INJURIES TO FISHERY RESOURCES, LOWER FOX RIVER/GREEN BAY NATURAL RESOURCE DAMAGE ASSESSMENT

Final Report

Prepared for:

U.S. Fish and Wildlife Service
U.S. Department of the Interior
U.S. Department of Justice

Prepared by:

Stratus Consulting Inc.
P.O. Box 4059
Boulder, CO 80306-4059
(303) 381-8000

November 8, 1999

William F. Hartwig
U.S. Fish and Wildlife Service, Regional Director and Authorized Official

CONTENTS

Figures v
Tables vii
Acronyms xi

Chapter 1 Introduction 1-1

Chapter 2 Fishery Resources in the Assessment Area

2.1 Introduction 2-1
2.2 General Description of the Fishery Resource 2-2
2.3 Commercial and Recreational Fishing 2-3
2.3.1 Commercial Fishery 2-4
2.3.2 Sport Fishery 2-6
2.4 Importance of the Fishery to the Oneida and Menominee Tribes 2-6
2.5 Conclusions 2-7

Chapter 3 PCB Pathway Determination and Exposure for Fishery Resources

3.1 PCB Pathway Determination 3-1
3.2 PCB Exposure and Accumulation in Fish 3-4

Chapter 4 Injury Assessment Approach

4.1 Overview of Adverse Changes in Fish Viability Caused by PCBs 4-1
4.2 Injury Definitions and Measures 4-3
4.2.1 Injury Definitions 4-3
4.2.2 Injury Measures 4-4
4.3 Injury Assessment Approach 4-6
4.3.1 Fish Consumption Advisories and FDA Tolerance Level Exceedences 4-6
4.3.2 Overview of Assessment of Adverse Changes to Fish Viability 4-7

Chapter 5 Fish Consumption Advisory Injuries

5.1 Introduction 5-1

5.2 Summary of 1998 Fish Consumption Advisory Report 5-1
5.3 Fish Consumption Advisory Update 5-8
5.3.1 Wisconsin Fish Consumption Advisories 5-8
5.3.2 Michigan Fish Consumption Advisories 5-13
5.4 Injury Determination 5-18
5.4.1 Injury Definitions 5-18
5.4.2 Injury Determination and Spatial/Temporal Extent of Injuries 5-18

Chapter 6 Adverse Health Effect Injuries to Walleye

6.1 Introduction 6-1
6.2 Summary of Data Reported in Barron et al. (1999) 6-1
6.3 Supplemental Liver PCB Analysis 6-7
6.4 Integration of Barron et al. (1999) Data and Supplemental PCB Analysis 6-7
6.5 Injury Determination 6-10
6.5.1 Injury Definition and Measurements 6-10
6.5.2 Injury Determination 6-10

Chapter 7 Reproductive Injuries to Lake Trout

7.1 Introduction 7-1
7.2 Description of the Lake Trout Resource 7-2
7.3 PCB Toxicity to Lake Trout 7-5
7.3.1 Description of Early Life Stage Toxicity of Coplanar PCBs and TCDD 7-5
7.3.2 The TCDD Toxicity Equivalence Approach to Expressing Toxic Doses 7-7
7.3.3 Thresholds for Early Life Stage Toxicity to Lake Trout 7-9
7.4 Previous Studies on Factors Associated With Lake Michigan Lake Trout Embryomortality 7-11
7.4.1 Contaminant Exposure 7-11
7.4.2 Thiamine Deficiency 7-15
7.5 USGS Studies on Lake Michigan Lake Trout Reproduction: 1996-1998 7-16
7.5.1 Methods 7-17
7.5.2 Results 7-21
7.5.3 Conclusions 7-21
7.6 Supplemental NRDA and USGS Studies on PCB/Thiamine Interaction 7-28
7.7 Comparison of Estimated 1975-1995 PCB Concentrations in Lake Trout Eggs with Injury Thresholds 7-29

7.7.1 Methods 7-29
7.7.2 Results 7-34
7.7.3 Conclusions 7-40
7.8 Summary and Injury Determination 7-41
7.8.1 Injury Definition and Measurements 7-41
7.8.2 Weight of Evidence Evaluation 7-41
7.8.3 Uncertainties 7-42
7.8.4 Injury Determination Conclusion 7-43

Chapter 8 Fish Injury Assessment Summary 8-1

Chapter 9 References 9-1

Appendices

A PCB Concentrations in Walleye Livers Collected in 1997
B Egg and Fry Viability and Contaminant Data in 1996-1998 USGS Studies

FIGURES

3-1 GBMBS PCB Pathways in Lower Fox River/Green Bay System 3-3
3-2 Fish Sampling Zones Used in Green Bay Mass Balance Study 3-7
3-3 Total PCB Concentrations in Green Bay Walleye, 1989 3-9
3-4 Mean PCB Concentrations in Lower Fox River/Green Bay Forage Fish and Predatory Fish, by Zone 3-10

5-1 Spatial Extent of PCB Fish Consumption Advisories in the Lower Fox River/ Green Bay Area. 5-22

6-1 1996-1997 Walleye Collection Locations 6-2
6-2 Individual Fish Liver PCB Concentrations in Fish with and without FCA or HT 6-9

