uncertainty inherent in the effects thresholds.

Figure 5.4-1 depicts hazard quotients for PSA fish tissue concentrations compared to the average
of the site-specific (Phase I and Phase II) fish effects thresholds derived for the PSA (i.e., 49
mg/kg tPCB). All mean HQs are below 3 and median HQs are below 2, indicative of an
ecologically significant but low magnitude of risk.

Figures 5.4-2 through 5.4-6 show the cumulative distribution plots for observed whole body tPCB concentrations for each species and reach within the PSA. The vertical lines represent effects concentrations from the literature (31 mg/kg tPCB), Phase II toxicity (52 mg/kg tPCB), and site-specific toxicity studies (<45 mg/kg tPCB). Table 5.4-1 displays the 95th percentile and exceedance probabilities for the threshold effects concentrations by species and PSA reach for observed fish tissue concentrations.

Within the PSA, Table 5.4-1 indicates a moderate to high probability of exceedance for most representative species and reaches. However, Figures 5.4-2 through 5.4-6 indicate that the magnitude of exceedance is low to moderate, depending on the effects threshold adopted.

16 **5.4.3.1.2 Downstream of PSA (Warmwater Fish)**

Table 5.4-2 displays the exceedance probabilities for the literature-based and site-specific effects concentrations, as well as the 95th percentile tPCB whole body tissue concentration for each species collected in Reach 8 during the EPA 1998-99 sampling. Although there were limited data for this portion of the river, significant risk to representative fish species (e.g., largemouth bass and yellow perch) is predicted from tPCBs downstream of Woods Pond (Reach 6) to Rising Pond (Reach 8). However, predicted risks are low for pumpkinseed and brown bullhead.

The analysis of the Stewart (1982) data, which required extrapolation from fillet concentrations to whole body concentrations, generally supports the evaluation of the EPA data. Specifically, there was a low probability of exceedance of the site-specific thresholds of 45 and 52 mg/kg tPCB, and a moderate probability of exceedance for the literature-based threshold of 31 mg/kg. The data also indicate a reduction in risk with distance downstream from the PSA.



1 2

MC75th and MC25th represent quartiles (i.e., 25th and 75th percentiles)

3 WB = whole body individuals; WB-R = whole body reconstituted; CM = multiple whole body fish composites

4 Figure 5.4-1 Hazard Quotients for tPCBs in PSA Fish Based on Comparison to the Mean Site-Specific Fish 5 Toxicity Threshold (49 mg/kg tPCB) (Logarithmic Scale)

MK01|O:\20123001.096\ERA_PB\ERA_PB_5.DOC











Table 5.4-1

Probabilities of Exceedances in the PSA for tPCBs

	Reach 5A				Reach 5BC				Reach 6			
Species	95th Percentile	Probability of Exceeding Thresholds		95 th Percentile	Probability of Exceeding Thresholds		95th Percentile	Probability of Exceeding Thresholds				
	(mg/kg)	31 mg/kg	45 mg/kg	52 mg/kg	(mg/kg)	31 mg/kg	45 mg/kg	52 mg/kg	(mg/kg)	31 mg/kg	45 mg/kg	52 mg/kg
BB	NA ^a	NA	NA	NA	73	47%	21%	21%	75	60%	28%	24%
LB	213	50%	50%	50%	209	80%	56%	56%	193	81%	58%	52%
PS	NA	NA	NA	NA	73	68%	36%	28%	59	56%	12%	12%
WS	170	88%	81%	75%	170	77%	62%	58%	169	87%	60%	53%
YP	228	96%	96%	96%	114	100%	100%	80%	123	84%	84%	80%

- 5 BB Brown Bullhead
- 6 LB Largemouth Bass
- 7 PS Pumpkinseed
- 8 WS White Sucker
- 9 YP Yellow Perch
- 10
- 11 Fish effects thresholds (vertical bars):
- 12 31 mg/kg tPCB Literature based threshold protective of PSA species
- 13 <45 mg/kg tPCB Phase I toxicity study threshold (unbounded)
- 14 52 mg/kg tPCB Phase II toxicity study effects threshold for warmwater species and rainbow trout

⁴ NA = not available

Table 5.4-2

Probabilities of Exceedances in Reach 8 for tPCBs and TEQ, Based on EPA Sampling

tPCBs	Species	95th Percentile	Probability of Exceeding Thresholds			
		(mg/kg)	31 mg/kg	45 mg/kg	52 mg/kg	
Observed	BB	9.94	0%	0%	0%	
	LB	60.4	32%	10%	10%	
	PS	23.7	0%	0%	0%	
	YP	130	67%	17%	17%	
TEQ	Species	95th Percentile (ng/kg)	Probability of Exceeding Thresholds			
			38 ng/kg	45 ng/kg	50 ng/kg	
0 Substitution	BB	16.4	0%	0%	0%	
	LB	123	48%	43%	33%	
	PS	29	0%	0%	0%	
	YP	213	100%	67%	67%	
DL Substitution	BB	28.2	0%	0%	0%	
	LB	131	71%	57%	43%	
	PS	44.5	15%	8%	0%	
	YP	222	100%	83%	67%	

5
6

7

8

9

10

11

12

1

2 3

4

BB Brown Bullhead; LB Largemouth Bass; PS Pumpkinseed; YP Yellow Perch

- Fish effects thresholds:
 - 45 ng/kg TEQ Phase II toxicity study threshold for warmwater species and rainbow trout
 - <38 ng/kg TEQ Phase I toxicity study threshold (unbounded)</p>
 - 50 ng/kg TEQ Literature based threshold protective of PSA species
 - 31 mg/kg tPCB Literature based threshold protective of PSA species
- 52 mg/kg tPCB Phase II toxicity study threshold for warmwater species and rainbow trout
- <45 mg/kg tPCB Phase I toxicity study threshold (unbounded)
- 13

There were limited data below Reach 8 (i.e., below Rising Pond), but the data suggest negligible
 to low risk from tPCBs to warmwater fish species.



4

5

6

7

3 5.4.3.2 2,3,7,8-TCDD TEQ

50 ng/kg TEQ – based on literature review (relevant to PSA fish species)
<38 ng/kg TEQ – based on Phase I study (largemouth bass)
45 ng/kg TEQ – based on Phase II study (warmwater species and rainbow trout)

Summary of Effects Thresholds for TEQ

8 5.4.3.2.1 PSA

9 Figure 5.4-7 depicts hazard quotients for PSA fish tissue concentrations compared to the average 10 site-specific effects threshold (i.e., 42 ng/kg TEQ). All 75th percentile-based HQs exceed 1, but 11 mean and median HQs for adult fish are below 3 for all species. These HQs are indicative of 12 ecologically significant but low magnitude risk.

Figure 5.4-8 shows the cumulative distribution plots for observed whole body TEQ tissue concentrations for each species (using DL substitution for ND congeners). The vertical lines represent the effects concentrations from the literature (50 ng/kg) and site-specific toxicity studies (45, 38 ng/kg). Table 5.4-3 displays the exceedance probabilities for the effects concentrations, as well as the 95th percentile for exposure for each species, substituting concentrations equal to zero or to the DL, respectively, for non-detected congeners. There was a moderate to high probability of exceedance for most fish species for all three effects thresholds.

20 5.4.3.3 PAHs

Table F.2-18 displays summary statistics for sediment chemistry concentrations for the eight individual PAHs identified as COCs. For the three individual PAHs for which thresholds could be determined, the median sediment concentration in each river reach was below the toxicity threshold, indicating negligible to low risk to fish from these contaminants. The median sediment concentrations for total PAH ranged from 3 to 8 mg/kg in the PSA, below the "most relevant" effects concentration of 10 mg/kg tPAH for fish.



MC75th/MC25th are quartiles (i.e., 25th/75th percentiles); WB = whole individuals; WB-R = whole body reconstituted; CM = multiple whole fish compositive
 Figure 5.4-7
 Hazard Quotients for TEQ for Fish in Primary Study Area (PSA) Based on Comparison to the Average Site-Specific Tissue Effects Threshold (42 ng/kg TEQ) (Logarithmic Scale)



Table 5.4-3

Probabilities of Exceedances for TEQ

Non-Detect	Species	95th Percentile	Probability of Exceeding Thresholds			
Methou		(ng/kg)	38 ng/kg	45 ng/kg	50 ng/kg	
	BB	125	75%	66%	56%	
0 Substitution	LB	117	61%	55%	41%	
0 Substitution	PS	58.9	26%	13%	10%	
	YP	151	96%	87%	87%	
	BB	130	88%	78%	72%	
DI Substitution	LB	126	77%	64%	57%	
DL Substitution	PS	66	58%	32%	29%	
	YP	163	98%	98%	96%	

BB	Brown Bullhead
LB	Largemouth Bass
PS	Pumpkinseed
YP	Yellow Perch
0 substitut	tion = non-detectable concentrations were substituted with zeroes
DL substit	tution = non-detectable concentrations were substituted with method detection limit
Fish effect 45 ng <38 n 50 ng	ts thresholds: /kg TEQ – Phase II toxicity study threshold for warmwater species g/kg TEQ – Phase I toxicity study threshold (unbounded) /kg TEQ – Literature based threshold protective of coldwater and warmwater species

1 5.4.4 Site-Specific Toxicity Studies

Results and interpretation of the site-specific toxicity studies are summarized in Sections 5.3.2
and 5.3.3 and detailed in Appendix F; therefore only a synopsis is provided here.

The fish toxicity studies indicate that PCBs are acting upon early life stages of fish, and causing various reproductive and developmental responses. The types of malformations and other effects observed are suggestive of an Ah-receptor (i.e., dioxin-like) etiology. However, the threshold effect levels (as identified in both the literature review and the site-specific studies) each have a moderately high uncertainty. For example, individual ED₅₀ values from Phase II site extracts data span the range of PCB concentrations found in PSA fish.

10 **5.4.5 Weight-of-Evidence Analysis**

A weight-of-evidence evaluation was conducted for the multiple measurement endpoints in the fish ERA to determine whether significant risk is posed to fish from tPCBs. The three-phase approach of Menzie et al. (1996) and the Massachusetts Weight-of-Evidence Workgroup was used. The weight-of-evidence approach involves: (a) assigning weights to each measurement endpoint, (b) determining the magnitude of response observed in the measurement endpoint, and (c) determining the concurrence among measurement endpoints.

The attributes considered in the weight of evidence are described in Section 2, and the rationale for weighting of the measurement endpoints are provided in Appendix F. A summary of the derived weightings for each attribute is provided in Table 5.4-4. The Phase II study yielded the highest overall rating because of the site specificity of the study and the connection to the exposure pathway of greatest interest (maternal transfer).

The magnitude of the response in the measurement endpoint is considered together with the measurement endpoint weight in judging the overall weight-of-evidence (Menzie et al 1996). This requires assessing the strength of evidence of ecological risk, as well as an indication of the magnitude of the response, if present. The weighting values, evidence of risk, and magnitude of responses were combined in a matrix format and are presented in Table 5.4-5.
Table 5.4-4

Weighting of Measurement Endpoints for Fish Weight-of-Evidence Evaluation

Measurement Endpoints:	Endpoint A: Site-	Specific Toxicity	Endpoint	B: Fish Tissue Ch	nemistry	Endpoint C:	Field Surveys
Attributes	A1. Phase I Study	A2. Phase II Study	B1. Observed / Literature Effects	B2. Observed / Phase I Effects	B3. Observed / Phase II Effects	C1. Community Studies	C2. Reproduction Study
I. Relationship Between Measuremen	t and Assessment En	dpoints					
1. Degree of Association	High	High	Mod	High	High	Low/Mod	Low/Mod
2. Stressor/Response	Mod	Mod	Low/Mod	Mod	Mod	Low	Low
3. Utility of Measure	High	High	Mod/High	Mod/High	Mod/High	Low/Mod	Low/Mod
II. Data Quality							
4. Data Quality	High	High	High	High	High	High	High
III. Study Design							
5. Site Specificity	High	High	Low/Mod	High	High	High	High
6. Sensitivity	Low/Mod	Mod/High	Low/Mod	Mod	Mod/High	Low	Low
7. Spatial Representativeness	Mod	Mod	Mod/High	Mod/High	Mod/High	Mod	Mod
8. Temporal Representativeness	High	High	High	High	High	Mod/High	Mod/High
9. Quantitative Measure	High	High	Mod	Mod	Mod	Mod	Low
10. Standard Method	High	High	Mod	Mod	Mod	Mod	Mod
Overall Endpoint Value	Mod/High	High	Mod	Mod/High	Mod/High	Low/Mod	Low/Mod

A: Site-Specific Toxicity

4

5

6

7

8

9

10

11

12

A1 - Reproductive success in site-specific toxicity tests, relative to reference condition

A2 – Reproductive success in site-specific toxicity tests, using dose-response analysis

B: Fish Tissue Chemistry

B1 - Observed fish tissue concentrations relative to literature toxicity threshold

B2 - Observed fish tissue concentrations relative to Phase I study toxicity threshold

B3 - Observed fish tissue concentrations relative to Phase II study toxicity threshold

C: Fish Community and Reproduction Studies

- C1 EPA and GE Fish Community Studies
- 13 C2 GE Fish Reproduction Study

Table 5.4-5

Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Fish Community

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
A. Site-Specific Toxicity			
A1. Reproductive success relative to reference	Mod/High	Yes	Low
A2. Reproductive success dose-response	High	Yes	Intermediate
B. Fish Tissue Chemistry			
B1. Observed fish tissue/ Literature toxic levels	Mod	Yes	Low
B2. Observed fish tissue/ Phase I toxic levels	Mod/High	Yes	Low
B3. Observed fish tissue/ Phase II toxic levels	Mod/High	Yes	Low
C: Fish Community and Reproduction Studies			
C1: EPA Study and GE Community Study	Low/Mod	Undetermined	-
C2: GE Reproduction Study	Low/Mod	Undetermined	-

5

A graphical method was used for displaying concurrence among measurement endpoints (Table 5.4-6); the method involved plotting the five symbols representing site-specific toxicity (A) and fish tissue chemistry (B) endpoints in a matrix, with the weight of the measurement endpoint and the degree of response as axes. This table illustrates that the majority of endpoints indicate, with a moderately high degree of confidence, that there are low magnitude risks to fish in the PSA.

6 **5.4.6 Sources of Uncertainty**

7 The assessment of risks to fish contains uncertainties. Each source of uncertainty can influence 8 the estimates of risk; therefore, it is important to describe and, when possible, specify the 9 magnitude and direction of such uncertainties. Appendix F contains a more complete list of 10 uncertainties; some of the most significant uncertainties are described below.

- Potential for seasonal variation in fish tissue concentrations The vast majority of the PCB data for this project were collected in the late summer and early fall (September October). Because maternal transfer to juveniles is expected to be the most important risk pathway for PCBs, changes in lipid content during the spawning period may affect the concentrations of PCBs delivered to the eggs.
- Fillet data There is some uncertainty with respect to risks for fish for which only
 fillet concentration data are available, since this required conversions to estimated
 whole body concentrations.
- Biological relevance of the injected doses used in Phase II toxicity study To develop a concentration-response relationship, it was assumed that the dose delivered to the egg was proportional to ovary concentration in the parent fish, and that these ovary concentrations would be related to whole body tissue concentrations on the basis of equilibrium partitioning to lipids. These assumptions required extrapolations and carried associated uncertainties.
- Lack of synopticity in Phase I toxicity study Because different fish were evaluated for tissue chemistry than were assessed for pathologies, the exposure and effects information for Phase I were not directly synoptic. However, the approach used to estimate the exposure concentrations was unbiased.
- Literature threshold uncertainty The literature effects thresholds for tPCBs and for TEQ have a number of uncertainties associated with them, including lack of information for Housatonic River representative species; limited number of studies that met the screening criteria used in the literature review; limited number of studies that replicated the maternal transfer of contaminants to offspring; and variability of effects thresholds identified.

Table 5.4-6

1 2 3

Risk Analysis for Risk Exposed to tPCBs and TEQ in the Housatonic River PSA

	Weighting Factors (increasing confidence of weight)				
Harm/Magnitude	Low	Low-Moderate	Moderate	Moderate-High	High
					۸2
					A2
	<u> </u>				
		C1, C2			

5 6

6 A: Site-Specific Toxicity

- 7 A1 Reproductive success in site-specific toxicity tests, relative to reference condition
- 8 A2 Reproductive success in site-specific toxicity tests, using dose-response analysis

9 B: Fish Tissue Chemistry

- 10 B1 Observed fish tissue concentrations relative to literature toxicity threshold
- 11 B2 Observed fish tissue concentrations relative to Phase I study toxicity threshold
- 12 B3 Observed fish tissue concentrations relative to Phase II study toxicity threshold

13 C: Fish Community and Reproduction Studies

- 14 C1 EPA and GE Community Studies
- 15 C2 GE Reproduction Study

- Toxicity study uncertainties The concentration-response relationships in the Phase I and Phase II studies were variable, and other organic contaminants (other than PCBs) may have been present that could confound the concentration-response curves for PCBs. In the Phase II studies, effects observed were variable for a given standard or extract among the different species and life stage combinations.
- Extrapolation of egg thresholds to whole body burdens The extrapolation of 6 7 concentrations of PCBs in egg to whole body concentrations has a degree of 8 associated uncertainty. The lipid content and type were not measured in eggs, which 9 can vary between species and between adults and eggs. PCB accumulation and 10 distribution in fish tissues is strongly influenced by the lipid content and lipid type (polar, neutral, nonpolar); thus accumulation and deposition of PCBs into tissues 11 including developing oocytes can be influenced by the percent and type of these 12 13 lipids (Monosson 1999).
- The exposure component for the field surveys is highly uncertain. The largemouth bass study did not quantify contaminant exposures in either sediment or fish tissue.
 Where chemistry data are available for relation to other field survey data (e.g., EPA biomass and abundance estimates), the exposure assessment is confounded by significant habitat variation across the PSA.
- The assessment of effects in the field survey studies was uncertain because these
 studies could not detect anything less than very large responses in the local
 population.
- 22

5.4.7 Downstream Extrapolation

23 5.4.7.1 Risks to Warmwater Fish Downstream of the PSA

As was done for the PSA, risks to warmwater fish were evaluated based on concentrations of tPCBs in fish tissue. A maximum acceptable threshold concentration (MATC) of 49 mg/kg tPCB in tissue (whole-body, wet weight) was developed for the PSA using the average of sitespecific (Phase I and Phase II) toxicity effects thresholds, and was also applied to areas downstream of Woods Pond using the available warmwater fish (e.g., bass, perch, sunfish) tissue data.

In the case of the fish, each downstream reach (Reaches 7 through 16) was evaluated as a unit, and the mean fish tissue concentration in the reach was compared with the threshold concentration to determine potential risk. Only data collected since 1998 were used in this analysis. Results are provided in Appendix F (Figure F.4-10). The moderate risks observed in the PSA
 decline to below levels of concern in Reaches 7 through 9, in the section of the river between
 Woods Pond and the Massachusetts/Connecticut state line. Potential risks were also not
 indicated in the Connecticut portion of the river.

5 5.4.7.2 Risks to Coldwater Fish (Trout) Downstream of the PSA

6 Trout were evaluated separately from PSA fish species because of apparent differences in the 7 sensitivity of trout to PCBs as indicated by the results of the site-specific fish toxicity studies 8 (Tillitt et al. 2003b) and the generally higher PCB concentrations in trout due to their increased 9 lipid content. Although the ED_{50} values for trout were within a factor of 2 of warmwater species 10 in the Phase II trials, other indications of toxicity (Tillitt et al. 2003b) suggest that rainbow trout 11 were slightly more toxic than the warmwater species. Furthermore, the rainbow trout strain 12 applied in the Phase II testing (Tillitt, personal communication 2003) is less sensitive than other 13 test strains, and the sensitivity of other downstream trout species (e.g., brown trout) has not been 14 assessed. Therefore, the PSA effects threshold of 49 mg/kg tPCB was divided by a factor of 4 to 15 account for potential increased sensitivity of downstream coldwater species (i.e., coldwater 16 MATC of 12 mg/kg tPCB whole body, wet weight). Because of the more limited database for 17 trout, a number of extrapolations were necessary to convert available warmwater fish data and/or 18 trout fillet data to estimated whole body concentrations for trout. These extrapolations are 19 summarized in Appendix F (Section F.4.6.2).

Results are provided in Appendix F (Figure F.4-11). In general, some potential risk to trout from PCBs was found in river reaches from Woods Pond Dam down to and including Reach 9. These risks were marginal, and are uncertain due to incertitude about the sensitivity differences for various trout species. Potential risk to trout was not evaluated downstream of Reach 12 due to lack of suitable trout habitat.

25 5.4.8 Risk Assessment Conclusions

Overall, evaluation of the fish assessment endpoint suggests ecologically significant but low magnitude risk to fish in the Housatonic River from both tPCBs and PCB TEQ, based on a weight-of-evidence evaluation of multiple endpoints. Other COCs, such as PAHs and metals, were not present in the PSA at concentrations expected to cause pronounced effects, although
 marginal PAH toxicity could not be conclusively ruled out.

3 The confidence in the numerical effects thresholds that support this conclusion is moderate. 4 Strength in the conclusions was derived from the concordance in predictions of risk from 5 multiple measurement endpoints; however, there is some uncertainty associated with several of the endpoints. Because the effects thresholds derived in this study span a similar range as the 6 7 observed fish tissue concentrations in the PSA, the uncertainty inherent in the threshold 8 derivation has large implications for the prediction of actual risks to the local fish population. 9 Use of a lower-bound threshold results in a prediction of significant adverse effects for the vast 10 majority of species in all PSA reaches. However, use of higher-end thresholds would lead to a 11 conclusion of low risks for the same fish. Catastrophic risks, such as total reproductive failure or 12 widespread direct mortality of adults, are not predicted for any species because the magnitude of 13 exceedance of conservative effects thresholds is generally marginal to moderate (i.e., within a 14 factor of 5).

15 **5.5 REFERENCES**

16 ASTM (American Society for Testing and Materials). 2002. Standard guide for conducting early

17 life-stage toxicity tests with fishes. In: Annual Book of ASTM Standards, E 1241-98, West

18 Conshohocken, PA.

19 BBL (Blasland, Bouck & Lee, Inc.) and QEA (Quantitative Environmental Analysis, LLC).

20 2003. *Housatonic River – Rest of River RCRA Facility Investigation Report*. Prepared for 21 General Electric Company. January 2003.

- Berlin, W.H., R.J. Hesselberg, and M.J. Mac. 1981. Growth and mortality of fry of Lake
 Michigan lake trout during chronic exposure to PCBs and DDE. Tech. Pap. U.S. Fish Wildl. Ser.
 105:11-22.
- Chadwick (Chadwick & Associates). 1993. Fisheries Investigation of the Housatonic River,
 Massachusetts. Prepared for General Electric Company, Pittsfield, MA.
- Chadwick (Chadwick & Associates). 1994. Aquatic Ecology Assessment of the Housatonic
 River, Massachusetts. Prepared for General Electric Company, Pittsfield, MA.
- 29 Coles, J.F. 1996. Organochlorine Compounds and Trace Elements in Fish Tissue and Ancillary
- 30 Data for the Connecticut, Housatonic, and Thames River Basin Study Unit, 1992-1994. U.S.
- 31 Geological Survey Open-File Report 96-358, 26 pp.

- 1 EPA (United States Environmental Protection Agency). 1999. Development of a tissue trigger
- 2 level from bioaccumulated tributyltin in marine benthic organisms: West Waterway, Harbor
- 3 Island Superfund Site, Seattle, WA. U.S. Environmental Protection Agency, Region 10, Seattle,
- 4 WA.
- 5 EPA (United States Environmental Protection Agency). 2000. Health of bullhead 6 remedial dredging. States Environmental in an urban fishery after United 7 Protection Agency, Great Lakes National Program Office. Chicago, Illinois. URL: 8 http://www.epa.gov/grtlakes/sediments/Bullhead/report.html.
- 9 Johnson, L, T. Collier, and J.E. Stein. 2002. An analysis in support of sediment quality
- 10 thresholds for polycyclic aromatic hydrocarbons (PAHs) to protect estuarine fish. Aquatic
- 11 Conservation: Marine and Freshwater Ecosystems 12:518-538.
- 12 Johnson, R.D., J.E. Tietge, K.M. Jensen, J.D. Fernandez, A.L. Linnum, D.B. Lothenbach, G.W.
- 13 Holcombe, P.M. Cook, S.A. Christ, D.L. Lattier, and D.A. Gordon. 1998. Toxicity of 2,3,7,8-
- 14 tetrachlorodibenzo-p-dioxin to early life stage brook trout (Salvelinus fontinalis) following
- 15 parental dietary exposure. *Environ. Toxicol. Chem.* 17(12):2408-2421.
- 16 Malins, D.C., B.B. McCain, D.W. Brown, S. Chan, M.S. Myers, J.T. Landahl, P.G. Prohaska,
- 17 A.J. Friedman, L.D. Rhodes, D.G. Burrows, W.D. Gronlund, and H.O. Hodgins. 1984. Chemical
- 18 pollutants in sediments and diseases of bottom-dwelling fish in Puget Sound, Washington.
- 19 Environmental Science and Technology 18(9):705-713.
- 20 Malins, D.C., M.M. Krahn, M.S. Myers, L.D. Rhodes, D.W. Brown, C.A. Krone, B.B. McCain 21 and Sin-Lam Chan. 1985. Toxic chemicals in sediments and biota from a creosote-polluted
- and Sin-Lam Chan. 1985. Toxic chemicals in sediments and blota from a creosote-polluted
- 22 harbor: relationships with hepatic neoplasms and other hepatic lesions in English sole
- 23 (Parophrys vetulus). Carcinogenesis 6(10):1463-1469.
- Mauck, W.L., P.M. Mehrle, and F.L. Mayer. 1978. Effects of the polychlorinated biphenyl
 Aroclor[®] 1254 on growth, survival, and bone development in brook trout (*Salvelinus fontinalis*) *J. Fish. Res. Board Can.* 35:1084-1088.
- Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S.
 Petron, B. Potocki, S. Svirsky, and P. Tyler. 1996. Special report of the Massachusetts Weightof-Evidence Workgroup: A weight-of-evidence approach for evaluating ecological risks. *Human and Ecological Risk Assessment* 2:277-304.
- Mitro, M.G. and A.V. Zale. 2000. Predicting fish abundance using single pass removal
 sampling. *Canadian Journal of Fisheries and Aquatic Sciences*. 57: 951-961.
- 33 Monosson, E. 1999. Reproductive, developmental and immunotoxic effects of PCBs in fish: a
- 34 summary of laboratory and field studies. Prepared for Damage Assessment Center, National
- 35 Oceanographic and Atmospheric Administration, Silver Spring, Maryland. NOAA Contract 50-
- 36 DSNC-7-90032. March 1999.

- 1 Oliver, B.G. and A.J. Niimi. 1985. Bioconcentration factors of some halogenated organics for 2 rainbow trout: limitations in their use for prediction of environmental residues. *Environ. Sci.*
- 3 *Technol*. 19:842-849.
- Papoulias, D. 2003a. Personal Communication (e-mail to M. Ptashynski, EVS Consultants,
 North Vancouver, BC, regarding interpretation of survival and pathology data). Fisheries
 Biologist, U.S. Geological Survey. Columbia Environmental Research Center. Columbia
 Missouri. June 16, 2003.
- Papoulias, D. 2003b. Personal Communication (e-mail to M. Ptashynski, EVS Consultants,
 North Vancouver, BC, regarding excerpt from final report describing statistical analyses for
 Phase II Studies). Fisheries Biologist, U.S. Geological Survey. Columbia Environmental
 Research Center. Columbia Missouri. June 18, 2003.
- Papoulias, D. 2003c. Personal Communication (e-mail to M. Ptashynski, EVS Consultants,
 North Vancouver, BC, regarding mortality in largemouth bass control fish). Fisheries Biologist,
 U.S. Geological Survey. Columbia Environmental Research Center. Columbia Missouri. June
 16, 2003.
- 16 PSWQAT (Puget Sound Water Quality Action Team). 2000. 2000 Puget Sound Update: Seventh
- 17 Report of the Puget Sound Ambient Monitoring Program. Puget Sound Water Quality Action
- 18 Team. Olympia, Washington.
- Reichert, W.L., B. French, T. Hom, H.R. Sanborn, J.E. Stein. 1996. Chronic exposure to
 sediment-associated polycyclic aromatic compounds: toxicokinetics of hepatic DNA adducts and
 biliary fluorescent aromatic compounds in English Sole. *Marine Environmental Research*42(1):279.
- Ricker, W.E. 1975. Computation and Interpretation of Biological Statistics of Fish Populations.
 Bulletin of the Fisheries Research Board of Canada. 191. 382 pp.
- 25 R2 (R2 Resource Consultants Inc.). 2002. Evaluation of Largemouth Bass Habitat, Population
- Structure, and Reproduction in the Upper Housatonic River, Massachusetts. Report prepared for
 General Electric Company.
- Smith, S.B. and J.F. Coles. 1997. Endocrine Biomarkers, Organochlorine Pesticides, and
 Congener Specific Polychlorinated Biphenyls (PCBs) in Largemouth Bass (Micropterus
 salmoides) from Woods Pond, Housatonic River, Massachusetts, September 1994 and May
 1995. U.S. Geological Survey Administrative Report. Prepared in cooperation with the U.S.
 Environmental Protection Agency. Reston, VA.
- Spies, R.B., D.W. Rice, Jr. and J.W. Felton. 1988. The effects of organic contaminants on
 reproduction of starry flounder, *Platichthys stellatus* (Pallas) in San Francisco Bay. Part I.
 Hepatic contamination and mixed-function oxidase (MFO) activity during the reproductive
 season. *Marine Biology* 98:181-189.
- 37 Spies, R.B. and D.W. Rice, Jr. 1988. The effects of organic contaminants on reproduction of 38 starry flounder, *Platichthys stellatus* (Pallas) in San Francisco Bay. Part II. Reproductive success

- of fish captured in San Francisco Bay and spawned in the laboratory. *Marine Biology* 98:191 202.
- 3 Stewart Laboratories, Inc. 1982. *Housatonic River Study 1980 and 1982*, Volumes I and II.
- 4 Tillitt, D. 2002. Personal communication (e-mail to Alice Shelly, TerraStat Consulting Group, 5 Everett, Washington regarding extrapolation of contaminant concentration from egg to ovary and
- 6 ovary to whole body). Columbia Environmental Research Center, USGS, Columbia, Missouri 7 July 31.
- 8 Tillitt, D. 2003. Personal communication (conference call with EVS Environmental Consultants,
- 9 USGS study team members, Weston Solutions, and US EPA regarding the Phase II studies).
- 10 USGS, Columbia Environmental Research Center, Columbia, Missouri. June 11, 2003.
- 11 Tillett, D., D. Papoulias, and D.R. Buckler. 2001. Fish Reproductive Health Assessment in PCB
- 12 Contaminated Regions of the Housatonic River, Massachusetts, USA: Investigations of Causal
- 13 Linkages Between PCBs and Fish Health. Prepared for U.S. Fish and Wildlife Service, Concord,
- 14 New Hampshire and U.S. Environmental Protection Agency, Boston, Massachusetts.
- 15 Tillett, D., D. Papoulias, and D.R. Buckler. 2002. Fish Reproductive Health Assessment in PCB
- 16 Contaminated Regions of the Housatonic River, Massachusetts, USA: Investigations of Causal
- 17 Linkages Between PCBs and Fish Health. Interim Report of Phase II Studies. Prepared for U.S.
- 18 Fish and Wildlife Service, Concord, New Hampshire and U.S. Environmental Protection
- 19 Agency, Boston, Massachusetts.
- 20 U.S. Army Corps of Engineers (USACE). 1988. Environmental effects of dredging technical 21 notes: relationship between PCB tissue residues and reproductive success of fathead minnows.
- U.S. Army Corps of Engineers, U.S. Army Engineer Waterways Experiment Station, EEDP-01 13.9 pp.
- Van den Berg, M., et al. 1998. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for
 humans and wildlife. *Environmental Health Perspectives*. 106(12): 775-792.
- Walker, M.K. and R.E. Peterson. 1991. Potencies of polychlorinated dibenzo-*p*-dioxin,
 dibenzofuran, and biphenyl congeners, relative to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, for
 producing early life stage mortality in rainbow trout (*Oncorhynchus mykiss*). *Aquat. Toxicol.*21:219-238.
- Walker, M.K., P.M. Cook, A.R. Batterman, B.C. Butterworth, C. Berini, J.J. Libal, L.C.
 Hufnagle and R.E. Peterson. 1994. Translocation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin from
 adult female lake trout (*Salvelinus namaycush*) to oocytes: effects on early life stage
 development and sac fry survival. *Can. J. Fish. Aquat. Sci.* 51:1410-1419.
- WESTON (Roy F. Weston, Inc). 2000a. Supplemental Investigation Work Plan. Prepared for
 U.S. Army Corps of Engineers and U.S. Environmental Protection Agency.
- 36 WESTON (Roy F. Weston, Inc). 2000b. *Quality Assurance Project Plan (QAPP)*. Prepared for 37 U.S. Army Corps of Engineers and U.S. Environmental Protection Agency.

- 1 Woodlot Alternatives. 2002. Fish Biomass Estimate for Housatonic River Primary Study Area. Prepared for U.S. Army Corps of Engineers. DCN: GE-061202-ABBF. 2
- 3 Zabel, E.W., P.M. Cook and R.E. Peterson. 1995a. Toxic equivalency factors of polychlorinated 4 dibenzo-p-dioxin, dibenzofuran and biphenyl congeners based on early life stage mortality in 5
- rainbow trout (Oncorhynchus mykiss). Aquat. Toxicol. 31:315-328.
- 6 Zabel, E.W., P.M. Cook and R.E. Peterson. 1995b. Potency of 3,3',4,4',5-pentachlorobiphenyl 7 (PCB-126), alone and in combination with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), to 8 produce lake trout early life-stage mortality. Environ. Toxicol. Chem. 14(12): 2175-2179.
- 9 Zippin, C. 1958. The removal method of population estimation. Journal of Wildlife Management 10 22:82-90.

1 6. WILDLIFE ASSESSMENT HIGHLIGHTS

2		Highlights
3 4 5		 Conceptual model for wildlife indicates diet is the major route of exposure to tPCBs and TEQ. Therefore, wildlife exposure modeling approach focuses only on dietary exposure.
6 7		 Probabilistic methods used to propagate uncertainty through wildlife exposure model.
8 9		 Selection of dietary concentration variables required consideration of spatial and temporal averaging of exposure.
10 11 12		 Options available to characterize exposure-effects relationships for tPCBs and TEQ include developing dose-response curves, NOAELs or LOAELs, field- based thresholds, or threshold ranges.
13		 Weight-of-evidence approach was used to characterize risks.
14		
15	6.1	OVERVIEW
16	The pu	urpose of this section is to describe the general approach and methods used to estimate
17	risks c	of contaminants of concern (COCs) to wildlife. Unlike the aquatic endpoints, the same
18	genera	al approach was appropriate for the majority of the wildlife endpoints. The endpoints for
19	which	this discussion applies include:
20		Insoctivarous Pirds (Section 7)
20 21		 Piscivorous Birds (Section 7) Piscivorous Birds (Section 8)
22		 Piscivorous Mammals (Section 9)
23		 Omnivorous and Carnivorous Mammals (Section 10)
24		 Threatened and Endangered Species (Section 11)
25		
26	An ove	erview of the following topics is included:
27		 Selection of COCs for wildlife
28		 Development of wildlife conceptual model
29		 Approach to wildlife exposure modeling
30		 Spatial and temporal averaging of exposure
31		 Probabilistic methods for propagating uncertainty
32		 Approach for effects assessment
22		

- 33 Approach for risk characterization
- 34

1 This section provides a comprehensive overview of the approaches used in modeling risk for 2 wildlife to reduce repetition in each of the subsequent sections, but does not provide the technical 3 details specific to each endpoint. The specific details are discussed in Appendix C and the 4 wildlife risk assessment appendices (Appendices G to K).

5	Representative Wildlife Species
6	Insectivorous Birds – Tree Swallow and American Robin
7	Piscivorous Birds – Belted Kingfisher and Osprey
8	Piscivorous Mammals – Mink and River Otter
9	Omnivorous and Carnivorous Mammals – Red Fox and Short-Tailed Shrew
10 11	Threatened and Endangered Species – Bald Eagle, American Bittern, and Small- Footed Myotis

12

13 Based on the results of the conservative, deterministic Pre-ERA (Section 2.4, Appendix B), the 14 contaminants of potential concern (COPCs) for wildlife include total polychlorinated biphenyls 15 (tPCBs), dioxins/furans, and several organochlorine pesticides. These pesticides were 16 subsequently screened out of the ERA because, in general, they were detected at low frequencies 17 and low concentrations, and are not considered to be from site-related sources. Further, the 18 actual concentrations of organochlorine pesticides are likely much lower than the measured 19 values due to laboratory interference. Dioxin, furan, and coplanar PCB congeners were 20 considered in the risk assessment by calculating 2,3,7,8-TCDD toxic equivalence (TEQ). 21 Methods for calculating TEQ concentrations are described in Section 6.4.

The conceptual model for the wildlife assessments is shown in Figure 6.1-1. The conceptual model outlines the ecosystem processes that qualitatively link stressor releases and primary, secondary, and tertiary exposure pathways to ecological receptors. Thus, the conceptual model provides a visual representation of the potential risk pathways to wildlife from COCs. Each representative species has a species-specific conceptual model. These are presented in Sections 7 to 11 and Appendices G to K.

The wildlife risk assessments have three main components: the exposure assessment, the effects
assessment, and risk characterization. The process used in each of these components is described
below.



1

Figure 6.1-1 Conceptual Model for the Assessment of Risks from tPCBs and TEQ to Wildlife in the Housatonic River Primary Study Area

4 Figure 6.1-2 depicts the framework for the exposure assessment.

5 During the exposure assessment, exposure of wildlife to tPCBs and TEQ in the Housatonic River 6 Primary Study Area (PSA) was determined, beginning with a description of the exposure model. 7 Input variables for the exposure model were established using life history information for the 8 representative species, and concentrations of tPCBs and TEQ in prey collected in the PSA. For 9 those input variables that were uncertain, variable, or both, distributions were used rather than 10 point estimates. Monte Carlo and probability bounds analyses were then performed to propagate 11 input variable uncertainties through the exposure model for each COC.

EXPOSURE



Figure 6.1-2 Framework Used to Model Exposure of Wildlife Species to Contaminants of Concern (COCs) in the Housatonic River PSA

Figure 6.1-3 shows the approach used in conducting the effects assessment. The effects assessment includes a comprehensive review of the literature on the effects of tPCBs and TEQ on survival, growth, and reproduction of representative wildlife species or reasonable surrogate species. Each of the studies was evaluated using acceptability criteria established for this ERA (see box below). Appropriate studies were then selected and used to derive the effects metric.

9	Acceptability Criteria for Wildlife Studies
10	Were appropriate controls used?
11	Were appropriate statistics applied?
12	Were acceptable methods (e.g., laboratory methods) used?
13	Was there an appropriate range of exposure doses?
14	Was the experimental effect attributable to the COC?

1



1

Figure 6.1-3 Approach Used to Model Effects of Contaminants of Concern (COCs) to Representative Species in the Housatonic River PSA

The final component of the wildlife risk assessments is the characterization of risk combining the results of the exposure and effects assessments, and other available lines of evidence (e.g., whole media or in situ studies, field surveys). Figure 6.1-4 presents the general approach used in the risk characterization process.

8

RISK CHARACTERIZATION



1

Figure 6.1-4 Approach Used to Characterize the Risks from Contaminants of Concern (COCs) to Representative Species in the Housatonic River PSA

5 In the risk characterization, the likelihood and magnitude of adverse effects occurring as a result 6 of exposure of the representative wildlife species to tPCBs and TEQ was evaluated. A weight-7 of-evidence approach (WOE) was used to make a risk determination for each representative 8 species. Several lines of evidence were available to characterize risks to wildlife from exposure 9 to tPCBs and TEQ, however, not all lines of evidence were available for each species:

- Modeled Exposure and Effects This line of evidence determines the extent to which the concentrations of tPCBs and TEQ ingested in the diet will cause adverse effects to the survival, reproduction, or growth of wildlife. Estimated exposures were compared to results of toxicological studies reported in the literature to determine if the representative wildlife species are exposed to tPCBs and TEQ at levels likely to induce adverse effects.
- Field Surveys When available, this line of evidence was used to determine the relationship between the concentrations of tPCBs and TEQ and the abundance of wildlife in the Housatonic River floodplain.
- Whole Media or In Situ Studies When available, this line of evidence was used to examine the relationship between tPCB and TEQ concentrations at specific sites or in whole media from the PSA and effects observed in wildlife species. This line of evidence is considered analogous to the site-specific toxicity testing line of evidence used in the assessments for aquatic receptors.

Each wildlife risk characterization includes a discussion of sources of uncertainty in the assessment of risks of COCs to wildlife, and the conclusions of the risk characterization.

17 6.2 WILDLIFE EXPOSURE MODEL

The approach for conducting the modeled exposure assessment for wildlife relies on the use of total daily intake models. The primary focus of the model is on ingestion of prey. The dietary exposure pathway is by far the most important exposure pathway for bioaccumulative substances such as tPCBs and TCDD and equivalentce (TEQ) (Moore et al. 1997, 1999). Thus, the wildlife exposure assessments do not include environmental media in the exposure model calculations. The wildlife exposure model follows the general form:

24
$$TDI = FIR \bullet FT \bullet \sum_{i=1}^{n} C_i \bullet P_i$$
 (Eq. 1)

25 where

- 26 TDI = Total daily intake (mg/kg bw/d tPCBs, ng/kg bw/d TEQ)
- 27 P_i = Proportion of i^{th} food item in the diet (unitless)
- 28 FIR = Normalized food intake rate (kg/kg bw/d)
- 29 C_i = Concentration in i^{th} food item (mg/kg tPCBs, ng/kg TEQ)

1 FT = Fraction of time in the contaminated area (unitless)

This general exposure model was customized accordingly for each representative species to reflect feeding habits, foraging range, habitat preferences, and life history. Extensive literature searches were conducted and data collected to determine the appropriate model inputs. Each of these inputs is briefly discussed below.

6 6.2.1 Food Intake Rate (FIR)

7 Data on food intake rate (FIR) are only available for a few species, primarily due to the 8 difficulties in measuring intake for free-ranging wildlife. This assessment does not use measured 9 food intake rates determined using captive animals, because such animals do not expend energy 10 foraging for food and water, avoiding predators, defending territories, etc. (EPA 1993). Thus, 11 food intake rates estimated for captive animals considerably underestimate expected food intake 12 rates for free-ranging animals. In this assessment, allometric equations developed from 13 measurements of free metabolic rate (FMR) in free-ranging animals (see text box below) were used to estimate food intake rate for each representative wildlife species. Food intake rate is 14 15 derived from *FMR* using the following equation:

16
$$FIR(g/day) = \frac{FMR}{\sum_{i=1}^{n} (AE_i \, x \, GE_i)}$$
(Eq. 2)

17 where AE_i is the assimilation efficiency of the *i*th food item (unitless) and GE_i is the gross energy 18 of the *i*th food item (kcal/g). Where measured food intake rates are available for free-ranging 19 animals for a representative species, the measured food intake rates are compared to the 20 corresponding food intake rate derived using the allometric modeling approach. Such 21 comparisons can be found in the wildlife sections and appendices.

1 Example Wildlife Free Metabolic Rate Equations 2 Birds $FMR(kJ/day) = 10.5 \cdot BW(g)^{0.681}$ (Coraciiformes) 3 4 Mammals $FMR(kJ/dav) = \log 0.221 \cdot BW(g)^{0.869}$ (Carnivores) 5 6 where 7 BW body weight (g). 8 (Nagy et al. 1999)

9 **6.2.2 Body Weight (BW)**

Body weights for each of the representative wildlife species were determined through data from the literature or data collected in the PSA of the Housatonic River. Data were combined from each relevant, acceptable study, and the mean and standard deviations calculated. Body weight is assumed to be a normally distributed parameter. The uncertainty associated with the variable is generally due to natural variability, rather than a lack of knowledge or data (i.e., body weight is easy to measure and data are available for each of the representative species).

16 6.2.3 **Proportions of Dietary Items**

17 Extensive literature searches were conducted to locate data and information on the dietary 18 preferences of the wildlife species assessed. The information in the literature on dietary 19 preferences was evaluated to determine relevance to representative species in the PSA and the 20 timing of their exposures to COCs. Some wildlife species have dietary preferences that can 21 include a large number of prey items. Therefore, only dietary items that comprise at least 10% 22 of the total diet of each species were included in the exposure model. In these cases, dietary 23 items for prey items comprising >10% of the diet were adjusted resulting in the sum of all 24 dietary components equaling 100%. Because diets vary between locations and individuals and 25 are also uncertain because of the limited data available for some species, distributions were used 26 for dietary variables.

1 6.3 SPATIAL AND TEMPORAL AVERAGING

2 Concentrations of COCs vary spatially and temporally in prey. The representative wildlife 3 species forage over distances ranging from tens of meters to greater than 10 km. Thus, 4 individuals tend to integrate spatial variation in the tissue concentrations of their prev over time. 5 Therefore, estimates of the central tendency (i.e., arithmetic means) are used in the exposure 6 model as an expression of the spatial and temporal averaging of concentrations of COCs in prey 7 tissues (EPA 1999). In the Monte Carlo analysis, it was assumed that the spatially and 8 temporally averaged exposure estimate did not vary between individuals foraging in the same 9 area. Thus, the point estimate of centrality was the minimum of:

10 11 1. The 95% upper confidence limit calculated using the Land H-statistic (assuming data are lognormally distributed), or

12 2. The maximum concentration measured.

In the probability bounds analyses, however, the uncertainty regarding the arithmetic mean wasaccounted for with a different procedure.

15 The procedure for the probability bounds analysis generally involved using the Land H-statistic 16 to estimate the lower and upper 95% confidence limits on the mean (Gilbert 1987), and then 17 using these lower and upper confidence limits to derive bounds on all possible distributions that 18 exist within this range. This approach results in an expression of the uncertainty about the true 19 value of the arithmetic mean that arises due to the small sample size. In cases where the 95% 20 upper confidence limit could not be estimated, or exceeded the maximum measured 21 concentration, other techniques were used to derive the bounds on the mean (see Appendices G 22 to K). Appendix C.5 describes the procedures for parameterizing prey concentration variables in 23 more detail.

EPA (1992) states that because of the uncertainty associated with estimating the true average concentration for a site, the 95% upper confidence limit (UCL) of the arithmetic mean should be used for this variable." For lognormal data, EPA (1992) recommends the Land method using the H-statistic. Several authors (e.g., Ott 1995; Seiler and Alvarez 1996; Hattis and Burmaster 1994) have argued that concentrations of contaminants in environmental media tend to be lognormally distributed and that this may be expected because of mechanistic reasons. Current EPA guidance

1 (EPA 1997; also see Haimes et al. 1994) states that distributions should be chosen for input 2 variables on the basis of mechanistic or theoretical reasons, if possible, because such 3 distributions have the highest degree of confidence. As a result, concentrations of contaminants 4 in prey were assumed to be lognormally distributed in this ERA, and hence the Land H-statistic was used to estimate the 95% UCL. To determine the reasonableness of this assumption, the 5 6 Shapiro-Wilk test was used to test for lognormality. Over two-thirds of the data sets used in the wildlife assessments passed the test for lognormality (i.e., p > 0.05), which supports the 7 8 assumption of lognormality for concentrations of contaminants. That said, it is recognized that 9 the Land method can produce high values for the UCL, particularly when data are not 10 lognormally distributed, sample size is small, or variation is high (Singh et al. 1997; Schultz and 11 Griffin 1999). EPA's (1992) guidance recognized this problem and recommended that the 12 maximum detected concentration be used when the calculated UCL exceeds this value. This 13 guidance was followed in this assessment.

14 6.4 TOXIC EQUIVALENCE (TEQ)

Some PCB congeners belong to a large class of chemicals called planar chlorinated hydrocarbons (PCH) that are regularly detected in the environment. The PCHs also include polychlorinated dibenzo-p-dioxins (PCDDs), and polychlorinated dibenzo-furans (PCDFs). PCHs have a common structural relationship that includes lateral halogenation (i.e., the addition of a halogen such as fluorine or chlorine to a compound), and the ability to assume a planar conformation (Figure 6.4-1).



21



1 This structure is important because it leads to a common mechanism of action in many animal 2 species that involves binding to the aryl hydrocarbon (Ah) receptor and elicitation of an Ah-3 receptor-mediated biochemical and toxic response (Van den Berg et al. 1998; Newsted et al. 4 1995; Safe 1994). Toxic responses include:

5	• Lethality.
6	 Hepatic lesions.
7	 Immunotoxicity.
8	 Tumor promotion.
9	 Adverse effects on reproduction.
10 11	 Induction of drug-metabolizing enzymes (Van den Berg et al. 1998; N 1995).

12 The planar structure determines the ability of the chemical to bind with the Ah receptor (Birnbaum and Devito 1995; Newsted et al. 1995). The Ah receptor facilitates the translocation 13 14 of PCHs into the nucleus of affected cells and the binding of the PCH-Ah receptor complex to sites on the DNA (Newsted et al. 1995). Environmental degradation of PCH congeners varies 15 16 due to their unique physical/chemical properties (Cogliano 1998) and thus there can be 17 substantial differences between the congeners detected in environmental samples and the congener makeup of the original product (Cogliano 1998; Van den Berg et al. 1998). The 18 19 congeners also have different toxic potencies. To address these issues and effectively estimate 20 the relative toxicity of these mixtures, various systems have been created involving the 21 development and use of toxic equivalency factors (TEFs) to derive toxic equivalence (TEQ) 22 (Van den Berg et al. 1998; Safe 1990, 1994; EPA 1987, 1989, 1991; Kennedy 1996; NATO 23 1988a, 1988b; Ahlborg et al. 1994). The TEQ approach is based on the in vivo and in vitro toxicity of each of the PCH congeners in relation to 2,3,7,8-tetrachlorodibenzo-p-dioxin 24 25 (TCDD). TCDD is considered to be the most toxic member of the PCH class of chemicals (Van den Berg et al. 1998; Birnbaum and DeVito 1995; Safe 1994). For this ERA, the TEFs proposed 26 27 by Van den Berg et al. (1998) (also referred to as the World Health Organization or WHO TEFs) 28 have been adopted (Table 6.4-1). These TEF values were developed for compounds that:

29

Show a structural relationship to PCDDs and PCDFs.

ewsted et al.

Table 6.4-1

World Health Organization Toxic Equivalency Factors (TEFs) for TCDD and Equivalents (Van den Berg et al. 1998)

	Mammals	Fish	Birds		
Congener	TEF				
PCB-77	0.0001	0.0001	0.05		
PCB-81	0.0001	0.0005	0.1		
PCB-126	0.1	0.005	0.1		
PCB-169	0.01	0.00005	0.001		
PCB-105	0.0001	< 0.000005*	0.0001		
PCB-114	0.0005	$< 0.000005^{*}$	0.0001		
PCB-118	0.0001	< 0.000005*	0.00001		
PCB-123	0.0001	< 0.000005*	0.00001		
PCB-156	0.0005	$< 0.000005^{*}$	0.0001		
PCB-157	0.0005	< 0.000005*	0.0001		
PCB-167	0.00001	< 0.000005*	0.00001		
PCB-189	0.0001	< 0.000005*	0.00001		
1,2,3,4,6,7,8-HpCDD	0.01	0.001	< 0.001*		
1,2,3,4,6,7,8-HpCDF	0.01	0.01	0.01		
1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.01		
1,2,3,4,7,8-HxCDD	0.1	0.5	0.05		
1,2,3,4,7,8-HxCDF	0.1	0.1	0.1		
1,2,3,6,7,8-HxCDD	0.1	0.01	0.01		
1,2,3,6,7,8-HxCDF	0.1	0.1	0.1		
1,2,3,7,8,9-HxCDD	0.1	0.01	0.1		
1,2,3,7,8,9-HxCDF	0.1	0.1	0.1		
1,2,3,7,8-PeCDD	1	1	1		
1,2,3,7,8-PeCDF	0.05	0.05	0.1		
2,3,4,6,7,8-HxCDF	0.1	0.1	0.1		
2,3,4,7,8-PeCDF	0.5	0.5	1		
2,3,7,8-TCDD	1	1	1		
2,3,7,8-TCDF	0.1	0.05	1		
OCDD	0.0001	< 0.0001*	0.0001		
OCDF	0.0001	< 0.001*	0.0001		

^{*} Values that are "less than" should be considered to be the upper limit for use in any TEQ calculation.

6	current scientific research (Dyke and Stratford 2002). They have been accepted and applied in
7	numerous jurisdictions worldwide (Dyke and Stratford 2002). Assumptions are made when
8	using the TEF approach, including:
9 10	 PCH congeners are Ah-receptor antagonists and their toxicological potency is mediated by their binding affinity.
11 12	 No interaction occurs between the congeners and thus, the sum of the individual congener effects accounts for the potency of the PCH mixture.
13	The overall effect of these assumptions is a potency estimate or toxic equivalence (TEQ) value.
14	To generate a TEQ the following equation (Equation 1- modified from Van den Berg et al. 1998)
15	is used:
16	$TEQ = \sum_{n=1}^{6} [PCDD_n \ x \ TEF_n] + \sum_{p=1}^{10} [PCDF_p \ x \ TEF_p] + \sum_{q=1}^{12} [PCB_q \ x \ TEF_q] $ (Eq. 3)
17	where
18	TEQ = Toxic equivalence
19	$PCDD_n$ = Polychlorinated dibenzo-p-dioxin congener concentration
20	$PCDF_p$ = Polychlorinated dibenzo-furan congener concentration
21	PCB_q = Polychlorinated biphenyl congener concentration
22 23	$TEF_{n,p,q}$ = Toxic equivalency factor for appropriate individual PCDD/PCDF and PCB congeners, respectively
24	Two circumstances often arise when calculating a TEQ value:
25 26 27	 Congener concentrations may be below the detection limit (i.e., non-detects), and Some congeners may not be resolved due to co-elution during analysis.

Bind to the Ah receptor.

Elicit an Ah-receptor-mediated biochemical and toxic response.

Birnbaum and DeVito 1995).

Are persistent and accumulate in the food chain (Van den Berg et al. 1998;

The WHO TEFs are the most recent estimates of 2,3,7,8-TCDD equivalence and are based on

3 4

5

1

2

The approach used to address each of these circumstances in the ERA is discussed in the
 following sections.

3 6.4.1 Non-Detects

4 Congeners detected at or below the detection limit (DL) were included in the TEQ calculations 5 by investigating three options: first, setting the value for the congener equal to zero (0), setting it 6 to half the DL, and, finally, setting it equal to the DL (Appendix C.2). A comparison of the 7 results of this bounding analysis provides a description of the uncertainty surrounding the TEQ 8 value due to concentrations of one or more congeners being below the detection limit. This 9 approach is also useful for determining the relative influence of individual non-detected 10 congeners on the estimated TEQ value. Concentrations of tPCBs in prev in the PSA were all 11 above the detection limit; therefore, there is no non-detect issue for tPCBs. However, treatment 12 of non-detects remains a concern for the TEQ congeners.

13 6.4.2 Congener Co-Elution

14 The development of a TEQ using the WHO approach requires the concentrations for each of 29 15 unique congeners (12 PCB and 17 PCDD/PCDF congeners). During analysis of many of the 16 tissue samples collected for the risk assessment, 2 of the 29 TEQ congeners (i.e., PCB-123 and 17 PCB-157) co-eluted with other congeners. PCB-123 co-eluted with PCB-149 (PCB-123/149) 18 and PCB-157 co-eluted with PCB-201 and PCB-173 (PCB-157/201/173). Assuming that the 19 concentration of the congener PCB-123 is equal to the doublet concentration and that the 20 concentration of PCB-157 is equal to the triplet concentration would likely overestimate the TEQ 21 concentration. Conversely, assuming that concentrations of the two congeners (i.e., PCB-123 22 and PCB-157) were equal to zero would likely underestimate the TEQ concentration. These two 23 approaches are useful to estimate TEQ bounds, but say little about the relative probabilities of 24 values between the bounds.

Where possible, independent data sets were located for tissue types where analytical results were available for the co-eluted congeners in the Housatonic River database. Priority was given to data sets with tissue samples taken from the Housatonic River to minimize uncertainty associated with congener metabolism and environmental degradation. Only one appropriate data set was

located, for fish tissue, in the Housatonic River that had unique results for each of the congeners 1 2 in the doublet and triplet. Ratios of the congeners found in the independent data sets were 3 generated and applied to the co-eluted congener data. The co-elution ratio was then multiplied 4 by the reported result for the doublet and triplet concentrations to estimate the PCB-123 and 5 PCB-157 concentrations for fish tissue samples. Uncertainty associated with the method for 6 treating the co-eluted congeners includes interlaboratory variance due to different analytical 7 methods, laboratory conditions, and analyst experience and expertise. The calculated ratios also 8 do not account for differences between species found in the tissue database. A full description of 9 the approach to developing the co-elution ratios from the independent data sets is provided in 10 Appendix C.10.

11	Co-Elution Ratios
12	PCB-123/149 – 0.003/0.997
13	PCB-157/201/173 – 0.195/0.632/0.174

14

6.4.3 Summary of Decision Criteria for Estimating Exposure Point Concentrations

To deal with the uncertainty arising from co-elution or non-detect congeners when estimating exposure point concentrations (EPCs) for use in the exposure analyses, the following decision criteria (Figure 6.4-2) were developed (also see Appendix C.2):

20 Concentrations of COCs in samples where the concentration was below the 21 **detection limit** – To determine whether this source of uncertainty was important, 22 arithmetic means were calculated for tissue concentrations assuming a concentration 23 of zero for non-detected COCs (ND = 0), and assuming a concentration equal to the 24 detection limit (ND = DL). If the ratio of the ND = DL mean to the ND = 0 mean 25 was less than 1.3, this source of uncertainty was deemed unimportant. In these cases, 26 exposure calculations were done assuming that concentrations of non-detected COCs were equal to half the detection limit (ND = $\frac{1}{2}$ DL).¹ In cases where the ratio 27 28

¹ This decision criterion supplements the procedures described in Appendix C.2.



UCL = Lower of the 95% UCL from the Land H-statistic or the dataset max LCL = Higher of the 95% LCL from the Land H-statistic or the dataset min

1 2 3

Figure 6.4-2 Decision Tree for Determining Appropriate Treatment of Data with Non-Detects and Co-Elution

exceeded 1.3, the source of uncertainty was considered sufficiently important to incorporate in the exposure analysis. In the Monte Carlo analysis, for samples with COC concentrations below the detection limit three estimates of the EPC (i.e., estimates assuming ND = 0, ND = $\frac{1}{2}$ DL, ND = DL) were used as parameters in a triangular distribution (i.e., minimum, best estimate, maximum). In the probability bounds analysis, the distribution-free range was the range spanning the LCL calculated assuming ND = 0 for the lower limit, and the UCL calculated assuming ND = DL for the upper limit.

9 Concentrations of TEO in tissue samples (other than fish) with co-eluted 10 congeners – In some tissue samples, two PCB congeners required in the TEQ calculation (PCB-157 and PCB-123) co-eluted with other congeners. As a result, the 11 concentrations of the triplet PCB-201/157/173 and the doublet PCB-149/123 are 12 13 known, but not the concentrations of PCB-157 and PCB-123. This source of 14 uncertainty was accounted for in the exposure calculations using an approach similar to that used to account for uncertainty stemming from non-detected COCs. For each 15 16 tissue concentration variable, a ratio was calculated for mean TEQ concentration 17 assuming that the concentration of PCB-157 and PCB-123 was zero, and the mean TEQ concentration assuming that the concentrations of these congeners were equal to 18 19 the triplet and doublet concentrations, respectively. If the ratio was less than 1.3, this 20 source of uncertainty was deemed unimportant. In these cases, exposure calculations were done assuming that concentrations of PCB-157 and PCB-123 were equal to the 21 22 triplet and doublet concentrations, respectively. In cases where the ratio exceeded 23 1.3, the source of uncertainty was considered sufficiently important to incorporate in 24 the exposure analysis. The procedures followed to accomplish this task were the same as used to deal with uncertainty due to non-detected COCs. 25

26 6.5 PROBABILISTIC RISK ASSESSMENT

27 6.5.1 Distribution Selection

28 Input distributions for the exposure analyses were generally assigned as follows: lognormal 29 distributions for variables that were right skewed with a lower bound of zero and no upper bound (e.g., amount of COC transferred from mother to offspring via egg tissue), beta distributions for 30 31 variables bounded by zero and one (e.g., proportion of a prey item in the diet), normal 32 distributions for variables that were symmetric and not bounded by one (e.g., body weight), and 33 point estimates for minor variables or variables with low coefficients of variation. In certain 34 situations (e.g., poor fit of data), other distributions were fit to the data or other approaches were 35 used. To quantify uncertainty, two approaches were used as described in Section 6.5.2, below.

36

1 2

3

4 5

6 7

8

6.5.2 Monte Carlo and Probability Bounds Analysis

2

3 4

5

6

7

/ 8

General Risk Assessment Approaches

Deterministic Methods – Methods in which all biological, chemical, physical, and environmental parameters are assumed to be constant and can be accurately specified.

Probabilistic Methods – Methods in which important biological, chemical, physical, and environmental parameters are assumed to vary or are uncertain and therefore, are specified using distribution of possible values.

9

10 Monte Carlo and probability bounds analysis are two uncertainty propagation techniques used in 11 the Housatonic River wildlife risk assessments. The use of probabilistic methods in risk analysis 12 is growing rapidly and EPA has produced guidance on how to conduct such analyses in Superfund and other programs (EPA 1997, 1999). The benefit of using probabilistic methods in 13 14 risk assessment is that they give the risk assessor the ability to fully characterize risk, rather than 15 providing a best estimate or a conservatively biased estimate of risk. For example, calculating a 16 mean risk (i.e., deterministic method) may exclude the potential for relatively rare, but serious, 17 extreme events (e.g., species extinction). This is generally undesirable, because although rare, 18 these events can occur and have significant impacts on individuals, communities, and 19 populations of species. By including the entire distribution for risk, all events are considered and 20 all of the data and information collected to characterize a situation are included. The remainder 21 of this section provides a short overview of Monte Carlo and probability bounds analysis as 22 applied in the wildlife risk assessments. Further technical detail on these methods can be found 23 in Appendix C.4.

24

25

26

27 28

29

30

Probabilistic Methods

Monte Carlo Analysis – A technique where parameter values are drawn at random from defined input probability distributions, combined according to a model equation, and the process repeated iteratively until a stable distribution of solutions results. It is most useful when input distributions are known precisely.

Probability Bounds Analysis – Separates uncertainty and variability to obtain bounds on the result that explicitly account for the uncertainty about the input distributions.

- 31
- 32 33

1 The primary goal of a Monte Carlo analysis in the risk assessment is to characterize 2 quantitatively, the uncertainty and variability in estimates of exposure and risk (EPA 1997). A 3 secondary goal is to identify key sources of variability and uncertainty and to quantify the 4 relative contribution of these sources to the overall range of wildlife exposure model results. 5 While Monte Carlo methods are appropriate for the determination of exposure risks when input 6 distributions are known precisely, they may not adequately represent the effects of uncertainty 7 about how to parameterize variability in the input distributions (Ferson 1996). In many 8 ecological risk assessments, the available data are limited and consequently the input 9 distributions used to calculate risks are uncertain. Probability bounds analysis is a tool for 10 separating variability and uncertainty to obtain bounds on the result that explicitly account for 11 uncertainty about the input distributions. As in Monte Carlo analysis, the overall slopes of the 12 bounds indicate how much variability exists in the system. The distance between the bounds, on 13 the other hand, is an indication of the uncertainty that exists due to lack of knowledge. An 14 example of exposure model outputs from Monte Carlo and probability bounds analyses is 15 presented in Figure 6.5-1.

16 The wildlife exposure models contain multiple variables, some of which may be correlated. The 17 assumption of independence can be inappropriate, because dependencies can affect the estimates 18 of exposure. If correlations are not accounted for, the variance and the tails of the exposure 19 distribution may be poorly estimated. The wildlife assessments use several approaches to 20 address correlations between variables. These approaches include simulation of observed 21 correlations, assumption of perfect covariance (e.g., when the diet consists of two prey items, the 22 proportion of one item in the diet is equal to one minus the other item), or no assumptions at all 23 about dependencies (e.g., all possible relationships between two variables can occur). The 24 specific approach used depends on the type of data and the application. In cases where 25 independence of variables seemed intuitively obvious (e.g., COC concentration in the prev item 26 and proportion of that item in the diet), independence was assumed.



Figure 6.5-1 Example Exposure Distribution from Monte Carlo and Probability
 Bounds Analyses (TDI = total daily intake of tPCBs)

5

2

1

6 6.6 EFFECTS ASSESSMENT

7 Effects data can be characterized and summarized in a variety of ways ranging from benchmarks 8 designed to be protective of most or all species to dose-response curves for the representative 9 species of interest. In this ERA, effects characterization preferentially relied on concentration-10 or dose-response curves, but defaulted to benchmarks or other estimates of effect (e.g., no 11 observed adverse effect level [NOAEL], or lowest observed adverse effect level [LOAEL]) when 12 insufficient data were available to derive dose-response curves. Effects associated with growth, 13 survival, and reproduction were generally the preferred measures of effect as they most closely 14 relate to the assessment endpoints for wildlife. This section provides an overview of the 15 procedures used for characterizing effects information and describes the decision criteria for 16 choosing among them for each receptor-COC combination.

Figure 6.6-1 displays the hierarchy of decision criteria used to characterize effects for each receptor-COC combination. In all cases, the units of the effects metrics were consistent with the

- 1 units of the exposure metrics. To the extent possible, effects metrics were based on long-term
- 2 studies to match expected exposure durations.
- 3 The remainder of this section provides details on how the effects metrics were derived from the
- 4 decision tree.



5 6 7

Figure 6.6-1 Decision Criteria Used to Characterize Effects for Each Wildlife Receptor-COC Combination

8 6.6.1 Dose-Response Relationships Using the Generalized Linear Model 9 Framework

Most probabilistic risk assessments previously conducted estimated the probability that exposure exceeded a specified no-observed-effects or lowest-observed-effects dose. An alternative approach is to estimate the probabilities of effects of varying magnitude. To do this, a concentration- or dose-response model is required. Generally, five or more treatments are required to develop concentration- or dose-response relationships, either from a single study or

from several studies that used a similar methodology. The Generalized Linear Model (GLiM) 1 framework described by Kerr and Meador (1996) and Bailer and Oris (1997) is a useful 2 3 framework for deriving these relationships. The framework involves using link functions to 4 transform effects metrics (e.g., probit or logit link functions for quantal responses) and assigning 5 appropriate error distributions (e.g., binomial distribution for quantal responses). Linear 6 regression can then be conducted on the transformed data to derive the dose-response 7 relationship. Thus, the framework can be used for all available types of response variables 8 (Moore et al. 2000). By adding a quadratic term to the linear model, the framework can be 9 adapted to incorporate simulation at low doses. The GLiM framework was used to derive dose-10 response relationships in this ERA when five or more treatments were available from a single 11 study for the receptor of interest or a reasonable surrogate. In some cases, it was necessary to 12 convert concentration-response relationships to dose-response relationships by multiplying the 13 former by the food intake rate of the species (Moore et al. 1999).

14 Dose-response relationships are combined with the corresponding exposure distribution in risk 15 characterization to derive risk curves that characterize the relationship between probability and 16 magnitude of effect.

17 6.6.2 Hypothesis Testing to Determine LOAEL and NOAEL

Analysis of variance (ANOVA) is the most common method of estimating low-level toxic effects from chronic tests. There are several reasons for this, including the wide availability of software capable of performing ANOVA and related nonparametric tests, and the familiarity of regulators with the technique. Until recently, most toxicity-testing protocols specified experimental designs more suited to hypothesis-testing methods such as ANOVA than to regression-based approaches. However, hypothesis testing as an approach for estimating lowlevel toxic effects has some limitations, including:

- NOAELs and LOAELs are test doses that do not correspond with specified effects
 levels from one test to the next.
- 27
 2. Poor experimental design may mistakenly indicate that a contaminant is less toxic than it really is.

- 1 2
- 3. Most information available from the toxicity test is not used (Stephan and Rogers 1985; Pack 1993; Suter 1996).

As a result, hypothesis testing was not the preferred method for analysis of toxicity data in thisERA.

5 However, in many cases, toxicity studies with five or more treatment levels are not available for 6 the representative species of interest or for a reasonable surrogate for tPCBs and TEQ. In those 7 cases, the use of hypothesis testing was necessary to estimate the NOAEL and LOAEL. In many 8 toxicological studies, these endpoints were previously determined and reported. Such studies 9 were evaluated to determine that proper statistical procedures were followed. Where the data 10 could be obtained from the reports or directly from the authors, the data were re-analyzed. In 11 cases where a re-analysis was conducted, information regarding the minimal difference required 12 to give a significant result was reported (e.g., number of replicates, test variance, α , β , test dose 13 intervals). The percent effect associated with the LOAEL, relative to the control, was also 14 reported.

15 6.6.3 Field-Based Measures of Effect and Threshold Ranges

16 Field-based measures of effect were derived from monitoring or in situ toxicity tests conducted 17 on the representative species or a reasonable surrogate. There are several methods available for 18 deriving field-based measures of effect. For benthic invertebrates, chemistry and effect data 19 from surveys of sediment and biota in various locations have been combined to develop sediment 20 concentrations that are generally protective or, conversely, likely associated with adverse effects 21 (Long et al. 1995; MacDonald et al. 1996). Similar approaches can be used with wildlife 22 species. With in situ studies, if sufficient data were available, regression-based approaches (e.g., 23 GLiM models) could be used to link concentrations or doses with effects observed in the field.

When data are lacking on the toxicity of a particular COC to the representative species or a reasonable surrogate, threshold ranges were developed. In these cases, it is not known whether the representative species is sensitive or tolerant. Therefore, a threshold range was developed that spanned the concentrations (or doses) that would be protective of sensitive species to those that would be protective only for tolerant species. The assumption is that the threshold for the representative species lies between these two extremes. To derive a threshold range, the toxicity literature was reviewed to determine the most sensitive and the most tolerant species for which studies have been conducted. Thresholds were derived for both the most sensitive and the most tolerant species using methods similar to those used in the Pre-ERA (see Section 2.4 and Appendix B). The two resulting thresholds become the threshold range, which was then compared to the exposure assessment results in the risk characterization.

6 6.7 RISK CHARACTERIZATION

7 6.7.1 Risk Categorization

8 Whenever possible, risk should be expressed quantitatively (Wentsel et al. 1997). For example, 9 a risk could be expressed as a 10% probability of >25% mortality for a particular species. In this 10 ERA, quantitative expressions of risk were derived for each of the wildlife assessment endpoints 11 (Appendices G to K) to facilitate discussion and to simplify comparisons of risk between species, 12 COCs, and locations. The following criteria were used to categorize risks to wildlife as high, 13 intermediate, or low:

14	•	Scenarios with effects data for the representative species (or a reasonable surrogate):
15 16		 If the probability of 10% or greater effect (or of exceeding the NOAEL) was less than 20%, then the risk was categorized as low (Figure 6.7-1).
17 18		- If the probability of 20% or greater effect (or of exceeding the LOAEL) was greater than 50%, then the risk was categorized as high (Figure 6.7-1).
19		- All other outcomes were categorized as intermediate risk (Figure 6.7-1).
20 21	•	Scenarios with effects data for the representative threatened and endangered species (or a reasonable surrogate):
22 23		 If the probability of 10% or greater effect (or of exceeding the NOAEL) was less than 20%, then the risk was categorized as low.
24 25		 If the probability of 10% or greater effect (or of exceeding the NOAEL) was greater than 50%, then the risk was categorized as high.


1

Note: Risk curves passing below the filled circle symbol indicate low risk, while those passing above the open circle
 symbol indicate high risk.

5 Figure 6.7-1 Example Risk Curves Indicating Low, Intermediate, and High Risk 6 Categories

1	 All other outcomes were categorized as intermediate risk.
2 3	 Scenarios with threshold concentrations for the representative species (or a reasonable surrogate):
4 5	 If the probability of exceeding the threshold for the most sensitive species was less than 20%, the risk was categorized as low.
6 7	 If the probability of exceeding the threshold for the most tolerant species was greater than 20%, the risk was categorized as high.
8	 All other outcomes were categorized as intermediate risk.
9	Each categorization of risk was derived from the results of the Monte Carlo exposure analyses
10	(Figure 6.7-1). To capture the uncertainty about a risk categorization, the results from the
11	corresponding probability bounds analysis were compared to the above criteria to determine a
12	risk range (risk category using lower probability bound to risk category using upper probability
13	bound).

14 These risk categorization criteria were based on several considerations including:

- Efroymson and Suter (1999) and others (e.g., Pack 1993) suggested that reductions in 15 16 survival, growth, or reproduction of 20% or greater is indicative of significant effects 17 to wildlife. Thus, a better than even chance (i.e., >50%) of exceeding this effect level 18 was deemed to represent a high risk situation. However, because effects at or above 19 the 20% level possibly may not be ecologically significant, these categorizations 20 should be considered further in each situation. For example, a stressor causing a 20% decline in reproductive fecundity of brook trout was shown to lead to a general 21 22 lowering of risks of population decline compared to unexposed conditions because 23 the negative consequences of overcrowding were diminished (Ferson et al. 1996). 24 Similar effects on other fish species, however, have led to population collapses 25 (Myers et al. 1995).
- Although there are exceptions (such as threatened and endangered species), an effect level of 10% is unlikely to be ecologically significant. Thus, if the probability of exceeding this effect level is relatively low (<20%), risk is deemed to be negligible to low.
- Several studies have shown that NOAELs are generally associated with effects of 10% or greater (85% of studies examined by Moore et al. 1997), and LOAELs are generally associated with effects of 20% or greater (79% of studies examined by Moore et al. 1997) (also see Hoeckstra and Van Ewijk 1993; Pack 1993). Therefore, the decision criteria above equated NOAELs with the 10% effect level, and LOAELs with the 20% effect level.

1 When toxicity data are lacking for representative species or reasonable surrogates, the 2 toxicity threshold for representative species is assumed to be between the thresholds of the most sensitive and tolerant species tested. Thus, if the probability of exceeding 3 4 the lowest threshold is low (< 20%), risk is deemed to be negligible to low. Tolerant 5 species may have thresholds one to several orders of magnitude higher than sensitive 6 species (see effects assessment sections in Appendices G to K). Thus, at the highest 7 threshold, it is likely that some representative species would be adversely affected, possibly quite seriously. Thus, even a relatively low probability (20% or greater) of 8 9 exceeding the upper threshold may be cause for concern.

Any effect to threatened and endangered species is cause for concern (Massachusetts Office of Environmental Affairs 1999; Massachusetts Division of Fisheries and Wildlife 2003; United States Congress 1973, Endangered Species Act). Because a LOAEL generally represents >20% effect, the criterion separating intermediate and high risk was adjusted for threatened and endangered species. Thus, a better than even (>50%) chance of exceeding a NOAEL was deemed to represent a high risk situation for threatened and endangered species.

The risk categories should not be used alone to determine whether risk management actions are necessary. Risk categories are based on the results of the Monte Carlo exposure analysis only. Risk categories are uncertain in cases where the risk range is wide (e.g., low to high). Risk categories are also uncertain for assessment endpoints without corroborating lines of evidence. Thus, the risk categories should be considered as a qualitative ranking of risk to facilitate comparisons between COCs, locations, and assessment endpoints. They are intended to contribute to weight-of-evidence assessments, not to replace them.

24 6.7.2 Weight-of-Evidence Assessment

25 A WOE approach was used in the risk assessments for wildlife. The WOE approach is a process 26 by which measurement endpoints are related to an assessment endpoint to evaluate whether 27 significant harm is posed to the environment (Menzie et al. 1996). The WOE approach used in 28 this ERA follows the approach originally described in the Massachusetts Weight of Evidence 29 Special Report (Menzie et al. 1996). A detailed review of the WOE approach used in the 30 Housatonic River ERA is provided in Section 2.9. In general, the WOE approach is an inclusive 31 process whereby multiple lines of evidence are considered prior to determining risk. For the 32 wildlife risk assessments, these lines of evidence included the exposure and effects modeling 33 results, field survey results, and/or in situ or whole media toxicity test results.

For the wildlife assessment endpoints, risk categories and risk ranges were developed for the modeling of exposure and effects line of evidence. The Massachusetts Weight-of-Evidence (WOE) approach requires a determination for evidence of harm and magnitude of effect for each assessment endpoint-COC scenario. For this assessment, criteria were developed for converting risk category and risk range to evidence of harm and magnitude of effect on the Massachusetts WOE scoring sheets (Table 6.7-1). Table 6.7-1

1 2

3

Decision Criteria for Converting Risk Category and Range to Evidence of Harm and Magnitude of Effect

Disk Catagory	Risk Range							
Kisk Category	Low	Low/Intermediate	Intermediate	Intermediate/High	High	Low/High		
Low	Evidence=No Magnitude=Low	Evidence=No Magnitude=Low				Evidence=Undetermined Magnitude=Low		
Intermediate		Evidence=Undetermined Magnitude=Intermediate	Evidence=Yes Magnitude=Intermediate	Evidence=Yes Magnitude=Intermediate		Evidence=Undetermined Magnitude=Intermediate		
High				Evidence=Yes Magnitude=High	Evidence=Yes Magnitude=High	Evidence=Undetermined Magnitude=High		

4 Evidence=Evidence of Harm (Yes, No, Undetermined), Magnitude=Magnitude (High, Intermediate, Low), --- Indicates outcome is not possible

1 6.8 REFERENCES

- Alhborg, U.S., G.C. Becking, L.S. Birnbaum, A. Brouwer, H.J.G.M. Derks, M. Feeley, G. Golor,
 A. Hanberg, J.C. Larsen, A.K.D. Liem, S.H. Safe, C. Schlatter, F. Wern, M. Younes, and E.
 Yrjanheikki. 1994. Toxic equivalency factors for dioxin-like PCBs: Report on a WHO-ECEH
 and IPCS consultation, December 1993. *Chemosphere* 28:1049-1067.
- 6 Bailer, A.J. and J.T. Oris. 1997. Estimating inhibition concentrations for different response 7 scales using generalized linear models. *Environmental Toxicology and Chemistry* 16:1554-1559.
- 8 Birnbaum, L.S. and M.J. DeVito. 1995. Use of toxic equivalency factors for risk assessment for 9 dioxins and related compounds. *Toxicology* 105:391-401.
- Cogliano, V.J. 1998. Assessing the cancer risk from environmental PCBs. *Environmental Health Perspectives* 106(6):317-323
- Dyke, P.H. and J. Stratford. 2002. Changes to the TEF schemes can have significant impacts on regulation and management of PCDD/F and PCB. *Chemosphere* 47:103-116.
- 14 Efroymson, R. and G.W. Suter II. 1999. Finding a niche for soil microbial toxicity tests in 15 ecological risk assessment. *Human and Ecological Risk Assessment* 5(4):635-868.
- 16 EPA (U.S. Environmental Protection Agency). 1987. *Interim Procedures for Estimating Risk* 17 *Associated with Exposure to Mixtures of Chlorinated Dibenzo-p-dioxins and Dibenzofurans* 18 *(CDD:CDFs)*. U.S. Environmental Protection Agency. EPA 625-3-87012.
- 19 EPA (U.S. Environmental Protection Agency). 1989. Interim Procedures for Estimating Risks
- 20 Associated with Exposure to Mixtures of Chlorinated Dibenzo-p-dioxins and Dibenzofurans and
- 21 *1989 Update*. Risk Assessment Forum, EPA/625/3-89/016.
- EPA (U.S. Environmental Protection Agency). 1991. Workshop Report on Toxicity Equivalence
 Factors for Polychlorinated Biphenyl Congeners Risk Assessment Forum. U.S. Environmental
- 24 Protection Agency. EPA/625/3-91/020.
- EPA (U.S. Environmental Protection Agency). 1992. A Supplemental Guidance to RAGS:
 Calculating the Concentration Term. Publication 9285.7-081. Office of Research and
 Development, Washington, DC. http://www.deq.state.ms.us/newweb/opchome.nsf/pages/
 HWDivisionFiles/ \$file/uclmean.pdf.
- EPA (United States Environmental Protection Agency). 1993. Wildlife Exposure Factors
 Handbook. Volumes I and II. Office of Research and Development. Washington, DC. EPA
 EPA/600/R-93/187a, EPA/600/R-93/187b.
- 32 EPA (U.S. Environmental Protection Agency). 1997. Guiding Principles for Monte Carlo
- 33 Analysis. U.S. Environmental Protection Agency, Office of Research and Development, 34 Washington DC ERA/630/R 07/001
- 34 Washington, DC. EPA/630/R-97/001.

- 1 EPA (U.S. Environmental Protection Agency). 1999. Risk Assessment Guidance for Superfund,
- 2 Volume 3 Part A, Process for Conducting Probabilistic Risk Assessment. Draft. Office of Solid
- 3 Waste and Emergency Response, U.S. Environmental Protection Agency, Washington, DC.
- Ferson, S. 1996. What Monte Carlo methods cannot do. *Human and Ecological Risk Assessment* 2(4):990-1007.
- Ferson, S., L.R. Ginzburg, and R.A. Goldstein. 1996. Inferring ecological risk from toxicity
 bioassays. *Journal of Water, Air and Soil Pollution* 90:71-82.
- 8 Gilbert, R.O. 1987. *Statistical Methods for Environmental Pollution Monitoring*. Van Nostrand
 9 Reinhold Company, New York.
- Haimes, Y.Y., T. Barry, and J.H. Lambert, Editors. 1994. When and how you can specify a
 probability distribution when you don't know much? *Risk Analysis* 14:661-706.
- Hattis, D. and D.E. Burmaster. 1994. Assessment of variability and uncertainty distributions for
 practical risk analyses. *Risk Analysis* 14:713-730.
- Hoeckstra, J.A. and P.H. Van Ewijk. 1993. Alternatives for the no-observed-effect-level.
 Environmental Toxicology and Chemistry 12:187-194.
- Kennedy, S.W., A. Lorenzen, and R.J. Norstrom. 1996. Chicken embryo hepatocyte bioassay for
 measuring cytochrome P4501A-based 2,3,7,8-tetrachlorodibenzo-p-dioxin equivalent
 concentrations in environmental samples. *Environmental Science and Technology* 30:706-715.
- Kerr, D.R. and J.P. Meador. 1996. Modeling dose response using generalized linear models.
 Environmental Toxicology and Chemistry 15:395-401.
- Long, E.R., D.D. MacDonald, S.L. Smith, and F.D. Calder. 1995. Incidence of adverse
 biological effects within ranges of chemical concentrations in marine and estuarine sediments.
 Environmental Management 19(1):81-97.
- MacDonald, D.D., R.S. Carr, F.D. Calder, E.R. Long, and C.G. Ingersoll. 1996. Development and evaluation of sediment quality guidelines for Florida coastal waters. *Ecotoxicology* 5:253-278.
- Massachusetts Division of Fisheries and Wildlife. 2003. 321 CMR 10.00 and 11.00.
 Massachusetts Endangered Species Act Regulations. Boston, MA.
- Massachusetts Office of Environmental Affairs. 1999. 310 CMR 40.00. Massachusetts
 Contingency Plan (MCP). Boston, MA.
- 31 Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S.
- 32 Petron, B. Potocki, S. Svirsky, and P. Tyler. 1996. Special report of the Massachusetts Weight-
- 33 of-Evidence Workgroup: A weight-of-evidence approach for evaluating ecological risks. *Human*
- 34 *and Ecological Risk Assessment* 2(2):277-304.

Moore, D.R.J., W. Warren-Hicks, B.R. Parkhurst, R.S. Teed, R.B. Baird, R. Berger,
 D.L. Denton, and J.J. Pletl. 2000. Intra- and intertreatment variability in reference toxicant tests:
 Implications for whole effluent toxicity testing programs. *Environmental Toxicology and Chemistry* 19(1):105-112.

Moore, D.R.J., B.E. Sample, G.W. Suter, B.R. Parkhurst, and R.S. Teed. 1999. A probabilistic
risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East
Fork Poplar Creek, Oak Ridge, Tennessee, USA. *Environmental Toxicology and Chemistry*18(12):2941-2953.

9 Moore, D.R.J., R.L. Breton, and K. Loyd. 1997. The effects of hexachlorobenzene on mink in 10 the Canadian environment: An ecological risk assessment. *Environmental Toxicology and*

- 11 *Chemistry* 16(5):1042-1050.
- Moore, D.R.J. and P.-Y. Caux. 1997. Estimating low toxic effects. *Environmental Toxicology and Chemistry* 16(4):794-801.
- 14 Myers, R.A., N.J. Barrowman, J.A. Hutchings, and A.A. Rosenberg. 1995. Population dynamics 15 of exploited fish stocks at low population levels. *Science* 269:1106-1108.
- Nagy, K.A., I.A. Girard, and T.K. Brown. 1999. Energetics of free-ranging mammals, reptiles,
 and birds. *Annual Reviews of Nutrition* 19:247-77
- 18 NATO (North Atlantic Treaty Organization). 1988a. International Toxicity Equivalence Factors
- 19 (I-TEF) Method of Risk Assessment for Complex Mixtures of Dioxins and Related Compounds.
- 20 Pilot Study on International Information Exchange on Dioxins and Related Compounds. Report
- 21 Number 176, August 1988. North Atlantic Treaty Organization, Committee on Challenges of
- 22 Modern Society.
- 23 NATO (North Atlantic Treaty Organization). 1988b. Scientific Basis for the Development of
- 24 International Toxicity Equivalency Factor (I-TEF), Method of Risk Assessment for Assessment of
- 25 Complex Mixtures of Dioxins and Related Compounds. Report No. 178.
- 26 Newsted, J.L., J.P. Giesy, G.T. Ankley, D.E. Tillitt, R.A. Crawford, J.W. Gooch, P.D. Jones, and
- 27 M.S. Denison. 1995. Development of toxic equivalency factors for PCB congeners and the
- 28 assessment of TCDD and PCB mixtures in rainbow trout. Environmental Toxicology and
- 29 *Chemistry* 14(5):861-871
- 30 Ott, W.R. 1995. Environmental Statistics and Data Analysis. Lewis Publishers, Boca Raton, FL.
- 31 Pack, S. 1993. A Review of Statistical Data Analysis and Experimental Design in OECD Aquatic
- 32 Toxicology Test Guidelines. Organization of Economic Cooperation and Development. Paris,
- 33 France.
- 34 Safe, S. 1990. Polychlorinated biphenyls (PCBs) dibenzo-p-dioxins (PCDDs), dibenzofurans
- 35 (PCDFs), and related compounds: environmental and mechanistic considerations which support
- 36 the development of toxic equivalence factors (TEFs). *Critical Reviews of Toxicology* 21:51-88.

- 1 Safe, S.H. 1994. Polychlorinated biphenyls (PCBs): Environmental impact, biochemical and 2 toxic responses, and implications for risk assessment. *Critical Reviews in Toxicology* 3 24(2):87-149.
- 4 Schulz, T.W. and S. Griffin. 1999. Estimating risk assessment exposure point concentrations 5 when the data are not normal or lognormal. *Risk Analysis* 19:577-584.
- 6 Seiler, F.A. and J.L. Alvarez. 1996. On the selection of distributions for stochastic variables. *Risk* 7 *Analysis* 16:5-18.
- 8 Singh, A.K., A. Singh, and M. Engelhardt. 1997. The lognormal distribution in environmental
 9 applications. EPA/600/R-97/006. U.S. Environmental Protection Agency, Washington, DC.
- 10 Stephan, C.E. and J.W. Rogers. 1985. Advantages of using regression analysis to calculate
- 11 results of chronic toxicity tests. In Aquatic Toxicology and Hazard Assessment. R.C. Bahner and
- 12 D.J. Hansen, Editors. STP 737. American Society for Testing and Materials, Philadelphia, PA.
- 13 pp. 377-387.
- Suter, G.W., II. 1996. Toxicological benchmarks for screening contaminants of potential concern
 for effects on freshwater biota. *Environmental Toxicology and Chemistry* 15(7):1232-1241.
- United States Congress. 1973. Endangered Species Act of 1973 (87 Statute 884). Washington,
 DC.
- 18 Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Freely, J.P. Giesy,
- 19 A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X.R van Leeuwen,
- 20 A.K. Djien Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt,
- M. Tysklind, M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives*
- 22 for PCBs, PCDDs, PCDFs for numans and windine. *Environmental Health Perspecti* 23 106(12):775-792.
- 24 Wentsel, R.S., M. Simini, R.T. Checkai, D. Ludwig, and T.W. La Point. 1997. Ecological Risk
- 25 Assessment Procedures for U.S. Army sites. Environmental Toxicology and Risk Assessment:
- 26 Modelling and Risk Assessment (Sixth Volume), ASTM STP 1317, F.J. Dwyer, T.R. Doane and
- 27 M.L. Hinman, Eds, American Society for Testing and Materials, West Conshohocken, PA.