7-1 Historical Lake Trout Yield/Area for Each of the Great Lakes 7-3
7-2 Annual Lake Trout Stocking in Green Bay and Wisconsin Waters of Lake Michigan near Green Bay 7-4
7-3 Regression for Estimating Thiamine Concentrations in Fertilized versus Unfertilized Eggs 7-18
7-4 Regression for Estimating TEQ Contributed by PCBs from Total PCB Concentration 7-20
7-5 Thiamine Concentration in Lake Trout Eggs vs Measures of Egg and Fry Viability . 7-22
7-6 PCB Concentration in Lake Trout Eggs vs Measures of Egg and Fry Viability 7-23
7-7 Total TEQ Concentration in Lake Trout Eggs vs Measures of Egg and Fry Viability 7-24
7-8 Thiamine Concentration in Lake Trout Eggs vs Fry Mortality 7-25
7-9 PCB Concentration in Lake Trout Eggs vs Measures of Egg and Fry Viability for Samples with Thiamine Concentrations of Less than 1000 pmol/g 7-26
7-10 Total TEQ Concentration vs Measures of Egg and Fry Viability for Samples with Thiamine Concentrations of Less than 1000 pmol/g 7-27
7-11 Relationship between the Mean and Standard Deviation and between the Mean and the Mean/Standard Deviation Ratio of Total PCB Concentration Measurements for Walleye Collected from the Fox River and Green Bay between 1978 and 1996 and for Carp Collected from Green Bay between 1977 and 1992. 7-32
7-12 Graphical Depiction of Method for Estimating Percent Exceedence of the LD10 and LD50 for Each Year 7-33
7-13 Estimated Mean Total PCB Concentrations in Lake Michigan Lake Trout Eggs, 1974-1998. 7-37

7-14 The Relative Contributions to the Total TEQ by PCBs and by PCDDs and PCDFs . . 7-38
7-15 Estimated exceedences of the LD10 and LD50 for PCB, PCDD,
and PCDF exposure since 1974. 7-39

TABLES

2-1 Average Annual Commercial Harvest on Green Bay, 1978-1990 2-5
2-2 1998 Commercial Harvest of Selected Species from the Wisconsin Waters of Lake Michigan and Green Bay 2-5

3-1 Lower Fox River/Green Bay Fish Species in Which PCBs Have Been Measured 3-5

4-1 Overview of Injury Assessment of Adverse Changes in Fish Viability 4-8

5-1 Summary of Fish Species in the Lower Fox River that Have Exceeded the FDA Tolerance for PCBs, 1971-1995 5-2
5-2 Summary of Fish Species in Green Bay that Have Exceeded the FDA Tolerance for PCBs, 1971-1995 5-3
5-3 Summary of Fish Species in Northern Lake Michigan that Have Exceeded the FDA Tolerance for PCBs, 1971-1995 5-4
5-4 Summary of Fish Species in the Lower Fox River for Which PCB Consumption Advisories Have Been Issued by Wisconsin, 1976-1997 5-5
5-5 Summary of Fish Species in Green Bay for Which PCB Consumption Advisories Have Been Issued by Wisconsin or Michigan, 1976-1997 5-6
5-6 Summary of Fish Species in Lake Michigan for Which PCB Consumption Advisories Have Been Issued by Wisconsin or Michigan, 1976-1997 5-7
5-7 Wisconsin Fish Consumption Advisories for the Lower Fox River from Little Lake Butte des Morts to the Dam at DePere 5-9
5-8 Wisconsin Fish Consumption Advisories for the Lower Fox River from the Mouth at Green Bay up to DePere Dam 5-10
5-9 Wisconsin Fish Consumption Advisories for Wisconsin Waters of Green Bay South of Marinette and its Tributaries including the Menominee, Oconto, and Peshtigo Rivers from Their Mouths Up to the First Dam 5-11
5-10 Wisconsin Fish Consumption Advisories for the Wisconsin Waters of Lake Michigan and its Tributaries up to the First Dam, including the Root River, Milwaukee River, Sheboygan River, Manitowoc River, and Kewaunee River 5-12
5-11 Michigan Fish Consumption Advisories for the Michigan Waters of Green Bay South of Cedar River 5-14
5-12 Michigan Fish Consumption Advisory for Little Bay de Noc 5-16
5-13 Michigan Fish Consumption Advisories for Lake Michigan North of Frankfurt 5-17
5-14 Summary of Fish Species in the Lower Fox River for Which PCB Consumption Advisories Have Been Issued by Wisconsin, 1976-1999 5-19

5-15 Summary of Fish Species in Green Bay for Which PCB Consumption Advisories Have Been Issued by Wisconsin or Michigan, 1976-1997 5-20
5-16 Summary of Fish Species in Lake Michigan for Which PCB Consumption Advisories Have Been Issued by Wisconsin or Michigan, 1976-1997 5-21

6-1 Mean and Standard Deviation of Total PCB Concentrations in Walleye Collected in 1996 6-4
6-2 Foci of Cellular Alteration and Hepatic Tumors in Male and Female Walleye 6-5
6-3 Mean and Standard Deviation of Total PCB Concentrations in Walleye Livers Collected in Assessment and Reference Areas in 1997 6-8
6-4 Mean and Standard Deviation of Total PCB Concentrations in Walleye Livers Collected in Assessment and Reference Areas in 1996 and 1997 Combined 6-8
6-5 Summary of Injury Determination Results for Walleye 6-11

7-1 TCDD LD50s for Lake Trout Embryomortality 7-10
7-2 Investigations of the Relationship Between Contaminants and Egg and Fry Viability of Lake Michigan Lake Trout 7-12
7-4 Toxic Equivalency Factors for PCDD, PCDF, and PCB Congeners 7-19
7-5 Model Parameters for Estimating Historical Egg Total PCB Concentrations 7-35
7-6 Mean Concentrations of Lake Trout Total PCBs, TEQ from PCBs, and Total TEQ Estimated by the Mixed-Order Model and the Nonzero Asymptote Model 7-36