7. ASSESSMENT ENDPOINT – SURVIVAL, GROWTH, AND 2 REPRODUCTION OF INSECTIVOROUS BIRDS

3	Highlights
4	Conceptual Model
5 6 7 8 9	The assessment endpoint is the survival, growth, and reproduction of insectivorous birds in the Housatonic River PSA. Insectivorous birds, including tree swallows and American robins, are exposed to tPCBs and TEQ via trophic transfer. These two species were selected as the representative species for the ecological risk assessment (ERA).
10	Exposure
11 12 13 14 15 16	Exposure of the representative species to tPCBs and TEQ was determined from: (1) concentrations found in prey items, (2) an estimation of the daily intake of these contaminants of concern (COCs) from consumption of prey, and (3) for tree swallows exposed to tPCBs only, tissue concentrations in 14-day-old nestlings. Site-specific nesting and reproduction studies were conducted to evaluate adverse effects to tree swallows and American robins from Housatonic River COCs.
17	Effects
18 19 20 21 22	Field data on the toxicity of tPCBs were available for tree swallows, but not American robins. No data were available on the toxicity of TEQ to tree swallows or American robins. Thus, surrogate species were used to estimate effects to American robins exposed to tPCBs and for both representative species exposed to TEQ. A threshold range spanning sensitive and tolerant surrogate species was used in these cases.
23	Risk
24 25 26 27 28 29 30 31 32 33 34 35 36	Modeled exposure and effects for tree swallows and American robins suggests that they are at intermediate to high risk as a result of exposure to tPCBs and TEQ in the Housatonic River PSA. However, the more highly weighted field study line of evidence suggests that if effects are occurring, they are minor for both species. Therefore, the weight-of-evidence (WOE) assessment favors a finding of low risk for insectivorous birds exposed to tPCBs and TEQ in the PSA. This conclusion, however, is uncertain because of the conflicting results in the WOE assessment. Other insectivorous bird species common to the PSA are expected to have either a similar to lower level of risk (e.g., cliff swallow, eastern kingbird, eastern bluebird, eastern towhee); a similar level of risk (e.g., barn swallow, common nighthawk, eastern phoebe, hermit thrush, northern mockingbird, veery, wood thrush); or a similar to higher level of risk (e.g., bank swallow, chimney swift, northern rough- winged swallow, gray catbird) compared to the representative species.

1 7.1 INTRODUCTION

2 The purpose of this section is to characterize and quantify the current and potential risks posed to 3 insectivorous birds exposed to contaminants of potential concern (COPCs) in the Housatonic 4 River and floodplain, focusing on total PCBs (tPCBs) and other COPCs originating from the 5 General Electric Company (GE) facility in Pittsfield, MA. The watershed is located in western 6 Massachusetts and Connecticut, discharging to Long Island Sound, with the GE facility located 7 near the headwaters of the watershed. The Primary Study Area (PSA) includes the river and 10-8 year floodplain from the confluence of the East and West Branches of the Housatonic River 9 downstream of the GE facility, to Woods Pond Dam.

10 A pre-ERA was conducted to narrow the scope of the ERA by identifying contaminants, other 11 than tPCBs, that pose potential risks to aquatic biota and wildlife in the PSA (Appendix B). A 12 three-tiered deterministic approach was used to screen COPCs. The deterministic assessments 13 compared potential conservative estimates of exposure with conservative adverse effects 14 benchmarks to identify COPCs for insectivorous birds in the Housatonic River. A hazard 15 quotient (total daily intake/effect benchmark) greater than 1 for insectivorous birds in the Housatonic River area resulted in the COPC being screened through to the next tier of the 16 17 assessment, and to the probabilistic ERA if necessary. Subsequent to the pre-ERA, several other 18 COPCs (primarily organochlorine pesticides) were screened out because their actual 19 concentrations in the PSA were likely much lower than the measured values due to laboratory 20 interference (see Section 2.4).

In summary, the COPCs that screened through to the probabilistic risk assessment for insectivorous birds were tPCBs and 2,3,7,8-TCDD toxic equivalence (TEQ). Total PCBs and TEQ were retained as contaminants of concern (COCs) for this assessment endpoint. Total PCBs detected in Housatonic River samples closely resemble the commercial PCB mixtures Aroclor 1260 and Aroclor 1254, which are similar in congener makeup. TEQ is calculated from coplanar PCB and dioxin and furan congeners using the toxic equivalency factor (TEF) approach developed by Van den Berg et al. (1998) (see Section 6.4). A step-wise approach was used to assess the risks of tPCBs and TEQ to insectivorous birds in
 the Housatonic River watershed. The four main steps in this process include:

3	1.	Derivation of a conceptual model (Figure 7.1-1).					
4	2.	2. Assessment of exposure of birds to COCs (Figure 7.1-2).					
5	3.	Assessment of the effects of COCs on birds (Figure 7.1-3).					
6	4	Characterization of risks to the insectivorous bird species (Figure 7 1-4)					
7							
8	This section	on is organized as follows.					
9 10	•	Section 7.2 presents the conceptual model for assessing the ecological risk to insectivorous birds.					
11	-	Section 7.3 describes the exposure models, input parameters, and techniques to					
12		propagate uncertainty. Also presented in this section are the exposure modeling					
13		results for tree swallows and American robins.					
14	•	Section 7.4 provides an overview of the literature on the effects of tPCBs and TEQ to					
15		survival, growth, and reproduction of tree swallows, American robins, and other bird					
16		species. Field studies on the reproduction of tree swallows (Custer 2002) and					
17		American robins (Henning 2002) are discussed. Key studies are then selected and					
18		used to derive the most appropriate effects metrics.					
19	-	The two lines of evidence for each representative species are discussed in the risk					
20		characterization, Section 7.5, followed by a discussion of the sources of uncertainty in					
21		this assessment, and the conclusions regarding risks of tPCBs and TEO to					
22		insectivorous birds in the Housatonic River PSA.					
a a							
23 24	l r	birds, which is presented in detail in Appendix G.					
	l						
25							





4

Figure 7.1-1 Conceptual Model Diagram: Exposure Pathways for Insectivorous Birds Exposed to Contaminants of Concern in the Housatonic River PSA

EXPOSURE



Figure 7.1-2 Overview of Approach Used to Assess Modeled Exposure of Insectivorous Birds to Contaminants of Concern in the Housatonic River PSA

6

1 2

3

4

EFFECTS



3

1

2

4



RISK CHARACTERIZATION



 Figure 7.1-4
 Figure 7.1-4
 Overview of Approach Used to Characterize the Risks of Contaminants of Concern to Insectivorous Birds in the Housatonic River PSA

1 7.2 CONCEPTUAL MODEL

2 The conceptual model presented in Figure 7.1-1 illustrates the exposure pathways for 3 insectivorous birds exposed to tPCBs and TEQ in the PSA. Total PCBs and TEQ are persistent, 4 lipophilic, and hydrophobic. Therefore, they are highly bioaccumulated by aquatic and 5 terrestrial biota directly through the consumption of contaminated prey as part of the food chain 6 (Haffner et al. 1994; Senthilkumar et al. 2001; Borga et al. 2001). Emergent aquatic insects and 7 terrestrial invertebrates are major dietary items for insectivorous birds. Insectivorous birds that 8 reside, or partially reside, within the study area are exposed to tPCBs and TEQ principally 9 through diet and trophic transfer. Other routes of exposure, considered to be less important to 10 overall exposure, include inhalation, water consumption, and soil ingestion (Moore et al. 1999).

The problem formulation (see Section 2) identified the tree swallow (*Tachycineta bicolor*; Figure 7.2-1) and American robin (*Turdus migratorius*; Figure 7.2-2) as the representative species for insectivorous birds potentially exposed to tPCBs and TEQ from consumption of contaminated prey. Life history profiles for these bird species are summarized in the following text boxes. Additional life history information for both species can be found in Sections G.2.1.3 and G.2.1.4.

Life History of the Tree Swallow

The tree swallow is distributed widely throughout northern and central North America and is one of the most widespread members of its genus. Tree swallows breed on both coasts and northward to the tree line.

Habitat - Prefer open habitat near water, including fields, marshes, shorelines, and wooded swamps. Tree swallows are hole nesters and depend on woodpeckers and other excavators to furnish nesting cavities. When defending a nest site, and especially when feeding nestlings, tree swallows show the greatest home range tenacity and typically remain within 100 to 200 m of their nest site. Large colonies can reach densities of 150 pairs per 0.7 acre (0.28 ha).

Diet – Tree swallows actively pursue flying insects, and occasionally glean insects from the water surface or vegetation. Food items include mosquitoes, midges, gnats, mayflies, and beetles.

Life History of the American Robin

The American robin is a common, wideranging North American bird, present throughout the continental United States and Canada, excluding the extreme north and high altitudes.

Habitat - Occupies a wide range of habitat types, from closed canopy forests to residential areas. In summer, commonly observed in cleared areas with short herbs, such as natural forest openings, lawns, and recently cleared or burned stands. Territory is small, ranging from 0.3 to 2.0 acres (0.1 to 0.8 ha).

Diet - Forages on the ground and in vegetation for invertebrates and fruits; proportions vary by season. In spring, diet comprises less than 10% fruits. In fall and early winter, fruits account for 80% to 99% of the diet, with the remainder of the diet invertebrates.



Figure 7.2-1 Tree Swallow (*Tachycineta bicolor*)

1





reproduction of insectivorous birds in the Housatonic River PSA. The measurement endpoints used to evaluate the assessment endpoint include: (1) determining, by comparisons of modeled exposure to doses reported in the literature to cause adverse effects, the extent to which the concentrations of tPCBs and TEQ ingested in the diet will cause adverse effects to the survival, 10 growth, and reproduction of insectivorous birds; and (2) determining, by conducting field 11 studies, the relationship between concentrations of tPCBs and TEQ in prey and the reproductive 12 performance of insectivorous birds in the Housatonic River floodplain. The 3-year tree swallow 13 field study measured clutch size, hatching success, and tissue concentrations of tPCBs and TEQ 14 and other variables at three locations in the PSA, and three reference locations. The American 15 robin field study evaluated the relationship between reproductive success and tissue 16 concentrations of tPCBs in eggs and young.

1 7.3 EXPOSURE ASSESSMENT

The exposure assessment for insectivorous birds focuses on both the PSA and several reference locations. Trophic transfer and exposure through ingestion of contaminated food items are the major exposure pathways for insectivorous birds exposed to tPCBs and 2,3,7,8-TCDD TEQ (TEQ). Other routes of exposure, considered to be negligible contributors to overall exposure, include inhalation, water consumption, and soil ingestion (Moore et al. 1999). PCBs and TEQ tend to bioaccumulate in the food chain because:

- 8
- PCBs and TEQ are persistent, and highly lipophilic and hydrophobic substances.
- When released to aquatic systems, these COCs form associations with dissolved and/or particulate matter in the water column and remain in the sediment; biodegradation is considered to be a relatively minor fate process in water (NRCC 12 1981; Howard et al. 1991).
- Aquatic sediment provides a sink for these compounds and may represent long-term sources to the aquatic food web (Kuehl et al. 1987; Muir 1988; Corbet et al. 1983; Tsushimoto et al. 1982). Both PCBs and TEQ are bioaccumulated by aquatic and terrestrial biota through the consumption of contaminated prey as part of the food chain (Haffner et al. 1994; Senthilkumar et al. 2001; Borga et al. 2001).

In summary, insectivorous birds that reside, or partially reside, within the PSA are exposed totPCBs and TEQ principally through diet and trophic transfer.

20 Exposure of tree swallows and American robins to tPCBs and TEQ was estimated using the 21 standard total daily intake model. Tree swallow exposure was also estimated using an explicit 22 microexposure model. The total daily intake model was adapted from the *Wildlife Exposure* 23 Factors Handbook (EPA 1993) and was also used in the other wildlife assessments. For the 24 microexposure model, accumulation of tPCBs and TEQ over the first 15 days of the birds' 25 development was estimated. The results of the two modeling approaches are compared, and the 26 tissue concentration data collected from tree swallow nestlings in the PSA and reference areas 27 compared to predicted tissue concentrations from the microexposure model.

The tree swallow field study was performed with nest boxes placed at six locations, three of which were located downstream of the GE facility within the PSA (Holmes Road, New Lenox Road, and Roaring Brook). Three other locations (Threemile Pond, Southwest Branch, and

Taconic Valley) were expected to serve as reference locations (Custer 2002). In this field study, 1 2 nest boxes were placed adjacent to the river and on the nearby floodplain. A map of the tree 3 swallow nest box locations is shown in Figure 7.3-1. The reference location at Threemile Pond 4 (not depicted on the map) is within the Housatonic River drainage, but is upgradient of the river. 5 This location was expected to be representative of background contaminant concentrations. Two 6 other reference locations (Southwest Branch and Taconic Valley sites) lie upstream of the major 7 source of PCB contamination. The exposure assessment focused on the six locations used in the 8 field study for tree swallows by Custer (2002).

9 Exposure of American robins was estimated for three areas (Locations 13, 14, and 15; see Figure

10 7.3-1 and text box) in the PSA.

An exposure assessment was not conducted for American robins in reference areas because concentrations of COCs in robin prey items were not available from these locations. In addition, concentrations of COCs in prey in the floodplain reference areas were not estimated because nearly all sediment samples from this area did not have detectable concentrations of tPCBs, therefore, it is not likely that the floodplain soil have detectable concentrations of PCBs.

16 Description of Sites 13, 14, and 15 17 Location 13 is a relatively flat area on the west shore of the river, in the floodplain 18 adjacent to river mile 133.2, situated at an elevation of 965 ft (294 m). The 19 community type is transitional floodplain forest that is flooded seasonally and is 20 moderately well drained, with extensive vegetation cover (80%) and alluvial silt-loam 21 soil. The PCB concentrations in floodplain soil averaged 55.2 mg/kg dw. 22 Location 14 is a relatively flat low-lying area located on the west shore of the river, in 23 the floodplain adjacent to river mile 129.9, situated at an elevation of 965 ft (294 m). 24 The community type is transitional floodplain forest that is flooded seasonally with 25 extensive vegetation cover (70%) and fluvial silt soil. The PCB concentrations in 26 floodplain soil averaged 26.1 mg/kg dw. 27 Location 15 is a flat area located on the west shore of the river, in the floodplain 28 adjacent to river mile 126.7, situated at an elevation of 965 ft (294 m). Community 29 types are circumneutral hardwood swamp and transitional floodplain forest that are 30 flooded seasonally. This site has 60% vegetation cover, 40% leaf litter cover, and a 31 primarily mineral soil. The PCB concentrations in floodplain soil averaged 0.484 32 ma/ka dw.

33



o:\gepitt\aprs\swallows.apr | Layout - swallow/soilinvert locs 7.3-1 | o:\gepitt\plots\in\tsnb_sis_locs_7-3-1.eps | 10:21 AM, 7/3/2003 |

This section begins with a description of the standard total daily intake (TDI) model used for tree swallows and American robins. For tree swallows, the TDI model was used as the basis for developing a microexposure model to estimate accumulation of tPCBs in tissues of nestlings from hatch to 14 days post-hatch. Subsequent sections describe the inputs used in the exposure analyses. The section concludes with a description of the Monte Carlo and probability bounds analyses conducted to estimate exposure of tree swallows and American robins to tPCBs and TEQ in the Housatonic River PSA and reference areas.

8 7.3.1 Exposure Models for Insectivorous Birds

9 7.3.1.1 Total Daily Intake Model

Exposure of the representative species, tree swallows and American robins, to tPCBs and TEQ
 was estimated using a total daily intake model adapted from the *Wildlife Exposure Factors Handbook* (EPA 1993) and related publications. The model used in the exposure analysis was:

13
$$TDI = FT \times FIR \sum_{i=1}^{n} C_i \times P_i$$
 (Eq. 1)

14 where

- 15 TDI = Total daily intake (mg/kg bw/d tPCBs, ng/kg bw/d TEQ).
- 16 FT = Foraging time in the area of interest (unitless and set equal to one for 17 insectivorous birds).
- 18 FIR = Normalized food intake rate (kg/kg bw/d).
- 19 P_i = Proportion of the *i*th dietary item (unitless).
- 20 C_i = Concentration in food item (mg/kg for tPCBs, ng/kg for TEQ).

The model considers the food intake rates of representative species (*FIR*), the concentrations of COCs in each food item (C_i), and the proportion of diet accounted for by that food item (P_i). For those input variables that are uncertain, variable, or both, distributions are used rather than point estimates. Monte Carlo and probability bounds analyses are used to propagate uncertainties about input variables through the exposure model for each COC. A description of these techniques and the methods used to parameterize input variables is presented in Section 6.5 and Appendix C.4. The results of the Monte Carlo analysis are used to estimate the probability of exposure exceeding an effects threshold or doses that cause adverse effects of differing magnitudes. The probability bounds analysis is conducted to determine how uncertainty regarding the distributions of the input variables influences the estimated exposure distribution. The results of these analyses are discussed in detail in Appendix G.

6 7.3.1.2 Microexposure Model

7 The exposure model is a simple model driven by the concentration of COCs in the diet and by 8 the food intake rate. In this section, a more complex, dynamic microexposure model is derived 9 for the tree swallow exposure assessment. This model determines the whole body contaminant 10 content in the swallows as a function of time over the first 15 days of their development.

11 A simulation length of 15 days was chosen because swallows reach adult body weight at 12 approximately this time. In addition, nestling swallow tissue samples were collected between 12 13 and 14 days in the Custer (2002) study.

The microexposure model equation for whole body tPCB and TEQ tissue concentrations at 15
days (*TOT BURDEN*) is given below:

16

$$TOT \ BURDEN = (TOT \ MT + \sum_{i} [(FIR_{i} \times C_{diet}) \times 1 \ d]) / BW$$
(Eq. 2)

17 where:

18 19 20	TOT_BURDEN	=	Total amount of COCs accumulated by tree swallows from maternal transfer plus their first 15 days of food consumption per gram of body weight (mg/kg for tPCBs, ng/kg for TEQ).
21	TOT_MT	=	COC tissue concentration (μg) from maternal transfer.
22	i	=	Refers to the time steps in the simulation (range from 0 to 14 days ¹).
23 24	<i>FIR</i> _i	=	Food intake rate (g/d) calculated using a variable body weight specific to each day of the simulation.

¹ Note that the first day of the simulation is day zero, and the last day of the simulation is day 14; therefore, the total simulation length is 15 days.

1 C_{diet} = Concentration in food item (mg/kg for tPCBs, ng/kg for TEQ).

2 BW = Body weight at day 14 (g).

3 All of the subscripts *i* refer to the time steps in the simulation, which range from 0 to 14 days. 4 C_{diet} (mg/kg) is the same as C_i in the TDI model. Intake rate of food (*FIR_i* measured in g/d) was 5 calculated in the same manner as in the TDI model (see Section G.2.1.3.2), except the body 6 weight variable was made specific to each day of the simulation. Because the value of food 7 intake rate is a function of body weight in the microexposure model, it is subscripted by day to 8 account for the effect of changing body weight over the simulation. The intake rate was 9 converted to the amount consumed on each day *i* by multiplying through by one day (1 d). The term TOT MT represents the COC tissue concentration from maternal transfer (see Section 10 11 G.2.1.3.2). The sum of maternal transfer and the expression in the summation was divided at the 12 end of the simulation by body weight at day 14 (BW). Thus, TOT BURDEN estimates the total 13 amount of COCs accumulated by tree swallows from maternal transfer plus their first 15 days of 14 food consumption per kilogram of body weight.

15 Two issues often arise when calculating a TEQ concentration in prey:

16 17

18

- Congener concentrations may be below the method detection limit (i.e., non-detects).
- Some congeners may not be resolved due to co-elution during analysis.

An approach was developed to address these issues and is presented in Section 6.4 and Appendix
C.2). Briefly, congeners detected at or below the detection limit (DL) were included in the TEQ
calculations by investigating three options:

- Setting the value for the congener equal to zero (0).
 - Setting it to half the DL.
- 23 24
- Setting it equal to the DL.
- 25
- Seame a equal to the I
- A comparison of the results of this bounding analysis provides a description of the uncertainty surrounding the TEQ value due to concentrations of one or more congeners being below the detection limit.

To resolve the co-elution issue, the concentrations of coplanar (TEQ) congeners that co-eluted with other congeners were assumed to be equal to the total concentration of the co-elutes (likely overestimate of TEQ concentration) or zero (likely underestimate of TEQ concentration). The

1 decision criteria in Section 6.4 were followed to address the uncertainty arising from co-elution 2 or non-detection of congeners when estimating exposure point concentrations (EPCs) for use in 3 the exposure analyses. 4 Input distributions to the exposure analyses were generally assigned as follows: 5 Lognormal distributions for variables that were right skewed with a lower bound of 6 zero and no upper bound (e.g., amount of COC transferred from mother to offspring via egg tissue for tree swallow). 7 8 Beta distributions for variables bounded by zero and one (e.g., proportion of a prey 9 item in the diet). 10 Normal distributions for variables that were symmetric and not bounded by one (e.g., 11 body weight). 12 Point estimates for minor variables or variables with low coefficients of variation. 13 In certain situations (e.g., poor fit of data), other distributions were fit to the data or other 14 approaches were used. To quantify uncertainty, two approaches were used as described in Section 6.5.2. The distributions used in the exposure analyses for tree swallows and American 15 16 robins are shown in Figures 7.3-2, 7.3-3, and 7.3-4 and are described in the following sections. 17 These distributions are also presented in greater detail in Appendix G.

18 Foraging Time

The foraging ranges of the two representative species are within the area of the PSA. Prey availability and an abundance of suitable foraging habitat suggest that the birds that forage in the PSA are able to meet their needs exclusively within this section of the river and floodplain. The assessment of risk to insectivorous birds inhabiting the PSA of the Housatonic River will, therefore, focus on those birds that spend 100% of their time foraging within the PSA. Foraging time is a point estimate; therefore, it is not shown in Figures 7.3-2, 7.3-3, and 7.3-4.







3 Figure 7.3-2 TDI Exposure Model Input Distributions for Tree Swallows



Maternal Transfer at Southwest Branch



2 Figure 7.3-3 Microexposure Model Input Distributions for Maternal Transfer for 3 Tree Swallows

4



2 Figure 7.3-4 TDI Exposure Model Input Distributions for American Robin

3

1 Body Weight (BW)

2 TDI Model

Tree swallows are small birds with an average adult body weight of about 20 g. Body weight can be as low as 16.5 g when food availability is low, and as high as 25.5 g for females during the mating season (Robertson et al. 1992). Newly hatched nestlings weigh about 1.5 g and achieve adult weight in about 14 days. Based on data cited in Dunning (1984), the mean adult body weight was estimated to be 20.1 g and standard deviation of 1.58 (Figure 7.3-2).

American robins are a sexually monomorphic species with similar male and female body weights (Clench and Leberman 1978; Wheelwright 1986; Marcum et al. 1998). Robins monitored in Delta Marsh, Manitoba, Canada, ranged from 72 to 86 g, with females gaining slightly more weight during the incubation period (Bierman and Sealy 1985). Clench and Leberman (1978) found an average mass of 77 g when data from both sexes were pooled. The distribution of the body weights of American robins is depicted in Figure 7.3-4.

14 Microexposure Model

The body weight of juvenile birds was modeled as a function of time, leveling off at 12 to 14 days, using a logistic model from Teather (1996). In the Monte Carlo version of the microexposure model, point values were used for body weight. In the probability bounds analysis, the uncertainty surrounding body weight as a function of time was taken into account. Tables G.2-4 and G.2-5 show the body weight distributions used in the Monte Carlo and probability bounds analyses, respectively.

21 Food Intake Rate (FIR)

22 TDI Model

The food intake rate of tree swallows and American robins has not been well characterized. The food intake rate for American robins has been measured in captive animals (Hazelton et al. 1984). Because the animals were captive, the measured food intake rates likely underestimated food intake rates of free-living robins (EPA 1993). Free-living robins, unlike captive robins, expend energy foraging for prey, avoiding predators, defending territories, etc. As a result, an
 allometric modeling approach, described below, was used to estimate food intake rate for
 American robins rather than the rates measured in the controlled study.

4 Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating the metabolic
5 rate of passerine birds using the following general equation:

6

$$FMR (kJ/day) = a x BW(g)^{b}$$
(Eq. 3)

The slope (*a*) and power (*b*) distributions were based on the error statistics reported in Nagy et al. (1999) and our own analyses of the raw data, assuming an underlying normal distribution for each. For passerine birds, the mean slope term for *log a* is equal to 1.02 with a standard error of 0.0883 in log_{10} units. The power term *b* had a reported mean of 0.680 and a standard error of 0.0682. The body weight (*BW*) distribution was described above. The results of the calculation were then converted to kcal/kg bw/d.

13 Food intake rate is derived from *FMR* using the following equation:

14
$$FIR(kg/kg bw/d) = \frac{FMR}{AE \cdot GE}$$
 (Eq. 4)

15 where AE is the assimilation efficiency of invertebrates (unitless) by birds and GE is the gross 16 energy of invertebrates (kcal/g). The gross energies of various wildlife food sources are 17 summarized in the Wildlife Exposure Factors Handbook (EPA 1993). For tree swallows, the mean assimilation efficiency was 77% (SD = 8.4%)(Karasov 1990; EPA 1993). The mean gross 18 19 energy for grasshoppers and crickets is 17 kcal/g (SD = 260) (Cummins and Wuycheck 1971; 20 Collopy 1975; Bell 1990), and for adult beetles, the mean is 1.5 kcal/g (Cummins and Wuycheck 21 1971; Collopy 1975; Bell 1990). Grasshoppers, crickets, and beetles were used as 22 representatives of emergent aquatic insects; their mean gross energy is 1.6 kcal/g. For American 23 robins, mean assimilation efficiencies were 72% for both earthworms and litter invertebrates, and 24 the mean gross energies were 0.81 kcal/g wet weight for earthworms and 1.6 kcal/g wet weight 25 for litter invertebrates. Point estimates were used for these variables in the Monte Carlo and 26 probability bounds analyses because of their relatively small coefficients of variation (i.e., CV 27 <10%). As a result, these input variables are not included in Figures 7.3-2, 7.3-3, and 7.3-4.

1 Microexposure Model

The allometric relationship for food intake rate was modeled as a function of body weight. This was recalculated as the microexposure model was stepped forward in time over the life of the swallow. For each day, the corresponding point estimate of body weight (in the case of the Monte Carlo analysis) or interval estimate of body weight (in the case of the probability bounds analysis) was substituted into the allometric equation for food intake rate. As with the intake rates used in the TDI model, the allometric model equation variables (the slope and power terms) were modeled as normally distributed.

9 Proportion of Dietary Items (P_i)

10 An analysis of the diet delivered to swallow nestlings indicated that it consisted of Diptera 11 (45.9%), mayflies (15.6%), and other insects (8.7%) by number, and Diptera (41.8%), mayflies 12 (21.3%), and moths and butterflies (9.2%) by total dry mass (Blancher and McNicol 1991). A 13 separate study also showed that mayflies and Diptera were common prey for swallows 14 (Robertson et al. 1992). Consumption of contaminated aquatic insects is presumed to be the 15 primary route of exposure of swallows to tPCBs. Direct stomach content samples were taken 16 from birds at nest sites within the Housatonic River PSA and at reference sites (Custer 2002). 17 These samples were used as the primary source of contaminant input concentrations for the 18 exposure models developed in this assessment.

19 American robin diets for the spring and summer were used in this assessment, because the focus 20 was on estimating reproductive effects to robins. Proportions of each prey item in the diet were 21 assumed to follow a beta distribution in the Monte Carlo analysis. For robins, the available 22 literature indicates that earthworms comprise about 15% of the diet on average during the spring 23 and summer, but may range from 10 to 20%. Litter invertebrates generally comprise about 60% 24 of the diet during spring and summer, with an approximate range of 45 to 75%. The proportion 25 of fruit in the robin diet during spring and summer was calculated as one minus the total of 26 earthworms and litter invertebrates.

1 Maternal Transfer (TOT_MT)

2 Maternal transfer refers to the total amount of COCs (in µg) transferred from the mother to the 3 offspring via egg tissue. Maternal transfer was estimated at each site using the concentrations of 4 COCs in the tissues of the newborn swallows (pippers) and egg samples. Table G.2-6 5 summarizes the ratios of the means of tPCB concentrations in eggs and pippers (due to maternal 6 transfer only) to the tPCB concentrations in nestlings (aged 12 to 14 days). Low ratios indicate 7 that the majority of tPCB tissue concentrations originated from feeding activity at the site. 8 Ratios approaching or greater than one indicate that the tPCB content was primarily due to 9 maternal transfer, and not from feeding locally over the period from birth to 14 days.¹ High 10 ratios of tPCB concentrations in pippers and eggs to total concentrations in nestlings occurred at 11 sites expected to be relatively uncontaminated locations. Those locations included Threemile 12 Pond, Taconic Valley, and Southwest Branch. At these locations, the ratios ranged from 0.57 at the Taconic Valley in 1999 to 1.89 at Threemile Pond in 2000. In contrast, the ratios at the more 13 14 contaminated locations ranged from 0.13 at Roaring Brook in 1998 to 0.60 at the same location 15 in 1999 (see Table G.2-6).

For the Monte Carlo simulation, maternal transfer was assumed to have a lognormal distribution, with location-specific mean and standard deviation. For the probability bounds analysis, probability bounds were derived using the site-specific lower 95% confidence limit (LCL) and the upper 95% confidence limit (UCL) around the means calculated using the Land H-statistic.

20 Concentrations of COCs in Food Items (C_f)

The concentrations of tPCBs and TEQ in the dietary items of insectivorous birds are illustrated in Figures 7.3-5 to 7.3-8. In these figures, the box and star symbols depict the median and arithmetic mean concentration of each COC in each dietary item in each of the areas for the risk assessment. The vertical line depicts the interquartile range for the concentration. The concentrations of COCs used in the exposure analyses are shown in Tables G.2-7, G.2-8, G.2-27, and G.2-28 of Appendix G. Total PCB concentrations in the prey of tree swallows are similar at

¹ Ratios greater than one, observed at Southwest Branch in 1998, Threemile Pond in 1999 and 2000, and Taconic Valley in 2000, would seem to indicate depuration or growth dilution over the 14 days of growth. These ratios are considered indicative of very high proportions of maternal transfer relative to intake of local contaminants.

Holmes Road, New Lenox Road, and Roaring Brook. All of the reference locations have lower
tPCB concentrations than sites in the PSA. TEQ concentrations in prey items of tree swallows
are highest at Holmes Road and lowest at the Taconic Valley reference area. Both tPCB and
TEQ concentrations in the prey items of American robins are highest at Location 13 and lowest
at Location 15.

6 Consumption of contaminated emergent aquatic insects is presumed to be the primary route of 7 exposure of swallows to tPCBs. Soil, water, and sediment exposure was not considered because 8 tree swallow exposure via these pathways was determined to be extremely limited. Both the TDI 9 model and the microexposure model used stomach content data collected from juvenile swallows during the Custer (2002) study. The microexposure model also used tissue data to estimate the 10 11 proportion of tPCB and TEQ concentrations in offspring tissue due to maternal transfer. Available data on contaminant concentrations in benthic invertebrates were not used because of 12 the availability of stomach content samples, which provide a more direct measure of 13 14 concentrations of COCs in tree swallow food.

15 Direct stomach content samples were taken from birds at nest sites within the Housatonic River 16 PSA and at reference sites. Stomach content samples were collected in 1998, 1999, and 2000: analytical results are included in Custer (2002). The median concentration of tPCBs in stomach 17 contents measured at Holmes Road was 3.24 mg/kg. The 25th and 75th percentiles were 2.56 and 18 10.7 mg/kg, respectively. The median, 25th, and 75th percentile concentrations of TEQ were 996, 19 20 669, and 1,324 ng/kg, respectively. Median tPCB concentrations in stomach contents at Holmes 21 Road, New Lenox Road, and Roaring Brook were approximately one order of magnitude higher 22 than at reference locations, except for Taconic Valley, which had comparable concentrations to 23 the PSA locations. Median TEQ concentrations in stomach contents at all locations, including 24 reference locations, were in the same order of magnitude. Statistics for concentrations of tPCBs 25 and TEQ in stomach contents at the six locations are shown in Tables G.2-7 and G.2-8.



2 Figure 7.3-5 Concentration of tPCBs in Prey of Tree Swallows


2 Figure 7.3-6 Concentration of TEQ in Prey of Tree Swallows



2 Figure 7.3-7 Concentration of tPCBs in Prey of American Robins



2 Figure 7.3-8 Concentration of TEQ in Prey of American Robins

3 7.3.2 TDI Model Results

Exposure distributions for the exposure of tPCBs and TEQ to tree swallows at Holmes Road,
New Lenox Road, Roaring Brook Road, Southwest Branch, Threemile Pond, and Taconic
Valley, and to American robins at Locations 13, 14 and 15 are presented in Figures 7.3-9 to 7.326.

8 The exposure distribution for tree swallows exposed to tPCBs at Holmes Road is presented in 9 Figure 7.3-9. The Monte Carlo analysis indicated that exposure of tree swallows to tPCBs could 10 range from a minimum of 6.87 to a maximum of 63.0 mg/kg bw/d. The mean exposure was 22.9 11 mg/kg bw/d, and the median exposure was 22.0 mg/kg bw/d; 80% of the exposure estimates 12 were between 15.3 and 32.1 mg/kg bw/d (Table G.2-9).



New Lenox Road







5 Figure 7.3-11 Tree Swallow TDI Exposure Model for tPCBs: Results of Monte 6 Carlo Analysis and Probability Bounds Analysis at Roaring Brook

7

1

2

3

Southwest Branch



Figure 7.3-12 Figure 7.3-12 Tree Swallow TDI Exposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Southwest Branch

8

1

2

3

Threemile Pond



- Pond



Holmes Road



5 Figure 7.3-15 Tree Swallow TDI Exposure Model for TEQ: Results of Monte 6 Carlo Analysis and Probability Bounds Analysis at Holmes Road

7

1

2

3



Figure 7.3-16 Figure 7.3-16 Tree Swallow TDI Exposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at New Lenox Road

8

1

2

3



7/10/2003

Southwest Branch



Figure 7.3-18 Figure 7.3-18 Tree Swallow TDI Exposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Southwest Branch

8

1

2

3



- Pond
- 8

7



5 Figure 7.3-20 Tree Swallow TDI Exposure Model for TEQ: Results of Monte 6 Carlo Analysis and Probability Bounds Analysis at Taconic Valley





5 Figure 7.3-22 Exposure of American Robins to tPCBs at Site 14 of the 6 Housatonic River PSA









2 Notes:

3 LPB = Lower probability bound

4 UPB = Upper probability bound

5 Figure 7.3-26 Exposure of American Robins to 2,3,7,8-TCDD TEQ at Site 15 of 6 the Housatonic River PSA

The probability bounds estimated for tree swallows foraging at Holmes Road are depicted in Figure 7.3-9. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 1.04 and 18.2 mg/kg bw/d. The 50th percentile ranged between 1.27 and 22.1 mg/kg bw/d, and the 90th percentile ranged between 1.81 and 32.3 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 15.3, the 50th percentile was 22.0, and the 90th percentile was 32.1 mg/kg bw/d (Table G.2-9).

Exposures of tree swallows to tPCBs at two other PSA locations, New Lenox Road and Roaring Brook, were lower than at Holmes Road, having mean total daily intakes of 11.0 and 13.3 mg/kg bw/d, respectively (Table G.2-9). Exposures at the reference locations, Southwest Branch and Threemile Pond, were very low with mean total daily intakes of 0.73 and 1.15 mg/kg bw/d. The third reference location, Taconic Valley, had relatively high exposure with a mean of 14.2 mg/kg bw/d. The highest concentrations of TEQ were at Holmes Road with a mean of 1,227 ng/kg bw/d. New Lenox Road and Roaring Brook had mean total daily intakes of 701 and 681 ng/kg bw/d, respectively (Table G.2-10). Mean exposure concentrations at the three reference
 locations ranged from 396 to 866 ng/kg bw/d.

American robins had the highest exposure to tPCBs and TEQ at Location 13, with mean concentrations of 5.66 mg/kg bw/d and 82.3 ng/kg bw/d, respectively. Exposure at Locations 14 and 15 was somewhat lower for both tPCBs and TEQ. Mean tPCB concentrations at Locations 14 and 15 were 4.69 and 1.22 mg/kg bw/d, respectively. Mean TEQ concentrations at Locations 14 and 15 were 57.5 and 33.0 ng/kg bw/d, respectively.

8 7.3.3 Microexposure Model Results

9 Exposure distributions for exposure of tree swallows to tPCBs and TEQ at Holmes Road, New
10 Lenox Road, Roaring Brook, Southwest Branch, Threemile Pond, and Taconic Valley are
11 presented in Figures 7.3-27 to 7.3-38.

Figure 7.3-27 depicts the cumulative distribution of tPCB intake rates for tree swallows at Holmes Road. The Monte Carlo analysis indicated that accumulation of tPCBs by tree swallows at Holmes Road could range from a minimum of 75.8 mg/kg in tissues of 14 d nestlings (5.06 mg/kg bw/d)¹ to a maximum of 595 mg/kg (39.7 mg/kg bw/d). The mean exposure was 222 mg/kg (14.8 mg/kg bw/d), and the median exposure was 215 mg/kg (14.3 mg/kg bw/d); 80% of the exposure estimates were between 154 (10.3) and 253 (15.6) mg/kg.

¹ Estimated by dividing tissue residues by 15, which was the number of days included in the microexposure model.

Holmes Road



5Figure 7.3-27Tree Swallow Microexposure Model for tPCBs: Results of Monte6Carlo Analysis and Probability Bounds Analysis at Holmes Road

1

2

3

New Lenox Road



Carlo Analysis and Probability Bounds Analysis at New Lenox

Roaring Brook



5 6 Carlo Analysis and Probability Bounds Analysis at Roaring Brook

7

1

2

3

Southwest Branch



1

2 Notes:

3 LPB = Lower probability bound

4 UPB = Upper probability bound

Figure 7.3-30 Tree Swallow Microexposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Southwest Branch

Threemile Pond



1

2 Notes:

- 3 LPB = Lower probability bound
- 4 UPB = Upper probability bound

Figure 7.3-31 Tree Swallow Microexposure Model for tPCBs: Results of Monte Carlo Analysis and Probability Bounds Analysis at Threemile Pond

Taconic Valley



2 Notes:

1

3 LPB = Lower

3 LPB = Lower probability bound

4 UPB = Upper probability bound

5Figure 7.3-32Tree Swallow Microexposure Model for tPCBs: Results of Monte6Carlo Analysis and Probability Bounds Analysis at Taconic Valley

Holmes Road



- 1
- 2 Notes:
- 3 LPB = Lower probability bound
- 4 UPB = Upper probability bound

5Figure 7.3-33Tree Swallow Microexposure Model for TEQ: Results of Monte6Carlo Analysis and Probability Bounds Analysis at Holmes Road

New Lenox Road



Tissue Residue (ng/kg)

1

2 Notes:

3 LPB = Lower probability bound

4 UPB = Upper probability bound

Tree Swallow Microexposure Model for TEQ: Results of Monte 5 Figure 7.3-34 6 Carlo Analysis and Probability Bounds Analysis at New Lenox Road 7

Roaring Brook



- 1
- 2 Notes:
- 3 LPB = Lower probability bound
- 4 UPB = Upper probability bound

5 Figure 7.3-35 Tree Swallow Microexposure Model for TEQ: Results of Monte 6 Carlo Analysis and Probability Bounds Analysis at Roaring Brook

Southwest Branch



Threemile Pond



- 1
- 2 Notes:
- 3 LPB = Lower probability bound
- 4 UPB = Upper probability bound

Figure 7.3-37 Figure 7.3-37 Tree Swallow Microexposure Model for TEQ: Results of Monte Carlo Analysis and Probability Bounds Analysis at Threemile Pond



1

2 Notes:

3 LPB = Lower probability bound

4 UPB = Upper probability bound

5Figure 7.3-38Tree Swallow Microexposure Model for TEQ: Results of Monte6Carlo Analysis and Probability Bounds Analysis at Taconic Valley

7 The probability bounds estimated for tree swallows foraging at Holmes Road are depicted in Figure 7.3-27. The 10th percentile of the probability envelope formed by the lower and upper 8 9 bounds ranged between 11.5 mg/kg (0.77 mg/kg bw/d) and 215 mg/kg (14.3 mg/kg bw/d). The 10 50th percentile ranged between 12.6 mg/kg (0.84 mg/kg bw/d) and 227 mg/kg (15.2 mg/kg bw/d), and the 90th percentile ranged between 14.4 mg/kg (0.96 mg/kg bw/d) and 273 mg/kg 11 (18.2 mg/kg bw/d). In comparison, the 10th percentile of the Monte Carlo output was 154 mg/kg 12 (10.3 mg/kg bw/d), the 50th percentile was 215 mg/kg (14.3 mg/kg bw/d), and the 90th percentile 13 14 was 253 mg/kg (15.6 mg/kg bw/d).

Accumulation of tPCBs by tree swallows at the other two PSA sites, New Lenox Road and 1 2 Roaring Brook, was lower than at Holmes Road, having mean tissue residues of 109 mg/kg (7.26 3 mg/kg bw/d) and 133 mg/kg (8.85 mg/kg bw/d), respectively (Tables G.2-13 and G.2-14). 4 Accumulation at the reference locations at Southwest Branch and Threemile Pond was very low 5 with mean tissue residues of 8.44 mg/kg (0.56 mg/kg bw/d) and 11.7 mg/kg (0.78 mg/kg bw/d). 6 The third reference location, Taconic Valley, had a relatively high accumulation of tPCBs with a 7 mean of 137 mg/kg (9.10 mg/kg bw/d). The highest tissue residues of TEQ were at Holmes 8 Road with a mean of 11,580 ng/kg (772 ng/kg bw/d). New Lenox Road and Roaring Brook had 9 mean tissue residues of 6,644 ng/kg (443 ng/kg bw/d) and 6,466 ng/kg (431 ng/kg bw/d), 10 respectively (Tables G.2-15 and G.2-16). Mean tissue residues at the three reference locations 11 ranged from 3,783 ng/kg (252 ng/kg bw/d) to 8,224 ng/kg (548 ng/kg bw/d). The results of the 12 microexposure model, when converted to daily intake units of tPCBs, correspond fairly closely 13 with the results of the TDI models.

14 **7.3.4** Tree Swallow Tissue Data from Field Study

15 7.3.4.1 Tissue Residue Data

Analyses of the COC concentrations in tree swallow tissues provided direct measures of tPCB and TEQ exposure. Tissue samples were collected from eggs or just hatched young (herein referred to as pippers) and 12- to 14-day-old nestlings in 1998 at four sites and in 1999 and 2000 at six sites (Custer 2002). These samples provided tPCBs concentrations data for pippers and 12- to 14- day old nestlings (i.e., pre-migratory full-grown birds) and TEQ concentrations for pippers.

22 7.3.4.2 12- to 14-day-old Bird Tissue Concentrations

The microexposure model estimated the whole body concentrations of tPCBs in 14-day-old nestlings. These estimates can be compared directly to the tPCB concentrations measured in 12to 14-day-old swallow tissue samples in 1998, 1999, and 2000 in the Custer (2002) study. Summary statistics by location are in Table G.2-20 (Custer 2002). The highest median concentrations, observed at Holmes Road, New Lenox Road, and Roaring Brook, ranged from 21.5 to 36.0 mg/kg ww during the 3-year period. Median concentrations at the upstream and the other reference locations (Threemile Pond) ranged from 0.77 to 3.30 mg/kg ww. Total PCB
concentrations typically found in the nestling tissue data at Holmes Road, New Lenox Road, and
Roaring Brook indicated that there was an accumulation above the concentrations found in the
eggs and pippers at these locations.

5 The nestling tissue data were compared to the results of the microexposure model directly 6 because the microexposure model estimated whole body concentrations of tPCBs (mg/kg) for 7 nestlings aged 14 days and the tissue samples were taken from nestlings aged 12 to 14 days. 8 Because of the maternal transfer term in the microexposure model, the tissue data and the 9 modeled estimates for exposure are not independent. The Kolmogorov-Smirnov test for 10 goodness of fit (Miller 1956) was used to compare the cumulative frequency distributions of 11 tPCBs estimated by the microexposure model and those observed in the tissue sample data at 12 each location. For all locations, the measured tissue concentration distributions were 13 significantly lower than the distributions estimated by the Monte Carlo analysis of the 14 microexposure model. This is likely partially due to the dissimilar exposure durations between 15 the location-specific data and modeled data. However, all measured distributions were within 16 the microexposure model probability bounds, as expected.

17 7.3.4.3 Pipper Tissue Concentrations

18 Total PCBs

Total PCB concentrations were generally higher in tree swallow pippers from locations in the Housatonic River PSA than at the reference locations (Table G.2-22). Median concentrations in pippers ranged from 44.9 to 80.2 mg/kg ww. In comparison, median concentrations ranged from 7.5 to 14.4 mg/kg ww in pippers at the reference sites. Concentrations of tPCBs were generally consistent among years at each site. However, pippers at Roaring Brook had higher concentrations in 1999 than in 1998. Also, pippers from Southwest Branch had lower tPCB concentrations in 2000 than in 1998 and 1999 (Custer 2002).
1 **TEQ**

Concentrations of TEQ were highest at sites in the Housatonic River PSA. The median
concentrations in pipper samples ranged from 1,390 ng/kg ww at Holmes Road to 2,890 ng/kg at
Roaring Brook. In comparison, median concentrations ranged from 562 to 730 ng/kg ww in
pippers at the reference sites (Table G.2-23) (Custer 2002).

1 7.4 EFFECTS ASSESSMENT

The effects assessment has two objectives. The first is to review the scientific literature for effects of tPCBs (mainly Aroclor 1254 and 1260 mixtures) and TEQ to insectivorous birds. Of primary interest are documented effects to the representative species in this assessment: tree swallows and American robins. In the absence of data for these species, other avian species are considered. The other objective is to derive the effects metrics that will be used, in conjunction with the exposure assessment results, to estimate risks to insectivorous birds from exposure to COCs in the Housatonic River PSA.

9 This section focuses on effects that have an influence on the long-term maintenance of bird 10 populations (i.e., mortality or impairment of reproduction or growth). Studies involving multiple 11 exposure treatments and where reported results were evaluated statistically to identify significant 12 differences from controls are preferred.

The COCs in this assessment are tPCBs and 2,3,7,8-TCDD TEQ. The congeners used to estimate TEQ concentration share the ability to bind to the Ah receptor protein (Bosveld and Van den Berg 1994) and elicit an Ah-mediated biochemical and toxic response. The toxic response to this group of chemicals is related to the three-dimensional structure of the molecule, including the degree of chlorination and positions of the chlorine on the aromatic frame (Van den Berg et al. 1998; Newsted et al. 1995; Safe 1994). Figures 7.4-1 and 7.4-2 illustrate the ranges of effects of tPCBs and TEQ, respectively, to various avian species.

20 Sensitivities of avian species to tPCBs and TEQ have been shown to vary greatly in literature 21 studies. Wild turkey embryos were found to be 20 to 100 times less sensitive than chicken 22 embryos to egg yolk injection of PCB-77. This difference in toxicity may be attributed to 23 differences in the availability of Ah receptors. Ah receptors were found in hepatocytes of 7-day-24 old chicken embryos, but not in liver cells of 9-day-old turkey embryos (Brunstrom and Lund 25 1988). Presented below is a brief review of the scientific literature for both field-based studies 26 on tree swallow exposure to tPCBs and the laboratory studies used to derive the threshold range 27 for TEQ for insectivorous birds, and tPCBs for American robin.



1 2 3

Figure 7.4-1 Effects of Aroclor 1254/1260 on Avian Species (mg/kg bw/d)



1 2

Figure 7.4-2 Effects of 2,3,7,8-TCDD TEQ on Avian Species (ng TEQ/kg bw/d)

1	Toxicity of tPCBs and TEQ to Avian Species				
2	Mode of Action				
3 4	Bind to the aryl-hydrocarbon (Ah) receptor, eliciting an Ah-receptor-mediated biochemical and toxic response.				
5	Types of Toxicity	Specific Effects			
6 7 8 9 10 11	hepatotoxicity immunotoxicity neurotoxicity embryotoxicity teratogenicity	mortality decreased growth weight loss porphyria reduced hatching embryo mortality			

12 7.4.1 Review of Effects of tPCBs

Heath et al. (1972) studied the effects of Aroclor 1254 on mortality of four avian species. The most sensitive (after oral dosing for 5 days with Aroclor 1254) was the bobwhite quail, with a median lethality response occurring at a dietary PCB concentration of 604 mg/kg. Other species tested, such as the Japanese quail, mallard duck, and ring-necked pheasant, were less sensitive, with oral LC_{50} s of 2,898, 2,699, and 1,091 mg/kg diet, respectively. Prestt et al. (1970) estimated the median lethal dietary dose rate of Aroclor 1254 to adult Bengalese finches to be 256 mg/kg/d.

20 Reproductive impairment of birds caused by tPCBs has been investigated in several species, in 21 dietary and egg injection studies, as well as field studies examining egg and hatchling 22 concentrations and hatching success. The most commonly noted effects to the reproduction of 23 avian species are reduced egg productivity, egg hatchability, and chick growth rates (CCME 24 1999). Of the species studied, chickens appear to be the most sensitive, followed by pheasants, 25 turkeys, ducks, and herring gulls (Bosveld and Van den Berg 1994). Total PCBs appear to have 26 no adverse effects on total egg weight, eggshell weight, or eggshell thickness (Lillie et al. 1974; 27 Britton and Huston 1973; Scott 1977). Lillie et al. (1974) exposed hens to 2 mg/kg Aroclor 1254 28 for 63 days in feed, giving birds a daily PCB dose of approximately 0.12 mg/kg bw/d. At this 29 treatment level, no significant effects were observed on fertility, egg production, shell thickness, 30 or hatchability, but the growth rate of chicks was slightly reduced. American kestrels were given 31 an approximate tPCB dose of 7 mg/kg bw/d for 100 days (Fernie et al. 2001) and birds

experienced a slight, but statistically insignificant, decrease in clutch size and the numbers of
 fertile eggs, hatchlings, and fledglings per breeding pair.

3 Large clutch sizes have been reported in other populations of bird species exposed to chemical 4 contamination (McCarty and Secord 1999a). The long-term impact of this effect is unknown, 5 but is considered an indicator of disturbed reproductive biology. McCarty and Secord (1999a) 6 indicated that the overall reproductive success of tree swallows along the Hudson River, which is 7 contaminated by PCBs, was generally low. Hatchability of eggs was among the lowest reported 8 for this species, nest abandonment was higher than expected, and the number of large clutches 9 was high. Additionally, nest quality based on nest weight and number of feathers lining the nest 10 was low relative to control sites (McCarty and Secord 1999b). Concentrations of tPCBs in 11 nestlings were as high as 62.2 mg/kg. Overall nest quality can have an important impact on 12 length of incubation, length of nestling period, nestling mass, and number of fledglings produced (McCarty and Secord 1999b). The reference data for this study were collected from an area 13 14 >230 km away from the Hudson River study area and 3 years previous (1990-91) (Custer 2002). 15 Comparing reproductive results from different years can be misleading (Custer 2002).

A field study conducted for GE examined the effects of ecological stressors (i.e., nest box density) on tree swallow reproduction (Robertson and Jones 2002). The study took place 50 km north of Kingston, Ontario, Canada. Ecological factors considered in the study included, but were not limited to, nest spacing and proximity to forest or shrub edge. The study was not considered in this risk assessment because the study did not evaluate the effects of tPCBs or TEQ on tree swallow reproduction; therefore, it was unrelated to the assessment endpoint.

22 7.4.2 2,3,7,8-TCDD and Equivalence (TEQ)

In single, oral doses of TCDD, bobwhite quail, mallards, and ringed turtledoves were found to have 37-day LD_{50} s of 15,000, 108,000, and 810,000 ng/kg bw (Hudson et al. 1984). Ringnecked pheasants given intraperitoneal TCDD doses weekly of 10, 100, or 1,000 ng/kg bw/week for 10 weeks (Nosek et al. 1992) experienced no mortality or body weight effects in the two lowest treatment groups, but the 1,000 ng/kg bw/week treatment group experienced 60% mortality by the 23rd week (13 weeks after the dosing period).

1 Nosek et al. (1992) also investigated reproductive effects of TCDD to ring-necked pheasant 2 hens. The two lower doses (10 and 100 ng/kg bw/week) caused no significant impairment to egg 3 production. The highest dose, 1,000 ng/kg bw/week, caused a decrease in cumulative egg production of approximately 70% over 7 weeks. The geometric mean of the LOAEL and 4 5 NOAEL from this study was 44 ng/kg bw/d. Hoffman et al. (1996) investigated the 6 developmental effects of TEQ on American kestrels and observed that skeletal growth was 7 significantly reduced at treatment levels of 25,000 ng TEQ/kg bw/d. This dose did not translate 8 into significant effects on hatchling success or weight gain.

9 7.4.3 Tree Swallow Field Study

The tree swallow reproduction field study was conducted between 1998 and 2000 in the PSA and reference areas. The study focused on nest box occupancy rate, clutch size, nesting success, and determination of tissue concentrations of tPCBs and TEQ.

13 Methods

14 Nest boxes were deployed in 1998 at four locations along the Housatonic River and its tributaries 15 and in 1999 at two additional reference locations. Nest boxes were placed on poles in early 16 spring and monitored thereafter for reproductive success. Samples of eggs or just hatched young (pippers) were collected for organochlorine contaminant analyses. Twelve-day-old nestlings 17 were collected from selected sites to quantify concentration and accumulation rates in nestlings 18 19 and to assay EROD activity. Stomach content samples were taken from birds in 1998, 1999, and 20 2000 (Custer 2002). EROD activity analysis of tPCBs and PCB congeners was conducted using standard methods. 2,3,7,8-TCDD equivalents (TEQ) were calculated using the toxic equivalency 21 22 factors (TEFs) of Van den Berg et al. (1998).

23 Results

Tree swallows nested at all study locations in the Housatonic River watershed. Nesting attempts increased from the first to second year and stabilized in the third year. Average clutch sizes in 1998, 1999, and 2000 were 5.43, 5.37, and 5.46, respectively. There was a significant negative relationship between tPCB concentrations in eggs and hatching success in 1999 (p = 0.044); however, the fit of the model was poor. In 1998 and 1999, clutches that contained dead embryos
had significantly higher concentrations of tPCBs than those that hatched normally (p < 0.001).
The geometric mean concentrations in clutches that had reduced hatching were 62.8 mg/kg ww
in eggs in 1998 and 69.1 mg/kg ww in eggs in 1999.

5 Differences in the geometric mean concentrations of dioxins and furans between sites were 6 similar to the pattern for tPCBs. EROD activity was significantly induced at sites in the 7 Housatonic River PSA compared to Threemile Pond.

8 **Conclusions**

9 The fecundity of tree swallows in the PSA was not severely impacted by contaminants. In 1998 10 and 1999, clutches with dead embryos had geometric means of 62.8 and 69.1 mg/kg ww tPCB in 11 eggs. These concentrations exceeded the field-based threshold of 62.2 mg/kg ww tPCBs in eggs 12 established from the studies by McCarty and Secord (1999a, 1999b) and Secord et al. (1999). 13 Multivariate regression models indicated that dioxins and furans in the PSA could be 14 contributing to the observed reduced hatching success.

15 7.4.4 American Robin Field Study (GE)

The reproductive output of American robins was studied in the PSA and reference areas during the 2001 breeding season (Henning 2002). The objective of the study was to evaluate the relationship between reproductive success and tissue concentrations of tPCBs in eggs and young.

19 Methods

The study was conducted in the Housatonic River 10-year floodplain and in nearby public and private lands. Active robin nests (having eggs or a fresh, wet mud lining) were monitored every three days to record the number of eggs and hatchlings, as well as depredation, abandonment, parental behavior, and development of young. Eggs and nestlings were analyzed for tPCB concentrations. A nest was deemed "successful" if at least one young fledged. Clutch size, hatching success, and fledging success were determined for successful nests.

1 Results

Clutch size averaged 2.91 eggs per nest in the PSA and 2.87 eggs per nest in the reference areas.
Clutch sizes in the PSA and reference areas were not significantly different according to Tukey's studentized range test. Mean hatching success rates were 0.81 hatchlings per nest in the PSA and 0.89 in the reference areas, and mean fledging success rates were 0.61 in the PSA and 0.62 in the reference areas. The differences were not significant according to Tukey's studentized range test.

8 Total PCB concentrations in robin eggs averaged 83.6 mg/kg ww in the PSA (n=9) and 0.153 9 mg/kg ww in the reference areas (n=2). Only nests from which egg samples were taken for tPCB 10 analysis were included in the test. The test indicated no relationship between tPCB 11 concentrations in eggs and clutch size.

12 Conclusions

Concentrations of tPCBs in American robin eggs and nestlings were significantly higher in the PSA compared to reference areas, with tPCB concentrations in robin eggs averaging 83.6 mg/kg ww in the PSA (n=9) and 0.153 mg/kg ww in the reference areas (n=2). There were, however, no significant differences in any of the measures of effects on reproduction included in this study. Further, clutch size, hatching success, and fledging success in the target and reference areas were within ranges reported for American robins (Brehmer and Anderson 1992; Kemper and Taylor 1981; Fluetsch and Sparling 1994).

20 7.4.5 Selection of Effects Metrics for Characterizing Risk

Effects data can be characterized and summarized in a variety of ways ranging from benchmarks designed to be protective of most or all species to concentration- or dose-response curves. A summary of the decision criteria used to derive effects metrics is provided in the text box. Further details on the decision criteria used in selecting effects metrics is provided in Section 6.6 of the ERA.

2	Decision Criteria for Derivation of Effects Metric					
3 4	The following is the hierarchy of decision criteria used to characterize effects for each receptor-COC combination:					
5 6 7	1. Have single-study bioassays with five or more treatments been conducted on the receptor of interest or a reasonable surrogate? If yes, estimate the concentration- or dose-response. If not, go to 2.					
8 9 0 1	 Are multiple bioassays with similar protocols, exposure scenarios, and effects metrics available that, when combined, have five or more treatments for the receptor of interest or a reasonable surrogate? If yes, estimate the dose- response relationship as in 1. If not, go to 3. 					
12 13 14	 Have bioassays with less than five treatments been conducted on the receptor of interest or a reasonable surrogate? If yes, conduct or report results of hypothesis testing to determine the NOAEL and LOAEL. If not, go to 4. 					
15 16 17 18	4. Are sufficient data available from field studies and monitoring programs to estimate concentrations or doses of the COC that are consistently protective or associated with adverse effects? If yes, develop field-based effects metrics. If not, go to 5.					
19 20 21 22 23 24 25	5. Derive a range where the threshold for the receptor of interest is expected to occur. Because information on the sensitivity of the receptor of interest is lacking, it is difficult to derive a threshold that is biased neither high nor low. If bioassay data are available for several other species, however, calculate a threshold for each to determine a threshold range that spans sensitive and tolerant species. That range is likely to include the threshold for the receptor of interest.					

26

1

Although much work has been done to investigate the effects of tPCBs and TEQ to various bird species in controlled laboratory settings, no suitable studies were available for tree swallows, American robins, or for bird species that could be considered reasonable surrogates. Therefore, no dose-response relationship could be established between exposure of tree swallows or American robins to tPCBs (either Aroclor 1254 or 1260) or TEQ and adverse effects on mortality, growth, or reproduction. It was also not possible to establish a NOAEL or LOAEL for adverse effects from available laboratory studies.

34 **7.4.5.1** Effects of tPCBs to Tree Swallows

Based on the results of the field-based effects studies reviewed, a field-based threshold range was derived for tree swallows exposed to tPCBs. The low threshold was based on the McCarty

and Secord (1999b) study where tPCB concentrations in whole body nestlings were 62.2 mg/kg 1 2 ww. At this concentration, reproductive effects were observed (e.g., abnormal nest abandonment 3 behavior, larger clutch sizes), but the ecological significance of these effects could not be 4 determined. The high effects threshold was based on the field study conducted by Custer (2002) 5 where tree swallow pipper tissue concentrations of 69 mg/kg ww were associated with hatching 6 problems. This value also has uncertainty as to whether it caused adverse effects to tree swallow 7 hatching success in the PSA (Custer 2002). Thus, despite the narrowness of the tPCB threshold 8 range, there is some uncertainty about this effects metric. In addition, field studies are subject to 9 a number of factors that are impossible to control, including weather, predation, disease, and 10 presence of other COPCs. Therefore, the effects threshold range for insectivorous birds for 11 tPCBs is 62.2 to 69 mg/kg ww.

12 7.4.5.2 Effects of tPCBs to American Robins

Based on a review of avian toxicity literature, white leghorn chickens were the most sensitive avian species to the reproductive effects of tPCBs and the most reproductively tolerant avian species to tPCBs was the American kestrel. The threshold range for the reproductive success of American robins exposed to tPCBs selected for this assessment is 0.12 to 7.0 mg/kg bw/d based on reproductive studies conducted on white leghorn chickens (Lillie et al. 1974) and American kestrels (Fernie et al. 2001), respectively.

19 7.4.5.3 Effects of TEQ to Tree Swallows and American Robins

The low toxicological threshold for effects of TEQ to sensitive birds is based on the study by Nosek et al. (1992) using ring-necked pheasants. A dose of 44 ng/kg bw/d World Health Organization (WHO) TEQ (geometric mean of LOAEL and NOAEL) was assumed to be the threshold for sensitive avian species exposed to TEQ. The threshold for tolerant avian species was derived from Hoffman et al. (1996) where 25,000 ng/kg bw/d TEQ was determined to be the reproductive threshold for American kestrels exposed to TEQ. Thus, the effects threshold range is 44 to 25,000 ng/kg bw/d¹ for TEQ.

¹ A tissue residue threshold range was derived for TEQ by multiplying this threshold range by 15 days, which is the number of days included in the microexposure model. However, this threshold range was not used in the final assessment of risk to tree swallows because the TDI model results were used to estimate risk of TEQ to tree swallows.

1 7.5 RISK CHARACTERIZATION

This section characterizes risk to insectivorous birds exposed to tPCBs and 2,3,7,8-TCDD TEQ in the PSA of the Housatonic River. The risk characterization uses two lines of evidence to determine potential ecological risks to tree swallows and American robins. The major lines of evidence are considered to be independent and will be combined in a weight-of-evidence assessment. The key risk questions and the lines of evidence are summarized in the text box.

7 8

9

10

11

12

13

14

Key Risk Questions

- Are the concentrations of tPCBs and TEQ present in the prey of insectivorous birds sufficient to cause adverse effects to individuals inhabiting the PSA of the Housatonic River?
- If so, how severe are the risks and what are their potential consequences?
- Lines of Evidence
 - Field-based tree swallow and American robin reproduction studies.
 - Probabilistic exposure and effects modeling.
- 15

Section 7.5.1 compares the modeled exposure of tree swallows and American robins to the effects metrics. Sections 7.5.2 and 7.5.3 present brief overviews of the field studies conducted for tree swallows and American robins, respectively. A more detailed presentation of this information is presented in Appendix G. A weight-of-evidence assessment is presented in Section 7.5.4 along with the sources of uncertainty (Section 7.5.5), and the overall findings of the risk characterization and extrapolation to other species (Section 7.5.6).

22 7.5.1 Comparison of Modeled Exposures to Effects

For tree swallows, exposure was assessed separately in the three PSA locations of Holmes Road, New Lenox Road, and Roaring Brook, and in the three reference locations of Southwest Branch, Threemile Pond, and Taconic Valley. The probabilistic exposure analysis for tree swallows used a total daily intake rate modeling approach for calculating exposure to tPCBs and TEQ. In addition to this model, a microexposure model was also employed to estimate tissue concentrations of tPCBs and TEQ in juvenile birds after the first 2 weeks of their development. The results from the microexposure model were used to estimate risk for tPCBs and the TDI 1 model was used to estimate the risk for TEQ, to match the type of effect metric developed for

2 each COC.

3 Exposure of American robins was estimated in three areas (Locations 13, 14, and 15, see Figure

4 7.3-1) in the PSA. The probabilistic exposure analysis for American robins used a total daily

5 intake model to estimate exposure to tPCBs and TEQ.

For each COC-location combination, a category of low, intermediate, or high risk was assigned,
using the guidance below, when integrating the exposure and effects distributions.

G	Guidance for Integrating the Exposure and Effects Distributions					
ŀ	If the probability of exceeding the lower toxicity threshold is less than 20%, the risk is considered to be low.					
·	If the probability of exceeding the upper toxicity threshold is greater than 20%, the risk is considered to be high.					
•	All other outcomes are considered to have intermediate risk.					

14

This exercise was done separately for the results of the Monte Carlo analyses and the lower and upper bounds from the probability bounds analyses. The "risk category" refers to the level of risk based on the results of the Monte Carlo analyses. The "risk range" refers to the levels of risk based on the results of the probability bounds analyses.

19 The results of the risk characterization are summarized in Table 7.5-1. The highest risk to tree 20 swallows is from exposure to tPCBs in the PSA sites and in the Taconic Valley reference site, with low risk in the other two reference areas. As shown in Figure 7.3-27, the tPCB exposure 21 22 distribution from the Monte Carlo analysis in the Holmes Road location is above the upper 23 toxicity threshold. This means that the estimated exposure of tree swallows to tPCBs is greater 24 than the upper bound threshold for adverse effects to this species. The risk category for tree 25 swallows is high at the three PSA locations, and the risk range is low-high, indicating high 26 uncertainty. The risk category and the risk range are low for the Southwest Branch and 27 Threemile Pond reference locations; for the Taconic Valley reference location, the risk category 28 is high and the risk range is low-high (Table 7.5-1). The highest risk to American robins is from 29 exposure to tPCBs at Location 13. The risk category at Locations 14 and 15 is intermediate.

The risk range from exposure to tPCBs at Locations 13 and 14 is low-intermediate, and at
 Location 15 the risk range is intermediate.

Tree swallows are at intermediate risk from exposure to TEQ in the PSA and in the reference locations, with risk ranges of intermediate for all sites except Southwest Branch, which had a risk range of low-intermediate (Table 7.5-1). Similarly, American robins are at intermediate risk from exposure to TEQ at Locations 13 and 14, and are at low risk at Location 15. The risk range from exposure to TEQ at Location 13 is intermediate, and at Locations 14 and 15 the risk range is low/intermediate.

9 The complete characterization of risks of tPCBs and TEQ to insectivorous birds is presented in10 Appendix G.

11

12 13

14

Table 7.5-1

Summary of Qualitative Risk Statements for Insectivorous Birds from the Housatonic River Study Area

	Qualitative 1			isk Statements		
	tPCBs			TEQ		
Bird/Location	Risk Category Risk Range			Risk Category Risk Range		
Tree Swallow						
Holmes Road	High	Low/High		Intermediate	Intermediate	
New Lenox Road	High	Low/High		Intermediate	Intermediate	
Roaring Brook	High	Low/High	Intermediate Int		Intermediate	
Southwest Branch	Low	Low		Intermediate	Low/Intermediate	
Threemile Pond	Low	Low		Intermediate	Intermediate	
Taconic Valley	High	Low/High		Intermediate	Intermediate	
American Robin						
Location 13	High	Intermediate/High		Intermediate	Intermediate	
Location 14	Intermediate	Intermediate/High		Intermediate	Low/Intermediate	
Location 15	Intermediate	Intermediate		Low	Low/Intermediate	

1 7.5.2 Tree Swallow Field Study

2 Custer (2002) conducted a tree swallow reproduction study in the years 1998 through 2000 in the 3 PSA and reference areas. Nest boxes were placed at three locations in the PSA (Holmes Road, 4 New Lenox Road, and Roaring Brook) and at three reference locations (Southwest Branch, 5 Taconic Valley, and Threemile Pond in 1999 and 2000 only). Several indicators of reproductive 6 performance were monitored, including clutch size, nesting success, and determination of tissue 7 concentrations of tPCBs and TEQ in eggs. Pippers and 12- to 14-day-old nestlings were also 8 measured. The field study indicated that tree swallows did not experience serious adverse effects, 9 despite high tissue concentrations of tPCBs and TEQ in nestlings in the PSA locations.

In central Massachusetts, the mean clutch size was 4.8 to 5.3 eggs/clutch over a 22-year period (Chapman 1955). The mean clutch sizes for tree swallows in the PSA in 1998, 1999, and 2000 were 5.43, 5.37, and 5.46 eggs/clutch, respectively. Thus, the fecundity of tree swallows in the PSA was unaffected by tPCBs and TEQ. McCarty and Secord (1999a) similarly reported large clutch sizes in tree swallows exposed to PCBs in the Upper Hudson River watershed, NY.

15 The geometric means of tPCB concentrations in tree swallow pippers and nestlings collected 16 from the Housatonic River ranged from 32 to 101 mg/kg ww whole body. These are the highest 17 concentrations reported in the literature (Custer 2002) and are substantially higher than 18 concentrations in samples obtained from reference sites (6 to 19 mg/kg ww whole body). Total 19 PCBs, dioxins, and furans were negatively correlated with hatching success in 1998 and 1999, 20 but the correlations were weak. Hatching success was not correlated with these COCs in 2000, 21 probably because concentrations were reduced in 2000 and because cold weather contributed to 22 poor hatching at all locations.

23 7.5.3 American Robin Field Study (GE)

A study of American robin reproduction was performed during the 2001 breeding season in the PSA and reference areas. The study evaluated the relationship between tissue concentrations and reproductive output. The study was conducted in the Housatonic River 10-year floodplain and in nearby public and private lands within the watershed. American robin nests were located by direct observation and identification, as well as by tracking robins in the area to their nests.

Eggs and nestlings were collected for analysis of tPCB residues. One egg that was viable and 1 2 had been incubated for at least 10 days, was randomly chosen from each nest having at least four 3 eggs; no eggs were taken from nests with fewer eggs. Some nonviable eggs were also collected 4 for chemical analysis. The largest 7-day old nestling was selected for analysis of tPCB residues 5 from successful nests with three or more nestlings; no nestlings were collected from nests with 6 fewer young. A successful nest was defined as a nest that fledged at least one young and the 7 percent of successful nests was determined by dividing the number of successful nests by the 8 number of active nests. Clutch size, and hatching and fledging success were also determined for 9 active nests.

10 Concentrations of tPCBs in American robin eggs and nestlings were significantly higher in the 11 PSA compared to reference areas, yet there were no significant differences in the measures of 12 effects included in this study. Clutch size, hatching success, and fledging success all exhibited 13 no difference in target and reference areas and were within ranges typical for American robins 14 (Brehmer and Anderson 1992; Kemper and Taylor 1981; Fleutsch and Sparling 1994). The 15 number of nonviable eggs per successful nest was higher in the PSA compared to the reference 16 areas, although this difference was not significant (p = 0.89). The difference in egg tPCB 17 concentrations at study and reference sites indicated that robins in the PSA were receiving a local 18 exposure. The principal concerns with the study are the inclusion of only active nests in the 19 measurement of reproductive success, the small number of eggs and nestlings that were collected 20 in the reference areas and analyzed for tPCBs, the non-random selection of eggs and nestlings, 21 the methods used to determine viability of eggs, and the use of some questionable statistical 22 methods to analyze the results. A reanalysis of the clutch size data was performed, including all 23 nests with these data, as opposed to only active nests. This relationship was examined with a 24 Chi-squared test for trend, but results showed no significant relationship between PCB 25 concentration in eggs and clutch size.

26 7.5.4 Weight-of-Evidence Analysis

A weight-of-evidence analysis procedure was used to assess risks of tPCBs and TEQ to insectivorous birds. The goal of this analysis was to determine whether significant risk is posed to insectivorous birds in the Housatonic River PSA as a result of exposure to tPCBs and TEQ. 1 The three-phase approach of Menzie et al. (1996) and the Massachusetts Weight-of-Evidence 2 Workgroup was applied for this purpose, in which weight-of-evidence was reflected in the 3 following three characteristics: (1) the weight assigned to each measurement endpoint, (2) the 4 magnitude of response observed in the measurement endpoint, and (3) the concurrence among 5 outcomes of the multiple measurement endpoints (see Section 2.9 for details).

A discussion of attributes considered in the WOE is provided in Section 2, and the rationale for weighting of measurement endpoints are provided in Appendix G. A summary of how attributes were weighted for the tree swallow and American robin lines of evidence is provided in Tables 7.5-2 and 7.5-3, respectively. The attribute values, evidence of harm, and magnitudes of responses for both tree swallows and American robins are presented in Table 7.5-4 (tPCBs) and Table 7.5-5 (TEQ).

For tree swallows exposed to tPCBs and TEQ, the field-based reproductive study line of evidence was given a high weighting, and the modeled exposure and effects line of evidence was given a moderate weighting.

For American robins, the field study was given a moderate/high weighting for both tPCBs and TEQ. The modeled exposure and effects line of evidence was given moderate value for tPCBs and TEQ.

Weighting of Measurement Endpoints for Tree Swallow Weight-of-Evidence Evaluation

Attributes	Field Study Custer (2002)	Modeled Exposure and Effects for tPCBs and TEQ			
I. Relationship Between Measurement and Assessment Endpoints					
1. Degree of Association	High	Moderate			
2. Stressor/Response	Moderate/High	Moderate			
3. Utility of Measure	High	Moderate			
II. Data Quality					
4. Data Quality	High	Moderate			
III. Study Design					
5. Site Specificity	High	Low/Moderate			
6. Sensitivity	Moderate/High	Low/Moderate			
7. Spatial Representativeness	High	Moderate			
8. Temporal Representativeness	High	Moderate			
9. Quantitative Measure	Moderate/High	Moderate/High			
10. Standard Method	High	Moderate			
Overall Endpoint Value	High	Moderate			

5 6

1 2

Weighting of Measurement Endpoints for American Robin Weight-of-Evidence Evaluation

Attributes	Field Study Henning (2002)	Modeled Exposure and Effects for tPCBs and TEQ			
I. Relationship Between Measurement and Assessment Endpoints					
1. Degree of Association	High	Moderate			
2. Stressor/Response	Moderate	Moderate			
3. Utility of Measure	Moderate/High	Moderate			
II. Data Quality					
4. Data Quality	Moderate/High	Moderate			
III. Study Design					
5. Site Specificity	High	Low/Moderate			
6. Sensitivity	Moderate/High	Low/Moderate			
7. Spatial Representativeness	High	Moderate			
8. Temporal Representativeness	Moderate	Moderate			
9. Quantitative Measure	Moderate/High	Moderate/High			
10. Standard Method	Moderate/High	Moderate			
Overall Endpoint Value	Moderate/High	Moderate			

5

1

2 3

4

6 The magnitude of the response in the measurement endpoint is considered together with the 7 measurement endpoint weight in judging the overall weight-of-evidence (Menzie et al. 1996). 8 This requires assessing the strength of evidence that ecological harm has occurred, as well as an 9 indication of the magnitude of response, if present. For both tree swallows and American robins exposed to tPCBs (Table 7.5-4) and TEQ (Table 7.5-5), the modeled exposure and effects line of 10 11 evidence indicated that there was evidence of harm, and that the magnitude was high risk for 12 tPCBs and intermediate risk for TEQ. For both tPCBs and TEQ, the tree swallow field study line 13 of evidence and the American robin field study line of evidence indicated that there was little 14 evidence of harm, and that the magnitude was low.

Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Study	High (Tree swallow) Moderate/High (American robin)	No (Tree swallow) No (American robin)	Low (Tree swallow) Low (American robin)
Modeled Exposure and Effects	Moderate	Yes	High

5

6 7

8

9

Table 7.5-5

Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Study	High (Tree swallow) Moderate/High (American robin)	No (Tree Swallow) No (American robin)	Low (Tree Swallow) Low (American robin)
Modeled Exposure and Effects	Moderate	Yes	Intermediate

10

The final component in the weight-of-evidence approach addresses the concurrence among the measurement endpoints as they relate to each assessment endpoint. The methodology for detecting concurrence involves the use of a graphical method where measurement endpoints are plotted on a matrix that also includes the weight of each endpoint and degree of response. Tables 7.5-6 and 7.5-7 depict the outcomes for tree swallows and American robins exposed to tPCBs and TEQ, respectively. The analyses were conducted separately for tPCBs and TEQ.

Risk Analysis Summary for Insectivorous Birds Exposed to tPCBs in the Housatonic River PSA

Assessment Endpoint: Survival, growth, and reproduction of insectivorous birds ► Weighting Factors (increasing confidence of weight) Harm/Magnitude Low Low/Moderate Moderate **Moderate/High** High MEE Yes/High Yes/Intermediate Yes/Low Undetermined/High Undetermined/Intermediate Undetermined/Low FS (American No/Low FS (Tree robin) swallow) No/Intermediate No/High

6

FS=Field study

MEE=Modeled exposure and effects

7 8

9

1

Risk Analysis Summary for Insectivorous Birds Exposed to TEQ in the Housatonic River PSA

Assessment Endpoint:	Survival	, growth, and reproduc	ction of insectivorous	birds	
Weighting Factors (increasing confidence of weight)					
Harm/Magnitude	Low	Low/Moderate	Moderate	Moderate/High	High
Yes/High					
Yes/Intermediate			MEE		
Yes/Low					
Undetermined/High					
Undetermined/Intermediate					
Undetermined/Low					
No/Low				FS (American robin)	FS (Tree swallow)
No/Intermediate					
No/High					

6 FS = Field Study

7 MEE = Modeled exposure and effects

8

9 The results from the modeled exposure and effects line of evidence suggest that tPCBs and TEQ 10 pose a intermediate to high risk to tree swallows living in the PSA. However, the field study 11 line of evidence suggests that, if effects are occurring, they are minor. The uncertainty 12 concerning the field-based threshold range for tPCBs likely means that risks of this COC are 13 overestimated for the PSA. Even the upper end of the tPCB range is associated with equivocal 14 evidence for adverse effects to tree swallows. For TEQ, the threshold range is quite broad. The 15 available evidence from field studies indicates that tree swallows are tolerant to exposure to 16 persistent organochlorines such as tPCBs and TEQ (Section G.3.2). If the tree swallow threshold 17 is near the upper end of the threshold range, then the current modeled exposure and effects line 18 of evidence is overestimating risks of TEQ to tree swallows. Thus, the WOE assessment

5

1

supports a finding of low risk for tree swallows exposed to tPCBs and TEQ in the PSA. This
 conclusion, however, is uncertain because of the conflicting results in the WOE assessment.

3 The results from the modeled exposure and effects lines of evidence suggest that tPCBs and TEQ 4 pose an intermediate to high risk to American robins inhabiting the PSA of the Housatonic River. 5 The American robin field study, however, suggests that reproductive success is not being impaired by the tPCBs and TEQ in the PSA. The uncertainty in the modeled exposure and 6 7 effects line of evidence, outlined below, likely means the approach overestimates the risks of tPCBs and TEQ to American robins in the PSA. The WOE assessment, therefore, supports a 8 9 conclusion of low risk to American robins exposed to tPCBs and TEQ in the PSA. This 10 conclusion, however, is uncertain because of the conflicting results in the WOE assessment.

11 7.5.5 Sources of Uncertainty

The assessment of risk to insectivorous birds contains uncertainties. Each source of uncertainty can influence the estimates of risk; therefore, it is important to describe and, when possible, specify the magnitude and direction of such uncertainties. The sources of uncertainty associated with the assessment of risks of tPCBs and TEQ to insectivorous birds are described below.

- In this assessment, it was assumed that dietary exposure represented the most important pathway for exposure of insectivorous birds to COCs. Although unlikely to provide a major contribution to the risk, other pathways could increase exposure and perhaps increase risk slightly (Moore et al. 1999). Deterministic calculations were conducted in which estimates of exposure to COCs via drinking water and soil ingestion were included in the exposure model. Inclusion of these routes did not substantially increase overall exposure of insectivorous birds to the COCs.
- The microexposure model used the ratio of tissue concentrations in pippers and nestlings to indirectly estimate the amount of tPCBs and TEQ transferred from mothers to offspring via egg tissue. The ratio was calculated using a sample of limited size. Variability in the ratio was high. Each of these parameters has associated uncertainties, but the potential magnitude and direction of uncertainty is unknown.
- The Monte Carlo sensitivity analyses suggested that the free metabolic rate (*FMR*) slope and power terms were generally the most influential variables on predicted total daily intakes of COCs. However, no direct measurements of free metabolic rate are available for tree swallows. Similarly, measured food intake rates are not available for free-living tree swallows and American swallows. Therefore, free metabolic rates

were estimated using allometric equations. The use of allometric equations introduced some degree of uncertainty into the exposure estimates because they have model-fitting error. The uncertainty due to model-fitting error was propagated in the uncertainty analyses by using distributions as input for the allometric slope and power terms.

- Sample sizes were limited for the analyses of COC concentrations in tree swallow stomach contents. Only four samples from each of 3 years were available for each location (2 years for Taconic Valley and Threemile Pond). In addition, these samples were pooled from collections from individual tree swallows to augment sample mass, but the exact number of individuals pooled was variable and reported only as a range.
- 11 Sample sizes were limited for the analyses of COC concentrations in some prey 12 items, including earthworms, litter invertebrates, and benthic invertebrates. In the case of TEQ analyses in earthworms, only one composite sample was available at 13 14 each location, composed of between 20 and 45 worm samples. To address this 15 uncertainty in the Monte Carlo analysis, the UCL or data set maximum (see Section 6.4 and Appendix C.5) was used as an estimate of COC concentrations in prey items. 16 The potential magnitude of the uncertainty associated with small sample sizes for 17 18 COC concentrations is unknown, but this approach likely overestimates exposure. 19 The probability bounds analysis used an unbiased approach (e.g., distribution free 20 range from lower confidence limit [LCL] to upper confidence limit [UCL]) to deal 21 with sample size uncertainty.
- PCB congeners 123 and 157 co-eluted with other congeners (PCB-123 with PCB-149; PCB-157 with PCB-173 and PCB-201). As a result, decision criteria were developed (see Section 6.4 of the ERA) for co-eluted congeners to determine TEQ concentrations used as distribution parameters in the Monte Carlo and probability bounds analyses. These criteria were designed to explicitly incorporate this source of uncertainty in the probabilistic analyses. Thus, this source of uncertainty has been incorporated in this risk assessment.
- 29 Many TEQ congeners were detected at or below the method detection limit (see 30 Appendix G and Section 6.4 of the ERA), particularly in the reference locations. In 31 the Monte Carlo analyses, for prey concentration distributions affected by non-32 detects, a triangular distribution was used with the minimum TEO assuming non-33 detects equal to zero, the best estimate TEQ assuming non-detected equal to one half 34 the detection limit, and the maximum TEQ assuming non-detects equal to the 35 detection limit. In the probability bounds analyses, a distribution-free range was used with the lower limit assuming non-detects equal to zero and the upper limit assuming 36 37 non-detects equal to the detection limit. Thus, this source of uncertainty has been 38 incorporated in this risk assessment. Concentrations of tPCBs in prey tissues in the 39 PSA were all above the detection limit. Thus, there was no non-detect issue for 40 tPCBs.
- 41 A source of uncertainty in the effects assessment for American robins exposed to 42 tPCBs and both representative species exposed to TEQ was the lack of controlled

1

2

3

4

laboratory studies involving tree swallows and American robins. As a result, laboratory studies on surrogate species were used to estimate the effects range of tPCBs and TEQ to insectivorous birds. This extrapolation introduced uncertainty in the tPCB and TEQ effects assessment because of the variations in physiological and biochemical factors such as uptake, metabolism, and disposition that can alter the potential toxicity of a contaminant. The sensitivity of birds to an environmental contaminant may differ from that of a laboratory or domestic species due to behavioral and ecological parameters, including stress factors (e.g., competition, seasonal changes in temperature or food availability), disease, and exposure to other contaminants. Inbred laboratory animal strains may also have an unusual sensitivity or resistance to a tested substance.

- 12 A source of uncertainty in the effects assessment for tPCBs was due to the lack of 13 controlled laboratory studies involving tree swallows exposed to tPCBs. As a result, 14 a field-based threshold range was used as the effects metric for effects of tPCBs to This extrapolation introduced uncertainty in the tPCB effects 15 tree swallows. 16 assessment for two reasons. First, the value selected for the lower end of the range 17 (62.2 mg/kg) has uncertainty as to whether it caused adverse effects to tree swallow reproduction, including abnormal nest abandonment behavior and larger clutch sizes, 18 19 in the upper Hudson River area (McCarty and Secord 1999a). The ecological 20 significance of these effects could not be determined. In addition, the value selected for the upper end of the range (69 mg/kg) has uncertainty as to whether it caused 21 22 adverse effects to tree swallow hatching success in the PSA (Custer 2002). Thus, 23 despite the narrowness of the tPCB threshold range, there is some uncertainty about 24 this effects metric. Second, field studies are subject to a number of factors that are impossible to control, including weather, predation, disease, and other COPCs. 25
- 26 Concentrations of tPCBs in tree swallow pippers and nestlings collected within the 27 PSA, although some of the highest ever reported and well above previously reported 28 thresholds, showed a weak negative correlation to hatching success in 1998 and 1999, 29 and no correlation in 2000. Poor hatching success in 2000 was attributed to cold 30 rainy weather in the nesting season. While reproductive effects may have been 31 observed in tree swallows nesting at other PCB-contaminated sites, evidence 32 indicating that these effects are occurring or are solely the result of tPCB exposures is 33 lacking for the PSA and other PCB-contaminated sites.

34 **7.5.6** Conclusions and Extrapolation to Other Species

The WOE analysis indicated that exposure of insectivorous birds, such as tree swallows and American robins, to tPCBs and TEQ in the PSA is unlikely to lead to adverse reproductive effects. This conclusion, however, is uncertain because the lines of evidence did not produce concordant results. The lines of evidence used in this conclusion were the field-based tree swallow and American robin reproductive studies and the comparison of modeled exposure with effects.

1 2

3

4

5

6

7

8

9

10

1 Custer (2002) conducted a field-based tree swallow reproduction study in the years 1998 through 2 2000 in the PSA and reference areas. The mean clutch sizes for tree swallows in the PSA in 3 1998, 1999, and 2000 were 5.43, 5.37, and 5.46 eggs/clutch, respectively, compared to central 4 Massachusetts, where the mean clutch size was 4.8 to 5.3 eggs/clutch over a 22-year period 5 (Chapman 1955). Thus, the fecundity of tree swallows in the PSA was unaffected by tPCBs and 6 TEQ. The geometric means of tPCB concentrations in tree swallow pippers and nestlings 7 collected from the Housatonic River ranged from 31.5 to 101 mg/kg ww whole body. These are 8 the highest concentrations reported in the literature (Custer 2002) and are substantially higher 9 than concentrations in samples obtained from reference sites (6 to 19 mg/kg ww whole body). 10 Total PCBs, dioxins, and furans were negatively correlated with hatching success in 1998 and 11 1999, but the correlations were weak.

The field-based tree swallow reproduction study did not detect obvious adverse effects to tree swallow reproduction, despite high tissue concentrations in nestlings. This study supports the conclusion that tree swallows are not being adversely affected due to exposure to tPCBs and TEQ in the PSA.

16 The American robin field study (Henning 2002) was conducted during the 2001 breeding season 17 in the PSA and reference areas. The study evaluated the relationship between tissue 18 concentrations and reproductive output. Concentrations of tPCBs in American robin eggs and 19 nestlings were significantly higher in the PSA compared to reference areas, with tPCB 20 concentrations in robin eggs averaging 83.6 mg/kg ww in the PSA (n=9) and 0.153 mg/kg ww in 21 the reference areas (n=2). There were, however, no significant differences in any of the 22 measures of effect in this study. Clutch size, hatching success, and fledging success all exhibited 23 no difference in target and reference areas and were within ranges typical for American robins (Brehmer and Anderson 1992; Kemper and Taylor 1981; Fleutsch and Sparling 1994). 24

The modeled exposure and effects line of evidence for tree swallows compared estimated body burdens of nestlings (for tPCBs) or estimated daily intake by adult females (for TEQ) with COC levels found in the literature. Field studies investigating the reproductive effects of tPCBs on tree swallows were available in the literature, so the effects characterization employed a fieldbased toxicity threshold range to describe the potential effects of tPCBs to tree swallows. The

most tolerant bird species to tPCBs found in the literature was the tree swallow, which had a 1 2 threshold tPCB tissue concentration range of 62.2 to 69 mg/kg ww, based on field-based 3 reproductive studies conducted on tree swallows by Custer (2002) and McCarty and Second (1999a). The upper threshold is the geometric mean tPCB concentration in tree swallows 4 5 associated with reduced hatching success (Custer 2002). The lower threshold represents the 6 whole body tPCB concentrations in tree swallow nestlings at which no adverse reproductive 7 effects were observed (McCarty and Secord 1999a). The microexposure model results indicated 8 that tree swallow nestlings are likely to attain body burdens greater than 69 mg/kg ww at the 9 three PSA study locations in this assessment.

10 Insufficient field-based data were available for American robins exposed to tPCBs. Thus, for 11 American robins a threshold range for surrogate species was used. White leghorn chickens were 12 the most sensitive avian species to the reproductive effects of tPCBs and the most reproductively 13 tolerant avian species to tPCBs was the American kestrel. The threshold range for the 14 reproductive success of insectivorous birds exposed to tPCBs selected for this assessment was 15 0.12 to 7.0 mg/kg bw/d based on reproductive studies conducted on white leghorn chickens 16 (Lillie et al. 1974) and American kestrels (Fernie et al. 2001b), respectively. The total daily 17 intake model results indicated that American robins are at high risk in the PSA.

18 A laboratory-based toxicity threshold range was used to describe the potential effects of TEQ to 19 insectivorous birds. The most sensitive and most tolerant bird species were used to develop the 20 TEQ toxicity threshold range, with the assumption that tree swallows and American robins 21 would begin to experience adverse effects in this range. The toxicity threshold range is very 22 wide (44 to 25,000 ng/kg bw/d). Most exposure estimates for tree swallows and American robins 23 fell within this range, placing the birds at an intermediate risk from exposure to TEQ. While the 24 modeled exposure and effects line of evidence suggested that tree swallows and American robins 25 are at risk in the PSA, confidence in this line of evidence is reduced compared to the field study.

The results from the modeled exposure and effects line of evidence suggest that tPCBs and TEQ pose intermediate to high risks to tree swallows and American robins living in the PSA. However, the more highly weighted field study line of evidence suggests that if effects are occurring, they are minor for both species. Thus, the WOE assessment favors a finding of low risk for insectivorous birds exposed to tPCBs and TEQ in the PSA. This conclusion, however, is
 uncertain because of the conflicting results in the WOE assessment.

3 Other insectivorous bird species common to the PSA include the bank swallow, northern rough-4 winged swallow, barn swallow, cliff swallow, chimney swift, common nighthawk, eastern 5 kingbird, eastern phoebe, eastern bluebird, eastern towhee, gray catbird, hermit thrush, northern 6 mockingbird, veery, and wood thrush. Effects data are not available for other insectivorous bird 7 species living in the Housatonic River area. As a result, the same effects data used to estimate 8 effects to tree swallows were used for other insectivorous species. A qualitative analysis was 9 conducted to compare exposure of tree swallows, American robins, and other insectivorous birds 10 to tPCBs and TEQ. The major factors that influence avian exposure to tPCBs and TEQ include:

11 12

13

14

- Foraging behavior and dietary composition.
- Foraging and home range size.
- Species body weight and other life history characteristics.

Tree swallows and other insectivorous bird species were compared using these factors and the results are provided in Appendix G.4.6. A qualitative analysis of risk to these species indicates that the cliff swallow, eastern kingbird, eastern bluebird, and eastern towhee have a similar to lower level of risk compared to the representative species; barn swallow, common nighthawk, eastern phoebe, hermit thrush, northern mockingbird, veery, and wood thrush have a similar level of risk; and bank swallow, chimney swift, northern rough-winged swallow, and gray catbird have a similar to higher level of risk compared to the tree swallow.

1

2

3

4

5

6

7

8

9

10

11

12

13

14

15

16

17

ERA Summary

The weight-of-evidence analysis indicates that insectivorous birds are likely at low risk in the PSA as a result of exposure to tPCBs and TEQ. This conclusion, however, is uncertain. Risks to tree swallows and American robins in the PSA are intermediate to high based on modeled exposure and effects, but field studies detected no obvious adverse reproductive effects in the PSA.

Other insectivorous bird species common to the PSA include the bank swallow, northern rough-winged swallow, barn swallow, cliff swallow, chimney swift, common nighthawk, eastern kingbird, eastern phoebe, eastern bluebird, eastern towhee, gray catbird, hermit thrush, northern mockingbird, veery, and wood thrush. A qualitative analysis of risk to these species indicates that the cliff swallow, eastern kingbird, eastern bluebird, and eastern towhee have a similar to lower level of risk compared to the tree swallow; barn swallow, common nighthawk, eastern phoebe, hermit thrush, northern mockingbird, veery, and wood thrush have a similar level of risk; and bank swallow, chimney swift, northern rough-winged swallow, and gray catbird have a similar to higher level of risk compared to the tree swallow.

18

19 **7.6 REFERENCES**

- Bell, G.P. 1990. Birds and mammals on an insect diet: A primer on diet composition analysis in relation to ecological energetics. *Studies in Avian Biology* 13:391-415. Cited in EPA 1993.
- 22 Bierman, G.C. and S.G. Sealy. 1985. Seasonal dynamics of body mass of insectivorous 23 passerines breeding on the forested dune ridge, Delta Marsh, Manitoba. *Canadian Journal of*
- 24 Zoology 63:1675-1682.
- Blancher, P.J. and D.K. McNicol. 1991. Tree swallow diet in relation to wetland acidity.
 Canadian Journal of Zoology 69:2629-2637.
- Borga, K., G.W. Gabrielsen, and J.U. Skaare. 2001. Biomagnification of organochlorines along
 a Barents sea food chain. *Environ. Pollut.* 113(2):187-198.
- Bosveld, A.T.C. and M. Van den Berg. 1994. Effects of polychlorinated biphenyls, dibenzo-pdioxins, and dibenzofurans on fish-eating birds. *Environmental Reviews* 2:147-166.
- Brehmer, P.M. and R.K. Andersen. 1992. Effects of urban pesticide application on nesting
 success of songbirds. Bulletin of Environmental Contamination and Toxicology 48:352-359.

Britton, W.M. and T.M. Huston. 1973. Influence of polychlorinated biphenyls in the laying hen.
 Poultry Science 52:1620-1624.

- 1 Brunström, B. and J. Lund. 1988. Differences between chick and turkey embryos in sensitivity
- 2 to 3,4'4,4'-tetrachlorobiphenyl and in concentration/affinity of the hepatic receptor for 2,3,7,8-
- 3 tetrachlorodibenzo-p-dioxin. Com. Biochem. Physiol. C 91(2):507-512.
- 4 Chapman, L.B. 1955. Studies of a tree swallow colony. *Bird Banding*. 26:45-70.
- 5 Clench, M.H. and R.C. Leberman. 1978. *Weights of 151 Species of Pennsylvania Birds* 6 *Analyzed by Month, Age, and Sex.* Bulletin of the Carnegie Museum of Natural History. 87 pp.
- CCME (Canadian Council of Ministers of the Environment). 1999. Canadian Tissue Residue
 Guidelines for the Protection of Wildlife Consumers of Aquatic Biota: Polychlorinated
 Biphenyls (PCBs). In *Canadian Environmental Quality Guidelines*, 1999. Canadian Council of
 Ministers of the Environment, Winnipeg.
- Collopy, M.W. 1975. Behavioral and predatory dynamics of kestrels wintering in the Arcata
 Bottoms [master's thesis]. Humboldt State University, Arcata, CA.
- 13 Corbet, R.L., D.G. Muir, and G.R.B. Webster. 1983. Fate of carbon-14 labeled 1,3,6,8-14 tetrachloro-dibenzo-*p*-dioxin in an outdoor aquatic system. *Chemosphere* 12:523-528.
- 15 Cummins, K.W. and J.C. Wuycheck. 1971. Caloric equivalents for investigations in ecological
- 16 energetics. International Association of Theoretical and Applied Limnology. Stuttgart, West
- 17 Germany, Cited in EPA 1993.
- 18 Custer, C.M. 2002. Exposure and Effects of Chemical Contaminants on Tree Swallows Nesting
- 19 Along the Housatonic River, Berkshire Co., Massachusetts, 1998-2000. Final Report to U.S.
- 20 Environmental Protection Agency. USGS, Upper Midwest Environmental Sciences Center, La
- 21 Crosse, WI.
- Dunning, J.B. 1984. Body Weights of 686 Species of North American Birds. Eldon Publishing,
 Cave Creek, Arizona.
- EPA (U.S. Environmental Protection Agency). 1993. Wildlife Exposure Factors Handbook.
 EPA/600/R-93/187a. Office of Research and Development. Washington, DC.
- 26 Fernie, K.J., J.E. Smits, G.R. Bortolotti, and D.M. Bird. 2001. Reproductive success of American
- 27 kestrels exposed to dietary polychlorinated biphenyls. *Environmental Toxicology and Chemistry*
- 28 20:776-781.
- 29 Fleutsch, K.M. and D.W. Sparling. 1994. Avian nesting success and iversity in conventionally
- 30 and organically managed apple orchards. Environmental Toxicology and Chemistry
- 31 13(10):1651-1659.
- 32 Haffner, G.D., M. Tomczak, and R. Lazar. 1994. Organic contaminant exposure in the Lake St.
- 33 Clair food web. *Hydrobiologia* 281:19-27.

- 1 Hazelton, P.K., R. J. Robel and A.D. Dayton. 1984. Preferences and influences of paired food
- 2 items on energy intake of American robins (Turdus migratorius) and gray catbirds (Dumetella
- 3 carolinensis) J. Wildl. Manage. 48:198-202.
- 4 Heath, R.G., J.W. Spann, E.F. Hill, and J.F. Kreitzer. 1972. Comparative Dietary Toxicities of
- *Pesticides to Birds.* U.S. Fish and Wildlife Service Special Scientific Report on Wildlife 152. 57
 pp.
- Henning, M.H. 2002. *Robin Productivity in the Housatonic River Watershed, Berkshire County, Massachusetts*. Prepared for: General Electric Company, Pittsfield, Massachusetts.
- 9 Hoffman, D.J., M.J. Melancon, P.N. Klein, C.P. Rice, J.D. Eisemann, R.K. Hines, J.W. Spann,
- and G.W. Pendleton. 1996. Developmental toxicity of PCB 126 (3,3,4,4,5-pentachlorobiphenyl)
- 11 in nestling American kestrels (Falco sparverius). Fundamental and Applied Toxicology 34:188-
- 12 200.
- Howard, P.H., R.S. Boethling, W.F. Jarvis, W.M. Meylan, and E.M. Michalenko. 1991.
 Handbook of Environmental Degradation Rates. Lewis Publishers, Chelsea, MI.
- Hudson, R., R. Tucker, and M. Haegele. 1984. *Handbook of Toxicity of Pesticides to Wildlife*.
 2nd Edition. U.S. Fish and Wildlife Service Resource Publication 153. Washington, DC.
- Karasov, W.H. 1990. Digestion in birds: Chemical and physiological determinants and
 ecological implications. *Studies in Avian Biology* 13:391-415.
- Kemper, D.L. and J.M. Taylor. 1981. Seasonal reproductive changes in the American robin
 (*Turdus migratorius*) of the Pacific Northwest. *Canadian Journal of Zoology* 59:212-217.
- Kuehl, D.W., P.M. Cook, A.R. Batterman, and B.C. Butterworth. 1987. Isomer dependent
 bioavailability of polychlorinated dibenzo-*p*-dioxins and dibenzofurans from municipal
 incinerator fly ash to carp. *Chemosphere* 16:657-666.
- Lillie, R.J., H.C. Cecil, J. Bitman, and G.F. Fries. 1974. Differences in response of caged white leghorn layers to various polychlorinated biphenyls (PCBs) in the diet. *Poultry Science* 53:726-732.
- Marcum, H.A., W.E. Grant, and F. Chavez-Ramirez. 1998. Simulated behavioral energetics of non-breeding American robins: The influence of foraging time, intake rate, and flying time on
- 29 weight dynamics. *Ecological Modeling* 106:161-175.
- McCarty, J.P., and A.L. Secord. 1999a. Nest-building behaviour in PCB-contaminated tree swallows. *Auk* 116:55-63.
- McCarty, J.P., and A.L. Secord. 1999b. Reproductive ecology of tree swallows (*Tachycineta bicolor*) with high levels of polychlorinated biphenyl contamination. *Environ. Toxicol. Chem.* 18:1433-1439.

Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S. Petron,
 B. Potocki, S. Svirsky, and P. Tyler. 1996. Special report of the Massachusetts Weight-of Evidence Workgroup: A weight-of-evidence approach for evaluating ecological risks. *Human Ecological Risk Assessment* 2:277-304.

Moore, D.R.J., B.E. Sample, G.W. Suter, B.R. Parkhurst, and R.S. Teed. 1999. A probabilistic
risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East
Fork Poplar Creek, Oak Ridge, Tennessee, USA. *Environmental Toxicology and Chemistry*18(12):2941-2953.

- 9 Muir, D.C.G., R.J. Nostrom, and M. Simon. 1988. Organochlorine contaminants in Arctic 10 marine food chains: Accumulation of specific polychlorinated biphenyls and chlordane-related 11 compounds. *Environmental Science and Technology* 22:1071-1079.
- Nagy, K.A. 1987. Free metabolic rate and food requirement scaling in mammals and birds.
 Ecological Monographs 57:111-128.
- Nagy, K.A., I.A. Girard, and T.K. Brown. 1999. Energetics of free-ranging mammals, reptilesand birds. *Annu. Rev. Nutr.* 19:247-277.
- 16 Newsted, J.L., J.P. Giesy, G.T. Ankley, D.E. Tillitt, R.A. Crawford, J.W. Gooch, P.D. Jones, and
- 17 M.S. Denison. 1995. Development of toxic equivalency factors for PCB congeners and the
- 18 assessment of TCDD and PCB mixtures in rainbow trout. Environmental Toxicology and
- 19 *Chemistry* 14(5):861-871.
- 20 Nosek, J.A., S.R. Craven, J.R. Sullivan, S.S. Hurley, and R.E. Peterson. 1992. Toxicity and
- 21 reproductive effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin in ring-necked pheasant. Journal of
- 22 *Toxicology and Environmental Health* 35:187-198.
- NRCC (National Research Council of Canada). 1981. Polychlorinated dibenzo-*p*-dioxins:
 Criteria for their effects on man and his environment. Publication NRCC No. 18574. National
 Research Council of Canada. Ottawa, Ontario. 251 p.
- Prestt, I., D.J. Jefferies, and N.W. Moore. 1970. Polychlorinated biphenyls in wild birds in
 Britain and their avian toxicity. *Environmental Pollution* 1:3-26.
- 28 Robertson, R.J., B.J. Stuchbury, and R.R. Cohen. 1992. Tree Swallow (*Tachycineta bicolor*). In
- 29 The Birds of North America, No. 11, A. Poole, P. Stettenheim, and F. Gill, Editors. Academy of
- 30 Natural Sciences, Philadelphia, and American Ornithologists Union, Washington, DC.
- Roberston, R.J., and J. Jones. 2002. Spatial and Demographic Effects on Tree Swallow Nest
 Quality and Reproductive Success. Department of Biology, Queen's University. Kingston,
 Ontario. Prepared for General Electric Company, April 2002.

Safe, S.H. 1994. Polychlorinated biphenyls (PCBs): Environmental impact, biochemical and
 toxic responses, and implications for risk assessment. *Critical Reviews in Toxicology* 24(2): 87 149.

- 1 Secord, A.L., J.P. McCarty, K.R. Echols, J.C. Meadows, R.W. Gale, and D.E. Tillitt. 1999.
- 2 Polychlorinated biphenyls and 2,3,7,8-tetrachlorodibenzo-p-dioxin equivalents in tree swallows
- 3 from the upper Hudson River, New York State, USA. *Environmental Toxicology and Chemistry*
- 4 18:2519-2525.
- 5 Senthilkumar, K., N. Iseki, S. Hayama, J. Nakanishi, and S. Masunaga. 2001. Polychlorinated
- 6 dibenzo-p-dioxins, dibenzofurans, and dioxin-like polychlorinated biphenyls in livers of birds
- 7 from Japan. Arch. Environ. Contam. Toxicol. 42:244-255.
- Scott, M.L. 1977. Effects of PCBs, DDT, and mercury compounds in chickens and Japanese
 quail. *Federation Proceedings* 36:1888-1893.
- Teather, K. 1996. Patterns of growth and asymmetry in nestling tree swallows. *Journal of Avian Biology* 27:302-310.
- 12 Tsushimoto, G., F. Matsumura, and R. Sago. 1982. Fate of 2,3,7,8-tetrachlorodibenzo-p-dioxin
- 13 (TCDD) in an outdoor pond and in model aquatic ecosystems. *Environmental Toxicology and*
- 14 *Chemistry* 1:61-68.
- 15 Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy,
- 16 A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X.R. van Leeuwen,
- 17 A.K.D. Liem, C. Nolt, R.E. Petersen, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind,
- 18 M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs) for tPCBs,
- 19 PCDDs, PCDFs for humans and wildlife. *Environ. Health Perspectives* 106:775-792.
- Wheelwright, N.T. 1986. The diet of American robins: An analysis of U.S. Biological Survey records. *The Auk* 103:710-725.

8. ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS BIRDS

3	Highlights
4	Conceptual Model
5 6 7 8	Piscivorous birds, including the osprey and belted kingfisher, selected as representative species for the ERA, are exposed to contaminants of concern (COCs) via diet and trophic transfer. The assessment endpoint is the survival, growth, and reproduction of piscivorous birds in the Housatonic River PSA.
9	Exposure
10 11	COC intake by ospreys and belted kingfishers was highest in Reaches 5 and 6 of the PSA, while exposure in the reference areas was much lower.
12	Effects
13 14 15	No information was available specifically on the toxicity of tPCBs and TEQ to belted kingfishers or ospreys. A threshold range spanning sensitive and tolerant surrogate species was used instead for both tPCBs and TEQ.
16	Risk
17 18 19 20 21 22	Ospreys are at high risk as a result of exposure to tPCBs and intermediate risk as a result of exposure to TEQ in the Housatonic River PSA. In these high-risk areas, modeled exposure of ospreys to PCBs is greater than doses that cause adverse effects in the most tolerant species studied. The weight-of-evidence (WOE) conclusion of high risk is uncertain because other lines of evidence (e.g., field surveys, in situ or whole media studies) were unavailable.
23 24 25 26 27 28	While modeled exposure and effects indicated high risk to belted kingfishers as a result of exposure to tPCBs and intermediate risk as a result of exposure to TEQ, a field study of kingfisher productivity indicated that the birds were reproducing in the PSA. The WOE assessment for belted kingfishers concluded that this species is at low risk. This conclusion, however, is uncertain because of the lack of concordance between the two lines of evidence.

29

30 8.1 INTRODUCTION

This section summarizes the current and potential risks to piscivorous birds exposed to contaminants of potential concern (COPCs) in the Housatonic River and floodplain. It focuses on tPCBs and other COPCs originating from the General Electric Company (GE) facility in Pittsfield, MA. The river is located in western Massachusetts and Connecticut, discharging to Long Island Sound, with the GE facility located near the headwaters. The Primary Study Area (PSA) includes the river and 10-year floodplain from the confluence of the East and West Branches of the Housatonic River, downstream of the GE facility, to Woods Pond Dam.

1 A Pre-ERA was conducted to narrow the scope of the ERA by identifying contaminants, other 2 than tPCBs, that pose potential risks to aquatic biota and wildlife in the PSA (Appendix B). A 3 three-tiered deterministic approach was used to screen COPCs. The deterministic assessments 4 compared conservative estimates of potential exposure with conservative adverse effects 5 benchmarks to identify which contaminants are of potential concern to piscivorous birds in the 6 Housatonic River. A hazard quotient (total daily intake/effect benchmark) greater than 1 for 7 piscivorous birds in the Housatonic River area resulted in the COPC being screened through to 8 the next tier assessment and to the probabilistic ERA, if necessary. Subsequent to the Pre-ERA, 9 several other COPCs (primarily organochlorine pesticides) were screened out because their 10 actual concentrations in the PSA were likely much lower than the measured values due to 11 laboratory interference (see Section 2.4).

In summary, the COCs that were retained for the probabilistic risk assessment for piscivorous birds were total PCBs (tPCBs) and 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) toxic equivalence (TEQ). Total PCBs detected in Housatonic River media closely resemble the commercial PCB mixtures Aroclor 1260 and Aroclor 1254, which are similar in congener makeup. TEQ is calculated from coplanar PCB and dioxin and furan congeners using the toxic equivalency factor (TEF) approach developed by Van den Berg et al. (1998) (see Section 6.4 of the ERA and Appendix C.10).

19 8.1.1 Overview of Approach

A step-wise approach was used to assess the risks of tPCBs and TEQ to piscivorous birds in the
Housatonic River watershed. The four main steps in this process include:

- 1. Derivation of a conceptual model (Figure 8.1-1).
 - 2. Assessment of exposure of birds to COCs (Figure 8.1-2).
 - 3. Assessment of the effects of COCs on birds (Figure 8.1-3).
 - 4. Characterization of risk to the piscivorous avian species (Figure 8.1-4).
- 25 26

23

1 This section is organized as follows:

2 3 4	•	Section 8.2 (Conceptual Model) - Describes the conceptual model for piscivorous birds, including selection of representative species and establishment of assessment and measurement endpoints.
5 6 7	•	Section 8.3 (Exposure Assessment) - Describes the exposure model, input variables, and techniques to propagate uncertainty. Also presented in this section are the input parameters and exposure modeling results for belted kingfishers and ospreys.
8 9 10	•	Section 8.4 (Effects Assessment) - Describes the potential effects to birds exposed to tPCBs and TEQ. This section reviews the belted kingfisher field study conducted in the PSA, as well as toxicity thresholds found in the literature.
11 12 13 14	•	Section 8.5 (Risk Characterization) - Presents the lines of evidence addressed in the risk characterization, followed by a discussion of the sources of uncertainty in this assessment, and the conclusions regarding risks of tPCBs and TEQ to piscivorous birds in the Housatonic River PSA.
15 16	[The detailed ecological risk assessment for piscivorous birds is provided in Appendix H.
17 18	-	


1 2 3

Figure 8.1-1 Conceptual Model for Exposure of Piscivorous Birds to tPCBs and TEQ in the Housatonic River PSA





7/10/2003



Figure 8.1-4 Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Piscivorous Birds in the Housatonic River PSA

1 8.2 CONCEPTUAL MODEL

2 The conceptual model presented in Figure 8.1-1 illustrates the exposure pathways for piscivorous 3 birds exposed to tPCBs and TEQ in the PSA. Total PCBs and TEQ are persistent, lipophilic, and 4 hydrophobic. Therefore, they are bioaccumulated by aquatic and terrestrial biota directly 5 through the consumption of contaminated prey as part of the food chain (Haffner et al. 1994; 6 Senthilkumar et al. 2001). Piscivorous birds that reside, or partially reside, within the study area 7 are exposed to tPCBs and TEQ principally through diet and trophic transfer. Other routes of 8 exposure, considered to be less important to overall exposure, include inhalation, water 9 consumption, and sediment ingestion (Moore et al. 1999).

10 The problem formulation (see Section 2 of the ERA) identified the belted kingfisher (Cervle 11 alcyon; Figure 8.2-1) and osprey (Pandion haliaetus; Figure 8.2-2) as the representative species 12 for piscivorous birds potentially exposed to tPCBs and TEQ from consumption of contaminated 13 prey in the PSA. Kingfishers have been observed nesting and breeding in the PSA, while 14 observations of ospreys suggest that birds foraging in the PSA are transients. The PSA contains 15 suitable habitat for ospreys, with abundant prey, so there is a high likelihood that as the 16 Massachusetts and Connecticut osprey population continues to expand, they may nest in the 17 PSA. Great blue herons were also considered as a representative species, but were not included 18 in the assessment because, although productivity data for herons in the vicinity of the PSA are 19 available (MDFW 1979, 1980, 1981, 1982, 1983, 1984, 1985, 1986a,b, 1987, 1989, 1991, 1996), 20 only a few of the birds from the rookery forage in the study area. Estimating exposure to these 21 individual birds, and estimating exposure to any contaminants for the remainder of the herons in 22 the area, would be difficult. Therefore, effects to great blue heron productivity, or lack thereof, 23 observed in the field would be difficult to attribute to specific COCs.

Life history profiles for belted kingfishers and ospreys are summarized in the following text boxes. Additional life history information on these species is provided in Appendix H.

The assessment endpoint that is the subject of this section is the survival, growth, and reproduction of piscivorous birds in the Housatonic River PSA. The measurement endpoints used to evaluate the assessment endpoint were based on the determination of the extent to which the concentrations of PCBs and TEQ ingested in the diet will impact the survival, reproduction, or growth of piscivorous birds. The assessment for piscivorous birds includes both a site-specific field study of kingfisher reproductive success, and comparisons of modeled exposures to doses reported in the literature to cause adverse effects.



Figure 8.2-1 Belted Kingfisher (Ceryle alcyon)

Life History of Belted Kingfisher

The belted kingfisher is a pigeon-sized member of the family Alcedinidae and is a common bird in North America, excluding the far north and the higher elevations of the Rocky Mountains.

Habitat - Prefers foraging areas with clear water and visibility unobstructed by turbidity or aquatic vegetation. Size of territory depends upon the availability of prey, ranging from 0.5 to 1.36 miles (0.8 to 2.2 km) of shoreline.

Diet - Principal prey is fish, but may also feed on berries and other small animals, including mollusks, crustaceans, insects, amphibians, reptiles, young birds, and small mammals.



Figuro	8 2-2	Osnrov	(Pandion	haliaotus	۱
гідиге	0.2-2	Osprey	(Fallulul	IIallaelus	,

Life	History	of	Ospr	еy
------	---------	----	------	----

Ospreys, also known as fish hawks or fishing eagles, are the only species in the family Pandionidae. The range of these raptorial birds covers almost all of North America, except for the extreme north.

Habitat - Use both fresh and saltwater ecosystems, but primarily the latter (Rattner et al. 2001). Ospreys are tree nesters, but have also adapted to man-made structures. Foraging ranges from 1.7 km to 15 km, depending on prey availability.

Diet - Almost exclusively piscivorous, preferring medium-sized fish (13 to 40 cm). On rare occasions, osprey will take dead fish or prey on small mammals, reptiles, and crustaceans.

1 8.3 EXPOSURE ASSESSMENT

The exposure assessment for piscivorous birds focuses on the PSA. Where possible, exposure assessments were also conducted for two reference locations: East Branch of the Housatonic River, upstream of Dalton (termed "upstream reference area"), and Threemile Pond in Sheffield, MA. This section describes the general model used to estimate exposure of the two representative species to tPCBs and TEQ in the PSA, as well as the inputs used for each representative species. A summary of the exposure analyses results concludes the section.

8 8.3.1 Exposure Model

9 The exposure model for piscivorous birds focuses on the ingestion of tPCBs and 2,3,7,8-TCDD 10 TEQ through the diet. Other exposure routes (e.g., water, air) were considered to be of much 11 less importance for tPCBs and TEQ, and were excluded from the analyses. The equation used to 12 estimate exposure was adapted from the *Wildlife Exposure Factors Handbook* (EPA 1993) and 13 related publications:

14
$$TDI = FT \times FIR\sum_{i=1}^{n} C_i \times P_i$$
 (Eq. 1)

15 where

- 16 TDI = Total daily intake (mg/kg bw/d tPCBs, ng/kg bw/d TEQ).
- 17FT= Foraging time in the PSA (unitless).18FIR= Normalized food intake rate (kg/kg bw/d).19 C_i = Concentration in the *i*th prey item (mg/kg for tPCBs, ng/kg for TEQ).20 P_i = Proportion of the *i*th prey item in the diet (unitless).2122

Because of differences in the size of their foraging ranges, exposure analyses for kingfishers
 were conducted separately for Reaches 5 and 6, and analyses for ospreys were conducted for the

PSA as a whole. The upstream reference area and Threemile Pond reference area were included
 for comparative purposes.

3 Monte Carlo and probability bounds analyses were used to propagate input variable uncertainties 4 through the exposure model for each COC. Descriptions of these techniques and the methods 5 used to parameterize input variables are presented in Section 6.5. The results of the Monte Carlo 6 analysis are used to estimate the probability of exposure exceeding an effects threshold or levels 7 that cause adverse effects of differing magnitudes. The probability bounds analysis is conducted 8 to determine how uncertainty regarding the distributions of the input variables influences the 9 estimated exposure distribution. The results of these analyses are discussed in detail in 10 Appendix H.

11 Input distributions to the exposure analyses were generally assigned as follows:

- Lognormal distributions for variables that were right skewed with a lower bound of zero and no upper bound (e.g., amount of COC transferred from mother to offspring via egg tissue for tree swallows).
- Beta distributions for variables bounded by zero and one (e.g., proportion of a prey item in the diet).
- Normal distributions for variables that were symmetric and not bounded by one (e.g., body weight).
- Point estimates for minor variables or variables with low coefficients of variation.

In certain situations (e.g., poor fit of data), other distributions were fit to the data or other approaches were used.

The input variable distributions used in the exposure models for piscivorous birds are depicted in Figures 8.3-1 and 8.3-2 and are summarized in the following sections. These distributions are also presented in greater detail in Appendix H.

25 **8.3.1.1** Foraging Time (FT)

The foraging ranges of the two representative species are within the size of the PSA. Prey availability and an abundance of suitable foraging habitat suggest that the birds that forage in the PSA are able to meet their needs exclusively within this section of the river and floodplain. The foraging range of kingfishers is relatively small (Salyer and Lagler 1946; Brooks and Davis 1987). Although the foraging radius of ospreys may be as large as 16 km (Clark 1995), foraging range is reduced when prey are readily available near the nesting site (Clark 1995; Newton 1979). The assessment of risk to piscivorous birds inhabiting the PSA of the Housatonic River therefore focuses on those birds that spend 100% of their time foraging within the PSA. Foraging time is a point estimate and is not shown in Figures 8.3-1 and 8.3-2.

8 8.3.1.2 Body Weight (BW)

Body weights of belted kingfishers vary only slightly with sex (Hamas 1994). Dunning (1993)
reported a body weight range of 125 to 215 g with a mean of 148 g. Mean body weights have
been reported close to this value by other investigators as well (Alexander 1977; Salyer and
Lagler 1946; Brooks and Davis 1987). The distribution of the body weights of belted kingfishers
is depicted in Figure 8.3-1.

Female ospreys are generally larger than males, weighing an average of 1.6 kg and 1.4 kg, respectively (Rattner et al. 2001). Poole (1985) studied ospreys in Massachusetts and found that females ranged from 1.7 to 1.9 kg in weight during the breeding season, while males were about 1.4 kg. Brown and Amadon (1968) observed body weights of 1.2 to 1.9 kg for females and 1.2 to 1.6 kg for males in Nova Scotia. The distribution of the body weights of ospreys is depicted in Figure 8.3-2.

20 8.3.1.3 Food Intake Rate (FIR)

The food intake rate of belted kingfishers has not been well characterized. Food ingestion rate data were available in EPA (1993), however this information was not appropriate for use in this exposure model. These data lacked body weight information, lacked statistical analyses, were estimates themselves, and/or were collected from young or captive birds. The field-based estimate of the daily food intake rate of free-living adult kingfishers (0.50 g/g-day, Alexander 1977) was close to the 30th percentile of the modeled food intake rate (see below) for these birds.



1 2

Figure 8.3-1 Exposure Model Input Distributions for Belted Kingfisher





1





Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating the metabolic
 rate of free-living birds using the following general equation:

3

$$FMR (kJ/day) = a \times BW(g)^{b}$$
(Eq. 2)

The slope (*a*) and power (*b*) terms in Equation 2 were based on the error statistics derived from regression analysis of the data reported in Nagy et al. (1999). There were insufficient data to generate an allometric equation for Coraciiformes, of which belted kingfishers are members, so the equation for all birds was used. The slope term *log a* had a mean of 1.02 and a standard error of 0.0393 in log₁₀ units, and the slope term *b* had a mean of 0.681 and a standard error of 0.0182 (Nagy et al. 1999).

10 The food intake rate of ospreys has not been well characterized either. Food ingestion rate data 11 were available in EPA (1993), however this information was not appropriate for use in this 12 exposure model. These data lacked body weight information, lacked statistical analyses, were 13 estimates themselves, and/or were collected from young or captive birds. The field-based measurements of the daily food intake rate of adult male ospreys (0.21 g/g-day, Poole 1983) 14 were close to the 25th percentile of the modeled food intake rate described below. There were 15 16 insufficient data to generate an allometric equation for Falconiformes, of which ospreys are 17 members, so the equation for Charadriiformes was used. This Order includes many piscivorous 18 birds and was thought to be a suitable surrogate group. The slope term log a had a mean of 19 0.928 and a standard error of 0.197 in \log_{10} units, and the power term b had a mean of 0.768 and 20 a standard error of 0.0874 (Nagy et al. 1999). These input variable distributions are depicted in 21 Figures 8.3-1 and 8.3-2. The body weights (BW) for these birds are described above. The results 22 of the calculation were then converted to kcal/kg bw/d.

1 Food intake rate is derived from *FMR* using the following equation:

$$FIR(kg / kg bw / day) = FMR / \sum_{i=1}^{n} (AE_i \times GE_i)$$
(Eq. 3)

3 where AE_i is the assimilation efficiency of the *i*th food item (unitless) and GE_i is the gross energy 4 in the *i*th food item (kcal/g). For kingfishers, mean assimilation efficiencies were 77% for 5 aquatic invertebrates and 79% for fish, and the mean gross energies were 1.1 kcal/g wet weight 6 for aquatic invertebrates and 1.2 kcal/g wet weight for fish. For ospreys, the mean assimilation efficiency of fish was 79% and the mean gross energy of fish was 1.2 kcal/g wet weight (EPA 7 1993). Point estimates were used for AE_i and GE_i in the Monte Carlo and probability bounds 8 9 analyses because of their relatively small coefficients of variation (i.e., CV < 10%). As a result, 10 these input variables are not included in Figures 8.3-1 and 8.3-2.

11 8.3.1.4 Proportion of Dietary Items (P_i)

2

12 The principal prey of kingfishers is fish, but they also feed on berries and a variety of other small 13 animals, including mollusks, crustaceans, insects, amphibians, reptiles, young birds, and small 14 mammals (Hamas 1994). Fish prey species are those that typically live in shallow water or near 15 the surface (Hamas 1994) and include trout, salmon, suckers, perch, minnows, killifish, sticklebacks, and others (EPA 1993). Fish and crayfish are the primary prey items for 16 17 kingfishers, with other items expected to contribute little to the diet. The exposure model, 18 therefore, uses a diet with a mean composition of 86% fish and 14% crayfish in Reach 5. 19 Distributions of the proportion of kingfishers' dietary items in Reach 5 are depicted in Figure 20 8.3-1. In Woods Pond and Threemile Pond, crayfish were not included in the kingfisher diet. 21 The primary reasons for this include:

The lack of observations of crayfish when conducting other field surveys during the last 3 years at these locations.
The presence of aquatic vegetation, which conceals crayfish from kingfishers.
The abundance of cyprinids and centrachids of forage size, which live in the shallow areas and are visually attractive to hunting kingfishers.

Given these factors, it was assumed that fish would replace crayfish in the diet of kingfishers
 foraging in the areas around Woods Pond and Threemile Pond. Therefore, the percent
 contribution of fish in the diet was assumed to be 100%, and is not depicted in Figure 8.3-1.

4 Ospreys prefer to forage in shallow waters in lakes and rivers where fish occur near the surface 5 and may be easily seen (DeGraaf and Yamasaki 2001). The birds are almost exclusively 6 piscivorous, preferring medium-sized fish (13 to 40 cm in length) (Vana-Miller 1987). On rare 7 occasions, ospreys will take dead fish or prey on small mammals, reptiles, and crustaceans 8 (Chubbs and Trimper 1998). For this exposure assessment, it was assumed that fish account for 9 100% of the osprey diet. Proportion of fish in the osprey diet is a point estimate and therefore, is 10 not shown in Figure 8.3-2.

11 8.3.1.5 Concentration of COCs in Dietary Items (C_i)

12 The concentrations of tPCBs and TEQ in the prey of piscivorous birds are illustrated in Figures 13 8.3-3 to 8.3-6. The bars in these figures depict the median concentration of each COC in each 14 dietary item in each of the areas for the risk assessment. The stars depict the arithmetic mean 15 and the vertical lines depict the interquartile ranges for the concentrations. The concentrations of 16 COCs used in the exposure analyses are shown in Tables H.2-4, H.2-5, H.2-12, and H.2-13 of 17 Appendix H. Rationales for the concentration variables are also provided in Appendix H. As 18 evident in these figures, the concentrations of tPCBs and TEQ in prey items of piscivorous birds 19 are highest within the PSA and substantially lower in the reference areas.

20 8.3.2 Exposure Model Results

The exposure model results for belted kingfishers and ospreys exposed to tPCBs and TEQ are
discussed in greater detail in Section 2 of Appendix H.

23 Belted kingfishers had the highest modeled exposure to tPCBs in Reach 5 (Figure 8.3-7) and

Reach 6 (Figure 8.3-8), whereas exposures in the reference areas (Figures 8.3-9 and 8.3-10) were

25 substantially lower. Belted kingfishers had the highest modeled exposure to TEQ in Reach 5



Notes: Error bars indicate interquartile range. URA = Upstream reference area; TMP = Threemile Pond reference area.

Figure 8.3-3 Concentrations of tPCBs in the Prey of Belted Kingfishers in the Housatonic River PSA and Reference Areas





13

14

Figure 8.3-4 Concentrations of TEQ in the Prey of Belted Kingfishers in the Housatonic River PSA and Reference Areas

URA = Upstream reference area; TMP = Threemile Pond reference area.



Notes: Error bars indicate interquartile range. URA = Upstream reference area; TMP = Threemile Pond reference area.

Figure 8.3-5 Concentrations of tPCBs in the Prey of Ospreys in the Housatonic River PSA and Reference Areas



Figure 8.3-6 Concentrations of TEQ in the Prey of Ospreys in the Housatonic River PSA and Reference Areas

1

7

8

9 10

11

12

13

(Figure 8.3-11) and Reach 6 (Figure 8.3-12), whereas exposures in the reference areas (Figures
 8.3-13 and 8.3-14) were again substantially lower.

3 The exposure distributions for ospreys exposed to tPCBs in the PSA, upstream reference area, 4 and the Threemile Pond reference area are presented in Figures 8.3-15, 8.3-16, and 8.3-17, 5 The exposure distributions for ospreys exposed to TEQ in the PSA, upstream respectively. 6 reference area, and the Threemile Pond reference area are presented in Figures 8.3-18, 8.3-19, 7 and 8.3-20, respectively. Ospreys had the highest modeled exposure to tPCBs and TEQ in the 8 PSA, while exposures in the reference areas were substantially lower. The differences in the 9 exposure estimates between the PSA and reference areas may be explained in great part by the 10 differences in COC concentrations in prey items at the sites, as depicted in Figures 8.3-3 to 8.3-6.

Overall, ospreys foraging in the PSA are expected to have the highest exposure to tPCBs and TEQ of the representative piscivorous bird species, followed by belted kingfishers. Tables H.2-6 and H.2-14 present a summary of tPCB exposure model results for belted kingfishers and ospreys, respectively. Results for the TEQ exposure model for piscivorous birds are presented in Tables H.2-7 and H.2-15, respectively. A complete account of the exposure model results, including Monte Carlo and probability bounds analyses and figures and tables, is presented in Appendix H.









UPB = Upper probability bound

Figure 8.3-8 Exposure of Belted Kingfishers to tPCBs in Reach 6 of the Housatonic River PSA











LPB = Lower probability bound UPB = Upper probability bound

Figure 8.3-10 Exposure of Belted Kingfishers to tPCBs in the Threemile Pond Reference Area





14

7/10/2003









Figure 8.3-14 Exposure of Belted Kingfishers to TEQ in the Threemile Pond Reference Area



MK01|O:\20123001.096\ERA_PB\ERA_PB_8.DOC

Reference Area





LPB = Lower probability bound UPB = Upper probability bound

Figure 8.3-17 Exposure of Ospreys to tPCBs in the Threemile Pond Reference Area



11Figure 8.3-18 Exposure of Ospreys to TEQ in Reaches 5 and 6 of the Housatonic12River PSA

13

7

8 9

Upstream Reference Area









1 8.4 EFFECTS ASSESSMENT

The purpose of the effects assessment is twofold. The first is to review the scientific literature for effects of PCB mixtures (mainly Aroclor 1254 and 1260 mixtures) and TEQ to piscivorous birds. Of primary interest are documented effects to the representative species in this assessment: ospreys and belted kingfishers. In the absence of data for these species, other avian species were considered. The other purpose is to derive the effects metrics that will be used, in conjunction with the exposure assessment results, to estimate risks to piscivorous birds from exposure to COCs in the Housatonic River PSA.

9 The COCs in this assessment are tPCBs and 2,3,7,8-TCDD toxic equivalence (TEQ). The 10 congeners used to estimate TEQ concentrations share the ability to bind to the aryl hydrocarbon 11 (Ah) receptor protein (Bosveld and Van den Berg 1994) and elicit an Ah-receptor-mediated 12 biochemical and toxic response. The toxic response to this group of chemicals is related to the 13 three-dimensional structure of the substance, including the degree of chlorination and positions 14 of the chlorine on the aromatic frame (Van den Berg et al. 1998; Newsted et al. 1995; Safe 15 1994).

Sensitivities of avian species to tPCBs and TEQ have been shown in the literature studies to vary greatly. Wild turkey embryos were found to be 20 to 100 times less sensitive than chicken embryos to the egg yolk injection of PCB-77. This difference in toxicity may be attributed to differences in availability of Ah receptors. Ah receptors were found in hepatocytes of 7-day-old chicken embryos, but not in liver cells of 9-day-old turkey embryos (Brunstrom and Lund 1988). Figures 8.4-1 and 8.4-2 illustrate the ranges of effects of tPCBs and TEQ, respectively, to various avian species.

1	Toxicity of tPCE	s and TEQ to Avian Species		
2	Mode of Action			
3 4	Binding to the aryl-hydrocarbon (Ah) receptor, eliciting an Ah-receptor-mediated biochemical and toxic response.			
5	Types of Toxicity	Specific Effects		
6 7 8 9 10 11	hepatotoxicity immunotoxicity neurotoxicity embryotoxicity teratogenicity	mortality decreased growth weight loss porphyria reduced hatching embryo mortality		

13 8.4.1 Total PCBs

Heath et al. (1972) studied the effects of Aroclor 1254 on mortality on four avian species. The most sensitive (after oral dosing for 5 days with Aroclor 1254) was the bobwhite quail, with a median lethality response occurring at a dietary PCB concentration of 604 mg/kg. Other species tested, such as the Japanese quail, mallard duck, and ring-necked pheasant, were less sensitive, with oral LC_{50} s of 2,898, 2,699, and 1,091 mg/kg diet, respectively. Prestt et al. (1970) estimated the median lethal dietary dose rate of Aroclor 1254 to adult Bengalese finches to be 256 mg/kg/d.

21 Reproductive impairment of birds caused by tPCBs has been investigated in several species, in 22 dietary and egg injection studies, as well as field studies examining egg and hatchling 23 concentrations and hatching success. The most commonly noted effects to the reproduction of 24 avian species are reduced egg productivity, egg hatchability, and chick growth rates (CCME 25 1999). Of the species studied, chickens appear to be the most sensitive, followed by pheasants, 26 turkeys, ducks, and herring gulls (Bosveld and Van den Berg 1994). Total PCBs appear to have 27 no adverse effects on total egg weight, eggshell weight, or eggshell thickness (Lillie et al. 1974; 28 Britton and Huston 1973; Scott 1977). Lillie et al. (1974) exposed hens to 2 mg/kg Aroclor 1254 29 for 63 days in feed, giving birds a daily PCB dose of approximately 0.12 mg/kg bw/d. At this 30



1 2 Figure 8.4-1 Effects of Aroclor 1254/1260 on Avian Species (mg/kg bw/d)



Figure 8.4-2 Effects of 2,3,7,8-TCDD TEQ on Avian Species (ng TEQ/kg bw/d)

treatment level, no significant effects were observed on fertility, egg production, shell thickness, or hatchability, but the growth rate of chicks was slightly reduced. American kestrels were given an approximate tPCB dose of 7 mg/kg bw/d for 100 days (Fernie et al. 2001) and birds experienced a slight, but statistically insignificant, decrease in clutch size and the numbers of fertile eggs, hatchlings, and fledglings per breeding pair.

6 8.4.2 TEQ

In single, oral doses of TCDD, bobwhite quail, mallards, and ringed turtledoves were found to have 37-day LD₅₀s of 15,000, 108,000, and 810,000 ng/kg bw (Hudson et al. 1984). Ringnecked pheasants given intraperitoneal TCDD doses weekly of 10, 100, or 1,000 ng/kg bw/week for 10 weeks (Nosek et al. 1992) experienced no mortality or body weight effects in the two lowest treatment groups, but the 1,000 ng/kg bw/week treatment group experienced 60% mortality by the 23rd week (13 weeks after the dosing period).

13 Nosek et al. (1992a) also investigated reproductive effects of TCDD to ring-necked pheasant hens. The two lower doses (10 and 100 ng/kg bw/week) caused no significant impairment to egg 14 15 production. The highest dose, 1,000 ng/kg bw/week, caused a decrease in cumulative egg 16 production of approximately 70% over 7 weeks. The geometric mean of the lowest observed adverse effect level (LOAEL) and no observed adverse effect level (NOAEL) from this study 17 18 was 44 ng/kg bw/d. Hoffman et al. (1996) investigated the developmental effects of TEQ on 19 American kestrels and observed that skeletal growth was significantly reduced at a treatment 20 level of 25,000 ng TEQ/kg bw/d. This dose did not translate into significant effects on hatchling 21 success or weight gain.

22 8.4.3 Effects Metrics

Effects data are ideally summarized as dose-response curves for each representative species. For this assessment, however, data were insufficient to generate dose-response curves, NOAELs and LOAELs, or field based measures of effect. Therefore, a threshold range for surrogate species was used to represent the effects of tPCBs and TEQ to piscivorous birds. This approach establishes a range of toxic effects thresholds for the most sensitive and tolerant avian species known and assumes that the thresholds for the representative species are within these bounds. 1 Further details on the decision criteria used in selecting effects metrics are presented below and

2 in Section 6.6 of the ERA.

3 8.4.3.1 Total PCBs

Based on a review of avian toxicity literature, white leghorn chickens were the most sensitive avian species to the reproductive effects of tPCBs (Lillie et al. 1974) and the most reproductively tolerant avian species to tPCBs was the American kestrel (Fernie et al. 2001). The resulting threshold range for the reproductive success of piscivorous birds exposed to tPCBs selected for this assessment is 0.12 to 7.0 mg/kg bw/d.

9	Decision Criteria for Derivation of Effects Metric
10 11	The following is the hierarchy of decision criteria used to characterize effects for each receptor-COC combination:
12	 Have single-study bioassays with five or more treatments been conducted on the
13	receptor of interest or a reasonable surrogate? If yes, estimate the
14	concentration- or dose-response relationship. If not, go to step 2.
15	 Are multiple bioassays with similar protocols, exposure scenarios, and effects
16	metrics available that, when combined, have five or more treatments for the
17	receptor of interest or a reasonable surrogate? If yes, estimate the dose-
18	response relationship as in step 1. If not, go to step 3.
19	 Have bioassays with less than five treatments been conducted on the receptor of
20	interest or a reasonable surrogate? If yes, conduct or report results of
21	hypothesis testing to determine the NOAEL and LOAEL. If not, go to step 4.
22	 Are sufficient data available from field studies and monitoring programs to
23	estimate concentrations or doses of the COC that are consistently protective or
24	associated with adverse effects? If yes, develop field-based effects metrics. If
25	not, go to step 5.
26	 Derive a range where the threshold for the receptor of interest is expected to
27	occur. Because information on the sensitivity of the receptor of interest is
28	lacking, it is difficult to derive a threshold that is neither biased high nor low. If
29	bioassay data are available for several other species, however, calculate a
30	threshold for each to determine a threshold range that spans sensitive and
31	tolerant species. That range is likely to include the threshold for the receptor of
32	interest.

1 8.4.3.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

The lower toxicological threshold for the effects of TEQ to sensitive birds is based on ringnecked pheasants (Nosek et al. 1992) and the upper threshold for tolerant species is based on the American kestrel (Hoffman et al. 1996). The resulting threshold range for the reproductive success of piscivorous birds exposed to TEQ is 44 to 25,000 ng/kg bw/d.

6 8.4.4 Belted Kingfisher Field Study

7 A belted kingfisher reproduction study was performed in the PSA during the 2002 breeding 8 season (Henning 2002). The objective of the study was to evaluate the relationship between 9 reproduction of kingfishers and exposure of adult and nestling kingfishers to tPCBs. Nine belted 10 kingfisher burrows were monitored during this study, three of which were depredated before the 11 young could fledge. In the remaining six nests, there was an average of 4.8 nestlings, or 87%, 12 that survived from egg to 26 days. When depredated nests were excluded, fledging rates were 13 consistent with the results of the only other kingfisher study reported in the literature (Brooks 14 and Davis 1987). Total daily intake of tPCBs was estimated based on prey concentrations and 15 food ingestion rates. No significant relationships were observed between estimated tPCB dose 16 and reproductive output (p>0.05), although this does not necessarily support a conclusion of no 17 adverse effects to the reproductive success of belted kingfishers. See Section 8.5.2 and Section 18 H.4.2 of Appendix H for discussion of this study.

1 8.5 RISK CHARACTERIZATION

This section characterizes risks to piscivorous birds exposed to tPCBs and 2,3,7,8-TCDD TEQ in the PSA of the Housatonic River. The risk characterization includes a comparison of probabilistic exposure estimates to relevant effect metrics, a review of the findings of the belted kingfisher field study, a summary of the weight-of-evidence (WOE) assessment, a discussion of the sources of uncertainty that may influence the estimates of risk, and a discussion of risks to other piscivorous birds foraging in the PSA.

	Risk Questions			
ŀ	Are the concentrations of tPCBs and TEQ present in the prey of piscivorous birds sufficient to cause adverse effects to individuals inhabiting the PSA of the Housatonic River?			
•	If so, how severe are the risks and what are their potential consequences?			
	Lines of Evidence			
•	Probabilistic exposure and effects modeling.			
	Field study of the reproductive success of belted kingfishers in the PSA.			

16

17 8.5.1 Comparison of Estimated Exposures to Laboratory-Derived Effect Doses

For piscivorous birds, exposure was assessed separately in Reaches 5 and 6 for belted kingfishers and in Reaches 5 and 6 combined for ospreys. For each receptor-COC-area combination, a category of low, intermediate, or high risk was assigned using the guidance below, when integrating the exposure and effects distributions.

22	Guidance for Integrating the Exposure and Effects Distributions
23 24	 If the probability of exceeding the lower toxicity threshold is less than 20%, the risk is considered to be low.
25 26	 If the probability of exceeding the upper toxicity threshold is greater than 20%, the risk is considered to be high.
27	 All other outcomes are considered to have intermediate risk.

1 This exercise was done separately for the results of the Monte Carlo analyses and the lower and 2 upper bounds from the probability bounds analyses. The "risk category" refers to the level of 3 risk based on the results of the Monte Carlo analysis. The "risk range" refers to the levels of risk 4 based on the results of the probability bounds analyses.

5 The results of the risk characterization, as described by the modeled exposure and effects line of 6 evidence, are summarized in Table 8.5-1. The highest risk to piscivorous birds is from exposure 7 to tPCBs in the PSA, with low-intermediate risk in the reference areas. As shown in Figure 8 8.3-7, the tPCB exposure curve for ospreys in the PSA is well above the upper toxicity threshold. 9 This means that the estimated daily intake of tPCBs by ospreys is greater than the intake known 10 to cause adverse effects in the most tolerant bird species studied. The risk category for ospreys is 11 high in the PSA and the risk range is also high (Table 8.5-1).

12 The highest risk to kingfishers is from exposure to tPCBs in Reaches 5 and 6. Both 13 representative piscivorous bird species were determined to be at intermediate risk to TEQ in the 14 PSA. The complete characterization of risks of piscivorous birds to tPCBs and TEQ is presented 15 in Appendix H.

16 8.5.2 Belted Kingfisher Field Study

A study of belted kingfisher reproduction in the PSA was performed by GE during the 2002
breeding season. The objective of the study was to determine the relationship between tPCB
dose and reproductive success. More information on this study can be found in Henning (2002).

20 Active kingfisher burrows were sought in the river banks and riparian zone of the PSA. In each 21 burrow, the number of nestlings and parental behavior were recorded. According to the report, 22 the total daily intake of tPCBs by kingfishers was estimated based on the concentration of COCs 23 in fish and crayfish samples taken within 1,200 m of each burrow and on prey ingestion rates of 24 adults and nestlings obtained from the literature. Crayfish samples were associated with specific 25 sampling locations, but fish samples were not. When developing the estimated dose, Henning 26 assumed a more precise location of a fish sample than is possible from the information 27 associated with the sample. River miles designated in the sample IDs in the database were

Table 8.5-1

Summary of Qualitative Risk Statements for Piscivorous Birds from the Housatonic River PSA

	Qualitative Risk Statements				
Bird / Location	tPCBs		TEQ		
Diru / Location	Risk Category Risk Range		Risk Category	Risk Range	
Belted Kingfisher					
Reach 5	High	High	Intermediate	Intermediate	
Reach 6	High	High	Intermediate	Intermediate	
Upstream Reference Area	Intermediate	Low/intermediate	Intermediate	Low/intermediate	
Threemile Pond	Low	Low	Low	Low	
	1				
Osprey					
Reaches 5 and 6	High	High	Intermediate	Intermediate	
Upstream Reference Area	Intermediate	Intermediate	Low	Low	
Threemile Pond	Low	Low	Low	Low	

5

6
representative of larger areas of the river, roughly corresponding to a segment of Reach 5A
 referred to in sampling records as "Shallow Reach" or Reaches 5B and 5C combined ("Deep
 Reach").

4 The total daily intake calculation did not result in a dose gradient necessary to evaluate a dose-5 response relationship for piscivorous birds consuming Housatonic River fish. First, the fish 6 sampling location was known only at the resolution of a range of river miles (i.e., a river reach 7 level); therefore, an actual river mile cannot be assumed for a sample. Second, fish species 8 sampled may be mobile between life stages and seasons, and have integrated contaminant 9 concentrations across these areas. These factors contribute to an averaging of the tPCB 10 concentrations in prey used to evaluate exposure by kingfishers, and result in a very narrow 11 exposure gradient, with total daily intake for adult birds ranging from 7.4 to 21 mg/kg bw/d in 12 the GE kingfisher field study (Henning 2002).

13 Although nine belted kingfisher burrows were monitored during this study, three of which were 14 depredated before the fledging date. For the remaining nests, there was an average of 4.8 15 nestlings, or 87%, that survived to 26 days. If depredated nests were included in the analysis, the 16 average decreased to 3.9 young per nest, or 58% surviving to 26 days. Estimated tPCB doses for 17 adults and young in the PSA were 13 and 35 mg/kg bw/d, respectively. Doses ranged from 7.4 18 to 21 mg/kg bw/d for adults and 20 to 57 mg/kg bw/d for nestlings. Henning (2002) reported 19 that there were no significant relationships between estimated tPCB dose and any of the 20 endpoints (p>0.05). The range of estimated total daily intakes in the PSA was narrow, however, 21 and could not be replicated from the data, providing insufficient basis on which to evaluate a 22 dose-response relationship. Multivariate models also indicated that combined independent 23 variables (e.g. nest density, tPCB dose) provided no significant relationship between stressors 24 and reproductive effects. The results were similar when depredated nests were included in the 25 analyses. The results of this analysis are similarly confounded as in the previously described 26 analyses, because of the same range of daily tPCB intakes in the PSA.

The kingfisher population in the Housatonic River appears to be breeding successfully, with fledging rates and population densities that, when depredated nests are excluded, are similar to what was reported in the only other comparable study from the literature (Brooks and Davis 1 1987). The lack of data from reference areas for comparison to the observations in the PSA 2 introduces uncertainty in interpretation of this study. Small sample size also introduces some 3 uncertainty, as only nine nests, six of which were successful, were observed in the study. The 4 model used to estimate the total daily intake of tPCBs has limited applicability as it was not 5 possible to attain a sufficiently wide dose gradient. One additional nest was observed during 6 oversight of the study by EPA contract staff (Woodlot Alternatives, Inc. 2002), but was 7 apparently not included in the study.

8 8.5.3 Weight-of-Evidence Analysis

9 A WOE analysis procedure was used to assess risks of tPCBs and TEQ to piscivorous birds. The 10 goal of this analysis was to determine whether significant risk is posed to piscivorous birds in the 11 Housatonic River PSA as a result of exposure to tPCBs and TEQ. The three-phase approach of 12 Menzie et al. (1996) and the Massachusetts Weight-of-Evidence Workgroup was applied for this 13 purpose, in which WOE was reflected in the following three characteristics: (1) the weight 14 assigned to each measurement endpoint; (2) the magnitude of response observed in the 15 measurement endpoint; and (3) the concurrence among outcomes of the multiple measurement 16 endpoints (see Section 2.9 for details).

The rationale for evaluating measurement endpoints is provided in Section 2.9 and Appendix H, along with a discussion of attributes considered in the WOE. The measurement endpoint weighting scores are presented in Table 8.5-2 and evidence of harm and magnitudes of responses are presented in Tables 8.5-3 and 8.5-4 for tPCBs and TEQ, respectively. For both tPCBs and TEQ, the modeled exposure and effects line of evidence was given a moderate weighting. The belted kingfisher field study was given a moderate/high weighting.

7

8 9

10

Table 8.5-2

Weighting of Measurement Endpoints for Piscivorous Birds Weight-of-Evidence Evaluation

Attributes	Modeled Exposure and Effects for tPCBs and TEQ	GE Kingfisher Field Study (Henning 2002)
I. Relationship Between Measuremen	t and Assessment Endp	oints
1. Degree of Association	М	Н
2. Stressor/Response	М	М
3. Utility of Measure	М	М
II. Data Quality		
4. Data Quality	M/H	М
III. Study Design		
5. Site Specificity	L/M	M/H
6. Sensitivity	L/M	L/M
7. Spatial Representativeness	М	M/H
8. Temporal Representativeness	М	М
9. Quantitative Measure	M/H	М
10.Standard Method	М	M/H
Overall Endpoint Value	М	M/H

11 12 13

H = high

Table 8.5-3

Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	
Modeled Exposure and Effects	М	Yes (Kingfisher) Yes (Osprey)	High (Kingfisher) High (Osprey)	
Belted Kingfisher Field Study (Henning 2002)	M/H	No (Kingfisher)	Low (Kingfisher)	

5

.

6

7

8 9

Table 8.5-4

Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	
Modeled Exposure and Effects	М	Yes (Kingfisher) Yes (Osprey)	Intermediate (Kingfisher) Intermediate (Osprey)	
Belted Kingfisher Field Study (Henning 2002)	M/H	No (Kingfisher)	Low (Kingfisher)	

10

The magnitude of the response in the measurement endpoint is considered together with the measurement endpoint weight in judging the overall WOE (Menzie et al. 1996). This requires assessing the strength of evidence that ecological harm has occurred, as well as an indication of the magnitude of response, if present. For both tPCBs (Table 8.5-3) and TEQ (Table 8.5-4), the modeled exposure and effects line of evidence indicated that there was evidence of harm, and that the magnitude was high. The belted kingfisher field study (Henning 2002) indicated that there was no evidence of adverse effects to productivity, and the magnitude was low.

A graphical method was used for displaying concurrence among measurement endpoints. Tables
 8.5-5 and 8.5-6 depict the outcome for belted kingfishers and ospreys exposed to tPCBs and
 TEQ. The analyses were conducted separately for ospreys and belted kingfishers exposed to

Table 8.5-5

Risk Analysis Summary for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSA

		Weighting Fa	ctors (increasin	g confidence of we	ight)
Harm/Magnitude	Low	Low/ Moderate	Moderate	Moderate/ High	High
Yes/High			MEE-KF MEE-O		
Yes/Intermediate					
Yes/Low					
Undetermined/High					
Undetermined/Intermediate					
Undetermined/Low					
No/Low				FS-KF	
No/Intermediate					
No/High					

MEE = Modeled exposure and effects FS = Field study KF = Kingfisher

12

O = Osprey

1	
2	Table 8.5-6
3	
4	Risk Analysis Summary for Piscivorous Birds Exposed to TEQ in the Housatonic
5	River PSA
6	
	Assessment Endpoint: Survival, growth, and reproduction of piscivorous birds.
7	

		Weighting Fa	ctors (increasin	g confidence of we	eight)	
Harm/Magnitude	Low	Low/ Moderate	Moderate	Moderate/ High	High	
Yes/High						
Yes/Intermediate			MEE-KF MEE-O			
Yes/Low						1
Undetermined/High						
Undetermined/Intermediate						
Undetermined/Low						
	•	•		· · ·		_
No/Low				FS-KF		Τ.

9

No/Low		FS-KF	┫
No/Intermediate			
No/High			

MEE = Modeled exposure and effects FS = Field study KF = Kingfisher

O = Osprey

tPCBs and TEQ. There is moderate confidence, because only one line of evidence is available, 1 2 that ospreys foraging in the PSA are subject to risk from exposure to tPCBs and TEQ (Appendix 3 H, Section 4.3). Belted kingfishers were judged, with moderate confidence, to be at low risk 4 from exposure to tPCBs and TEQ in the PSA based on the Kingfisher field study. Based on the 5 modeled exposure and effects analysis, there is moderate confidence that Kingfishers are 6 adversely affected by exposure to tPCBs and TEQ. Confidence in this conclusion is not high 7 because the two lines of evidence (modeled exposure and effects, kingfisher field study) 8 produced conflicting risk estimates. Risks in the reference areas for these COCs are generally 9 low.

10 8.5.4 Sources of Uncertainty

The assessment of risk to piscivorous birds contains uncertainties. Each source of uncertainty can influence the estimates of risk, therefore, it is important to describe and, when possible, specify the magnitude and direction of such uncertainties. The sources of uncertainty associated with the assessment of risks of tPCBs and TEQ to piscivorous birds are described as follows.

- 15 The Monte Carlo sensitivity analyses suggested that the free metabolic rate (FMR) slope and power terms were generally the most influential variables on predicted total 16 daily intakes of COCs. However, no suitable direct measurements of free metabolic 17 rate are available for the representative wildlife species. Similarly, suitable measured 18 19 food intake rates are not available for free-living belted kingfisher and osprey. 20 Therefore, free metabolic rates were estimated using allometric equations. The use of 21 allometric equations introduces some degree of uncertainty into the exposure 22 estimates because they are subject to model-fitting error, and are based on species different from the representative species used in this assessment. Given the lack of 23 24 empirical data on species specific to this assessment, it is difficult to judge the 25 magnitude of the uncertainty introduced by the use of the allometric models. The 26 uncertainty due to model-fitting error was propagated in the uncertainty analyses by 27 using distributions as input for the allometric slope and power terms.
- 28 Sample sizes were limited for the analyses of COC concentrations in some prey 29 items, specifically, crayfish. To address this uncertainty in the Monte Carlo analysis, 30 the UCL or data set maximum (see Section 6.4 and Appendix C.5) was used as an 31 estimate of COC concentrations in prey items. The potential magnitude of the 32 uncertainty associated with small sample sizes for COC concentrations is unknown, 33 but this approach likely overestimates exposure. The probability bounds analysis 34 used an unbiased approach (e.g., distribution free range from lower confidence limit [LCL] to upper confidence limit [UCL]) to deal with sample size uncertainty. 35

- PCB congeners 123 and 157 co-eluted with other congeners (PCB-123 with PCB-149; PCB-157 with PCB-173 and PCB-201). As a result, decision criteria were developed (see Section 6.4) for co-eluted congeners to determine TEQ concentrations used as distribution parameters in the Monte Carlo and probability bounds analyses. These criteria were designed to explicitly incorporate this source of uncertainty in the probabilistic analyses. Thus, this source of uncertainty has been incorporated in this risk assessment.
- 8 The greatest source of uncertainty of the effects assessment was associated with the 9 lack of toxicity studies involving the representative species. There were no toxicity 10 studies available for belted kingfishers or ospreys exposed to tPCBs or TEQ. As a result, laboratory studies involving other species were used to estimate effects to 11 piscivorous birds. This extrapolation introduced uncertainty in the effects assessment 12 13 because of the variations in physiological and biochemical factors that can alter the 14 potential toxicity of a contaminant. The sensitivity of wild birds to an environmental contaminant may differ from that of a laboratory or domestic species due to 15 16 behavioral and ecological parameters including stress factors (e.g., competition, 17 seasonal changes in temperature or food availability), disease, and exposure to other 18 contaminants. Inbred laboratory animal strains may also have an unusual sensitivity 19 or resistance to a tested substance. To address uncertainty in the effects assessment, a 20 threshold range was used in which effects to tolerant and sensitive species were considered. It is assumed that the toxicity thresholds for the representative species lie 21 22 within these ranges.
- The belted kingfisher field study methods appeared to generally follow accepted protocols, however EPA was not provided with an opportunity to review these protocols prior to receiving the study. There were several shortcomings of the approach used. For example, there was no reference site, no information was provided regarding nest search intensity, the researchers were unable to determine clutch size, and there were too few visits to the nests during the reproductive cycle. These shortcomings limit the ability to draw rigorous conclusions.
- The statistics used in the belted kingfisher field study were not clearly stated. Student
 t-tests were apparently performed even though there were no reference sites to
 compare to. A power analysis of the results would have been useful. The sample
 sizes were very small (i.e., n=6) for the statistics used (i.e., t-test and regression).
- 34 The approach used to estimate dose in the belted kingfisher field study had a number 35 of shortcomings. The investigators assumed a foraging radius of 1,200 m and 36 attempted to identify prey samples within this radius of each burrow. The fish 37 samples had a "river mile" location associated with each sample, but this is an imprecise measure that does not allow the location of the sample with sufficient 38 39 precision to assign specific fish to a specific 1,200-m foraging radius. Fish are also 40 mobile within the PSA, meaning that they receive their total exposure from many 41 areas of the river and that concentrations in fish do not vary substantially within the 42 PSA. As a result, the dose gradient achieved by this approach is likely too narrow to 43 detect a significant dose-response relationship.

2

3

4

5

6

1 The belted kingfisher field study results do not definitively support the conclusions of 2 low risk because the data are limited. There are several conclusions drawn by the 3 authors that are not strongly supported by the information presented in the report. 4 The conclusion that the kingfisher population is consistent with the quality of habitat 5 present is speculative. Survival to 26 days and densities were compared with the 6 results from only one study (Brooks and Davis 1987). It is inappropriate to conclude 7 that the Housatonic River kingfishers fall within the range reported for other 8 kingfisher populations when only one study is referenced. Although the GE study 9 provides no evidence of impaired reproduction or population density attributable to 10 PCBs, it fails to acknowledge the limitations associated with the use of only one 11 metric to evaluate reproduction.

12 8.5.5 Extrapolation to Other Species

Belted kingfisher and osprey are the only piscivorous birds common to the area. Other, less
common, piscivorous birds (e.g., pied-billed grebe, great blue heron), are addressed via
extrapolation in Section 11 and Appendix K.

16 **8.5.6 Summary and Conclusions**

The WOE analysis indicated that exposure of piscivorous birds, such as the belted kingfisher and osprey, to tPCBs and TEQ in the PSA, could lead to adverse reproductive effects in some species. The two lines of evidence used to support this conclusion were the field study of kingfisher productivity and the comparison of modeled exposure with effects to piscivorous birds.

22 For the assessment of risks to kingfishers, both lines of evidence were employed. The modeled 23 exposure and effects line of evidence indicated that kingfishers in the PSA are likely to receive a 24 tPCB dose greater than what the most tolerant species known can endure. For TEQ, the risk is 25 less clear because the threshold range for this COC is very wide and the exposure estimates for 26 kingfishers fell within this range. Thus, without effects data specific to kingfishers, it is difficult 27 to make definitive conclusions about the risks of TEQ to this species. The field study of 28 kingfisher productivity, however, indicated that these birds are able to reproduce in the PSA. 29 This line of evidence was given a higher weighting than the exposure and effects modeling, 30 despite concerns about the field study. Therefore, kingfishers are considered to be at low risk in

the PSA as a result of exposure to tPCBs and TEQ. The conclusion of low risk to kingfishers is 1 2 uncertain because the two lines of evidence did not give concordant results.

3 For ospreys, only the modeled exposure and effects line of evidence was available to assess risk 4 to these birds. As with kingfishers, this line of evidence indicated that ospreys in the PSA are 5 likely to receive a tPCB dose that is greater than what the most tolerant species known can bear. 6 The risks due to exposure to TEQ are unclear, as the estimates for exposure also fell within 7 toxicity threshold range. Ospreys, however, lack a site-specific study that investigated the 8 effects of COCs in the PSA. The PSA contains suitable habitat for ospreys, with abundant prey, 9 raising the possibility that they are not resident in the area because of contaminants. Ospreys are 10 therefore considered to be at risk in the PSA as a result of exposure to tPCBs and TEQ.

11

ERA Results for Piscivorous Birds

The WOE analysis suggests that ospreys may be at high risk from exposure to tPCBs and intermediate risk from exposure to TEQ in the Housatonic River PSA. In the PSA, exposure of piscivorous birds to tPCBs is greater than concentrations that caused adverse effects in the most tolerant species studied. The conclusion of high risk to ospreys is uncertain because only one line of evidence was available. Belted kingfishers are considered to be at low risk as a result of exposure to tPCBs and TEQ in 14 the Housatonic River PSA. While modeled exposure and effects indicated high risk for tPCBs and intermediate risk for TEQ, a field study of kingfisher productivity indicated that the birds were reproducing in the PSA. The conclusion of low risk to kingfishers is uncertain because the two 15 lines of evidence did not give concordant results.

16

17 8.6 REFERENCES

18 Alexander, G.R. 1977. Food and vertebrate predators on waters in north central lower Michigan. 19 Michigan Academician 10:187-195.

20 Bosveld, A.T.C. and M. Van den Berg. 1994. Effects of polychlorinated biphenyls, dibenzo-p-21 dioxins, and dibenzofurans on fish-eating birds. Environmental Reviews 2:147-166.

22 Britton, W.M. and T.M. Huston. 1973. Influence of polychlorinated biphenyls in the laying hen. 23 Poultry Science 52:1620-1624.

24 Brooks, R.P. and W.J. Davis. 1987. Habitat selection by breeding belted kingfishers. American

25 Midland Naturalist 117:63-70.

- 1 Brown, L. and D. Amadon. 1968. Eagles, Hawks, and Falcons of the World. McGraw-Hill, New
- 2 York, NY, USA.
- 3 Brunström, B. and J. Lund. 1988. Differences between chick and turkey embryos in sensitivity to
- 4 3,4'4,4'-tetrachlorobiphenyl and in concentration/affinity of the hepatic receptor for 2,3,7,8-
- 5 tetrachlorodibenzo-p-dioxin. Com. Biochem. Physiol. C 91(2):507-512.
- 6 CCME (Canadian Council of Ministers of the Environment). 1999. Canadian Tissue Residue 7 Guidelines for the Protection of Wildlife Consumers of Aquatic Biota: Polychlorinated 8 Biphenyls (PCBs). In Canadian Environmental Quality Guidelines, 1999. Canadian Council of
- 9 Ministers of the Environment, Winnipeg.
- 10 Chubbs, T.E. and P.G. Trimper. 1998. The diet of nesting osprey, Pandion haliaetus, in 11 Labrador. Canadian Field-Naturalist 112:502-505.
- 12 Clark, K.E. 1995. Osprey. In *Living Resources of the Delaware Estuary*. L.E. Dove and R.M.
- Nyman (eds.). The Delaware Estuary Program, DE, USA. p. 395-400. 13
- 14 DeGraaf, R.M. and M. Yamasaki. 2001. New England Wildlife: Habitat, Natural History, and Distribution. University Press of New England, Hanover, NH, USA. 15
- 16 Dunning, J.C. Jr. 1993. CRC Handbook of Avian Body Masses. CRC Press, Boca Raton, FL.
- EPA (U.S. Environmental Protection Agency). 1993. Wildlife Exposure Factors Handbook. 17 18 EPA/600/R-93/187a. Office of Research and Development, Washington, DC.
- 19 Fernie, K.J., J.E. Smits, G.R. Bortolotti, and D.M. Bird. 2001. Reproductive success of American 20 kestrels exposed to dietary polychlorinated biphenyls. Environmental Toxicology and Chemistry 21 20:776-781.
- 22 Haffner, G.D., M. Tomczak, and R. Lazar. 1994. Organic contaminant exposure in the Lake St. 23 Clair food web. Hydrobiologia 281:19-27.
- 24 Hamas, M.J. 1994. Belted Kingfisher (Cervle alcoon). In: The Birds of North America, No. 84. 25 A. Poole and G. Gill, Editors. The Birds of North America, Inc., Philadelphia, PA.
- 26 Heath, R.G., J.W. Spann, E.F. Hill, and J.F. Kreitzer. 1972. Comparative Dietary Toxicities of 27 Pesticides to Birds. U.S. Fish and Wildlife Service Special Scientific Report on Wildlife 152. 28 57 pp.
- 29 Henning, M.H. 2002. Productivity and Density of Belted Kingfishers on the Housatonic River,
- 30 Berkshire County, Massachusetts. Prepared for: General Electric Company, Pittsfield,
- 31 Massachusetts.
- 32 Hoffman, D.J., M.J. Melancon, P.N. Klein, C.P. Rice, J.D. Eisemann, R.K. Hines, J.W. Spann,
- 33 and G.W. Pendleton. 1996. Developmental toxicity of PCB-126 (3,3,4,4,5-pentachlorobiphenyl)
- 34 in nestling American kestrels (Falco sparverius). Fundamental and Applied Toxicology
- 35 34:188-200.

- 1 Hudson, R., R. Tucker, and M. Haegele. 1984. Handbook of Toxicity of Pesticides to Wildlife.
- 2 2nd Edition. U.S. Fish and Wildlife Service Resource Publication 153. Washington, DC.
- Lillie, R.J., H.C. Cecil, J. Bitman, and G.F. Fries. 1974. Differences in response of caged white leghorn layers to various polychlorinated biphenyls (PCBs) in the diet. *Poultry Science* 53:726-732.
- MDFW. 1979. Field Investigation Report: Great Blue Heron Rookery Inventory. Commonwealth
 of Massachusetts Division of Fisheries and Wildlife. October 1.
- MDFW. 1980. Field Investigation Report: Great Blue Heron Rookery Inventory, 1980.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife. June 30.
- MDFW. 1981. Field Investigation Report: Great Blue Heron Rookery Inventory, 1981.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife. June 30.
- MDFW. 1982. Field Investigation Report: Great Blue Heron Rookery Inventory, 1982.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife. July 16.
- MDFW. 1983. Field Investigation Report: Great Blue Heron Rookery Inventory, 1983.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife. July 3.
- MDFW. 1984. Field Investigation Report: Great Blue Heron Rookery Inventory Results.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife. October 16.
- MDFW. 1985. Field Investigation Report: Great Blue Heron Rookery Inventory Results.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife. December 16.
- MDFW. 1986a. Field Investigation Report: Great Blue Heron Rookery Inventory Results.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife. February 19.
- MDFW. 1986b. Field Investigation Report: Great Blue Heron Rookery Inventory, 1986.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife. November 25.
- MDFW. 1987. Field Investigation Report: Great Blue Heron Rookery Inventory, 1987.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife. October 9.
- MDFW. 1989. Field Investigation Report: Great Blue Heron Rookery Inventory, 1989.
 Commonwealth of Massachusetts Division of Fisheries and Wildlife.
- MDFW. 1991. Memorandum: 1991 Great Blue Heronry Survey. Commonwealth of
 Massachusetts Division of Fisheries & Wildlife. August 19.
- MDFW. 1996. Memorandum: 1996 Great Blue Heronry Survey. Commonwealth of
 Massachusetts Division of Fisheries & Wildlife. November 1.
- Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S. Petron, B. Potocki, S. Svirsky, and P. Tyler. 1996. Special report of the Massachusetts Weight-of-

- Evidence Workgroup: A weight-of-evidence approach for evaluating ecological risks. *Human Ecological Risk Assessment* 2:277-304.
- Moore, D.R.J., B.E. Sample, G.W. Suter, B.R. Parkhurst and R.S. Teed. 1999. A probabilistic risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East Fork Poplar Creek, Oak Ridge, Tennessee, USA. *Environ. Toxicol. Chem.* 18:2941-2953.
- Nagy, K.A. 1987. Free metabolic rate and food requirement scaling in mammals and birds.
 Ecological Monographs 57:111-128.
- Nagy, K.A., I.A. Girard, and T.K. Brown. 1999. Energetics of free-ranging mammals, reptiles,
 and birds. *Annual Review of Nutrition* 19:247-277.
- 10 Newsted J.L., J.P. Giesy, G.T. Ankley, D.E. Tillitt, R.A. Crawford, J.W. Gooch, P.D. Jones, and
- 11 M.S. Denison. 1995. Development of toxic equivalency factors for PCB congeners and the
- 12 assessment of TCDD and PCB mixtures in rainbow trout. Environmental Toxicology and
- 13 *Chemistry* 14:861-871.
- 14 Newton, I. 1979. *Population Ecology of Raptors*. Buteo Books, Vermillion, SD. 399 pp.
- 15 Nosek, J.A., S.R. Craven, J.R. Sullivan, S.S. Hurley, and R.E. Peterson. 1992. Toxicity and
- 16 reproductive effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin in ring-necked pheasant. Journal of
- 17 Toxicology and Environmental Health 35:187-198.
- 18 Poole, A.F. 1983. Poole, A. F. (1983) Courtship feeding, clutch size, and egg size in ospreys: A
- 19 preliminary report. In: Bird, D. M.; Seymour, N. R.; Gerrard, J. M., eds. Biology and
- 20 management of bald eagles and ospreys. St. Anne de Bellvue, Quebec: Harpell Press; pp.
- 21 243-256.
- 22 Poole, A.F. 1985. Courtship, feeding, and osprey reproduction. *The Auk* 102:479-492.
- Prestt I., D.J. Jefferies, and N.W. Moore. 1970. Polychlorinated biphenyls in wild birds in Britain
 and their avian toxicity. *Environmental Pollution* 1:3-26.
- 25 Rattner, B.A., N.H. Golden, J.L. Pearson, J.B. Cohen, L.J. Garrett, M.A. Ottinger, and R.M.
- 26 Erwin. 2001. Biological and Ecotoxicological Characteristics of Terrestrial Vertebrate Species
- 27 *Residing in Estuaries*. <u>http://www.pwrc.usgs.gov/bioeco/default.htm</u>.
- Safe, S. 1994. Polychlorinated biphenyls (PCBs): Environmental impact, biochemical and toxic
 responses, and implications for risk assessment. *Critical Reviews in Toxicology* 24:87-149.
- 30 Salyer, J.C. and K.F. Lagler. 1946. The eastern belted kingfisher, *Megaceryle alcyon alcyon* 31 (Linneaus), in relation to fish management. *Transactions of the American Fisheries Society*
- 32 76:97-117.
- 33 Scott, M.L. 1977. Effects of PCBs, DDT, and mercury compounds in chickens and Japanese
- 34 quail. Federation Proceedings 36:1888-1893.

- Senthilkumar, K., N. Iseki, S. Hayama, J. Nakanishi, and S. Masunaga. 2001. Polychlorinated
 dibenzo-p-dioxins, dibenzofurans, and dioxin-like polychlorinated biphenyls in livers of birds
 from Japan. *Arch. Environ. Contam. Toxicol.* 42:244-255.
- Vana-Miller, S. 1987. *Habitat Suitability Index Models: Osprey*. U.S. Fish and Wildlife Service
 Biological Report 82 (10.154). 46 pp.
- Van den Berg, M., L. Birnbaum, A.T.C. Bosveld., B. Brunström, P. Cook, M. Feeley, J.P. Giesy,
 A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X. Rolaf van Leeuwen,
 A.K.D. Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind,
 M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs) for PCBs,
- 10 PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives* 106(12):775–792.
- 11 Woodlot Alternatives, Inc. 2002. Draft Report, Oversight of GE Field Studies for Housatonic
- 12 River Primary Study Area. Prepared for Weston Solutions, Contract No. DACW33-00-D-
- 13 0006/004, DCN: GE-082902-ABEG.

9. ASSESSMENT ENDPOINT – SURVIVAL, GROWTH, AND REPRODUCTION OF PISCIVOROUS MAMMALS

3	Highlights
4	Conceptual Model
5 6 7 8	The assessment endpoint is the survival, growth, and reproduction of piscivorous mammals in the Housatonic River PSA. Piscivorous mammals (mink and river otter), selected as representative species for the ERA, are exposed to tPCBs and TEQ via diet and trophic transfer.
9	Exposure
10 11 12	Exposure of the representative species to COCs (tPCBs and TEQ) was determined from concentrations found in prey items and an estimation of the daily intake of COCs from consumption of prey.
13	Effects
14 15 16 17 18 19	Data on toxicity of tPCBs to mink were used to derive a dose-response relationship. The existing data on toxicity of TEQ to mink were insufficient to derive a dose-response relationship; therefore, upper and lower toxicity thresholds were derived. No tPCB or TEQ toxicity data were available for otter. River otter were assumed to have a similar sensitivity to tPCBs and TEQ. A site-specific feeding study was conducted to evaluate adverse effects to mink from Housatonic River COCs.
20	Risk
21 22 23	Mink and river otter are at high risk as a result of exposure to tPCBs and TEQ in the Housatonic PSA. The risk remains high even for those individuals who forage only a fraction of their time in the PSA.

24 9.1 INTRODUCTION

25 The purpose of this section is to characterize and quantify the current and potential risks posed to 26 piscivorous mammals exposed to contaminants of potential concern (COPCs) in the Housatonic 27 River and floodplain, focusing on total PCBs (tPCBs) and other COPCs originating from the 28 General Electric Company (GE) facility in Pittsfield, MA. The river is located in western 29 Massachusetts and Connecticut, discharging to Long Island Sound, with the GE facility located 30 near the headwaters. The Primary Study Area (PSA) includes the river and 10-year floodplain 31 from the confluence of the East and West Branches of the Housatonic River downstream of the 32 GE facility, to Woods Pond Dam (Figure 1.1-2).

A Pre-ERA was conducted to narrow the scope of the ecological risk assessment (ERA) by identifying contaminants, other than tPCBs, that pose potential risks to aquatic biota and wildlife in the PSA (Appendix B). A three-tiered deterministic approach was used to screen COPCs. The deterministic assessments compared conservative estimates of potential exposure with conservative adverse effects benchmarks to identify contaminants that are of potential concern to piscivorous mammals in the Housatonic River. A hazard quotient (total daily intake/effect benchmark) greater than one resulted in the COPC being screened through to the next Tier assessment and to the probabilistic ecological risk assessment, if necessary.

7 Subsequent to the Pre-ERA, several other COPCs (primarily organochlorine pesticides) were 8 screened out because their actual concentrations in the PSA were likely much lower than the 9 measured values due to laboratory interference problems (see Section 2.4). These COPCs were 10 evaluated further for each assessment endpoint, and the contaminants of concern (COCs) that 11 were retained for the probabilistic risk assessment for piscivorous mammals were tPCBs and 12 2,3,7,8-TCDD toxic equivalence (TEQ). Total PCBs detected in Housatonic River samples 13 closely resemble the commercial PCB mixtures Aroclor 1260 and Aroclor 1254, which are 14 similar in congener makeup. TEQ is calculated from coplanar PCB and dioxin and furan 15 congeners using the toxic equivalency factor (TEF) approach developed by Van den Berg et al. 16 (1998)(see Section 6.4).

A step-wise approach was used to assess the risks of tPCBs and TEQ to piscivorous mammals in
the Housatonic River watershed. The four main steps in this process include:

- 19
 - Derivation of a conceptual model (Figure 9.1-1).
 Assessment of exposure of piscivorous mammals to COCs (Figure 9.1-2).
 - 3. Assessment of the effects of COCs on piscivorous mammals (Figure 9.1-3).
 - 4. Characterization of risks to the piscivorous mammalian community (Figure 9.1-4).
- 22 23

20

21

24 This section is organized as follows:

- Section 9.2 (Conceptual Model)—Describes the conceptual model for piscivorous mammals, including selection of representative species and establishment of measurement and assessment endpoints.
- Section 9.3 (Exposure Assessment)—Describes the exposure model, input variables, and techniques to propagate uncertainty. Also presented in this section are the exposure modeling results for mink and river otter.

- Section 9.4 (Effects Assessment)—Describes the effects to mammals exposed to tPCBs and TEQ and derives the effects metrics.
- Section 9.5 (Risk Characterization)—Integrates the exposure and effects assessments to quantify risk to piscivorous mammals in the PSA for each line of evidence. This section contains brief descriptions of field surveys, feeding study, and modeled exposure and effects measurement endpoints. The feeding and field studies were used in the risk characterization only and were not used to develop effect levels for comparison to modeled results. The risk information from three lines of evidence is combined in the weight-of-evidence analysis. This section also describes the sources of uncertainty in the ERA for piscivorous mammals, followed by the conclusions regarding risks of tPCBs and TEQ to piscivorous mammals in the Housatonic River PSA.
 - The detailed ecological risk assessment for piscivorous mammals is provided in Appendix I.



Figure 9.1-1 Conceptual Model Diagram: Exposure Pathways for Piscivorous Mammals Exposed to COCs in the Housatonic River PSA

4

1

2

EXPOSURE



Figure 9.1-2 Overview of Approach Used to Assess Modeled Exposure of
 Piscivorous Mammals to Contaminants of Concern (COCs) in the Housatonic
 River PSA

EFFECTS



Figure 9.1-3 Overview of Approach Used to Assess the Modeled Effects of Contaminants of Concern (COCs) to Piscivorous Mammals in the Housatonic River PSA

RISK CHARACTERIZATION



Figure 9.1-4 Overview of Approach Used to Characterize the Risks of Contaminants of Concern (COCs) to Piscivorous Mammals in the Housatonic River PSA

1 2 3

1 9.2 CONCEPTUAL MODEL

2 The conceptual model presented in Figure 9.1-1 illustrates the exposure pathways for piscivorous 3 mammals exposed to tPCBs and TEQ in the PSA. Total PCBs and TEQ are persistent and 4 hydrophobic and lipophilic. Therefore, they are bioaccumulated by aquatic and terrestrial biota 5 through the consumption of contaminated prey as part of the food chain (Haffner et al. 1994; 6 Senthilkumar et al. 2001). Fish, small mammals, crayfish, waterfowl, and amphibians are the 7 major dietary items for piscivorous mammals. Piscivorous mammals that reside, or partially 8 reside, within the study area are exposed to tPCBs and TEQ principally through diet and trophic 9 transfer. Other routes of exposure, considered to be less important to overall exposure, include 10 inhalation, water consumption, and sediment ingestion (Moore et al. 1999).

The problem formulation (see Section 2) identified mink (*Mustela vison*) and river otter (*Lutra canadensis*) (Figures 9.2-1 and 9.2-2) as the representative species for piscivorous mammals exposed to tPCBs and TEQ from consumption of contaminated prey. Life history profiles for mink and river otter are summarized in the following text boxes. Additional life history information on these species is presented in Sections I.2.1.5 and I.2.1.6, respectively.

16 The assessment endpoint that is the subject of this section is the survival, growth, and reproduction of piscivorous mammals in the Housatonic River PSA. The measurement 17 18 endpoints used to evaluate the assessment endpoint included: (1) determining the extent to 19 which the concentrations of tPCBs and TEQ ingested in the diet impact the survival, 20 reproduction, or growth of piscivorous mammals by comparisons to doses reported in the 21 literature to cause adverse effects; (2) determining, by conducting quantitative field surveys, the 22 abundance of piscivorous mammals in the Housatonic River relative to appropriate 23 uncontaminated reference areas within the watershed; and (3) determining, by conducting a 24 feeding study using fish collected from the PSA, whether a diet of site-specific fish has an 25 adverse effect on the survival and reproduction of farm-raised mink.



Figure 9.2-1 Mink (Mustela vison)

Life History of Mink

Mink are small, fur-bearing animals with characteristic elongated bodies, short legs, and long tails. Mink are one of the most widespread mammalian carnivores, with a range spanning much of the continental USA and Canada.

Habitat – Require access to open water such as streams, tidal flats, marshes, shallow rivers, lakes, and swamps. Also suitable cover in the form of overhanging vegetation, rock crevices, exposed roots, log jams, and undercut banks.

Home Range - Adult males occupy home ranges exclusive of other adult males, and may include the home ranges of one or more females. Males range from 309 to 776 ha, and females range from 7.8 to 20.4 ha. Riverine home ranges are linear (between 1.0 and 6.0 km of shoreline); those in marsh habitats tend to be more circular.

Dietary Habits - Primary food items include fish, small mammals, benthic invertebrates, birds, and amphibians. Opportunistic; diet varies depending upon the availability of prey items. Mean percentage of prey items in diet: fish, 23%; mammals, 15%; birds, 11.0%; invertebrates, 36%; and amphibians and reptiles, 15.0%.



Figure 9.2-2 River Otter (Lutra canadensis)

Life History of River Otter

River otter are long-bodied, short-legged, semi-aquatic mustelids that occur throughout most of Canada and the continental United States. Male otter in the eastern United States are quite large and range in weight from 8 to 11 kg. Females range from 7.5 to 8 kg.

Habitat - Remain close to aquatic habitats such as lakes, marshes, streams, seashores, rivers, creeks, and bayous. In New England, preferentially select riverine and lacustrine systems. Have numerous denning and nesting sites within home range, used over the course of the year. Denning and resting sites may be located in log jams, riparian vegetation, snow or ice cavities, riprap, talus rock, boulders, brush and log piles, undercut banks, and dens constructed by other animals.