ACRONYMS

AhR	aryl hydrocarbon receptor
EMS	early life stage mortality syndrome
EPA	Environmental Protection Agency
EROD	ethoxyresorufin-O-deethylase
FCA	hepatic foci of cellular alteration
FDA	Food and Drug Administration
GBMBS	Green Bay Mass Balance Study
HT	hepatic tumors
LD10	dose lethal to 10% of the population
LD50	dose lethal to 50% of the population
NOAA	National Oceanic and Atmospheric Administration
NRDA	natural resource damage assessment
PCBs	polychlorinated biphenyls
PCDD	polychlorinated dibenzodioxin
PCDF	polychlorinated dibenzofuran
REP	relative potency
TCDD	2,3,7,8-tetrachloro-*p*-dibenzodioxin
TEF	toxicity equivalency factor
TEQ	toxicity equivalents
USFWS	U.S. Fish and Wildlife Service
USGS	United States Geological Survey

CHAPTER 1
INTRODUCTION

This document presents the determination and quantification of injuries to fishery resources that have resulted from releases of polychlorinated biphenyls (PCBs) from paper company facilities along the Lower Fox River, Wisconsin. This injury assessment is part of the natural resource damage assessment (NRDA) being performed for the Lower Fox River/Green Bay environment by the U.S. Department of the Interior (the Department) through the U.S. Fish and Wildlife Service (USFWS, or the Service), the National Oceanic and Atmospheric Administration (NOAA), the Oneida Tribe of Indians of Wisconsin (Oneida Tribe), and the Menominee Indian Tribe of Wisconsin (Menominee Tribe) (collectively, the Trustees).

The Trustees have issued several NRDA reports that address injuries to natural resources of the Lower Fox River/Green Bay ecosystem. These reports provide documentation of:

- PCB releases and transport pathways from Lower Fox River paper companies to the Lower Fox River/Green Bay environment (Stratus Consulting Inc., 1999d)
- injuries to avian resources in the Lower Fox River/Green Bay environment that result from the PCB releases and transport (Stratus Consulting Inc., 1999b)
- injuries to surface water resources in the Lower Fox River/Green Bay environment that result from the PCB releases and transport (Stratus Consulting Inc., 1999c)
- PCB fish consumption advisories and exceedences of federal PCB tolerance levels in fish of the Lower Fox River/Green Bay environment (Stratus Consulting Inc., 1998)
- the methods, results, and conclusions of studies conducted by the Service on the adverse effects of PCBs on walleye fish health in the Lower Fox River/Green Bay environment (Stratus Consulting Inc., 1999a).

The purpose of this report is to present an injury determination and quantification for fishery resources of the Lower Fox River/Green Bay ecosystem. This injury determination and quantification relies on the reports previously prepared by the Trustees, on additional information that supplements these previous reports, and on data from the scientific literature.

This report will be used by the Trustees to assist in the determination of the amount and type of restoration required to compensate the public for the injuries. A companion report prepared by the Service describes the nature and magnitude of damages associated with recreational fishing

losses resulting from the fish injuries described in this report (Stratus Consulting Inc., 1999e). This process was described in the initial Restoration and Compensation Determination Plan, published September 21, 1998 [63 FR 50,254] and will result in completion of the Restoration and Compensation Determination Plan.

This report is organized as follows (with brief chapter summaries also provided):

Chapter 2 describes the fishery resources of the Lower Fox River/Green Bay environment, and includes descriptions of the ecological, economic, and tribal cultural importance of the resource. The resource is a nationally significant resource that is vital in providing important ecological services, human use services, and tribal cultural services.

Chapter 3 presents a PCB pathway determination for the fishery resources and describes PCB exposure to fish in the Lower Fox River/Green Bay system. The chapter demonstrates that PCB releases from Lower Fox River paper company facilities are the primary source of PCBs in the Lower Fox River and Green Bay. Fish throughout the river and bay are exposed to the released PCBs. Fish exposure to PCBs occurs through direct contact with the water and sediments and through consumption of prey items that are contaminated with PCBs. The accumulation of PCBs tends to be highest in predatory species and in areas of the river and bay with the highest sediment and surface water PCB concentrations. The chapter also concludes that PCB transport and exposure pathways continue to result in fish exposure to PCBs.

Chapter 4 describes the injury assessment approach used for fishery resources. The approach, which follows the Service's Assessment Plan [61 FR 43558] and addenda [62 FR 33804; 63 FR 25144], includes evaluation of injuries according to the following three definitions of injury for biological resources:

- PCB concentrations that exceed action or tolerance levels established under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342, in edible portions of organisms [43 CFR § 11.62(f)(1)(ii)]; or

- PCB concentrations that exceed levels for which an appropriate State health agency has issued directives to limit or ban consumption of fish [CFR § 11.62(f)(1)(iii)]; or

- PCB concentrations sufficient to cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction), or physical deformations [43 CFR § 11.62(f)(1)(i)].

The assessment of fish consumption advisory and exceedence of tolerance level injuries is based on the Service's 1998 report (Stratus Consulting Inc., 1998), updated with more recent information. Adverse changes in viability are assessed in terms of adverse effects on fish health in walleye and adverse reproductive effects in lake trout. A preliminary assessment was also

conducted for adverse effects on fish health in brown trout and lake trout. These injuries were assessed through a combination of field and laboratory studies and information from the scientific literature. Chapter 4 also includes documentation that the injury assessment approach and measured biological responses meet the Department's injury determination criteria.