Home Range - Average size of the home range for adult otter is about 30 km of shoreline. Lactating females have the smallest home ranges. Other than family groups, are typically solitary. Will form temporary associations that may consist of related or unrelated individuals. Home ranges shown to overlap extensively, with some otter sharing essentially the same home range.

Dietary Habits - Diet somewhat variable; primarily consists of aquatic animals, particularly fish; other prey includes crayfish, amphibians, turtles, birds, small mammals, and insects. Prefer to forage in shallow water and eat primarily slow-moving, shallow-dwelling fish, such as chubs, suckers, catfish, daces, darters, and schooling fish such as bluegill and other sunfish.

1 9.3 EXPOSURE ASSESSMENT

2	This exposure assessment evaluates exposure of piscivorous mammals to tPCBs and 2,3,7,8-
3	TCDD toxic equivalence (TEQ) in Reaches 5 (confluence to Woods Pond) and 6 (Woods Pond),
4	together referred to as the Primary Study Area (PSA) of the Housatonic River. Exposure
5	assessments were also conducted for two reference areas for comparative purposes. One of the
6	reference areas is located upstream of the GE facility on the East Branch of the Housatonic River
7	in Dalton, MA (herein referred to as the "upstream reference area"). The other reference area is
8	Threemile Pond located in Sheffield, MA, which is in the Housatonic River drainage, but at a
9	higher elevation, draining to the river. The representative species for piscivorous mammals are
10	mink and river otter. These mammals occur in the Housatonic River watershed and feed on prey
11	exposed directly to tPCBs and TEQ and through trophic transfer. The ingestion of contaminated
12	prey is the major exposure pathway for piscivorous mammals exposed to tPCBs and TEQ.
13	Total PCBs and TEQ tend to bioaccumulate in the food chain because:
14	 Total PCBs and TEQ are persistent, hydrophobic, and lipophilic substances.
15 16 17 18	• When released to aquatic systems, the majority of these compounds form associations with dissolved and/or particulate matter in the water column and settle to the sediment bed; biodegradation is considered to be a relatively minor fate process in water (NRCC 1981; Howard et al. 1991).
19 20 21 22 23	 Aquatic sediment provides a sink for these compounds and may represent long-term sources to the aquatic food web (Kuehl et al. 1987; Muir 1988; Corbet et al. 1983; Tsushimoto et al. 1982). Both of these COCs are bioaccumulated by aquatic and terrestrial biota directly through the consumption of contaminated prey as part of the food chain (Haffner et al. 1994; Senthilkumar et al. 2001; Borga et al. 2001).
24	In summary, piscivorous mammals that reside, or partially reside, within the PSA are exposed to
25	tPCBs and TEQ principally through diet.
26	The exposure analysis for mink was carried out separately for Reach 5 and Reach 6 of the DSA
20 27	because the foreging range of mink enprovimetes the lengths of these river sections. However
	- DULAUNG HIG TOLAYINY TANYE OF HIMIK ADDITIXTILIATES THE TENYINS OF HIMSE TIVEL SECTIONS - HOWEVEL

28 the foraging range of river otter is larger; therefore, the exposure analysis for river otter was

29 conducted with Reaches 5 and 6 combined.

1 This section begins with a description of the exposure model used for the representative species.

2 Subsequent sections describe the inputs used in the exposure analyses for each representative

3 species. The section concludes with a presentation of the results of the exposure analyses.

4 9.3.1 Exposure Model

Exposure of the representative species, mink and river otter, to tPCBs and TEQ was estimated
using a total daily intake model adapted from the *Wildlife Exposure Factors Handbook* (EPA
1993) and related publications. The model used in the exposure analysis was:

8
$$TDI = FT \cdot FIR \sum_{i=1}^{n} C_i \cdot P_i$$
 (Eq. 1)

9 where

10 TDI Total daily intake (mg/kg bw/d tPCBs, ng/kg bw/d TEQ). = Normalized food intake rate (kg/kg bw/d). 11 FIR = 12 FT= Foraging time in PSA (unitless). Concentration in *i*th food item (mg/kg tPCBs, ng/kg TEQ). 13 C_i =14 P_i Proportion of the *i*th food item in the diet (unitless). =

15

The models consider the food intake rates of the representative species (FIR), the concentrations 16 17 of COCs in each food item (C_i) , and the proportion of the diet accounted for by that food item 18 (P_i) . For those input variables that are uncertain, variable, or both, distributions are used rather 19 than point estimates. Monte Carlo and probability bounds analyses are the methods used to 20 propagate uncertainties about input variables in the exposure model for each COC. Α 21 description of these techniques and methods used to parameterize input variables is presented in 22 Section 6.5 and Appendix C. The results of the Monte Carlo analysis are used to estimate the 23 probability of exposure exceeding an effects threshold or doses that cause adverse effects of 24 differing magnitudes. The probability bounds analysis is conducted to determine how 25 uncertainty regarding the distributions of the input variables influences the estimated exposure 26 distribution. The results of these analyses are discussed in detail in Appendix I.

27 Two issues arose when calculating a TEQ concentration in prey:

1	 Congener concentrations may be below the method detection limit (DL) (i.e., non- detects)
$\frac{2}{3}$	 Some congeners may not be resolved due to co-elution during analysis.
4 5	An approach was developed to address these issues. This approach is presented in Section 6.4
6	and Appendix C.2. Briefly congeners detected at or below the DL were included in the TEO
7	calculations by investigating three options:
8 9 10 11	 Setting the concentration for the congener equal to zero (0). Setting it to half the DL. Setting it equal to the DL.
12	A comparison of the results of this bounding analysis provides a description of the uncertainty
13	surrounding the TEQ value due to concentrations of one or more congeners being below the
14	detection limit.
15 16	To resolve the co-elution issue, the concentrations of congeners that co-eluted with other congeners were assumed equal to the total concentration (likely an overestimate of TEO)
10	congeners were assumed equal to the total concentration (intery an overestimate of TEQ
1/	Concentration) of zero (interval to dool with the upcontainty origing from as obtion or non detection
18	Section 6.4 were followed to deal with the uncertainty arising from co-elution or hon-detection
19 20	of congeners when estimating exposure point concentrations (EPCs) for use in the exposure
20	anaryses.
21	Input distributions to the exposure analyses were generally assigned as follows:
22 23	 Lognormal distributions were assigned to variables that were right skewed with a lower bound of zero and no upper bound (e.g., amount of COC in fish).
24 25	 Beta distributions for variables bounded by zero and one (e.g., proportion of a prey item in the diet).
26 27	 Normal distributions for variables that were symmetric and not bounded by one (e.g., body weight).
28	 Point estimates for minor variables or variables with low coefficients of variation.
29	In certain situations (e.g., poor fit of data), other distributions were fit to the data or other
30	approaches were used. To quantify uncertainty, two approaches were used as described in
31	Section 6.5.2 and Appendix C. The distributions used in the exposure analyses for mink and

river otter are shown in Figures 9.3-1 and 9.3-2. A brief description of these variables is
 provided below.

3 9.3.1.1 Body Weight (BW)

4 Average body weights (wet weight of wild animals) of female mink range from 550 g (Mitchell 5 1961) to 970 g (Hornshaw et al. 1983) and males range from 630 to 1,000 g (Whitaker and 6 Hamilton 1998). For the Monte Carlo analysis, the mean weight of females was estimated to be 7 685 g with a standard deviation of 122. Body weights were assumed to be distributed normally. 8 There is low uncertainty associated with this variable. The uncertainty in this variable is due to 9 variability, rather than lack of knowledge or data (i.e., the variable is easily measured and many 10 studies have been conducted that measured this variable). Accordingly, the same distribution 11 was used in the probability bounds analysis.

12 Body weight is not used in the model directly, but is a required variable in allometric models 13 (e.g., Nagy 1987) to estimate food intake or free metabolic rates. Whitaker and Hamilton (1998) 14 reported that body weights of river otter ranged from 8 to 11 kg (average of 9.2 kg) for males and from 7.5 to 8.0 kg (average of 7.9 kg) for females in eastern United States populations. In the 15 16 Monte Carlo analysis, body weight was assumed to be normally distributed with a mean of 8,630 17 g and a standard deviation of 1,600 g. The same distribution was used in the probability bounds 18 analysis for this input variable. The uncertainty in this variable is small and is likely due to 19 variability, rather than lack of knowledge or data gaps.





Figure 9.3-1 Input Distributions Used in Exposure Modeling for Mink



Figure 9.3-2 Input Distributions Used in Exposure Modeling for River Otter

2

9.3.1.2 Food Intake Rate (FIR)

5 The daily energy requirements of mink vary depending on environmental conditions and the 6 stage of the reproductive cycle. However, the long-term average daily consumption of dry 7 matter is approximately 0.040 kg/kg of body mass for males and 0.0530 kg/kg of body mass for 8 female captive mink (Bleavins and Aulerich 1981; Lariviere 1999). A 1.0-kg mink living in a 9 laboratory requires approximately 150 kilocalories (kcal) of digestible energy every day for 10 maintenance. A nursing female can require 3 times that amount at 3 weeks post-partum 11 (Lariviere 1999). However, a nursing female food intake rate was not considered in this 12 assessment because nursing is a short-term event relative to the extended time scale of this 13 assessment (1 year). The time scale of this exposure assessment was chosen to be approximately 14 1 year based on the extended reproductive cycle of mink (mating starts in early March) and the 15 duration of the mink feeding study, which evaluated the effects on young mink until they were 6 16 months old.

For the purpose of the ERA for piscivorous mammals, *FIR* was estimated using an allometric equation rather than using literature-reported values for captive mink. An allometric modelderived *FIR* better approximates the increased energy demand of wild mink resulting from higher activity levels incurred while foraging, defending and inspecting territory, and avoiding predators (Lamprey 1964; Buechner and Golley 1967; Koplin et al. 1980).

6 Food intake rate (FIR) is derived using the following equation:

 $FMR \ (kJ/d) = a \cdot BW(g)^b$ (Eq. 2)

8 where *FMR* is the free-living metabolic rate and *BW* is the body weight. The slope (*a*) and 9 power (*b*) distributions were based on the error statistics from regression analysis of the data 10 reported in Nagy et al. (1999). For carnivorous mammals, the mean slope term log (*a*) had a 11 mean of 0.367 and a standard error of 0.223. The power term (*b*) had a reported mean of 0.850 12 and a standard error of 0.055(Nagy et al. 1999).

13 Food intake rate is derived from *FMR* using the following equation:

14
$$FIR = \frac{FMR}{\sum_{i=1}^{n} AE_i \cdot GE_i}$$
 (Eq. 3)

where AE_i is the assimilation efficiency of *i*th food item (unitless) and *GE* is the gross energy of *i*th food item (kcal/kg).

The gross energies of various wildlife food sources are summarized in the *Wildlife Exposure Factors Handbook* (EPA 1993) and were as follows: fish and amphibians (assumed for amphibians) 1.20 kcal/g (Thayer et al. 1973; EPA 1993), invertebrates 1.10 kcal/g (Jorgensen et al. 1991; Minnich 1982; Thayer et al. 1973), and birds and mammals 1.8 kcal/g (EPA 1993). These variables were treated as point estimates in Monte Carlo simulations because of their relatively small coefficients of variation. Gross energy is easily measured and thus measurement error is likely to be low.

Average assimilation efficiency for mammals consuming fish and amphibians is 0.91, for invertebrates it is 0.87, and for birds and mammals it is 0.84 (EPA 1993; Grodzinski and Wunder 1 1975; Barrett and Stueck 1976). No data were available for assimilation efficiency of mammals 2 consuming amphibians, but it is likely to be similar to that for mammals consuming fish. These 3 variables were treated as point estimates in Monte Carlo simulations and probability bounds 4 analyses because of their relatively small coefficients of variation. As a result, these input 5 variables are not included in Figures 9.3-1 and 9.3-2.

6 9.3.1.3 Proportions of Dietary Items (P_i)

7 The primary food items in the mink diet include small mammals, fish, benthic invertebrates 8 (crayfish), birds (waterfowl), and amphibians (Alexander 1977; Burgess and Bider 1980; Cowan 9 and Reilly 1973; Gilbert and Nanckivell 1982; Hamilton 1959, 1940; Melquist et al. 1981; 10 Proulx et al. 1987) (Table I.2-2). Combining the available data, an average of 23% (range of 0 to 11 64.7%) of the mink diet consists of fish. Mammals on average comprise 15% of the diet (range 12 of 0 to 25%). Reptiles and amphibians also constitute an average of 15% (range of 0 to 30%) of 13 the diet, and birds (i.e., waterfowl) 11% (range of 0 to 39%) of the diet. Invertebrates constitute 14 an average of 36% of the diet (range of 0 to 54%).

15 Melquist et al. (1981) found that fish taken by mink were mostly cyprinids between 7 and 12 cm 16 long. Similarly, Hamilton (1940) recorded that the average length of fish taken by mink ranged from 7.6 to 10.2 cm. According to Alexander (1977), mink in rivers and streams in lower 17 18 Michigan and New York consume fish ranging from 15 to 18 cm. Based on this information, 19 fish used in the exposure analyses were limited to a minimum length of 7 cm and a maximum 20 length of 20 cm. Fish prey of river otter can range from 2 to 50 cm in length (Melquist and 21 Hornocker 1983). In some areas, fish captured were typically less than 15 cm (Hamilton 1961; 22 Lagler and Ostenson 1942; Alexander 1977). Greer (1956), however, indicated that fish 23 captured by otter ranged from 15 to 25 cm. Based on these observations, the exposure analysis 24 for otter included tissue samples for fish ranging in length from 2 to 50 cm.

The proportion of each prey type in the diet was assumed to follow a beta distribution in the Monte Carlo analysis and was parameterized to approximate the above averages and ranges (Table I.2-3). The beta distribution is not an available option in RiskCalc, the software used for conducting the probability bounds analyses. As an alternative, minimum, mean, and maximum values were specified for each dietary item using the means and ranges described above. The minimum, mean, and maximum values were then included as a distribution-free statement in
 RiskCalc. The results bound all possible distributions, given the minimum, mean, and maximum
 values specified for the dietary items.

4 9.3.1.4 Concentrations of COCs in Prey

5 The median concentrations of tPCBs in mink prey from the PSA range from 2.45 mg/kg in amphibians from Reach 5 to 29.9 mg/kg in fish from the same location. The 25th and 75th 6 percentiles are 1.13 and 5.37 mg/kg for amphibians and 24.6 and 39.2 mg/kg for fish from Reach 7 8 5. Median TEQ levels in mink prey range from 91.6 ng/kg in amphibians from Reach 5 to 858 ng/kg in birds from the same location. The 25th and 75th percentiles are 58.8 and 123 ng/kg for 9 10 amphibians and 532 and 1,596 ng/kg for birds from Reach 5. The distributions for 11 concentrations of tPCBs and TEQ in prey of mink are presented in Figures 9.3-3 and 9.3-4, 12 respectively. The distributions for concentrations of tPCBs and TEQ in prey of river otter are 13 presented in Figures 9.3-5 and 9.3-6, respectively. The input variables for concentrations of 14 COCs in prey of mink and river otter are shown in Tables I.2-4, I.2-5, I.2-12, and I.2-13.









Figure 9.3-4 Concentrations of TEQ in Prey of Mink







5 9.3.2 Results of Exposure Assessments

1

Exposure distributions for exposure of mink and river otter to tPCBs and TEQ in Reaches 5 and
6, and reference areas are presented in Figures 9.3-7 through 9.3-20.

8 Figure 9.3-7 depicts the cumulative distribution of tPCB intake rates for mink in Reach 5. The 9 Monte Carlo analysis indicated that exposure of mink to tPCBs could range from a minimum of 10 0.308 to a maximum of 82.5 mg/kg bw/d. The mean exposure was 5.29 mg/kg bw/d and the 11 median exposure was 3.97 mg/kg bw/d. Of the exposure estimates, 90% were between 1.15 and 13.6 mg/kg bw/d. The probability bounds estimated for mink foraging in Reach 5 are depicted in 12 Figure 9.3-7. The 10th percentile of the probability envelope formed by the lower and upper 13 bounds ranged between 0.0244 and 4.11 mg/kg bw/d. The 50th percentile ranged between 0.292 14 and 8.47 mg/kg bw/d, and the 90th percentile ranged between 1.72 and 22.5 mg/kg bw/d. In 15 16 comparison, the 10th percentile of the Monte Carlo output was 1.52, the 50th percentile was 17 3.97, and the 90th percentile was 10.4 mg/kg bw/d.








4 Figure 9.3-8 Exposure of Mink to tPCBs in Reach 6 of the Housatonic River



Figure 9.3-9 Exposure of Mink to tPCBs in the Housatonic River Upstream Reference Area

6

4



Threemile Pond Reference Area



4 Figure 9.3-10 Exposure of Mink to tPCBs in the Threemile Pond Reference Area

5





Figure 9.3-11 Exposure of Mink to 2,3,7,8-TCDD TEQ in Reach 5 of the Housatonic River

6

1

2 3



LPB = Lower probability bound	l
UPB = Upper probability bound	

Figure 9.3-12 Exposure of Mink to 2,3,7,8-TCDD TEQ in Reach 6 of the Housatonic River

6

1

2 3







2 3

LPB = Lower probability bound

UPB = Upper probability bound

Figure 9.3-14 Exposure of Mink to 2,3,7,8-TCDD TEQ in the Threemile Pond Reference Area

6 Figure 9.3-15 depicts the cumulative distribution of tPCB intake rates for river otter in Reaches 5 7 and 6. The Monte Carlo analysis indicated that exposure of otter to tPCBs foraging in the PSA 8 100% of the time could range from a minimum of 0.251 to a maximum of 111 mg/kg bw/d. The 9 mean exposure was 8.42 mg/kg bw/d and the median exposure was 6.02 mg/kg bw/d (Table I.2-10 14). Of the exposure estimates, 90% were between 1.60 and 22.8 mg/kg bw/d. The probability bounds estimated for river otter foraging in Reaches 5 and 6 are depicted in Figure 9.3-15. The 11 12 10th percentile of the probability envelope formed by the lower and upper bounds ranged 13 between 1.59 and 8.12 mg/kg bw/d. The 50th percentile ranged between 3.27 and 14.2 mg/kg 14 bw/d, and the 90th percentile ranged between 5.78 and 53.0 mg/kg bw/d. In comparison, the 15 10th percentile of the Monte Carlo output was 2.15, the 50th percentile was 6.03, and the 90th 16 percentile was 17.1 mg/kg bw/d.







Figure 9.3-15 Exposure of River Otter to tPCBs in Reaches 5 and 6 of the Housatonic River





LPB = Lower probability boundLIPB = Lippor probability bound

UPB = Upper probability bound

5 Figure 9.3-16 Exposure of River Otter to tPCBs in the Housatonic River 6 Upstream Reference Area

7

1 2





5 Figure 9.3-17 Exposure of River Otter to tPCBs in the Threemile Pond Reference 6 Area

7

1 2



_	

4 5

LPB = Lower probability bound
UPB = Upper probability bound

Figure 9.3-18 Exposure of River Otter to 2,3,7,8-TCDD TEQ in Reaches 5 and 6 of the Housatonic River





Figure 9.3-19 Exposure of River Otter to 2,3,7,8-TCDD TEQ in the Upstream Reference Area

6

1

2 3

4



Threemile Pond Reference Area

LPB = Lower probability bound UPB = Upper probability bound

Figure 9.3-20 Exposure of River Otter to 2,3,7,8-TCDD TEQ in the Threemile Pond Reference Area

6

1

1 9.4 EFFECTS ASSESSMENT

2 The purpose of the effects assessment is to review the scientific literature and derive appropriate 3 effects metrics for effects of tPCBs and TEQ to piscivorous mammals. An effects metric can be 4 represented by a dose-response relationship or a daily dose for a COC that represents a threshold 5 beyond which toxic effects may appear in piscivorous mammals. The effects metrics are used, in 6 conjunction with the exposure assessment, to estimate risks to piscivorous mammals exposed to 7 tPCBs and TEQ in the Housatonic River PSA. This section focuses on effects that have an 8 influence on the maintenance of local populations (i.e., mortality, or impairment of reproduction 9 or growth). Studies involving multiple exposure treatments and where reported results were 10 evaluated statistically to identify significant differences from controls were preferred. This 11 section also presents the results of a study where farm-raised mink were exposed to a diet 12 containing fish collected from the PSA.

Studies that document effects of tPCBs and TEQ were available only for mink, not otter. However, given the close similarities between mink and otter in their feeding preferences and phylogeny, an assumption was made that toxicity data for mink can be used to approximate toxicity to river otter.

9.4.1 Review of Toxicity from the Literature

Presented below is a brief review of the scientific literature on the effects of dietary tPCBs and TEQ to piscivorous mammals. The discussion focuses on ecologically relevant effects endpoints such as survival, growth, and reproduction. A summary of reproduction effects for tPCBs and TEQ is presented in Figures I.3-1 and I.3-2 and Table I.3-1.

22 9.4.1.1 Total PCBs

23 **9.4.1.1.1** Mortality

In a study where the diet was prepared from cattle that had consumed feed contaminated with Aroclor 1254 (Figure I.3-1; Table I.3-1; Platonow and Karstad 1973), a dose of 0.0896 mg/kg bw/d consumed by female mink over 160 days of exposure caused 100% mortality in the 1 offspring. The treatment also caused 17% mortality in adult females, but not in males. 2 Hornshaw et al. (1983) fed female mink with contaminated carp containing a dose of 0.210 3 mg/kg bw/d of PCBs identified as Aroclor 1254. After 7 months of this feeding regime, the 4 mink were allowed to reproduce. None of the young were born alive. A slightly higher dose of 5 0.280 mg/kg bw/d caused 100% kit mortality 4 weeks after birth. Adult female mink 6 experienced 12% mortality after 10 months of continuous exposure to this treatment (Aulerich 7 and Ringer 1977).

8 Total mortality in adults was observed at a dose of 0.500 mg/kg bw/d (Platonow and Karstad 9 1973). Only 105 days of dietary exposure at this concentration were required to kill all the 10 adults. In another study, female mink were exposed to a dose of 0.700 mg/kg bw/d. Of these 11 individuals, 30% died after 9 months of exposure (Aulerich and Ringer 1977). Mortality 12 increased to 71% in response to a dose of 1.40 mg/kg bw/d.

Ranch-raised mink exposed to 0.140 mg/kg bw/d reported as Aroclor 1254 from field-collected carp experienced lower survival in lactating offspring (Wren et al. 1987b). However, the carp contained other contaminants that could have contributed to the toxic response. Dietary LC_{50} tests with mink performed by Hornshaw et al. (1986) using Aroclor 1254 revealed average LC_{50} s from 6.58 mg/kg bw/d to 8.12 mg/kg bw/d. One of the highest estimates of acute doses was reported by Aulerich et al. (1973), who found a 48-hour LD_{50} of 140 mg/kg bw/d.

19 Dietary exposure of female mink to a dose of 0.004 mg/kg bw/d tPCBs (42 to 60 % chlorine) in 20 carp for 3 to 6 weeks resulted in 15% mortality in kits (Heaton et al. 1995). Mortality increased 21 to 69% at a dose of 0.1 mg/kg bw/d after 3 weeks of exposure and 71% after 6 weeks of 22 exposure. At the dose of 0.210 mg/kg bw/d, kit mortality was 71% after 3 weeks of exposure 23 and 89% after 6 weeks of exposure. Total kit mortality was observed at a dose of 0.360 mg/kg 24 bw/d, with death being observed in as little as 24 hours after receiving the dose. Jensen et al. 25 (1977) exposed female mink to a dose of 1.54 mg/kg bw/d of Aroclor 1254 for 66 days. After 26 the treatment, no live kits were born to exposed females. Ringer et al. (1972) exposed mink to a 27 diet spiked with 4.20 mg/kg bw/d PCBs (equal amounts of Aroclors 1242, 1248, and 1254). All 28 adult mink died prior to whelping.

1 **9.4.1.1.2** Reproduction

2 Farm-raised mink exposed to 0.140 mg/kg bw/d Aroclor 1254 experienced reduced survival of 3 lactating offspring. However, no declines in fertility, whelping, or fecundity were observed 4 (Wren et al. 1987b). Kihlstrom et al. (1992) exposed female mink to 1.64 mg of Aroclor 1254 per individual (1.28 mg/kg bw/d) in food for 105 days. The exposure caused all kits to be 5 6 stillborn. The dose also increased the number of interrupted pregnancies. Aulerich and Ringer 7 (1977) reported that exposure of mink to 0.280 mg/kg bw/d of Aroclor 1254 did not affect birth 8 rate, birth weight, or survival. However, a dose of 2.80 mg/kg bw/d caused reduced whelping 9 and growth rate of kits. At 0.7 mg/kg bw/d, no whelping was observed, although survival was 10 unaffected (Bleavins et al. 1980).

11 Decreased mink fecundity has been observed following exposure to 0.08 mg/kg bw/d (0.7 mg/kg 12 diet) (Brunstrom et al. 1991). In another study, Aulerich et al. (1985) exposed mink to dietary concentrations of Aroclor 1254 over extended exposure periods (several weeks). 13 А 14 concentration of 2.5 mg/kg was associated with reduced fecundity. Only one female whelped 15 and the kit that was born died after birth. This dietary exposure is equivalent to a dose of 0.288 16 mg/kg bw/d given the food intake rate of 115 g/day. Male and female mink fed PCB-17 contaminated diets (Saginaw Bay carp) had decreased breeding performance. Kit body weight 18 and survival were reduced at birth following exposure to 0.140 mg/kg bw/d of tPCBs in the diet 19 (Restum et al. 1998).

20 9.4.1.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

21	Effects of TEQ
22	Types of effects to mammals from exposure to TEQ include:
23	Hormone induction
24	 Decreases in body and organ weight
25	 Reduced fertility
26	Reduced litter size
27	 Reduced survival at birth or weaning
28	 Mortality

1	Mode of Action of TEQ Congeners
2 3 4 5 6	Congeners that comprise the TEQ group have the ability to bind with the aryl hydrocarbon (Ah) receptor and elicit similar toxic responses. The most toxic congeners tend to be those that have a planar shape and are chlorinated in the 2,3,7, and 8 positions for dioxins and furans, and in the meta and para positions for PCBs.
7 8 9 10 11 12	This structural configuration best fits the receptor and leads to a common mechanism of action in many animal species involving binding to the Ah receptor and elicitation of an Ah-receptor-mediated biochemical and toxic response. The toxic response of this group of chemicals is, therefore, related to the three-dimensional structure of the substance, including the degree of chlorination and positions of the chlorine on the aromatic frame.
13 14 15 16 17 18	Planar chlorinated hydrocarbons are found in the environment as a mixture of congeners. The congeners can have different toxic potencies. To address this issue and effectively estimate the relative toxicity of these mixtures, various systems have been created involving the development and use of toxic equivalency factors (TEFs) to derive toxic equivalence (TEQ). The approach used for this assessment is described in Section 6.4.

20 **9.4.1.2.1** Mortality

21 Mature female mink fed diets with 0.600, 16.0, 53.0, 180, and 1,400 ng/kg of 2,3,7,8-TCDD 22 (equivalent to a dose of 0.0840, 2.24, 7.42, 25.2, and 196 ng/kg bw/d) for a maximum of 132 23 days exhibited 17% mortality, as well as lethargy and bloody stools at the highest dose 24 concentration (Hochstein et al. 2001). Final body weights were inversely related to dietary 25 TCDD concentration and there was a dose-dependent drop in kit weight from birth to week three 26 of exposure. At the highest dose concentration of 196 ng/kg bw/d various physiological 27 functions were depressed. Hochstein et al. (1998) exposed female mink to 1, 10, 100, 1,000, 28 10,000, and 100,000 ng/kg of TCDD in the diet (daily dose equivalent of 0.14, 1.4, 14, 140, 1,400, and 14,000 ng/kg bw/d) for 125 days. A dose-dependent wasting syndrome (decrease in 29 30 body weight) was observed. Mortality reached 12.5%, 62.5%, and 100% after 28 days of 31 exposure to 140, 1,400, and 14,000 ng/kg bw/d, respectively. After 125 days of exposure, 32 mortality reached 100% in the 1,400 and 14,000 ng/kg exposure groups. Newborn mink given 33 doses (intraperitoneal injection) of 100 and 1,000 ng TCDD/kg bw experienced 100% mortality 34 at the higher dose after 12 days. The lower dose caused depressed body weight and 62% 35 mortality (Aulerich et al. 1988). Adult mink administered a single oral dose of 2,500 ng/kg bw 36 TCDD had significantly reduced body weights after 3 weeks (Hochstein et al. 1988). At 0.250

ng/kg bw/d, Heaton et al. (1995) observed 15% mortality to mink kits after exposure for 3
weeks. At a dose of 3.6 ng/kg bw/d, 69% mortality in kits was reported. Mortality increased to
100% at a dose of 10.7 ng/kg bw/d.

4 9.4.1.2.2 Reproduction

5 Adult mink exposed to 0.6, 16, 53, 180, and 1,400 ng/kg TCDD in a diet of field-collected fish 6 (daily dose equivalent of 0.084, 2.24, 7.42, 25.2, and 196 ng/kg bw/d) for up to 132 days 7 produced offspring that had reduced survival from birth to week three of exposure (Hochstein et 8 al. 2001). There is some evidence that TCDD interferes with ovulation. Ushinohama et al. 9 (2001) administered a dose of 32,000 ng/kg by gavage to female rats. The treatment led to 10 reduced body weight gains as well as to reduced ovarian weights. Infertility and fetal loss have 11 been observed at a dose of 0.01 mg/kg/day (10 ng/kg bw/d TEQ) administered to rats (Murray et 12 al. 1979). A lowest observed adverse effect level (LOAEL) of 1 ng/kg/day was estimated by 13 Nisbet and Paxton (1982) using Murray et al. (1979) data. Ovulation was delayed and fewer ova produced. A dose of 0.350 mg/kg bw/d of 2,3,6,2',3',6'-HxCB caused reduced litter size 14 (Aulerich et al. 1985). Female mink exposed to 0.00140 mg/kg bw/d of the isomer 3,4,5,3',4',5-15 16 HxCB for 120 days did not experience adverse effects on reproduction (Aulerich et al. 1987). A 17 dose of 0.0140 mg/kg bw/d was associated with a total absence of whelping.

18 9.4.2 Mink Feeding Study

19 It was hypothesized, when developing the conceptual model for the ERA, that contaminants in 20 the prey of piscivorous mammals foraging in the PSA may have caused adverse effects on the 21 survival, reproduction, and/or growth of exposed individuals, based on a lack of observations of 22 mink or otter (or sign) during EPA field investigations. To test this hypothesis, a long-term 23 feeding study was performed by researchers at Michigan State University (MSU) (Bursian et al. 2002), the results of which are described below.

25 **9.4.2.1** *Methodology*

In this study, fish were collected from the PSA, frozen, and sent to MSU. These fish were mixed with ocean herring in varying proportions to derive a control diet formulated to meet the

nutritional requirements of farm-raised mink (all diets were 30% fish, 70% formulated mink diet) 1 2 and five treatment diets containing target concentrations ranging from 0.25 to 4 mg/kg tPCBs. 3 These concentrations were established during the study design to span the range of known effects 4 thresholds from previous studies of the effects of PCBs on mink. The diets were fed to captive adult female mink for approximately 160 days. The exposure period for adult females began 5 6 approximately 2 months prior to mating, and continued through mating and whelping of the kits. 7 Some kits were exposed for an additional 6 months following whelping. A variety of endpoints 8 were measured during the study including feed consumption rate, mating success, gestation 9 length, number of kits born, adult and kit survival, body weights, organ weights, and tissue 10 histology. Biochemical parameters and the histopathology of the jaws of mink kits were also 11 measured. The latter endpoints are discussed separately in Sections 9.4.2.3 and 9.4.2.4.

12 9.4.2.2 Results and Discussion

13 The presence of COCs in the diet did not have a significant effect on food intake rate of adult 14 female mink. Consumption of diets containing COCs derived from Housatonic River fish had no 15 significant effect on breeding success (number of females bred/total number of females) or 16 whelping success (number of females whelping/number of females bred) of female mink. 17 Gestation length was not significantly altered by exposure treatments. Average litter size and kit 18 survival at birth and 3 weeks of age were also not affected by the exposure treatments. However, 19 decreased survival of kits in the 3.7 mg/kg tPCB treatment group at 6 weeks of age (i.e., 46% 20 lower compared to controls) was statistically significant compared to kits in the control and 1.6 21 mg/kg tPCB treatment groups (Table I.3-2).

22 There were no significant differences between treatments for adult female body weights at the 23 beginning of the study, during the pre-breeding period, and at 3 and 6 weeks post whelping. 24 However, there was a significant treatment by date interaction for kit body weights from birth to 25 6 weeks of age. At 3 weeks of age, kits in the 0.0.61 mg/kg tPCB treatment group had 26 significantly higher body weights when compared to kits in the other five groups. Kits in the 3.7 27 mg/kg tPCB treatment group had significantly lower body weights when compared to kits in the 28 other groups. At 6 weeks of age, however, mean kit body weight in the 3.7 mg/kg tPCB 29 treatment group was only slightly lower than mean kit body weights observed in the control treatment (251 ± 16.2 g versus 293 ± 11.3 g, respectively). From 10 to 30 weeks of age,
 differences in kit body weights between treatments were minor and did not have a dose dependent relationship with either tPCBs or TEQ.

4 Absolute and relative (expressed as a percentage of body weight) brain, heart, spleen, liver, 5 kidney, and adrenal gland weights of adult females and kits were not significantly different 6 between treatment groups at necropsy. Differences in absolute heart and liver weights of kits 7 between treatments were minor and did not have a dose-dependent relationship with either 8 tPCBs or TEQ. The results of the histological examination of the tissues of major internal 9 organs of the adult female mink and their kits did not show remarkable changes attributable to 10 the treatment diets.

11 9.4.2.3 Mink Jaw Lesion Study

The purpose of this study was to examine the histopathology of jaws of mink from the feeding study by Bursian et al. (2002). The objective was to determine whether the dietary treatments induced lesions that have been previously observed in other studies of mink fed PCB-126 and TCDD. The evaluation was conducted on 6-month kits necropsied at the end of the mink feeding study.

17 **9.4.2.3.1** Methodology

The skulls of 6-month-old mink kits (36 kits collected) were fixed in a 10% formalin-saline solution at necropsy, decalcified in 5% nitric acid, rinsed, trimmed, processed using a routine histotechnologic method, and embedded in paraffin. Tissues were sectioned at 6 microns and stained with hematoxylin and eosin. Jaws from 36 kits were examined for pathologies. There were 6 jaw samples from each of the control and 0.34 0.61 0.96, 1.6, and 3.7 mg tPCBs/kg treatments. The observed lesions were graded as mild, moderate, or severe based on the number and size of foci of squamous cell proliferation in maxilla and mandibles.

25 9.4.2.3.2 Results and Discussion

While none of the mink kits had gross abnormalities of the maxilla and mandible, histological evidence in the form of proliferation of periodontal squamous epithelial cells was present. Nests

1 of squamous epithelium were found adjacent to the teeth and some had cystic centers. The 2 proliferation resulted in focal loss of alveolar bone. Squamous cell proliferation was apparent in 3 17%, 33%, and 100% of kits in the 0.96, 1.6 and 3.7 mg tPCBs/kg treatments, respectively. No 4 lesions were observed in the controls, 0.34, and 0.61 mg tPCBs/kg treatments. The lesions 5 appeared to start from the caudal molar region of the jaw and advanced to the pre-molar, canine, 6 and incisor regions. The initial lesions in the molar region usually consisted of large cysts lined 7 with thick layers of stratified squamous epithelium and filled with floating, sloughed squamous 8 cells. The subsequent lesions in the pre-molar, canine, and incisor regions of the jaw were 9 characterized as multiple nodules of compact stratified squamous epithelium.

These results indicate that dietary concentrations of PCB-126 as low as 54 ng/kg in the diet (0.96 mg tPCBs/kg diet) can induce maxillary and mandibular squamous cell proliferation. Exposure of mink to higher concentrations of PCB-126 for longer periods of time, as would be expected in the Housatonic River ecosystem, would undoubtedly cause increased severity of the lesions leading to erosion of the mandible and maxilla with concomitant loss of teeth. Such an effect could ultimately cause the animal to die of starvation (Bursian et al. 2002; 2003).

16 9.4.2.4 Mink Enzyme Study

17 Tillitt et al. (2003) performed a study to measure hepatic O-dealkylase activities associated with 18 cytochrome P450 (CYP) isozymes induced in mink fed diets containing fish collected from the 19 PSA as part of the MSU feeding study by Bursian et al. (2002). Specific activities were 20 measured against four separate substrates to measure the induction of CYP enzymes in maternal 21 and F1 generations of the exposed mink. The induction of CYP enzymes is a good indicator of 22 exposure to coplanar PCBs, dioxins, and furans, and indicates a first level of toxicological 23 response (Aulerich et al. 2003). Hepatic activities were measured because the majority of 24 detoxification of xenobiotics occurs in the liver.

25 **9.4.2.4.1** Methodology

In the feeding study by Bursian et al. (2002), 36 offspring (kits) along with the adults were used at 6 weeks after whelping. Another 36 kits were used at 6 months post whelping. The livers were removed and placed in 1.2-ml cryovials and frozen in liquid nitrogen. Frozen liver samples from the parental generation, 6-week-old offspring, and 6-month-old offspring were transmitted to the Columbia Environmental Research Center (CERC) for analysis. The analyses consisted of microsomal preparation and various O-dealkylase assays. All procedures were executed according to CERC Standard Operating Procedures (SOPs) and QA/QC procedures.

5 9.4.2.4.2 Results and Discussion

6 Induction of CYP2B-related activity in mink (benzyloxyresorufin-O-deethylase or BROD and 7 pentoxyresorufin-O-deethylase or PROD) was not substantial at any of the doses of fish from the 8 Housatonic River. Only a few dose-age treatment combinations had significant inductions 9 toward BROD or PROD activities. Further, none of the increases in BROD or PROD activities 10 occurred in a dose-dependent fashion. Thus, the amounts of di- to tetra-ortho-chloro-substituted 11 PCBs (PCB congeners thought to be responsible for CYP2B-related enzyme inductions) were 12 either below a threshold of activation of these enzymes in the dietary treatments or the enzyme induction pathways were saturated. Further analysis (protein content or message) would be 13 14 required to discern which of these occurred in these studies.

15 Induction of CYP1A1-related hepatic enzyme activities (ethoxycoumarin-O-deethylase or ECOD 16 and ethoxyresorufin-O-deethylase or EROD) was observed to occur in a dose-dependent manner 17 in all ages of mink examined. Significant increases in these Ah-receptor-regulated enzymes 18 were observed even in treatments with only a small amount of fish from the Housatonic River 19 (0.44%) in their diets. These results confirm the known sensitivity of mink to the effects of 20 tPCBs and other related dioxin-like compounds. The results also confirm that only a small 21 amount of fish (< 0.5%) from the Housatonic River would be required in the diets of mink to 22 activate Ah receptor pathways and processes in mink (Tillitt et al. 2003).

23 9.4.2.5 PCB Congener Comparison in Diet

The composition of PCB congeners in the diet-fish blend used in the mink feeding study was compared to the congener composition measured in Housatonic River fish likely to be consumed by mink, which were used to determine modeled exposure concentrations. This comparison was performed to determine whether there were potential differences in toxicity between the diet blend used in the feeding experiment and the fish that would be consumed by wild mink in the
 PSA. See Appendix C.7 for more detail.

3 **9.4.2.5.1** Methodology

4 The fish component of the mink diet from the feeding study was analyzed by the USGS 5 Columbia Environmental Research Center (CERC) laboratory for 136 individual congeners plus 6 2 co-eluting pairs of congeners. These congeners collectively total over 95% of Aroclor 1260 7 (Frame et al. 2001). The fish used in the exposure analyses for mink were analyzed by the 8 CERC laboratory for 71 individual congeners, 22 co-eluting pairs, and 2 co-eluting triplets, 9 which also represented over 95% of Aroclor 1260. The individual congeners or congener groups 10 common to the two analyses were used for this evaluation, and included 61 individual congeners, 11 20 pairs, and 2 triplicate congener groups for a total of 83 congeners/congener groups.

12 9.4.2.5.2 Results and Discussion

The congener patterns in the feeding study diet were comparable to those in the fish used in the exposure analyses (Figure I.3-5). There were some exceptions, however. The following congeners were higher (difference between means greater than 2 standard errors) in the feeding study diet relative to at least one species of fish used in the exposure analyses:

- 17 PCB-149/123.
- 18 PCB-170/190.
- 19 PCB-174.
- 20 PCB-136.
- PCB-42/37/59.
 - PCB-130.
 - PCB-22/51.
 - PCB-209.
- 24 25

22

- The following congeners were lower in the feeding study diet relative to the fish samples used in the exposure analyses:
- 28
 PCB-82.

 29
 PCB-56.

 30
 PCB-67.
- 31

When the analysis was repeated for all fish species combined, there was an increase in the number of congener concentrations that differed by more that two standard errors between the feeding study diet and exposure analysis fish. That increase was attributed to a drop in standard error due to the increased number of samples (N = 92). There were 15 congeners that were higher and 27 congeners that were lower in the feeding study diet than in the exposure analysis fish.

7 The percent contribution of several coplanar congeners to the tPCB mixture differs slightly in the 8 fish used in the feeding study versus the fish used in the exposure analyses. The mean percent 9 contribution of the most toxic congener, PCB-126, in the fish used in the exposure analyses was 10 approximately 0.022% compared to approximately 0.005% in the fish used in the feeding study 11 (Figure I.3-6). However, because the error bars (± 2 standard errors) for the two means overlap, 12 it is unlikely that these differences are statistically meaningful. Therefore, the PCB composition 13 in fish from the feeding study can be treated as similar to that in fish used in the exposure 14 analyses and the results from both studies are directly comparable.

15 9.4.3 Effects Metrics for Characterizing Risk

Effects data can be summarized in a variety of ways ranging from benchmarks designed to be protective of most or all species to dose-response curves. A summary of the decision criteria used to derive effects metrics is provided in the text box. Further details on the decision criteria used in selecting effects metrics is provided in Section 6.6.

2	Decision Criteria for Derivation of Effects Metrics
3 4	The following is the hierarchy of decision criteria used to characterize effects for each receptor-COC combination:
5 6 7	1. Have single-study bioassays with five or more treatments been conducted on the receptor of interest or a reasonable surrogate? If yes, estimate the concentration- or dose-response relationships. If not, go to 2.
8 9 10 11	2. Are multiple bioassays with similar protocols, exposure scenarios and effects metrics available that, when combined, have five or more treatments for the receptor of interest or a reasonable surrogate? If yes, estimate the dose-response relationship as in 1. If not, go to 3.
12 13 14	3. Have bioassays with less than five treatments been conducted on the receptor of interest or a reasonable surrogate? If yes, conduct or report results of hypothesis testing to determine the NOAEL and LOAEL. If not, go to 4.
15 16 17 18	 Are sufficient data available from field studies and monitoring programs to estimate concentrations or doses of the COC that are consistently protective or associated with adverse effects? If yes, develop field-based effects metrics. If not, go to 5.
19 20 21 22 23 24 25	5. Derive a range where the threshold for the receptor of interest is expected to occur. Because information on the sensitivity of the receptor of interest is lacking, it is difficult to derive a threshold that is biased neither high nor low. If bioassay data are available for several other species, however, calculate a threshold for each to determine a threshold range that spans sensitive and tolerant species. That range is likely to include the threshold for the receptor of interest.

1

In this ERA, data were available to derive dose-response curves for mink exposed to tPCBs.
There were insufficient data to derive dose-response relationships for TEQ. Field-based
threshold range was derived instead. There were no toxicity data for river otter. Mink toxicity
data were used as surrogate estimates of toxicity of tPCBs and TEQ to river otter.

31 9.4.3.1 Effects of tPCBs to Mink and River Otter

Derivation of a dose-response curve requires long-term feeding studies that singly or combined have at least five dose treatments for sensitive endpoints such as mortality or reproductive success. The acceptable studies that met these criteria were the Bleavins et al. (1980) and Aulerich et al. (1985) studies. Because both studies used similar protocols, exposure duration, and species (a similar strain of farm-raised mink), they were combined to yield a data set with nine treatments for fecundity. Figure 9.4-1 presents the dose-response curve for reduced
 fecundity of mink exposed to tPCBs. The dose-response curve indicates that 10% and 20%
 declines in fecundity would be expected at doses of 0.0128 and 0.0272 mg/kg bw/d, respectively.



4 5 Note: Symbols indicate raw data.

6 Figure 9.4-1 Dose Response Curve for Effects of tPCBs on Fecundity of Mink

7 9.4.3.2 Effects of TEQ to Mink and River Otter

The studies by Heaton et al. (1995), Hochstein et al. (1998, 2001), and Aulerich et al. (1988) involved exposing mink to fish contaminated with TEQ and other contaminants. Based on a review of these studies, adverse effects on growth in kits begin to occur at concentrations of 3.6 ng/kg bw/d (lower toxicity threshold; Heaton et al. 1995). The highest dose that did not cause adverse effects was 36 ng/kg bw/d (upper toxicity threshold; Hochstein et al. 2001). Thus, the threshold range, based on studies that used field-collected fish, is 3.6 to 36 ng/kg bw/d for piscivorous mammals.

1 9.5 RISK CHARACTERIZATION

This section characterizes the risk to piscivorous mammals exposed to tPCBs and TEQ in the PSA of the Housatonic River. The risk characterization uses three lines of evidence to determine ecological risks to this group of mammals. The three major lines of evidence are considered to be independent and are combined in a weight-of-evidence (WOE) assessment. The key risk questions and the three lines of evidence are summarized in the text box.

7	Key Risk Questions			
8 9 10	•	Are the concentrations of tPCBs and TEQ present in the prey of piscivorous mammals sufficient to cause adverse effects to individuals inhabiting the PSA of the Housatonic River?		
11	•	If so, how severe are the risks and what are their potential consequences?		
12		Lines of Evidence		
13	•	Use of semi-qualitative field surveys.		
14	•	Probabilistic exposure and effects modeling.		
15	•	Feeding study using fish from the PSA.		

16

17 Section 9.5.1 presents a brief overview of the methodology, results, and interpretation of the 18 mammal surveys conducted from 1998 to 2001 in the Housatonic PSA. A more detailed 19 presentation of this information can be found in Appendix A. In Section 9.5.2, the dose-response 20 curves are combined with the corresponding exposure distributions to derive risk curves that 21 characterize the relationship between probability and magnitude of effect. The results of the 22 mink feeding study are summarized in Section 9.5.3. A WOE assessment is presented is Section 23 9.5.4, along with sources of uncertainty (Section 9.5.5) and the overall findings of the risk 24 characterization (Section 9.5.8).

25 9.5.1 Field Surveys

26 9.5.1.1 EPA Study

The mammalian community in the PSA was studied by EPA over a 4-year period, from 1998 to 28 2001. Surveys were conducted to record presence, relative abundance, and habitat usage for 29 small and large mammals including mink and river otter. A variety of field survey techniques including small mammal trapping, snow tracking, and scent-post station surveys were used to characterize the mammalian community. Mink tracks and scats were observed at several locations in the PSA during snow-tracking surveys. Tracks were observed at the northern and southern-most portions of the PSA; no observations occurred in the middle portions. Mink were also observed at the Washington Mountain Lake and Ashley Lake reference areas during the 1999 and 2000 surveys. On a per effort basis, mink were observed in the reference areas twice as frequently as in the PSA (Table 9.5-1).



0	

Table 9.5-1 Results of Snow Tracking and Scent Post Station Surveysin the PSA and Reference Areas

10	Primary Study Area	Mink	Otter
	Hours of survey effort	260.5	260.5
11	Number of sightings	5	4
	No. sightings/hour	0.019	0.015
12	Reference Areas		
12	Hours of survey effort	108.0	108.0
13	Number of sightings	4	14
14	No. sightings/hour	0.037	0.130

15

River otter signs were observed on only four occasions in three locations in the PSA. Otter tracks, slides, and scats were observed in the reference areas relatively frequently. A nearly 9:1 ratio of observations per unit effort occurred between the reference areas and the PSA during the tracking surveys (Table 9.5-1) and, additionally, families of river otter were repeatedly observed in the reference areas during the course of other field surveys.

The Housatonic River in the PSA offers an abundance of habitat that meets the requirements for mink and river otter. Their occurrence in the PSA, however, was much lower than would be expected, considering the large amount of available habitat and food resources. Despite hundreds of hours specifically conducting track and scent post surveys for these species and thousands of person-days spent conducting other field surveys and sampling, only a handful of observations of each species occurred. Based on the experience of the field personnel and the
substantial number of hours spent in the study area from 1998 to 2001, far fewer observations of
mink and otter, or their sign, occurred than would be normally expected in a riverine system such
as the PSA.

5 9.5.1.2 GE Study

6 The General Electric (GE) company studied the presence or absence and possible distribution of 7 wild mink in the Housatonic River Study Area from the spring of 2001 to the spring of 2003 8 (Bernstein et al. 2003). River otter (Lutra canadensis) were included in the study of winter of 9 2002/2003. The methods were similar to those used in the EPA surveys, and consisted of 10 looking for tracks in soft sand at scent post stations in the spring, summer, and fall (mink only), 11 and snow tracking (mink and otter) during the winter months. The survey efforts were 12 concentrated in suitable mink habitat, irregular shorelines and backwaters with dense, wooded 13 cover near the water. Additional efforts included setting traps (100 in total), scented burrows, 14 and motion sensitive camera sites. The observations were conducted along the Housatonic River 15 between New Lenox Road and Woods Pond (the midpoint of Reach 5B, and Reach 5C and 6; no 16 work was performed in the upstream half of the PSA).

17 The study reported 35 sets of mink tracks between April 2001 and March 2002. A total of 33 18 mink track sets and 41 river otter track sets were observed in 2003. However, only 4 out of the 19 35 mink track sets were observed in the snow-free months. In 2003, all mink and river otter 20 tracks were observed in winter. This suggests that either the observation methods used in the 21 spring, summer, and fall were not effective or that mink were not present in the PSA. The 22 former could have been due to issues with methods used during the construction of scent posts 23 (e.g., failure to wear rubber boots and gloves during the construction of the scent posts) 24 (Woodlot Alternatives, Inc. 2003). If the lack of sightings during the spring, summer, and fall 25 was due to lack of mink in the PSA (a hypothesis supported by very few tracks at post stations, 26 no photographs recorded by the motion-sensitive camera in the snow-free months, and no tracks 27 in scented burrows), then the tracks detected in winter likely belonged to transient mink rather 28 than to local residents. The 2003 survey discovered one confirmed and one suspected river otter 29 den site in winter, suggesting that that some river otter might be present in the PSA for extended periods of time. However, the presence of one, perhaps a temporary den site, in winter is
 insufficient as evidence for a healthy and reproducing river otter population in the PSA.

3 In summary, the GE report cited incidences of mink and river otter signs in the PSA. However, 4 the study had several limitations, which lead to conclusions that are not supported by the data. 5 These limitations included the failure to discuss the implications of the disproportionate number 6 of sightings in winter versus other seasons, apparent ineffectiveness of scent posts, no 7 established reference areas outside the PSA, lack of tracking expertise and experience, empty 8 traps, no results from motion-sensitive camera trials (i.e., no mink or river otter observed in the 9 snow-free months trials), uncertainty in determining sex of mink from tracks, and uncertainty in 10 attributing a different sets of tracks to separate individuals.

9.5.2 Comparison of Estimated Exposures to Laboratory-Derived Effects Doses

12 Exposure was assessed for mink and river otter in the PSA. Because Reaches 5 and 6 combined 13 roughly correspond to the size of the home range for otter, these reaches were combined for the 14 river otter analysis. For mink, the assessment was conducted separately for each reach because 15 of their smaller foraging range. For comparative purposes, exposure was also estimated for mink 16 and otter in two reference areas: the upstream reference area and Threemile Pond. Moreover, 17 exposure was also estimated for mink and river otter foraging 50, 25, and 10% of time in the 18 PSA. For each receptor-COC combination, a category of low, intermediate, or high risk was 19 assigned following integration of the exposure and effects distributions. This exercise was done 20 separately for the results of the Monte Carlo analyses and each of the lower and upper bounds 21 from the probability bounds analyses. The "risk category" refers to the level of risk based on the 22 results of the Monte Carlo analysis. The "risk range" refers to the levels of risk based on the 23 results of the probability bounds analyses.

	Guidance for Determining Level of Risk to Representative Species				
F	Risk Curves for Mink and River Otter Available				
•	If the probability of 10% or greater effect is less than 20%, then the risk to piscivorous mammals is low.				
•	If the probability of 20% or greater effect is greater than 50%, then the risk to piscivorous mammals is high.				
-	All other outcomes are considered to have intermediate risk.				
F	Risk Curves for Mink and River Otter Not Available				
•	If the probability of exceeding the lower toxicity threshold was less than 20%, th risk to piscivorous mammals was low.				
•	If the probability of exceeding the upper toxicity threshold was greater than 20% the risk to piscivorous mammals was high.				
•	All other outcomes for the lower and upper thresholds were considered to have intermediate risk.				

The results of the risk characterization are summarized in Table 9.5-2. Figures 9.5-1 through to 9.5-14 depict the risk curves for mink and river otter exposed to tPCBs in Reaches 5 and 6, including the Monte Carlo estimate and both the lower and upper probability bounds (LPB and UPB, respectively). The highest risk to mink and river otter is from exposure to tPCBs in Reaches 5 and 6. Risks were much lower in the reference areas. The exposure in Reaches 5 and 6 is so high that individuals foraging 10% of their time at those locations would experience high risk.

25

Table 9.5-2

Summary of Qualitative Risk Statements for Piscivorous Mammals from the

Housatonic River Study Area

	Qualitative Risk Statements				
	PCBs		TEQ		
Location	Risk Category	Risk Range	Risk Category	Risk Range	
Mink					
Reach 5	High	High	High	Intermediate/High	
Reach 6	High	Intermediate/High	High	Intermediate/High	
Upstream Reference Area	Intermediate	Low/High	Intermediate	Low/High	
Threemile Pond	High	Low/High	Low	Low/High	
River Otter					
Reaches 5 and 6	High	High	High	High	
Upstream Reference Area	Intermediate	Low/Intermediate	Intermediate	Low/Intermediate	
Threemile Pond	Low	Low/Intermediate	Low	Low/Intermediate	

5

6 The degree of risk for mink (and otter) associated with exposure to tPCBs downstream of Woods 7 Pond was assessed by comparing concentrations of tPCBs in prey fish of mink (5 to 20 cm) in 8 Reaches 7 to 16 to a maximum acceptable threshold concentration (MATC) developed 9 specifically for mink (Appendix I). Fish residue data were obtained from sampling efforts from 10 1998 to 2002. The MATC of 2.65 mg/kg tPCBs in fish (whole body, wet weight) was developed 11 as the geometric mean of the NOAEL and LOAEL developed by Bursian et al. (2002) in the site-12 specific study of the toxicity of Housatonic River fish to mink. The LOAEL was based on the 13 observation of significantly reduced mink kit survivability at 6 weeks of age. The value of this 14 LOAEL was estimated at 3.7 mg/kg feed supplied to reproducing females. The NOAEL was 15 based on the same endpoint and its value was 1.6 mg/kg feed.



2 3

Figure 9.5-1 Total PCB Risk to Mink Exposed to tPCBs in Reach 5 of the Housatonic River





2 3 4

Figure 9.5-2 Total PCB Risk to Mink (10% Foraging Time in Reach 5)



Figure 9.5-3 Total PCB Risk to Mink Exposed to tPCBs in Reach 6 of the Housatonic River

2 3





23
Upstream Reference Area











Figure 9.5-6 Total PCB Risk to Mink (10% Foraging Time in the Upstream Reference Area)





2

3

4

Figure 9.5-7 Total PCB Risk to Mink Exposed to tPCBs in the Threemile Pond Reference Area

Threemile Pond Reference Area



Figure 9.5-8 Total PCB Risk to Mink (10% Foraging Time in the Threemile Pond Reference Area)

4

1

2 3





1 2 3 4 5

Note: The LPB and UPB overlap the Monte Carlo line.

Figure 9.5-9 Total PCB Risk to River Otter Exposed to tPCBs in Reaches 5 and 6 of the Housatonic River





2 3

4





Figure 9.5-11 Total PCB Risk to River Otter Foraging in the Upstream Reference Area

Upstream Reference Area



Upstream Reference Area)

2 3

1

Threemile Pond Reference Area



Total PCB Risk to River Otter Exposed to tPCBs in the Threemile Pond Reference Area

2 3

4

Threemile Pond Reference Area



Figure 9.5-14 Total PCB Risk to River Otter (10% Foraging Time in the Threemile Pond Reference Area)

4 5

1 2

3

9.5.3 Mink Feeding Study

6 Consumption of diets containing tPCBs and TEQ derived from fish collected from the 7 Housatonic River did not have an adverse effect on adult mink reproduction as assessed by 8 breeding success, whelping success, and gestation length. Kit survival at 6 weeks of age, 9 however, was significantly decreased in the 3.7 mg/kg tPCBs (68.5 ng/kg TEQ) treatment group. 10 In this treatment, less than 4% of the diet was derived from Housatonic River fish, which is well 11 below what mink typically consume in the wild (23% on average with a range from 0 to 65%). 12 The enzyme induction analysis indicated that ECOD and EROD enzymes were induced even 13 when the Housatonic River fish content in the diet was very low (0.44%; 0.50 mg/kg tPCBs) 14 attesting to the high sensitivity of mink to tPCBs and TEQ. The histopathological examination 15 of kit jawbones revealed that jaw lesions were apparent at tPCB treatments as low as 0.96 mg/kg 16 diet (0.88% Housatonic River fish)(Bursian 2002, 2003).

1 9.5.4 Weight-of-Evidence Analysis

2 A weight-of-evidence (WOE) analysis was used to combine the three major lines of evidence 3 described in the preceding sections for mink and river otter. The goal of this analysis was to 4 determine whether significant risk is posed to piscivorous mammals in the Housatonic River 5 PSA as a result of exposure to tPCBs and TEQ. The three-phase approach of Menzie et al. 6 (1996) and the Massachusetts Weight-of-Evidence Workgroup was used, in which WOE was 7 expressed with the following three characteristics: (1) the weight assigned to each measurement 8 endpoint; (2) the magnitude of response observed in the measurement endpoint; and (3) the 9 concurrence among outcomes of the multiple measurement endpoints.

Each measurement endpoint was evaluated and assigned a qualitative weight in Appendix I, along with a discussion of the reason for the value assigned (Table 9.5-3). The EPA field survey had a moderate to high value, the GE field survey had a moderate value, the feeding study had a high value, and the modeled exposure and effects line of evidence for tPCBs and TEQ had a high and moderate/high values in the overall WOE analysis.

The magnitude of the response in the measurement endpoint is considered together with the measurement endpoint value in developing the overall WOE (Menzie et al. 1996). This requires assessing the strength of evidence that ecological harm has occurred, as well as an indication of the magnitude of response, if present. The weighting values, evidence of harm, and magnitude of response were combined in a matrix format and are presented in Tables 9.5-4 and 9.5-5.

Table 9.5-3

Weighting of Measurement Endpoints for Piscivorous Mammals Weight-of-Evidence Evaluation

Attributes	Field Surveys		Feeding Study	Modeled Exposure and Effects		
	EPA	GE	Study	tPCBs	TEQ	
I. Relationship Between Measur	rement and As	ssessment En	dpoints			
1. Degree of Association	L/M	L/M	Н	Н	Н	
2. Stressor/Response	М	L/M	M/H	Н	М	
3. Utility of Measure	М	L/M	Н	M/H	М	
II. Data Quality						
4. Data Quality	M/H	L	Н	M/H	M/H	
III. Study Design	III. Study Design					
5. Site Specificity	Н	M/H	М	L/M	L/M	
6. Sensitivity	М	L/M	Н	Н	Н	
7. Spatial Representativeness	Н	M/H	M/H	M/H	M/H	
8. Temporal Representativeness	Н	M/H	M/H	M/H	M/H	
9. Quantitative Measure	L	L	Н	Н	Н	
10. Standard Method	Н	M/H	Н	M/H	M/H	
Overall Endpoint Value	M/H	М	Н	Н	M/H	

L = low; M = moderate; H = high

Table 9.5-4

Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints		Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	
Field Surveys	EPA	Moderate/High	Yes	High	
Tield Surveys	GE	Moderate	No	Low	
Feeding Study		High	Yes	High	
Modeled Exposure and Effects		Moderate/High	Yes	High	

5

6

7

8

9

10

Table 9.5-5

Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints		Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	
Field Surveys	EPA	Moderate/High	Yes	High	
Field Surveys	GE	Moderate	No	Low	
Feeding Study		High	Yes	High	
Modeled Exposure and Effects		Moderate/High	Yes	High	

All three lines of evidence indicated that the elevated concentrations of tPCBs and TEQ in the 1 2 PSA of the Housatonic River are causing adverse effects of high magnitude to mink and river 3 otter. The field surveys indicated that mink and river otter are rarely present in the PSA, except 4 during winter, and likely have not established home territories close to the main channel despite 5 suitable mink and otter habitat. The MSU feeding study indicated that feeding adult female mink 6 with a diet containing as little as 3.51% fish from the PSA caused a statistically significant 7 reduction (46% compared to controls) in kit survival to 6 weeks of age. Because mink in the 8 wild typically consume between 20% or more fish in their diet, the associated risk is 9 correspondingly higher. In addition, other components of the mink diet in the PSA (e.g., 10 crayfish) have high concentrations of tPCBs and TEQ. Further, the jaw lesion study indicated 11 that erosion of the jaw occurs at even lower doses and exhibits a dose-response relationship. 12 Such effects could eventually lead to starvation. The occurrence of jaw lesions coincides with 13 the induction of Ah-receptor-regulated enzymes (ECOD and EROD) also in a dose-response 14 manner.

The high risks evident from the feeding study are further supported by the modeled exposure and effects line of evidence. The estimated potential for exposure is so high that even individual mink and otter that only forage in the PSA for short periods of time (less than or equal to 10% of foraging time) are at an intermediate or higher risk from tPCBs and TEQ.

A graphical method was used for displaying concurrence among measurement endpoints. Tables
9.5-6 and 9.5-7 depict the outcome for piscivorous mammals exposed to tPCBs and TEQ,
respectively. The three measurement endpoints have a high degree of concurrence.

Table 9.5-6

Risk Analysis Summary for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Assessment Endpoint: Survival, growth, and reproduction of piscivorous mammals ► Weighting Factors (increasing confidence of weight) Harm/Magnitude Low Low/Moderate Moderate Moderate/High High Yes/High MEE, FS-EPA MFS Yes/Indeterminate Yes/Low Undetermined/High Undetermined/Intermediate Undetermined/Low -----_____ No/Low FS-GE ▼ No/Intermediate No/High

6

7 FS-EPA = Field surveys by EPA

8 FS-GE = Field surveys by GE

9 MFS = Mink feeding study

10 MEE = Modeled exposure and effects

11

1

2

3

4

Table 9.5-7

Risk Analysis Summary for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSA

Assessment Endpoint:	Survival,	growth, and reprodu	ction of piscive	orous mammals		
	Weighting Factors (increasing confidence of weight)]	
Harm/Magnitude	Low	Low/Moderate	Moderate	Moderate/High	High	
Yes/High				MEE, FS-EPA	MFS	٦.
Yes/Intermediate] ↑
Yes/Low						
						!
Undetermined/High						
Undetermined/Intermediate						
Undetermined/Low						
No/Low			FS-GE]↓
No/Intermediate						
No/High						

6 FS-EPA = Field surveys by EPA

7 FS-GE = Field surveys by GE

- 8 MFS = Mink feeding study
- 9 MEE = Modeled exposure and effects
- 10

1

2 3

4

5

11 9.5.5 Sources of Uncertainty

The assessment of risk to piscivorous mammals contains uncertainties. Each source of uncertainty can influence the estimates of risk. Therefore, it is important to describe, and when possible, specify the magnitude and direction of such uncertainties. Some of the major sources of uncertainty associated with the assessment of risks of tPCBs and TEQ to piscivorous mammals are briefly described below. An expanded discussion is presented in Appendix I.

In this assessment, it was assumed that dietary exposure represented the most important pathway of exposure for piscivorous mammals exposed to COCs.
 Although unlikely to provide a major contribution to the risk, other pathways could increase the exposure and perhaps increase risk slightly (Moore et al. 1999). Other

pathways include drinking water intake, incidental ingestion of sediment, inhalation, transdermal uptake, and preening activity. When drinking water was included in a screening level analysis for piscivorous mammals, the results showed negligible increases in exposure due to drinking water and their likely low importance. The remaining pathways were not assessed due to the difficulty in quantifying intake via those routes.

- 7 The Monte Carlo sensitivity analyses indicated that the free metabolic rate (FMR) 8 slope and power terms were generally the most influential variables on predicted total 9 daily intakes of COCs. However, no measurements of free metabolic rate were 10 available for the representative wildlife species. Similarly, measured food intake rates were not available for free-living mink and river otter or reasonable surrogate 11 species. Therefore, free metabolic rates were estimated using allometric equations. 12 13 The use of allometric equations introduces some uncertainty into the exposure 14 estimates because they have model-fitting error and are based on species different 15 from the representative species used in this assessment. For mink and river otter, the 16 carnivora model of Nagy et al. (1999) was selected as the most appropriate allometric model to estimate free metabolic rate. Examples of other species used in the model 17 18 included the cat, fox, dog, and wolf. Given the lack of data on representative species 19 used in the current assessment, it is difficult to judge the magnitude of the uncertainty 20 introduced by the use of the allometric models. The uncertainty due to model-fitting 21 error was propagated in the uncertainty analyses by using distributions as inputs for 22 the allometric slope and power terms.
- 23 Because no stomach contents or other dietary analyses were available for mink in the 24 PSA, dietary compositions were derived from those reported in the literature from other similar geographical locations. The potential magnitude and direction of the 25 uncertainty associated with lack of information on diet are unknown. The uncertainty 26 27 due to lack of knowledge on diet of mink in the PSA was partially addressed by using distributions to represent variability in diets observed at other similar sites. Small 28 29 mammals were the most contaminated prey, thus, any increases in the proportion of this type of prey consumed would lead to increases in exposure. Conversely, 30 31 amphibians were the least contaminated prey, thus increases in the intake of this prey 32 item would lead to decreases in exposure to tPCBs and TEQ.
- 33 Sample sizes were limited for the analyses of COC concentrations in some prey items. For example, there were only four amphibian samples for tPCBs from the 34 upstream reference area, only one amphibian sample for TEQ from the upstream 35 reference area and the Threemile Pond reference area, and only three invertebrate 36 37 samples for TEQ from the upstream reference area. Uncertainty due to sample size 38 was explicitly addressed in the uncertainty analyses. In the Monte Carlo analysis, 39 sample size uncertainty was addressed by use of the 95% upper confidence limit (UCL) on the mean. The use of the UCL addressed uncertainty, but it was biased 40 41 toward overestimating exposure. In the probability bounds analysis, uncertainty was addressed by specifying concentration variables as a range from the 5% lower 42 43 confidence limit (LCL) to the UCL. This treatment of uncertainty was unbiased.

1 2

3

4

5

- Data on concentrations of tPCBs and TEQ in crayfish and mammals were not available for Reach 6. In those cases, the concentrations of tPCBs and TEQ in prey were estimated using ratios between prey items at other locations for which full data sets existed. This type of extrapolation introduces some uncertainty regarding the concentration of COCs in prey tissue, although the magnitude and direction of this uncertainty is difficult to judge.
- In some instances, data on concentrations of tPCBs and TEQ in crayfish, birds, and mammals from reference areas were missing. In those cases, an assumption was made that these prey items contained no detectable residues of the two contaminants.
 The magnitude of uncertainty introduced by this assumption is likely to be small because sediment data indicate that detectable tPCB and TEQ residues are rare at those sites.
- The base exposure scenario for mink and river otter assumed that these animals would forage 100% of their time in the PSA. This assumption is reasonable given the similarity between the size of the PSA and the foraging ranges of these species. However, some individuals might forage part of their time outside the PSA on less contaminated prey. The exposure and risk analyses indicated that individual mink and river otter that forage even a small fraction of time in the PSA (10%) are at high risk, particularly for tPCBs.
- 20 The effects metrics used to estimate risk to piscivorous mammals via exposure 21 models were derived for Aroclor 1254 mixtures. Some uncertainty is inherent in 22 extrapolating from studies using the Aroclor 1254 mixture to the specific congener 23 patterns observed in weathered mixtures in the PSA of the Housatonic River. The 24 feeding study with mink using fish from the PSA suggested that the PCB mixture in 25 fish (most closely resembling Aroclor 1260) was less toxic than the PCB mixture 26 reported in literature (Aroclor 1254). Thus, the risk to mink and river otter estimated 27 by the exposure model may be slightly overestimated. This overestimate, however, 28 does not affect the final risk conclusion due to the very high exposure rates for mink 29 and river otter.
- 30 The comparison of PCB congeners in the diet-fish blend used in the MSU study to the 31 congener composition measured in Housatonic River fish revealed (with few 32 exceptions) that the congener patterns (and potency) in the feeding study diet were 33 comparable to those in the fish used in the exposure analyses. However, there was 34 some uncertainty as to the influence of PCB-126 on the toxicity of the treatment diet 35 (PCB-126 content of 0.005%) vs. exposure analysis fish (PCB-126 content of 0.022%). Although the percentages were within two standard errors of each other 36 37 (criteria for similarity), the difference might have contributed to the lower than expected toxicity of tPCBs observed in the feeding study. 38
- There was some uncertainty whether other COCs present in Saginaw Bay fish contributed to the increased toxicity of those fish compared to fish from the Housatonic River.

- It was uncertain whether the food intake rates of the mink fed fish from the Housatonic River were comparable to the corresponding rates observed for mink in the Saginaw Bay study.
- There was uncertainty whether the congener mixture in the Housatonic River fish has
 the same potency as the mixture in the Saginaw Bay fish.
- The GE mink and otter study lacks critical information needed to confirm track identification (i.e., multiple measurements with a scale) and sex determination (i.e., photographs of tracks, most without a scale, were sent out of state to scientists in Louisiana). In addition, data on spacing between paired tracks and on lope distance were not presented to segregate male long-tailed weasels from female mink. Without this supporting information, the results and interpretation remain questionable.
- In the GE study, the use of a study area that represents only a portion of Housatonic River and adjacent floodplain known to be affected by PCB contamination, and the lack of reference areas creates uncertainty, and limits the ability to draw inferences about whether the number of individual mink and otter observed was comparable to other uncontaminated sites in this area.
- 17 9.5.6 Comparison to Other Piscivorous Mammals
- 18 There are no piscivorous mammals other than mink and river otter in the PSA.

19 9.5.7 Risk Downstream of PSA

20 The risk for mink and river otter associated with exposure to tPCBs downstream of Woods Pond 21 was assessed by comparing concentrations of tPCBs in prey fish (5 to 20 cm) in Reaches 7 to 16 22 to a maximum acceptable threshold concentration (MATC) developed specifically for mink (also 23 used for river otter). For the downstream assessment for mink, it was assumed that the fish 24 constituted 23% of the diet and invertebrates (mostly crayfish) 36% of the diet. The remaining 25 41% of the diet consisted of other uncontaminated dietary items. No crayfish data were available 26 for the downstream reaches. However, crayfish residues in the PSA were similar to the fish 27 residues. Therefore, it was assumed that 59% of the diet of mink foraging downstream of Woods 28 Pond was composed of fish. For the downstream assessment of river otter, it was assumed that 29 100% of the diet was composed of fish. On average, however, river otter consume 30 approximately 80% fish and 20% crayfish. Crayfish data were not available for the downstream 31 reaches and tissue residue concentrations in crayfish were approximated using fish residues

1 (which were similar in the PSA). Thus, the assumption of 100% fish diet for downstream otter
2 was reasonable.

The MATC of 2.65 mg/kg tPCBs in fish (whole body, wet weight) was developed as the geometric mean of the NOAEL and LOAEL developed by Bursian et al. (2002) in a site-specific study of the toxicity of Housatonic River fish to mink. The LOAEL was based on the observation of significantly reduced mink kit survivability at 6 weeks of age. The value of this LOAEL was estimated at 3.7 mg/kg feed supplied to reproducing dams. The NOAEL was based on the same endpoint and its value was 1.6 mg/kg feed.

9 To determine the extent and types of habitats available for mink and river otter downstream, U.S. 10 Fish and Wildlife Service National Wetland Inventory, U.S. Geological Survey Topographical 11 Quadrangle maps, and aerial photos of the river were examined in detail. The species-habitat 12 matrix in Appendix A.2 (Ecological Characterization of the Housatonic River Downstream of 13 Woods Pond) identified potential habitat for mink and otter. According to this analysis, potential 14 mink habitat is ubiquitous and includes all areas except high gradient stream, calcareous rock 15 cliff, cultural grassland, agricultural cropland, and residential/industrial development. Potential 16 river otter habitat is less abundant and centers more on larger wetland systems, with slower 17 flowing water, or with impounded water. Any places where the river is impounded, or near a 18 lake or pond, there is potential river otter habitat.

Fish tissue data were obtained from sampling efforts conducted during 1998 to 2002. The results of the analysis are presented in Figures I.5-15 and I.5-16. Potential risk to mink and river otter exists in river sections from Woods Pond to the end of Reach 10 (mink) and 12 (river otter).

22 9.5.8 Conclusions

For piscivorous mammals, data from three major lines of evidence were available, including field surveys, mink feeding study, and exposure and effects modeling. In general, the weight-ofevidence analysis indicates an intermediate to high risk for mink and river otter to tPCBs and TEQ in the PSA. Field surveys by EPA and GE were conducted to determine the presence of mink and river otter in the PSA. The surveys were not designed to provide a quantitative evaluation of the relationship between exposure to COCs and the survival, growth, and reproduction of piscivorous mammals in the PSA. Instead, the surveys determined the presence and relative abundance of piscivorous mammals. Signs of mink and river otter were observed in the PSA, but mostly in winter, suggesting that mink and river otter that are present in the PSA are there on a transient basis.

8 The mink feeding study was designed to determine the effects on growth and reproduction of 9 captive mink fed a diet containing fish from the PSA. The results from this study indicated that 10 feeding adult female mink with a diet containing as little as 3.51% fish from the PSA caused a 11 statistically significant reduction (46% compared to controls) in kit survival to 6 weeks of age. 12 Because mink in the wild typically consume between 0 and 65% fish in their diet (mean 23%), 13 the associated risk is correspondingly higher. Further, the jaw lesion study indicates that erosion 14 of the jaw occurs at even lower doses and exhibits a dose-response. Such effects could 15 eventually lead to starvation. The occurrence of jaw lesions coincides with the induction of Ah-16 receptor-regulated enzymes (ECOD and EROD) also in a dose-responsive manner.

17 The modeling of exposure and effects line of evidence was used to determine the level of risk to 18 the representative mammal species, mink and river otter. The effects characterization developed 19 a dose-response curve to describe the potential effects of tPCBs to piscivorous mammals. 20 Toxicity benchmarks based on mink studies were developed for TEQ. The dose-response curve 21 for effects of tPCBs to piscivorous mammals indicated that 10% and 20% declines in fecundity 22 would be expected at doses of 0.0128 and 0.0272 mg/kg bw/d, respectively. For TEQ 23 benchmarks (reduction in kit growth), the lower threshold was set at 3.6 ng/kg bw/d and the 24 upper threshold was set at 36 ng/kg bw/d. The modeled exposure results indicated that the daily 25 intake rates of tPCBs by mink and river otter were far greater than the toxicity thresholds. This 26 means that mink and river otter feeding in the PSA receive tPCB doses that cause adverse 27 reproductive effects. A similar conclusion was reached for TEQ.

2	ERA Summary
3 4	The weight-of-evidence analysis indicates an intermediate to high risk for mink and river otter exposed to tPCBs and TEQ in the PSA.
5 6	The risk continues to be elevated for individuals that forage only a small fraction of their time in the PSA.
7 8 9	Downstream of Woods Pond (Reach 6), a screening level ERA indicated that mink and river otter may be at risk from exposure to tPCBs and TEQ as far as Reach 10 and 12, respectively.
10	

12 9.6 REFERENCES

1

Alexander, G.R. 1977. Food of vertebrate predators on trout waters in north central lower Michigan.
 The Michigan Academician 10:181-195.

Allen, A.W. 1986. *Habitat Suitability Index Models: Mink, Revised*. U.S. Fish and Wildlife Service
Biological Report 82(10.127). 23 pp.

Anderson, E.A., and A. Woolf. 1987. River otter food habits in northwestern Illinois. *Transactions of the Illinois State Academy of Science* 80:115-118.

Arnold, T.W., and E.K. Fritzell. 1987. Activity patterns, movements, and home ranges of prairie
 mink. *Prairie Naturalist* 19:25-32.

- Arnold, T.W., and E.K. Fritzell. 1990. Habitat use by male mink in relation to wetland characteristics and avian prey abundance. *Canadian Journal of Zoology* 68:2205-2208.
- Aulerich, R.J., R.K. Ringer, and S. Iwamoto. 1973. Reproductive failure and mortality in mink
 fed on Great Lakes fish. *Journal of Reproduction and Fertility Supplements* 19:365-376.
- 25 Aulerich, R.J., S.J. Bursian, W.J. Breslin, B.A. Olson, and R.K. Ringer. 1985. Toxicological
- 26 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. *Journal of Toxicology and Environmental Health* 15:63-79.
- Aulerich, R.J., S.J. Bursian, M.G. Evans, J.R. Hochstein, K.A. Koudele, B.A. Olson, and A.C.
 Napolitano. 1987. Toxicity of 3,4,5,3',4',5'-hexachlorobiphenyl to mink. *Archives of Environmental Contamination and Toxicology* 16:53-60.
- 31 Aulerich, R.J., S.J. Bursian, and A.C. Napolitano. 1988. Biological effects of epidermal growth
- 32 factor and 2,3,7,8-tetrachlorodibenzo-p-dioxin on developmental parameters of neonatal mink.
- 33 Archives of Environmental Contamination and Toxicology 17:27-31.

- 1 Aulerich, R.J., S. Bursian, and B. Yamini. 2003. Hepatic 0-dealkylase activities from induction of
- 2 cytochrome P-450 in mink and their offspring fed diets containing contaminated fish from the
- 3 Housatonic River. USGS Biochemistry and Physiology Branch Final Laboratory Report FY 2003-
- 4 30-4. 18 pp.
- 5 Barrett, G.W., and K.L. Stueck. 1976. Caloric ingestion rate and assimilation efficiency of the 6 short-tailed shrew, *Blarina brevicauda*. *Ohio Journal of Science* 76:25-26.
- Birks, J.D.S., and N. Dunstone. 1985. Sex-related differences in the diet of the mink, *Mustela vison. Holarctic Ecology* 8:245-252.
- Birks, J.D., and I.J. Linn. 1982. Studies of home range of the feral mink, *Mustela vison. Symp. Zool. Soc. Lond.* 49: 231-257.
- 11 Bjerke, D.L. and R.E. Peterson. 1994. Reproductive toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin
- 12 in male rats: different effects of in utero versus lactational exposure. *Toxicol. Appl. Pharmacol.*
- 13 127:241-249.
- Bjerke, D.L., R.J. Sommer, R.W. Moore, and R.E. Peterson. 1994. Effects of in utero and lactational 2,3,7,8-tetrachlorodibenzo-p-dioxin exposure on responsiveness of the male rat
- reproductive system to testosterone stimulation in adulthood. *Toxicol. Appl. Pharmacol.* 127:250-257.
- 18 Bleavins, M.R., R.J. Auerlich, and R.K. Ringer. 1980. Polychlorinated biphenyls (Aroclors 1016
- and 1242): Effects on survival and reproduction in mink and ferrets. Archives of Environmental
 Contamination and Toxicology 9:627-635.
- Borga, K., G.W. Gabrielsen, and J.U. Skaare. 2001. Biomagnification of organochlorines along a
 Barents Sea food chain. *Environ. Pollut.* 113(2):187-198.
- 23 Brunstrom, B., H. Hakansson, and K. Lundberg. 1991. Effects of a technical PCB preparation and
- 24 fractions thereof on ethoxyresorufin O-deethylase activity, vitamin A levels and thyn development
- 25 in the mink (*Mustela vison*). Annual Review of Pharmacology and Toxicology 69:421-26.
- Buechner, H.K and F.B. Golley. 1967. Preliminary estimation of energy flow in Uganda kob. In:
 Petrusiewicz, L. ed. *Secondary Productivity of Terrestrial Ecosystems*. Warszawa-Krakow; pp. 243-254.
- Burgess, S.A. and J.R. Bider. 1980. Effects of stream habitat improvements on invertebrates,
 trout populations, and mink activity. *Journal of Wildlife Management* 44(4):871-880.
- 31 Bursian, S.J., R.J. Aulerich, B. Yamini, and D.E. Tillitt. 2002. Dietary Exposure of Mink to Fish
- 32 from the Housatonic River: Effects on Reproduction and Survival. Michigan State University,
- 33 Department of Animal Science. Final Draft Report. March 28, 2002.
- 34 Bursian, S.J., and B. Yamini. 2003. Dietary Exposure of Mink to Fish from the Housatonic River.
- Inducement of Manidibular and Maxillary Squamos Cell Profileration. Report submitted to Weston
 Solutions, Inc. March 18, 2003.

- 1 Burt, W.H., and R.P. Grossenheider. 1976. A Field Guide to the Mammals. Field Marks of all
- 2 North American Species Found North of Mexico. Sponsored by the National Wildlife Federation
- 3 and National Audubon Society. Houghton Mifflin Company, Boston, MA. 290 pp.
- Choate, J.R., J.K. Jones, Jr., and C. Jones. 1994. *Handbook of Mammals of the South-Central States*. Louisiana State University Press, Baton Rouge, LA. 304 pp.
- 6 Corbet, R.L., D.G. Muir, and G.R.B. Webster. 1983. Fate of carbon-14 labeled 1,3,6,8-7 tetrachloro-dibenzo-p-dioxin in an outdoor aquatic system. *Chemosphere* 12:523-528.
- 8 Cowan, W.F. and J.R. Reilly. 1973. Summer and fall foods of mink on the J. Clark Salyer
 9 National Wildlife Refuge. *Prairie Naturalist* 5:20-24.
- 10 Eagle, T.C. and J.S. Whitman. 1987. Mink. In Wild Furbearer Management and Conservation,
- 11 M. Novak, J.A. Baker, M.E. Obbarel, et al. eds. University of Pittsburgh Press, Pittsburgh, PA.
- 12 pp. 615-624.
- 13 EPA (U.S. Environmental Protection Agency). 1993. Wildlife Exposure Factors Handbook.
- 14 Volume 1 of 2. EPA/600/R-93/187a. U.S. Environmental Protection Agency, Office of Research
- 15 and Development, Washington, DC.
- Frame, G. M. 2001. The current state-of-the-art of comprehensive, quantitative, congenerspecific PCB analysis, and what we now know about the distributions of individual congeners in commercial Aroclor mixtures. In *PCBs: Recent Advances in Environmental Toxicology and Health Effects* L. W. Pobertson and L. G. Hanson, Eds
- 19 *Health Effects*, L. W. Robertson and L.G. Hansen, Eds.
- French, Jr., J.B., M.B. Voltura, and T.E. Tomasi. 2001. Effects of pre- and postnatal
 polychlorinated biphenyl exposure on metabolic rate and thyroid hormones of white-footed mice.
 Environmental Toxicology and Chemistry 20:1704-1708.
- Gerell, R. 1970. Home ranges and movements of the mink *Mustela vison* Schreber in southern
 Sweden. *Oikos* 21(2):160-173.
- Giavini, E., M. Prati, and C. Vismara. 1983. Embryonic effects of 2,3,7,8-tetrachlorodibenzo-pdioxin administered to female rats before mating. *Environ. Res.* 31:105-110.
- Gilbert, F. F. and E.G. Nancekivell. 1982. Food habits of mink (*Mustela vison*) and otter (*Lutra canadensis*) in northeastern Alberta. *Canadian Journal of Zoology* 60:1282-1288.
- Gray, L.E. Jr. and J.S. Ostby. 1995. In utero 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) alters
 reproductive morphology and function in female rat offspring. *Toxicol. Appl. Pharmacol.* 133:285 294.
- Gray, L.E. Jr., W.R. Kelce, E. Monosson, J.S. Ostby, and L.S. Birnbaum. 1995. Exposure to TCDD during development permanently alters reproductive function in male Long Evans rats and
- 34 hamsters: Reduced ejaculated and epididymal sperm numbers and sex accessory weights in
- 35 offspring with normal androgenic status. *Toxicol. Appl. Pharmacol.* 131:108-118.

- 1 Green, S., F.M. Sauro, and L. Friedman. 1975. Lack of dominant lethality in rats treated with 2 polychlorinated biphenyls (Aroclors 1242 and 1254). *Fd. Cosmet. Toxicol.* 13:507-510.
- Greer, K.R. 1956. Fur Resources and Investigations: Study of the Otter Food Habits Along a
 Segment of the Gallatin River. Montana Fish and Game Department, Job Comp. Rep. W-049-R06:35-59.
- 6 Greer, K.R. 1955. Yearly food habits of the river otter in the Thompson Lakes region, 7 northwestern Montana, as indicated by scat analysis. *American Midland Naturalist* 54:299-313.
- 8 Grodzinski, W., and B.A. Wunder. 1975. Ecological Energetics of Small Mammals. In Small
- 9 Mammals: Their Productivity and Population Dynamics. F.B. Golley, K. Petrusewicz, and L.
- 10 Ryszkowksi, Editors. Cambridge University Press, Cambridge, MA.
- Haffner, G.D., M. Tomczak, and R. Lazar. 1994. Organic contaminant exposure in the Lake St.
 Clair food web. *Hydrobiologia* 281:19-27.
- Hamilton, W.J. Jr. 1940. The summer food of minks and raccoons on the Montezuma Marsh, New
 York. *Journal of Wildlife Management* 4:80-84.
- Hamilton, W.J. Jr. 1959. Foods of mink in New York. New York Fish and Game Journal6(1):77-85.
- Hamilton, W.J. Jr. 1961. Later fall, winter and early spring food of 141 otters from New York, NY. *Fish and Game Journal* 8:106-109.
- 19 Heaton, S.N., S.J. Bursian, J.P. Giesy, D.E. Tillitt, J.A. Render, P.D. Jones, D.A. Verbrugge, T.J.
- Kubiak, and R.J. Aulerich. 1995. Dietary exposure of mink to carp from Saginaw Bay,
 Michigan. 1. Effects on reproduction and survival, and the potential risks to wild mink
 populations. *Archives of Environmental Contamination and Toxicology* 28:334-343.
- Hochstein, J.R., R.J. Aulerich, and S.J. Bursian. 1988. Acute toxicity of 2,3,7,8tetrachlorodibenzo-p-dioxin to mink. *Archives of Environmental Contamination and Toxicology*17:33-37.
- 26 Hochstein, J.R., S.J. Bursian, and R.J. Aulerich. 1998. Effects of dietary exposure to 2,3,7,8-
- tetrachlorodibenzo-p-dioxin in adult female mink (*Mustela vison*). Archives of Environmental
 Contamination and Toxicology 35:348-353.
- Hochstein, J.R., J.A. Render, S.J. Bursian, and R.J. Aulerich. 2001. Chronic toxicity of dietary 2,3,7,8-tetrachlorodibenzo-p-dioxin to mink. *Veterinary and Human Toxicology* 43:134-39.
- 31 Hornshaw, T.C., R.J. Aulerich, and H.E. Johnson. 1983. Feeding Great Lakes fish to mink:
- 32 Effects on mink and accumulation and elimination of PCBs by mink. Journal of Toxicology and
- 33 Environmental Health 11:933-946.

- Hornshaw, T.C., J. Safronoff, R.K. Ringer, and R.J. Aulerich. 1986. LC₅₀ test results in
 polychlorinated biphenyl-fed mink: Age, season, and diet comparisons. *Archives of Environmental Contamination and Toxicology* 15:717-23.
- Howard, P.H., R.S. Boethling, W.F. Jarvis, W.M. Meylan, and E.M. Michalenko. 1991.
 Handbook of Environmental Degradation Rates. Lewis Publishers, Chelsea, MI.
- 6 Huuskonen, H., M. Unkila, R. Pohjanvirta, and J. Tuomisto. 1994. Developmental toxicity of 7 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the most TCDD-resistant and –susceptible rat 8 strains. *Toxicol. Appl. Pharmacol.* 124:174-180.
- Jensen, S., J.E. Kihlstrom, M. Olsson, C. Lundberg, and J. Orberg. 1977. Effects of PCB and
 DDT on mink (*Mustela vison*) during the reproductive season. *Ambio* 6:239.
- Jorgensen, S.E., S.N. Nielsen, and L.A. Jorgensen. 1991. *Handbook of Ecological Parameters and Ecotoxicology*. Elsevier Science Publishers, Amsterdam, The Netherlands.
- Khera, K.S. and J.A. Ruddick. 1973. Polychlorodibenzo-p-dioxins: Perinatal effects and the
 dominant lethal test in Wistar rats. *Toxicol*. 120:70-84.
- 15 Kihlstrom, J.E., M. Olsson, S. Jensen, A. Johansson, and A. Bergman. 1992. Effects of PCB and 16 different fractions of PCB on the reproduction of the mink (*Mustela vison*). *Ambio* 21:563-69.
- Kimbrough, R.D., R.E. Linder, and T.B. Gaines. 1972. Morphological changes in livers of rats
 fed polychlorinated biphenyls. *Archives of Environmental Health* 25:354-364.
- Koplin, J.R., Collopy, M.W., and A.R. Bammann. 1980. Energetics of two wintering raptors.
 Auk 97 : 795-806.
- Kuehl, D.W., P.M. Cook, A.R. Batterman, and B.C. Butterworth. 1987. Isomer dependent
 bioavailability of polychlorinated dibenzo-p-dioxins and dibenzofurans from municipal
 incinerator fly ash to carp. *Chemosphere* 16:657-666.
- Lagler, K.F., and B.T. Ostensen. 1942. Early spring food of the otter in Michigan. *Journal of Wildlife Management* 6:244-254.
- Lamprey, H.F. 1964. Estimation of the large mammal densities, biomass, and energy exchange in the Tarangire Game Reserve and the Masai Steppe in Tanganyika. *E. Afr. Wild. J.* 2: 1-46.
- 28 Lariviere, S. 1999. Mustela vison. Mammalian Species 608:1-9.
- 29 Liers, E.E. 1951. Notes on the river otter (Lutra canadensis). Journal of Mammalogy 32:1-9.
- 30 Linn, I.J., and J.D. Birks. 1981. Observations on the home ranges of feral American mink
- 31 (Mustela vison) in Devon, England, as revealed by radio-tracking. In Proceedings Worldwide
- 32 Furbearer Conference: Vol. 1. J.A. Chapman, and J.A. Pursley, Editors. Frostbury, MD. pp.
- 33 1088-1102.Lowery, G.H. 1974. The Mammals of Louisiana and Its Adjacent Waters. Louisiana
- 34 State University Press, Baton Rouge, LA. 565 pp.

- 1 Mably, T.A., R.W. Moore, and R.E. Peterson. 1992. In utero and lactational exposure of male
- 2 rats to 2,3,7,8-tetrachlorodibenzo-p-dioxin. 1. Effects on androgenic status. *Toxicol. Appl.*
- 3 Pharmacol. 144:97-107.
- Melquist, W.E., and M.G. Hornocker. 1983. Ecology of river otters in west central Idaho. *Wildlife Monograph* 83. 60 pp.
- Melquist, W.E., J.S. Whitman, and M.G. Hornocker. 1981. Resource partitioning and coexistence of
 sympatric mink and river otter populations. In *Worldwide Furbearer Conference Proceedings*, Vol.
 I. J.A. Chapman and D. Pursley, Editors. Frostberg, MD.
- 9 Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S. 10 Petron, B. Potocki, S. Svirsky, and P. Tyler. 1996. Special report of the Massachusetts Weight-
- of-Evidence Workgroup: A weight-of-evidence approach for evaluating ecological risks. *Human*
- 12 and Ecological Risk Assessment 2:277-304.
- Minnich, J.E. 1982. The Use of Water. In *Biology of the Reptilia*. Vol. 12. C. Gans, F.H. Pough,
 Editors. Academic Press, New York, NY.
- Mitchell, J.L. 1961. Mink movements and populations on a Montana river. *Journal of Wildlife Management* 25:48-54.
- 17 Moore, D.R.J., B.E. Sample, G.W. Suter, B.R. Parkhurst, and R.S. Teed. 1999. A probabilistic 18 risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East
- 19 Fork Poplar Creek, Oak Ridge, Tennessee, USA. Environmental Toxicology and Chemistry
- 20 18:2941-2953.
- 21 Muir, D.C.G., R.J. Nostrom, and M. Simon. 1988. Organochlorine contaminants in Arctic

22 marine food chains: Accumulation of specific polychlorinated biphenyls and chlordane-related

- 23 compounds. *Environmental Science and Technology* 22:1071-1079.
- 24 Murray, F.J., F.A. Smith, K.D. Nitschke, C.G. Humiston, R.J. Kociba, and B.A. Schwetz. 1979.
- 25 Three-generation reproduction study of rats given 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)
- 26 in the diet. *Toxicology and Applied Pharmacology* 50:241-252.
- Nagy K.A. 1987. Field metabolic rate and food requirement scaling in mammals and birds.
 Ecological Monographs 57:111-128.
- Nagy, K.A., I.A. Girard, and T.K. Brown. 1999. Energetics of free-ranging mammals, reptiles,
 and birds. *Annual Reviews in Nutrition* 19:247-77.
- Nisbet, I.C.T., and M.B. Paxton. 1982. Statistical aspects of three-generation studies of the reproductive toxicity of TCDD and 2,4,5-T. *Journal American Statistical Association* 36:290-298.
- 34 NRCC (National Research Council of Canada). 1981. Polychlorinated dibenzo-p-dioxins:
- Criteria for their effects on man and his environment. Publication NRCC No. 18574. National
 Research Council of Canada, Ottawa, Ontario. 251 p.

- Palmer, E.L. and H.S. Fowler. 1975. *Fieldbook of Natural History*. McGraw-Hill Book
 Company, New York, NY.
- Platanow, N.S., and L.H. Karstad. 1973. Dietary effects of polychlorinated biphenyls on mink.
 Canadian Journal of Comparative Medicine 37:391-400.
- 5 Pohjanvirta, R., M. Unkila, and J. Tuomisto. 1993. Comparative acute lethality of 2,3,7,8-6 tetrachlorodibenzo-p-dioxin (TCDD), 1,2,3,7,8-pentachlorodibenzo-p-dioxin and 1,2,3,4,7,8-7 hexachlorodibenzo-p-dioxin in the most TCDD susceptible and the most TCDD-resistant rat 8 strain. *Pharmacol. and Toxicol.* 73:52-56.
- Proulx, G., J.A. McDonnell, and F.F. Gilbert. 1987. The effect of water level fluctuations on
 muskrat, *Ondatra zibethicus*, predation by mink, *Mustela vison. Canadian Field-Naturalist*101(1):89-92.
- 12 Restum, J.C., S.J. Bursian, J.P. Giesy, J.A. Render, W.G. Helferich, E.B. Shipp, D.A. Verbrugge,
- 13 and R.J. Aulerich. 1998. Multigenerational study of the effects of consumption of PCB-
- contaminated carp from Saginaw Bay, Lake Huron, on mink. 1. Effects on mink reproduction,
 kit growth and survival, and selected biological parameters. *Journal of Toxicology and*
- 16 Environmental Health 54:343-375.
- Ringer, R.K., R.J. Aulerich, and M. Zabik. 1972. Effects of dietary polychlorinated biphenyls on
 growth and reproduction in mink. *Proceedings of the 164th National Meeting, American Chemical*
- 19 *Society* 12:149-154.
- Safe, S.H. 1994. Polychlorinated biphenyls (PCBs): Environmental impact, biochemical and toxic
 responses, and implications risk assessment. *Critical Reviews in Toxicology* 24:87-149.
- Senthilkumar, K., N. Iseki, S. Hayama, J. Nakanishi, and S. Masunaga. 2001. Polychlorinated
 dibenzo-p-dioxins, dibenzofurans, and dioxin-like polychlorinated biphenyls in livers of birds
 from Japan. *Arch. Environ. Contam. Toxicol.* 42:244-255.
- Sheldon, W.G., and W.G. Toll. 1964. Feeding habits of the river otter in a reservoir in central
 Massachusetts. *Journal of Mammalogy* 45:449-455.
- Stahl, B.U., A. Kettrup, and K. Rozman. 1992. Comparative toxicity of four chlorinated dibenzo-pdioxins (CDDs) and their mixture. *Arch. Toxicol.* 66:471-477.
- Thayer, G.W., W.E. Schaaf, J.W. Angelovic, and M.W. LaCroix. 1973. Caloric measurements of
 some estuarine organisms. *Fishery Bulletin* 71:289-296.
- 31 Tillitt, D. E., S. R. Birke, and D. K. Nicks. 2003. Hepatic O-dealkylase activities from induction
- 32 of cytochrome P-450 in mink and their offspring fed diets containing contaminated fish from the
- 33 Housatonic River. USGS, Columbia Environmental Research Center, Biochemistry and
- 34 Physiology Branch, Final Laboratory Report FY 2003 30 4, 2003. 16 p.

- 1 Ushinohama, K., D-S. Son, K.F. Roby, K.K. Rozman, and P.F. Terranova. 2001. Impaired 2 ovulation by 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) in immature rats treated with equine 3 chorionic gonadotropin. *Reproductive Toxicology* 15:275-280.
- 4 Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy,
- 5 A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X.R. van Leeuwen, A.K.D.
- 6 Liem, C. Nolt, R.E. Petersen, L. Poellinger, S. Safe, D. Scenk, D. Tillitt, M. Tysklind, M.
- 7 Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs) for PCBs,
- 8 PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives* 106:775-792.
- 9 Whitaker, J.O., Jr., and W.J. Hamilton Jr. 1998. *Mammals of the Eastern United States*. 3rd edition, Cornell University Press, Ithaca, NY, USA.
- 11 Wren, C.D., D.B. Hunter, J.F. Leatherland, and P.M. Stokes. 1987b. The effects of
- 12 polychlorinated biphenyls and methylmercury, singly and in combination on mink. II.
- 13 Reproduction and kit development. Archives of Environmental Contamination and Toxicology
- 14 16:449-454.

110.ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND2REPRODUCTION OF OMNIVOROUS AND CARNIVOROUS3MAMMALS

4 **Highlights** 5 **Conceptual Model** 6 The assessment endpoint is the survival, growth, and reproduction of omnivorous 7 and carnivorous mammals in the Housatonic River PSA. Common omnivorous and 8 carnivorous mammals, including red fox and northern short-tailed shrew, are 9 exposed to tPCBs and TEQ via trophic transfer. These two species were selected as 10 representative species for the ecological risk assessment (ERA). 11 Exposure 12 Exposure of the representative species to tPCBs and TEQ was determined from 13 concentrations found in prey items and an estimation of the daily intake of these 14 contaminants of concern (COCs) from consumption of prev. 15 Effects 16 No data were available on toxicity of tPCBs and TEQ to red fox and northern short-17 tailed shrew. Surrogate species were used to estimate effects to these species. 18 Sufficient surrogate data were available to generate dose-response curves for each 19 species. 20 Risk 21 Modeled exposure and effects for red fox and short-tailed shrew suggest that they are at an intermediate risk as a result of exposure to tPCBs and TEQ in the 22 23 Housatonic PSA. Other omnivorous and carnivorous mammal species common to 24 the PSA are expected to have either higher levels of risk (e.g., smoky shrew), similar 25 levels of risk (e.g., masked shrew, gray fox), and in one case (covote), a lower level of risk compared to the representative species. 26

27

28 10.1 INTRODUCTION

29 The purpose of this section is to characterize and quantify the current and potential risks posed to 30 omnivorous and carnivorous mammals exposed to contaminants of potential concern (COPCs) in the Housatonic River and floodplain, focusing on total PCBs (tPCBs) and other COPCs 31 32 originating from the General Electric Company (GE) facility in Pittsfield, MA. The watershed is 33 located in western Massachusetts and Connecticut, discharging to Long Island Sound, with the 34 GE facility located near the headwaters of the watershed. The Primary Study Area (PSA) 35 includes the river and 10-year floodplain from the confluence of the East and West Branches of 36 the Housatonic River downstream of the GE facility, to Woods Pond Dam (Figure 1.1-2).

A Pre-ERA was conducted to narrow the scope of the ecological risk assessment by identifying 1 2 contaminants, other than tPCBs, that pose potential risks to aquatic biota and wildlife in the PSA 3 (Appendix B). A three-tiered deterministic approach was used to screen COPCs. The 4 deterministic assessments compared potential conservative estimates of exposure with 5 conservative adverse effects benchmarks to identify which contaminants are of potential concern 6 to omnivorous and carnivorous mammals in the Housatonic River. A hazard quotient (total daily 7 intake/effect benchmark) greater than 1 in the Housatonic River area resulted in the COPC being 8 screened through to the next tier assessment, and to the probabilistic ecological risk assessment, 9 if necessary. In the COPC screening specific to this endpoint, several other COPCs (primarily 10 organochlorine pesticides) were screened out because their actual concentrations in the PSA 11 were likely much lower than the measured values due to laboratory interference (see Section 12 2.4). In summary, the COPCs that screened through to the probabilistic risk assessment for 13 omnivorous and carnivorous mammals were the contaminants of concern (COCs), tPCBs and 14 2,3,7,8-TCDD TEQ (TEQ). Total PCBs detected in Housatonic River media samples closely 15 resemble the commercial PCB mixtures Aroclor 1260 and Aroclor 1254, which are similar in 16 congener makeup. TEQ is calculated from coplanar PCB and dioxin and furan congeners using 17 the toxic equivalency factor (TEF) approach developed by Van den Berg et al. (1998)(see 18 Section 6.4).

A step-wise approach was used to assess the risks of tPCBs and TEQ to omnivorous and
 carnivorous mammals in the Housatonic River watershed. The four main steps in this process
 include the following:

- 1. Derivation of a conceptual model (Figure 10.1-1).
- 23
- 2. Assessment of exposure of mammals to COCs (Figure 10.1-2).
- 24
- 3. Assessment of the effects of COCs on mammals (Figure 10.1-3).
- 25 26
- 4. Characterization of risks to the omnivorous and carnivorous mammalian species (Figure 10.1-4).

- 1 This section is organized as follows:
- 2 Section 10.2 presents the conceptual model for assessing the ecological risk to 3 omnivorous and carnivorous mammals. 4 Section 10.3 describes the exposure model, input variables, and uncertainty propagation techniques. Also presented in this section are the exposure modeling 5 results for red fox and northern short-tailed shrew. 6 7 Section 10.4 describes the effects to mammals exposed to tPCBs and TEQ and 8 derives the effects metrics 9 Section 10.5 describes the lines of evidence, followed by a discussion of the sources 10 of uncertainty in this assessment, and the conclusions regarding risks of tPCBs and 11 TEQ to omnivorous and carnivorous mammals in the Housatonic River PSA. 12 13 This section provides a summary of the ERA for omnivorous and carnivorous mammals, 14 which is presented in detail in Appendix J. 15



1 2

3

4

Figure 10.1-1 Conceptual Model Diagram: Exposure Pathways for Omnivorous and Carnivorous Mammals Exposed to Contaminants of Concern (COCs) in the Housatonic River PSA



Contaminants of Concern (COCs) in the Housatonic River PSA







20 the Housatonic River PSA
1 10.2 CONCEPTUAL MODEL

2 The conceptual model presented in Figure 10.1-1 illustrates the exposure pathways for 3 omnivorous and carnivorous mammals exposed to tPCBs and TEQ in the PSA. Total PCBs and 4 TEQ are persistent, hydrophobic, and lipophilic. Therefore, they are bioaccumulated by aquatic 5 and terrestrial biota directly through the consumption of contaminated prey as part of the food 6 chain (Haffner et al. 1994; Senthilkumar et al. 2001; Borga et al. 2001). Small mammals, 7 earthworms and other invertebrates, and plants comprise the major dietary items for omnivorous 8 Carnivorous mammals primarily feed on mammals, although fruits, birds, and mammals. 9 invertebrates can supplement their diet. In summary, omnivorous and carnivorous mammals that 10 reside, or partially reside, within the study area are exposed to tPCBs and TEQ principally 11 through diet as a result of trophic transfer. Other routes of exposure, considered to be less 12 important to overall exposure, include inhalation, water consumption, and soil/sediment 13 ingestion (Moore et al. 1999).

The problem formulation (see Section 2) identified the red fox (*Vulpes vulpes*) (Figure 10.2-1) as the representative species for carnivorous mammals potentially exposed to tPCBs and TEQ from consumption of contaminated prey. The northern short-tailed shrew (*Blarina brevicauda*) (Figure 10.2-2) was selected as the representative species for omnivorous mammals. Life history profiles for the red fox and short-tailed shrew are provided in the text boxes. Additional life history information on these species is presented in Section J.2.1.

20 The assessment endpoint is the survival, growth, and reproduction of omnivorous and 21 carnivorous mammals in the Housatonic River PSA. The measurement endpoints used to 22 evaluate the assessment endpoint include: (1) determining, by comparisons to doses reported in 23 the literature to cause adverse effects, the extent to which the concentrations of tPCBs and TEQ 24 ingested in the diet will cause adverse effects to the survival, growth, or reproduction of omnivorous and carnivorous mammals, and (2) determining, by conducting field surveys, the 25 26 relationship between the concentrations of tPCBs and TEQ and survival, reproduction, and 27 relative abundance of omnivorous and carnivorous mammals in the Housatonic River floodplain. 28 As part of the EPA field survey, placental scars in small mammals were analyzed as an 29 indication of past reproductive performance.





Life History of Red Fox

The red fox is a common dog-sized canine that occurs in many habitats throughout its range and is the most widely distributed carnivore in the world. In North America, the red fox is found throughout the United States and Canada, but not in the southeast coastal region, extreme southwest, parts of the central states, or the Pacific coastal regions. The typical pelage color of fox is red and it can be identified by its characteristic bushy, white-tipped tail, pointed muzzle, and prominent ears.

Habitat - Occupies a variety of habitats, but preferred habitat is a matrix of forest, cropland, and pastureland, habitats common in the PSA. The availability of suitable prey as well as suitable den sites is also important. Prefer to locate dens in forested areas, but within a short distance of open areas and usually within 100 meters of a source of open water.

Home Range - Maintains territory throughout the year and is considered nonmigratory. Average home range for adults in Maine was 14.7 km² (range = 6.0-27.5 km²), average home range in Ontario was 9 km² with a range of 5 to 20 km². Mean territory sizes reported in EPA (1993) ranged from 100 to 2,000 hectares (1 to 20 km²). Adults traverse most of their territory on a routine basis, but focus activities around dens, preferred hunting areas, food supplies, and resting areas.

Dietary Habit - Diet varies throughout the year depending on food availability. Includes almost all available animals as prey, such as insects, fish, reptiles, amphibians, birds, small mammals, and carrion. Although typically identified as carnivores, can consume considerable amounts of plant materials, particularly in the summer and fall. Plant material in the diet includes berries, apples, and nuts.

1

2

3

4 5

6

7

8

9

10

11

12

13

14

15

16

17

18 19

20

21

22

23

24

25



Figure 10.2-2 Northern Short-Tailed Shrew (*Blarina brevicauda*)

Life History of Short-Tailed Shrew

The northern short-tailed shrew is a small energetic mouse-like animal with dark slate-colored pelage found throughout the northcentral and northeastern United States extending into southern Canada. It is easily identified as a shrew by its long pointed snout, small black eyes, concealed ears, and five toes on each foot. The northern short-tailed shrew has a short tail, which is approximately 20% of total animal length.

Habitat - Can be found in a variety of habitats, including wetlands and uplands, and are common in areas with abundant vegetative cover, occur in both forested and open habitats.

Home Range - Home range of 0.06 acre (0.024 ha) in central New York State. Other estimates of home range size vary from 0.25 to 0.5 acres (0.1 to 0.2 ha) in areas of low prey density in winter months during nonbreeding periods to 0.07 to 0.17 acres (0.03 to 0.07 ha) in areas of high prey density with a minimum of territory overlap. Do not migrate seasonally, remaining in home range.

Dietary Habits - Earthworms and insects comprise most of the diet, earthworms reported to be the most important item in the diet. Invertebrates in the diet are mainly obtained from the leaf litter layer, and consist of millipedes, insect larvae, spiders, slugs, snails, and other mollusks. Plant materials, including nuts, berries, roots, and fungi, and occasional small mammals are also a component of the diet.

1 10.3 EXPOSURE ASSESSMENT

2 This exposure assessment for omnivorous and carnivorous mammals focuses on the PSA. The 3 representative species for omnivorous and carnivorous mammals are the northern short-tailed 4 shrew and the red fox. These mammals occur in the PSA and feed on prey exposed directly to 5 tPCBs and TEQ and through trophic transfer. Trophic transfer and exposure through ingestion 6 of contaminated prey are the major exposure pathways for omnivorous and carnivorous 7 mammals exposed to tPCBs and TEQ. Other routes of exposure, considered to be negligible 8 contributors to overall exposure, include inhalation, water consumption, and soil/sediment 9 ingestion (Moore et al. 1999). Total PCBs and TEQ tend to bioaccumulate in the food chain 10 because of the following:

- Total PCBs and TEQ are persistent, and hydrophobic and highly lipophilic
 substances.
- When released to aquatic systems, the majority of these compounds form associations with dissolved and/or particulate matter in the water column and remain in sediment layers; biodegradation is considered to be a relatively minor fate process in water (NRCC 1981; Howard et al. 1991).
- Aquatic sediment provides a sink for these compounds and may represent long-term sources to the aquatic food web (Kuehl et al. 1987; Muir 1988; Corbet et al. 1983; Tsushimoto et al. 1982). Both of these COCs are bioaccumulated by aquatic and terrestrial biota directly through the consumption of contaminated prey as part of the food chain (Haffner et al. 1994; Senthilkumar et al. 2001; Borga et al. 2001).

Foxes were observed throughout the PSA from 1998 to 2001 (Appendix A). The exposure analysis was carried out for all of Reach 5 of the PSA because the foraging range of red fox is fairly large. Because short-tailed shrews have a much smaller foraging range, the exposure analysis was performed for three locations in the PSA (Locations 13, 14, and 15, Figure J.2-3) that represent the range of COC concentrations found in the PSA.

27

Description of Sampling Locations 13, 14, and 15

Location 13 is a relatively flat area on the west shore of the river, in the floodplain adjacent to river mile
 133.2, situated at an elevation of 965 ft (294 m). The community type is transitional floodplain forest that
 is flooded seasonally and is moderately well drained, with extensive vegetation cover (80%) and alluvial
 silt-loam soil. PCB concentrations in floodplain soil averaged 55.2 mg/kg dw.

Location 14 is a relatively flat low-lying area on the west shore of the river, in the floodplain adjacent to
river mile 129.9, situated at an elevation of 965 ft (294 m). The community type is transitional floodplain
forest that is flooded seasonally, with extensive vegetation cover (70%) and fluvial silt soil. PCB
concentrations in floodplain soil averaged 26.1 mg/kg dw.

Location 15 is a flat area on the west shore of the river, in the floodplain adjacent to river mile 126.7,
 situated at an elevation of 965 ft (294 m). Community types are circumneutral hardwood swamp and
 transitional floodplain forest that are flooded seasonally. This site has 60% vegetation cover, 40% leaf
 litter cover, and a primarily mineral soil. PCB concentrations in floodplain soil averaged 0.484 mg/kg dw.

14 This section begins with a description of the exposure model used for both of the representative

15 species. Subsequent sections describe the inputs used in the exposure analyses for each

16 representative species. The section concludes with a presentation of the results of the exposure

17 analyses.

18

1 10.3.1 Exposure Model

Exposure of the representative species, red fox and northern short-tailed shrew, to tPCBs and
TEQ was estimated using a total daily intake model adapted from the *Wildlife Exposure Factors Handbook* (EPA 1993) and related publications. The model used in the exposure analysis was:

$$TDI = FT \cdot FIR \sum_{i=1}^{n} C_i \cdot P_i$$
 (Eq. 1)

6 where

5

7	TDI	=	total daily intake	(mg/kg bw/d	tPCBs, ng/kg bw/d T	EQ)
---	-----	---	--------------------	-------------	---------------------	-----

8 FIR = normalized food intake rate (kg/kg bw/d)

9 FT = foraging time in PSA (unitless)

10
$$C_i$$
 = concentration in *i*th food item (mg/kg tPCBs, ng/kg TEQ)

11 P_i = proportion of the *i*th food item in the diet (unitless)

12 The models consider the food intake rates of the representative species (FIR), the concentrations 13 of COCs in each food item (C_i) , and the proportion of the diet accounted for by that food item 14 (P_i) . For those input variables that are uncertain, variable, or both, distributions are used rather 15 than point estimates. Monte Carlo and probability bounds analyses are the methods used to 16 propagate uncertainties about input variables through the exposure model for each COC. A 17 description of these techniques and the methods used to parameterize input variables is presented 18 in Section 6.5 and Appendix C.4. The results of the Monte Carlo analysis are used to estimate 19 the probability of exposure exceeding an effects threshold or doses that cause adverse effects of 20 The probability bounds analysis is conducted to determine how differing magnitudes. 21 uncertainty regarding the distributions of the input variables influences the estimated exposure 22 distribution. The results of these analyses are discussed in detail in Appendix J.

23 Two issues often arise when calculating a TEQ concentration in prey:

- 24 25
- Congener concentrations may be below the detection limit (DL) (i.e., non-detects).
- Some congeners may not be resolved due to co-elution during analysis.

An approach was developed to address these issues and is presented in Section 6.4 and Appendix
 C.2. Briefly, congeners detected at or below the DL were included in the TEQ calculations by
 investigating three options:

- 4 5
- First, setting the concentration for the congener equal to zero (0),
- Setting it to half the DL, and,
- 6 7
- Finally, setting it equal to the DL.

8 A comparison of the results of this bounding analysis provides a description of the uncertainty 9 surrounding the TEQ value due to concentrations of one or more congeners being below the 10 detection limit.

To resolve the co-elution issue, the concentration of congeners that co-eluted with other congeners was assumed equal to the total concentration of the co-elutes (overestimate of TEQ concentration) or zero (underestimate of TEQ concentration). The decision criteria in Section 6.4 were followed to deal with the uncertainty arising from co-elution or non-detection of congeners when estimating exposure point concentrations for use in the exposure analyses.

16 Input distributions to the exposure analyses were generally assigned as follows:

- Lognormal distributions for variables that were right skewed with a lower bound of zero and no upper bound (e.g., amount of COC transferred from mother to offspring via egg tissue for tree swallows).
- Beta distributions for variables bounded by zero and one (e.g., proportion of a prey item in the diet).
- Normal distributions for variables that were symmetric and not bounded by one (e.g., body weight).
- 24
- Point estimates for minor variables or variables with low coefficients of variation.

In certain situations (e.g., poor fit of data), other distributions were fit to the data or other approaches were used. To quantify uncertainty, two approaches were used as described in Section 6.5.2 and Appendix C.4, Monte Carlo analyses and Probability Bounds analyses. The distributions used in the exposure analyses for red fox and northern short-tailed shrews are shown in Figures 10.3-1 and 10.3-2. A brief description of these variables is provided below. A discussion of the concentrations of COCs in prey follows.



- 3







Figure 10.3-1 Input Distributions for the Exposure Modeling of the Red Fox





1 10.3.1.1 Body Weight (BW)

As with many mammalian species, the red fox exhibits sexual dimorphism in body size. Males are typically 10% larger and 20% to 30% heavier than females (Storm et al. 1976; Lariviere and Pasitschniak-Arts 1996; Voigt 1987). In a study conducted in Indiana, males weighed an average of 4.9 kg and females weighed an average of 4.0 kg (Lariviere and Pasitschniak-Arts 1996). Voigt (1987) found that male red fox in Ontario averaged 4.1 kg in weight (n = 37) and females averaged 3.4 kg in weight (n = 37).

8 The northern short-tailed shrew can weigh over 22 grams (George et al. 1986; Burt and 9 Grossenheider 1980, as cited in EPA 1993). Whitaker and Hamilton (1998) reported a mean 10 body weight for adult males and females of 19.3 g. As part of the ecological characterization of 11 the PSA (Appendix A), 58 adult short-tailed shrews of both sexes were caught during small 12 mammal trapping in 1998 to 2001. The body weights ranged from 15 to 27 g (mean = 21.9 g). 13 The average weight of adult female shrews was 22.3 g (SD = 2.87 g).

14 10.3.1.2 Food Intake Rate (FIR)

The food intake rate of red fox and northern short-tailed shrew were measured in laboratory and captive animals (Sargeant 1978; Barrett and Stuek 1976; Morrison et al. 1957). Because the animals were captive or kept in a laboratory, the measured food intake rates likely underestimated food intake rates of free-living fox and shrew (EPA 1993). Free-living fox and shrew, unlike captive fox and shrew, expend energy foraging for prey, avoiding predators, defending territories, etc. As a result, an allometric modeling approach, described below, was used to estimate food intake rate for red fox and short-tailed shrew.

Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating the free
metabolic rate (FMR) of free-living mammals in kilojoules (kJ) per day using the following
general equation:

$$FMR \ (kJ/d) = a \cdot BW(g)^{b}$$
(Eq. 2)

The slope (*a*) and power (*b*) distributions were based on the error statistics derived from regression analysis of the data reported in Nagy et al. (1999). For red fox, the carnivore equation 1 was used and had a mean slope term *log a* equal to 1.67 and a standard error of 2.65 in log_{10} 2 units. The power term (*b*) had a reported mean of 0.869 and a standard error of 0.116 (Nagy et 3 al. 1999). For short-tailed shrew, the insectivore equation was used. The slope term *log a* had a 4 reported mean of 6.98 and a standard error of 1.32 in log_{10} units, and the power term (*b*) had a 5 reported mean of 0.622 and a standard error of 0.0630 (Nagy et al. 1999). The body weight 6 (*BW*) distribution was described above. The results of the calculation were then converted to 7 kcal/kg bw/d.

8 Food intake rate (FIR) is derived from *FMR* using the following equation:

$$FIR(kg/kgbw/d) = \frac{FMR}{\sum_{i=l}^{n} AE_i \cdot GE_i}$$
(Eq. 3)

where AE_i is the assimilation efficiency of *i*th food item (unitless) and GE_i is the gross energy of *i*th food item (kcal/kg).

12 The gross energies of various wildlife food sources are summarized in the *Wildlife Exposure* 13 Factors Handbook (EPA 1993). The gross energy of earthworms ranges from 780 to 830 14 kcal/kg (mean = 805; SD = 141) (Cummins and Wuycheck 1971; Thayer et al. 1973). The mean 15 gross energy for grasshoppers and crickets is 1,700 kcal/kg (SD = 260) (Cummins and 16 Wuycheck 1971; Collopy 1975; Bell 1990), and for adult beetles, the mean is 1,500 kcal/kg 17 (Cummins and Wuycheck 1971; Collopy 1975; Bell 1990). Grasshoppers, crickets, and beetles 18 were used as representatives of litter invertebrates; their mean gross energy is 1,600 kcal/kg. 19 Mammals have a gross energy of 1,700 kcal/kg (SD = 280) (Koplin et al. 1980).

The assimilation efficiency for mammals consumed by mammals is 84% (SD = 6.5%) (Castro et al. 1989). The assimilation efficiency of earthworms consumed by mammals is not known. The mean assimilation efficiency for insects consumed by small mammals is 87% (Bryant and Bryant 1988). This value was used to represent the assimilation efficiency for earthworms consumed by mammals. Point estimates were used for these variables in the Monte Carlo and probability bounds analyses because of their relatively small coefficients of variation (i.e., CV < 10%). As a result, these input variables are not included in Figures 10.3-1 and 10.3-2.

1 10.3.1.3 Proportions of Dietary Items (P_i)

2 The red fox can occupy a variety of habitats in the PSA and may use a variety of food sources. 3 Available studies reporting the dietary composition of the red fox in North America show that 4 the proportion of dietary items varies according to season (Table J.2-1). Most studies found that 5 mammals constitute the majority of the diet of the red fox, with the percentage in the diet as high 6 as 92% in the spring (Knable 1974). For this assessment, mammals represent approximately 7 76% of the average diet for all seasons (Figure 10.3-1). However, the distributions used in the 8 exposure analyses for mammals were sufficiently wide to incorporate the range of variation 9 reported in the literature (Appendix J). Other food items including birds, invertebrates, and 10 vegetation were not included as part of the exposure model because the dietary items represent a 11 relatively small portion of the diet (e.g., birds and invertebrates) or the contribution to overall 12 exposure is negligible (e.g., vegetation).

13 As with the red fox, there is variation in the proportion of dietary items reported for the short-14 tailed shrew (Table J.2-9). Earthworms comprised between 5% and 31% of the diet of short-15 tailed shrew, whereas insects and small mammals were reported as high as 61% and 24% of the 16 diet, respectively (Hamilton 1941, as cited in EPA 1993; Linzey and Linzey 1973; Eadie 1944). 17 Averaging the available data for the winter and summer diets of the short-tailed shrew indicates 18 that earthworms, litter invertebrates (all combined), and small mammals comprise the major 19 dietary items for the short-tailed shrew. For this exposure assessment, the diet of the shrew was 20 on average 19% for earthworms, 60% for litter invertebrates, and 12% for small mammals 21 (Figure 10.3-2). However, the distributions used in the exposure analyses for each dietary 22 component were sufficiently wide to incorporate the range of variation reported in the literature 23 (Appendix J).

24 10.3.1.4 Foraging Time (FT)

The red fox visits all parts of its territory on a regular basis (Ables 1974). The home range used in this assessment was 9 km² (Voigt and Tinline 1980) and the width of the floodplain for Reach 5 ranges from 200 to 600 m. Therefore, the red fox is expected to spatially and temporally average exposure inside and outside the PSA within its home range, potentially experiencing areas of high contamination along with areas of low or no contamination. As a result, the PSA represents only a portion of the home range of red fox. It was estimated that red fox spend up to
 50% of their time foraging in the PSA (see Appendix J.2).

The foraging range for northern short-tailed shrew is small. Whitaker and Hamilton (1998) and Degraaf and Yamasaki (2001) found that the northern short-tailed shrew had a home range size of 0.06 acre (0.024 ha), while Platt (1976) reported that the home range size varies from 0.25 to 0.5 acres (0.1 to 0.2 ha) in areas of low prey density. The sizes of Locations 13, 14, and 15 are approximately 2 to 3 acres. Therefore, shrews are expected to have 100% of their foraging range within each of Locations 13, 14, and 15 in the PSA.

9 10.3.1.5 Concentrations of COCs in Prey

Mammals such as white-footed mouse and short-tailed shrew are the major dietary items for red fox. The median concentration of tPCBs in mammals measured in Reach 5 is 4.98 mg/kg. The 25th and 75th percentiles are 1.78 and 29.3 mg/kg, respectively. The median, 25th and 75th percentile concentrations of TEQ are 290, 179, and 1,107 ng/kg, respectively.

14 The diet for northern short-tailed shrew includes earthworms, litter invertebrates, and mammals. 15 Similar statistics for concentrations of tPCBs and TEQ in these prey at Locations 13, 14, and 15 16 are presented in Figures 10.3-3 and 10.3-4, respectively. TEQ concentration in earthworms was 17 measured in one composite sample of 20 to 45 earthworms at each location. Data on 18 concentrations of TEQ in litter invertebrate prev were not available to estimate exposure to short-19 tailed shrew. In this case, the concentrations of TEQ in prey were extrapolated using measured 20 concentrations in earthworms. Concentrations of tPCBs and TEQ in all prev items were highest 21 at Locations 13 and 14. At these locations, earthworms had the highest concentrations of tPCBs, 22 whereas concentrations of TEQ were highest in mammals.



Figure 10.3-3 Concentrations of tPCBs in Prey of Northern Short-Tailed Shrew (n=1 for invertebrates and earthworms)

6





Note: Error bars indicate interquartile range.

5 Figure 10.3-4 Concentrations of TEQ in Prey of Northern Short-Tailed Shrew (n=1 6 for invertebrates and earthworms)

7

8 The input variables for concentrations of COCs in prey of red fox and short-tailed shrew are 9 shown in Tables J.2-12 and J.2-13.

10 10.3.2 **Results of Exposure Assessments**

Figures 10.3-5 to 10.3-12 present exposure distributions for red fox and short-tailed shrew to 11 12 tPCBs and TEQ.

13 Figure 10.3-5 depicts the cumulative distribution of tPCB intake rates for red fox in Reach 5.

14 The Monte Carlo analysis indicated that exposure of red fox to tPCBs could range from a

15 minimum of 0.0220 to a maximum of 82.5 mg/kg bw/d. The mean exposure was 6.25 mg/kg

16 bw/d and the median exposure 2.68 mg/kg bw/d. Ninety percent of the exposure estimates were

17 between 0.321 and 25.0 mg/kg bw/d.



Reach 5

1 Notes: LPB = Lower Probability Bound

2 . UPB = Upper Probability Bound

Figure 10.3-5 Exceedance Probability Distribution for Red Fox Exposed to tPCBs in Reach 5 of the PSA



Reach 5



2 . UPB = Upper Probability Bound

Figure 10.3-6 Exceedance Probability Distribution for Red Fox Exposed to TEQ in Reach 5 of the PSA



- 1 Notes: LPB = Lower Probability Bound
- 2 . UPB = Upper Probability Bound

Figure 10.3-7 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to tPCBs at Location 13 of the PSA





Figure 10.3-9 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to tPCBs at Location 15 of the PSA





3 . UPB = Upper Probability Bound

Figure 10.3-10 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to TEQ at Location 13 of the PSA





3 . UPB = Upper Probability Bound

Figure 10.3-11 Exceedance Probability Distribution for Short-Tailed Shrew Exposed to TEQ at Location 14 of the PSA



The probability bounds estimated for red fox foraging in Reach 5 are depicted in Figure 10.3-5. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 0.106 and 0.702 mg/kg bw/d. The 50th percentile ranged between 0.607 and 3.54 mg/kg bw/d, and the 90th percentile ranged between 3.52 and 23.5 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 0.434, the 50th percentile was 2.68, and the 90th percentile was 16.7 mg/kg bw/d (Table J.2-6).

Short-tailed shrew living at Locations 13 and 14 had the highest exposure to tPCBs. Red fox
foraging in Reach 5 had slightly less exposure to tPCBs than shrews at Locations 13 and 14.
Red fox in Reach 5 and short-tailed shrew at Locations 13 and 14 had the highest exposures to
TEQ. For both tPCBs and TEQ, short-tailed shrew foraging at Location 15 had the lowest
exposure.

1 10.4 EFFECTS ASSESSMENT

2 The objective of the effects assessment is to review the scientific literature and derive the most 3 appropriate effects metrics for effects of tPCBs and TEQ to omnivorous and carnivorous 4 mammals. An effects metric can be represented by a dose-response relationship or a daily dose 5 for a COC that represents a threshold beyond which toxic effects may appear in omnivorous and 6 carnivorous mammals. The effects metrics will be used, in conjunction with the exposure 7 assessment, to estimate risks to omnivorous and carnivorous mammals exposed to tPCBs and 8 TEQ in the Housatonic River PSA. This section focuses on effects that have an influence on the 9 long-term maintenance of mammal populations (i.e., mortality, or impairment of reproduction or 10 growth). Studies involving multiple exposure treatments that employed statistical evaluations to 11 determine whether treatment results were different from controls are preferred. Studies that 12 document effects of tPCBs and TEQ on the representative species, red fox and northern short-13 tailed shrew, were preferred but unavailable. As a result, laboratory studies involving surrogate 14 species were used to estimate effects to the representative species. For short-tailed shrew and red 15 fox, the rat was used as a surrogate species for effects due to exposure to tPCBs. In the case of 16 exposure to TEQ, the mouse was used as a surrogate species for short-tailed shrew, while the rat 17 was used for red fox.

Exposure of mammals to tPCBs and 2,3,7,8-TCDD (TEQ) causes a range of effects (see text box). The congeners that comprise the TEQ group have the ability to bind to the aryl hydrocarbon receptor protein (Bosveld and van den Berg 1994) and elicit an Ah-mediated biochemical and toxic response. A discussion of the chemical features that elicit the toxic response and the mode of action are shown below.

23	Effects of tPCBs and TEQ on Mammals			
24	Types of effects to mammals from exposure to tPCBs and TEQ include:			
25 26 27 28 29 30	 Hormone induction Decreases in body and organ weight Reduced fertility Reduced litter size Reduced survival at birth or weaning Mortality 			

1	
2	Mode of Action of TEQ Congeners
3 4 5 6 7 8 9 10 11 12	Congeners that comprise the TEQ group have the ability to bind with the Ah receptor and elicit similar toxic responses. The most toxic congeners tend to be those that have a planar shape and are chlorinated in the 2,3,7, and 8 positions for dioxins and furans, and in the meta and para positions for PCBs. This structural configuration best fits the receptor and leads to a common mechanism of action in many animal species involving binding to the aryl hydrocarbon (Ah) receptor and elicitation of an Ah receptor-mediated biochemical and toxic response. The toxic response of this group of chemicals is, therefore, related to the three-dimensional structure of the substance, including the degree of chlorination and positions of the chlorine on the aromatic frame.
13 14 15 16 17 18	Planar chlorinated hydrocarbons are found in the environment as a mixture of congeners. The congeners can have different toxic potencies. To address this issue and effectively estimate the relative toxicity of these mixtures, various systems have been created involving the development and use of toxic equivalency factors (TEFs) to derive toxic equivalence (TEQ). The approach used for this assessment is described in Section 6.4.

Presented below is a brief review of the scientific literature on the effects of dietary tPCBs and TEQ to mammals. The discussion focuses on ecologically relevant effects endpoints such as survival, growth, and reproduction. A summary of reproductive effects for tPCBs and TEQ is presented in Figures J.3-1 and J.3-2 and Tables J.3-2 and J.3-3.

24 **10.4.1** Review of Effects of tPCBs and TEQ

25 10.4.1.1 tPCBs—Mortality

Linder et al. (1974) studied the effects of Aroclor 1254 and 1260 on 3- to 4-week-old Shermanstrain male rats. Oral LD₅₀s were 1,295 and 1,315 mg/kg bw/d, respectively (Linder et al. 1974). Under similar test conditions, groups of 10 female Sherman-strain rats were treated with Aroclor 1260 doses of 7.2, 38.2, and 72.4 mg/kg bw/d for 8 months (Kimbrough et al. 1972). During this time period, one, two, and eight females died, respectively. Bruckner et al. (1973) estimated a 14-day LD₅₀ of 4,250 mg/kg bw/d for Aroclor 1242 for rats.

1 10.4.1.2 tPCBs—Reproduction

Many of the available studies focus on determining effects to offspring of mammals following in utero and/or lactational exposure to tPCBs. Impaired reproductive performance as a result of maternal PCB exposure has been reported for many mammals, including rats and mice. In general, females are administered contaminants by gavage or in the diet prior to or during gestation. Endpoints studied included pre- and post-natal survival and development, fertility, and other effects (Linder et al. 1974; Brezner et al. 1984; Overmann et al. 1987; Linzey 1987, 1988; Masuda et al. 1979; Allen and Barsotti 1976; Bleavins et al. 1981).

9 A two-generation reproduction study was performed in which groups of 20 female and 10 male 10 Sherman rats were exposed to diets of Aroclor 1254 at doses of 0.06, 0.32, 1.5, and 7.6 mg/kg 11 bw/d (Linder et al. 1974). Exposure to Aroclor 1254 caused significantly reduced litter sizes at 12 the 1.5 and 7.6 mg/kg bw/d doses. Survival to weaning was reduced by 77.8% in the second 13 Spencer (1982) investigated reproductive effects of Aroclor 1254 using eight generation. 14 treatment concentrations on Sprague Dawley strain rats fed treated diets on days 6 through 15 of 15 gestation. Statistically significant reductions were observed in fetal weight at birth (11.8%; p<0.05) and fetal survival (28%; p<0.05) for rats fed diets of Aroclor 1254 at 7.47 and 17.1 16 mg/kg bw/d, respectively. Reproductive impairment in white-footed mice was also observed in 17 several studies (Linzey 1987; McCoy et al. 1995; Merson and Kirkpatrick 1976). Effects 18 19 included longer intervals between births, smaller litter sizes at birth, smaller litter sizes at weaning, reduced mean birth and weaning weight in offspring, and reduced litter production rate. 20

21 10.4.1.3 TEQ—Mortality

22 The 2,3,7,8-tetrachlorodibenzo-p-dioxin acute lethality of (TCDD), 1,2,3,7,8-23 pentachlorodibenzo-p-dioxin (PCDD), and 1,2,3,4,7,8-hexachlorodibenzo-p-dioxin (HCDD) was 24 investigated for Long-Evans (LE) rats and Han/Wistar (H/W) (Pohjanvirta et al. 1993). The 25 H/W rats were approximately 1,000-fold more resistant to TEQ than LE rats. For example, the LD₅₀ values for exposure of female and male LE rats were 9,800 and 17,700 ng/kg bw/d TEQ, 26 27 respectively, whereas LD₅₀s for H/W female rats were greater than 7,200,000 ng/kg bw/d TEQ.

In long-term exposure studies, 100% mortality occurred at 57.1 ng/kg bw/d TEQ when Sprague Dawley rats were continuously exposed to TCDD for 31 weeks. Kociba et al. (1978) conducted a 2-year study by feeding diets containing TCDD at 1, 10, and 100 ng/kg bw/d TEQ to male and female Sprague Dawley rats. At 100 ng/kg bw/d TEQ, they observed a cumulative increase in mortality in the latter half of the study period and a decrease in mean body weight from 6 to 24 months compared to controls.

7 10.4.1.4 TEQ—Reproduction

Among the 209 possible PCB congeners, the non-ortho-substituted (planar) congeners are the most toxic due to their structural similarity to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). The most toxic of these congeners include 3,3',4,4'-tetrachlorobiphenyl (PCB-77), 3,3',4,4',5,5'hexachlorobiphenyl (PCB-169), and 3,3',4,4',5-pentachlorobiphenyl (PCB-126) (Safe 1984).

12 The prenatal toxicity of PCB-77 was determined in rats and mice fed contaminated diets between 13 days 6 and 18 of gestation (Marks et al. 1989; d'Argy et al. 1987; Wardell et al. 1982; Rands et 14 al. 1982a; 1982b). Marks et al. (1989) reported a PCB-77 dose-related increase in the percentage 15 of implants that resorbed, at concentrations ranging from 400 (7% increase) to 6,400 (82.5% 16 increase) ng/kg bw/d TEQ; a significant increase (16.4%) was determined at 1,600 ng/kg bw/d 17 TEQ and above. In addition, the average number of live fetuses per female mouse was 18 significantly reduced (21.5%) at 1,600 ng/kg bw/d TEQ and above (Marks et al. 1989). Khera 19 and Ruddick (1973) treated pregnant Wistar rats with eleven doses of TCDD on gestation days 6 20 to 15. A dose-related decrease in live fetuses was observed; 100% embryonic lethality was 21 reported when animals were exposed to a dose of 4,000 ng/kg bw/d TEQ or higher. Similar 22 observations were made by Sparschu et al. (1971) in Sprague Dawley rats fed six doses of 23 TCDD on days 6 through 15 of gestation. The number of viable fetuses decreased and the total 24 number of resorptions increased starting at 125 ng/kg bw/d TEQ. Giavini et al. (1983) and 25 Huuskonen et al. (1994) also observed a reduction in the number of living fetuses per litter when 26 rats were fed TCDD.

In similar studies on rats and mice, other TEQ effects observed included significant increases in
mortality of offspring, resorptions, mortality of embryos and of offspring, reduced fecundity,
reduced litter size, reduced body weight at birth, reduced in vitro fertilizing ability of the eggs,

and reduced survival to weaning (Rands et al. 1982a; Linzey 1987; d'Argy et al. 1987; Wardell
 et al. 1982; Huang et al. 1998; Neubert and Dillman 1972; Murray et al. 1979; Bjerke and
 Peterson 1994; Mably et al. 1992; Bjerke et al. 1994; Flaws et al. 1997; Gray and Ostby 1995;
 Thomas and Hinsdill 1979; Khera and Ruddick 1973).

5 **10.4.2 Effects Metrics for Characterizing Risk**

Effects data can be characterized and summarized in a variety of ways ranging from benchmarks
designed to be protective of most or all species to concentration- or dose-response curves. A
summary of the decision criteria used to derive effects metrics is provided in the text box.
Further details on the decision criteria used in selecting effects metrics is provided in Section 6.6
of the ERA.

2	Decision Criteria for Derivation of Effects Metric	
3 4	The following is the hierarchy of decision criteria used to characterize effects for eac receptor-COC combination:	
5	 Have single-study bioassays with five or more treatments been conducted on the	
6	receptor of interest or a reasonable surrogate? If yes, estimate the	
7	concentration- or dose-response. If not, go to 2.	
8	 Are multiple bioassays with similar protocols, exposure scenarios and effects	
9	metrics available that, when combined, have five or more treatments for the	
10	receptor of interest or a reasonable surrogate? If yes, estimate the dose-	
11	response relationship as in 1. If not, go to 3.	
12	 Have bioassays with less than five treatments been conducted on the receptor of	
13	interest or a reasonable surrogate? If yes, conduct or report results of	
14	hypothesis testing to determine the NOAEL and LOAEL. If not, go to 4.	
15	4. Are sufficient data available from field studies and monitoring programs to	
16	estimate concentrations or doses of the COC that are consistently protective or	
17	associated with adverse effects? If yes, develop field-based effects metrics. If	
18	not, go to 5.	
19 20 21 22 23 24 25	5. Derive a range where the threshold for the receptor of interest is expected to occur. Because information on the sensitivity of the receptor of interest is lacking, it is difficult to derive a threshold that is neither biased high or low. If, however, bioassay data are available for several other species, calculate a threshold for each to determine a threshold range that spans sensitive and tolerant species. That range is likely to include the threshold for the receptor of interest.	

In this ERA, data were available to derive dose-response curves using surrogate mammals for the
 representative species.

28 **10.4.2.1** Effects of tPCBs to Red Fox and Short-Tailed Shrew

The Spencer (1982) study was used for the derivation of a dose-response curve based on mortality at birth. Figure 10.4-1 presents the dose-response curve for mortality of rats at birth. The dose-response curve indicates that 10% and 20% declines in mortality at birth would be expected at doses of 3.05 and 5.37 mg/kg bw/d, respectively.



Figure 10.4-1 Dose-Response Curve for Effects of tPCBs on Mortality at Birth of Rats

6 10.4.2.2 Effects of TEQ to Red Fox

1 2

3 4

5

The Khera and Ruddick (1973) and Sparschu et al. (1971) studies were combined, because of the similarity of the protocols, for the derivation of a dose-response curve based on reproductive effects. Figure 10.4-2 presents the dose-response curve for reproductive fecundity of rats exposed to TEQ. The dose-response curve indicates that 10% and 20% declines in reproductive fecundity would be expected at doses of 156 and 330 ng/kg bw/d TEQ, respectively.



2 Note: Symbols indicate raw data.

Figure 10.4-2 Dose-Response Curve for Effects of TEQ on Reproductive Fecundity of Rat

5 10.4.2.3 Effects of TEQ to Short-Tailed Shrew

6 The Marks et al. (1989) study was used for the derivation of a dose-response curve based on 7 reproductive effects. Figure 10.4-3 presents the dose-response curve for reproductive fecundity 8 of mice exposed to TEQ. The dose-response curve indicates that 10% and 20% declines in 9 reproductive fecundity would be expected at doses of 570 and 1207 ng/kg bw/d TEQ, 10 respectively.



Note: Symbols indicate raw data

Figure 10.4-3 Dose Response Curve for Effects of TEQ on Reproductive Fecundity of Mouse

1 10.5 RISK CHARACTERIZATION

This section characterizes risk to omnivorous and carnivorous mammals exposed to tPCBs and TEQ in the PSA of the Housatonic River. The risk characterization uses two and three lines of evidence to determine potential ecological risks to red fox and short-tailed shrew, respectively. The major lines of evidence are considered to be independent and will be combined in a weightof-evidence assessment. The key risk questions and the lines of evidence are summarized in the text box.

8	Key Risk Questions			
9 10 11	•	Are the concentrations of tPCBs and TEQ present in the prey of omnivorous and carnivorous mammals sufficient to cause adverse effects to individuals inhabiting the PSA of the Housatonic River?		
12	•	If so, how severe are the risks and what are their potential consequences?		
13		Lines of Evidence		
14	-	Use of semiquantitative biological field surveys.		
15	-	Probabilistic exposure and effects modeling.		
16		Population demography field study for short-tailed shrew.		

17

18 Section 10.5.1 presents a brief overview of the methodology, results, and interpretation of the 19 mammal surveys conducted from 1998 to 2001 in the Housatonic PSA. A more detailed 20 presentation of this information is presented in Appendix A. In Section 10.5.2, the dose-21 response curves are combined with the corresponding exposure distributions to derive risk curves 22 that characterize the relationship between probability and magnitude of effect. A brief overview 23 of the population demographics field study is described in Section 10.5.3. A weight-of-evidence 24 assessment is presented is Section 10.5.4 along with sources of uncertainty (Section 10.5.5), 25 extrapolation of risk to other species (10.5.6) and the overall findings of the risk characterization 26 (Section 10.5.6).

27 10.5.1 Field Surveys (Performed by EPA)

The mammalian community in the PSA was studied by EPA over a 4-year period, from 1998 to 2001. Surveys were conducted to record presence, relative abundance, and habitat usage for small and large mammals including short-tailed shrew and red fox. A variety of field survey
 techniques including small mammal trapping, snow tracking, and scent-post station surveys were
 used to characterize the mammalian community.

4 Forty-two mammal species were documented in the PSA during the 4 years of field surveys (see 5 Appendix A for more details). Many species were observed throughout the PSA in a variety of 6 habitats. Forested communities, such as red maple swamp, black ash-red maple-tamarack, 7 transitional floodplain forest, and high-terrace floodplain forest supported the greatest number of 8 species. Observations of omnivorous and carnivorous mammals including covotes, red fox, 9 raccoons, white-footed mice, short-tailed shrews, and little brown bats were common, all of 10 which were observed in forested and nonforested habitats as well as riverine, shoreline, wetland, 11 upland, and residential habitats. Other carnivorous mammals observed in the PSA included 12 bobcats, fishers, and long-tailed weasels. Omnivorous mammals were one of the most abundant groups of mammals observed in the PSA. Common omnivores included white-footed mice, 13 14 raccoons, striped skunks, Virginia opossums, and black bears. The short-tailed shrew was the 15 most abundant insectivorous mammal observed in the PSA. The semiguantitative nature of the 16 field surveys and lack of reference locations in the surveys make it difficult to develop 17 relationships between abundance of representative species and concentrations of COCs.

18 During the small mammal surveys, females were checked for evidence of lactation; some 19 individuals were euthanized and the uterus was removed for placental scar analysis. Number of 20 placental scars has been used in a variety of mammals to estimate litter sizes (Hensel et al. 1969; 21 Sanderson 1950; Oleyar and McGinnes 1974; Nixon et al. 1975). Placental scar identifications 22 of small female mammals were performed on four species including short-tailed shrew, white-23 footed mouse, meadow jumping mouse, and masked shrew at Locations 13, 14, and 15. Sample 24 sizes were small for each species. In some cases, placental scars were difficult to identify, 25 particularly for white-footed mouse. Although the data have uncertainties due to these 26 limitations, it appeared that there were no differences in numbers of placental scars between 27 locations (Table J.4-1). The white-footed mouse was the most frequently captured species with 28 sample sizes ranging from 6 to 11 females amongst the locations. The average number of placental scars per white-footed mouse female was 6.33, 6.27, and 6.50 at Locations 13, 14, and 29 30 15, respectively. Mean soil tPCB concentrations vary over 50-fold among these three locations.

The number of females for the other three species combined ranged from two to six, and had a
 lower average number of scars ranging from zero to 2.50 (Table J.4-1).

3 10.5.2 Comparison of Estimated Exposures to Laboratory-Derived Effects 4 Doses

Red fox exposure was assessed for all of Reach 5 and short-tailed shrew exposure was estimated
in three areas (Locations 13, 14, and 15, Figure J.2-3) in the PSA.

For each receptor-COC combination, a category of low, intermediate, or high risk was assigned following integration of the exposure and effects distributions. This exercise was done separately for the results of the Monte Carlo analyses and each of the lower and upper bounds from the probability bounds analyses. The "risk category" refers to the level of risk based on the results of the Monte Carlo analysis. The "risk range" refers to the levels of risk based on the results of the probability bounds analyses. The 10% and 20% effects doses for each species and COC are presented in Section 10.4.

			۹.
14		Guidance for Determining Level of Risk to Representative Species	
15 16		If the probability of 10% or greater effect is less than 20%, then the risk to omnivorous and carnivorous mammals was considered low.	
17 18		If the probability of 20% or greater effect is greater than 50%, then the risk to omnivorous and carnivorous mammals was considered high.	
19		 All other outcomes were considered to have intermediate risk. 	
20 21	The	results of the risk characterization are summarized in Table 10.5-1. Figures 10.	- 5-1

10.5-8 present the risk curves for red fox exposed in Reach 5 and short-tailed shrew exposed at
Locations 13, 14, and 15 to tPCBs and TEQ.

to
Table 10.5-1

Summary of Qualitative Risk Statements for Omnivorous and Carnivorous Mammals from the Housatonic River Study Area

	Qualitative Risk Statements					
	tPCBs			TEQ		
Mammal / Location	Risk Category	Risk Range		Risk Category	Risk Range	
Red Fox						
Reach 5	Intermediate	Low-Intermediate		Intermediate	Low- Intermediate	
Short-Tailed Shrew						
Location 13	High	Intermediate -High		Low	Low-Intermediate	
Location 14	High	Intermediate-High		Low	Low- Low	
Location 15	Low	Low-Intermediate		Low	Low-Low	

5

1 2



Figure 10.5-1 Risk Function for Red Fox Exposed to tPCBs in Reach 5 of the Housatonic River



2 3

Figure 10.5-2 Risk Function for Red Fox Exposed to TEQ in Reach 5 of the Housatonic River





Figure 10.5-3 Risk Function for Short-Tailed Shrew Exposed to tPCBs at Location
 13 of the PSA





Figure 10.5-4 Risk Function for Short-Tailed Shrew Exposed to tPCBs at Location
 14 of the PSA



Site 15

Figure 10.5-5 Risk Function for Short-Tailed Shrew Exposed to tPCBs at Location
 15 of the PSA

4



Figure 10.5-6 Risk Function for Short-Tailed Shrew Exposed to TEQ at Location
 13 of the PSA



Figure 10.5-7 Risk Function for Short-Tailed Shrew Exposed to TEQ at Location
 14 of the PSA



1

3

Figure 10.5-8 Risk Function for Short-Tailed Shrew Exposed to TEQ at Location 15 of the PSA

1 The results of the risk characterization showed that the highest risk to omnivorous and 2 carnivorous mammals is from exposure to tPCBs at Locations 13 and 14. The risk category for 3 short-tailed shrew at Locations 13 and 14 was high, and the risk range, as determined by the 4 probability bounds analysis, ranged from intermediate to high. Risk to shrews at Location 15 5 was low. The risk category for exposure of short-tailed shrew to TEQ at Location 13 is low; the 6 risk range is low to intermediate. Short-tailed shrew exposed to TEQ at Locations 14 and 15 7 have a risk category of low. Both the upper and lower bound of the risk ranges for Locations 14 8 and 15 are low. Red fox had an intermediate risk category for both exposure to tPCBs and TEQ. 9 The risk range for both COCs for red fox is low to intermediate.

10 **10.5.3** Population Demography Field Study (Performed by GE)

11 A population demography field study was performed in 2001 along a 16-km reach of the 12 Housatonic River between Pittsfield and Woods Pond (Boonstra 2002). The study objectives 13 included evaluating population density, survival, rates of reproduction, sex ratio, and growth 14 rates of short-tailed shrew. More information on this study is provided in Boonstra (2002).

15 Six sites were selected based on tPCB concentrations, habitat uniformity, and sufficient area to 16 permit a 1-hectare (ha) trapping grid to be located within each site. All sites were located within 17 the eastern deciduous temperate forest biome in primarily palustrine habitat, with portions of two 18 grids also including upland habitat. Two grid classes were selected, designated as high and low 19 concentrations of tPCBs, with three sites in each class. Habitat varied across the grids, 20 particularly between the northern sites and the southern sites (e.g., the former had much more 21 vegetative biomass than did the latter). Comparison of the northern and southern sites is an 22 indirect way of determining whether habitat quality has an effect on the survival, growth, and 23 reproduction of shrews. The six areas trapped in this study, in fact, varied in habitat quality. 24 Without habitat and microhabitat data at the six trapping sites, it is difficult to determine if 25 differences in habitat explain variation in population parameters among the six sites. Three 26 trapping sessions were conducted in spring, summer, and fall (trapping sessions one, two, and 27 three, respectively) over the course of the study, with each session lasting 3 consecutive days.

Boonstra (2002) suggested that population characteristics of short-tailed shrew living on more
 contaminated tPCB sites in the PSA are not negatively affected compared to those living on less

1 contaminated sites. The results showed that exposure of short-tailed shrew to tPCBs had no 2 apparent effect on population density, sex ratio, reproduction, and growth rate. In general, shrew 3 populations showed high monthly survival. Although there was grid-to-grid variability in survival, this variability could not be explained by differences in tPCB concentrations among the 4 5 grids. The only significant effect was on body mass of males but, in this case, the males living in 6 the highly contaminated sites weighed more, not less, than those living in the low contaminated 7 sites. In summary, variations in tPCB concentrations among the sites resulted in no differences 8 in population demography of short-tailed shrew according to Boonstra (2002).

9 There were several confounding factors in the Boonstra (2002) study, including flooding of the 10 floodplain, which prevented trapping on three of the six grids during the first of the three 11 trapping events. While spring flooding is a natural phenomenon within the Housatonic River 12 PSA, and shrew populations are very likely accustomed to such events, it is difficult to determine 13 the impact of flooding on the study results. Habitat quality varied across the grids. Without 14 identifying habitat and microhabitat data at the six trapping sites, it is difficult to tell if 15 differences in habitat explain variation in population parameters between the six sites. The lack 16 of reproduction rate data in the Boonstra (2002) study creates uncertainty regarding population 17 maintenance. Even with high adult survivorship, without reproduction rate data, it is impossible 18 to know if shrew populations are maintaining themselves through natural production or 19 immigration. The use of body weight to imply reproductive status may not be appropriate 20 because it is insensitive to potential reproductive impairments. These factors limit the strength 21 of conclusions from the study.

22 Comparison of the spatially weighted concentrations of tPCBs in soil from Boonstra (2002) and 23 the spatially weighted mean concentrations of tPCBs in soil derived as part of this ERA (see 24 Appendix J) showed that the Boonstra (2002) estimates in the six grids appear to be in error. 25 Additional analyses were carried out to verify the relationship between shrew survival and 26 concentrations of tPCBs derived as part of this ERA. The results of the analyses indicated a 27 significant relationship between concentrations of tPCBs in soil and survival of shrews from 28 summer to autumn for males, females, and males and females combined. Although the results of 29 the analyses indicated a significant relationship between soil concentrations of tPCBs and shrew 30 survival, the confidence limits indicated that the relationships were not strong. Some of the

"noise" in the relationships may be attributed to the influence of habitat differences among the
grids, small sample sizes, the effects of flooding, the analytical methods used to measure tPCBs,
the selection of the correct soil samples for inclusion in the analyses, and the relatively small
number of treatments.

5 10.5.4 Weight-of-Evidence Analysis

6 A weight-of-evidence analysis was used to combine the two major lines of evidence described in 7 the preceding sections for red fox and short-tailed shrew. The goal of this analysis was to 8 determine whether significant risk is posed to omnivorous and carnivorous mammals in the 9 Housatonic River PSA as a result of exposure to tPCBs and TEQ. The three-phase approach of 10 Menzie et al. (1996) and the Massachusetts Weight-of-Evidence Workgroup was applied for this 11 purpose, in which weight-of-evidence was reflected in the following three characteristics: (a) the 12 weight assigned to each measurement endpoint, (b) the magnitude of response observed in the 13 measurement endpoint, and (c) the concurrence among outcomes of the multiple measurement 14 endpoints.

A discussion of attributes considered in the WOE is provided in Section 2, and the rationales for weighting of measurement endpoints are provided in Appendix J. A summary of the derived weightings is provided in Table 10.5-2. For both tPCBs and TEQ, the field surveys, the population demography field study, and the modeled exposure and effects lines of evidence were given a moderate/high value.

Table 10.5-2 Weighting of Measurement Endpoints for Omnivorous and
Carnivorous Mammals Weight-of-Evidence Evaluation

Attributes	Field Surveys	Population Demography Field Study*	Modeled Exposure and Effects for tPCBs and TEQ
I. Relationship Between Measurement a	and Assess	ment Endpoints	
1. Degree of Association	L	M/H	М
2. Stressor/Response	М	М	M/H
3. Utility of Measure	L/M	M/H	M/H
II. Data Quality			
4. Data Quality	Н	M/H	M/H
III. Study Design			
5. Site Specificity	Н	Н	L/M
6. Sensitivity	М	М	Н
7. Spatial Representativeness	Н	Н	М
8. Temporal Representativeness	M/H	Н	М
9. Quantitative Measure	М	M/H	Н
10. Standard Method	Н	Н	M/H
Overall Endpoint Value	M/H	M/H	M/H

3

1

2

* Field study only for short-tailed shrew.

L = low; M = moderate; H = high

4

5 6 The magnitude of the response in the measurement endpoint is considered together with the 7 measurement endpoint weight in judging the overall weight-of-evidence (Menzie et al. 1996). 8 This requires assessing the strength of evidence that ecological harm has occurred, as well as an 9 indication of the magnitude of response, if present. The weighting values, evidence of harm, and 10 magnitudes of responses were combined in a matrix format and are presented in Tables 10.5-3 11 and 10.5-4. The field surveys indicated that red fox and short-tailed shrew are likely common in 12 the PSA. However, it is not known whether these species would be more abundant in the 13 absence of COCs, or if they are abundant because of immigration from less contaminated areas.

1 The objectives of the population demography field study (Boonstra 2002) were to measure 2 population demography of short-tailed shrews directly, including reproduction, growth, and 3 survival in the PSA. The responses were quantitatively compared with magnitude of exposure. 4 However, confounding factors such as flooding, area (i.e., location within the floodplain), habitat 5 quality, and the use of body weight to imply reproductive status may have had significant effects 6 on population demographics and the results of the field study. Additional analyses of data 7 generated in the population demography field study showed that tPCBs may be having effects on 8 survival of short-tailed shrews. Other demographic parameters, however, do not appear to be 9 affected by tPCB concentrations in soil. The results from the modeled exposure and effects line 10 of evidence suggest that there is a high risk to short-tailed shrew exposed to tPCBs at Locations 11 13 and 14, and a low risk at Location 15. There is an intermediate risk for fox exposed to tPCBs 12 foraging in Reach 5 (Table J.4-8). There is an intermediate risk to red fox exposed to TEQ in the 13 PSA, and low risk to short-tailed shrew exposed to TEQ at Locations 13, 14, and 15 (Table 14 J.4-9).

Table 10.5-3

Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	
Field Surveys	Moderate/High	Undetermined	Low	
Population Demography Field Study	Moderate/High	Undetermined (Shrew)	Intermediate	
Modeled Exposure and Effects	Moderate/High	Yes (Shrew) Undetermined (Red Fox)	High Intermediate	

5

6

7 8

9

Table 10.5-4

Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	
Field Surveys	Moderate/High	Undetermined	Low	
Population Demography Field Study	Moderate/High	Undetermined (Shrew)	Intermediate	
Modeled Exposure and Effects	Moderate/High	No (Shrew) Undetermined (Red Fox)	Low Intermediate	

10

11 A graphical method was used for displaying concurrence among measurement endpoints. Tables 12 10.5-5 and 10.5-6 depict the outcome for omnivorous and carnivorous mammals exposed to 13 tPCBs and TEQ, respectively. The uncertainty concerning the modeled exposure and effects line 14 of evidence for tPCBs and TEQ, particularly because surrogate species were used for estimating 15 effects, could mean that risks of these COCs are being under- or over-estimated for the PSA. 16 The field survey line of evidence, although inconclusive in terms of demonstrating effects and 17 risk, indicated that omnivorous and carnivorous mammals, including short-tailed shrew and red 18 fox, were commonly observed in hardwood forests, palustrine forested areas, and shorelines of 19 the PSA. In addition, according to Boonstra (2002), the population demography field study line

1 of evidence suggests that no effects attributable to tPCBs are occurring to short-tailed shrews. 2 However, the results of the latter study are confounded by factors such as flooding, quality of 3 habitat, and using body weight to imply reproductive status, all of which likely introduced a 4 large amount of uncertainty. In addition, the soil concentration data used in the Boonstra (2002) 5 study appear to be in error. Additional analyses with revised soil concentration data do not 6 support the conclusion in the Boonstra (2002) study that shrew survival in the study grids was 7 not affected by soil tPCB concentration, but indicates that there is a statistically significant 8 relationship between PCB concentrations and survival, although not strong.

9 With the exception of modeled exposure and effects for shrew, the potential for harm to shrew 10 and red fox based on the remaining lines of evidence was undetermined. Based on the modeled 11 exposure and effects assessment for shrew, there is a high potential for adverse effects resulting 12 from tPCB exposure and very limited potential for adverse effects resulting from exposure to 13 TEQ.

5

6

Assessment Endpoint: Su	irvival, grov ammals	wth, and reprod	luction of omni	vorous and carni	vorous	
	Weighting Factors (increasing confidence of weight)					
Harm/Magnitude	Low	Low/ Moderate	Moderate	Moderate/ High	High	
Yes/High				MEE-S		
Yes/Intermediate						
Yes/Low						4
						-I
Undetermined/High						
Undetermined/Intermediate				MEE-F, PDFS		
Undetermined/Low				FS		
No/Low]↓
No/Intermediate						
No/High]

Table 10.5-5

Risk Analysis Summary for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA

7

8 FS = Field surveys

9 MEE-S = Modeled exposure and effects – shrew

10 MEE-F = Modeled exposure and effects – red fox

11 PDFS = Population demography field study for short-tailed shrew only

2 3

4

5 6

0

7

Assessment Endpoint: Survival, growth, and reproduction of omnivorous and carnivorous mammals

► Weighting Factors (increasing confidence of weight) Low/ Moderate/ Low Moderate High Harm/Magnitude Moderate High Yes/High Yes/Intermediate Yes/Low Undetermined/High MEE-F, Undetermined/Intermediate PDFS Undetermined/Low FS No/Low MEE-S No/Intermediate No/High

Table 10.5-6

Risk Analysis Summary for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSA

8

9 FS = Field surveys

10 MEE-S = Modeled exposure and effects – shrew

11 MEE-F = Modeled exposure and effects – red fox

12 PDFS = Population demography field study for short-tailed shrew only

13

14 10.5.5 Sources of Uncertainty

The assessment of risk to omnivorous and carnivorous mammals contains uncertainties. Each source of uncertainty can influence the estimates of risk. Therefore, it is important to describe and, when possible, specify the magnitude and direction of such uncertainties. Some of the major sources of uncertainty associated with the assessment of risks of tPCBs and TEQ to

Т _1 omnivorous and carnivorous mammals are briefly described below. A more complete list is
 presented in Appendix J.

- 3 The Monte Carlo sensitivity analyses suggest that the free metabolic rate (FMR) slope 4 and power terms were generally the most influential variables on predicted total daily 5 intakes of COCs. However, no measurements of free metabolic rate were available 6 for the representative wildlife species. Similarly, measured food intake rates were not 7 available for free-living red fox or northern short-tailed shrew or reasonable surrogate 8 species. Therefore, free metabolic rates were estimated using allometric equations. 9 The use of allometric equations introduces some uncertainty into the exposure 10 estimates because they have model-fitting error, and are based on species different from the representative species used in this assessment. Given the lack of data on 11 12 representative species used in the current assessment, it is difficult to judge the magnitude of the uncertainty introduced by the use of the allometric models. The 13 14 uncertainty due to model-fitting error was propagated in the uncertainty analyses by 15 using distributions as input for the allometric slope and power terms.
- Sample sizes were limited for the analyses of COC concentrations in some prey 16 17 items. Only one composite sample of earthworm (comprising between 20 and 45 worms) and four samples of mammals were available to estimate exposure of shrews 18 19 to TEQ at each location. Similarly, only two or three samples of litter invertebrates 20 were available to estimate concentrations of tPCBs at each of Locations 13, 14, and 21 15. Uncertainty due to sample size was explicitly incorporated in the uncertainty 22 analyses. In the Monte Carlo analysis, sample size uncertainty was addressed by use 23 of the 95 upper confidence limit (UCL) on the mean. Use of the UCL addressed 24 uncertainty, but is biased toward overestimating exposure. In the probability bounds 25 analysis, uncertainty was addressed by specifying concentration variables as a range 26 from the 95% lower confidence limit (LCL) to the UCL. This treatment of 27 uncertainty is unbiased.
- 28 PCB congeners 123 and 157 co-eluted with other congeners (PCB-123 with PCB-29 149; PCB-157 with PCB-173 and PCB-201), leading to uncertainty about TEO concentrations in prey sample. This source of uncertainty was addressed in the 30 31 uncertainty analyses by estimating prey concentrations assuming concentrations of 32 PCB-123 and PCB-157 were equal to zero, and assuming that concentrations of PCB-123 and PCB-157 were equal to the doublet and triplet concentrations, respectively. 33 The resulting TEQ estimates were then compared. If the ratio of the upper to lower 34 35 bound TEQ estimates was less than 1.3, this source of uncertainty was deemed 36 unimportant and disregarded. If the ratio exceeded 1.3, the uncertainty due to co-37 elution was propagated through the uncertainty analyses.
- The largest source of uncertainty in the effects assessment was associated with the lack of toxicity studies involving the representative species. There were no toxicity studies available for red fox and short-tailed shrew exposed to tPCBs or TEQ. As a result, laboratory studies involving surrogate species were used to estimate effects to these species. These extrapolations introduced uncertainty in the effects assessment

because of the variation in sensitivities of mammal species to tPCBs and TEQ. The sensitivity of wildlife to an environmental contaminant may also differ from that of a laboratory or domestic species due to behavioral and ecological parameters, including stress factors (e.g., competition, seasonal changes in temperature or food availability), disease, and exposure to other contaminants.

- For omnivorous and carnivorous mammals, data for two and three major lines of evidence were available for red fox and short-tailed shrew, respectively. For these assessments, feeding studies involving prey and food items from the PSA would have improved the weight-of-evidence assessment. Such studies would have accounted directly for the specific characteristics of the Housatonic River ecosystem, and the toxicity of the PCB mixture found on-site.
- The lack of reproduction rate data in the Boonstra (2002) study creates uncertainty regarding population maintenance. Even with high adult survivorship, without reproduction rate data, it is impossible to know if shrew populations are maintaining themselves through natural production or immigration.
- 16 In the Boonstra (2002) study, the use of body weight to imply reproductive fitness is a limitation because it is insensitive to potential reproductive impairments. 17 For 18 example, 6 of 10 female short-tailed shrews greater than 18 grams in weight trapped 19 by EPA in August 1999 had no evidence of breeding history upon placental scar 20 analysis. Although some of these six could have been young animals that had not 21 bred yet, others could have been animals with reproductive impairments due to various factors, including PCB exposure. Additionally, during the 1999 trapping, of 22 23 those specimens submitted for tissue analysis (8 of the 10 females over 18 grams), 24 five females with no evidence of breeding history had body burdens up to 135 mg/kg 25 (average 74 mg/kg), whereas those with direct evidence of breeding had body 26 burdens up to 93 mg/kg (average 57 mg/kg). This sample size is quite small; 27 however, along with laboratory evidence of PCB-induced reproductive impairments 28 to mammals, it suggests that animal weight alone may not be representative of the 29 reproductive status of individual animals.

30 **10.5.6 Conclusions**

1

2

3

4

5

31 For omnivorous and carnivorous mammals, data from three major lines of evidence were 32 available, including field surveys, a population demography field study of short-tailed shrew, and 33 exposure and effects modeling. The weight-of-evidence analysis indicates an intermediate risk 34 for short-tailed shrews exposed to tPCBs and TEQ in the PSA. This conclusion, however, is 35 uncertain because of the lack of definitive findings as to whether effects are occurring in the field 36 surveys and population demography field study, and the lack of species-specific measures of 37 effects. The WOE also suggests, based on two lines of evidence for red fox, an intermediate risk 38 to fox exposed to tPCBs and TEQ in the PSA. This finding is also uncertain because although

fox were commonly observed during the field surveys, a foraging rate of 50% in Reach 5 was
 used, and species-specific measures of effects were not available.

Field surveys were conducted (in part) to determine which omnivorous and carnivorous mammal species were present in the PSA. The surveys were not designed to provide a quantitative evaluation of the relationship between exposure to COCs and the survival, growth, and reproduction of omnivorous and carnivorous mammals in the PSA. Instead, the surveys determined the presence, relative abundance, size, and reproductive status of omnivorous and carnivorous mammals. Red fox, short-tailed shrew, and other omnivorous and carnivorous mammals were observed frequently in several areas in the PSA.

10 The objectives of the population demography field study were to determine population density, 11 survival, rates of reproduction, sex ratio, and growth rates of short-tailed shrew measured at six 12 sites having different concentrations of tPCBs in the PSA (Boonstra 2002). The Boonstra (2002) 13 results found that variation in tPCB concentrations among the sites resulted in no differences in 14 population demographic parameters of short-tailed shrew. However, confounding factors such 15 as flooding, area (i.e., location within the floodplain), and habitat quality may have had a 16 significant impact on shrew population demography. The lack of reproduction rate data in the 17 Boonstra (2002) study creates uncertainty regarding population maintenance. Even with high 18 adult survivorship, without reproduction rate data it is impossible to know if shrew populations 19 are maintaining themselves through natural production or immigration. In addition, the use of 20 body weight to imply reproductive fitness may not be appropriate because it is insensitive to 21 potential reproductive impairments.

22 In contrast to the findings in the Boonstra (2002) study, the results of the supplemental analyses 23 conducted for this ERA indicated a significant relationship between tPCB spatially weighted and 24 measured concentrations in soil and survival of shrews from summer to autumn for males, 25 females, and males and females combined, although the confidence limits indicate that the 26 relationships are not strong. Some of the "noise" in the relationships may be attributed to factors 27 listed above. The additional analyses do not support the Boonstra (2002) conclusion that "there 28 is no evidence that this variability [in shrew survival] can be explained by differences in tPCB 29 concentrations among the grids." The modeling of exposure and effects line of evidence was

used to determine the level of risk to the representative mammal species, short-tailed shrew, and red fox. The effects characterization developed dose-response curves to describe the potential effects of tPCBs and TEQ to omnivorous and carnivorous mammals. There were no toxicity studies available for red fox and short-tailed shrew. Surrogate species were used to estimate effects with the assumption that representative omnivorous and carnivorous mammal species would experience adverse effects similar to the surrogate species.

7 The dose-response curve for effects of tPCBs to omnivorous and carnivorous mammals indicated 8 that 10% and 20% declines in mortality at birth would be expected at doses of 3.05 and 5.37 9 mg/kg bw/d, respectively. The modeled exposure results indicated that the daily intake of tPCBs 10 by red fox fell within this range while the daily intake of tPCBs by northern short-tailed shrew 11 was greater than 5.37 mg/kg bw/d at Locations 13 and 14. This means that shrews, and possibly 12 red fox in the PSA, are likely to receive tPCB doses that would cause adverse reproductive 13 effects. The daily intakes of short-tailed shrews at Locations 15 were below the 10% effects 14 dose, meaning that shrews are likely not at risk from exposure to TEQ at that site.

15 For TEQ, the dose-response curve for red fox indicated that 10% and 20% declines in 16 reproductive fecundity would be expected at doses of 156 and 330 ng/kg bw/d, respectively. The 17 modeled exposure results indicated that the daily intake of TEQ by red fox fell within this range. 18 It is, therefore, difficult to make definitive conclusions about the risks of TEQ to this species. 19 For northern short-tailed shrews, the dose-response curve indicated that 10% and 20% declines 20 in reproductive fecundity would be expected at doses of 570 and 1207 ng/kg bw/d TEQ, 21 respectively. The daily intakes of short-tailed shrews at Locations 13, 14, and 15 were below the 22 10% effects dose, meaning that shrews are likely not at risk from exposure to TEQ in the PSA.

The field surveys and conclusions made in the Boonstra (2002) study contradict the results from the modeling of exposure and effects line of evidence. However, the results of the supplemental analyses of the data from the Boonstra study (2002) on survival of short-tailed shrews are in agreement with the modeling results, suggesting that there is a high potential for adverse effects from exposure to COCs in the contaminated areas of the PSA.

Population dynamics of mammals are affected by processes such as growth, reproduction, death
of predators, immigration, and emigration. As a result, a number of mechanisms exist to

possibly compensate for the adverse effects of a toxic chemical. For example, a toxic chemical 1 2 may lead to an increase of a mammal population by reducing abundance of competitors or by 3 eliminating predators. Other mechanisms could also compensate for the direct effects of a toxic 4 chemical (e.g., increased immigration from uncontaminated sites). In the Housatonic River PSA, 5 such compensating mechanisms could exist for the local populations of short-tailed shrew, and 6 red fox. Thus, a possible explanation for the lack of concordance between the field survey 7 results, the Boonstra (2002) field study results, the additional analyses on survival of shrew, and 8 the modeling results is that other mechanisms (e.g., reduced competition, elimination of 9 predators) compensated for the direct effects due to tPCBs. No information, however, is 10 available to support or test this supposition.

11 Other omnivorous and carnivorous species common to the area include smoky shrews, masked 12 shrews, coyotes, gray fox, fishers, short-tailed weasels, and long-tailed weasels (see Appendix 13 A). Exposure and sensitivity to COCs are the two factors used to estimate risk to omnivorous 14 and carnivorous mammals. As noted in this ERA, effects studies conducted on short-tailed 15 shrew and red fox are not available. Similarly, effects data are not available for other 16 omnivorous and carnivorous species living in the Housatonic River area. As a result, the same 17 surrogate effects data used to estimate effects to short-tailed shrew and red fox would be used for 18 other omnivorous and carnivorous species. A qualitative analysis was conducted to compare 19 exposure of representative species and other omnivorous and carnivorous mammals to tPCBs 20 and TEQ. The major factors that influence mammalian exposure to tPCBs and TEQ include the 21 following:

22 23

24

25

- Foraging behavior and dietary composition.
- Foraging and home range size.
- Species body weight and other life history characteristics.
- Representative species and other mammal species were compared using these factors. Resultsare provided in the text box.

1	ERA Summary
2 3 4 5	The weight-of-evidence analysis indicates a potential risk for short-tailed shrews exposed to tPCBs and TEQ in the PSA. This conclusion, however, is uncertain because of the uncertainty about whether effects are occurring in two of the lines of evidence (i.e., field survey, population demography field study).
6 7	Risk to carnivorous mammals, such as red fox, exposed to tPCBs and TEQ are undetermined in the PSA.
8 9 10 11 12 13	Other omnivorous and carnivorous mammal species common to the PSA include smoky shrew, masked shrew, coyote, gray fox, fisher, short-tailed weasel, and long- tailed weasel. A qualitative analysis of risk on these species indicates that smoky shrew, short-tailed weasel, and long-tailed weasel have higher levels of risk; masked shrew, gray fox, and fisher have similar levels of risk; and coyote has a lower level of risk compared to the representative species.
14	

1 10.6 REFERENCES

- Ables, E.D. 1974. Ecology of the red fox in North America. In: Fox, M.W. ed. *The Wild Canids*.
 Van Nostrand Reinhold, New York, NY. pp 148-163.
- 4 Allen, J.R. and D.A. Barsotti. 1976. The effects of transplacental and mammary movement of 5 PCBs on infant rhesus monkeys. *Toxicology* 6:331-340.
- 6 Barret, G.W. and K.L. Stueck. 1976. Caloric ingestion rate and assimilation efficiency of the 7 short-tailed shrew, *Blarina brevicauda*. *Ohio J. Sci.* 76:25-26.
- 8 Bell, G.P. 1990. Birds and mammals on an insect diet: A primer on diet composition analysis in 9 relation to ecological energetics. *Studies in Avian Biology* 13:391-415. Cited in EPA 1993.
- 10 Bjerke, D.L. and R.E. Peterson. 1994. Reproductive toxicity of 2,3,7,8-tetrachlorodibenzo-p-
- 11 dioxin in male rats: different effects of in utero versus lactational exposure. *Toxicol. Appl.*
- 12 *Pharmacol.* 127:241-249.
- Bjerke, D.L., R.J. Sommer, R.W. Moore and R.E. Peterson. 1994. Effects of *in utero* and lactational 2,3,7,8-tetrachlorodibenzo-p-dioxin exposure on responsiveness of the male rat reproductive system to testosterone stimulation in adulthood. *Toxicol. Appl. Pharmacol.*
- 16 127:250-257.
- Bleavins, M.R., R.J. Aulerich and R.K. Ringer. 1981. Placental and mammary transfer of
 polychlorinated and polybrominated biphenyl in the mink and ferret. In *Avian and mammalian wildlife toxicology*. D.W. Lamb and E.E. Kenaga, Editors. pp. 121-131. Second conference,
 ASTM STP 757. American Society for Testing and Materials, 1916 Race Street, Philadelphia,
 PA.
- Boonstra, R. 2002. Demography of short-tailed shrew population living on PCB contaminated
 sites. Division of Life Sciences, University of Toronto at Scarborough, Ontario, Canada.
- Borga, K., G.W. Gabrielsen, and J.U. Skaare. 2001. Biomagnification of organochlorines along
 a Barents sea food chain. *Environ. Pollut.* 113(2):187-198.
- Bosveld, A.T.C. and M. van den Berg. 1994. Effects of polychlorinated biphenyls, dibenzo-*p*dioxins and dibenzofurans on fish eating birds. Environmental Reviews 2:147-165.
- Brezner, E., J. Terkel and A.S. Perry. 1984. The effect of Aroclor 1254 (PCB) on the physiology
 of reproduction in the female rat-I. *Comp. Biochem. Physiol.* 77C:65-70.
- 30 Bruckner, J.V., K.L. Khanna, and H.H. Cornish. 1973. Biological responses of the rat to 31 polychlorinated biphenyls. *Toxicol. Appl. Pharmacol.* 24:434-448.
- 32 Bryant, D.M. and V. M. Bryant. 1988. Assimilation efficiency and growth of nestling 33 insectivores. *Arch. Hydrobiol*. 130: 268-274.

- 1 Burt, W.H., and R.P. Grossenheider. 1980. *A field guide to the mammals of North America north* 2 *of Mexico*. Houghton Mifflin Co., Boston, MA.
- 3 Castro, G., N. Stoyan, and J.P. Myers. 1989. Assimilation efficiencies in birds: A function of 4 taxon or food type? *Comp. Biochem. Physiol.* 92:271-278.
- Collopy, M.W. 1975. Behavioral and predatory dynamics of kestrels wintering in the Arcata
 Bottoms [master's thesis]. Humboldt State University, Arcata, CA.
- Conaway, C.H. 1955. Embryo resorption and placental scar formation in the rat. *J. Mammal.*36:516-532.
- 9
- 10 Corbet, R.L., D.G. Muir, and G.R.B. Webster. 1983. Fate of carbon-14 labeled 1,3,6,8-11 tetrachloro-dibenzo-*p*-dioxin in an outdoor aquatic system. Chemosphere 12:523-528.
- 12 Cummins, K.W. and J.C. Wuycheck. 1971. Caloric equivalents for investigations in ecological
- 13 energetics. International Association of Theoretical and Applied Limnology. Stuttgart, West
- 14 Germany, Cited in EPA 1993.
- 15 d'Argy, R., L. Dencker, E. Klasson-Wehler, Å Bergman, P.O. Darnerud, and I. Brandt. 1987.
- 3,3',4,4'-tetrachlorobiphenyl in pregnant mice: Embryotoxicity, teratogenicity and toxic effectson the cultured embryonic thymus. *Pharm. Toxicol.* 61:53-57.
- 18 DeGraaf, R.M., and M. Yamasaki. 2001. *New England Wildlife: Habitat, Natural History, and* 19 *Distribution*. University Press of New England, Hanover, NH.
- DiBello, F., S. Arthur, and W. Krohn. 1990. Food habits of sympatric coyotes, red foxes, and bobcats in Maine. *Canadian Field Naturalist* 104:403-408.
- Eadie, W.R. 1944. The short-tailed shrew and field mouse predation. *Journal of Mammalogy* 25:359-364.
- Eadie, W.R. 1948. Shrew-mouse predation during low mouse abundance. *Journal of Mammalogy* 29:35-37.
- 26 EPA (U.S. Environmental Protection Agency). 1993. Wildlife Exposure Factors Handbook.
- Volumes I and II. Office of Research and Development. Washington, DC. EPA EPA/600/R-93/187a, EPA/600/R-93/187b. (99-0040)
- Flaws, J.A., R.J. Sommer, E.K. Silbergeld. R.E. Peterson, and A. Hirshfield. 1997. In utero and lactational exposure to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) induces genital dysmorphogenesis in the female rat. *Toxicol. Appl. Pharmacol.* 147:351-362.
- 32 French, Jr., J.B., M.B. Voltura, and T.E. Tomasi. 2001. Effects of pre- and postnatal
- 33 polychlorinated biphenyl exposure on metabolic rate and thyroid hormones of white-footed mice.
- 34 Environmental Toxicology and Chemistry 20:1704-1708.