Chapter 5 describes fish injuries based on exceedences of the Food and Drug Administration (FDA) PCB tolerance level in fish tissue and based on the establishment of state fish consumption advisories because of PCBs in fish tissue. The chapter concludes that PCB concentrations in the edible tissue of many fish species in the Lower Fox River/Green Bay environment exceed the FDA PCB tolerance level. The exceedences have occurred since PCBs were first measured in Lower Fox River/Green Bay fish and continue to the present. The chapter also summarizes the fish consumption advisory programs, approaches, and methods of the State of Michigan and the State of Wisconsin, and presents the PCB advisories issued by the two states for fish from the Lower Fox River/Green Bay environment. PCB advisories have been in place since the mid-1970s, and cover many fish species throughout the Lower Fox River/Green Bay environment.

Chapter 6 presents information on adverse health effect injuries to walleye in the Lower Fox River/Green Bay environment. The information in the chapter demonstrates that walleye from the assessment area have a higher prevalence of liver tumors and pre-tumors compared with walleye from control areas. Assessment area walleye also have higher PCB concentrations, and fish liver tumors and pre-tumors are known to be caused by PCB exposure. The association of increased tumor frequency with PCB exposure in walleye demonstrates that environmental exposure to PCBs in the Lower Fox River and Green Bay has resulted in injury to walleye.

Chapter 7 presents the results of an assessment of injuries to lake trout in Green Bay and Lake Michigan resulting from PCB exposure. Based on the information and analyses presented in this chapter, the Trustees conclude that there is indication that PCBs caused lake trout reproductive failure in the 1970s. However, given that evidence suggests little to no reproductive effects of PCBs since 1980 and that other factors appear to be substantially more important to the survival of lake trout fry, the Trustees determine that current data do not support the conclusion that lake trout in Green Bay and Lake Michigan are injured by the PCBs released from Fox River paper companies.

Chapter 8 summarizes the injury determination and quantification for fishery resources of the Lower Fox River/Green Bay. Fish throughout the Lower Fox River and Green Bay are injured as a result of the extensive PCB fish consumption advisories established by the states of Michigan and Wisconsin, and by PCB concentrations in fish tissue that exceed the FDA tolerance level. These injuries have occurred from the mid-1970s to the present and cover many different fish species throughout the entire area of the Lower Fox River and Green Bay. The magnitude of the service losses that result from these injuries is quantified in a separate report (Stratus Consulting, 1999e). In addition, walleye of the Lower Fox River and Green Bay are injured as a result of higher incidences of liver tumors and pre-tumors associated with PCB exposure than in reference populations.

CHAPTER 2
FISHERY RESOURCES IN THE ASSESSMENT AREA

2.1 INTRODUCTION

The Green Bay fishery provides vital ecologic, economic, recreational, and tribal services. As part of the larger Lake Michigan and Great Lakes ecosystem, Green Bay provides important fish habitat and supports a diverse and productive fishery. The fishery is essential to the Green Bay food web, providing food for the region's piscivorous birds and mammals. The fishery also supports a variety of human uses including commercial fishing, recreational fishing, and tribal cultural resource use.

To address the complex issues of fisheries restoration across the Great Lakes, resource agencies from the United States and Canada chose to develop a common strategy for fisheries management. In 1980, the Service and the National Marine Fisheries Service joined 10 other state, provincial, or Federal fishery agencies as signatories to the Joint Strategic Plan for Management of Great Lakes Fisheries (Great Lakes Fishery Commission, 1980). Newly formed tribal fishery agencies became signatories to the strategic plan in 1989 and a third revision of the plan was approved in 1997 (Great Lakes Fishery Commission, 1997). Lost fishing opportunities, instability of fish communities, inadequate environmental quality, and competition and conflict among users of the fishery resources remain as the primary issues addressed by the strategic plan to achieve the common goal statement for Great Lakes Fishery Agencies:

> *To secure fish communities, based on foundations of stable self-sustaining stocks, supplemented by judicious plantings of hatchery-reared fish, and provide from these communities an optimum contribution of fish, fishing opportunities and associated benefits to meet the needs identified by society for:*
>
> *wholesome food,*
> *recreation,*
> *cultural heritage,*
> *employment and income,*
> *and a healthy aquatic ecosystem.*

Recognizing that the economic and cultural value of the Great Lakes fisheries continued to be damaged by excessive harvest, loss of critical habitat, invasions of nonindigenous species, and contaminant burdens, the U.S. Congress enacted the Great Lakes Fish and Wildlife Restoration Act of 1990 (16 U.S.C. 941). The Act directed the Service to conduct a comprehensive study of the status of, and the assessment, management, and restoration needs of, the fishery resources of

the Great Lakes basin. The study was to include, in part, recommendations regarding 1) an action plan to analyze the effects of contaminant levels on fishery resources, 2) an action plan for the cooperative restoration and enhancement of depleted, nationally significant fish stocks, including lake trout, yellow perch, lake sturgeon, walleye, forage fish, and Atlantic salmon, and 3) important fishery resource habitat and other areas that should be protected, restored, or enhanced for the benefit of the Great Lakes fishery resources. The completed study report provides findings for Lake Michigan and 32 recommendations for priority activities to achieve restoration of the Great Lakes (Burkett et al., 1995). The Act also directed the Service to establish coordination and fishery resources offices to enhance the operational and coordinating activities of the Service related to fishery protection, restoration, maintenance, and enhancement of the Great Lakes.