- 1 George, S.B., J.R. Choate, and H.H. Genoways. 1986. *Blarina brevicauda*. p. 1-9. *In* T.E. Lawlor
- 2 (ed.) Mammalian Species Number 261. American Society of Mammalogists, Baltimore, MD.
- Giavini, E., M. Prati, and C. Vismara. 1983. Embryonic effects of 2,3,7,8-tetrachlorodibenzo-pdioxin administered to female rats before mating. *Environ. Res.* 31:105-110.
- Gray, L.E. Jr. and J.S. Ostby. 1995. In utero 2,3,7,8-tetrachrlorodibenzo-p-dioxin (TCDD) alters
 reproductive morphology and function in female rat offspring. *Toxicol. Appl. Pharmacol.*133:285-294.
- Gray, L.E. Jr., W.R. Kelce, E. Monosson, J.S. Ostby and L.S. Birnbaum. 1995. Exposure to
 TCDD during development permanently alters reproductive function in male Long Evans rats
 and hamsters: Reduced ejaculated and epididymal sperm numbers and sex accessory weights in
 offspring with normal androgenic status. *Toxicol. Appl. Pharmacol.* 131:108-118.
- Green, S., F.M. Sauro and L. Friedman. 1975. Lack of dominant lethality in rats treated with polychlorinated biphenyls (Aroclors 1242 and 1254). *Fd. Cosmet. Toxicol.* 13:507-510.
- Haffner, G.D., M. Tomczak, and R. Lazar. 1994. Organic contaminant exposure in the Lake St.
 Clair food web. *Hydrobiologia* 281:19-27.
- Hamilton, W.J., Jr. 1941. The foods of small forest mammals in eastern United States. *Journal of Mammalogy* 22:250-263.
- Harrison, S.J., J.A. Bissonnette and J.A. Sherburne. 1989. Spatial relationships between coyotes
 and red foxes in eastern Maine. *J. Wild. Manag.* 53:181-185.
- Howard, P.H., R.S. Boethling, W.F. Jarvis, W.M. Meylan, and E.M. Michalenko. 1991.
 Handbook of environmental degradation rates. Lewis Publishers. Chelsea, Michigan.
- Hensel, R.J., W.A. Troyer and A.W. Erickson. 1969. Reproduction in the female brown bear. J.
 Wildl. Manage. 33:357-365.
- Huang, Y-W. M.J. Melancon, R.E. Jung, and W.H. Karasov. 1998. Induction of cytochrome
 P450-associated monooxygenases in northern leopard frogs, *Rana pipiens*, by 3,3',4,4',5pentachlorobiphenyl. *Environ. Toxicol. Chem.* 17: 1564-1569.
- Huuskonen, H., M. Unkila, R. Pohjanvirta and J. Tuomisto. 1994. Developmental toxicity of
 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in the most TCDD-resistant and –susceptible rat
 strains. *Toxicol. Appl. Pharmacol.* 124:174-180.
- 30 Keenan, R.J. 1981. Spatial use of home range among red foxes (*Vulpes vulpes*) in south-central
- 31 Ontario. In: Chapman, J.A.; Pursley, D. eds. Worldwide furbearer conference proceedings,
- 32 August 1980, Frostburg, Maryland. pp 1041-1063.
- 33 Khera, K.S. and J.A. Ruddick. 1973. Polychlorodibenzo-p-dioxins: Perinatal effects and the 34 dominant lethal test in Wistar rats. *Toxicol*. 120:70-84.

- Kimbrough, R.D., R.E. Linder and T.B. Gaines. 1972. Morphological changes in livers of rats
 fed polychlorinated biphenyls. *Archives of Environmental Health* 25:354-364.
- Knable, A.E. 1974. Seasonal trends in the utilization of major food groups by the red fox (*vulpes fulva*) in Union County, Illinois. Trans. III. State Acad. Sci. 66:113-115.
- Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, C.E. Wade, D.A. Dittenber, R.P. Kalnins,
 L.E. Frauson, C.N. Park, S.D. Barnard, R.A. Hummel, and C.G. Humiston. 1978. Results of a
- 7 two-year chronic study and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rats.
- 8 Toxicol. Appl. Pharmacol. 46:279-303.
- 9 Kolb, H. H. 1986. Some observations on the home ranges of vixens in the suburbs of Edinburg.
 10 *Journal of Zoology* 210:636-639.
- 11 Koplin, J.R., M.W. Collopy, A.R. Bammann and H. Levenson. 1980. Energetics of two 12 wintering raptors. *Auk* 97:795-806.
- 13 Korschgen, L.J. 1959. Food habits of the red fox in Missouri. J. Wild. Manage. 23:168-176.
- 14 Kuehl, D.W., P.M. Cook, A.R. Batterman, and B.C. Butterworth. 1987. Isomer dependent
- bioavailability of polychlorinated dibenzo-*p*-dioxins and dibenzofurans from municipalincinerator fly ash to carp. Chemosphere 16:657-666.
- Lariviere, S. and M. Pasitschniak-Arts. 1996. *Vulpes vulpes*. Mammalian Species No. 537.
 American Society of Mammalogist. 11pp.
- Linder, R.E., T.B. Gaines, and R. Kimbrough. 1974. The effect of polychlorinated biphenyls onrat reproduction. *Fd. Cosmet. Toxicol.* 12:63-77.
- Linzey, A.V. 1987. Effects of chronic polychlorinated biphenyls exposure on reproductive
 success of white-footed mice (*Peromyscus leucopus*). Arch. Environ. Contam. Toxicol. 16:455460.
- Linzey, A.V. 1988. Effects of chronic polychlorinated biphenyls exposure on growth and
 reproduction of second-generation white-footed mice (Peromyscus leucopus). *Arch. Environ. Contam. Toxicol.* 17:39-45.
- Linzey, D.W., and A.V. Linzey. 1973. Notes on food of small mammals from Great Smokey
 Mountains National Park, Tennessee-North Carolina. J. Elisha Mitchell Science Society 89:6-14.
- 29 Mably, T.A., R.W. Moore, and R.E. Peterson. 1992. In utero and lactational exposure of male
- 30 rats to 2,3,7,8-tetrachlorodibenzo-p-dioxin. 1. Effects on androgenic status. *Toxicol. Appl.*
- 31 Pharmacol. 144:97-107.
- 32 Marks, T.A., G.L. Kimmel and R.E. Staples. 1989. Influence of symmetrical polychlorinated
- biphenyl isomers on embryo and fetal development in mice. *Fund. Appl. Tox.* 13:681-693.

- Masuda, Y., R. Kagawa, H. Kuroki, S. Tokudome and M. Kuratsune. 1979. Transfer of various 1 2 polychlorinated biphenyls to the fetuses and offspring of mice. Fd. Cosmet. Toxicol. 17:623-627.
- 3 McCoy, G., M.F. Finlay, A. Rhone, K. James, and G.P. Cobb. 1995. Chronic polychlorinated 4 biphenyls exposure on three generations of oldfield mice (*Peromyscus polionotus*): Effects on 5 reproduction, growth, and body burdens. Arch. Environ. Contam. Toxicol. 28:431-435.
- Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S. Petron, 6 7 B. Potocki, S. Svirsky and P. Tyler. 1996. Special report of the Massachusetts weight-of -8 evidence workgroup: A weight-of-evidence approach for evaluating ecological risks. Human Ecol. 9
- Risk Assess. 2:277-304.
- 10 Merson, M.H. and R.L. Kirkpatrick. 1976. Reproductive performance of captive white-footed 11 mice fed a PCB. Bull. Environ. Contam. Toxicol. 16(4):392-398.
- 12 Miller, H. and L.L. Getz. 1977. Factors influencing local distribution and species diversity of 13 forest small mammals in New England. Can. J. Zool. 55:806-814.
- 14 Moore, D.R.J., B.E. Sample, G.W. Suter, B.R. Parkhurst and R.S. Teed. 1999. A probabilistic 15 risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East
- 16 Fork Poplar Creek, Oak Ridge, Tennessee, USA. Environ. Toxicol. Chem. 18: 2941-2953.
- 17 Morrison, P.R., M. Pierce and F.A. Ryser. 1957. Food consumption and body weight in the 18 masked and short-tailed shrews (genus Blarina) in Kansas, Iowa, and Missouri. Ann. Carnegie 19 Mus. 51 : 157-180.
- 20 Muir, D.C.G., R.J. Nostrom, and M. Simon. 1988. Organochlorine contaminants in Arctic 21 marine food chains: Accumulation of specific polychlorinated biphenyls and chlordane-related
- 22 compounds. Environmental Science and Technology 22:1071-1079.
- 23 Mumford, R.E., and J.O. Whitaker Jr. 1982. Mammals of Indiana. Indiana University Press, 24 Bloomington, IN.
- 25 Murray, F.J., F.A. Smith, K.D. Nitschke, C.G. Humiston, R.J. Kociba, and B.A. Schwetz. 1979. 26 Three-generation reproduction study in rats given 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) 27 in diet. Toxicol. Appl. Pharmacol. 50:241-252.
- 28 Nagy K.A. 1987. Free metabolic rate and food requirement scaling in mammals and birds. 29 Ecological Monographs 57:111-128.
- 30 Nagy, K.A., I.A. Girard, and T.K. Brown. 1999. Energetics of free-ranking mammal, reptiles, and birds. Annu. Rev. Nutr. 19:247-277. 31
- 32 Neubert, D. and I. Dillmann. 1972. Embryonic effects in mice treated with 2,4,5-33 trichlorophenoxyacetic acid and 2,3,7,8-tetrachlorodibenzo-p-dioxin. Arch. Pharmacol. 272:243-34 264.

- 1 Newsted, J.L., J.P. Giesy, G.T. Ankley, D.E. Tillitt, R.A. Crawford, J.W. Gooch, P.D. Jones, and
- 2 M.S. Denison. 1995. Development of toxic equivalency factors for PCB congeners and the
- 3 assessment of TCDD and PCB mixtures in rainbow trout. Environmental Toxicology and
- 4 *Chemistry* 14(5):861-871
- 5 Nixon, C.M., M.W. McClain and R.W. Donohoe. 1975. Effects of hunting and mast crops on a 6 squirrel population. *J. Wildl. Manage*. 39:1-25.
- NRCC (National Research Council of Canada). 1981. Polychlorinated dibenzo-*p*-dioxins:
 Criteria for their effects on man and his environment. Publication NRCC No. 18574. National
 Research Council of Canada. Ottawa, Ontario. 251 p.
- 10 Oleyar, C.M. and B.S. McGiness. 1974. Field evaluation of diethulstillbestrol for suppressing 11 reproduction in foxes. *J. Wildl. Manage*. 38:101-106.
- 12 Overmann, S.R., J. Kostas, L.R. Wilson, W. Shain and B. Bush. 1987. Neurobehavioral and 13 somantic effects of perinatal PCB exposure in rats. *Environ. Res.* 44:56-70.
- 14 Platt, W. J. 1976. The social organization and territoriality of short-tailed shrew (*Blarina* 15 *brevicauda*) populations in old-field habitats. *Anim. Behav.* 24: 305-318.
- Pohjanvirta, R., M. Unkila and J. Tuomisto. 1993. Comparative acute lethality of 2,3,7,8tetrachlorodibenzo-p-dioxin (TCDD), 1,2,3,7,8-pentachlorodibenzo-p-dioxin and 1,2,3,4,7,8hexachlorodibenzo-p-dioxin in the most TCDD susceptible and the most TCDD-resistant rat strain. *Pharmacol. and Toxicol.* 73:52-56.
- Rands, P.L., R.D. White, M.W. Carter, S.D. Allen, and W.S. Bradshaw. 1982a. Indicators of
 developmental toxicity following prenatal administration of hormonally active compounds in the
 rat. I. Gestational length. *Teratology* 25:37-43.
- Rands, P.L., C.L. Newhouse, J.L. Stewart, and W.S. Bradshaw. 1982b. Indicators of
 developmental toxicity following prenatal administration of hormonally active compounds in the
 rat. II. Pattern of maternal weight gain. *Teratology* 25:45-51.
- Safe, S. 1984. Polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs):
 Biochemistry, toxicology, and mechanism of action. *CRC Critical Reviews in Toxicology* 13:
 319-395.
- Samuel, D.E. and B.B. Nelson. 1982. In: Chapman, J.A., Feldhammer, G.A. eds. *Wild Mammals of North America*. Johns Hopkins University Press, Baltimore, MD. pp 475-490.
- Sanderson, G.C. 1950. Methods of measuring productivity in raccoons. J. Wildl. Manage.
 14:389-402.
- Sargeant, A.B. 1978. Red fox prey demands and implications to prairie duck production. J.
 Wildl. Manage. 42(3):520-527.

- Sargeant, A. B., S. H. Allen, and R. T. Eberhardt. 1984. Red fox predation on breeding ducks in
 mid-continent North America. *Wildlife Monographs* 89:1-41.
- Senthilkumar, K., Iseki, N., Hayama, S., Nakanishi, J., and Masunaga, S. 2001. Polychlorinated
 Dibenzo-p-Dioxins, Dibenzofurans, and Dioxin-like Polychlorinated Biphenyls in Livers of
- 5 Birds from Japan. Arch. Environ. Contam. Toxicol. 42:244-255.
- 6 Sparschu, G.L., F.L. Dunn, and V.K. Rowe. 1971. Study of the Terotogenicity of 2,3,7,8-7 tetrachlorodibenzo-p-dioxin in the rat. *Fd. Cosmet. Toxicol.* 9:405-412.
- 8 Spencer, F. 1982. An assessment of the reproductive toxic potential of Aroclor 1254 in female
 9 Sprague Dawley rats. *Bull. Environ. Contam. Tox.* 28-290-297.
- 10 Stahl, B.U., A. Kettrup, and K. Rozman. 1992. Comparative toxicity of four chlorinated dibenzo-11 p-dioxins (CDDs) and their mixture. *Arch. Toxicol.* 66:471-477.
- Storm, G.L., R.D. Andrews, R.L. Phillips, R.A. Bishop, D.B. Siniff and J.R. Tester. 1976.
 Morphology, reproduction, dispersal and mortality of Midwestern red fox populations. *Wild. Monogr.* 49, 82 pp.
- Thayer, G.W., W.E. Schaaf, J.W. Angelovic, and M.W. LaCroix. 1973. Caloric measurements of some estuarine organisms. *Fishery Bull*. 71: 289-296.
- 17 Thomas, P.T. and R.D. Hinsdill. 1979. The effect of perinatal exposure to tetrachlorodibenzo-p-18 dioxin on the immune response of young mice. *Drug and Chemical Toxicol.* 2(1-2):77-98.
- 19 Tsushimoto, G., F. Matsumura, and R. Sago. 1982. Fate of 2,3,7,8-tetrachlorodibenzo-p-dioxin
- 20 (TCDD) in an outdoor pond and in model aquatic ecosystems. Environmental Toxicology and 21 Chemistry 1:61-68
- 21 Chemistry 1:61-68.
- Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Freely, J.P. Giesy,
 A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X. Rolan van Leeuwen,
 A.K. Djien Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Twilit, M.
- Tickling, M. Younest, F. Warn, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs)
 for PCBs, PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives*106(12):775-792.
- 28 Voigt, D.R. 1987. Red fox In M. Novak, J. A. Baker, M. E. Obbard, and B. Mallock, eds., Wild
- 29 Furbearer Management and Conservation in North America, pp. 378-392. Toronto: Ontario
- 30 Ministry of Natural Resources, Toronto, Ontario.
- Voigt, D.R. and J. Broadfoot. 1983. Locating pup-rearing dens of red foxes with radio-equipped
 woodchucks. *J. Wild. Manage.* 47:858-859.
- 33 Voigt, D.R. and R.L. Tinline. 1980. Strategies for analyzing radio tracking data. In: Amlaner,
- C.J., Jr. and D.W. MacDonald eds. *A Handbook on Biotelemetry and Radio Tracking*. Permagon
 Press, Oxford, United Kingdom. pp. 387-404.

- 1 Wardell, R.E., R.E. Seegmiller, and W.S. Bradshaw. 1982. Induction of prenatal toxicity in the
- 2 rat by diethylstilbestrol, zeranol, 3,4,3',4'-tetrachlorobiphenyl, cadmium and lead. 1982.
- 3 *Teratology* 26:229-237.
- Whitaker, J.O., Jr., and W.J. Hamilton Jr. 1998. *Mammals of the Eastern United States*. 3rd ed.
 Cornell University Press, Ithaca, NY. 538 p.
- 6 Whitaker, O.J., Jr., and M.G. Ferraro. 1963. Summer food of 220 short-tailed shrews from 7 Ithaca, New York. *Journal of Mammalogy* 44:419.
- 8 Wrigley, R.E., J.E. Dubois, and H.W.R. Copland. 1979. Habitat, abundance, and distribution of
- 9 six species of shrews in Manitoba. *Journal of Mammalogy* 60:505-520.

1 11. ASSESSMENT ENDPOINT—SURVIVAL, GROWTH, AND 2 REPRODUCTION OF THREATENED AND ENDANGERED 3 SPECIES

Highlights 4 5 Conceptual Model 6 The assessment endpoint is the survival, growth, and reproduction of T&E species in 7 the Housatonic River PSA. The measurement endpoints include comparisons to 8 doses reported in the literature to cause adverse effects and conducting field surveys 9 to determine the abundance of T&E species in the Housatonic River floodplain. T&E 10 species, including bald eagle, American bittern, and small-footed myotis, selected as 11 representative species for the ERA, are exposed to these COCs via diet and trophic 12 transfer. 13 Exposure 14 Exposure of the representative species to tPCBs and TEQ was determined from 15 concentrations found in prey items and an estimation of the daily intake of these COCs from consumption of prey. 16 17 Effects 18 Limited data were available on the toxicity of tPCBs and TEQ to bald eagle. 19 American bittern, and small-footed myotis. Surrogate species were used to develop 20 toxicity thresholds for bald eagle and American bittern. Sufficient surrogate data 21 were available to generate effects dose-response curves for small-footed myotis. 22 Risk 23 Bald eagle, American bittern, and small-footed myotis are at risk as a result of 24 exposure to tPCBs and TEQ in the Housatonic PSA. In particular, bald eagles are at 25 high risk in the PSA. Other similar, but not T&E, species common to the PSA have 26 either higher levels of risk (e.g., least bittern, green heron); similar levels of risk (e.g., great blue heron, Indiana bat, little brown bat); or a lower level of risk (e.g., sora) 27 28 compared to the representative species.

29

30 11.1 INTRODUCTION

The purpose of this section of the ecological risk assessment (ERA) is to characterize and quantify the current and potential risks posed to rare, threatened, and endangered (T&E) species exposed to contaminants of potential concern (COPCs) in the Housatonic River and floodplain, focusing on total polychlorinated biphenyls (tPCBs) and other COPCs originating from the General Electric Company (GE) facility in Pittsfield, MA. The Housatonic River watershed is located in western Massachusetts and Connecticut, discharging to Long Island Sound, with the GE facility located near the headwaters of the watershed. The Primary Study Area (PSA) includes the river and 10-year floodplain from the confluence of the East and West Branches of
 the Housatonic River downstream of the GE facility to Woods Pond Dam (Figure 1.1-2).

3 A pre-ERA was conducted to narrow the scope of the ERA by identifying contaminants, other 4 than tPCBs, that pose potential risks to aquatic biota and wildlife in the PSA (Appendix B). A 5 three-tiered deterministic approach was used to screen COPCs. The deterministic assessments 6 compared potential conservative estimates of exposure with conservative adverse effects 7 benchmarks to identify which contaminants are of potential concern to T&E species in the 8 Housatonic River. A risk quotient (total daily intake/effect benchmark) for T&E species greater 9 than 1 in the Housatonic River area resulted in the COPC being screened through to the next tier 10 assessment, and to the probabilistic ERA, if necessary.

In summary, the contaminants of concern (COCs) that were retained in the probabilistic risk assessment for T&E species were tPCBs and 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) toxic equivalence (TEQ). Total PCBs detected in Housatonic River media samples closely resemble the commercial PCB mixtures Aroclor 1260 and Aroclor 1254, which are similar in congener makeup. TEQ is calculated from coplanar PCB and dioxin and furan congeners using the toxic equivalency factor (TEF) approach developed by Van den Berg et al. (1998) (see Section 6.4 of the ERA).

18 **11.1.1 Overview of Approach**

A step-wise approach was used to assess the risks of tPCBs and TEQ to T&E species in theHousatonic River watershed. The four main steps in this process include:

- 21
- 22
- 23
- 24 25
- 26

1. Derivation of a conceptual model (Figure 11.1-1).

- 2. Assessment of exposure of T&E species to COCs (Figure 11.1-2).
 - 3. Assessment of the effects of COCs on T&E species (Figure 11.1-3).
 - 4. Characterization of risks to the T&E species community (Figure 11.1-4).
 - The detailed ecological risk assessment for T&E species is provided in Appendix K.





Figure 11.1-1 Conceptual Model Diagram: Exposure Pathways for T&E Species Exposed to COCs in the Housatonic PSA



Figure 11.1-2 Approach Used to Assess Modeled Exposure of T&E Species to COCs

5


1

Figure 11.1-3 Approach Used to Assess the Modeled Effects of COCs to T&E Species



3 Figure 11.1-4 Approach Used to Characterize Risks of COCs to T&E Species

1 11.1.2 Conceptual Model

2 The conceptual model presented in Figure 11.1-1 illustrates the exposure pathways for T&E species exposed to tPCBs and TEQ in the PSA. Total PCBs and TEQ are persistent and highly 3 4 hydrophobic and lipophilic. Therefore, they are bioaccumulated by aquatic and terrestrial biota 5 directly through the consumption of contaminated prey as part of the food chain (Haffner et al. 6 1994; Senthilkumar et al. 2001; Borga et al. 2001). Fish, amphibians, invertebrates, mammals, 7 and birds comprise the major dietary items for T&E species. In summary, T&E species that 8 reside, or partially reside, within the study area are exposed to tPCBs and TEQ principally 9 through diet and trophic transfer. Other routes of exposure, considered to be less important to 10 overall exposure, include inhalation, water consumption, and sediment ingestion (Moore et al. 11 1999).

12 The problem formulation (Section 2) identified the bald eagle (*Haliaeetus leucocephalus*), 13 American bittern (Botaurus lentiginosus), and small-footed myotis (Myotis leibii) as the 14 representative T&E species potentially exposed to tPCBs and TEQ from consumption of 15 contaminated prey. American bitterns have been observed during the breeding season in suitable 16 nesting habitat; therefore, they were chosen for inclusion because of the potential for nesting. 17 Similarly, bald eagles nest downstream, have attempted to nest in the PSA, and have ample 18 habitat available for nesting in the PSA. Small-footed myotis may occur in the PSA as well 19 because of their known range and the suitability of habitat. Life history profiles for the bald 20 eagle, American bittern, and small-footed myotis are presented in the following text boxes. 21 Additional life history information on these species is provided in Appendix K, Sections K.2.1.5, 22 K.2.1.6, and K.2.1.7, respectively.

The assessment endpoint, which is the subject of this section, is the survival, growth, and reproduction of T&E species in the Housatonic River PSA. The potential lines of evidence considered in the evaluation of the assessment endpoint included (1) comparing modeled exposure to doses of tPCBs and TEQ ingested in the diet reported in the literature to cause adverse effects to the survival, reproduction, or growth of omnivorous and carnivorous mammals; and (2) determining, by conducting field surveys, the qualitative abundance of T&E species in the Housatonic River floodplain.



Photo by: Karol Worden

Life History of Bald Eagle

The bald eagle is one of the largest and most conspicuous birds of prey in North America. Weights of adults and juveniles vary from 3 kg to over 7 kg. The bald eagle is currently federally listed as Threatened in all of the 48 lower states, but is more restrictively listed as Endangered by several New England states, including Massachusetts, Vermont, New Hampshire, and Connecticut.

- Habitat Habitat use varies depending on the region, but proximity to large bodies of water with suitable foraging opportunities is critical; thus bald eagles are generally restricted to coastal areas, lakes, and rivers. Relatively open canopies, some type of habitat edge, and the availability of super-story trees with stout horizontal perching branches providing good access to nests are preferred habitat features for breeding pairs.
- Home Range –Large home ranges, minimum size of 1,730 acres (700 ha) and an average size of 4,645 ± 2,224 acres (1,879.76 ± 900.02 ha); linear (riverine) foraging distances of 1.9 to 4.3 miles (3.1 to 6.9 km). Nesting bald eagles were reported to generally forage within 0.3 mile (0.5 km) of the nest, ranging up to 1.9 miles (3.0 km), and as far as 5.0 miles (8.0 km) from their nest.
- Dietary Habits –Feed primarily over water on aquatic prey; opportunistic feeders, consuming a variety of live prey and scavenging carrion. Fish taken primarily from shallow water form the largest percentage of diet. Fish consumption is 17.1% to 90.1%, depending on location, season, and prey availability. Birds, particularly waterfowl, can form large portions of the diet, more commonly during the winter and in coastal habitats. Mammal species average 4.9% of prey; but are reported to be as much as 11.7%, or as little as 0%.

3



Photo by: Scott Robinson

Life History of American Bittern

The American bittern is a mid-sized, stocky heron of freshwater marshes. It is identified by its heavily streaked breast with vertical brown and white stripes below. American bittern populations have been declining since the 1960s primarily as a result of habitat loss and wetland degradation. The Commonwealth of Massachusetts has included the American bittern on its list of Endangered species.

- Habitat Use a wide range of freshwater wetlands with diversity of vegetation classes (i.e., aquatic bed, emergent, and scrub-shrub) and high interspersion of open water and plant cover.
- Home Range Varies with geographic area and availability of preferred habitat and prey species. Average home ranges of 315 acres (127 ha) in Minnesota, with the birds using a 61-acre (25 ha) core area more than 50% of the time. In Massachusetts breeding occurs in scattered localities in Berkshire County. Nests built in dense emergent vegetation over water with depths ranging from 5 to 20 cm (2 to 8 inches), consisting of a 15- to 25-cm (6- to 10-inch) high platform of reeds, sedges, or grasses bent down and lined with fine grasses.
- Dietary Habits Prey upon insects, crayfish, amphibians, fish, and small mammals. Insect prey consists primarily of adult and nymphal dragonflies, giant waterbugs, water scorpions, water beetles, and grasshoppers. Fish species vary with availability and include eels, catfish, pickerel, sunfish, suckers, killifish, sticklebacks, and perch, typically from 10 to 100 mm in length.

	3	
	4 5 6 7 8	
1 1	9 0 1	
1	23	
1	4 5 6	
1	7 8	
12	9 0 1	
-	1	

22

23

 $\frac{1}{2}$



Photo Credit: Merlin Tuttle (http://www.dcnr.state.pa.us/wrcf/myopic.htm)

Life History of Small-Footed Myotis

The small-footed myotis is a small bat. It is identified by its golden brown fur and black mask. The small-footed myotis is listed as a Species of Special Concern by the Massachusetts Natural Heritage and Endangered Species Program (MNHESP) (1984).

- Habitat Use buildings, overhanging rocks, and caves as summer roosts and maternity sites. Females and young roost in small (typically less than 20 individuals) maternity colonies in rock crevices and crevice-like places on buildings; males are solitary. Hibernate hanging from walls or underneath fallen rock and rubble from November to March, usually in the foothills of mountains up to 610 m (2,000 feet) in elevation, in coniferous woodlands.
- **Home Range** Home range is unknown. It is assumed that home ranges are similar to other *Myotis* species (Indiana bat that has a home range of 52 to 95 ha for pregnant and lactating females).
- Dietary Habits Little is known about feeding habits; however, believed to be similar to other *Myotis* species. Flies, beetles, bugs, leafhoppers, and flying ants have been found in their stomachs. Many species are opportunistic feeders, exploiting available food resources. They fly low to the ground (1 to 3 m) when feeding, along forest openings, including waterways.

 $\frac{1}{2}$

20

1 **11.1.3 Organization**

2 This section is organized as follows:

3 Section 11.2 (Exposure Assessment) - Describes the exposure model, input 4 variables, and techniques to propagate uncertainty. This section also presents the exposure modeling results for T&E species. 5 6 Section 11.3 (Effects Assessment) – Describes the effects to T&E species exposed to 7 tPCBs and TEQ and summarizes the ranges of benchmarks (toxicity thresholds) 8 derived from the literature. 9 Section 11.4 (Risk Characterization) - Integrates the exposure and effects 10 assessments, and makes conclusions regarding risk for T&E species in the Housatonic River PSA using the two potential lines of evidence. A discussion of the sources of 11 uncertainty regarding risk estimates follows. This section also presents an 12 extrapolation of risks beyond the PSA to areas downstream of Woods Pond. 13 14

1 11.2 EXPOSURE ASSESSMENT

The exposure of T&E species to tPCBs and TEQ in the Housatonic River PSA is estimated in 2 3 this section. The representative T&E species are the bald eagle, American bittern, and small-4 footed myotis. These species are T&E species that occur in the PSA, potentially breed within the 5 PSA, and feed on prey exposed directly to the COCs and through trophic transfer (see Appendix 6 A). Trophic transfer and exposure through ingestion of contaminated prey are the major 7 exposure pathways for T&E species exposed to tPCBs and TEQ. Other routes of exposure, 8 considered to be negligible contributors to overall exposure, include inhalation, water 9 consumption, and sediment ingestion (Moore et al. 1999).

All of the bald eagle sightings in the PSA occurred south of New Lenox Road, primarily at Woods Pond and the backwaters north of Woods Pond (Appendix A). Bald eagles would not be expected to regularly utilize the more shallow and narrow northern sections of the river. Therefore, the exposure area assumed for bald eagles was the southern portion of the PSA, from the more downstream portion of Reach 5B to Woods Pond. This entire area was not subdivided because individual bald eagles would likely forage throughout this area.

American bittern habitat occurs throughout the PSA, and bitterns have been observed from Canoe Meadows Wildlife Sanctuary south to Woods Pond (Appendix A). In addition, home range sizes and habitat requirements for this species are such that individuals forage predominantly within one subreach. Therefore, for the PCB analyses, the PSA was split into four reaches: Reach 5A, Reach 5B, Reach 5C, and Reaches 5D and 6 combined. For the TEQ analyses, samples from the PSA were combined into one analysis because the smaller sample sizes did not allow for statistically robust analyses to be conducted for each subreach.

Little is known about the home range size of the small-footed myotis. The Indiana bat (*Myotis* sodalis), a similar Myotis species, has a range averaging 128 acres (52 ha), but this range may be as large as 232 acres (94 ha) for lactating female bats (Kurta 1995; DeGraaf and Yamasaki 2001). The exposure area assumed for small-footed myotis was the entire PSA because of the small number of dietary samples from each reach.

This section begins with a description of the exposure model used for the representative species. 1

2 Subsequent sections describe the inputs used in the exposure analyses for each species. The 3 section concludes with a presentation of the results of the exposure analyses.

11.2.1 4 Exposure Model

5 Exposure of T&E species to tPCBs and TEQ was estimated using a total daily intake model 6 adapted from the Wildlife Exposure Factors Handbook (EPA 1993) and related publications. 7 The model used in the exposure analysis was:

8
$$TDI = FT \cdot FIR \sum_{i=1}^{n} C_i \cdot P_i$$
 (Eq. 1)

9 where

10	TDI	=	total daily intake (mg/kg bw/d tPCBs, ng/kg bw/d TEQ)
11	FIR	=	normalized food intake rate (kg/kg bw/d)
12	FT	=	foraging time in Primary Study Area (unitless)
13	C_i	=	concentration in <i>i</i> th food item (mg/kg tPCBs, ng/kg TEQ)
14	P_i	=	proportion of the <i>i</i> th food item in the diet (unitless)
15			```````

16 The models consider the food intake rates (FIRs) of the representative species (FIR), the 17 concentrations of COCs in each food item (C_i) , and the proportion of the diet accounted for by 18 that food item (P_i) . For those input variables that are uncertain, variable, or both, distributions 19 are used rather than point estimates. Monte Carlo and probability bounds analyses are the 20 methods used to propagate uncertainties about input variables through the exposure model for 21 each COC. A description of these techniques and the methods used to parameterize input 22 variables is presented in Section 6.5. The results of the Monte Carlo analysis are used to 23 estimate the probability of exposure exceeding an effects threshold or doses that cause adverse 24 effects of differing magnitudes. The probability bounds analysis is conducted to determine how 25 uncertainty regarding the distributions of the input variables influences the estimated exposure 26 distribution. The results of these analyses are discussed in detail in Appendix K.

27 Two circumstances often arose when calculating a TEQ concentration in prey:

28

Congener concentrations may be below the method detection limit (i.e., non-detects).

29

1 An approach was developed to address these circumstances and is presented in Section 6.4 and 2 Appendix C.2. Briefly, congeners detected at or below the detection limit (DL) were included in 3 the TEQ calculations by investigating three options: first, setting the value for the congener equal 4 to zero (0); setting it to half the DL; and, finally, setting it equal to the DL. A comparison of the 5 results of this bounding analysis provides a description of the uncertainty surrounding the TEQ 6 value due to the concentrations of one or more congeners being below the detection limit. To 7 resolve the co-elution issue, the concentration of congeners that co-eluted with other congeners 8 were assumed to equal to the total concentration of the co-elutes (potential overestimate of TEQ) 9 concentration) or zero (potential underestimate of TEQ concentration).

10 Input distributions to the exposure analyses were generally assigned as follows:

- Lognormal distributions for variables that were right skewed with a lower bound of zero and no upper bound (e.g., amount of COC transferred from mother to offspring via egg tissue).
 - Beta distributions for variables bounded by zero and one (e.g., proportion of a prey item in the diet).
- 16 17

14

15

- Normal distributions for variables that were symmetric and not bounded by one (e.g., body weight).
- 18

Point estimates for minor variables or variables with low coefficients of variation.

In certain situations (e.g., poor fit of data), other distributions were fit to the data or other approaches were used. To quantify uncertainty, two approaches were used as described in Section 6.5.2, Monte Carlo Analysis and Probability Bounds Analysis. The distributions used in the exposure analyses for bald eagle, American bittern, and small-footed myotis are shown in Figures 11.2-1, 11.2-2, and 11.2-3. A brief description of these variables is provided below.

24 11.2.1.1 Input Variables

25 11.2.1.1.1 Body Weight *(BW)*

The typical weight of an adult bald eagle ranges from 3.0 kg to over 7.0 kg. Adult males average 4.13 kg and adult females average 5.4 kg (Dunning 1992; EPA 1993; Buehler 2000; Canadian Wildlife Service 2000). For this risk assessment, the weight of female bald eagles was used because the effects endpoint is reproductive impairment, and the female will have the greatest effect on survival of the young through transfer of PCBs to the egg. The mean weight was 5.35
 kg with a standard deviation of 0.40.

The typical weight of an adult American bittern ranges from 370 g to >800 g (Gibbs et al. 1992;
Dunning 1992). In the Monte Carlo analyses, the mean BW of 707 g with a standard deviation
of 183 was used (Dunning 1992).

6 Small-footed myotis typically weigh 5 to 7 g, although their weights can range from 3 to 8 g
7 (Kurta 1995). A mean BW of 6 g with a standard deviation of 0.7 g was used in the exposure
8 analyses for small-footed myotis.

9 11.2.1.1.2 Food Intake Rate (FIR)

10 In the EPA Wildlife Exposure Factors Handbook (EPA 1993), three studies were used to 11 determine FIR. The first study (Stalmaster and Gessaman 1982) used captive eagles (obtained 12 from a zoological garden), housed in $3x_3x_2.5$ -m chambers, in their feeding study. The second 13 study (Stalmaster and Gessaman 1984) estimated feeding rates by remote observation of the 14 amount of food consumed at feeding stations. Estimates were averages of the total estimated 15 food consumed by the total number of eagles observed feeding, and assumed that the eagles fed 16 exclusively at the stations, although the authors acknowledged that some birds fed elsewhere. In 17 the third study (Craig et al. 1988), Stalmaster and Gessaman's (1984) data were used to estimate 18 prey consumption. Because of the issues associated with estimating food consumption in each of 19 these studies, the FIR was derived from the estimated metabolic rate of free-living eagles using 20 data from Nagy (1987) and Nagy et al. (1999).

The FIR developed from the Nagy studies was compared with those from EPA (1993). The measured FIRs reported in the Wildlife Exposure Factors Handbook (EPA 1993) are consistent with the FIR distribution estimated from the allometric equation. Estimated values for freeflying eagles from Connecticut (Craig et al. 1988) were 0.12 to 0.14 g/g bw/d, while the median FIR from the allometric equation was 0.158 g/g bw/d.

Nagy (1987) and Nagy et al. (1999) derived allometric equations for estimating the free metabolic rate (FMR) of free-living mammals in kilojoules per day using the following general equation:





7/10/2003





Figure 11.2-2

0.4 0.2 0

0 200 400 600 800 1000

Input Distributions for the Exposure Modeling of American Bittern



1200 1400

Weight (g)

Figure 11.2-3
 Figure 11.2-3
 Input Distributions for the Exposure Modeling of Small-Footed Myotis

1.2 1.4

$$FMR \ (kJ/d) = a \cdot BW(g)^b \tag{Eq. 2}$$

where *FMR* (kcal/kg bw/d) is the free-living metabolic rate, and *BW* (g) is the body weight in
grams. The slope (a) and power (b) distributions were based on the error statistics reported in
Nagy et al. (1999). For birds, the equation was used and had a mean slope term (a) equal to 8.47
and a standard error of 1.57. The power term (b) had a reported mean of 0.768 and a standard
error of 0.087 (Nagy et al. 1999). For small-footed myotis, the insectivore equation was used.
The slope term (a) had a reported mean of 6.98 and a standard error of 4.19, and the power term
(b) had a reported mean of 0.622 and a standard error of 0.0630 (Nagy et al. 1999). The BW

9 distribution was described above.

1

11

10 FIR is derived from FMR using the following equation:

$$FIR = \frac{FMR}{\sum_{i=1}^{n} AE_i \cdot GE_i}$$
(Eq. 3)

where *AE* is the assimilation efficiency of *i*th food item (unitless) and *GE_i* is the gross energy of *i*th food item (kcal/kg).

The gross energies of various wildlife food sources are summarized in the *Wildlife Exposure Factors Handbook* (EPA 1993). The mean gross energy is 1.6 for invertebrates, 1.2 for fish and amphibians, and 1.8 for birds and mammals.

The mean assimilation efficiency for fish and amphibians consumed by birds is 79%. For the consumption of mammals by birds, the mean assimilation efficiency is 78%. For the consumption of invertebrates by birds, the mean assimilation efficiency is 72%. Point estimates were used for these variables in the Monte Carlo and probability bounds analyses because of their relatively small coefficients of variation (i.e., CV<10%).

22 **11.2.1.1.3** Proportions of Dietary Items (*P_i*)

The proportions of prey items in bald eagle diets are listed in Table K.2-1. Studies of bald eagles in habitat similar to the PSA have found mean fish consumption to be 77.5% (range 71.0 to 90.1)

1 of the prey species taken during the breeding season. Bird species comprise on average 16.9% of 2 the bald eagle diet in habitats similar to the PSA, but can be as little as 7.9% and up to 26.1%. 3 Consumption of mammals averages 4.8% and can range from 1.3% to 11.7%. Reptiles make up 0.24% of the diet and can range from 0 to 0.6%. Invertebrates such as cravfish, crabs, and 4 5 mussels make up 0.12% of the diet on average and can range from 0 to 0.6% (Havwood and 6 Ohmart 1986; Dunstan and Harper 1975; Todd et al. 1982; Watson et al. 1991; Stratus 1999). 7 Reptiles and invertebrates were not included in the exposure analysis, however, because of their 8 small contribution to the overall diet. The proportion in the diet for the Monte Carlo analysis 9 was parameterized to allow the diet to equal 1. This resulted in a diet of 50.3% bottom fish, 10 16.1% predatory fish, 11.8% forage fish, 16.3% birds, and 5% mammals (Table K.2-1). For the 11 probability bounds analysis, the minimum, mean, and maximum values were used as specified 12 above (Table K.2-3).

13 An analysis of the stomach contents of 160 individuals reported that the American bittern diet 14 consisted of invertebrates (23%), amphibians (21%), fish (21%), crayfish (19%), small mammals 15 (10%), and snakes (5%) (Cottam and Uhler 1945, as cited in Gibbs et al. 1992). It was assumed 16 that American bitterns would consume the same proportion of prey items in each reach for this 17 assessment. Reptiles were not included in the exposure analysis, however, because of their small 18 contribution to the overall diet. The proportion in the diet for the Monte Carlo and probability 19 bounds analysis was parameterized to allow the diet to equal 1. This resulted in a diet of 24.5% 20 invertebrates, 20.2% macroinvertebrates (crayfish), 22.3% fish, and 22.3% amphibians (Table 21 K.2-10).

22 Along the Housatonic River, the small-footed myotis likely forages on small emergent aquatic 23 insects, as does the little brown bat. Adult little brown bats in New York were found to consume 24 Chironomidae (76.4% of food volume), Trichoptera (18.2%), Lepidoptera (4.2%), and 25 Coleoptera (1.2%) (Belwood and Fenton 1976). Other studies conducted in the northeast found 26 these to be commonly consumed species along with other Diptera, such as Tipulidae, Culicidae, 27 Homoptera, Hymenoptera, Neuroptera, Plecoptera, and Ephemeroptera (Griffith and Gates 1985; 28 Anthony and Kunz 1977). For this exposure assessment, the proportion of invertebrates in the 29 diet was assumed to be a point estimate, with invertebrates accounting for 100% of the small-30 footed myotis diet.

1 **11.2.1.1.4 Foraging Time** *(FT)*

Bald eagles nesting in the PSA would be expected to forage entirely within the PSA, as they generally forage within 0.3 miles (0.5 km) of the nest, with a maximum reported foraging distance of up to 5.0 miles (8.0 km) from their nest (Bowerman et al. 1995; Stratus 1999). As a result, for the purpose of modeling COC exposure, it was assumed that bald eagles would spend 100% of their time foraging in the PSA, based on their feeding habits and availability of fish, waterfowl, and mammals in the PSA.

8 American bittern nesting in the PSA would be expected to forage entirely within the PSA, as 9 they have territories averaging 315 acres of wetlands (DeGraaf and Yamasaki 2001). As a result, 10 it was assumed that American bitterns would spend 100% of their time foraging in the PSA. The 11 foraging time was specified as a point estimate.

Little is known about home range size of the small-footed myotis. The Indiana bat, a similar *Myotis* species, has a range averaging 128 acres (52 ha), but this range may be as large as 232 acres (94 ha) for lactating female bats (Kurta 1995; DeGraaf and Yamasaki 2001). Small-footed myotis feed predominantly over water on emergent insects; therefore, it was assumed that smallfooted myotis would forage 100% of the time in the PSA.

17 **11.2.1.2 Concentrations of COCs in Prey**

Fish, birds, and mammals are the major dietary items for bald eagles. The median concentration of tPCBs in bottom feeding fish is 59.1 mg/kg (mean = 88.7 mg/kg), 64.8 mg/kg (mean = 79.1 mg/kg) for predatory fish, and 34.5 mg/kg (mean = 36.9 mg/kg) for forage fish. The median concentration of tPCBs in birds is 6.09 mg/kg (mean = 7.18 mg/kg). The median concentration of tPCBs in mammals is 4.98 mg/kg (mean = 28.2 mg/kg). The median, 25th and 75th percentile concentrations of tPCBs and TEQ are presented in Figures 11.2-4 and 11.2-5.



7/10/2003

1 The diet for American bittern includes fish, amphibians, small mammals, invertebrates, and 2 macroinvertebrates. Similar statistics for concentrations of tPCBs and TEQ in American bittern 3 prey from Reaches 5A, 5B, 5C, and 5D and 6 are presented in Figures 11.2-6 and 11.2-7, 4 respectively.



Small-footed myotis prey items were not directly sampled in the PSA. Concentrations of tPCBs 1 2 in invertebrates were obtained from samples of tree swallow (Tachycineta bicolor) gut contents (Custer 2002). Tree swallow gut content samples were used for small-footed myotis prey 3 4 because these samples contain prey species that are more representative of actual myotis prey 5 items than are the benthic invertebrate samples. Small-footed myotis and tree swallows are both 6 aerial insectivores that forage primarily over open water and consume similar types of 7 invertebrates; therefore, gut contents of tree swallows are likely to be similar to that of the small-8 footed myotis. The median concentration of these samples was 7.10 mg/kg for tPCBs and 564 9 ng/kg for TEQ.

In the Monte Carlo analysis, it was assumed that the spatially and temporally averaged exposure estimate did not vary between individuals foraging in the same area. Thus, the point estimate of centrality was the minimum of:

- The 95% upper confidence limit (UCL) calculated using the Land H-statistic (assuming data are lognormally distributed), or
- 15
 2. The maximum concentration measured. In the probability bounds analyses, however,
 the uncertainty regarding the arithmetic mean was accounted for with a different
 procedure.

The procedure generally involved using the Land H-statistic to estimate the lower and upper 95% confidence limits on the mean (Gilbert 1987), and then using these lower and upper confidence limits to derive bounds on all possible distributions that exist within this range. This approach results in an expression of the uncertainty about the true value of the arithmetic mean that arises due to the small sample size.

25 **11.2.2** Results of Exposure Assessments

Examples of exposure distributions for exposure to tPCBs and TEQ for bald eagles in the southern PSA, American bittern in Reaches 5 and 6, and small-footed myotis in the PSA are presented in Figures 11.2-8 through 11.2-23.

The input variables for concentrations of COCs in prey of bald eagle, American bittern, and small-footed myotis are shown in Tables K.2-4, K.2-5, K.2-12, K.2-13, K.2-20, and K.2-21.

1 **11.2.2.1 Bald Eagle**

Figure 11.2-8 depicts the cumulative distribution of tPCB intake rates by bald eagles in the southern PSA. The Monte Carlo analysis indicated that exposure of bald eagles to tPCBs ranges from a minimum of 6.23 to a maximum of 25.4 mg/kg bw/d. The mean exposure was 13.2 mg/kg bw/d, and the median exposure 13.0 mg/kg bw/d. Eighty percent of the exposure estimates were between 10.2 and 16.6 mg/kg bw/d (Table K.2-6).

The probability bounds estimated for bald eagles foraging in the southern PSA are depicted in Figures 11.2-8 and 11.2-9. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 3.73 and 13.4 mg/kg bw/d. The 50th percentile ranged between 5.41 and 17.9 mg/kg bw/d, and the 90th percentile ranged between 8.27 and 24.2 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 10.2, the 50th percentile was 13.0, and the 90th percentile was 16.6 mg/kg bw/d (Table K.2-6).

Female bald eagles present in the PSA for 30 days prior to egg laying are estimated to have a mean tPCB egg concentration of 35.3 mg/kg, a low egg concentration of 23.0 mg/kg, and a high concentration of 51.5 mg/kg (Figure 11.2-10). Female bald eagles present in the PSA for 30 days prior to egg laying are estimated to lay eggs with a mean TEQ concentration of 683 ng/kg, a low concentration of 440 ng/kg, and a high concentration of 997 ng/kg (Figure 11.2-11).











1 11.2.2.2 American Bittern

The Monte Carlo analysis indicated that exposure of American bittern to tPCBs ranges from a minimum of 4.70 to a maximum of 18.6 mg/kg bw/d. The mean exposure was 9.24 mg/kg bw/d, and the median exposure was 9.07 mg/kg bw/d. Eighty percent of the exposure estimates were between 7.30 and 11.4 mg/kg bw/d (Table K.2-14).

6 The probability bounds estimated for American bittern foraging in the PSA are depicted in 7 Figures 11.2-12 through 11.2-21. In Reach 5A, the 10th percentile of the probability envelope 8 formed by the lower and upper bounds ranged between 3.53 and 7.44 mg/kg bw/d. The 50th 9 percentile ranged between 4.42 and 9.19 mg/kg bw/d, and the 90th percentile ranged between 10 5.49 and 11.7 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 11 7.30, the 50th percentile was 9.07, and the 90th percentile was 11.4 mg/kg bw/d (Table K.2-14).

Exposures of American bittern to tPCBs in Reaches 5B, 5C, and 5D, and 6 were similar or lower than in Reach 5A, having a mean TDI of 7.84, 9.03, and 6.53 mg/kg bw/d, respectively (Table K.2-14). Thus, exposure of American bittern to tPCBs is similar for all reaches of the PSA. The uncertainty of these exposure estimates, as illustrated by the probability bounds distributions, indicates a similar degree of uncertainty for all four reaches.

Mean exposure of American bittern to TEQ was 372 for the PSA (Table K.2-16). Figures 11.216 and 11.2-21 depict the cumulative distribution for TEQ intake, as well as the probability
bounds.

The lowest egg concentrations after 45 days in the PSA were 24.7, 21.0, 24.2, and 17.5 mg/kg for Reaches 5A, 5B, 5C, and 6, respectively. Mean egg concentrations in the PSA after 45 days were 37.0, 31.4, 36.2, and 26.2 mg/kg for Reaches 5A, 5B, 5C, and 6, respectively. High egg concentrations after 45 days in the PSA were 53.0, 44.9, 51.8, and 37.4 mg/kg for Reaches 5A, 5B, 5C, and 6, respectively. The estimated TEQ egg concentration for American bitterns over time is shown in Figure 11.2-21. The lowest egg concentrations after 45 days in the PSA was 898 ng/kg, the mean concentration was 1,490 ng/kg, and the high concentration was 2,290 ng/kg.







4

Reaches 5D and 6



5









Figure 11.2-17 American Bittern Egg Exposure to tPCBs in Reach 5A of the Housatonic River Primary Study Area

5 6 7





Figure 11.2-18 American Bittern Egg Exposure to tPCBs in Reach 5B of the
 Housatonic River Primary Study Area

















1 11.2.2.3 Small-Footed Myotis

2 11.2.2.3.1 Total PCBs

The Monte Carlo analysis indicated that exposure of small-footed myotis to tPCBs could range from a minimum of 2.05 to a maximum of 96.0 mg/kg bw/d. The mean exposure was 16.7 mg/kg bw/d, and the median exposure was 14.5 mg/kg bw/d (Table K.2-22). Eighty percent of the exposure estimates were between 7.17 and 28.8 mg/kg bw/d. Figure 11.2-22 depicts the cumulative distribution for small-footed myotis in Reach 5.

The probability bounds estimated for small-footed myotis foraging in Reach 5 are depicted in Figure 11.2-22. The 10th percentile of the probability envelope formed by the lower and upper bounds ranged between 1.96 and 7.67 mg/kg bw/d. The 50th percentile ranged between 4.05 and 15.0 mg/kg bw/d, and the 90th percentile ranged between 8.09 and 32.2 mg/kg bw/d. In comparison, the 10th percentile of the Monte Carlo output was 7.17, the 50th percentile was 14.5, and the 90th percentile was 28.8 mg/kg bw/d (Table K.2-22).





14

Figure 11.2-22 Total Daily Intake (TDI) of tPCBs by Small-Footed Myotis in Reach
 5 of the Housatonic River Primary Study Area

11.2.2.3.2 TEQ 1

2 The Monte Carlo analysis indicated that exposure of small-footed myotis to TEQ ranges from a 3 minimum of 61.4 to a maximum of 7,020 ng/kg bw/d. The mean exposure was 1,130 mg/kg 4 bw/d, and the median exposure was 936 ng/kg bw/d (Table K.2-24). Eighty percent of the 5 exposure estimates were between 381 and 2,120 ng/kg bw/d. Figure 11.2-23 depicts the cumulative distribution for small-footed myotis in Reach 5. 6

7 The probability bounds estimated for small-footed myotis foraging in the PSA are depicted in Figure 11.2-23. The 10th percentile of the probability envelope formed by the lower and upper 8 bounds ranged between 18.8 and 985 ng/kg bw/d. The 50th percentile ranged between 38.1 and 9 1,910 ng/kg bw/d, and the 90th percentile ranged between 74.8 and 3,900 ng/kg bw/d. In 10 comparison, the 10th percentile of the Monte Carlo output was 381, the 50th percentile was 936, 11 and the 90th percentile was 2,120 ng/kg bw/d (Table K.2-24). 12



Reach 5

13 14

Figure 11.2-23 Total Daily Intake (TDI) of TEQ by Small-Footed Myotis in Reach 5 of the Housatonic River Primary Study Area 15

1 11.3 EFFECTS ASSESSMENT

2 The purpose of the effects assessment is to review the scientific literature and to derive the most 3 appropriate metrics for effects of tPCBs and TEQ to T&E species. An effects metric can be 4 represented by a dose-response relationship or a daily dose of a COC that represents a threshold 5 beyond which toxic effects may appear in T&E species. The effects metric is used, in 6 conjunction with the exposure assessment, to estimate risks to T&E species exposed to tPCBs and TEQ in the Housatonic River PSA. This section focuses on effects that have an influence on 7 8 the long-term maintenance of T&E species populations (i.e., mortality or impairment of 9 reproduction or growth).

10	Toxicity of tPCBs and TEQ to Avian Species				
11	Mode of Action				
12 13	Binding to the aryl-hydrocarbon (Ah) receptor, eliciting an Ah receptor-mediated biochemical and toxic response.				
14	Types of Toxicity	Specific Effects			
15 16 17 18 19 20	hepatotoxicity immunotoxicity neurotoxicity embryotoxicity teratogenicity	mortality decreased growth weight loss porphyria reduced hatching embryo mortality			
21					
22	Toxicity of tPCBs and TEQ to Mammal Species				
23	Mode of Action				
24 25	Binding to the Ah receptor, eliciting an Ah receptor-mediated biochemical and toxic response.				
26	Types of Toxicity	Specific Effects			
27 28 29 30 31	hepatotoxicity immunotoxicity neurotoxicity embryotoxicity teratogenicity	mortality decreased growth decreased body and organ weight reduced survival at birth and weaning reduced fertility			

1 Mode of Action of TEQ Congeners 2 Congeners that have been assigned a 2,3,7,8-TCDD TEF have the ability to bind 3 with the Ah receptor and elicit similar toxic responses. The most toxic congeners 4 tend to be those that have a planar shape and are chlorinated in the 2,3,7, and 8 5 6 7 positions for dioxins and furans, and in the meta and para positions for PCBs. This leads to a common mechanism of action in many animal species involving binding to the Ah receptor and elicitation of an Ah receptor-mediated biochemical and toxic 8 9 response. The toxic response of this group of chemicals is, therefore, related to the three-dimensional structure of the substance, including the degree of chlorination 10 and positions of the chlorine on the aromatic frame.

11

A brief review of the scientific literature on the effects of tPCBs and TEQ to T&E species from dietary exposure is presented in the following sections. The discussion focuses on ecologically relevant effect endpoints such as survival, growth, and reproduction of T&E species. A summary of reproduction effects for tPCBs and TEQ is presented in Figures K.3-1 and K.3-2 and Table K.3-1. The effects metrics used for this assessment are also presented.

17 **11.3.1 Total PCBs**

18 Laboratory studies on the toxicity of PCBs to bald eagles and American bittern have not been 19 conducted. However, studies using other avian species were available. Appendix H provides 20 detailed descriptions of dietary and in ovo exposures of PCBs and TEQ to surrogate bird species.

21 Laboratory studies on raptor species demonstrated that PCBs cause adverse effects. American 22 kestrels (Falco sparverius) dosed in ovo to produce a mean PCB tissue concentration of 34.1 23 mg/kg on a whole egg wet weight (ww) basis (PCBs were a 1:1:1 mixture of Aroclors 24 1248:1254:1260) had decreased reproductive success, including suppression of egg laying, 25 delays in clutch initiation, smaller clutch sizes, and reduced fledgling survival (Fernie et al. 26 2001a). Twenty-five percent of exposed females failed to lay any eggs compared to 9% of the 27 control females. PCB-exposed females also had lower fledgling success: 55% compared to 28 93.3% in the control group. Males exposed to PCBs in ovo also showed reduced reproductive 29 success, with 63.5% of their broods experiencing complete mortality compared to 0% complete 30 mortality in the control group (Fernie et al. 2001a).

Numerous field studies have found that organochlorine compounds negatively impact the
 reproductive success of raptors and piscivorous birds (see overview in Donaldson et al. 1999).
 Toxicological effects include reduced hatching success, malformation, edema, and reduced organ
 and body weight (Elliott et al. 1996).

5 Wiemeyer et al. (1993) reported a significant reproductive decline in bald eagles with egg 6 concentrations greater than 13 mg/kg. However, PCB concentrations were highly correlated 7 with DDE concentrations, thus this threshold must be considered with caution. For sensitivity to 8 PCBs, American kestrels can also be considered as a surrogate species for bald eagles. A daily 9 intake rate of 7 mg/kg bw/d was shown to cause an increase in laying lag and a decrease in the 10 number of fledglings per breeding pair (Fernie et al. 2001a and b). These birds had a long 11 exposure period (100 days) and the study covered a sensitive life stage.

12 Hoffman et al. (1986) found a negative correlation between embryonic weight and tPCB residues 13 in eggs of black-crowned night herons nesting in the San Francisco Bay. Heron eggs had a mean 14 PCB concentration of 4.1 mg/kg (ww). PCB-contaminated embryos, with the yolk sac removed, 15 had a significantly lower (15%) weight than embryos from clean sites. Concentrations of other 16 organochlorines were low (mean DDE concentration of 1.7 mg/kg). Other effects associated 17 with PCB exposure in black-crowned night herons included reduced femur to body weight ratio, 18 increased edema, and increased hepatic aryl hydrocarbon hydroxylase activity (Hoffman et al. 19 1993); some of these effects may have been related to the presence of other contaminants, 20 although the authors stated that concentrations of these other contaminants were not high enough 21 to account for the observed effects. Laporte (1982) reported that mean tPCB concentrations of 22 15 mg/kg in eggs negatively impacted great blue heron reproductive success in Quebec.

Great blue herons in Indiana showed no observable effects at 4.9 mg/kg tPCBs in their eggs (predominantly PCB congeners 118/106, 105, and 156) (Custer et al. 1998). Great blue herons in Texas had a mean of 6.2 mg/kg PCBs in their eggs and fledged 1.6 young per nest, a value within the range of stable populations (Mitchell et al. 1981). Total egg PCB concentrations of 1 mg/kg were found to have no effect on great blue heron productivity (Elliott et al. 1989).

No PCB toxicology studies have been conducted on small-footed myotis; however, studies have
been conducted for little brown bats and big brown bats. The little brown bat is a closely related

species that has similar habitat, behavior, diet, and size. The big brown bat also shares many
 traits with the small-footed myotis (see Appendix A).

3 Studies have shown that bats accumulate PCBs from their diet (Clark and Lamont 1976a; Clark 4 and Lamont 1976b; Clark and Prouty 1976; Clark 1978; Clark and Stafford 1981). Clark and 5 Prouty (1976) found that little brown bats accumulated higher concentrations of organochlorine 6 compounds (including PCBs, DDT, and DDE) than did other bat species at the same location. 7 Little brown bats also accumulated brain PCB concentrations that were higher than brain 8 concentrations of other bat species, which may make them more susceptible to PCB poisoning 9 (Clark and Prouty 1976). Like the little brown bat, small-footed myotis may also be more 10 susceptible to PCBs because they have similar physiology, diet, and habitat.

Adult little brown bats fed mealworms with 15 mg/kg PCBs (Aroclor 1260) for 40 days accumulated a mean PCB concentration of 92 mg/kg (carcass ww) (Clark and Stafford 1981). In the same study five little brown bats were fed a diet of mealworms containing 1,000 mg/kg PCBs; four of the bats died before 40 days. These four bats had a mean PCB concentration of 3,300 mg/kg (ww). The one surviving bat had a PCB concentration of 940 mg/kg (Clark and Stafford 1981).

17 Residues of PCBs in bat brains are a linear function of the amount of fat and residues in the 18 carcass (Clark and Prouty 1977; Clarke et al. 1978; Clark and Stafford 1981). During 19 hibernation, the percent of lipids in the body decreases, but the lipid percentage in the brain does 20 not change, which results in elevated concentrations of PCBs in the brain (Clark and Prouty 21 1977; Clark and Stafford 1981). Elevated concentrations of PCBs in the brain may lead to 22 tremoring, a common symptom of organochlorine poisoning (Clark and Stafford 1981). Bats in 23 hibernation have energy stores that are closely balanced against needs, and any disturbance, such 24 as tremoring, that increases metabolic rates can cause mortality through starvation (Clark and 25 Stafford 1981).

PCBs are also known to have adverse reproductive effects on bats (Clark and Lamont 1976a;
Clark and Lamont 1976b; Clark 1978). Female bats transfer PCBs to their young through the
placenta (Clark et al. 1975; Clark 1978). Clark and Lamont (1976a) found that neonates contain
16.8% to 31.8% as much PCBs as their parents. Organochlorine contaminants are also passed to

the young through the mother's milk (Clark et al. 1975; Clark and Lamont 1976a). Milk collected from the stomachs of young big brown bats contained a PCB (Aroclor 1260) concentration of 13 mg/kg. The young had a mean PCB concentration of 0.7 mg/kg (Clark and Lamont 1976a). Wild captured female big brown bats that produced dead young contained significantly higher concentrations of PCBs (1.99 mg/kg ww) than those that produced live young (0.56 mg/kg ww) (Clark and Lamont 1976b).

7 11.3.2 2,3,7,8-TCDD Toxic Equivalence (TEQ)

8 Several researchers estimated NOAEL and LOAEL values for TEQ for bald eagles (Giesy et al. 9 1995, Bowerman et al. 1995, Elliott et al. 1996). Giesy et al. (1995) and Bowerman et al. (1995) 10 sampled the impact of contaminated prey fish on bald eagles in the Great Lakes region and 11 derived a NOAEL for bald eagle eggs of 7 ng/kg TEQ. This value is based on toxicity studies 12 conducted using other avian species, including the chicken, wood duck, and American kestrel. 13 The bald eagle is less sensitive to TEQ compared to the chicken, ducks, and other gallinaceous 14 species; therefore, this value may be low (Elliott et al. 1996; Elliott and Harris 2002 in press). 15 Elliott et al. (1996) reported a NOAEL of 135 ng TEQ/kg egg and a LOAEL of 400 ng TEQ/kg 16 egg based on studies of incubated bald eagle eggs taken from nests in British Columbia. Studies 17 conducted on ospreys (Pandion haliaetus) found a similar NOAEL on hatching success of 136 18 ng/kg (Elliott et al. 2001), and Woodford et al. (1998) found that 162 ng/kg had no effect on 19 productivity, but may have been influencing growth of young. Using the Elliott et al. (1996) 20 study, the NOAEL is 135 ng/kg TEQ in eggs and the corresponding LOAEL is 400 ng/kg TEQ 21 in eggs. A chronic LOAEL of 25,000 ng/kg bw/d can be derived from the oral dose of PCB-126 22 to American kestrels, which caused significantly reduced skeletal growth in hatchlings. 23 Hatchlings at this exposure dose also had decreased spleen weight, increased liver weight, and 24 lymphoid depletion in the spleen and bursa (Hoffman et al. 1996).

Black-crowned night heron pipping embryos had TEQ concentrations of 30, 622, and 272 ng/kg (Rattner et al. 2000). Benzyloxyresorufin-O-deethylase (BROD) and 7-ethoxyresorufin-Odeethylase (EROD) activity were elevated at the 272 and 622 ng/kg TEQ concentration. Elliott et al. (1989) found that a TEQ of 230 ng/kg in the eggs of great blue herons caused reduced reproductive success. The same study found TEQ concentrations of 11, 14, 34, 64, and 79 ng/kg in eggs to have no effect on hatching success. The NOAEL for TEQ in eggs is 79 ng/kg, and the
 LOAEL is 230 ng/kg TEQ in eggs.

3 The dose-response curve for TEQ is derived using the results of Khera and Ruddick (1973) and 4 Sparschu et al. (1971). Khera and Ruddick (1973) treated pregnant Wistar rats with several 5 doses of TEQ on gestation days 6 to 15. Animals were sacrificed on day 22 of gestation. A 6 dose-related decrease in live fetuses was observed; 100% embryonic lethality was reported when 7 animals were exposed to a dose of 4,000 ng TEQ/kg bw/d. Sparschu et al. (1971) made similar 8 observations in Sprague Dawley rats fed several doses of TCDD on days 6 to 15 of gestation. 9 The number of viable fetuses decreased and the total number of resorptions increased dose 10 dependently, starting at 125 ng TEQ/kg bw/d.

11 **11.3.3 Effects Metrics for Characterizing Risk**

Effects data can be characterized and summarized in a variety of ways ranging from benchmarks designed to be protective of most or all species to concentration- or dose-response curves. A summary of the decision criteria used to derive effects metrics is provided in the text box. Further details on the decision criteria used in selecting effects metrics is provided in Section 6.6 of the ERA.

In this ERA, data were available to derive dose-response curves using surrogate mammals for the
small-footed myotis. Toxicity threshold ranges were developed for bald eagles and American
bittern.

20 11.3.3.1 Effects of tPCBs to Bald Eagle

American kestrels can be considered as a surrogate species for bald eagles when evaluating toxicity studies. A daily intake rate of 7 mg/kg bw/d was shown to cause an increase in laying lag, and a decrease in the number of fledglings per breeding pair of kestrels (Fernie et al. 2001a and b). These birds had a long exposure period (100 days) and the study covered a sensitive life stage. Therefore, this dose is considered to be the LOAEL for tPCBs. A chronic NOAEL was estimated by applying a factor of 10 to the LOAEL, resulting in a NOAEL of 0.7 mg/kg bw/d. This NOAEL is used as the toxicity threshold for bald eagles.
1	Decision Critoria for Derivation of Effects Matric
1	Decision Criteria for Derivation of Effects Wetric
2 3	The following is the hierarchy of decision criteria used to characterize effects for each receptor-COC combination:
4 5 6	 Have single-study bioassays with five or more treatments been conducted on the receptor of interest or a reasonable surrogate? If yes, estimate the concentration- or dose-response. If not, go to 2.
7 8 9 10	 Are multiple bioassays with similar protocols, exposure scenarios and effects metrics available that, when combined, have five or more treatments for the receptor of interest or a reasonable surrogate? If yes, estimate the dose- response relationship as in 1. If not, go to 3.
11 12 13	 Have bioassays with less than five treatments been conducted on the receptor of interest or a reasonable surrogate? If yes, conduct or report results of hypothesis testing to determine the NOAEL and LOAEL. If not, go to 4.
14 15 16 17	 Are sufficient data available from field studies and monitoring programs to estimate concentrations or doses of the COC that are consistently protective or associated with adverse effects? If yes, develop field-based effects metrics. If not, go to 5.
18 19 20 21 22 23 24	5. Derive a range where the threshold for the receptor of interest is expected to occur. Because information on the sensitivity of the receptor of interest is lacking, it is difficult to derive a threshold that is neither biased high or low. If bioassay data are available for several other species, however, calculate a threshold for each to determine a threshold range that spans sensitive and tolerant species. That range is likely to include the threshold for the receptor of interest.
۲ <i>۲</i>	

26 Wiemeyer et al. (1993) reported a significant reproductive decline in bald eagles with egg 27 concentrations greater than 13 mg/kg. However, PCB concentrations were highly correlated 28 with DDE concentrations, thus this threshold must be considered with caution. A threshold 29 value of 20 mg/kg in bald eagle eggs was suggested in the recent assessment of the Fox 30 River/Green Bay system (Stratus 1999). That value is consistent with other raptor studies that 31 suggest tPCBs have higher egg thresholds for reproductive effects than does DDE (Helander et 32 al. 1982; Peakall et al. 1990; Nobel and Elliott 1990). Therefore, the field-based threshold 33 selected for tPCB in bald eagle eggs was 20 mg/kg. If the probability of exceeding the toxicity 34 threshold was less than 20%, the risk to T&E species was considered to be low. If the probability of exceeding the toxicity threshold was greater than 50%, the risk to T&E species 35 was considered to be high. All other outcomes are considered to have intermediate risk. 36

1 11.3.3.2 Effects of TEQ to Bald Eagle

2 A chronic LOAEL of 25,000 ng/kg bw/d can be derived from the oral dose of PCB-126 to 3 American kestrels, which caused significantly reduced skeletal growth in hatchlings. Hatchlings 4 at this exposure dose also had decreased spleen weight, increased liver weight, and lymphoid 5 depletion in the spleen and bursa (Hoffman et al. 1996). This dose is considered the chronic 6 LOAEL. A dose of 5,000 ng/kg bw/d TEQ did not produce any adverse effects on American 7 kestrel chicks, and therefore, is the NOAEL. This NOAEL is used as the toxicity threshold for 8 bald eagles exposed to TEQ. Using the Elliott et al. (1996) study, the toxicity threshold for TEQ 9 in eggs is the NOAEL of 135 ng/kg.

10 **11.3.3.3** Effects of tPCBs to American Bittern

There were insufficient data available to develop a field-based threshold for American bitterns exposed to tPCBs. In the absence of such data, the threshold range estimated for sensitive and tolerant species, developed for piscivorous birds (Appendix H), exposed to tPCBs was applied. For American bitterns, the NOAEL for sensitive species (0.12 mg/kg bw/d) was the lower toxicity threshold, and the NOAEL for tolerant species (0.7 mg/kg bw/d) was the upper toxicity threshold.

This review indicates that the threshold for toxic effects to herons is in the range of 4 to >6 mg/kg PCBs in eggs. American bitterns can be reasonably represented by black-crowned night and great blue herons. For this assessment, a NOAEL of 4.9 mg/kg PCBs in eggs was selected for American bitterns.

21 11.3.3.4 Effects of TEQ to American Bittern

There were insufficient data to develop a field-based threshold for American bitterns exposed to TEQ. In the absence of such data, a range of toxic effects was estimated to span the range for sensitive to tolerant avian species, as was done for piscivorous birds (Appendix H). The threshold range estimated for American bittern exposed to TEQ was 14 ng/kg bw/d to 5,000 ng/kg bw/d.

Elliott et al. (1989) found that a TEQ of 230 ng/kg in the eggs of great blue herons caused reduced reproductive success. The same study found TEQ concentrations of 11, 14, 34, 64, and 79 ng/kg in eggs to have no effect on hatching success. The NOAEL selected for TEQ in eggs
 was 79 ng/kg and the LOAEL was 230 ng/kg TEQ in eggs. The toxicity threshold for American
 bittern eggs exposed to TEQ is the NOAEL, 79 ng/kg.

4 11.3.3.5 Effects of tPCBs to Small-Footed Myotis

5 The Spencer (1982) study was used for the derivation of a dose-response curve based on 6 mortality at birth. Figure 11.3-1 presents the dose-response curve for mortality of rats at birth. 7 The dose-response curve indicates that 10% and 20% declines in mortality at birth would be 8 expected at doses of 3.05 and 5.37 mg/kg bw/d, respectively.



10Figure 11.3-1Dose-Response Curve for Effects of tPCBs on Mortality at Birth of11Rats

12 **11.3.3.6 Effects of TEQ to Small-Footed Myotis**

Because of the similarity of the protocols, the Khera and Ruddick (1973) and Sparschu et al. (1971) studies were combined for the derivation of a dose-response curve based on reproductive effects. Figure 11.3-2 presents the dose-response curve for reproductive fecundity of rats exposed to TEQ. The dose-response curve indicates that 10% and 20% declines in reproductive fecundity would be expected at doses of 156 and 330 ng/kg bw/d TEQ, respectively.



1 11.4 RISK CHARACTERIZATION

This section characterizes risk to T&E species exposed to tPCBs and TEQ in the PSA of the Housatonic River. The risk characterization discusses two potential lines of evidence, field surveys and modeled exposure and effects, to determine potential ecological risks to T&E species. The key risk questions and the two potential lines of evidence are summarized in the text box.

7		Key Risk Questions
8 9 10	•	Are the concentrations of tPCBs and TEQ present in the prey of T&E species sufficient to cause adverse effects to individuals inhabiting the PSA of the Housatonic River?
11	•	If so, how severe are the risks and what are their potential consequences?
12		Lines of Evidence
13 14	•	Use of qualitative field surveys (not considered in the weight-of-evidence analysis).
15	•	Probabilistic exposure and effects modeling.

16

Section 11.4.1 presents a brief overview of the methodology, results, and interpretation of the bird and bat surveys conducted from 1998 to 2001 in the Housatonic PSA. A more detailed presentation of this information is provided in Appendix A. In Section 11.4.2, the dose-response curves are combined with the corresponding exposure distributions to derive risk curves that characterize the relationship between probability and magnitude of effect. A weight-of-evidence analysis is presented is Section 11.4.3 along with sources of uncertainty (Section 11.4.4) and the overall findings of the risk assessment (Section 11.4.5).

24 11.4.1 Field Survey

T&E species in the Housatonic River study area were surveyed from 1998 to 2001. Field data were collected using methods targeted at specific species or family groups, as well as more general, reconnaissance-level investigations of species' presence, relative abundance, and habitat use. Surveys for T&E species were conducted as part of broader survey efforts. Throughout this period, any observations were recorded along with notes on habitat use, breeding signs, and behavior.

The avian community in the PSA was studied over a 4-year period, from 1998 to 2001. Surveys 1 2 were conducted to record presence, abundance, and habitat usage for each major group of birds. 3 These surveys included wading and marsh bird surveys, hawk and owl surveys, and forest bird 4 surveys. Additional studies were conducted to sample animal tissues (i.e., waterfowl sampling, 5 tree swallow study). Observations recorded in the field were used to refine the matrix to depict 6 habitat use and seasonality of occurrence. Marsh and wading bird surveys were conducted in 7 1998 using playback point counts to identify species using the PSA wetlands and reference areas 8 (Appendix A). Playback point counts were also used in 1999 to survey hawks and owls (raptors) 9 in the PSA and in three reference areas. In the PSA, raptor transects were positioned along the 10 Housatonic River from the confluence of the East and West Branches to Woods Pond (Appendix 11 A).

12 The mammalian community in the PSA was studied from 1998 to 2001. Field data included 13 methods targeted at specific species, as well as more general, reconnaissance-level investigations 14 of species presence, relative abundance, and habitat use.

15 Bat surveys were conducted to determine presence in the PSA by recording their echolocation 16 calls. Three transects were established along the river in the northern, central, and southern 17 sections of the PSA (Appendix A). There is a large amount of overlap between the call 18 characteristics of the little brown bat, small-footed myotis, and Indiana bat, which makes it 19 difficult to distinguish between these *Myotis* species using echolocation. When recording the 20 results, these three species were all labeled as *Myotis* sp. The majority of these calls were likely 21 little brown bat; however, a small number of the calls had parameters that suggested small-footed 22 myotis rather than little brown bats or Indiana bats. Small-footed myotis cannot be confirmed 23 without having animals in hand for visual identification.

Bald eagles were not observed during raptor surveys in the PSA or any of the three reference areas. However, incidental bald eagle observations were made in the PSA (primarily in the vicinity of Woods Pond) and at the Threemile Pond reference area. The PSA provides suitable nesting and foraging habitat for bald eagles. In the mid 1990s, a pair of bald eagles constructed a nest at Woods Pond (T. Gulo, MDFW, personal communication 2001). The nest was reportedly destroyed during an April snowstorm and the pair did not attempt to re-nest. In 2001, a bald eagle pair nested along the Housatonic River in Connecticut, below Interstate 84, and raised one chick. The pair returned in 2002 and displayed breeding activity (i.e., adding sticks to previous
 year's nest); however, no nesting was observed (J. Bictoria, CTDEP, personal communication
 2002).

American bitterns were not observed during marsh bird surveys in the PSA, and no marsh bird surveys were conducted outside of the PSA. Incidental observations of American bitterns occurred in the PSA and at Washington Mountain Lake during the breeding season, and one individual was heard calling in the PSA, indicating intent to breed in the area. These observations were incidental observations, occurring while researchers were on-site for other surveys. During timed bird surveys (i.e., playback surveys), the results in the PSA were the same for reference areas; no bald eagles or American bitterns were observed in either location.

11 Small-footed myotis observations in the PSA have not been confirmed, and no bat surveys were 12 conducted in reference areas. Suitable summer habitat for small-footed myotis is present in and 13 adjacent to the study area, and it is likely that the species occurs there. The small-footed myotis 14 has been recorded in western Massachusetts and has been documented twice since 1978 in 15 Hampden County, MA (MNHESP 1984; Godin 1977), making their presence in the study area 16 possible. It is believed that this species was recorded during bat surveys; however, as previously 17 mentioned, limitations of echolocation technology prevent this species from being definitively 18 identified. Other studies conducted in the region have reported small-footed myotis observations 19 (Zimmerman and Glanz 2000; Krusic et al. 1996).

Any differences in population structure between PSA and reference locations cannot be evaluated due to the overall low population size of T&E species, few numbers of sightings, and qualitative study design. The number of observations for these species by definition is expected to be low and the lack of observations in one location does not necessarily reflect the suitability of that habitat or the absence of an individual.

25 11.4.2 Comparison of Estimated Exposures to Laboratory-Derived Effects 26 Doses

For the bald eagle, exposure was assessed for the southern PSA, while exposure of American bittern was estimated for each reach, and exposure to small-footed myotis was estimated for the entire PSA. For each receptor-COC combination, a category of low, intermediate, or high risk was assigned using the guidance in the text box following integration of the exposure and effects distributions. This exercise was done separately for the results of the Monte Carlo analyses and each of the lower and upper bounds from the probability bounds analyses. The "risk category" refers to the level of risk based on the results of the Monte Carlo analyses. The "risk range" refers to the levels of risk based on the results of the probability bounds analyses. The toxicity thresholds or the 10% and 20% effects doses for each species and COC are presented in Section 11.3.

8			Guidance for Determining Level of Risk to Bald Eagle
9 10		•	If the probability of exceeding the toxicity threshold was less than 20%, the risk to bald eagle was considered to be low.
11 12		•	If the probability of exceeding the toxicity threshold was greater than 50%, the risk to bald eagle was considered to be high.
13		•	All other outcomes are considered to have intermediate risk.
14			
15			Guidance for Determining Level of Risk to American Bittern
16 17		•	If the probability of exceeding the lower toxicity threshold was less than 20%, the risk to American bittern was considered to be low.
18 19		•	If the probability of exceeding the upper toxicity threshold was greater than 20%, the risk to American bittern was considered to be high.
20		•	All other outcomes are considered to have intermediate risk.
21			
22			Guidance for Determining Level of Risk to Small-Footed Myotis
23 24		•	If the probability of 10% or greater effect is less than 20%, then the risk to small- footed myotis is low.
25 26		•	If the probability of 20% or greater effect is greater than 50%, then the risk to small-footed myotis is high.
27		•	All other outcomes are considered to have intermediate risk.
28 29	The r	esul	ts of the risk characterization are summarized in Table 11.4-1. Figures 11.4-1 th
30	11.4-	4 ar	e risk curves for bald eagles exposed to tPCBs and TEQ in the PSA. Figures

31 through 11.4-14 show American bittern exposed to tPCBs and TEQ in the PSA, and Figures

32 11.4-15 and 11.4-16 show small-footed myotis exposed to tPCBs and TEQ in the PSA.

Table 11.4-1

Summary of Qualitative Risk Statements for T&E Species from the Housatonic **River Study Area**

	Qualitative Risk Statements					
Bird / Location	tPC	Bs	TEQ			
	Risk Category ^a	Risk Range ^b	Risk Category ^a	Risk Range ^b		
Bald Eagle						
Southern PSA	High	High	Low	Low		
American Bittern						
Reach 5A	High	High	Intermediate	Intermediate		
Reach 5B	High	High	Intermediate	Intermediate		
Reach 5C	High	High	Intermediate	Intermediate		
Reach 5D and 6	High	High	Intermediate	Intermediate		
Small-Footed Myotis						
Reaches 5 and 6	High	Intermediate - High	High	Low - High		

5 6 ^aRisk category is the risk level based on First Order Monte Carlo (FOMC).

^bRisk range is the range of risk encompassed by the upper and lower probability bounds (UPB and LPB).



Figure 11.4-1 Risk Curves for Bald Eagles Exposed to tPCBs in the Housatonic **River Primary Study Area**



Figure 11.4-2 Risk Curves for Bald Eagles Exposed to TEQ in the Housatonic 6 **River Primary Study Area** 7

MK01\O:\20123001.096\ERA_PB\ERA_PB_11.DOC



Primary Study Area

tPCB egg concentration (mg/kg)



Primary Study Area





Figure 11.4-4 Risk for Bald Eagle Eggs Exposed to TEQ in the Housatonic River Primary Study Area



Figure 11.4-5
 Risk Curves for American Bittern Exposed to tPCBs in Reach 5A
 of the Housatonic River Primary Study Area





Figure 11.4-6 Risk Curves for American Bittern Exposed to tPCBs in Reach 5B of the Housatonic River Primary Study Area



Figure 11.4-7 Risk Curves for American Bittern Exposed to tPCBs in Reach 5C of the Housatonic River Primary Study Area



Reaches 5D & 6









 Figure 11.4-9
 Risk Curves for American Bittern Exposed to TEQ in the Housatonic River Primary Study Area



4 5 6

Figure 11.4-10 Risk for American Bittern Eggs Exposed to tPCBs in Reach 5A of the Housatonic River Primary Study Area



Figure 11.4-11 Risk for American Bittern Eggs Exposed to tPCBs in Reach 5B of the Housatonic River Primary Study Area







Figure 11.4-12 Risk for American Bittern Eggs Exposed to tPCBs in Reach 5C of the Housatonic River Primary Study Area





Figure 11.4-13 Risk for American Bittern Eggs Exposed to tPCBs in Reaches 5D and 6 of the Housatonic River Primary Study Area

Primary Study Area





Figure 11.4-14 Risk for American Bittern Eggs Exposed to TEQ in the Housatonic River Primary Study Area



Figure 11.4-15 Risk Curves for Small-Footed Myotis Exposed to tPCBs in Reach 5 of the Housatonic River Primary Study Area



4 5 6

Figure 11.4-16 Risk Curves for Small-Footed Myotis Exposed to TEQ in Reach 5 of the Housatonic River Primary Study Area

The results of the risk characterization showed that the highest risk to T&E species is to bald 1 2 eagles and American bitterns from exposure to tPCBs. The risk for bald eagles exposed to TEQ 3 was low; however, risk to bald eagle eggs exposed to TEQ was high. The analysis for bald 4 eagles associated with exposure to tPCBs downstream of Woods Pond indicated that bald eagles 5 would only potentially be at risk in Reach 8 (Rising Pond). The risk to bald eagles nesting and 6 wintering downstream of the PSA is low. The risk category for American bittern was 7 intermediate for TEQ and high for eggs exposed to TEQ. The risk category for small-footed 8 myotis was high for both tPCB and TEQ. The risk range for small-footed myotis, as determined 9 by the probability bounds analysis, ranged from intermediate to high for tPCBs and low to high 10 for TEQ.

11 11.4.3 Weight-of-Evidence Analysis

A weight-of-evidence analysis was used to evaluate the lines of evidence described in the 12 13 preceding sections for T&E species. The goal of this analysis was to determine whether 14 significant risk is posed to T&E species in the Housatonic River PSA as a result of exposure to 15 tPCBs and TEQ. The three-phase approach of Menzie et al. (1996) and the Massachusetts 16 Weight-of-Evidence Workgroup was applied for this purpose, in which weight-of-evidence was 17 reflected in the following three characteristics: (a) the weight assigned to each measurement 18 endpoint, (b) the magnitude of response observed in the measurement endpoint, and (c) the 19 concurrence among outcomes of the multiple measurement endpoints. As noted previously, field 20 surveys were qualitative and therefore not used in this analysis.

The rationale for weighting of measurement endpoints is provided in Appendix K, along with a discussion of attributes considered in the weight-of-evidence. A summary of how attributes were weighted for the bald eagle, American bittern, and small-footed myotis lines of evidence is provided in Table 11.4-2. For both tPCBs and TEQ, the modeled exposure and effects lines of evidence were given a moderate/high value.

Table 11.4-2

Weighting of Measurement Endpoints for T&E Species Weight-of-Evidence Evaluation

Attributes	Modeled Exposure and Effects for Bald Eagles Exposed to tPCBs and TEQ	Modeled Exposure and Effects for American Bitterns Exposed to tPCBs and TEQ	Modeled Exposure and Effects for Small- Footed Myotis Exposed to tPCBs and TEQ	Rationale
I. Relationship Between Measur	rement and Assessment En	dpoints		
1. Degree of Association	H (tPCBs) M/H (TEQ)	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Exposure models were species-specific, but effects metrics for bald eagle (body burden), American bitterns, and small- footed myotis were derived from studies of surrogate species. Effects metrics for bald eagle eggs were species- specific for tPCBs.
2. Stressor/Response	M/H (tPCBs) M (TEQ)	M (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Exposure modeling was species- and stressor-specific. Effects metrics for representative species were available only for bald eagle eggs exposed to tPCBs. A dose- response curve was used for small-footed myotis, rather than thresholds.
3. Utility of Measure	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Modeled exposure and effects procedures used were standardized and widely accepted; the primary limitation was lack of species-specific effects data, except for bald eagle eggs exposed to tPCBs.
II. Data Quality				
4. Data Quality	H (tPCBs and TEQ)	H (tPCBs and TEQ)	H (tPCBs and TEQ)	The field surveys were performed according to well- defined and documented protocols. The low numbers of individuals observed, and the inability to confirm identification of the Myotis without handling, limited the ability to observe site-specific effects. The DQOs for the sampling analysis and tissue samples were met for the tissue residue data used in the exposure analysis for both tPCBs and TEQ.

Table 11.4-2

Weighting of Measurement Endpoints for T&E Species Weight-of-Evidence Evaluation (Continued)

Attributes	Modeled Exposure and Effects for Bald Eagles Exposed to tPCBs and TEQ	Modeled Exposure and Effects for American Bitterns Exposed to tPCBs and TEQ	Modeled Exposure and Effects for Small- Footed Myotis Exposed to tPCBs and TEQ	Rationale
III. Study Design				
5. Site Specificity	M (tPCBs and TEQ)	M (tPCBs and TEQ)	M (tPCBs and TEQ)	Biological tissue data used in exposure models were site specific, and other exposure parameters were representative of site conditions. However, effects measures were not site specific.
6. Sensitivity	M/H (tPCBs) H (TEQ)	H (tPCBs and TEQ)	H (tPCBs and TEQ)	Modeled exposure and effects directly assessed exposure- response relationship. Laboratory studies from which effects data were derived were stressor-specific.
7. Spatial Representativeness	H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	H (tPCBs and TEQ)	Modeled exposures relied on tissue data collected throughout the study area and areas of actual exposure. American bittern exposure based on small mammals trapped in unfavorable foraging area.
8. Temporal Representativeness	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Modeled exposure and effects lines of evidence spanned critical life stages and, in general, tissue data used were collected when exposure was expected to be high.
9. Quantitative Measure	H (tPCBs and TEQ)	H (tPCBs and TEQ)	H (tPCBs and TEQ)	Probabilistic exposure and effects modeling were highly quantitative and propagated uncertainty associated with modeling procedures.
10. Standard Method	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	Generally accepted exposure and effects modeling procedures were followed, but probability bounds analysis is a relatively new technique for propagating uncertainty.
Overall Endpoint Value	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	M/H (tPCBs and TEQ)	

M = Moderate

H = High

2 3

1 The magnitude of the response in the measurement endpoint is considered together with the 2 measurement endpoint weight in judging the overall weight-of-evidence (Menzie et al. 1996). 3 This requires assessing the strength of evidence that ecological harm has occurred, as well as an 4 indication of the magnitude of response, if present. For bald eagles and American bitterns, 5 exposure and effects for both tPCBs and TEQ were estimated for body burden and eggs 6 separately. In the weight-of-evidence analysis, the risks for both of these factors were combined 7 and presented together because ecologically if there is risk to either life stage, there is risk to the 8 organism.

9 The results from the modeled exposure and effects line of evidence indicate that there is no 10 evidence of harm to adult bald eagles exposed to TEQ in the PSA, but high risk to bald eagle 11 eggs. There is, however, evidence of harm to bald eagles and American bitterns exposed to 12 tPCBs, and an undetermined risk for American bittern exposed to TEQ and small-footed myotis 13 exposed to tPCBs and TEQ in the PSA.

The weighting, evidence of harm, and magnitudes of responses were combined in a matrixformat and are presented in Tables 11.4-3 and 11.4-4.

16

17

Table 11.4-3

Evidence of Harm and Magnitude of Effects for T&E Species Exposed to tPCBs in Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled exposure and effects, Bald Eagle	Moderate/High	Yes	High
Modeled exposure and effects, American Bittern	Moderate/High	Yes	High
Modeled exposure and effects, Small-Footed Myotis	Moderate/High	Undetermined	High

Table 11.4-4

2 3 4

1

Evidence of Harm and Magnitude of Effects for T&E Species Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled exposure and effects, Bald Eagle	Moderate/High	Yes	Intermediate
Modeled exposure and effects, American Bittern	Moderate/High	Undetermined	High
Modeled exposure and effects, Small-Footed Myotis	Moderate/High	Undetermined	High

5

6 A graphical method was used for displaying concurrence among measurement. Tables 11.4-5

7 and 11.4-6 depict the outcome for T&E species exposed to tPCBs and TEQ, respectively. The

8 field survey line of evidence was not included as it is inconclusive.