The emphasis and effort expended by governmental agencies in managing the fishery resources of Green Bay and Lake Michigan are indications of the importance of the resource in supplying vital ecologic, cultural, and economic services. The following sections briefly describe some of these services provided by the fishery resource.

2.2 GENERAL DESCRIPTION OF THE FISHERY RESOURCE

Green Bay has one of the most productive fisheries in the Great Lakes (U.S. EPA and Environment Canada, 1995), supporting a diversity of habitats and forage that, in turn, support a complex food web. Each region of Green Bay offers different habitats to its resident fish (Bertrand et al., 1976). Northern Green Bay, north of Chambers Island, is characterized by deeper water, with about 85% of the area under more than 30 feet of water and maximum depths of 160 feet. The northern bay thus provides mostly deep, cold-water habitat. Little and Big Bay de Noc, on Michigan's Upper Peninsula, are large open bays of moderate depths and are suitable to both warm-water fish and some colder water fish. Southern Green Bay, south of Chambers Island, provides habitat for warm-water fish, with half of the region's area less than 30 feet deep. Estuaries and sandbars, occurring especially on the west shore and south end of Green Bay, are warm water habitats found primarily at the delta fans of rivers. Finally, the shoreline of the eastern bay along the Door Peninsula is rocky and steep, and the bays along this shoreline tend to have relatively warm shallow waters (Bertrand et al., 1976).

The Fox River has three primary habitat types, differentiated by substrate (Exponent, 1999). The river downstream of DePere Dam consists primarily of soft, silty substrate (composed of silty aqueous sediments), while above the dam a mix of sand, cobble and rock predominates. The third type of habitat is characterized by submerged aquatic vegetation and can be found throughout the Lower Fox River (Exponent, 1999). Gravel/cobble-dominated habitats generally support greater fish species diversity than sand or silt substrate and are used by many fish species as spawning habitat. For example, walleye and smallmouth bass spawn exclusively on rock or gravel beds (Exponent, 1999). Aquatic vegetation provides forage, cover, spawning, and nursery habitat to fish and support increased species richness by increasing the structural complexity of shallow water habitats (Exponent, 1999). For example, species such as northern pike utilize beds of

submerged aquatic vegetation in the Lower Fox River as nursery habitats and feeding grounds (Exponent, 1999).

Shorelines offer a wide diversity of habitat to warmer-water fish, while the deep, open waters found in parts of Green Bay attract fish such as trout and salmon which prefer colder temperatures. Small forage fish, including alewives, gizzard shad, and spottail shiners, feed on insects, zooplankton, and bottom-dwelling invertebrates. They tend to occupy nearshore habitats in the littoral zone, including wetlands, where aquatic vegetation provides cover and forage. Forage fish provide an important trophic link between zooplankton and game fish (University of Wisconsin-Green Bay, 1993) such as walleye and northern pike. Beaches, with fewer plants, more sediments, and stronger wave action, appear to be preferred by fewer species than wetlands for nearshore habitat (Brazner and Magnuson, 1994). Finally, bottom feeders such as channel catfish are omnivores that scavenge in the sediments of the bay, providing another trophic link between bottom dwelling invertebrates and predator fish.

Several fish species, including northern pike, walleye, smallmouth bass, yellow perch, and lake sturgeon, have been documented to migrate between the waters of Green Bay and tributaries to Green Bay (Cogswell, 1998; Cogswell and Bougie, 1998; Green Bay Fisheries Resources Office, 1998). Fish migration has also been documented between Green Bay and Lake Michigan (Wisconsin Department of Natural Resources, 1996) and within Green Bay itself (U.S. Fish and Wildlife Service, 1998).

The species composition and trophic relationships of the Green Bay fishery resource are much different today than they were before the onset of significant human impacts in the 19th century (University of Wisconsin-Green Bay, 1993). Overfishing, the introduction of exotic species, habitat destruction, and pollution have all contributed to shifts in the fish community composition (Bertrand et al., 1976; Wisconsin Department of Natural Resources, 1988).

Despite these historical changes in the fish community, the fishery resource continues to provide valuable ecological services. The Green Bay ecosystem supports a wide diversity of piscivorous birds that depend on fish as a food source, including bald eagles, terns, herons, ducks, and double-crested cormorant (Stratus Consulting Inc., 1999b). Piscivorous mammals that depend on the fishery resource in the area include otter and mink (Linscombe et al., 1982; Toweill and Tabor, 1982; Allen et al., 1987).

2.3 COMMERCIAL AND RECREATIONAL FISHING

Commercial fishing and sport fishing are both important human uses of the Lower Fox River/Green Bay fishery resource (Colborn et al., 1990). Commercial fishing dominated historically and is still a major industry in Green Bay. However, recreational fishing currently contributes more to the economy than does commercial fishing. For example, in 1985, the landed value of commercial fishing in the Great Lakes was estimated to be $41 million, compared to

estimated spending by sport anglers of $2 billion (Colborn et al., 1990). Since sport fishing is considered to be more valuable economically, Great Lakes resource agencies have adopted policies that favor recreational fishing over commercial fishing. The Trustees' NRDA report on recreational fishing damages associated with fish consumption advisories in the assessment area presents a more thorough evaluation of recreational fishing use (Stratus Consulting Inc., 1999e).

2.3.1 Commercial Fishery

The commercial fishing industry began in Green Bay in the early 19th century (Bertrand et al., 1976). Species important in the northern bay were lake whitefish and lake trout, while Little and Big Bay de Noc offered plentiful lake sturgeon, northern pike, lake herring, perch, suckers, and black bass. In the southern bay, popular species were lake herring, lake whitefish, lake trout, walleye, perch, suckers, pickerel, and lake sturgeon. Catfish and suckers, along with muskellenge, carp, white bass, crappies, sunfish, and shad, were also harvested in the southern bay and the Fox River (Bertrand et al., 1976).