9

10

11 12

13

Risk Analysis Summary for T&E Species Exposed to tPCBs in the Housatonic River PSA

Table 11.4-5

	We	eighting Factors	(increasing co	nfidence of weig	ght)
Harm/Magnitude	Low	Low/ Moderate	Moderate	Moderate/ High	High
Yes/High				BE, AB	
Yes/Intermediate					
Yes/Low					
Undetermined/High				SFM	
Undetermined/Intermediate					
Undetermined/Low					
No/Low					
No/Intermediate					
No/High					

14 BE = bald eagle

15 AB = American bittern

16 SFM = small-footed myotis

4

5

Risk Analysis Summary for T&E Species Exposed to TEQ in the Housatonic River PSA

Table 11.4-6

Weighting Factors (increasing confidence of weig				nfidence of weight)	
Harm/Magnitude	Low	Low/Moderate	Moderate	Moderate/High	High
Yes/High					
Yes/Intermediate				BE	
Yes/Low					
Undetermined/High				AB, SFM	
Undetermined/Intermediate					
Undetermined/Low					
				·	
No/Low					
No/Intermediate					
No/High					

6

BE = bald eagle

7 AB = American bittern

8 SFM = small-footed myotis

9

10 11.4.4 Sources of Uncertainty

11 The assessment of risk to T&E species contains uncertainties. Each source of uncertainty can 12 influence the estimates of risk. Therefore, it is important to describe and, when possible, specify 13 the magnitude and direction of such uncertainties. Some of the major sources of uncertainty 14 associated with the assessment of risks of tPCBs and TEQ to T&E species are briefly described 15 below. A more complete list is presented in Appendix K.

16 The Monte Carlo sensitivity analyses suggest that the FMR slope and power terms were generally the most influential variables on predicted total daily intakes of COCs. 17 18 However, no measurements of free metabolic rate were available for the 19 representative wildlife species. Similarly, measured food intake rates were not 20 available for bald eagles, American bitterns, small-footed myotis, or reasonable

surrogate species. Therefore, free metabolic rates were estimated using allometric equations. The use of allometric equations introduces some uncertainty into the exposure estimates because they have model-fitting error, and are based on species different from the representative species used in this assessment. Given the lack of data on representative species used in the current assessment, it is difficult to judge the magnitude of the uncertainty introduced by the use of the allometric models. The uncertainty due to model-fitting error was propagated in the uncertainty analyses by using distributions as input for the allometric slope and power terms.

- 9 Sample sizes were limited for the analyses of COC concentrations in some prey 10 items. Only two to four invertebrate samples for Reaches 5B, 5C, and 6 were available to estimate exposure of American bittern to PCBs. Uncertainty due to 11 sample size was explicitly addressed in the uncertainty analyses. In the Monte Carlo 12 13 analysis, sample size uncertainty was addressed by use of the 95% UCL on the mean. Use of the UCL addressed uncertainty, but is biased towards overestimating 14 In the probability bounds analysis, uncertainty was addressed by 15 exposure. 16 specifying concentration variables as a range from the 95% LCL to the 95% UCL. 17 This treatment of uncertainty is unbiased.
- PCB congeners 123 and 157 co-eluted with other congeners (PCB-123 with PCB-18 19 149; PCB-157 with PCB-173 and PCB-201) leading to uncertainty about TEQ 20 concentrations in prey sample. This source of uncertainty was addressed in the 21 uncertainty analyses by estimating prey concentrations assuming concentrations of 22 PCB-123 and PCB-157 were equal to zero, and assuming that concentrations of PCB-23 123 and PCB-157 were equal to the doublet and triplet concentrations, respectively. 24 The resulting TEQ estimates were then compared. If the ratio of the upper to lower bound TEQ estimates was less than 1.3, this source of uncertainty was deemed 25 unimportant and disregarded. If the ratio exceeded 1.3, the uncertainty due to the co-26 27 elution was propagated through the uncertainty analyses.
- The adult body burden and associated egg concentration was estimated assuming that avian species do not metabolize PCBs, to simplify the estimated accumulation by bald eagles. This assumption may result in an overestimate of the amount of PCBs accumulated.
- 32 The adult body burden was estimated assuming that a breeding adult would arrive in 33 the PSA with no tPCBs in the body. New breeding pairs colonizing the PSA would 34 be expected to have low concentrations of tPCBs in their tissue. However, eagles 35 return to the same breeding area, and often the same nest, each year. Bald eagles returning to the PSA during subsequent years would have COC body burdens 36 37 accumulated during previous breeding seasons. Therefore, body burdens and egg 38 concentrations are likely underestimated for eagles that have previously bred in the 39 PSA.
- The largest source of uncertainty in the effects assessment was associated with the
 lack of, or limited, toxicity studies involving the representative species. There were
 no toxicity studies available for American bittern and small-footed myotis exposed to

1

2

3

4

5

6 7

1 tPCBs or TEQ. As a result, laboratory studies involving surrogate species were used 2 to estimate effects to these species. These extrapolations introduced uncertainty in 3 the effects assessment because of the variation in sensitivities of species to tPCBs and 4 TEQ. The sensitivity of wildlife to an environmental contaminant may also differ 5 from that of a laboratory or domestic species due to behavioral and ecological 6 parameters including stress factors (e.g., competition, seasonal changes in 7 temperature, or food availability), disease, and exposure to other contaminants.

- For T&E species, data for two potential lines of evidence were available. For these assessments, toxicity studies performed in situ in the PSA of the Housatonic River or feeding studies involving prey and food items from the PSA would have improved the weight-of-evidence assessment. Such studies would have accounted directly for the specific characteristics of the Housatonic River ecosystem and the toxicity of the PCB mixture found on site.
- 14 **11.4.5 Conclusions**
- 15 **11.4.5.1** Risks in the PSA

The weight-of-evidence analysis indicates that T&E species such as bald eagles, American bitterns, and small-footed myotis are at some risk in the PSA as a result of exposure to tPCBs and TEQ. The risks for bald eagles and American bitterns exposed to tPCBs are high. The risk for small-footed myotis exposed to tPCBs and TEQ are undetermined.

20 21

22

23

24

25

26

ERA Results for Representative T&E Species

The weight-of-evidence analysis indicates that T&E species such as bald eagle and American bittern are at risk in the PSA as a result of exposure to tPCBs. Risks to bald eagles and American bittern exposed to tPCBs are high. There are intermediate risks to bald eagles exposed to TEQ, and risks to American bittern exposed to TEQ are undetermined. Risks to small-footed myotis exposed to tPCBs and TEQ are undetermined.

27

The bald eagle, American bittern, and small-footed myotis were chosen to represent T&E species inhabiting the Housatonic River PSA. Other T&E species that occur in the area include one mussel (triangle floater); three dragonflies (riffle snaketail, zebra clubtail, and arrow clubtail); a turtle (wood turtle); three salamanders (Jefferson salamander, four-toed salamander, and northern spring salamander); three hawks (northern harrier, sharp-shinned hawk, and Cooper's hawk); two warblers (northern parula and blackpoll warbler); a wading bird (common moorhen); and a shrew (northern water shrew). Some of these species were assessed in other appendices, and the risks were compared to other, more appropriate assessment endpoints (i.e., salamanders assessed in
 Appendix E, Amphibians).

A qualitative analysis was conducted to compare exposure of representative species and other similar species to tPCBs and TEQ. The major factors that influence exposure to tPCBs and TEQ include the following:

6 7

8

9

- Foraging behavior and dietary composition.
- Foraging and home range size.
- Species body weight and other life history characteristics.

As noted in this ERA, effects studies conducted on bald eagles, American bittern, and smallfooted myotis are not available. Similarly, effects data are not available for other similar species living in the Housatonic River area. As a result, the surrogate effects data used to estimate effects to bald eagles were also used to estimate risk for other piscivorous raptors, data for American bittern were used for other wading birds, and data from small-footed myotis for other bat species.

16 Results are provided in the following text box.

17 18	ERA Results for Other Piscivorous Raptors, Wading Birds, and Bats Living in the PSA
19 20	The other piscivorous raptor that occurs in the PSA is the osprey. Risk to osprey is characterized in Appendix H.
21 22 23 24	Other piscivorous wading bird species that could occur in the PSA include the least bittern, green heron, great blue heron, king rail, least rail, sora, and pied-billed grebe. A qualitative analysis of risk to these species indicates that the great blue heron and king rail are expected to have a similar level of risk compared to the American bittern.
25 26 27 28 29	The wading birds that have similar diets but are smaller and have higher metabolisms—such as least bittern, green heron, Virginia rail, and pied-billed grebe—are expected to have a higher level of risk than the American bittern. Wading birds that consume plant material, such as the sora, are expected to have low levels of risk.
30 31 32	Other bat species, especially those in the myotis family (little brown bat, Indiana bat, and northern myotis) are expected to have a similar level of risk as the small-footed myotis.

1 **11.4.5.2** *Risk Estimates Downstream of Woods Pond*

Risks to bald eagles due to contaminants in the river and floodplain below Woods Pond were
also assessed. Total PCBs measured in sediment, floodplain soil, and fish tissue from the river
above and below Woods Pond are presented in Appendix H. The data indicate that
contamination in these media declines substantially below Woods Pond Dam.

6 **11.4.5.2.1** Risk for Bald Eagles Wintering Downstream of Woods Pond

7 The risk for bald eagles from exposure to tPCBs downstream of Woods Pond was assessed by 8 comparing concentrations of tPCBs in prey fish in Reaches 7 to 16 to a maximum acceptable 9 threshold concentration (MATC) developed specifically for bald eagles. The MATC of 30.41 10 mg/kg tPCBs in fish (whole body, wet weight) was developed as the concentration at which bald 11 eagle TDI would exceed the toxicity threshold for eggs. The TDI was calculated assuming that 12 eagles wintering downstream of Woods Pond would consume 83.4% fish and 16.1% waterfowl 13 (Stalmaster and Plettner 1992). The waterfowl concentration was assumed to be zero, as 14 waterfowl wintering on the Housatonic are likely to have migrated there from northern locations 15 outside the study area. The results of the analysis that indicate that bald eagles would be at risk 16 only in Reach 8 (Rising Pond), are presented in Figure K.4-5. This conclusion is conservative, in 17 that it assumes bald eagles would consume fish only from Rising Pond. However, this is 18 unlikely because Rising Pond is considerably smaller than a typical bald eagle foraging area.

19 **11.4.5.2.2** Risk for Bald Eagles Breeding Downstream of Woods Pond

Figure K.4-5 presents the assessment of risk to bald eagles exposed to tPCBs downstream of Woods Pond. Bald eagles are known to breed downstream of Woods Pond. In particular, one bald eagle pair nested and raised one chick in Reach 15, just south of Interstate 84, in 2001. In 2002, the pair returned to the nest and displayed breeding activity but did not nest (J. Bictoria, CTDEP, personal communication 2002).

Risk from exposure to tPCBs was estimated for bald eagles nesting at this location. Bald eagles
have a linear (riverine) foraging distance of 1.9 to 4.3 miles (3.1 to 6.9 km) (Craig et al. 1988).

Therefore, bald eagles nesting near Interstate 84 could potentially be foraging in Reach 15 (Lake
 Zoar) and the southern section of Reach 14 (Lake Lillinonah).

3 Total PCB concentrations for prey items from Reaches 14 and 15 were available only for fish. 4 Because of the small number of fish samples for Reaches 14 and 15, all fish were combined 5 instead of separating them into classes (i.e., predatory fish, bottom feeder, forage fish). Fish in 6 Reaches 14 and 15 had an average concentration of 0.717 mg/kg. Bald eagles on average 7 consume a summer diet consisting of 78.6% fish, 16.8% birds, and 5.1% mammals (see 8 Appendix K). Mammal and bird tPCB concentrations were not available for downstream 9 reaches. Total PCB concentrations for these prey items were estimated in three ways to give high, moderate, and low concentrations. High concentrations assumed that waterfowl and 10 11 mammals from downstream would have tPCB concentrations equal to those in the PSA. Low 12 concentrations assumed that waterfowl and mammals from downstream would have tPCB 13 concentrations of zero. A moderate concentration was developed by determining fish-to-14 mammal and fish-to-bird ratios based on concentrations in the PSA. Mammal tPCB 15 concentrations in the PSA are on average 75% of the total fish concentration, and waterfowl 16 tPCB concentrations averaged 15% of the total fish concentration. Therefore, moderate tPCB 17 concentrations downstream were 0.538 mg/kg for mammals and 0.108 mg/kg for birds.

The estimated low, moderate, and high tPCB intake rates averaged 0.022 mg/kg bw/d, 0.025
mg/kg bw/d, and 0.243 mg/kg bw/d, respectively. These values fall below the lower toxicity
threshold of 0.7 mg/kg bw/d.

Risks from TEQ to adult bald eagles in the PSA were intermediate. Because TEQ concentrations
in downstream prey species are reduced to the same degree as PCB concentrations, it is assumed
that risk from TEQ to bald eagles breeding downstream would be low.

1 11.5 REFERENCES

- Anthony, E.P.L. and T.H. Kunz. 1977. Feeding strategies of the little brown bat in southern New
 Hampshire. *Ecology* 58:775-786.
- 4 Belwood, J.J. and M.B. Fenton. 1976. Variation in the diet of Myotis lucifugus. *Canadian* 5 *Journal of Zoology* 54:1674-1678.
- Bictoria, J. 2002. Connecticut Department of Environmental Protection. Personal
 Communication to Woodlot Alternatives, Inc. 12 December 2002.
- Borga, K., G.W. Gabrielsen, and J.U. Skaare. 2001. Biomagnification of organochlorines along a
 Barents sea food chain. *Environmental Pollution* 113(2):187-198.
- 10 Bowerman, W.W., J.P. Giesy, D.A. Best, and V.J. Kramer. 1995. A review of factors affecting
- 11 productivity of bald eagles in the Great Lakes region: Implications for recovery. *Environmental*
- 12 *Health Perspectives* 103(Suppl 4):51-59.
- 13 Buehler, D.A. 2000. Bald Eagle (Haliaeetus leucocephalus). pp. 1-39. In Poole, A. and F. Gill
- (ed.) *The Birds of North America*. Vol. 506. The Birds of North America, Inc., Philadelphia, PA,
 USA.
- Canadian Wildlife Service. 2000. Hinterland Who's Who: Bald Eagle. URL http://www.cws scf.ec.gc.ca/hww-fap/bald/bald.html (accessed 13 May 2002).
- Clark, D.R., Jr. 1978. Uptake of dietary tPCB by pregnant big brown bats and their fetuses.
 Bulletin of Environmental Contamination and Toxicology 19:707–714.
- Clark, D.R., Jr., and C.J. Stafford. 1981. Effects of DDE and PCB (Aroclor 1260) on
 experimentally poisoned female little brown bats (*Myotis lucifugus*): Lethal brain concentrations.
 Journal of Toxicology and Environmental Health 7:925–934.
- Clark, D.R., Jr., C.O. Martin, and D.M. Swineford. 1975. Organochlorine insecticide residues in
 the free-tailed bat (*Tadarida brasiliensis*) at Braken Cave, Texas. *Journal of Mammalogy*56(2):429-443.
- Clark, D.R., Jr., and R.M. Prouty. 1976. Organochlorine residues in three bat species from four localities in Maryland and West Virginia, 1973. *Pesticides Monitoring Journal* 10:44-53.
- Clark, D.R., Jr., and R.M. Prouty. 1977. Experimental feeding of DDE and PCB to female big
 brown bats (*Eptesicus fuscus*). *Journal of Toxicology and Environmental Health* 2:917-928.
- Clark, D.R., Jr., and T.G. Lamont. 1976a. Organochlorine residues in females and nursing young of the big brown bat. *Bulletin of Environmental Contamination and Toxicology* 15(1):1-8.
- Clark, D.R., Jr., and T.G. Lamont. 1976b. Organochlorine residues and reproduction in the big brown bat. *Journal of Wildlife Management* 40(2):249-254.

- 1 Clark, D.R., Jr., T.H. Kunz, and T.E. Kaiser. 1978. Insecticides applied to a nursery colony of little
- 2 brown bats (*Myotis lucifugus*): lethal concentrations in brain tissues. Journal of Mammalogy
- 3 59:84-91.
- 4 Cottam, C. and F.M. Uhler. 1945. Birds in Relation to Fishes. U.S. Fish and Wildlife Service 5 Leaflet, No. 272.
- 6 Craig, R.J., E.S. Mitchell, and J.E. Mitchell. 1988. Time and energy budgets of bald eagles 7 wintering along the Connecticut River. *Journal of Field Ornithology* 59:22-32.
- 8 Custer, C.M. 2002. Exposure and effects of chemical contaminants on tree swallows nesting
- 9 along the Housatonic River, Berkshire Co., Massachusetts, 1998-2000. Final Report to U.S.
- 10 Environmental Protection Agency. USGS, Upper Midwest Environmental Sciences Center, La
- 11 Crosse, WI.
- 12 Custer, T.W., R.K. Hines, P.M. Stewart, M.J. Melancon, D.S. Henshel, and D.W. Sparks. 1998.
- 13 Organochlorines, mercury, and selenium in great blue heron eggs from Indiana Dunes National
- 14 Lakeshore, Indiana. Journal of Great Lakes Research 24(1):3-11.
- DeGraaf, R.M. and M. Yamasaki. 2001. New England Wildlife: Habitat, Natural History, and
 Distribution. University Press of New England, Hanover, NH, USA. 482 pp.
- Donaldson, G.M., J.L. Shutt, and P. Hunter. 1999. Organochlorine contamination in bald eagle
 eggs and nestlings from the Canadian Great Lakes. Archives of Environmental Contamination and
 Toxicology 36:70-80.
- Dunning, J.B., Jr. (ed.) 1992. CRC Handbook of Avian Body Mass. CRC Press, Boca Raton, FL,
 USA.
- Dunstan, T.C. and J.F. Harper. 1975. Food habits of bald eagles in north-central Minnesota.
 Journal of Wildlife Management 39:140-143.
- Elliott, J.E., R.W. Butler, R.J. Norstrom, and P.E. Whitehead. 1989. Environmental contaminants
 and reproductive success of great blue herons in British Columbia 1986-87. *Environmental Pollution* 59:91-114.
- 27 Elliott, J.E., R.J. Norstrom, A. Lorenzen, L.E. Hart, H. Philibert, S.W. Kennedy, J.J. Stegeman,
- G.D. Bellward, and K.M. Cheng. 1996. Biological effects of polychlorinated dibenzo-p-dioxins,
 dibenzofurans, and biphenyls in bald eagle (*Haliaeetus leucocephalus*) chicks. *Environmental Toxicology and Chemistry* 15:782-793.
- 31 Elliott, J.E., M.L. Harris, L.K. Wilson, P.E. Whitehead, and R.J. Norstrom. 2001.
- Monitoring Temporal and Spatial Trends in Polychlorinated Dibenzo-p-dioxins (PCDDs) and Dibenzofurans (PCDFs) in Eggs of Great Blue Heron (*Ardea herodias*) on the Coast of British
- 34 Columbia, Canada, 1983–1998. Ambio 30(7):416–428.
- 35 Elliott, J.E., and M.L. Harris. In Press. An ecotoxicological assessment of chlorinated 36 hydrocarbon effects on bald eagle populations. *Reviews in Toxicology*.

- 1 EPA (U.S. Environmental Protection Agency). 1993. Wildlife Exposure Factors Handbook.
- 2 Volume 1 of 2. EPA/600/R-93/187a. Office of Research and Development, Washington, DC,
- 3 USA.
- 4 Fernie, K.J., J.E. Smits, G.R. Bortolotti, and D.M. Bird. 2001a. In ovo exposure to 5 polychlorinated biphenyls: reproductive effects on second-generation American kestrels.
- 6 Archives of Environmental Contamination and Toxicology 40:544-550.
- Fernie, K.J., J.E. Smits, G.R. Bortolotti, and D.M. Bird. 2001b. Reproductive success of
 American kestrels exposed to dietary polychlorinated biphenyls. *Environmental Toxicology and Chemistry* 20:776-781.
- 10 Gibbs, J.P., S. Melvin, and F.A. Reid. 1992. American Bittern (Botaurus lentiginosus). In The
- 11 Birds of North America, No. 18. A. Poole, P. Stettenheim, and F. Gill (eds). The Birds of North
- 12 America, Inc. Philadelphia, PA, USA.
- 13 Giesy, J.P., W.W. Bowerman, M.A. Mora, D.A. Verbrugge, R.A. Othoudt, J.L. Newsted, C.L.
- 14 Summer, R.J. Aulerich, S.J. Bursian, J.P. Ludwig, G.A. Dawson, T.J. Kubiak, D.A. Best, and
- 15 D.E. Tillitt. 1995. Contaminants of fishes from Great Lakes-influenced sections and above dams
- 16 of three Michigan rivers: III. Implications for health of bald eagles. Archives of Environmental
- 17 *Contamination and Toxicology* 29(3):309-321.
- 18 Gilbert, R.O. 1987. *Statistical Methods for Environmental Pollution Monitoring*. Van Nostrand19 Reinhold Company, New York, NY. 320 pp.
- Godin, A.J. 1977. *Wild Mammals of New England*. The John Hopkins University Press, Baltimore,
 MD, USA, and London, UK.
- Griffith, L.A. and J.E. Gates. 1985. Food habits of cave-dwelling bats in the central
 Appalachians. *Journal of Mammalogy* 66:451-460.
- Gulo, T. 2001. Massachusetts Division of Fisheries and Wildlife. Personal Communication toWoodlot Alternatives, Inc.
- Haffner, G.D., M. Tomczak, and R. Lazar. 1994. Organic contaminant exposure in the Lake St.
 Clair food web. *Hydrobiologia* 281:19-27.
- Haywood, D.D. and R.D. Ohmart. 1986. Utilization of benthic-feeding fish by inland breeding
 bald eagles. *Condor* 88:35-42.
- 30 Helander, B., M. Olsson, and L. Reuterhard. 1982. Residue levels of organochlorine and mercury
- 31 compounds in unhatched eggs and their relationship to breeding success in white-tailed sea
- 32 eagles in Sweden. *Holarctic Ecology* 5:349-366.
- 33 Hoffman, D.J., B.A. Rattner, C.M. Bunck, A. Krynitsky, H.M. Ohlendorf, and R.W. Lowe. 1986.
- 34 Association between PCBs and lower embryonic weight in black-crowned night herons in San
- 35 Francisco Bay. Journal of Toxicology and Environmental Health 19:383-391.

- 1 Hoffman, D.J., G.J. Smith, and B.A. Rattner. 1993. Biomarkers of contaminant exposure in
- 2 common terns and black-crowned night herons in the Great Lakes. *Environmental Toxicology and*
- *Chemistry* 12:1095-1103.
- 4 Hoffman, D.J., M.J. Melancon, P.N. Klein, C.P. Rice, J.D. Eisemann, R.K. Hines, J.W. Spann,
- 5 and G.W. Pendleton. 1996. Developmental toxicity of PCB 126 (3,3,4,4,5-pentachlorobuphenyl)
- 6 in nestling American kestrels (*Falco sparverius*). *Fundamental and Applied Toxicology* 34:188-
- 7 200.
- Khera, K.S. and J.A. Ruddick. 1973. Polychlorodibenzo-p-dioxins: Perinatal effects and the
 dominant lethal test in Wistar rats. *Toxicology* 120:70-84.
- 10 Krusic, R.A., Y. Mariko, C.D. Neefus, and P.J. Pekins. 1996. Bat habitat use in the White 11 Mountain National Forest. *Journal of Wildlife Management* 60(3):625–631.
- Kurta, A. 1995. *Mammals of the Great Lakes Region*. The University of Michigan Press. Ann
 Arbor, MI. 392 pp.
- Laporte, P. 1982. Organochlorine residues and eggshell measurements of great blue heron eggsfrom Quebec. *Colonial Waterbirds* 5:95-103.
- 16 Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S. Petron,
- 17 B. Potocki, S. Svirsky, and P. Tyler. 1996. Special report of the Massachusetts Weight-of-
- 18 Evidence Workgroup: A weight of evidence approach for evaluating ecological risks. *Human and*
- 19 Ecological Risk Assessment 2(2):277-304.
- Mitchell, C.A., D.H. White, and T.E. Kaiser. 1981. Reproductive success of great blue herons at
 Nueces Bay, Corpus Christi, Texas. *Bulletin of Texas Ornithological Society* 14:18-21.
- MNHESP (Massachusetts Natural Heritage and Endangered Species Program). 1984. Special
 Concern Species of Massachusetts Small-footed Myotis Fact Sheet prepared by the
 Commonwealth of Massachusetts, Division of Fisheries and Wildlife. Westborough, MA, USA.
- Moore, D.R.J., B.E. Sample, G.W. Suter, B.R. Parkhurst, and R.S. Teed. 1999. A probabilistic
 risk assessment of the effects of methylmercury and PCBs on mink and kingfishers at East Fork
 Poplar Creek, Oak Ridge, TN. *Environmental Toxicology and Chemistry* 18:2941-2953.
- Nagy, K.A., I.A. Girard, and T.K. Brown. 1999. Energetics of free-ranging mammals, reptiles,
 and birds. Annu. Rev. Nutr. 19:247-277.
- Nagy, K.A. 1987. Free metabolic rate and food requirement scaling in mammals and birds.
 Ecological Monographs 57:111-128.
- Nobel, D.G. and J.E. Elliott. 1990. Levels of contaminates in Canadian raptors, 1966 to 1988; effects and temporal trends. *Canadian Field Naturalist* 104:222-243.

- 1 Peakall, J.D., D.G. Noble, J.E. Elliott, J.D. Somers, and G. Erickson. 1990. Environmental 2 contaminants in Canadian Peregrine Falcons, *Falco peregrines*: a toxicological assessment.
- 3 *Canadian Field-Naturalist* 104:244–254.
- 4 Rattner, B.A., D.J. Hoffman, M.J. Melancon, G.H. Olsen, S.R. Schmidt, and K.C. Parsons. 2000.
- 5 Organochlorine and metal contaminant exposure and effects in hatching black-crowned night
- 6 herons (Nycticorax nycticorax) in Delaware Bay. Archives of Environmental Contamination and
- 7 *Toxicology* 39:38-45.
- 8 Senthilkumar, K., N. Iseki, S. Hayama, J. Nakanishi, and S. Masunaga. 2001. Polychlorinated
- 9 Dibenzo-p-Dioxins, Dibenzofurans, and Dioxin-like Polychlorinated Biphenyls in Livers of
- 10 Birds from Japan. Archives of Environmental Contamination and Toxicology 42:244-255.
- 11 Sparschu G.L., F.L. Dunn, R.W. Lisowe, and V.K. Rowe. 1971. Study of the effects of high
- 12 levels of 2.4.5-trichlorophenoxyacetic acid on fetal development in the rat. *Food and Cosmetics*
- 13 *Toxicology* 9:527-530.
- 14 Spencer, F. 1982. An assessment of the reproductive toxic potential of Aroclor 1254 in female 15 Sprague-Dawley rats. *Bulletin of Environmental Contamination and Toxicology* 28:290-297.
- 16 Stalmaster, M.V. and J.A. Gessaman. 1982. Food consumption and energy requirements of 17 captive bald eagles. *Journal of Wildlife Management* 46(3):646-654.
- 18 Stalmaster, M.V. and J.A. Gessaman. 1984. Ecological energetics and foraging behavior of 19 overwintering bald eagles. *Ecological Monographs* 54(4):407-428.
- 20 Stalmaster, M.V. and R.G. Plettner. 1992. Diets and foraging effectiveness of bald eagles during 21 extreme winter weather in Nebraska. *Journal of Wildlife Management* 56:355-367.
- 22 Stratus (Stratus Consulting, Inc.). 1999. Injuries to Avian Resources, Lower Fox River/Green Bay
- 23 Natural Resource Damage Assessment. Final Report. Prepared for U.S. Fish & Wildlife Services,
- U.S. Department of the Interior, and the U.S. Department of Justice. May 7, 1999.
- Todd, C.S., L.S. Young, R.B. Owen, and F.J. Gramlich. 1982. Food habits of bald eagles in Maine.
 Journal of Wildlife Management 46:636–645.
- 27 Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, P. Giesy,
- 28 A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larson, F.W.R. Leeuwen, D. Tillitt,
- M. Tysklind, M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives*
- 31 106:775-792.
- Watson, J.W., M.G. Garrett, and R.G. Anthony. 1991. Foraging ecology of bald eagles in the Columbia River estuary. *Journal of Wildlife Management* 55:492-499.
- 34 Wiemeyer, S.N., C.M. Bunck, and C.J. Stafford. 1993. Environmental contaminants in bald
- 35 eagle eggs 1980–84 and further interpretations of relationships to productivity and shell
- 36 thickness. Archives of Environmental Contamination and Toxicology 24:213-227.

- 1 Woodford, J.E., W.H. Karasov, M.W. Meyer and L. Chambers. 1998. Impacts of 2,3,7,8-TCDD
- 2 exposure on survival, growth, and behavior of ospreys breeding in Wisconsin, USA.
- 3 Environmental Toxicology and Chemistry 17:1323-1331.
- 4 Zimmerman, G.S. and W.E. Glanz. 2000. Habitat use by bats in eastern Maine. Journal of Wildlife
- 5 *Management* 64:1032-1040.

1 12. RISK SUMMARY

2 Highlights of Risk Summary 3 Total PCBs and other COCs in the PSA of the Housatonic River pose 4 unacceptable risks to some assessment endpoints. 5 Risk is high for benthic invertebrates, amphibians, and piscivorous mammals. . 6 7 Confidence in this conclusion is high because (1) multiple lines of evidence with concordant results were available; (2) models used to estimate risk were not 8 conservative; and (3) consideration of uncertainty indicates a high probability of 9 effects. 10 Risk is moderate to high for some piscivorous birds, omnivorous and carnivorous 11 mammals, and high for selected threatened and endangered bird and mammal species. There is uncertainty regarding these conclusions because corroborating 12 13 lines of evidence were generally not available. 14 Risk is low to moderate for fish and confidence in this conclusion is moderate. 15 Risk is low for insectivorous birds, but confidence in this conclusion is not high. 16 Other species not included in the quantitative risk assessments may also be at 17 risk in the PSA. 18 Assessment of risks to the most susceptible endpoints downstream of the PSA 19 indicates that benthic invertebrates, amphibians, warmwater and coldwater fish. 20 mink, river otter, and bald eagles may be at risk.

21 **12.1 OVERVIEW**

The assessment of ecological risks of COCs in the Housatonic River to aquatic life and wildlife is described in Sections 3 through 11 and in more detail in Appendices D through K. The amount of information considered in this assessment is large, and the analyses and interpretation complex. The purpose of this section is to summarize the major findings of the ERA and to discuss the implications of these findings for biota in the Primary Study Area (PSA) and downstream of the PSA.

Section 12.2 summarizes the risk assessment findings for each assessment endpoint. The first part of this presentation (Section 12.2.1) discusses the results of the weight-of-evidence approach for each of the 8 assessment endpoints evaluated in the risk assessment. The WOE approach is a process by which measurement endpoints are related to an assessment endpoint to evaluate whether significant risk is posed to the environment (Menzie et al. 1996). A formal WOE can range from a simple qualitative assessment to a highly quantitative evaluation; however, no matter what form the WOE takes, it should provide documentation of the thought process used
 when assessing potential ecological risk.

The term "line of evidence" as used in this ERA follows the definition of "Information derived from different sources or by different techniques that can be used to describe and interpret risk estimates" provided in the *Guidelines for Ecological Risk Assessment* (EPA 1998). Unlike the term "weight-of-evidence," this definition does not imply assignment of qualitative or quantitative weightings to information. The three general lines of evidence under which most measurement endpoints fall are (Hull and Suter 1994; Suter et al. 1995):

- 9
- Biological (field) survey data that indicate the state of the receiving environment.
- 10 11
- Media toxicity data that indicate whether the contaminated media are toxic (i.e.,
- laboratory or in situ toxicity testing).
- 12 13

 Single contaminant toxicity data that indicate the toxic effects of the concentration measured in site media (e.g., exposure modeling).

Two or three general lines of evidence were considered in evaluating potential risk for most assessment endpoints. The WOE approach used in this ERA for each of the assessment endpoints follows the approach originally described in the *Massachusetts Weight-of-Evidence Special Report* (Menzie et al. 1996).

Following the WOE discussion, Section 12.2.2 presents a discussion of hazard quotients (HQs) that were calculated for each receptor for the two COCs of greatest concern, tPCBs and TEQ, to facilitate comparison of risks between assessment endpoints in the PSA. The HQ analysis includes estimates of uncertainty to provide an indication of both the magnitude of risk for each COC receptor combination and the amount of uncertainty about each risk estimate.

Following the HQ analysis, the assessment of risks conducted for areas downstream of the PSA is described in Section 12.2.3. As is apparent from the preceding sections of the ecological risk assessment, risks to some assessment endpoints vary within the PSA as well as downstream, due to the small-scale variability in sediment and, to a lesser degree, floodplain soil concentrations. Section 12.2 concludes with a brief discussion of possible reasons for the differences in risk between assessment endpoints for the most influential COCs, tPCBs and TEQ.
Section 12.3 discusses the broader implications of the risk assessment findings summarized in
 Section 12.2. Issues addressed include:

- The risk assessment described in Sections 3 through 11 and Appendices D through K focused the majority of quantitative analyses on selected species, termed "representative species." There are, however, many other species that occur in the watershed of the Housatonic River. Section 12.3 begins with a discussion of estimates of the potential risks posed by COCs to these other species.
- In addition to effects on survival, growth, and reproduction of individuals in the Housatonic River, there are a number of other possible impacts of COCs on aquatic life and wildlife that were not addressed in the individual assessments (i.e. indirect effects, narrowing of the genetic pools for exposed species). These topics are briefly addressed in Section 12.3.

13 Section 12.4 provides a discussion acknowledging that there are many sources of uncertainty in 14 an ecological risk assessment, even in assessments (such as this ERA) that have a great deal of 15 available information. These sources of uncertainty can have important consequences during the 16 risk management process; therefore, it is important to describe them. The preceding sections and 17 the appendices described the sources of uncertainty for each assessment endpoint and their 18 potential influence on risk estimates and the confidence in those risk estimates. Section 12.4 19 summarizes the most important sources of uncertainty, particularly those that were common to 20 many assessment endpoints.

Section 12.5 concludes with a listing of the major findings of the Housatonic River ecological
risk assessment.

1 12.2 SUMMARY OF THE ASSESSMENT ENDPOINT CONCLUSIONS

The problem formulation stage of the ERA (Section 2.8) identified the assessment endpoints considered important in the Housatonic River ERA. Each of the assessment endpoints was evaluated and conclusions made regarding the potential for adverse effects (see Sections 3 through 11 and Appendices D through K). Table 12.2-1 provides a short summary for each of the assessment endpoints and the conclusions reached in the ERA for the PSA. Tables 12.2-2 to 12.2-16 indicate the results of the weight-of-evidence assessments for each assessment endpoint.

8 12.2.1 Results of Weight-of-Evidence Evaluation

9 12.2.1.1 Benthic Invertebrates

The WOE results for the benthic invertebrate assessment endpoint are shown in Table 12.2-2. In this WOE table, the measurement endpoints for the three lines of evidence: water, sediment, and tissue chemistry (C), toxicity tests (T), benthic community measures (B) are listed, as are the weighting of the measurement endpoint, evidence of harm, and magnitude of response. This table indicates that the majority of endpoints suggest some risk for benthic communities in both coarse- and fine-grained sediment. The conclusion is that there is an intermediate to high risk to much of the benthic community, as indicated by the WOE evaluation.

17 12.2.1.2 Amphibians

18 The results of the WOE assessment for amphibians are presented in Table 12.2-3. In the 19 amphibian WOE matrix, the measurement endpoints for the three lines of evidence: tissue 20 chemistry (C); wood frog toxicity tests (W) and leopard frog toxicity tests (L); and field surveys 21 (B) are listed. As shown in the table, many of the measurement endpoints indicated some degree 22 of risk. The potential for two amphibian studies conducted for GE to determine risk to amphibians was judged to be undetermined due to limitations in the study designs. The only 23 24 endpoint that did not indicate potential risk was the earliest life stage wood frog toxicity 25 endpoint, for which there is a mechanistic explanation for the lack of response. Four endpoints 26 exhibited a high degree of risk combined with a moderate to high confidence rating.

Ecological Assessment Endpoints and Conclusions for the Primary Study Area Portion of the Lower Housatonic River

Receptor	Assessment Endpoint	Conclusions
Benthic Invertebrates	Community structure, survival, growth, and reproduction	The benthic invertebrate ERA demonstrates significant risk from tPCBs based on a weight-of-evidence evaluation of multiple effects endpoints. The pronounced toxicity (laboratory and in situ) observed in PSA sediment was supported by toxicity identification evaluation (TIE) findings, alterations to macroinvertebrate community structure, and large exceedances of effects benchmark values for invertebrate tissues, sediment, and water.
Amphibians	Community condition, survival, reproduction, development, and maturation	The amphibian ERA indicates significant risk to frogs from tPCBs based on a weight-of-evidence evaluation of multiple effects endpoints. The literature-derived tissue thresholds for tPCBs were supported by site-specific toxicity studies, skewed sex ratios, malformations, and other effects that implicated tPCBs as the causal agent. Sediment toxicity tests indicated a correlation between level of effect and tPCB concentration.
Fish	Survival, growth, and reproduction	The fish ERA found significant potential risk to fish from tPCBs and TEQ based on a weight-of-evidence evaluation. The tissue thresholds identified in the literature and from site-specific toxicity studies were exceeded by fish tissue concentrations measured in the PSA for all representative species. For some species (e.g., yellow perch), the majority of individual fish concentrations exceeded the respective benchmarks for both tPCBs and TEQ. Despite the high probability of risk, the magnitude of adverse responses is expected to be low to intermediate. The observed fish tissue tPCB concentrations did not exceed the derived effects levels by a large factor (i.e., factor of 5 or more) in many samples, and studies suggest that current effects to fish are not severe. The findings from the literature reviews and site-specific toxicity studies are consistent with the results of field studies, which indicate that populations of fish in the PSA are not experiencing catastrophic effects.
Insectivorous Birds	Survival, growth, and reproduction	The weight-of-evidence analysis indicates that insectivorous birds, such as tree swallows and American robins, are likely at low risk in the PSA as a result of exposure to tPCBs and TEQ. Risks to tree swallows and robins in the PSA are predicted to be intermediate to high based on modeling of exposure and effects, but field studies of tree swallows and American robins detected no obvious adverse reproductive effects to these birds in the PSA. The weight-of- evidence assessment relied more heavily on the results of the site- specific field studies, but the conclusion of low risk is uncertain because the lines of evidence did not give concordant results.

Ecological Assessment Endpoints and Conclusions for the Primary Study Area Portion of the Lower Housatonic River (Continued)

Receptor	Assessment Endpoint	Conclusions
Piscivorous Birds	Survival, growth, and reproduction	The weight-of-evidence analysis indicates that ospreys are at high risk from exposure to tPCBs and intermediate risk from exposure to TEQ in the Housatonic River PSA. In the PSA, exposure of ospreys to tPCBs is greater than doses that caused adverse effects in the most tolerant bird species studied. The conclusion of high risk to ospreys is uncertain because only one line of evidence was available. Belted kingfishers are considered to be at low risk as a result of exposure to tPCBs and TEQ in the Housatonic River PSA. While modeled exposure and effects indicated high risk for tPCBs and intermediate risk for TEQ, a field study of kingfisher productivity indicated that the birds were reproducing in the PSA. The conclusion of low risk to kingfishers is uncertain because the two lines of evidence did not give concordant results.
Piscivorous Mammals	Survival, growth, and reproduction	The weight-of-evidence analysis indicates that piscivorous mammals (i.e., mink and river otter) are at intermediate to high risk in the PSA as a result of exposure to tPCBs and TEQ. Evidence for this conclusion includes limited sightings of mink and otter in the PSA, except during winter, despite availability of appropriate habitat and evidence that they are common in nearby reference areas; results of the feeding study which showed effects on kit survival and jaw lesions in surviving mink at a much smaller fraction of fish in the diet (3.5%) than would be expected of mink foraging in the PSA; and modeling of exposure and effects which predicted severe risks to mink and otter foraging in the PSA. Risks to mink and otter are likely to be elevated even for mink and otter that forage only a small fraction (e.g., 10%) of their time in the PSA.
Omnivorous and Carnivorous Mammals	Survival, growth, and reproduction	The weight-of-evidence analysis indicates an intermediate risk for red fox and short-tailed shrews exposed to tPCBs and TEQ in the PSA. The field survey indicated that omnivorous and carnivorous mammals, including short-tailed shrew and red fox, are common in some areas of the PSA. In contrast, modeling of exposure and effects predicts these animals to be at low to high risk as a result of exposure to tPCBs and TEQ in the PSA. The population demography field study suggested that short-tailed shrews are not seriously affected by tPCB contamination, although a reanalysis of the data did not fully support this conclusion. The conclusion of intermediate risk is uncertain because of the uncertainty about whether effects are occurring for two of the three lines of evidence.
Threatened and Endangered Species	Survival, growth, and reproduction	Based on modeling of exposure and effects, bald eagles and American bitterns are at risk as a result of exposure to tPCBs. Risk from TEQ is intermediate for bald eagles and undetermined for American bittern. The risks to small-footed myotis exposed to tPCBs and TEQ are undetermined.

Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Benthic Community

	Weighting	Coarse-Grained Sediment		Fine-Grained Sediment	
Measurement Endpoints	Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
C. Chemical Measures					
C-1. Concentration of PCB in overlying water in relation to levels reported to be harmful to benthic invertebrates	Low/Moderate	Yes	Intermediate	Yes	Intermediate
C-2. Concentration of PCB in the sediment in relation to levels reported to be harmful to benthic invertebrates	Low/Moderate	Yes	High	Yes	High
C-3. Concentration of PCB in invertebrate tissues in relation to levels reported to be harmful to benthic invertebrates	Moderate	Yes	Intermediate	Yes	Intermediate
T. Toxicological Measures					
T-1. Sediment toxicity to multiple invertebrate species, as measured in laboratory toxicity tests	Moderate/ High	Yes	High	Yes	High
T-2. Sediment toxicity to multiple invertebrate species, as measured in in situ toxicity tests	Moderate/ High	Yes	Intermediate	Yes	High
T-3. Indications of PCB as toxicity driver in toxicity identification evaluations	Moderate	Undetermined	_	Yes	Intermediate
B. Benthic Community Measures					
B-1. Abundance, richness, and biomass of invertebrates, relative to reference stations of comparable substrate and habitat (ANOVA)	Moderate	Yes	Intermediate	No	_
B-2. Benthic community structure, as assessed using a multivariate assessment of key benthic metrics (rank analysis and MDS)	Moderate	Yes	Intermediate	No	—
B-3. Water quality assessment using modified Hilsenhoff Biotic Index (MHBI) indicator of organic pollution	Moderate	No	_	No	—

Table 12.2-3Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of Amphibian
Populations in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
C. Chemical Measures			
C. Concentration of PCB in frog tissues in relation to levels reported to be harmful to amphibians.	Moderate	Yes	Low
W. Wood Frog Toxicological Measures			
W-1. Sediment toxicity to hatchling/late embryo life stages.	Mod/High	No	-
W-2. Sediment toxicity to larval life stages.	Mod/High	Yes	Intermediate
W-3. Sediment toxicity to late larval/metamorph life stage.	Mod/High	Yes	High
W-4. GE Study (juvenile wood frogs)	Low	Undetermined	-
L. Leopard Frog Toxicological Measures			
L-1. Sediment toxicity to hatchling/late embryo life stages.	Mod/High	Yes	Low
L-2. Sediment toxicity to larval life stages.	Mod/High	Yes	High
L-3. Sediment toxicity to late larval/metamorph life stage.	Mod/High	Yes	High
L-4. Sediment toxicity to adult leopard frogs (reproductive health).	Mod/High	Yes	High
B. Biology			
B-1. Vernal pool community study.	Mod/High	Yes	Low
B-2. GE leopard frog egg mass survey	Low/Mod	Undetermined	-
B-3. Anecdotal observations during collections for reproductive study.	Moderate	Yes	Low

In addition, a population model was constructed for wood frogs to determine whether effects from PCBs on individual wood frogs influence the populations within the PSA. A ten-year simulation both with and without the effects of PCBs was conducted. The model demonstrated that effects observed in the toxicity studies would result in population level impacts.

5 The conclusion is that there is a significant risk to amphibians as indicated by the preponderance 6 of the evidence, the relative weights of the measurement endpoints, and the population modeling. 7 The "no risk" value of measurement endpoint W-1 does not diminish the overall conclusion, 8 because the study demonstrated that the embryo/early larval life stages are fairly insensitive to 9 the effects of maternally transferred PCBs relative to later juvenile life stages exposed to 10 contaminated media.

11 12.2.1.3 Fish

12 The WOE results for fish in the PSA are shown in Table 12.2-4. In the fish WOE matrix, the 13 measurement endpoints for the three lines of evidence: site-specific toxicity tests (A); fish tissue 14 chemistry (B); and field surveys (C) are listed. This table illustrates that the majority of 15 endpoints indicate, with a moderately high degree of confidence, that there are low magnitude 16 risks to fish in the PSA. Although a high probability of adverse impacts to fish from tPCBs 17 and/or TEQ is predicted throughout the PSA, the impacts predicted are for sensitive sublethal 18 endpoints (reproduction and development); mortality of adults is unlikely. Therefore, the 19 magnitude of impact is not predicted to be catastrophic in any reach; adverse effects, although 20 high in probability, are generally expected to be subtle. The field studies conducted in the PSA 21 (fish community and reproduction studies) support lack of catastrophic effects, but cannot be 22 used to assess lesser impacts.

23 12.2.1.4 Insectivorous Birds

The WOE results for exposure of insectivorous birds to tPCBs are presented in Table 12.2-5 and for exposure to TEQ in Table 12.2-6. Two lines of evidence are presented in the table; the field studies, and modeled exposure and effects. The results from the modeled exposure and effects line of evidence suggest that tPCBs and TEQ pose intermediate to high risk to tree swallows living in the PSA. However, the field study line of evidence suggests that, if effects are

Evidence of Harm and Magnitude of Effects for Measurement Endpoints Related to Maintenance of a Healthy Fish Community

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	
A. Site-Specific Toxicity				
A1. Reproductive success relative to reference	Mod/High	Yes	Low	
A2. Reproductive success dose-response	High	Yes	Intermediate	
B. Fish Tissue Chemistry				
B1. Observed fish tissue/Literature toxic levels	Mod	Yes	Low	
B2. Observed fish tissue/Phase I toxic levels	Mod/High	Yes	Low	
B3. Observed fish tissue/Phase II toxic levels	Mod/High	Yes	Low	
C: Fish Community and Reproduction Studies				
C1: EPA Study and GE Community Study	Low/Mod	Undetermined	-	
C2: GE Reproduction Study	Low/Mod	Undetermined	-	

Table 12.2-5

Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Study	High (Tree swallow) Moderate/High (American robin)	No (Tree swallow) No (American robin)	Low (Tree swallow) Low (American robin)
Modeled Exposure and Effects	Moderate	Yes	High

Table 12.2-6

Evidence of Harm and Magnitude of Effects for Insectivorous Birds Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Study	High (Tree swallow) Moderate/High (American robin)	No (Tree Swallow) No (American robin)	Low (Tree Swallow) Low (American robin)
Modeled Exposure and Effects	Moderate	Yes	Intermediate

occurring, they are minor. The uncertainty concerning the field-based threshold range for tPCBs 1 2 likely means that risks of this COC are overestimated for the PSA. Even the upper end of the 3 tPCB range is associated with equivocal evidence for adverse effects to tree swallows. For TEQ, 4 the threshold range is quite broad. The available evidence from field studies indicates that tree 5 swallows are tolerant to exposure to persistent organochlorines such as tPCBs and TEQ. If the 6 tree swallow threshold is near the upper end of the threshold range, then the current modeled 7 exposure and effects line of evidence is overestimating risks of TEQ to tree swallows. Thus, the 8 WOE assessment supports a finding of low risk for tree swallows exposed to tPCBs and TEQ in 9 the PSA. This conclusion, however, is uncertain because of the conflicting results in the WOE 10 assessment.

11 The results from the modeled exposure and effects lines of evidence suggest that tPCBs and TEQ 12 pose an intermediate to high risk to American robins inhabiting the PSA of the Housatonic River. 13 The American robin field study, however, suggests that reproductive success is not being impaired by the tPCBs and TEQ in the PSA. The uncertainty in the modeled exposure and 14 15 effects line of evidence, outlined below, likely means the approach overestimates the risks of 16 tPCBs and TEQ to American robins in the PSA. The WOE assessment therefore supports a 17 conclusion of low risk to American robins exposed to tPCBs and TEQ in the PSA. This 18 conclusion, however, is uncertain because of the conflicting results in the WOE assessment.

19 12.2.1.5 Piscivorous Birds

The WOE analysis indicates that exposure of piscivorous birds, such as the belted kingfisher and osprey (Tables 12.2-7 and 12.2-8), to tPCBs and TEQ in the PSA, could lead to adverse reproductive effects in some species. The two lines of evidence used to support this conclusion were the field study of kingfisher productivity and the comparison of modeled exposure with effects to piscivorous birds.

For the assessment of risks to kingfishers, both lines of evidence were available. The modeled exposure and effects line of evidence indicated that kingfishers in the PSA are likely to receive a tPCB dose greater than what the most tolerant species known from the literature can be exposed to without effects. For TEQ, the risk picture is less clear because the threshold range for this COC is very wide and the exposure estimates for kingfishers fell within this range. Thus,

Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled Exposure and Effects	М	Kingfisher – Yes Osprey – Yes	Kingfisher – High Osprey – High
Belted Kingfisher Field Study (Henning 2002)	M/H	Kingfisher - No	Kingfisher - Low

5 6 7

1

2 3

4

8

9

Table 12	2.2-8
----------	-------

Evidence of Harm and Magnitude of Effects for Piscivorous Birds Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Modeled Exposure and Effects	М	Kingfisher – Yes Osprey – Yes	Kingfisher – Intermediate Osprey – Intermediate
Belted Kingfisher Field Study (Henning 2002)	M/H	Kingfisher - No	Kingfisher - Low

10

without effects data specific to kingfishers, it is difficult to make definitive conclusions about the risks of TEQ to this species. The field study of kingfisher productivity, however, indicated that these birds are able to reproduce in the PSA. This line of evidence was given a higher weighting than the exposure and effects modeling, despite concerns about the field study. Therefore, kingfishers are considered to be at low risk in the PSA as a result of exposure to tPCBs and TEQ. The conclusion of low risk to kingfishers is uncertain because the two lines of evidence did not give concordant results.

For ospreys, only the modeled exposure and effects line of evidence was available to assess risk to these birds. As with kingfishers, this line of evidence indicated that ospreys in the PSA are likely to receive a tPCB dose that is greater than what the most tolerant species known in the literature can be exposed to without effects. The risks due to exposure to TEQ are unclear, as the estimates for exposure also fell within the toxicity threshold range. Ospreys, however, lack a site-specific study that investigated the effects of COCs in the PSA. The PSA contains suitable habitat for ospreys, with abundant prey, raising the possibility that they are not resident in the area because of contaminants. Ospreys are, therefore, considered to be at risk in the PSA as a result of exposure to tPCBs and TEQ.

5 12.2.1.6 Piscivorous Mammals

6 The results of the WOE assessment for piscivorous mammals are presented for tPCB and TEQ, 7 respectively, in Tables 12.2-9 and 12.2-10. All three lines of evidence-field studies, feeding 8 study, and modeled exposure and effects-indicate that the elevated concentrations of tPCBs and 9 TEQ in the PSA of the Housatonic River are causing adverse effects of high magnitude to mink 10 and river otter. The field surveys indicate that mink and river otter are rarely present in the PSA, 11 except during winter, and likely have not established home territories close to the main channel despite suitable mink and otter habitat. The MSU site-specific feeding study indicated that 12 13 feeding adult female mink with a diet containing as little as 3.51% fish from the PSA caused a 14 statistically significant reduction (46% compared to controls) in kit survival to 6 weeks of age. 15 Because mink in the wild typically consume between 20% or more fish in their diet, the 16 associated risk is correspondingly higher. In addition, other components of the mink diet in the 17 PSA (e.g., crayfish) have high concentrations of tPCBs and TEQ. Further, the jaw lesion study 18 indicated that erosion of the jaw occurs at even lower doses and exhibits a dose-response 19 relationship. Such effects could eventually lead to starvation. The occurrence of jaw lesions 20 coincides with the induction of Ah-receptor-regulated enzymes (ECOD and EROD) also in a 21 dose-response manner.

The high risks evident from the feeding study are further supported by the modeled exposure and effects line of evidence. The estimated potential for exposure is so high that even individual mink and otter that only forage in the PSA for short periods of time (less than or equal to 10% of foraging time) are at an intermediate or higher risk from tPCBs and TEQ.

26 12.2.1.7 Omnivorous and Carnivorous Mammals

The WOE results for omnivorous and carnivorous mammals are shown in Table 12.2-11 for tPCB and Table 12.2-12 for TEQ. Data from three lines of evidence were available, including

Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Measureme Endpoints	nt	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Surveys	EPA	Moderate/High	Yes	High
	GE	Moderate	No	Low
Feeding Study		High	Yes	High
Modeled Exposure and Effects		Moderate/High	Yes	High

5

6

7 8

Evidence of Harm and Magnitude of Effects for Piscivorous Mammals Exposed to TEQ in the Housatonic River PSA 9

Table 12.2-10

Measureme Endpoints	nt	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)
Field Surveys	EPA	Moderate/High	Yes	High
Field Surveys	GE	Moderate	No	Low
Feeding Study		High	Yes	High
Modeled Exposur Effects	re and	Moderate/High	Yes	High

10

Table 12.2-11

Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to tPCBs in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low) Low	
Field Surveys	Moderate/High	Undetermined		
Population Demography Field Moderate/High Study		Undetermined (Shrew)	Intermediate	
Modeled Exposure and	Moderate/High	Yes (Shrew)	High	
Effects		Undetermined (Red Fox)	Intermediate	

5

6

7 8

9

Table 12.2-12

Evidence of Harm and Magnitude of Effects for Omnivorous and Carnivorous Mammals Exposed to TEQ in the Housatonic River PSA

Measurement EndpointsWeighting Value (High, Moderate, Low)Field SurveysModerate/High		Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low) Low	
		Undetermined		
PopulationDemography FieldModerate/HighStudy		Undetermined (Shrew)	Intermediate	
Modeled Exposure and Effects	Moderate/High	No (Shrew) Undetermined (Red Fox)	Low Intermediate	

field surveys, a population demography field study of short-tailed shrew and exposure and 1 2 effects modeling. The weight-of-evidence analysis indicates an intermediate risk for short-tailed 3 shrews exposed to tPCBs and TEQ in the PSA. This conclusion, however, is uncertain because 4 of the lack of definitive findings as to whether effects are occurring in the field surveys and 5 population demography field study, and the lack of species-specific measures of effects in the 6 literature. The WOE also suggests, based on one line of evidence for red fox an intermediate 7 risk to fox exposed to tPCBs and TEQ in the PSA using a foraging rate of 50% in Reach 5, in 8 addition, measures of effects for fox were not available in the literature.

9 The field surveys, and conclusions made in a study of short-tailed shrew populations at the site 10 conducted for GE are not in agreement with the results from the modeling of exposure and 11 effects line of evidence. However, the results of the supplemental analyses of the data from the 12 GE study on survival of short-tailed shrews are in agreement with the modeling results, 13 suggesting that there are intermediate effects from exposure to COCs in the contaminated areas 14 of the PSA.

15 12.2.1.8 Threatened and Endangered Species

The results of the WOE evaluation for threatened and endangered species using a single line of evidence, modeled site-specific exposures and effects, are shown in Table 12.2-13 and Table 12.2-14 for tPCBs and TEQ, respectively. The results of the risk characterization showed that the highest risk to T&E species is to bald eagles and American bitterns from exposure to tPCBs. The risk for adult bald eagles exposed to TEQ is low; however, risk to bald eagle eggs exposed to TEQ is high.

The risk to American bittern was high for TEQ and high for eggs exposed to TEQ. The risk category for small-footed myotis was high for both tPCB and TEQ. The risk range for smallfooted myotis, as determined by the probability bounds analysis, ranged from intermediate to high for tPCBs and low to high for TEQ.

The weight-of-evidence analysis indicates that T&E species such as bald eagles, American bitterns, and small-footed myotis are at risk in the PSA as a result of exposure to tPCBs 1 2 3

4

Table 12.2-13

Evidence of Harm and Magnitude of Effects for T&E Species Exposed to tPCBs in Housatonic River PSA

Measurement Endpoints Weighting Value (High, Moderate, Low)		Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low)	
Modeled exposure and effects, Bald Eagle	Moderate/High	Yes	High	
Modeled exposure and effects, American Bittern	Moderate/High	Yes	High	
Modeled exposure and effects, Small-Footed Myotis Moderate/High		Undetermined	High	

5

6 7

8

9

Table 12.2-14

Evidence of Harm and Magnitude of Effects for T&E Species Exposed to TEQ in the Housatonic River PSA

Measurement Endpoints	Weighting Value (High, Moderate, Low)	Evidence of Harm (Yes, No, Undetermined)	Magnitude (High, Intermediate, Low) Intermediate	
Modeled exposure and effects, Bald Eagle	Moderate/High	Yes		
Modeled exposure and effects, American Moderate/High Bittern		Undetermined	High	
Modeled exposure and effects, Small-Footed Myotis	Moderate/High	Undetermined	High	

10

11	and TEQ. The risks for bald eagles and American bitterns exposed to tPCBs are high. The risks
12	to small-footed myotis exposed to tPCBs and TEQ are undetermined. Risk from exposure to
13	TEQ is intermediate for bald eagles and undetermined for American bittern.

14 **12.2.2 Hazard Quotient Analyses**

15 The assessments described in Sections 3 through 11 and Appendices D through K were 16 conducted using various lines of evidence including, in many cases, different measurement

endpoints and effects metrics. It is clear that risks posed by COCs in the PSA vary between 1 2 species; however, the degree of variability is not clear from these discussions because of the 3 differing endpoints and metrics used. To facilitate comparison of risks among aquatic life and 4 wildlife receptors and to give a broad overview of the findings of the risk assessment, assessment 5 results were converted to probabilistic hazard quotients (HQs). An HQ is the expected 6 environmental concentration or dose of a contaminant divided by its estimated low or no toxic 7 effect concentration or dose. Higher quotients indicate greater risk. The methods used to 8 calculate the probabilistic HQs and the results of these analyses for each endpoint are discussed 9 in this section.

10 12.2.2.1 Aquatic Assessment Endpoints

11 12.2.2.1.1 Benthic Invertebrates

12 For benthic invertebrates, HQs were calculated for Reaches 5A, 5B, 5C, 5D, and 6. Using data on concentrations of tPCBs in sediment, medians, means, 25th and 50th percentiles, and minimum 13 14 and maximum concentrations were calculated for each reach. Hazard quotients were calculated by dividing each of these summary statistics by the effects benchmark for benthic invertebrates 15 16 exposed to tPCBs in sediment. The sediment effects threshold used in the derivation of the HQs 17 was 3 mg/kg tPCB, which represents a concentration above which significant adverse responses 18 were observed in site-specific toxicity tests (Section 3 and Appendix D). The results are plotted 19 in Figure 12.2-1. These results indicate that significant risk was observed in all reaches of the 20 PSA, with HQs for tPCBs above 1 for both mean and median tPCB concentrations. Predicted 21 risks were greatest in the upstream (Reach 5A) and Woods Pond (Reach 6) sediment. Due to the 22 considerable small-scale variation in sediment tPCB concentrations, HQs for the reaches span 23 about 4 orders of magnitude (approximately 0.01 to 100). Because benthic invertebrates are 24 much less mobile than fish and wildlife, they do not spatially and temporally integrate their 25 exposures. Thus, the hazard quotient results for benthic invertebrates indicate that the majority 26 of individuals are at risk (i.e., HQ > 1), but that some individuals in less contaminated areas are 27 not.



WB-R = Whole body, reconstituted fish tissue.

Figure 12.2-1 Hazard Quotients for Aquatic Biota Exposed to tPCBs in the Housatonic River PSA

WB = Whole body fish tissue.

1 12.2.2.1.2 Amphibians

2 For amphibians, HOs were calculated for Reaches 5A, 5B, 5C, 5D, and 6, using methods similar 3 to those used for benthic invertebrates. Hazard quotients were calculated by dividing summary 4 statistics for vernal pool sediment concentrations by the effects benchmark for amphibians 5 exposed to tPCBs in sediment (3 mg/kg tPCBs) (Section 4 and Appendix E). This approach does 6 not address adult leopard frog exposures that likely occur in river and backwater sediment. The 7 results are plotted in Figure 12.2-1. These results indicate significant risk in all reaches of the 8 PSA, with HQs above 1 for both mean and median tPCB concentrations. Predicted risks were 9 greatest in the upstream (Reach 5A) vernal pool habitats. Because of the variation in sediment 10 PCB concentrations between the vernal pools, HQs for the subreaches span about 4 orders of 11 magnitude (approximately 0.01 to 100). The hazard quotient results for amphibians indicate that 12 the majority of individuals are at risk (i.e., HO > 1), with higher levels of risk (i.e., HO > 5) in a 13 large percentage of vernal pools (about 50% in Reaches 5A and 5B).

14 **12.2.2.1.3** Fish

For fish, HQs were calculated separately for the five representative warmwater species (Section 15 16 5 and Appendix F) by dividing summary statistics for exposure by the tissue effects benchmark 17 protective of all species of PSA fish (49 mg/kg tPCB; derived from site-specific toxicity studies). 18 The results are plotted in Figure 12.2-1. These results indicate that risk occurs in all reaches of 19 the PSA, with both mean and median HQs for tPCBs above 1 for adult fish (i.e., whole body 20 reconstituted tissue concentrations of some species). Predicted risks were greatest in adult fish 21 and in predator fish at the top of the food web. Due to the variation in fish tissue tPCB 22 concentrations, hazard quotients for the reaches span about an order of magnitude, with most HQ 23 values between 0.3 and 3; the lower bound of this range represents primarily younger age classes 24 that have not yet accumulated their maximum tPCB burdens and fish species near the lower end 25 of the food chain. Thus, the hazard quotient results for fish indicate that predatory species are at 26 risk (i.e., HQ > 1) once they reach their maximum adult tPCB concentrations. The ERA 27 indicates that these HQs are indicative of sublethal (e.g., reproductive and developmental) 28 responses to offspring; the pathway for the manifestation of effects is through the maternal 29 transfer of tPCBs to eggs. Acute mortality to adults is not expected for most fish.

In addition to tPCBs, fish HQs were derived and plotted for TEQ (Figure 12.2-2). The effects
 benchmark applied in this analysis was derived from the site-specific toxicity studies (42 ng/kg
 TEQ) (Appendix F). The magnitudes and probabilities of risk for TEQ are generally similar to
 tPCB risks.

5 **12.2.2.1.4 Summary**

6 For aquatic receptors (benthic invertebrates, amphibians, and fish), the HQs presented in Figures 7 12.2-1 and 12.2-2 are not conservative. Although sensitive species were considered in the 8 derivation of the effects thresholds, no additional safety factors were used to estimate the effects 9 metrics. The thresholds used in HQ calculations represent levels demonstrated to cause adverse 10 responses to organisms in site-specific studies. Thus, HQ exceedances of 1 are cause for 11 concern.

12 **12.2.2.2** Wildlife Assessment Endpoints

13 For wildlife, probabilistic HQs were calculated as follows:

14 The distributions from the Monte Carlo analyses for total daily intake of COCs by • 15 representative species were each divided by the corresponding effects metrics used to estimate risks in Sections 7 through 11 and Appendices G through K. In the case of a 16 dose-response curve effects metric (e.g., mink exposed to tPCBs), the effects metric 17 was specified as a uniform distribution of dose ranging from 10 to 20% effect. A 18 similar approach was used for NOAEL-LOAEL ranges (e.g., bald eagles exposed to 19 tPCBs), field-based effects metrics (e.g., tree swallows exposed to tPCBs), and 20 21 threshold ranges (e.g., kingfishers exposed to TEQ).

- A similar approach was used with the results of the probability bounds analysis,
 except that the effects metric was specified as a distribution-free range.
- The analyses were done for both tPCBs and TEQ.



Figure 12.2-2 Hazard Quotients for Fish Exposed to 2,3,7,8-TCDD TEQ in the Housatonic River PSA

- Modified box-and-whisker plots were developed for each representative species in the 1 2 PSA. For species with smaller foraging ranges, the analyses were done for different 3 areas within the PSA. Included in the plots (Figures 12.2-3 and 12.2-4) are the 4 median HQ from the Monte Carlo analysis (the thick line bisecting each box), the mean HQ from the Monte Carlo analysis (star symbol), the 25th and 75th percentile 5 6 HQs from the Monte Carlo analysis (the bottom and top of each box), the 10th 7 percentile HQ from the lower bound of the probability bounds analysis (bottom 8 whisker), and the 90th percentile from the upper bound of the probability bounds 9 analysis (top whisker).
- 10 Probabilistic HQ plots were also developed for tree swallows exposed to tPCBs using measured concentrations in 12-day nestlings (data from Custer 2002) as an estimate 11 of exposure. This was done to facilitate comparison of risks using the microexposure 12 13 modeling approach and using measured concentrations from birds in the PSA. 14 Insufficient data were available to perform a similar analysis for TEQ. For the 15 measured concentrations data, an empirical histogram was specified for each PSA 16 location using the available 3 years of data for the Monte Carlo portion of the 17 analyses. The empirical histograms were divided by the same threshold range as used for HQs based on the microexposure model outputs. For the probability bounds 18 19 analyses, 95% Kolmogorov-Smirnov confidence limits were calculated for each 20 empirical histogram. Both the upper and lower confidence limits were divided by the 21 same threshold range as used for HQs based on the microexposure model outputs. 22 Using the results of these analyses, modified box-and-whisker plots were developed 23 as previously described (Figures 12.2-3 and 12.2-4).
- In addition to plots developed for mink and otter exposed to tPCBs and TEQ using the results of literature-based dose-response curves, plots were also developed using the results of the mink feeding study conducted by Bursian et al. (2003). In this case, the denominator was the NOAEL to LOAEL range from Bursian et al. (2003), rather than the 10 and 20% effects doses from the literature-based dose-response curve. Otherwise, the approach was as described above. The results are shown in Figures 12.2-3 and 12.2-4.
- For wildlife species other than tree swallow, mink, and river otter, it was not possible to derive field-based or feeding-study-based HQs because the required data were not available.
- Unlike traditional HQs, the probabilistic HQs presented in Figures 12.2-3 and 12.2-4 are not biased to be conservative. No safety factors were used to estimate the effects metrics (except in the case of the bald eagle), and uncertainties regarding exposure model inputs were explicitly propagated through the probability bounds and Monte Carlo analyses. Thus, HQ exceedances of 1 are cause for concern.



Figure 12.2-3 Hazard Quotients for Wildlife Exposed to tPCBs in the Housatonic River PSA



Figure 12.2-4 Hazard Quotients for Wildlife Exposed to 2,3,7,8-TCDD TEQ in the Housatonic River PSA

MK01|O:\20123001.096\ERA_PB\ERA_PB_12.DOC

1

2

7/11/2003

2 • Wildlife risks from tPCBs are generally higher than risks from TEQ by 1 to several orders of magnitude. 3 4 The comparison of HO plots for tree swallows indicates that risks are higher using the • 5 results of the microexposure model than is the case using measured concentrations in 6 tree swallow nestlings. Thus, the microexposure model appears to be overestimating 7 exposure for tPCBs. The HQ plots using measured concentrations in nestlings 8 supports the weight-of-evidence conclusion that risks of tPCBs to tree swallows is 9 low (see Section 7, Appendix G and Table 12.2-1). 10 • Wildlife risks from tPCBs and TEQ are highest for mink and river otter, with mean 11 and median HQs between 100 and 500 for tPCBs, and between 5 and 10 for TEQ using the results of the literature-based dose-response curve. The HQs for tPCBs 12 13 were lower when the results of the feeding study were used to derive the effects 14 range. However, this difference is no longer apparent for TEO, supporting the 15 hypothesis presented in Section 9 and Appendix I that the congener mixture in Housatonic River prey in the PSA is less toxic than the commercial mixtures Aroclor 16 17 1260 and 1254. 18 The risks from tPCBs and TEQ to most wildlife species are uncertain to the extent 19 that the bottom or top whiskers span 1. The whiskers are the extreme representations 20 of uncertainty because they are tail outputs from the probability bounds analyses, a 21 technique designed to propagate all forms of uncertainty (e.g., inability to precisely 22 specify distribution type or parameter values for a distribution). Thus, the boxes in 23 Figures 12.2-3 and 12.2-4 should be interpreted as representing a reasonable range 24 within which the HQ estimate occurs for the receptor of interest, and the whiskers 25 should be interpreted as representing the extremes within which the HO could occur.

In summary, the wildlife HQs presented in Figures 12.2-3 and 12.2-4 indicate the following:

The HQ analyses for aquatic biota and wildlife indicate that risks of tPCBs vary widely between representative species and assessment endpoints. Section 12.2.3 explores the fundamental reasons why this might occur.

29 **12.2.3** Risk Assessment Downstream of Woods Pond

Because of the reduced levels of contaminant concentrations downstream of the PSA and significant shifts in aquatic habitat types associated both with river gradient and location of dams, a different approach than that applied in the PSA was followed to assess potential ecological risks of tPCBs to biota in areas downstream of Woods Pond. The assessment of potential ecological risks was conducted using mapping (GIS) techniques and threshold concentrations that indicate potential risk for six taxonomic groups selected based on the

outcome of the evaluations performed in the PSA and the habitat characteristics found
 downstream. These groups are benthic invertebrates, amphibians, warmwater fish, trout, mink,
 otter, and bald eagles.

For each of these groups, a maximum acceptable threshold concentration (MATC) for tPCBs was developed based primarily on the detailed risk assessment performed for the PSA. Each MATC was then compared to media-specific data for areas downstream of Woods Pond to Long Island Sound. Areas with MATC exceedances, indicating potential risk, were plotted on maps of the river. The methods used for each of the six representative groups and the results of the analyses are discussed in the following sections (see Table 12.2-15 for a summary of results).

Where insufficient data for the medium of interest were available for a reach to estimate the risk for a species, other available data were examined to determine the likelihood of the concentrations of tPCBs in the medium of interest being high enough to pose a risk. In all cases, concentrations were lower than in other reaches with available data for the medium of interest; therefore, it is unlikely that the receptor is at risk in those reaches for which data were not available.

16 12.2.3.1 Benthic Invertebrates

17 For benthic invertebrates, the medium of interest was river sediment. An MATC of 3 mg/kg 18 tPCBs was used as a conservative measure of the potential for adverse affects to benthic 19 invertebrates downstream of Woods Pond (see Section 3 and Appendix D for a description of the 20 derivation of the MATC). This concentration was developed using multiple lines of evidence 21 (e.g., benthic community studies, in situ and laboratory toxicity testing, bioaccumulation testing, 22 Sediment Quality Triad) and was selected as the concentration at which some sensitive endpoints 23 exhibited adverse responses, but the magnitude of responses was not large. Above a 24 concentration of 3 mg/kg tPCBs, numerous endpoints indicated ecologically significant 25 responses, with many LC_{50}/EC_{50} values falling in the range of 3 to 30 mg/kg tPCBs.

The MATC of 3 mg/kg tPCBs was compared to recent surficial sediment data downstream of Woods Pond, and the results were plotted (Figure 12.2-5) to indicate samples above and below

Summary of the Assessment of Risks Conducted for Biota Exposed to tPCBs in the Lower Housatonic River Below Woods Pond

	Potentially at Risk (MATC Exceeded)						
Reach	Benthic Invertebrates	Amphibians	Warmwater Fish	Trout	Mink	Otter	Eagle
7	Yes	Yes	No	Yes	Yes	Yes	No
8	Yes	Yes	No	No suitable habitat	Yes	Yes	Yes*
9	No	Yes	No	Yes	Yes	Yes	No
10	No	Limited suitable habitat	No	No suitable habitat	Yes	Yes	No
11	No	Limited suitable habitat	No	No	No	Yes	No
12	No	Limited suitable habitat	No	No	No	Yes	No
13	No	Limited suitable habitat	No	No suitable habitat	No	No	No
14	No	Limited suitable habitat	No	No suitable habitat	No	No	No
15	No	Limited suitable habitat	No	No suitable habitat	Insufficient data but unlikely	No	No
16	No	Limited suitable habitat	Insufficient data but unlikely	No suitable habitat	Insufficient data but unlikely	Insufficient data but unlikely	Insufficient data but unlikely

5 6

1 2

3 4

* Reach 8 is Rising Pond. Although eagles would be at risk foraging there, Rising Pond is smaller than an eagle's foraging area; therefore, there is uncertainty about this risk estimate.

the MATC. Inverse distance weighting was used to interpolate sediment concentrations between
discrete sampling points, and the potential for risk to benthic invertebrates was shown by shading
the corresponding sections of the river channel (Figure 12.2-5).

In general, potential risks to benthic invertebrates occur in limited areas downstream of Woods
Pond to Rising Pond. These areas are depositional and tend to have higher concentrations of
tPCBs. Below Rising Pond, sediment does not contain concentrations of tPCBs that represent a
potential risk to benthic invertebrates.

8 **12.2.3.2** Amphibians

9 Many species of amphibians are primarily exposed to PCBs in the floodplain, particularly in 10 vernal pools and other low-lying wet areas. A sediment/floodplain soil MATC of 3.0 mg/kg 11 tPCB (dry weight) was derived from the amphibian risk assessment conducted for the PSA 12 (Section 4 and Appendix E). Inverse distance weighting was used to interpolate tPCB 13 concentrations to the limit of the 100-year floodplain (10-year floodplain contours are not 14 available downstream of Woods Pond) using the 0 to 6 inch (0 to 15 cm) depth data from the 15 floodplain downstream of Woods Pond; a separate analysis was conducted for sediment in a 16 manner analogous to that described above for benthic invertebrates.

17 Areas where the 3.0 mg/kg tPCB threshold was exceeded, indicating potential risks to 18 amphibians due to PCBs in floodplain soil and sediment, are indicated in Figures 12.2-6 and 19 12.2-7. Several large floodplain areas of potential risk are located in the area between Woods 20 Pond and Rising Pond, with only small isolated areas of potential risk downstream of Rising 21 Pond. Downstream of the Massachusetts/Connecticut state line the risk mapping for amphibians 22 was conducted for sediment only because the concentrations in floodplain soil had decreased 23 below the MATC throughout Reach 9 upstream, and the extent of the floodplain is limited in 24 Connecticut.



O:\gepitt\aprs\era_species.apr | layout - benthos d4-7 | o:\gepitt\epsfiles\plots\in\benthos_risk_12-2-5.eps | 10:42 AM, 7/10/2003 |



| O:\gepitt\aprs\era_species.apr | layout - amphibs FLD e4-4 | o:\gepitt\epsfiles\plots\in\amphibs_fid_risk_12-2-6.eps | 10:38 AM, 7/10/2003 |



O:\gepitt\aprs\era_species.apr | layout - amphibs SEDS e4-5 | o:\gepitt\epsfiles\plots\in\amphibs_sed_risk_12-2-7.eps | 10:40 AM, 7/10/2003 |

1 12.2.3.3 Warmwater Fish

2 As was done for the PSA, risk to fish was evaluated based on concentrations of tPCBs in fish 3 tissue. An MATC of 49 mg/kg tPCB in tissue (whole body, wet weight) developed for the PSA 4 based on site-specific effects (Section 5 and Appendix F) was applied to areas downstream of 5 Woods Pond using the available (e.g., bass, perch, sunfish) tissue data for warmwater species. 6 Each downstream reach (Reaches 7 through 16) was evaluated as a unit, and the mean adult fish 7 tissue concentration in each reach was compared with the MATC to determine potential risk. 8 Only data collected since 1998 were used in this analysis. The results are shown in Figure 9 12.2-8.

10 In some cases, it was necessary to extrapolate from the available raw data to develop an adult 11 tissue concentration for comparison with the MATC. Young-of-year (YOY) largemouth bass 12 data were extrapolated up to estimated adult concentrations by applying a factor of 3.5; similarly, 13 yellow perch YOY data were scaled up by a factor of 2.5. Both ratios were calculated from the 14 more extensive database available for the PSA. In addition, the majority of filet data were 15 extrapolated to estimated whole body concentrations using the factor of 2.3 developed by 16 Bevelhimer et al. (1997). Brown bullhead filet data were extrapolated to estimated whole body 17 concentrations using the factor of 1.5 developed by EPA (1999a).

18 The evaluation of risk to warmwater fish species downstream of Woods Pond indicated that no 19 risks were indicated in any of the reaches below the PSA.

20 12.2.3.4 Trout

21 Trout were evaluated separately from warmwater fish species because of significant differences 22 in habitat requirements and in the sensitivity of some trout species to tPCBs documented in the 23 literature. Trout also tend to have higher tPCB concentrations because of their higher lipid 24 content. The site-specific studies did not indicate large differences between the effects threshold 25 for rainbow trout and warmwater species (Section 5 and Appendix F), but the strain of rainbow 26 trout used in the site-specific toxicity tests (Buckler 2002) is less sensitive than other strains used 27 widely in toxicity testing. Furthermore, there are other trout species found downstream of the 28 PSA (e.g., brown trout) for which sensitivity has not been assessed. Given





| O:\gepitt\aprs\era_species.apr | layout - fish warm f4-10 | o:\gepitt\epsfiles\plots\in\warm_fish_12-2-8.eps | 10:48 AM, 7/10/2003 |

that some trout species have been documented to have greater sensitivity to PCBs and dioxins (Walker et al. 1994; Zabel et al. 1995), relative to the warmwater species considered in the development of the 49 mg/kg tPCB warmwater MATC, a factor of 4 was applied in recognition of these potential interspecies differences. Therefore, a tissue MATC of 12 mg/kg tPCBs (whole body, wet weight) was derived for trout.

6 Because of the more limited database for trout, a number of extrapolations were necessary to 7 convert available warmwater fish data and/or trout filet data to estimated whole body 8 concentrations for trout. These extrapolations included all of the extrapolation factors discussed 9 above for warmwater fish species and an additional extrapolation factor of 2.0 to estimate trout 10 whole body tPCB concentrations from measured tPCB concentrations in warmwater fish 11 samples. This factor was developed from the available smallmouth bass and trout data from the 12 Trout Management Area (Reach 11, in part) in Connecticut. The Amrhein et al. (1999) factor of 13 1.47 was used to convert the trout filet data to estimated whole body concentrations.

The results of this evaluation are shown in Figure 12.2-9. Trout are potentially at risk in Reaches 7 and 9, but not in reaches with suitable habitat downstream. This assessment has high uncertainty due to the number of extrapolations required and the low magnitude of exceedance of the MATC value. Potential risk to trout was not evaluated in Reach 8, Reach 10, and downstream of Reach 12 due to lack of suitable trout habitat.

19 12.2.3.5 Mink

An MATC for mink of 2.65 mg/kg tPCBs in fish (whole body, wet weight) was derived from the the geometric mean of the NOAEL and LOAEL developed by Bursian et al. (2003) in a sitespecific study of the toxicity of a diet containing Housatonic River fish to mink (Section 9 and Appendix I).

Habitat suitability for mink was determined for the reaches downstream of the PSA (Appendix A). According to this analysis, potential mink habitat is ubiquitous along Reaches 7 to 16 and includes all areas except high gradient stream, calcareous rock cliff, cultural grassland, agricultural cropland, and residential/industrial development.





| O:\gepitt\aprs\era_species.apr | layout - fish cold f4-11 | o:\gepitt\epsfiles\plots\in\trout_risk_12-2-9.eps | 10:46 AM, 7/10/2003 |

Mean fish concentrations were calculated for each river reach downstream of Woods Pond using available whole body fish tissue data from samples collected since 1998. The analysis was restricted to fish with an overall body length between 7 and 20 cm, corresponding to the size commonly preyed on by mink. In some cases, it was necessary to use YOY data for bass and perch, and these were extrapolated to adult concentrations using the factors discussed above.

6 For this analysis, it was assumed that the mink were exposed to the mean fish concentration in 7 the downstream reaches for 59 % of the diet because the mink diet contains on average 23% fish 8 and 36% invertebrates, the majority of which are crayfish. No crayfish data were available for 9 the downstream reaches; however, within the PSA, crayfish tPCB concentrations were similar to 10 fish concentrations in the size range consumed by mink. Therefore, the assumption of 59% of 11 the dietary exposure at the mean fish concentrations in the downstream reaches is reasonable. 12 This risk estimate likely underestimates the mink exposure in the downstream reaches, as it was 13 assumed that the concentration of tPCBs in the remaining 41% of the diet was 0.

14 The results of the evaluation for mink are shown in Figure 12.2-10. Potential risk to mink due to 15 consumption of contaminated fish occurs from the Woods Pond Dam downstream to the Great 16 Falls Dam, corresponding to Reaches 7 through 10.

17 12.2.3.6 River Otter

18 The mink MATC of 2.65 mg/kg tPCB in fish (whole body, wet weight) was also used for river 19 otter. Potential river otter habitat downstream of Woods Pond is less abundant than for mink and 20 is associated with larger wetland systems, with slower flowing water, or with impounded water. 21 Any places where the river is impounded, or near a lake or pond, there is potential river otter 22 habitat. Mean fish concentrations were calculated for such areas in river reaches downstream of 23 Woods Pond using available whole body fish tissue data from samples collected since 1998. The 24 analysis was restricted to fish with an overall body length between 5 and 50 cm, corresponding 25 to the size commonly preved on by otter. In some cases, it was necessary to use YOY data for 26 bass and perch, and these were extrapolated to adult concentrations using the factors discussed 27 above.



| O:\gepitt\aprs\era_species.apr | layout - mink i4-15 | o:\gepitt\epsfiles\plots\in\mink_risk_12-2-10.eps | 10:47 AM, 7/10/2003 |
For this analysis it was assumed that otter were exposed to the mean fish concentrations in the downstream reaches for 100% of the diet because the majority (80%) of the otter diet is fish, with most of the remainder (8 to 20%) composed of crayfish. No crayfish data were available for the downstream reaches; however, within the PSA, crayfish tPCB concentrations were similar to fish concentrations in the size range consumed by otter. Therefore, the assumption of 100% of the dietary exposure at the mean fish concentrations in the downstream reaches was reasonable.

8 The results of this evaluation for otter are shown in Figure 12.2-11. Potential risk to otter due to 9 consumption of contaminated fish occurs from the Woods Pond Dam downstream to the Bulls 10 Bridge Dam, corresponding to Reaches 7 through 12.

11 12.2.3.7 Bald Eagle

12 An MATC of 30.4 mg/kg tPCBs (whole body fish tissue, wet weight) (Appendix K) was 13 developed for wintering bald eagles, assuming that the eagle diet was composed of 78% fish, and 14 that the remainder of the diet included other non-aquatic species that were assumed, for the 15 purpose of this analysis, to be uncontaminated.

This concentration was compared with the available fish tissue concentration data from areas downstream of the PSA, in some cases applying scaling factors as discussed above for other receptors. Only data from samples collected since 1998 were used, and fish less then 12 cm total length were excluded from the analysis (unless appropriately scaled) to reflect the common size of fish preyed on by eagles.

21 A more in-depth analysis was performed for Reaches 14 and 15 where bald eagles have nested. 22 Bald eagles on average consume a summer diet consisting of 78.2% fish, 16.3% birds, and 5% 23 mammals (see Section K2.1.5). Mammal and bird tPCB concentrations were not available for 24 downstream reaches. Total PCB concentrations for these prey items were estimated in three 25 ways to give high, moderate, and low concentrations. High concentrations assumed that 26 waterfowl and mammals from downstream would have tPCB concentrations equal to those in the 27 PSA. Low concentrations assumed that waterfowl and mammals from downstream would have 28 tPCB concentrations of zero. A moderate concentration was developed by determining fish-to-



| O:\gepitt\aprs\era_species.apr | layout - otter i4-16 | o:\gepitt\epsfiles\plots\in\otter_risk_12-2-11.eps | 10:43 AM, 7/10/2003 |

mammal and fish-to-bird ratios based on concentrations in the PSA. Mammal tPCB
concentrations in the PSA are on average 75% of the total fish concentration, and waterfowl
tPCB concentrations averaged 15% of the total fish concentration. Therefore, moderate tPCB
concentrations downstream were 0.539 mg/kg for mammals and 0.108 mg/kg for birds.

5 The results of the evaluation are shown in Figure 12.2-12. Potential risks to bald eagles from 6 consuming contaminated fish in areas downstream of Woods Pond are restricted to Reach 8, 7 corresponding to Rising Pond. However, Rising Pond is smaller than the typical eagle foraging 8 area so this estimate of risk is conservative. In addition, the more in-depth analysis specific to 9 Reaches 14 and 15 also did not show risk in the foraging area of the nesting bald eagles.



| O:\gepitt\aprs\era_species.apr | layout - eagle k4-5 | o:\gepitt\epsfiles\plots\in\eagle_risk_12-2-12.eps | 10:49 AM, 7/10/2003 |

1 12.3 SPECIES SENSITIVITY AND MECHANISMS OF TOXICITY

There is a large amount of variability in the toxicokinetic responses of different species to tPCBs
and 2,3,7,8-TCDD equivalence (TEQ). Numerous studies have shown that tPCBs and TEQ may
cause a variety of adverse effects (e.g., Bosveld and Van den Berg 1994; Newsted et al. 1995;
Van den Berg et al. 1998). Effects may include:

6 Lethality. 7 Hepatic lesions. 8 Immunotoxicity. 9 Tumor promotion. • 10 • Adverse effects on reproduction. Induction of drug-metabolizing enzymes. 11 12

How PCB and TEQ congeners cause these effects, and the ability of different species to defend against these contaminants is less clear. A brief description of the primary toxic mechanism of coplanar PCBs, dioxins, and furans is provided in Section 12.2.3.1 (see also Section 2), as is information on the relative sensitivities of biota to tPCBs and TEQ.

17 Although sensitivity to COCs undoubtedly explains some of the differences in effects and 18 resulting risk experienced by biota in the PSA (see Figures 12.2-1 through 12.2-4), other factors 19 also play a role. For example, higher trophic level biota that may forage exclusively in the PSA 20 (e.g., kingfishers, mink) have higher exposures to tPCBs and TEQ than do biota with foraging 21 areas of which only a portion is in the PSA (e.g., red fox).

Also, the composition and toxicity of the congener mixture that biota are exposed to changes
with trophic level. The latter issue is briefly discussed in Section 12.2.3.2 and in more detail in
Appendix C.7.

25 12.3.1 Mechanism of Action and Sensitivity of Species to tPCBs and TEQ

Some chlorinated PCBs, dioxins, and furans belong to a large class of chemicals called planar chlorinated hydrocarbons (PCHs) that are regularly detected in the environment. PCHs have a common structural relationship that includes lateral halogenation (i.e., the addition of chlorine to the compound) and the ability to assume a planar conformation. This structure is important as it leads to a common mechanism of action in many animal species involving binding to the aryl hydrocarbon (Ah) receptor and elicitation of an Ah-receptor-mediated biochemical and toxic
response (Van den Berg et al. 1998; Newsted et al. 1995; Safe 1994). The planar conformation
is the factor that controls the ability of the the chemical to bind with the Ah receptor (Birnbaum
and Devito 1995; Newsted et al. 1995). The Ah receptor facilitates the translocation of PCHs
into the nucleus of affected cells and the binding of the PCH-Ah receptor complex to sites on the
DNA (Newsted et al. 1995).

Exposure of PCBs and other organic toxins in vertebrates elicits a response of the cytochrome P450 system with associated mixed function oxidases (MFO). MFOs enhance the elimination of some hydrophobic chemicals through a series of oxidative reactions (Eisler 2000). However, the MFO system is less capable of breaking down congeners with chlorine substitution at the 2, 3, 7, and 8 positions. As a result, these coplanar congeners show resistance to metabolic breakdown in many higher organisms (Bosveld and Van den Berg 1994).

13 The development of the cytochrome P450 system varies between species of vertebrates. 14 Therefore, some species may be more sensitive to tPCBs and TEQ than others, even within 15 taxonomic families. For example, mustelids may vary widely in sensitivity to PCBs (Leonards et 16 al. 1997). Mink are among the most sensitive species to PCBs known (Aulerich et al. 1985; 17 Giesy and Kannan 1998). Conversely, ferrets are much less sensitive to PCBs than mink 18 (Bleavins et al. 1980). There are, however, few data available for other mustelid species. Foxes 19 and dogs have been shown to have an unusual P450 isoenzyme that allows them to degrade 20 PCB-153 more efficiently than rats and monkeys (Georgii et al. 1994). In general, fish are less 21 capable of metabolizing PCBs than most birds and mammals (Van den Berg et al. 1998). 22 Despite their reduced ability to metabolize PCBs, fish are relatively insensitive to mono-ortho 23 PCBs, compared to birds and mammals (Van den Berg et al. 1998).

Fewer studies have been conducted on amphibians than on mammals, fish, and birds. The ability of amphibians to metabolize organic contaminants appears to be comparable to that of fish, but lower than that of rats (Eisler 2000). In amphibians, effects on the neutrophil function (i.e., immunosuppression) may be important (Angermann and Matsumura 1999). There is evidence that indicates that amphibians contain the Ah receptor, but it is not as well described because of limited research (Jung 1997). PCB-126 induces cytochrome P450 activity in both leopard frogs and green frogs (Huang et al. 2001). Amphibians have a cytochrome P450 mixed function oxidase that is less active and well developed than mammals, but that does not appear to be significantly different from other vertebrates (Eisler 2000). Benthic invertebrate toxicity is not mediated by an Ah receptor mechanism, and TEF systems have not been developed for amphibians or benthic invertebrates.

6 CCME (1999) reviewed the toxicology literature for mammals and birds and suggested that bird 7 species may be less sensitive to the effects of PCBs than mammals. Some bird species such as 8 tree swallows appear to be quite tolerant of elevated exposures to tPCBs and TEQ (Custer et al. 9 1998; Bishop et al. 1995, 1999; McCarty and Secord 1999). Substantial differences in sensitivity 10 to PCBs between bird species have also been noted. Barron et al. (1995) determined that 11 differences in the genetic expression of the Ah receptor were the dominant factor explaining differences in PCB sensitivity of the bird species examined. Brunström (1988, 1989) examined 12 13 the sensitivity of numerous species of avian embryos to coplanar PCBs and concluded that 14 interspecies differences in sensitivity were due to differences in the Ah receptor ligands.

15 Therefore, the available literature indicates that there are differences in sensitivity of biota, and 16 that these differences may be partially attributed to differences in development of the cytochrome P450 system and other factors. The differences in sensitivity of biota partially 17 18 explain why, for example, mink are experiencing much greater effects from exposure to tPCBs 19 and TEQ in the PSA than are kingfishers (Figures 12.2-3 and 12.2-4), despite the two species 20 having similar diets. Similarly, tree swallows and small-footed myotis have similar diets, yet the 21 more tolerant tree swallows are likely to experience lower risks from exposure to tPCBs and 22 TEQ than does small-footed myotis (Figures 12.2-3 and 12.2-4).

Although differences in sensitivity of biota can partly be explained on the basis on differential development of Ah receptor and cytochrome P450 systems, toxicity from non-coplanar congeners not associated with these systems is also important. Observations of effects in aquatic receptors that are either unrelated to Ah receptor interactions (e.g., benthic invertebrates) or are greater than would be predicted on the basis of congeners with TEF values only (e.g., fish) are evidence of this. Detailed knowledge of PCB toxicity for all 209 PCB congeners does not exist; differential toxicity to non-coplanar congeners may explain some of the interspecies differences
 in toxicity observed.

3 **12.3.2** Congener Composition and Toxicity to Biota

4 Environmental degradation (or weathering) of PCH congeners varies due to the unique physical/chemical properties of each congener (Cogliano 1998). This can cause differences 5 6 between the congeners detected in environmental samples and the congener makeup of the 7 original product or Aroclor (Cogliano 1998; Van den Berg et al. 1998). In the Housatonic River 8 PSA, PCB composition exhibits little spatial variability within a medium (e.g., in sediment 9 between reaches), although there are shifts in composition across media (Appendix C.7). 10 Between receptor groups, PCB congener composition may exhibit considerable variation. The 11 change in the congener composition in prey tissue can produce differences in toxicological 12 responses in exposed predator species relative to the effects observed in the laboratory for 13 species exposed to technical mixtures (e.g., Aroclor 1254 or 1260).

14 In the site-specific fish studies conducted for this assessment, the congener composition in fish 15 was found to be more toxic than would be expected from studies exposing fish to Aroclor 1254 16 and 1260 commercial mixtures (Appendix F). Conversely, the results of the mink feeding study 17 (Appendix I) indicated that the congener composition in fish tissue from the PSA was less toxic 18 than would be expected from mink toxicity studies conducted with mixtures expected to be 19 similar. For example, the highest dose treatment in the mink feeding study caused effects on 20 survival of mink kits that would have been expected at about a 4-fold lower dose based on 21 chronic feeding studies conducted with Aroclor 1254 (see Figure I.3-5 in Appendix I).

1 12.4 BROADER IMPLICATIONS

2 The weight-of-evidence assessments briefly described in Sections 3 through 11, and in more 3 detail in Appendices D through K, indicate that COCs in the PSA of the Housatonic River, 4 particularly tPCBs, are causing risks to many of the species chosen to represent the assessment 5 endpoints (see Figures 12.2-1 through 12.2-4, Table 12.2-1). Risks from COCs, however, may 6 potentially extend beyond adverse effects to survival, growth, and reproduction of representative 7 species. The purpose of this section is to explore the implications of the risks of COCs to 8 representative species demonstrated in the preceding sections. This section begins by extending 9 the ecological risk assessment to species that occur in the Housatonic River watershed, but that 10 had not been considered explicitly in the quantitative ecological risk assessments previously 11 described in Sections 3 through 11. This section is followed by a general discussion of the 12 possible broader ecological implications of this risk assessment.

13 **12.4.1** Implications for Other Species in the Primary Study Area

The purpose of this section is to qualitatively compare exposure of the representative species to other species that were identified in the Ecological Characterization (Appendix A) to occur in the PSA for tPCBs and TEQ. The major factors that influence exposure to tPCBs and TEQ and that were considered in the analysis include:

- 18 Dietary composition.
 - Foraging behavior and home range.
 - Size, metabolism, and life history characteristics.
- 20 21 22

19

Sensitivity to COCs.

The following sections briefly compare these factors between the representative species and other species in their foraging groups. The comparison highlights similarities and differences, and their potential to influence exposure and thus risks to tPCBs and TEQ. This comparison does not consider differences in sensitivity between representative species and other species in their foraging groups because toxicity data to make this comparison are lacking. Table 12.4-1 summarizes this qualitative risk assessment. More discussion is presented in Appendices D through K.

	Representative							Level of	Risk Com	pared to Re	presentative	Species	
Category	Species and Risk Category in PSA*	Other Species	Size	Residency	Foraging/ Home Range	Diet	Life History/ Miscellaneous	Lower	Lower to Similar	Similar	Similar to Higher	Higher	References
									1	[
Top Predator Fish	Largemouth bass/ Intermediate to		10-16 inches (21-41 cm)	Year- round	n/a	Aquatic insects, fish, crayfish				n/a			Hartel et al. 2002
	High	Smallmouth bass	8-13 inches (20-33 cm)	Year- round	n/a	Aquatic invertebrates, fish	Prefers cooler, clearer, rockier areas than largemouth bass (LMB), but similar			Х			Hartel et al. 2002
		Black crappie	8-12 inches (20-30 cm)	Year- round	n/a	Aquatic invertebrates, fish	Prefers cooler, clearer, rockier areas than LMB, but diet is similar			Х			Hartel et al. 2002
		Rock bass	6-8 inches (15-20 cm)	Year- round	n/a	Aquatic invertebrates, fish, crayfish	Diet and some habitat preferences are similar, particularly to young (3-4 y.o.) LMB			Х			Hartel et al. 2002
		Chain pickerel	15-24 inches (38-61 cm)	Year- round	n/a	Invertebrates, fish	Similar diet compared to LMB			Х			Hartel et al. 2002
Omnivorous Bottom Feeders	Brown bullhead/ Intermediate to High		8-14 inches (20-36 cm)	Year- round	n/a	Animal and plant material (up to 40% plants, up to 60% filamentous algae)				n/a			Hartel et al. 2002; Gunn et al. 1977
		Yellow bullhead	8-12 inches (20-30 cm)	Year- round	n/a	Insects, crustaceans, mollusks, small fish, plant material (up to 40% plants)	Similar habitat, although typically prefers clearer water than brown bullhead. Diet is similar			X			Hartel et al. 2002

	Representative							Level of	f Risk Com	pared to Re	presentative	e Species	
Cottooren	Species and Risk Category	Other	6:	Desidentes	Foraging/ Home	D:-4	Life History/		Lower to	~	Similar to		Defense
Category	in PSA*	Species	Size	Residency	Kange	Diet	Miscellaneous	Lower	Similar	Similar	Higher	Higher	Keterences
		Common carp	24 inches (61 cm)	Year- round	n/a	Animal and plant material	Habitat and diet similar to brown bullhead			Х			Hartel et al. 2002
		Goldfish	5-13 inches (13-33 cm)	Year- round	n/a	Animal and plant material	Habitat and diet similar to brown bullhead			Х			Hartel et al. 2002
	White sucker/ Intermediate to High		12-24 inches (30-61 cm)	Year- round	n/a	Aquatic invertebrates, larval insects, detritus				n/a			Hartel et al. 2002
		Longnose sucker	12-15 inches (30-38 cm)	Year- round	n/a	Aquatic invertebrates, algae	Prefers cooler, cleaner stream reaches than white sucker. Diet is similar to white sucker			Х			Hartel et al. 2002
		Creek chubsucker	9 inches (23 cm)	Year- round	n/a	Omnivorous	Habitat and diet similar to white sucker			Х			Hartel et al. 2002
Forage Fish	Pumpkinseed/ Intermediate to High		4-5 inches (10-13 cm)	Year- round	n/a	Aquatic invertebrates				n/a			Hartel et al. 2002
		Bluegill	5-7 inches (13-18 cm)	Year- round	n/a	Aquatic invertebrates, some small fish	Habitat and diet similar to pumpkinseed			Х			Hartel et al. 2002
		Redbreast sunfish	4-8 inches (10-20 cm)	Year- round	n/a	Larvae and adult aquatic insects, terrestrial insects, some small fish	Habitat and diet similar to pumpkinseed			Х			Hartel et al. 2002

	Representative							Level of	f Risk Com	pared to Re	presentativo	e Species	
Category	Species and Risk Category in PSA*	Other Species	Size	Residency	Foraging/ Home Range	Diet	Life History/ Miscellaneous	Lower	Lower to Similar	Similar	Similar to Higher	Higher	References
Amphibians	Wood frog/ High		18 g	Year- round, larvae: 3 months	64.5 m ²	Insects, beetles, flies, slugs, snails, spiders, bugs, moth larvae, and earthworms	Small home range, but establishes territories >1,000 m from breeding pools			n/a			DeGraaf and Yamasaki 2001; Hunter et al. 1999
		Northern spring peeper	1.0-2.7 g	Year- round, larvae: 3 months	23 m ²	Spiders (up to 50%), ants, beetles, mites, ticks, springtails, caterpillars, slugs, and snails	Territories usually established near suitable breeding sites. Late summer/fall migrations to hibernation sites may be further away				X		DeGraaf and Yamasaki 2001; Hunter et al. 1999
		Spotted salamander	14 g	Year- round, larvae: 3 months	10-14 m ²	Forest floor invertebrates: earthworms, slugs, snails, spiders, millipedes, centipedes, larval and adult insects	Home ranges usually established within 200 m of breeding site				X		Petranka 1998; DeGraaf and Yamasaki 2001; Hunter et al. 1999
Amphibians (cont.)	Wood frog/ High (cont.)	Jefferson salamander	11 g	Year- round, larvae: 3 months		Small invertebrates, worms, spiders, insects, crustaceans	Home ranges typically within 250 m from breeding pond, but have been recorded up to 624 m away				Х		Petranka 1998; DeGraaf and Yamasaki 2001; Hunter et al. 1999
	Northern leopard frog/ High		38 g	Year- round, larvae: 3 months	5 to 53 m nightly movement	Beetles (up to 50%), lepidopteran larvae, bugs, grasshoppers, ants, spiders, crayfish, snails	Semi-terrestrial spending summer month in damp fields and woods, hibernates and breeds in permanent bodies of water			n/a			DeGraaf and Yamasaki 2001; Hunter et al. 1999

	Representative							Level of	f Risk Com	pared to Re	presentative	e Species	
Category	Species and Risk Category in PSA*	Other Species	Size	Residency	Foraging/ Home Range	Diet	Life History/ Miscellaneous	Lower	Lower to Similar	Similar	Similar to Higher	Higher	References
		Pickerel frog	Not found	Year- round, larvae: 3 months	Not found	Insects, beetles, caterpillars, true bugs, ants, spiders, snails, crayfish, and amphipods	Habitat preferences and feeding habits similar to leopard frog				Х		DeGraaf and Yamasaki 2001; Hunter et al. 1999
		Green frog	30-70 g	Year- round, larvae: 1-2 years	61 m ² home range, 2-3 m breeding territory	Adults: plants, spiders, beetles, true bugs, wasps and bees, mosquitoes, flies, midges and gnats, mayflies, moths, and butterflies	Green frogs are more aquatic than leopard frogs. They do enter the floodplain to access seasonally available food resources				X		DeGraaf and Yamasaki 2001; Hunter et al. 1999; Stewart and Sandison 1973; Jenssen and Klimstra 1966.

	Representative							Level of	f Risk Com	pared to Re	presentativo	e Species	
Category	Species and Risk Category in PSA*	Other Species	Size	Residency	Foraging/ Home Range	Diet	Life History/ Miscellaneous	Lower	Lower to Similar	Similar	Similar to Higher	Higher	References
Amphibians (cont.)	Northern leopard frog/ High (cont.)	Eastern newt	2-3 g	Year- round, larvae: 2 months, efts 3-7 years	Adults: captured within 7 m of original capture sites, efts: 270 m ² up to 800 m from breeding sites	Adults: mayflies, damselflies, dragonflies, mosquitoes, midges, gnats, water fleas, amphipods, bivalves, and clams Newt larvae efts: snails, slugs, mites, ticks, beetles, beetle larvae, flies, mosquitoes, midges, gnats, maggots, wasps, and bees	Larval period is spent in pools; metamorph into terrestrial eft stage that lasts 2-7 years. A second metamorphosis occurs when adults return to pools and transform into aquatic adults				X		Petranka 1998; DeGraaf and Yamasaki 2001; Hunter et al. 1999; Burton 1977; MacNamara 1977; Bellis 1968; Healy 1975
Insectivorous Birds	Tree swallow/ Intermediate to High		20.1 g (range 16.5-25.5 g)	6 months	Defend 10-15 m around nest; feed within 300-400 m of nest	Primarily emergent insects, such as flies, mosquitoes, midges, gnats, mayflies, and beetles	Feed on emergent insects over bodies of water			n/a			DeGraaf and Yamasaki 2001; Robertson et al. 1992; Ehrlich et al. 1988; Martin et al. 1951
		Bank swallow	13.5 g	5 months	Territory limited to immediate vicinity of the nest entrance	Almost entirely insects	Nest in exposed and eroding riverbanks and in gravel pits				X		DeGraaf and Yamasaki 2001; Ehrlich et al. 1992

	Representative							Level of	f Risk Com	pared to Re	presentative	e Species	
	Species and Bisk Catagory	Other			Foraging/		Life History/		Lower		Similar		
Category	in PSA*	Species	Size	Residency	Range	Diet	Miscellaneous	Lower	to Similar	Similar	to Higher	Higher	References
Insectivorous Birds (cont.)	Tree swallow/ Intermediate to High (cont.)	Northern rough- winged swallow	16 g	5 months	Nest within 1 km of water	Entirely insects	Nest in exposed and eroding riverbanks and in gravel pits				X		DeGraaf and Yamasaki 2001; Ehrlich et al. 1992
		Barn swallow	19 g	5 months	Seldom feed more than 0.8 km from nest site	Insects, occasionally berries and seeds	Nest under bridges in PSA and feed over river and fields			Х			DeGraaf and Yamasaki 2001; Ehrlich et al. 1992
		Cliff swallow	21 g	5 months	Foraging range typically within 1 km	Almost entirely insects, but occasionally gorge on berries	Nest under bridges in PSA and feed over river and fields	х					DeGraaf and Yamasaki 2001; Ehrlich et al. 1992
		Chimney swift	23 g	5 months	Foraging range up to several kilometers	Flying insects					X		DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Sibley 2001
		Common nighthawk	62 g	5 months	Territory 2-23 ha	Flying insects, especially flying ants, mosquitoes, moths, grasshoppers	Nests on rooftops in town, feeds over fields and water			Х			DeGraaf and Yamasaki 2001; Ehrlich et al. 1992
		Eastern kingbird	40 g	6 months	Territory 5.7-14.2 ha	Flying insects, some fruit	Commonly nests in trees overhanging the river in the PSA, capture insects over the river. Also occurs in agricultural areas, over fields	х					DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951

	Representative							Level of	f Risk Com	pared to Re	presentative	e Species	
Category	Species and Risk Category in PSA*	Other Species	Size	Residency	Foraging/ Home Range	Diet	Life History/ Miscellaneous	Lower	Lower to Similar	Similar	Similar to Higher	Higher	References
Insectivorous Birds (cont.)	Tree swallow/ Intermediate to High (cont.)	Eastern phoebe	20 g	7 months	0.3 ha in an Illinois flood- plain; 1.3- 2.9 ha in settled area	92-97% flying insects from spring through fall, mostly berries and seeds in winter				Х			DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951
	American robin/High		77 g	Year- round	Territory: 0.4 ha; foraging range for nestlings and fledg- lings; 0.15 and 0.81 ha	50-90% animal matter in spring and summer, switches to plants (berries) in fall and winter. Prey includes earthworms, butterflies, moths, beetles, and ants				n/a			DeGraaf and Yamasaki 2001; Sallabanks and James 1999; Ehrlich et al. 1992; Weatherhead and McRae 1990; Martin et al. 1951
		Eastern bluebird	31 g	8 months	Territory 2.2-3.5 ha	60% or more animal matter year-round, up to 80-95% in spring and summer. Prey includes beetles, grasshoppers, crickets, and caterpillars	Diet similar to robin except earthworms rarely consumed		х				DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951
		Eastern towhee	40 g	6 months	Territory 0.26-2.4 ha	50:50 plant and animal in summer and fall. Mostly terrestrial insects, seeds, and berries	Tends to consume considerably less animal matter than robin	Х					DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951

	Representative							Level of	Risk Com	pared to Re	presentative	e Species	
	Species and				Foraging/				Lower		Similar		
Catalan	Risk Category	Other	C!	Destidences	Home	D:-4	Life History/	-	to	~	to		Defense
Category	in PSA*	Species	Size	Residency	Kange	Diet	Miscellaneous	Lower	Similar	Similar	Higher	Higher	References
	American robin/High (cont.)	Gray catbird	37 g	7 months	Territory 0.06-0.32 ha	40-80% animal matter in spring and summer, fall diet nearly 80% plants (berries). Prey includes largely terrestrial insects (ants, beetles), caterpillars, and grasshoppers	Diet similar to robin except earthworms rarely consumed				X		DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951
		Hermit thrush	31 g	7 months	Territory 0.06-3.34 ha	93 and 85% animal matter in spring and summer, respectively. Dominant prey includes beetles, ants, caterpillars, flies, and insects	Diet similar to robin with the exception of earthworms			X			DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951
		Northern mockingbird	49 g	Year- round	Territory 0.25-0.5 ha in summer	70-85% animal matter in spring and summer. Dominant prey includes beetles, ants, bees, wasps, and grasshoppers	Diet similar to robin except earthworms rarely consumed			X			DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951

	Representative							Level of	Risk Com	pared to Re	presentative	e Species	
Category	Species and Risk Category in PSA*	Other Species	Size	Residency	Foraging/ Home Range	Diet	Life History/ Miscellaneous	Lower	Lower to Similar	Similar	Similar to Higher	Higher	References
	American robin/High (cont.)	Veery	31 g	5 months	Territory 0.1-3+ ha	60-95% animal matter in spring and summer. Dominant prey includes beetles, ants, caterpillars, spiders, and grasshoppers	Diet similar to robin except earthworms rarely consumed			х			DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951
		Wood thrush	47 g	6 months	Territory 0.08-2.8 ha	60-95% animal matter in spring and summer. Dominant prey includes beetles, ants, caterpillars, spiders, and grasshoppers	Diet similar to robin except earthworms rarely consumed			х			DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951
Omnivorous and Carnivorous Mammals	Northern short- tailed shrew/High		20.5 g	Year- round	0.024-0.2 ha	Common prey includes insects and earthworms. Also forage on snails, slugs, crustaceans, small mammals, fungi, and, rarely, vegetation	Occurs in damp woodlands and fields			n/a			DeGraaf and Yamasaki 2001; Whitaker and Hamilton 1998; Kurta 1995; EPA 1993; Whitaker and Ferraro 1963; Hamilton 1941; Linzey and Linzey 1973; Eadie 1944; Eadie 1948
		Smoky shrew	6.1-11 g	Year- round	Not found	Insectivorous; also salamanders, young mice, vegetable matter	Habitat preferences and diet similar to short-tailed shrew					X	DeGraaf and Yamasaki 2001; Whitaker and Hamilton 1998; Kurta 1995

	Representative							Level of	f Risk Com	pared to Re	presentative	e Species	
	Species and Disk Catagory	Other			Foraging/		I :fo History/		Lower		Similar		
Category	in PSA*	Species	Size	Residency	Range	Diet	Miscellaneous	Lower	to Similar	Similar	to Higher	Higher	References
Omnivorous and Carnivorous Mammals (cont.)	Short-tailed shrew/High (cont.)	Masked shrew	4.0-6.5 g	Year- round	0.16-0.28 ha	Predominantly insectivorous; also mollusks, annelids, dead bodies of larger animals, salamanders, young mice, <i>Endogone</i> , vegetable matter	Similar diet but more commonly in dryer uplands, meadows, old fields, and fencerows			X	- mg.c.	- mgirri	DeGraaf and Yamasaki 2001; Whitaker and Hamilton 1998; Kurta 1995
	Red fox/ Intermediate		3.4-6.4 kg	Year- round	60-600 ha	Up to 30% plants in summer and fall, the remainder being small mammals, birds, and insects. Mammals average 76% of diet for all seasons	Prefers open agricultural land and forest edges			n/a			DeGraaf and Yamasaki 2001; Whitaker and Hamilton 1998; Kurta 1995; EPA 1993; Martin et al. 1951; Powell and Case 1982; Knable 1974; Korschgen 1959; Hockman and Chapman 1983; Dibello et al. 1990
		Coyote	9.1-22.7 kg	Year- round	1000- 4000 ha	78% mammals, 21% fruit, 10% insects, and 3% birds by frequency in 1,500 scats from Adirondacks	Broad habitat requirements, open fields, agricultural land, forested areas	Х					DeGraaf and Yamasaki 2001; Whitaker and Hamilton 1998; Kurta 1995; Martin et al. 1951

	Representative							Level of	f Risk Com	pared to Re	presentative	e Species	
	Species and	Other			Foraging/		I :fo History/		Lower		Similar		
Category	in PSA*	Species	Size	Residency	Range	Diet	Miscellaneous	Lower	to Similar	Similar	to Higher	Higher	References
Omnivorous and Carnivorous Mammals (cont.)	Red fox/ Intermediate (cont.)	Gray fox	3.2-5.9 kg	Year- round	85-3200 ha	85-95% animal matter throughout the year (e.g., rabbit, squirrel)	Most common in forested areas			Х			DeGraaf and Yamasaki 2001; Whitaker and Hamilton 1998; Kurta 1995; Martin et al. 1951
		Fisher	3.6-5.5 kg	Year- round	1500- 3500 ha	Nearly 100% animal matter, including small mammals, squirrels, rabbits, porcupine, birds, reptiles, and amphibians	Prefer forested areas with closed canopies				х		DeGraaf and Yamasaki 2001; Whitaker and Hamilton 1998; Kurta 1995; Martin et al. 1951
		Long-tailed weasel	85-270 g	Year- round	31.9-160 ha	78% small mammals (mice, voles, shrews), 17% rabbits; also birds (up to 10%), squirrels, snakes, invertebrates	Terrestrial				X		DeGraaf and Yamasaki 2001; Whitaker and Hamilton 1998; Kurta 1995
		Short-tailed weasel	50-150 g	Year- round	Males: 17.0-25.0 ha Females: 10.1-14.9 ha	75% small mammals (mice, voles, shrews); also squirrels, rabbits, birds, amphibians, snakes, invertebrates	Terrestrial				x		DeGraaf and Yamasaki 2001; Whitaker and Hamilton 1998; Kurta 1995

	Representative							Level o	f Risk Com	pared to Re	presentative	e Species	
	Species and Disk Catagory	Other			Foraging/		I :fo History/		Lower		Similar		
Category	in PSA*	Species	Size	Residency	Range	Diet	Miscellaneous	Lower	to Similar	Similar	to Higher	Higher	References
Threatened and Endangered Species**	American bittern/High		370-500 g	5 months	Varies with geo- graphic area, preferred habitat avail- ability and prey species >3 ha reported for Michigan and 24.7 ha core use reported for Minnesota	Amphibians, small snakes, crayfish, insects, small fish	Consume a wide variety of prey items allowing them to hunt in varying habitats		Sima	n/a	Ingitti	Ingiti	Gibbs et al. 1992; Brown and Dinsmore 1986; Azure 1998 in Deschant et al. 2001; Gibbs et al. 1992 in DeGraaf and Yamasaki 2001
		Great blue heron	2,400 g	Year- round	Will feed kilometers from nest	Approximately 70% fish, also insects, crayfish, small mammals, and amphibians				Х			DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951
		Green heron	210 g	5 months	Defends territory a few feet from nest	Crayfish approximately 50% of diet, 25% aquatic insects, and 20% small fish	Separate feeding territories may be vigorously defended					Х	DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951

	Representative							Level of Risk Compared to Representative Species					
	Species and Risk Category	Other			Foraging/ Home		Life History/		Lower to		Similar to		
Category	in PSA*	Species	Size	Residency	Range	Diet	Miscellaneous	Lower	Similar	Similar	Higher	Higher	References
Threatened and Endangered Species (cont.)	American bittern/High (cont.)	Pied-billed grebe	450 g	7 months	Defends area 50 m around nest	Primarily animal matter, including crayfish, small fish, mollusks, aquatic insects						Х	DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951
		Sora	75 g	5 months	Distances between nest ranges from 1.2 to 25 m	60% animal matter spring and fall. Prey includes beetles, snails, spiders, and crustaceans		X					DeGraaf and Yamasaki 2001; Odum 1977; Tanner and Hendrickson 1956 in DeGraaf and Yamasaki 2001; Berger 1951 in Degraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951
		Virginia rail	85 g	5 months	Territorial during pair formation and nest establish- ment	90-95% animal matter spring and fall. Prey includes beetles, snails, spiders, true bugs, and diptera larvae. Also crustaceans, dragonfly and damselfly larvae, ants, grasshoppers, crickets, and small fish						X	DeGraaf and Yamasaki 2001; Ehrlich et al. 1992; Martin et al. 1951.

	Representative							Level of Risk Compared to Representative Species					
	Species and Bisk Category	Other			Foraging/		Life History/		Lower		Similar		
Category	in PSA*	Species	Size	Residency	Range	Diet	Miscellaneous	Lower	to Similar	Similar	to Higher	Higher	References
Threatened and Endangered Species (cont.)	Small-footed myotis/High		6 g, range 5- 7 g	Year- round, but hibernate	Travel < 40 km between summer and winter grounds	Primarily midges, caddisflies, moths, butterflies, and beetles	Observed longevity of 12 years in the wild			n/a			DeGraaf and Yamasaki 2001; Griffith and Gates 1985; Anthony and Kunz 1977; Belwood and Fenton 1976; Kurta 1995; van Zyll de Jong 1985 in DeGraaf and Yamasaki 2001
		Big brown bat	15-24 g	Year- round, but hibernate	Forages 1 to 2 km from day roosts	Specialize in capturing beetles, but also take true bugs, wasps, bees, flies, mosquitoes, midges, gnats, moths, and butterflies	Travels short distances, usually no more than 48 to 80 km from maternity colony to hibernaculum site			Х			DeGraaf and Yamasaki 2001; Whitaker 1995; Griffith and Gates 1985; Barbour and Davis 1969 in DeGraaf and Yamasaki 2001; Mills et al. 1975 in DeGraaf and Yamasaki 2001; Kurta 1995; Kurta and Baker 1990 in Degraaf and Yamasaki 2001; Kurta 1995
		Indiana myotis	6-11 g	Year- round, but hibernate.	Female range over 52- 95 ha	Includes terrestrial insects as well as emergent aquatic insects	Forages in foliage of tree crowns along river and lake shores			Х			DeGraaf and Yamasaki 2001; Kurta 1995

Comparison of Risks of tPCBs and TEQ to Representative and Other Species in the Housatonic River PSA (Continued)

	Representative							Level of Risk Compared to Representative Species					
Category	Species and Risk Category in PSA*	Other Species	Size	Residency	Foraging/ Home Range	Diet	Life History/ Miscellaneous	Lower	Lower to Similar	Similar	Similar to Higher	Higher	References
Threatened and Endangered Species (cont.)	Small-footed myotis/High (cont.)	Little brown bat	6-12 g	Year- round, but hibernate	Unknown	Midges, mayflies, caddisflies, and mosquitoes. Also beetles, moths, stoneflies, true bugs, and termites	Consume 1.8-3.7 g of food per night			х			DeGraaf and Yamasaki 2001; Griffith and Gates 1985; Anthony and Kunz 1977; Belwood and Fenton 1976; Kurta 1995

For representative species with multiple assessments in the PSA, the highest risk category is listed here.

** Several of the species included in this section (i.e., sora, great blue heron, green heron, Virginia rail, northern myotis, little brown bat) are not threatened and endangered species either federally or in Massachusetts and Connecticut (Appendix A). They are included in the discussion of T&E species because they are taxonomically and ecologically similar to either American bittern or to small-footed myotis.

n/a = not applicable.

1 12.4.1.1 Benthic Invertebrates

2 The benthic invertebrate ERA included the entire benthic community; benthic community 3 composition analysis was a measurement endpoint considered in the weight-of-evidence 4 assessment. Individual species were also used in toxicity tests as surrogates for the Housatonic 5 River freshwater benthic community (i.e., Chironomus tentans, Hyalella azteca, Lumbriculus 6 variegatus, Daphnia magna, Ceriodaphnia dubia). Both the status of sensitive taxa and 7 community composition are considered indicators of the overall health and productivity of the 8 benthic community. Thus, there is no need to extrapolate the findings of the benthic invertebrate 9 assessment described previously to other benthic invertebrate species in the PSA.

10 **12.4.1.2** Amphibians

11 Certain amphibian species that were not studied may be more susceptible to the effects of tPCBs 12 because of their life history characteristics. For example, blue-spotted (Ambystoma laterale) and 13 spotted salamanders (Ambystoma maculatum) have a lifestage as aquatic carnivorous, bottom-Thus, they could potentially bioaccumulate PCBs more quickly than 14 dwelling larvae. 15 herbivorous amphibians. The larvae of these two species forage on the bottom of vernal pools, 16 and have greater opportunities to be in contact with contaminated sediment than do pelagic frog 17 tadpoles. The salamander larvae feed on an assortment of planktonic animals, and then shift to 18 larger aquatic worms, insect larvae, small crustaceans, and tadpoles as they grow larger (Hunter 19 et al. 1999).

20 Blue-spotted and spotted salamanders also have longer larval periods, lasting between 70 to 100 21 days (Whitford and Vinegar 1966), than do wood frogs, which have a larval period averaging 67 22 days (Hunter et al. 1999). Salamanders appeared in lower numbers in vernal pools with high 23 sediment tPCB concentrations (Woodlot Alternatives, Inc. 2003). Several salamander species 24 occur in contaminated habitat in the PSA, including the spotted salamander, the Jefferson 25 salamander (Ambystoma jeffersonianum, formerly considered a variety of blue-spotted salamander), and the four-toed salamander (Hemidactylium scutatum), the latter two of which are 26 27 Species of Special Concern (www.state.ma.us/dfwele/dfw/nhesp/nhrare.htm).

1 12.4.1.3 Fish

Five fish species—largemouth bass (*Micropterus salmoides*), yellow perch (*Perca flavescens*),
brown bullhead (*Ameiurus nebulosis*), white sucker (*Catostomus commersoni*), and pumpkinseed
(*Lepomis gibbosus*)—were selected as the representative species for the ERA. The fish species
were selected to include representatives of the principal trophic levels and exposure routes for
fish in the PSA.

7 There is evidence in the literature that salmonid species may have a higher sensitivity to the 8 effects of PCBs and other dioxin-like COPCs (Walker et al. 1994; Zabel et al. 1995). The use of 9 rainbow trout in the site-specific toxicity testing program (Phase II), combined with effects data 10 from the literature (Appendix F), provides a high degree of confidence that the ERA included an 11 evaluation of fish species with equal or greater sensitivities than the representative species listed 12 above. However, the procedure used to establish MATCs for fish in the PSA placed a low 13 weight on studies conducted with fish species known to be highly sensitive (e.g., lake trout) to 14 avoid an overly conservative assessment. Risks to coldwater fisheries (e.g., trout) downstream 15 of the PSA were explicitly evaluated using benchmarks developed for salmonids; the uncertainty 16 in these downstream risk estimates is high due to the number of extrapolations required. The 17 risk of COCs to the occasional salmonid occurring within the PSA is considered to be moderate. 18 The PSA, however, is considered to be a warmwater fishery, and thus salmonid abundance is 19 expected to be low in this portion of the river, even in the absence of chemical stressors.

20 12.4.1.4 Insectivorous Birds

The weight-of-evidence assessment indicated that exposure of insectivorous birds, such as tree swallows and American robins, to tPCBs and TEQ is high but unlikely to lead to adverse reproductive effects. Confidence in this conclusion, however, is not high because the two available lines of evidence for both species did not produce concordant results.

The tree swallow was chosen as the one of the representative species for insectivorous birds. This species is common in the PSA and other areas in the watershed of the Housatonic River (Appendix A). Other insectivorous bird species that are comparable to tree swallows and are common to the PSA include the bank swallow (*Riparia riparia*), northern rough-winged swallow (Stelgidopteryx serripennis), barn swallow (Hirundo rustica), cliff swallow (Hirundo
 pyrrhonota), chimney swift (Chaetura pelagica), common nighthawk (Chordeiles minor),
 eastern kingbird (Tyrannus tyrannus), and eastern phoebe (Sayornis phoebe).

4 Compared to the tree swallow, the cliff swallow is expected to have a similar to lower level of 5 risk from exposure to tPCBs and TEQ. The cliff swallow has a similar diet and is of similar size, 6 but has a much larger foraging range that may dilute exposure compared to tree swallows. The 7 eastern kingbird is also expected to have a similar to lower level of risk because it is twice the 8 size of the tree swallow and, therefore, has a lower metabolic and food intake rate. The lower 9 food intake rate will likely lead to reduced exposure. Eastern kingbirds and tree swallows have a 10 similar diet and foraging range. The barn swallow and eastern phoebe have a similar level of 11 risk compared to tree swallows because they have similar body sizes and diet. The bank 12 swallow, chimney swift, and northern rough-winged swallow have a similar to higher level of 13 risk from exposure to tPCBs and TEQ because they have a higher proportion of insects in the 14 diet and/or are smaller than tree swallows.

Insectivorous birds that are more comparable to American robins and are common to the area include the eastern bluebird (*Sialia sialis*), eastern towhee (*Pipilo erythrophthalmus*), gray catbird (*Dumetella carolinensis*), hermit thrush (*Catharus guttatus*), northern mockingbird (*Mimus polyglottos*), veery (*Catharus fuscescens*), and wood thrush (*Hylocichla mustelina*) (see Appendix A).

20 Compared to American robins, eastern bluebirds and eastern towhees are expected to experience 21 lower to similar levels of risk from exposure to tPCBs and TEQ. Eastern bluebirds consume 22 similar prey compared to American robins, but have a larger foraging range that would dilute 23 exposure to tPCBs and TEQ in the PSA. Eastern towhees consume less animal matter than 24 American robins. Because animal matter contains higher concentrations of COCs than 25 vegetation, eastern towhee exposure to tPCBs and TEQ will likely be lower.

The level of risk for the hermit thrush, northern mockingbird, veery, and wood thrush is expected to be similar to American robins. With the exception of earthworms in the robin diet, the dietary preferences of these birds are similar to the American robin. The absence of earthworms, a major dietary source of contaminants, will decrease their exposure to tPCBs and TEQ. However, their smaller body sizes result in higher food intake rates and thus greater exposure to tPCBs and
 TEQ through diet compared to American robins.

Gray catbirds are expected to experience similar to higher levels of risk compared to American robins. With the exception of earthworms in the robin diet, gray catbirds consume similar prey and have a foraging range comparable to American robins. Their smaller body size results in a higher food intake rate and greater exposure to tPCBs and TEQ through diet compared to American robins.

8 **12.4.1.5** *Piscivorous Birds*

9 The weight-of-evidence assessment indicates that risks of tPCBs and TEQ to belted kingfisher 10 are low; however, risks of these COCs to osprey are high and could lead to adverse reproductive 11 effects.

The belted kingfisher and osprey were chosen to represent piscivorous birds inhabiting the Housatonic River area. Belted kingfisher and osprey are common piscivorous birds in the PSA. Great blue herons are also found in the PSA, and are discussed in Appendix K with other piscivorous birds (e.g., American bittern).

16 **12.4.1.6** *Piscivorous Mammals*

Mink and river otter, the representative species for piscivorous mammals, are the only
piscivorous mammals commonly found in the watershed of the Housatonic River (EPA 2001)
(Appendix A).

20 12.4.1.7 Omnivorous and Carnivorous Mammals

The weight-of-evidence assessment indicates that omnivorous and carnivorous mammals, such as red fox and short-tailed shrew, are at risk in the PSA as a result of exposure to tPCBs and TEQ. Risks to short-tailed shrews exposed to tPCBs at Sites 13 and 14 are high.

The northern short-tailed shrew and red fox were chosen to represent omnivorous and carnivorous mammals inhabiting the Housatonic River area. Other omnivorous and carnivorous species common to the area include the smoky shrew (*Sorex fumeus*), masked shrew (*Sorex cinereus*), coyote (*Canis latrans*), gray fox (*Urocyon cinereoargenteus*), fisher (*Martes pennanti*), long-tailed weasel (*Mustela frenata*), and short-tailed weasel (*Mustela erminea*) (see Appendix A).

5 Masked shrews are expected to experience a level of risk similar to northern short-tailed shrews. 6 Both animals have similar foraging behaviors and ranges. Masked shrews are smaller than 7 northern short-tailed shrews; therefore, they have a higher metabolism that increases exposure to 8 contaminants. Masked shrews prefer to inhabit dry upland areas that are less contaminated than 9 the damp woodlands and fields of the PSA where northern short-tailed shrews are found.

10 Compared to northern short-tailed shrews, smoky shrews are expected to experience higher 11 levels of risk from exposure to tPCBs and TEQ. These shrews have similar foraging preferences 12 and life history characteristics. Smoky shrews are much smaller than northern short-tailed 13 shrews and thus have a higher metabolic rate that increases exposure to tPCBs and TEQ.

14 Coyotes have a larger body size and foraging range that decreases their exposure to tPCBs and 15 TEQ. Considering these characteristics, coyotes are expected to experience lower risks from 16 exposure to tPCBs and TEQ.

Gray and red foxes are expected to experience similar risks from exposure to tPCBs and TEQ.
Gray fox have a larger foraging range than red fox and that may decrease their exposure to
tPCBs and TEQ. Gray fox, however, have a greater reliance on animal matter and thus greater
exposure to tPCBs and TEQ.

21 Fisher, long-tailed weasels, and short-tailed weasels are expected to experience similar to higher 22 levels of risk from exposure to tPCBs and TEQ compared to the red fox. Fisher and red fox have 23 similar body weights. Animal matter constitutes nearly 100% of the fisher diet compared to an 24 average of 76% of the red fox diet. This greater consumption of animal matter increases the 25 fisher's exposure to tPCBs and TEQ. The diets of long- and short-tailed weasels are similar to 26 red fox, but weasels have smaller foraging ranges, which increases their exposure to tPCBs and 27 TEQ in the PSA. Their smaller body weight results in a higher metabolism that further increases 28 exposure to tPCBs and TEQ.

1 12.4.1.8 Threatened and Endangered Species

The weight-of-evidence assessment indicates that threatened and endangered (T&E) species such as bald eagles, American bitterns, and small-footed myotis are at risk in the PSA as a result of exposure to tPCBs and, to a lesser extent, TEQ. The risk for bald eagles exposed to tPCBs is high. The risks for American bittern and small-footed myotis exposed to tPCBs and TEQ are intermediate.

7 The bald eagle, American bittern, and small-footed myotis were chosen to represent T&E species 8 that are likely to be highly exposed to COCs in the Housatonic River PSA. Other T&E species 9 that occur in the area include one mussel (triangle floater); three dragonflies (riffle snaketail, 10 zebra clubtail, and arrow clubtail); a turtle (wood turtle); three salamanders (Jefferson 11 salamander, four-toed salamander, and northern spring salamander); three hawks (northern 12 harrier, sharp-shinned hawk, and Cooper's hawk); two warblers (northern parula and blackpoll 13 warbler); a wading bird (common moorhen); and a shrew (northern water shrew). Some of these species were qualitatively assessed in other appendices and compared to other, more appropriate, 14 15 assessment endpoints (e.g., amphibians for salamanders).

The level of risk for soras¹ is expected to be lower than for American bitterns. The sora and American bittern have similar life history characteristics and habitat preferences. However, the sora consumes more vegetable matter and less animal matter than the American bittern. This decreases the sora's exposure to tPCBs and TEQ.

Great blue herons and king rails are expected to experience a similar level of risk as American bitterns. Great blue herons consume more fish than American bitterns. Fish contain higher concentrations of tPCBs than other prey. However, great blue herons have a larger body size than American bitterns resulting in a slower metabolism and lower accumulation of contaminants. In addition, the reproductive strategy for great blue herons suggests that few individuals from an entire rookery would be exposed in the PSA, lessening the risk to the local

MK01|O:\20123001.096\ERA_PB\ERA_PB_12.DOC

¹ Several of the species included in this section (i.e., sora, great blue heron, green heron, Virginia rail, northern myotis, little brown bat) are not threatened and endangered species either federally or in Massachusetts and Connecticut (Appendix A). They are included in the discussion of T&E species because they are taxonomically and ecologically similar to either American bittern or to small-footed myotis.

population. King rails consume prey that would have lower concentrations of COCs than
 American bitterns. Therefore, although they have a smaller body size and higher metabolism,
 their exposure to tPCBs and TEQ is expected to be lower.

The least bittern, green heron, Virginia rail, and pied-billed grebe are expected to experience higher levels of risk compared to the American bittern. The foraging and life history characteristics of these birds are similar to the American bittern. However, these birds are much smaller than the American bittern. Their smaller body sizes result in a higher metabolism and greater exposure to tPCBs and TEQ.

9 The Indiana bat, northern myotis, and little brown bat are expected to have similar levels of risk 10 as the small-footed myotis. These species belong to the same genus (*Myotis*) and have similar 11 foraging behaviors and life histories.

12 All of the above information is summarized in greater detail in Table 12.4-1.

13 **12.4.2** Ecological Implications and Other Concerns

As with most ecological risk assessments of contaminated sites, the ecological risk assessment for the Housatonic River is an assessment of the direct effects of COCs on a species-by-species basis. The following discussion places the ecological risk assessment in a broader ecological context by examining populations, ecological interactions and functions, and other issues of concern to decision makers and the public. The section begins with a brief discussion of the regulatory objectives of EPA and other agencies as they pertain to ecological protection goals for contaminated sites.

21 12.4.2.1 Protection Goals

Recently, there has been considerable interest in regulatory agencies and elsewhere for assessing risks at higher levels of organization, such as the population, community, or ecosystem (e.g., Environment Canada 1997; EPA 1997; Landis et al. 1998; EPA 1999b). Assessment of risks at higher levels of organization is useful because it furthers the understanding of the seriousness of risks posed by COCs, an important consideration in developing appropriate risk management responses. Assessment of risk at higher levels of organization, however, is not an easy task 1 (Moore and Bartell 2000). The desire to understand risk at higher levels of organization should
2 not be misinterpreted to mean that risks must be demonstrated at higher levels of organization
3 (e.g., population or higher) to be of concern to EPA and other agencies at the Housatonic River,
4 or other assessments of contaminated sites. As stated in EPA 1999b:

5 Levels that are expected to protect local populations and communities can be 6 estimated by extrapolating from effects on individuals and groups of individuals 7 using a line-of-evidence approach. The performance of multi-year field studies at 8 Superfund sites to try to quantify or predict long-term changes in local 9 populations is not necessary for appropriate risk management decisions to be 10 made. Data from discrete field and laboratory studies, if properly planned and appropriately interpreted, can be used to estimate local population or community 11 level effects. 12

- 13 In addition, the Massachusetts Contingency Plan at 310 CMR 40.0995 (4) states:
- (b) The Stage II Environmental Risk Characterization shall identify
 environmental resources associated with the disposal site, such as wetlands,
 aquatic and terrestrial habitat, fisheries, or rare and endangered species, and shall
 evaluate whether the release of oil and/or hazardous material has adversely
 impacted, or may adversely impact the ecological functions which support those
 resources.
- The evaluation shall focus on ecological functions at the spatial scale of the disposal site.
- 22
 2. The relevance of potential impacts shall be judged at the spatial scale of the disposal site (e.g., effects on subpopulations that use the site as habitat) rather
 24
 25
 26
 27
 28
 29
 29
 20
 20
 21
 22
 23
 24
 25
 25
 26
 27
 28
 29
 29
 20
 20
 21
 22
 20
 21
 22
 21
 22
 22
 23
 24
 25
 26
 27
 28
 29
 29
 20
 21
 21
 22
 21
 22
 22
 23
 24
 25
 24
 25
 25
 26
 27
 28
 29
 29
 20
 20
 21
 21
 22
 21
 22
 22
 23
 24
 24
 25
 24
 25
 26
 27
 28
 29
 29
 20
 21
 21
 21
 22
 22
 23
 24
 24
 25
 24
 25
 25
 26
 27
 27
 28
 29
 29
 20
 21
 21
 21
 21
 22
 23
 24
 24
 24
 25
 24
 25
 26
 27
 28
 29
 29
 29
 20
 21
 <l

26 The concentrations of contaminants at this site, compared to most sites assessed under hazardous 27 waste regulatory standards, are very high. By assessing aquatic life and wildlife that are exposed 28 to COCs, the risk assessment evaluated whether the contaminated habitats (i.e., the Primary 29 Study Area) are functioning as would normal habitats in the absence of contaminants. Vital 30 functions include providing adequate food and shelter and sustaining normal reproductive 31 success. The central question, for purposes of this assessment, is whether the exposed local 32 populations are thriving in the contaminated habitat, not whether the larger regional population is 33 surviving in spite of it.

1 12.4.2.2 Ecological Implications

Populations of organisms are controlled by the balance between positive processes (e.g., growth, reproduction, immigration) and negative processes (e.g., starvation, death from predation, toxicant effects, emigration) (Taub 1989). Growth and reproduction are often controlled by food supply and availability of adequate habitat. Mortality is often caused by predators and other stressors in the environment. The dynamics of populations exposed to COCs have important implications toward other interacting organisms and functions (Landis et al. 1998). Examples include:

- 9 **Removal of Predators Compensates for Direct Effects of Contaminants**—A toxic 10 chemical may increase food supply by reducing abundance of competitors or by eliminating predators. In the Housatonic River ERA, the modeling of exposure and 11 12 effects line of evidence indicated that some species are experiencing risk from 13 exposure to tPCBs and TEQ (e.g., short-tailed shrews, largemouth bass), yet are fairly abundant in the PSA. One possible explanation for this lack of concordance between 14 measurement endpoints is that elimination of predators (e.g., mink, river otter) may 15 16 be compensating for the direct effects due to tPCBs and TEQ.
- For fish, the fishing ban imposed on the Housatonic River limits human predation on the fish stocks, therefore likely compensating for the effects of tPCBs on recruitment to older age classes. Elimination of predators, however, does not necessarily benefit each prey population. For example, some prey populations may decline as a result of increased competition from other prey populations (Bartell et al. 1992).
- 22 Immigration Compensates for Direct Effects of Contaminants—The elimination 23 of individuals from a habitat creates openings that may be exploited by individuals 24 emigrating from surrounding habitats. Many species can migrate to the PSA or 25 within the PSA from areas of lower contamination to areas of higher contamination 26 and compensate for losses of organisms due to toxic effects; therefore, the presence of 27 a normal number of animals in a contaminated area does not necessarily demonstrate 28 that the population is unaffected or that the habitat is providing normal support 29 functions. If contaminant concentrations are such that organisms cannot reproduce 30 normally or thrive in the affected area without immigration from other areas, then 31 those effects are viewed by EPA as unacceptable. Immigration is likely the process 32 that explains the infrequent sightings of mink tracks in winter on the edges of the 33 PSA. In winter, juvenile and young adult mink often emigrate to other habitats, 34 particularly those that are not already occupied by mink, have an abundant food supply, and offer ideal habitat (i.e., the PSA). The risk assessment for the PSA 35 36 indicates, however, that these mink are unlikely to survive and reproduce in the PSA.
- Populations Exposed to COCs May Be More Vulnerable to Other Stressors in
 the Future—The studies conducted to support the ERA were done during a period of

regulatory restrictions, such as the fish, turtle, and frog advisories that have been in 1 2 place in the Housatonic River in Massachusetts since 1982. Several authors (e.g., 3 Evans et al. 1990; Edwards et al. 1990) have surmised that lake trout populations in 4 the Great Lakes remained stable in the first half of the century despite fishing 5 pressure and the influence of other anthropogenic stressors. Lake trout populations declined to very low levels by the 1970s, however, because of additional stressors 6 7 such as contaminants and introduction of sea lampreys. Thus, a fish population can 8 often tolerate some anthropogenic stresses, but if the combination of stresses becomes 9 too great, the population crashes. Density-independent stressors, such as toxic 10 chemicals that reduce fitness at all population densities, lower the capacity of populations to respond to otherwise favorable conditions or to tolerate other stressors 11 12 (Evans et al. 1990). Control of sea lampreys, reductions in contaminant concentrations, and reduced fishing pressure have not restored lake trout populations 13 14 to their historical levels (Edwards et al. 1990).

15 Many other indirect effects may occur as a result of the presence of tPCBs and TEQ in the PSA.

16 For example, Wu et al. (1993) and Spromberg et al. (1998) have shown that subpopulations in

17 patches removed from contamination may be affected by contaminated patches even if there is

18 no transfer of contaminant (the so-called "action at a distance"). Alternatively, COCs transferred

19 outside the PSA (e.g., by downstream transport, migration of birds) can augment exposures or

20 cause effects to organisms outside the PSA (e.g., hawks preying on migrating birds).

21 12.4.2.2.1 Genetic Diversity

Chemical adaptation has been cited as a compensatory mechanism that can enable populations to survive at a site (Shugart 1996). This mechanism leads to selection of genotypes that are resistant to a COC as a result of the elimination of sensitive individuals. The result is a population with an altered genetic makeup, generally with a less diverse genetic pool. It is likely that such reductions in the genetic pool cause alterations that may reduce a population's resilience, making it more susceptible to other stresses in the future.

28 12.4.2.2.2 Immune System Effects

Non-coplanar PCBs can influence the activity of neutrophils (a type of white blood cell) through mechanisms unrelated to the Ah receptor. In studies with rat- and human-derived neutrophils, researchers have found that non-coplanar PCBs can activate biochemical pathways that lead to the production of reactive oxygen species (ROS)(Fischer et al. 1998). Although the production of ROS is a normal function of neutrophils (it is designed to destroy bacteria and viruses and to break down tissue damaged by burns, chemicals, and physical injuries), when inappropriately activated by PCBs, this neutrophil function can initiate harmful effects on healthy tissues because of the destructive nature of ROS. Because neutrophils are among the first white blood cells sent to sites of infection or inflammation, exposure to PCBs may weaken an animal's immune and inflammatory responses.

1 12.5 SOURCES OF UNCERTAINTY

2 The assessment of risks of COCs to aquatic and wildlife species in the Housatonic River contains 3 uncertainties. Each source of uncertainty can influence the estimates of risk; therefore, it is 4 important to describe and, when possible, specify the magnitude and direction of such 5 uncertainties. The sources of uncertainty associated with the assessment of risks of tPCBs and 6 TEQ to each assessment endpoint were summarized in Sections 3 through 11 and Appendices D 7 through K. This material is not repeated here. In this section, the most significant sources of 8 uncertainty commonly encountered throughout the ERA are described. The sources of 9 uncertainty are grouped by phase of the ERA (i.e., problem formulation, exposure assessment, 10 effects assessment, risk assessment).

11 **12.5.1 Problem Formulation**

12 The problem formulation is intended to define the linkages between stressors, potential exposure, and predicted effects on ecological receptors. As such, the conceptual model provides the 13 14 scientific basis for selecting assessment and measurement endpoints to support the risk 15 assessment process. Potential uncertainties arise from lack of knowledge regarding ecosystem 16 functions, failure to adequately address spatial and temporal variability in the evaluations of 17 sources, fate and effects, omission of stressors, and overlooking secondary effects (EPA 1998). 18 The types of uncertainties associated with the conceptual model that links contaminant sources to 19 effects include those associated with the identification of COCs, environmental fate and transport 20 of COCs, exposure pathways, receptors at risk, and ecological effects. Of these, the 21 identification of exposure pathways probably represents the primary source of uncertainty in the 22 conceptual model. The detailed ecological characterization performed at this site has greatly 23 reduced the uncertainties associated with problem formulation, yet some remain and are 24 described below:

26 27 28

25

29 30 The Housatonic River and surrounding floodplain have received chemical inputs since the industrialization of the area. In addition to tPCBs and TEQ, other contaminants identified in water, soil, and sediment samples include metals (e.g., mercury, lead, chromium); pesticides (e.g., aldrin, DDT, toxaphene, parathion, 2,4-D); and semivolatile organic compounds (e.g., PAHs, chlorinated benzenes, anilines, phenols). The conservative screening level assessment indicated that only tPCBs and
TEQ present a potential risk to wildlife species; therefore, only these COCs were included in the probabilistic risk assessments for wildlife. Several other COCs were screened through for aquatic life, although none were as influential as tPCBs. Additive, synergistic, and antagonistic effects due to exposure to multiple contaminants were not considered.

- 6 In this assessment, it was assumed that dietary exposure represented the most 7 important pathway for the exposure of wildlife to COCs. Although unlikely to 8 provide a major contribution to the risk, other pathways could increase exposure and 9 perhaps increase risk slightly (Moore et al. 1999). Deterministic calculations were 10 conducted in which estimates of exposure to COCs via drinking water and inhalation were included in the exposure model, but were not included in the assessment 11 because inclusion of these routes did not substantially increase overall exposure of 12 13 wildlife to the COCs. This issue was less important for aquatic life because these 14 assessments were conducted for multiple media exposures. The aquatic life endpoints 15 considered tissue burdens that integrated exposures from all sources and/or evaluated 16 exposures from the abiotic media (sediment, overlying water, porewater) deemed 17 most relevant to exposure.
- 18 **12.5.2 Exposure Assessment**

1 2

3

4

5

19 The exposure assessment is intended to describe the actual or potential co-occurrence of stressors 20 with receptors. As such, the exposure assessment identifies the exposure pathways and the 21 intensity and extent of contact with stressors for each receptor or group of receptors at risk. The 22 exposure models for wildlife were energetics-based models requiring information on body 23 weight, free living metabolic rate, proportions of food items in the diet, and the concentrations of 24 COCs in these food items. Each of these variables has associated uncertainties, most of which 25 were propagated through the exposure models. Exposure of fish species to COCs was assessed using measured wet weight whole body tissue concentrations. Exposure of benthos to COCs was 26 27 assessed as either the COC concentrations in abiotic site media (i.e., sediment, water) or as the 28 tissue body burdens that represent integrated exposure from all sources.

29 The greatest uncertainty in the benthic invertebrate and amphibian exposure 30 assessments was the potential for small-scale variability in exposure concentrations to 31 complicate the development of concentration-response relationships, additionally confounded by analytical variability (Appendix C.11). For studies that had replicate 32 33 measurements of tPCBs at a given station over a short period (e.g., benthic 34 macroinvertebrate sampling), the spatial variability was quantified and considered 35 explicitly in the derivation of concentration-response relationships. Where spatial 36 replication was not available, characterization of variability required the incorporation 37 of additional data sets.

- Tissue chemistry data (tPCBs, TEQ) were relied upon in the characterization of 1 2 exposures to fish species and piscivorous wildlife. Total PCB concentrations in fish 3 exhibit seasonal fluctuations; these are sometimes related to lipid content changes that 4 occur during reproductive life history stages. To minimize confounding effects of 5 short-term variations in PCB concentrations due to spawning events, the vast majority of the PCB data for this project were collected in the late summer and early fall. 6 7 Potential uncertainties (over time and space) in fish tissue chemistry were ameliorated 8 through the collection of a large number of samples (multiple species, ages, and 9 spanning multiple years of collections). Therefore, the ERA was conducted with a 10 robust data set (including a confirmatory analysis with non-EPA data sets) that limited the probability of spurious outcomes in the exposure assessments. 11
- 12 The Monte Carlo sensitivity analyses suggested that the parameters of the free 13 metabolic rate (FMR) allometric equation were generally the most influential variables on predicted total daily intakes of COCs. However, no direct measurements 14 15 of free metabolic rate or food intake rate (other than for captive animals) were 16 available for most of the representative wildlife species. Therefore, free metabolic 17 rates were estimated using allometric equations. The use of allometric equations introduces some degree of uncertainty into the exposure estimates because they are 18 19 subject to model-fitting error and are based on species different from the 20 representative species used in this assessment. Given the lack of data on species specific to this assessment, it is difficult to judge the magnitude of the uncertainty 21 22 introduced by the use of the allometric models. The uncertainty due to model-fitting 23 error was propagated in the uncertainty analyses by using distributions as inputs for 24 the allometric slope and power terms.
- 25 Sample sizes, while composites, were limited for the analyses of COC concentrations in some prey items, including earthworms, litter invertebrates, and benthic 26 27 invertebrates. To address this uncertainty in the Monte Carlo analyses for wildlife, 28 the upper confidence limit (UCL) or data set maximum (see Section 6.4 and 29 Appendix C.5) was used as an estimate of COC concentrations in prey items. The potential magnitude of the uncertainty associated with small sample sizes for COC 30 31 concentrations is unknown, but this approach likely overestimated exposure. The 32 probability bounds analysis used an unbiased approach (e.g., distribution free range 33 from LCL to UCL) to deal with sample size uncertainty.

34 12.5.3 Effects Assessment

The effects assessment is intended to describe the effects caused by stressors, link them to the assessment endpoints, and evaluate how effects change with fluctuations in the levels (i.e., concentrations or doses) of the various stressors. In this assessment, the effects of tPCBs and other COCs to representative species were assessed. There are several sources of uncertainty in the assessment of effects, including measurement errors, extrapolation errors, and data gaps.

- For benthos and amphibians, the effects benchmarks derived from the literature had a high degree of uncertainty due to the need to extrapolate across sites and species. This uncertainty was explicitly addressed in the weight-of-evidence evaluation.
- The site-specific fish toxicity studies indicated variations in the concentrationresponse relationships observed across species, reaches, and treatments, and introduced uncertainty into the development of effects thresholds.
- The methodology used to mimic maternal transfer used in the fish Phase II studies has been recently developed and has not been widely applied as an environmental monitoring technique; therefore, there are potential uncertainties inherent to extrapolating these laboratory-based results to Housatonic River fish. Similarly, the extrapolation of concentrations of tPCBs in egg to whole body concentrations has a degree of uncertainty associated with it. The magnitude and direction of the uncertainty is unknown.
- 14 The greatest potential source of uncertainty for the fish and wildlife effects 15 assessments was associated with the lack of toxicity studies involving the representative species. The direct assessment of effects to benthos, amphibians, 16 largemouth bass, and mink included studies on the effects of tPCBs to reproduction 17 and/or survival for these representative species. There were, however, no toxicity 18 19 studies available for many other representative species exposed to tPCBs or TEQ. As 20 a result, laboratory studies involving other species were often used to estimate effects 21 to representative species. To address uncertainty in the effects assessments, threshold 22 ranges were used in which effects to tolerant and sensitive species were considered. 23 It was assumed that the toxicity thresholds for the representative species lie within 24 these ranges.
- The effects metrics used to estimate risk from the literature were derived for Aroclor
 1254 and Aroclor 1260 mixtures, and more information was available for Aroclor
 1254 than for Aroclor 1260. Some uncertainty is inherent in extrapolating from
 studies using commercial Aroclor mixtures to the specific congener patterns observed
 in weathered mixtures in the PSA of the Housatonic River. The potential magnitude
 and direction of the uncertainty associated with this extrapolation are unknown.
- 31 TEO is an expression of the planar chlorinated hydrocarbons (PCHs). TEO are 32 derived from an equation that combines the relative potency of each congener into a 33 single concentration. The potencies of individual congeners are not known precisely 34 and were estimated based on a combination of data and professional judgment (Van den Berg et al. 1998). Although there is uncertainty in these calculations, this 35 approach has been accepted and applied in numerous jurisdictions worldwide. The 36 37 potential magnitude and direction of the uncertainty associated with this approach are 38 unknown. The potential toxic effects of congeners not evaluated by this method are 39 poorly understood and can not be quantified.

1

2

3

4

5

6

1 12.5.4 Risk Characterization

A weight-of-evidence procedure was used to assess risks of tPCBs and TEQ to the assessment
endpoints in the Housatonic River PSA. The analysis follows the methodology proposed by the
Massachusetts Weight-of-Evidence Workgroup (Menzie et al. 1996; see Section 2.9 for details).

5 In general, the weight-of-evidence approach is an inclusive process whereby multiple lines of 6 evidence are considered prior to determining risk. For the wildlife risk assessments, these lines 7 of evidence included the exposure and effects modeling results and, in some cases, field survey 8 results, and/or in situ or whole media toxicity test results. For the fish and benthic invertebrate 9 risk assessments, available lines of evidence included field survey results (e.g., community 10 evaluation for benthos), site-specific toxicity tests, and comparison of tissue and sediment 11 concentrations to benchmarks (both from the literature and site-specific benchmarks).

- 12 Uncertainty in the risk characterization arises from the absence of one or more of the 13 available lines of evidence. In the case of piscivorous birds, data on two of the three 14 major lines of evidence were available, i.e., comparison of modeled exposure and 15 effects and the field study. Threatened and endangered species had only the modeled 16 exposure and effects line of evidence to support the risk assessment. The 17 consequence of the lack of multiple concurring lines of evidence is less confidence in 18 the conclusion regarding risk. For example, the risk characterization of piscivorous 19 mammals had three major lines of evidence available, thus providing high confidence 20 in the risk conclusions. The risk characterization for fish and benthic invertebrates 21 also had three major lines of evidence available.
- Uncertainty for individual lines of evidence was sometimes sufficiently large to render the line of evidence of limited use in the ERA. For example, the community evaluation for fish was confounded by large habitat variations combined with small overall gradients and large small-scale variation in PCB concentrations. These factors made derivations of concentration-response relationships unfeasible (i.e., due to very low statistical power for determining contaminant-induced effects) and limited the studies to qualitative assessments.

1 12.6 ERA CONCLUSIONS

2

3

4

5

6

- Weight-of-evidence assessments indicated that aquatic life and wildlife in the Primary Study Area of the Housatonic River are experiencing unacceptable risks as a result of exposure to tPCBs and other COCs. Confidence in this conclusion is high for benthic invertebrates, amphibians, and piscivorous mammals because multiple lines of evidence gave concordant results.
- The risks of tPCBs and other COCs likely extend beyond the representative species considered in the quantitative risk assessments described herein. Qualitative risk assessments indicated that many other species in the PSA are potentially at risk.
 Further, there are likely indirect effects (e.g., changes in predator-prey relationships, changes in metapopulation dynamics) occurring inside and outside the PSA as a result of the direct impacts caused by tPCBs and other COCs.
- An assessment of risk downstream of the PSA indicated that tPCBs could potentially be causing adverse effects to benthic organisms in depositional areas as far as Reach 8, amphibians in floodplain areas as far as Reach 8, trout in Reaches 7 and 9, mink as far as Reach 10, otter as far as Reach 12, and bald eagle in Reach 8.

1 12.7 REFERENCES

Amrhein, J.F., C.A. Stow and C. Wible. 1999. Whole-fish versus filet polychlorinated-biphenyl
 concentrations: An analysis using classification and regression tree models. *Environmental Toxicology and Chemistry* 18:1817-1823.

- 5 Angermann, J.E., and F. Matsumura. 1999. Effects of PCBs and Cold Exposure on Immune 6 Function and Hepatic Glycogenolysis in *Rana pipiens*. Society of Environmental Toxicology and
- 7 Chemistry (SETAC). Presentation, SETAC 20th Annual Meeting, Nashville, Tennessee.
- Anthony, E.P.L. and T.H. Kunz. 1977. Feeding strategies of the little brown bat in southern New
 Hampshire. *Ecology* 58:775-786.
- 10 Aulerich, R.J., S.J. Bursian, W.J. Breslin, B.A. Olson, and R.K. Ringer. 1985. Toxicological
- 11 manifestations of 2,4,5-, 2',4',5'-, 2,3,6,2',3',6'-, and 3,4,5,3',4',5'-Hexachlorobiphenyl and
- 12 Aroclor 1254 in mink. Journal of Toxicology and Environmental Health 15:63-79.
- 13 Azure, D.A. 1998. Aspects of American Bittern ecology in northwest Minnesota. M.S. Thesis. 14 University of North Dakota, Grand Forks, North Dakota. 139 pp. Cited in: J.A. Deschant, M.L. Sondreal, D.H. Johnson, L.Dl, Igl, C.M. Goldade, A.L. Zimmerman, and B. R. Euliss. 2001. 15 16 Effects of Management Practices on Grassland Birds: American Bittern. Northern Prairie 17 Wildlife Research Center. Jamestown. ND. USA. Available at: 18 http://www.npwrc.usgs.gov/resource/literatr/grasbird/ambi/ambi.htm (Version 17FEB2000).
- 19 Barbour, R.W. and W.H. Davis. 1969. Bats of America. University of Kentucky Press.
- 20 Lexington, Kentucky. 286 pp. Cited in: R.M. DeGraaf, and M. Yamasaki. 2001. New England
- Wildlife: Habitat, Natural History, and Distribution. University Press of New England, Hanover,
 New Hampshire.
- Barron, M.G., H. Galbraith, and D. Beltman. 1995. Comparative reproductive and developmental
 toxicology of PCBs in birds. *Comparative Biochemistry and Physiology* 112C(1)1-14.
- Bartell, S.M., R.H. Gardner and R.V. O'Neill. 1992. *Ecological Risk Estimation*. Lewis
 Publishers, Boca Raton, FL.
- Bellis, E.D. 1968. Summer movement of red-spotted newts in a small pond. *Journal Herpetology*1:86-91.
- Belwood, J.J. and M.B. Fenton. 1976. Variation in the diet of *Myotis lucifugus. Canadian Journal of Zoology* 54:1674-1678.
- 31 Berger, A.J. 1951. Nesting density of Virginia and sora rails in Michigan. *Condor* 53:202. Cited
- 32 in: R.M. DeGraaf, and M. Yamasaki. 2001. New England Wildlife: Habitat, Natural History, and
- 33 Distribution. University Press of New England, Hanover, New Hampshire.

- 1 Bevelhimer, M.S., J.J. Bauchamp, B.E. Sample and G.R. Southworth. 1997. Estimation of
- 2 Whole-Fish Contaminant Concentrations From Fish Fillet Data. ES/ER/TM-202. Oak Ridge
- 3 National Laboratory, Oak Ridge, Tennessee.
- 4 Birnbaum, L.S., and M.J. DeVito. 1995. Use of toxic equivalency factors for risk assessment for 5 dioxins and related compounds. *Toxicology* 105:391-401.
- 6 Bishop, C.A., M.D. Koster, A.A. Chek, D.J.T. Hussell, and K. Jock. 1995. Chlorinated
- 7 hydrocarbons and mercury in sediments, red-winged blackbirds (Agelaius phoeniceus) and tree
- 8 swallows (Tachycineta bicolor) from wetlands in the Great Lakes-St. Lawrence River basin.
- 9 Environmental Toxicology and Chemistry 14:491-501.
- 10 Bishop, C.A., H.A. Mahony, S. Trudeau, and K.E. Pettit. 1999. Reproductive success and
- 11 biochemical effects in tree swallows (*Tachycineta bicolour*) exposed to chlorinate hydrocarbon

12 contaminants in wetlands of the Great Lakes and St. Lawrence River basin, USA and Canada.

- 13 Environmental Toxicology and Chemistry 18:263-271.
- 14 Bleavins, M.R., R.J. Aulerich, and R.K. Ringer. 1980. Polychlorinated biphenyls (Aroclor 1016
- 15 and 1242): effects on survival and reproduction in mink and ferret. Archives of Environmental
- 16 *Contamination and Toxicology* 9:627-635.
- Bosveld, A.T.C, and M. Van den Berg. 1994. Effects of polychlorinated biphenyls: dibenzo-pdioxins and dibenzofurans on fish-eating birds. *Environmental Reviews* 2:147-166.
- Brown, M. and J.J. Dinsmore. 1986. Implications of marsh size and isolation for marsh bird
 management. *Journal of Wildlife Management* 50:392-397.
- Brunström, B. 1988. Sensitivity of embryos from duck, goose, herring gull, and various chicken
 breeds to 3,3', 4,4'-tetrachlorobiphenyl. *Poultry Science* 67:52-57.
- Brunström, B. 1989. Toxicity of coplanar polychlorinated biphenyls in avian embryos.
 Chemosphere 19(1-6):765-768.
- Buckler, DR. 2002. Fish reproductive health assessment in PCB contaminated regions of the
 Housatonic River, Massachusetts, USA: Investigations of causal linkages between PCBs and fish
 health. Interim Report of Phase II Studies. Prepared by D.R. Buckler for U.S. Fish and Wildlife
 Service, Concord, New Hampshire and U.S. Environmental Protection Agency, Boston,
 Massachusetts. September 27, 2002.
- Bursian, S.J., R.J. Aulerich, B. Yamini, and D.E. Tillitt. 2003. Dietary exposure of mink to fish
 from the Housatonic River: Effects on reproduction and survival. Michigan State University,
 Department of Animal Science. Final Report. June 10, 2003.
- Burton, T.M. 1977. Population estimates, feeding habits, and nutrient and energy relationships of
 Notophthalmus v. viridescens, in Mirror Lake, New Hampshire. *Copeia*. 1977:139-143.
- 35 CCME (Canadian Council of Ministers of the Environment). 1999. Canadian Tissue Residue
 36 Guidelines for the Protection of Wildlife Consumers of Aquatic Biota: Polychlorinated

- 1 Biphenyls (PCBs). In: Canadian Environmental Quality Guidelines 1999. Canadian Council of
- 2 Ministers of the Environment, Winnipeg.
- Cogliano, V.J. 1998. Assessing the cancer risk from environmental PCBs. *Environmental Health Perspectives* 106(6):317-323
- 5 Conway, C.J. 1995. Virginia rail (Rallus limicola). In The Birds of North America. No. 421. A.
- 6 Poole and F. Gill, Editors. Academy of Natural Sciences, Washington, DC.
- 7 Custer, C.M., T.W. Custer, P.D. Allen, K.L. Stromborg, and M. Melancon. 1998.
- 8 Organochlorine contaminants and tree swallows nesting along the Fox River and in Green Bay,
- 9 Wisconsin. Environmental Toxicology and Chemistry 17:1786-1798.
- DeGraaf, R.M. and M. Yamasaki. 2001. New England Wildlife: Habitat, Natural History, and
 Distribution. University Press of New England, Hanover, New Hampshire.
- 12 Deschant, J.A., M.L. Sondreal, D.H. Johnson, L.Dl, Igl, C.M. Goldade, A.L. Zimmerman, and B.
- 13 R. Euliss. 2001. Effects of Management Practices on Grassland Birds: American Bittern.
- 14 Northern Prairie Wildlife Research Center, Jamestown, North Dakota. Available at:
- 15 http://www.npwrc.usgs.gov/resource/literatr/grasbird/ambi/ambi.htm (Version 17FEB2000).
- DiBello, F., S. Arthur, and W. Krohn. 1990. Food habits of sympatric coyotes, red foxes, and
 bobcats in Maine. *Canadian Field Naturalist* 104:403-408.
- 18 Eadie, W.R. 1944. The short-tailed shrew and field mouse predation. *Journal of Mammalogy*19 25:359-364.
- Eadie, W.R. 1948. Shrew-mouse predation during low mouse abundance. *Journal of Mammalogy* 29:35-37.
- Edwards, C.J., R.A. Ryder, and T.R. Marshall. 1990. Using lake trout as a surrogate of
 ecosystem health for oligotrophic waters of the Great Lakes. *Journal of Great Lakes Research*16:591-608.
- Ehrlich, P.R., D.S. Dobkin, and D. Wheye. 1988. *The Birder's Handbook A Field Guide to the Natural History of North American Birds*. Fireside Press, New York.
- Ehrlich, P.R., D.S. Dobkin, and D. Wheye. 1992. *The Birder's Handbook A Field Guide to the Natural History of North American Birds*. Fireside Press. New York.
- Eisler, R. 2000. Handbook of Chemical Risk Assessment: Health Hazards to Humans, Plants,
 and Animals. Volume 2 Organics. Lewis Publishers, Boca Raton, FL. ISBN 1-56670-506-1.
- 31 Environment Canada. 1997. Environmental Risk Assessments of Priority Substances und the
- 32 Canadian Environmental Protection Act: Guidance Manual. Chemicals Evaluation Division,
- 33 Commercial Chemicals Evaluation Branch, Environment Canada, Ottawa, Canada.

- 1 EPA (U.S. Environmental Protection Agency). 1993. Wildlife Exposure Factors Handbook.
- 2 Volumes I and II. Office of Research and Development, Washington, DC. EPA EPA/600/R-
- 3 93/187a, EPA/600/R-93/187b.
- 4 EPA (U.S. Environmental Protection Agency). 1997. Ecological Risk Assessment Guidance for
- 5 Superfund: Process for Designing and Conducting Ecological Risk Assessments. Interim Final
- 6 Report. Solid Waste and Emergency Response. EPA 540-R-97-006. June 1997.
- 7 EPA (U.S. Environmental Protection Agency). 1998. *Guidelines for Ecological Risk Assessment*.
 8 EPA/630/R-95/002F. Risk Assessment Forum, Washington, DC.
- 9 EPA (U.S. Environmental Protection Agency). 1999a. Hudson River PCBs Reassessment RI/FS;
- 10 Volume 2E- Baseline Ecological Risk Assessment. Prepared for EPA, Region 2 and the US
- 11 Army Corps of Engineers, Kansas City District. Prepared by TAMS/MCA. August 1999.
- 12 EPA (U.S. Environmental Protection Agency). 1999b. Issuance of Final Guidance: Ecological
- 13 Risk Assessment and Risk Management Principles for Superfund Sites; Signed 10/7/99.
- 14 Memorandum from Stephen D. Luftig, Office of Emergency and Remedial Response,
- 15 Washington, DC.
- 16 EPA (U.S. Environmental Protection Agency). 2000. Methods for Measuring the Toxicity and
- Bina (e.g. Environmental Protection Figure), 2000. Includes for Incust ing the Foundary and
 Bioaccumulation of Sediment-Associated Contaminants with Freshwater Invertebrates. EPA
 600/R-99/064. 192 pp.
- 19 EPA (U.S. Environmental Protection Agency). 2001. Ecological Characterization of the
- 20 Housatonic River: Revised Preliminary Draft. DCN to be assigned. Boston, Massachusetts.
- 21 Prepared by Woodlot Alternatives, Inc. with Roy F. Weston under EPA contract No. DACW33-
- 22 94-D-0009/032.
- Evans, D.O., G.J. Warren, and V.W. Cairns. 1990. Assessment and management of fish
 community health in the Great Lakes: synthesis and recommendations. *Journal of Great Lakes Research* 16:639-669.
- Fischer, L., R.F. Seegal, P. Ganey, I.N. Pessah, and P.R.S. Kodavanti. 1998. Toxicity of noncoplanar PCBs. *Toxicological Sciences* 41:49-61.
- 28 Georgii, S., G. Bachour, K. Failing, U. Eskins, I. Emadfa, and W.L. Budde. 1985. Mass
- 29 spectrometric determination of polychlorinated biphenyl congeners in foxes in Germany from
- 30 1983 to 1991. Archives of Environmental Contamination and Toxicology 26:1-6.
- Gibbs, J.P., S. Melvin, and F.A. Ried. 1992. American Bittern (*Botaurus lentiginosus*). In: *The Birds of North America*, No. 18. A. Poole, P. Stettenheim, and F. Gill, Editors. The Birds of
 North America, Inc. Philadelphia, PA. pp.1-11.
- Giesy, J.P., and K. Kannan. 1998. Dioxin-like and non-dioxin-like toxic effects of
 polychlorinated biphenyls (PCBs): implications for risk assessment. *Critical Reviews in Toxicology* 28(6):511-569.

- Griffith, L.A. and J.E. Gates. 1985. Food habits of cave-dwelling bats in the central
 Appalachians. *Journal of Mammalogy* 66:451-460.
- Gunn, J.M., S.U. Qadri, and D.C. Mortimer. 1997. Filamentous algae as a food source for the
 brown bullhead. *Journal of the Fisheries Research Board of Canada* 34:396-401.
- Hamilton, W.J., Jr. 1941. The foods of small forest mammals in eastern United States. *Journal of Mammalogy* 22:250-263.
- Hartel, K.E., D.B. Halliwell, and A.E. Launer. 2002. *Inland Fishes of Massachusetts. Massachusetts Audubon Society*. Lincoln, Massachusetts.
- 9 Healy, W.R. 1975. Terrestrial activity and home range in efts of *Notophthalmus v. viridescens*.
 10 *American Midland Naturalist* 93:131-138.
- 11 Hockman, J.G., and J.A. Chapman. 1983. Comparative feeding habits of red foxes (Vulpes
- 12 vulpes) and gray foxes (Urocyon cinereoargentus) in Maryland. American Midland Naturalist
- 13 110:267-285.
- Huang, Y., D.J. Hoffman, and W.H. Karasov. 2001. Oxidative stress in PCB 126-exposed frogs,
 Rana pipiens. Presentation, SETAC 22nd Annual Meeting, Baltimore, MD.
- Hull, R.N. and G.W. Suter II. 1994. Using the Weight-of-Evidence Approach for Ecological Risk
 Assessment at a DOE Facility. SETAC Denver, CO. November 1, 1994.
- Hunter, M.L., A.J.K. Calhoun, and M. McCollough. 1999. *Maine Amphibians and Reptiles*. The
 University of Maine Press, Orono, Maine. 252 pp.
- Jenssen, T.A., and W.D. Klimstra. 1966. Food habits of the green frog, *Rana clamitans*, in
 southern Illinois. *American Midland Naturalist* 76:169-182.
- Jung, R.E., and M.K. Walker. 1997. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on
 development of anuran amphibian. *Environmental Toxicology and Chemistry* 16:230-240.
- Knable, A.E. 1974. Seasonal trends in the utilization of major food groups by the red fox (*vulpes fulva*) in Union County, Illinois. *Trans. III. State Acad.* Sci. 66:113-115.
- Korschgen, L.J. 1959. Food habits of the red fox in Missouri. *Journal of Wildlife Management*23:168-176.
- Kurta, A. 1995. *Mammals of the Great Lakes Region*. The University of Michigan Press. Ann
 Arbor, MI. 392 pp.
- 30 Kurta, A. and R.H. Baker. 1990. *Eptesicus fuscus*. Mammalian Species No. 356:1-10. American
- 31 Society of Mammalogists. Shippensburg, Pennsylvania.

- 1 Landis, W.G., D.R.J. Moore, and S.B. Norton. 1998. Ecological risk assessment: looking in,
- 2 looking out. In: Pollution Risk Assessment and Management. P.E.T. Douben (Ed). John Wiley &
- 3 Sons, Toronto.
- 4 Leonards, P.E.G., Y. Zierikzee, U.A. Brinkman, W.P. Cofino, N.M. van Straalen, and B. van
- 5 Hattum. 1997. The selective dietary accumulation of planar polychlorinated biphenyls in the
- 6 otter (*Lutra lutra*). *Environmental Toxicology and Chemistry* 16(9):1807-1815.
- Linzey, D.W. and A.V. Linzey. 1973. Notes on food of small mammals from Great Smokey
 Mountains National Park, Tennessee-North Carolina. *J. Elisha Mitchell Science Society* 89:6-14.
- 9 MacNamara, M.C. 1977. Food habits of terrestrial adult migrants and immature red efts of the 10 red-spotted newt, *Notophthalmus viridescens. Herpetologica* 33:127-132.
- Martin, A.C., H.S. Zim, and A.L. Nelson. 1951. American Wildlife and Plants A Guide to
 Wildlife Food Habits. Dover Publications, Inc., New York.
- McCarty, J.P., and A.L. Secord. 1999. Reproductive ecology of tree swallows (*Tachycineta bicolor*) with high levels of polychlorinated biphenyl contamination. *Environmental Toxicology and Chemistry* 18:1433-1439.
- Menzie, C., M.H. Henning, J. Cura, K. Finkelstein, J. Gentile, J. Maughan, D. Mitchell, S.
 Petron, B. Potocki, S. Svirsky, and P. Tyler. 1996. Special report of the Massachusetts Weightof-Evidence Workgroup: A weight-of-evidence approach for evaluating ecological risks. *Human and Ecological Risk Assessment* 2:277-304.
- Mills, R.S., G.W. Barrett, and M.P. Farrell. 1975. Population dynamics of the big brown bat
 (*Eptesicus fuscus*) in southwestern Ohio. *Journal of Mammalogy* 56:591-604. Cited in: R.M.
 DeGraaf, and M. Yamasaki. 2001. *New England Wildlife: Habitat, Natural History, and Distribution*. University Press of New England, Hanover, New Hampshire.
- Moore, D.R.J., and S.M. Bartell. 2000. Estimating ecological risk of multiple stressors: advanced
 methods and difficult issues. In: *Multiple Stressors in Ecological Risk and Impact Assessment: Approaches to Risk Estimation.* S.A. Ferenc and J.A. Foran (Eds). Society of Environmental
 Toxicology and Chemistry (SETAC).
- 28 Moore, D.R.J., B.E. Sample, G.W. Suter, B.R. Parkhurst, and R.S. Teed. 1999. A probabilistic
- risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East
 Fork Poplar Creek, Oak Ridge, Tennessee. *Environmental Toxicology and Chemistry* 18:29412953.
- Newsted, J.L., J.P. Giesy, G.T. Ankley, D.E. Tillitt, R.A. Crawford, J.W. Gooch, P.D. Jones, and
 M.S. Denison. 1995. Development of toxic equivalency factors for PCB congeners and the
- 34 assessment of TCDD and PCB mixtures in rainbow trout. Environmental Toxicology and
- 35 *Chemistry* 14(5):861-871
- 36 Odum, R.R. 1977. Sora (*Porzana carolina*). In: *Management of migratory shore and upland* 37 *game birds in North America*, pp. 57-65. G.C. Sanderson, Editor. Washington, D.C.

- 1 Petranka, J.W. 1998. Salamanders from the United States and Canada. Smithsonian Institution
- 2 Press. Washington, DC.
- Powell, D.G. and R.M. Case. 1982. Food habits of the red fox in Nebraska. *Trans. Nebr. Acad. Sci. and Affil. Soc.* 10:13-16.
- 5 Robertson, R.J., B.J. Stutchbury, and R.R. Cohen. 1992. Tree Swallow (*Tachycineta bicolor*). In:
- 6 The Birds of North America, No. 11, pp. 1-26. A. Poole, P. Stettenheim, and F. Gill, Editors. The
- 7 Birds of North America, Inc., Philadelphia, Pennsylvania.
- 8 Safe, S.H. 1994. Polychlorinated Biphenyls (PCBs): Environmental impact, biochemical and
 9 toxic responses, and implications for risk assessment. *Critical Reviews in Toxicology* 24(2):8710 149.
- 11 Sallabanks, R. and F.C. James. 1999. American Robin (Turdus migratorius) In: The Birds of
- 12 North America, No. 642, pp. 1-27. A. Poole, P. Stettenheim, and F. Gill, Editors. The Birds of
- 13 North America, Inc., Philadelphia, Pennsylvania.
- Shugart, L.R. 1996. Molecular markers to toxic agents. In: *Ecotoxicology: A Hierarchical Treatment*, pp. 154-156. M.C. Newman and C.H. Jagoe, Editors. CRC Lewis Publishers, New
 York.
- 17 Sibley, D.A. *The Sibley Guide to Bird Life & Behavior*. 2001. Alfred A. Knopf, Inc., New York.
- 18 Spromberg, J.A., B.M. John, and W.G. Landis. 1998. Metapopulation dynamics: Indirect effects
- 19 and multiple discrete outcomes in ecological risk assessment. Environmental Toxicology and
- 20 *Chemistry* 17(8):1640-1649.
- Stewart, M.M. and P. Sandison. 1973. Comparative food habits of sympatric mink frogs,
 bullfrogs, and green frogs. *Journal of Herpetology* 6:241-244.
- 23 Suter, G.W., II, B.E. Sample, D.S. Jones, T.L. Ashwood, and J.M. Loar. 1995. Approach and
- 24 Strategy for Performing Ecological Risk Assessments for the U.S. Department of Energy's Oak
- 25 *Ridge Reservation.* Oak Ridge National Laboratory, Oak Ridge, TN. ES/ER/TM-33/R2.
- 26 Tanner, W.D. and G.O. Hendrickson. 1956. Ecology of the sora in Clay County, Iowa. Iowa Bird
- 27 Life 26(4):78-81. Cited in: R.M. DeGraaf, and M. Yamasaki. 2001. New England Wildlife:
- Habitat, Natural History, and Distribution. University Press of New England, Hanover, New
 Hampshire.
- Taub, F.B. 1989. Standardized aquatic microcosms. *Environmental Science and Technology* 23(9):1064-1066.
- 32 Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Freely, J.P. Giesy,
- 33 A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X.R van Leeuwen, A.K.
- 34 Djien Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind,
- 35 M. Younes, F. Waern, and T. Zacharewski. 1998. Toxic equivalency factors (TEFs) for PCBs,
- 36 PCDDs, PCDFs for humans and wildlife. *Environmental Health Perspectives* 106(12):775-792.

- 1 van Zyll de Jong, C.G. 1985. Handbook of Canadian Mammals Bats. National Museum of
- 2 Natural Sciences. Ottawa, Canada. 212 pp. Cited in: R.M. DeGraaf, and M. Yamasaki. 2001.
- 3 New England Wildlife: Habitat, Natural History, and Distribution. University Press of New
- 4 England, Hanover, New Hampshire.
- 5 Walker, M.K., P.M. Cook, A.R. Batterman, B.C. Butterworth, C. Berini, J.J. Libal, L.C.
- 6 Hufnagle, and R.E. Peterson. 1994. Translocation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin from
- 7 adult female lake trout (Salvelinus namaycush) to oocytes: effects on early life stage
- 8 development and sac fry survival. *Can. J. Fish. Aquat. Sci.* 51:1410-1419.
- 9 Weatherhead, P.J. and S.B. McRae. 1990. Brood care in American robins: implications for 10 mixed reproductive strategies by females. *Anim. Behav.* 39:1179-1188.
- 11 WESTON (Roy F. Weston, Inc.). 2000a. Supplemental Investigation Work Plan for the Lower
- 12 Housatonic River. Prepared for U.S. Army Corps of Engineers and U.S. Environmental
- 13 Protection Agency. 22 February 2000. DCN GEP2-020900-AAME.
- 14 WESTON (Roy F. Weston, Inc.). 2001a. Quality Assurance Project Plan, Vol. I Text, Vol. II –
- 15 Appendix A, Vol. IIA Appendix A, cont'd., Vol. IV Appendices E and F. Prepared for U.S.
- 16 Army Corps of Engineers and U.S. Environmental Protection Agency. DCN GE-021601-
- 17 AAHM.
- 18 Whitford, A.G., and A. Vinegar. 1966. Homing, survivorship, and overwintering of larvae in 19 *Ambystoma maculatum. Copeia.* 1966:515-519.
- 20 Whitaker, J.O. 1995. Food of the big brown bat *Eptesicus fuscus* from maternity colonies in 21 Indiana and Illinois. *American Midland Naturalist* 134:346-360.
- Whitaker, J.O., Jr. and M.G. Ferraro. 1963. Summer food of 220 short-tailed shrews from Ithaca,
 New York. *Journal of Mammalogy* 44:419.
- Whitaker, J.O. and W.J. Hamilton. 1998. *Mammals of the Eastern United States*. Cornell
 University Press. Ithaca, New York.
- Woodlot Alternatives, Inc. 2003. Amphibian reproductive success within vernal pools associated
 with the Housatonic River, Pittsfield to Lenoxdale, Massachusetts. DCN: 99-1275.
- Wu, J., J.L. Vankat, and Y. Barlas. 1993. Effects of patch connectivity and arrangement on animal metapopulation dynamics: a simulation study. *Ecological Modeling* 65:221-254.
- 30 Zabel, E.W., P.M. Cook, and R.E. Peterson. 1995. Toxic equivalency factors of polychlorinated
- 31 dibenzo-*p*-dioxin, dibenzofuran and biphenyl congeners based on early life stage mortality in
- 32 rainbow trout (Oncorhynchus mykiss). Aquat. Toxicol. 31:315-328.