The commercial fishery of the Great Lakes and Green Bay has declined in recent years (U.S. EPA and Environment Canada, 1995). Degraded by a combination of factors, including overfishing, pollution, and introduction of exotic species, the fishery has changed such that the more valuable larger fish have given way to smaller and relatively low-value species. Alewives, harvested primarily for animal feed and also for fish food and fertilizer, were a crucial commercial species in Green Bay through the early 1970s, constituting between 50% and 85% of the total annual alewife harvest from Lake Michigan (Bertrand et al., 1976). However, alewife harvest has declined in recent years and the alewife harvest in Green Bay from 1978 to 1990 comprised only about 12% of the average annual Lake Michigan catch (Table 2-1).

Table 2-1 summarizes the average annual harvest of the major commercial fish species from Green Bay between 1978 and 1990. Average annual harvest between these years included approximately 2.4 million pounds for alewife and approximately 2.5 million pounds for lake whitefish. Other species for which the annual harvest for these same years exceeded 100,000 pounds are smelt, suckers, perch, and carp.

More recent data reflect changes in the commercial fishery. The 1998 commercial catch for several important commercial species in the Wisconsin waters of Green Bay is summarized in Table 2-2. Chubs and lake whitefish dominated the 1998 harvest, and the smelt harvest reflects a continuing lakewide decline in the smelt population (Wisconsin Department of Natural Resources, 1999). The commercial harvest of yellow perch was limited by quota to 200,000 pounds from Green Bay (Wisconsin Department of Natural Resources, 1999).

Table 2-1
Average Annual Commercial Harvest on Green Bay, 1978-1990[a]

Species	Annual Catch from Green Bay (1,000s of pounds)[b]	Percent of Lake Michigan Catch from Green Bay
Lake whitefish	2500	46.90
Alewives	2446	12.46
Smelt	2246	80.89
Suckers	1211	97.54
Perch	440	28.40
Carp	381[c]	98.71
Chubs	21	0.77
Round whitefish (*Prosopium cylindraceum*)	18	8.59
Lake trout	11	3.76

a. Data provided by Randy Eshenroder, Great Lakes Fisheries Commission, 1995.
b. Calculated from annual Lake Michigan catch and percent of Lake Michigan catch from Green Bay.
c. Carp catch average includes both the 1978-1983 period when fishing was active and the 1984-1990 period when carp fishing was closed on Green Bay. Total Lake Michigan carp catch after 1984 was typically less than 1,000 pounds per year.

Table 2-2
1998[a] Commercial Harvest of Selected Species from the Wisconsin Waters of Lake Michigan and Green Bay

Species	1998 Catch (1000s of pounds)
Chubs	1,891
Lake whitefish	1,557
Rainbow smelt	272 (125 from Green Bay)
Yellow perch	204[b,c]

a. 1998 quota year, from July 1, 1997, to June 30, 1998.
b. From Green Bay only. Harvest was closed on Lake Michigan.
c. Reported value for the 1998 calendar year.

Source: Wisconsin Department of Natural Resources (1999).

2.3.2 Sport Fishery

The Lower Fox River/Green Bay sport fishery is a very important economic resource to the region (Stratus Consulting Inc., 1999e). In 1963, the Wisconsin Department of Natural Resources introduced 9,000 rainbow trout into several Door County tributaries to manage the alewife population and to provide a sport fishery (Eggold, 1995). Because of the success of this initial stocking, the program was expanded to include other salmonids: brown trout, brook trout, lake trout, chinook salmon, and coho salmon (Hansen et al., 1990; Eggold, 1995). Since stocking began, the sport fishery has become an important industry (Colborn et al., 1990). The Wisconsin Department of Natural Resources (1999) estimates that anglers spent nearly 3 million hours fishing on Lake Michigan and Green Bay in 1998. A walleye fish stocking program below the DePere Dam from 1977 through 1984 attracted sport fishers to the region, and today this area is an established, regionally famous walleye fishing area (Wisconsin Department of Natural Resources, 1988).

Additional details on the economic importance of the Green Bay recreational fishery is presented in the Trustees' report on damages associated with PCB fish consumption advisories (Stratus Consulting Inc., 1999e).

2.4 IMPORTANCE OF THE FISHERY TO THE ONEIDA AND MENOMINEE TRIBES

The Fox River/Green Bay fishery is vital to the Oneida Tribe and the Menominee Tribe because of direct and indirect relationships to the Oneida and Menominee lifestyles. The fishery resource is an integral part of the culture of both tribes, and has served as a vital food source for tribal members.

The Oneida Tribe begins the fishing season with a ceremony giving thanks to the Creator for the annual fish runs. The ceremony is followed by a social dance to celebrate the return of the fish. The ceremony and dance continue to be celebrated on the reservation even though the fish are not considered safe to eat because of PCB contamination. Historically, the annual fish migrations were a community event. Family, clans, and neighborhood groups would camp at their traditional locations along reservation waters for days at a time. These annual gatherings that revolved around the fish migrations were important community events and helped to refine the traditional Oneida culture and to teach the Oneida children a way to sustain and provide for themselves. Similarly, the lake sturgeon has played an important role in the Menominee tribal culture. The tribe celebrates the return of the sturgeon every spring with a ceremony and community feasts, and lake sturgeon play an important role in the creation stories of the Menominee Tribe.

The fish supply historically was a major source of protein for many tribal members. Fish would be dried, canned, salted, or smoked for use throughout the year. One of the local tribal delicacies was fish head soup, a seasonal dish prepared only during the spawning runs. Many different fish species were utilized; lake sturgeon were historically a significant food source for the Menominee

Tribe. The annual fish migrations were also a way to supplement the income. Tribal members would take a portion of their catch to Green Bay or Appleton to sell, providing a source of income for the family.

The importance of the fishery resources to the tribes is indicated by the changes in tribal culture that have resulted from the PCB contamination. For example, the issuance of fish consumption advisories in the 1970s reduced the consumption of fish from the Oneida reservation, removing fish as a staple of the Oneida diet. With the decline in fish consumption came a loss in the ability to share fish catches with other members of the tribal community, one of the most important lessons taught to the children at the annual gatherings. Some tribal members believe that this loss of sharing has contributed to the disconnect between the elders and the younger tribal members today.

2.5 CONCLUSIONS

The Green Bay fishery is a vital resource that provides important ecological, economic, and cultural services. As part of the Great Lakes, Green Bay is part of the largest area of fresh surface water on earth and supports a diverse and significant fishery. Despite degradation brought on by various human impacts, the fishery remains a vital resource used by commercial, sport, and tribal fishermen. Fish from the Lower Fox River and Green Bay, supported by the diverse habitats provided by the bay, comprise an important food source for piscivorous birds and mammals. The importance of the fishery resources of the bay is reflected in the extensive efforts expended by various government agencies to manage and protect the fishery resource of Green Bay.

CHAPTER 3
PCB PATHWAY DETERMINATION AND EXPOSURE FOR FISHERY RESOURCES

This chapter discusses the pathways by which the fishery resources of the Lower Fox River/Green Bay ecosystem have come to be exposed to PCBs released from Lower Fox River paper companies. The chapter includes a PCB pathway determination (Section 3.1) and a description of PCB exposure and accumulation in assessment area fish (Section 3.2).

3.1 PCB PATHWAY DETERMINATION

Pathway determination is one of the injury determination steps in the Department's regulations for conducting NRDAs at 43 CFR Part 11. A pathway is defined in the regulations as "the route or medium through which oil or a hazardous substance is or was transported from the source of the discharge or release to the injured resource" [43 CFR §11.14(dd)]. The pathway is determined by

> either demonstrating the presence of the oil or hazardous substance in sufficient concentrations in the pathway resource or by using a model that demonstrates that the conditions existed in the route and in the oil or hazardous substance such that the route served as the pathway [43 CFR §11.63(a)(2)].

The Trustees have published a PCB pathway determination report for the Lower Fox River/Green Bay environment (Stratus Consulting Inc., 1999d). The Trustees based the pathway determination on evaluations of the following:

- PCB releases into the Lower Fox River from paper company facilities
- PCB pathway models used in the Green Bay Mass Balance Study (GBMBS), a multi-million dollar, multi-agency effort to understand and model the transport, fate, and bioaccumulation of PCBs in the Lower Fox River and Green Bay
- PCB transport processes in the Lower Fox River and Green Bay, including downstream transport in the Lower Fox River, water circulation patterns in Green Bay, and sediment transport and deposition in the bay
- the spatial and temporal distributions of PCBs in Green Bay surface water, sediment, and biota in relation to releases into the bay from the Lower Fox River

- PCB congener patterns in Green Bay sediments compared with congener patterns in the Lower Fox River and Lake Michigan.

The PCB pathway determination uses both measurements of PCBs in pathway media and the results of the GBMBS models to determine PCB pathways in the system. The conclusions of the PCB pathway determination are as follows (Stratus Consulting Inc., 1999d):

1. Paper manufacturing and processing facilities released large quantities of PCBs into the Lower Fox River. An estimated 300,000 kg of PCBs have been released into the Lower Fox River from paper company facilities. These releases are the primary PCB source into the river and Green Bay.

2. The Fox River is the dominant source of PCBs to Green Bay. For example, mass balance models show that in 1989-1990, the Fox River contributed 92% of the PCBs that entered the bay from all tributary and atmospheric sources.

3. Surface water is the primary pathway by which PCBs are transported in the Lower Fox River/Green Bay system.

4. Fox River PCBs are transported throughout Green Bay.

5. Surface water, sediment, plankton, and forage fish serve as PCB pathways for different fish species of the Lower Fox River and Green Bay.

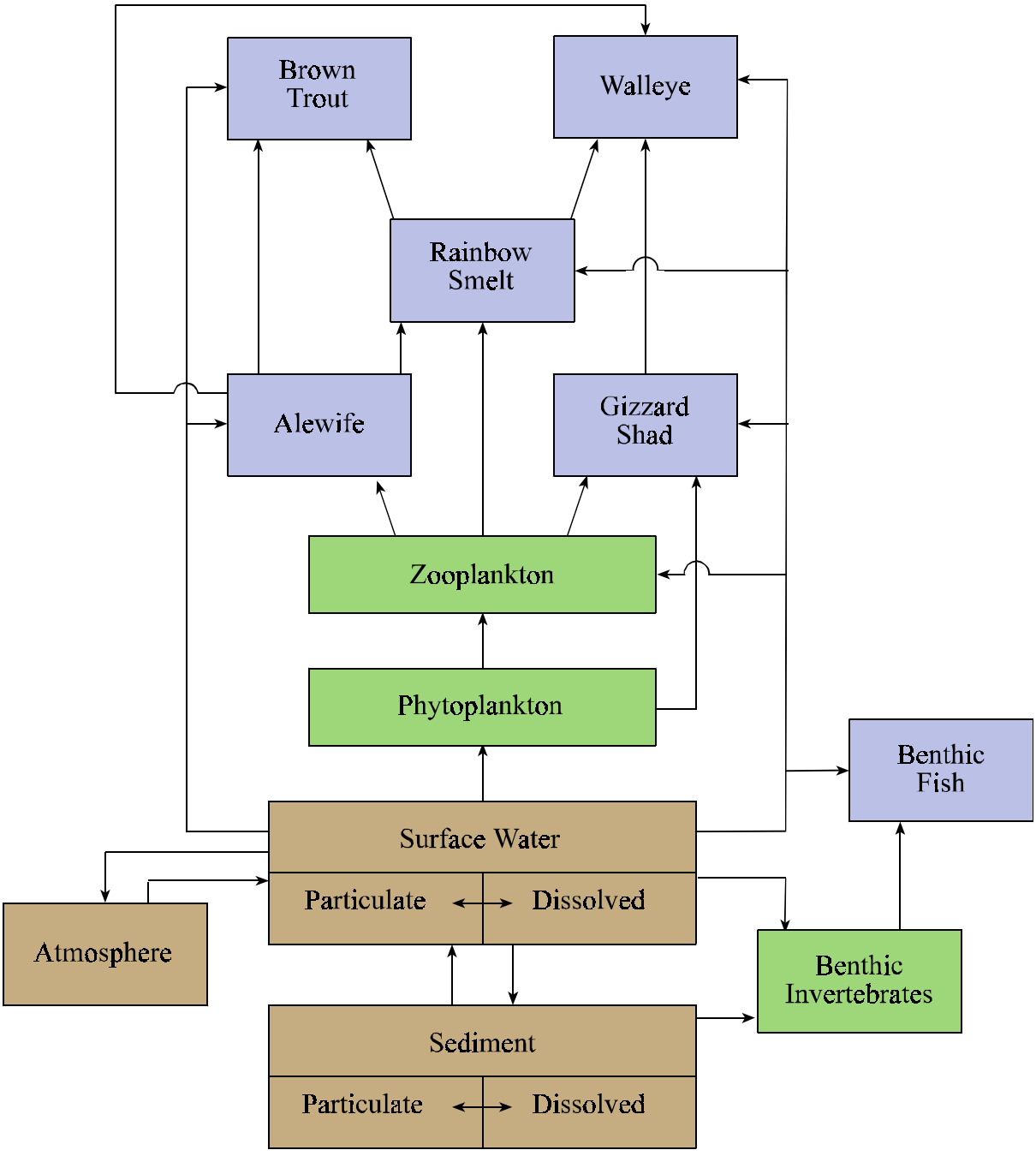
6. PCB concentrations in Green Bay have declined since the 1970s, but remain high because of the environmental persistence and continued environmental release of PCBs.

7. PCBs are transported from Green Bay into Lake Michigan.

Figure 3-1, which is taken from the Trustees' pathway report, presents a PCB exposure pathway diagram for selected fish species in Green Bay. The pathway diagram shows that PCBs enter the aquatic food chain from contaminated surface water and sediment. Fish are exposed to PCBs through the food chain and through direct uptake from surface water and sediment. Elevated concentrations of PCBs have been documented in the pathway resources shown in Figure 3-1, including surface water, sediment, plankton, and forage fish (Connolly et al., 1992; Stratus Consulting Inc., 1999d).

Another PCB pathway in the system is fish migration. Several fish species, including northern pike, walleye, smallmouth bass, yellow perch, and lake sturgeon, have been documented to migrate between the waters of Green Bay and tributaries to Green Bay (Cogswell, 1998; Cogswell and Bougie, 1998; Green Bay Fisheries Resources Office, 1998). Fish migration has also been documented between Green Bay and Lake Michigan (Wisconsin Department of Natural Resources, 1996) and within Green Bay itself (U. S. Fish and Wildlife Service, 1998).

Figure 3-1
GBMBS PCB Pathways in Lower Fox River/Green Bay System
(abiotic media are in brown, primary producers and invertebrates are in green, and fish species are in blue)



Source: Stratus Consulting Inc., 1999d.

This migration of contaminated biota may serve as a particularly important transport pathway for natural resources on the reservation of the Oneida Tribe. The Oneida reservation is located immediately west and southwest of the city of Green Bay, near the southern end of Green Bay. The reservation is connected to the waters of the Lower Fox River and Green Bay through several creeks that run through the reservation. These creeks include Duck Creek, which flows through the reservation and enters Green Bay just northwest of the Fox River mouth, and Dutchman's Creek, which flows through the reservation and enters the Fox River a few miles upstream of its mouth.

Several studies have shown that fish migrate from Green Bay up Duck Creek and into the reservation, thereby transporting PCBs to the reservation. In a study conducted by the Service between 1995 and 1998, fish marked with floy tags in Green Bay were found in Duck Creek within the reservation boundaries, and fish marked in Duck Creek were found in Green Bay (Cogswell, 1998; Cogswell and Bougie, 1998). For example, 46% of the northern pike tagged in Duck Creek were recaptured in Green Bay. The species documented to migrate between Duck Creek and Green Bay include northern pike, walleye, smallmouth bass, and yellow perch.

PCB concentrations measured in walleye, white sucker, and northern pike collected in 1998 confirm that fish from the reservation have elevated concentrations of PCBs (Battelle, 1999). Average total PCB concentrations for fish from Duck Creek were 1199 ng/g (wet weight) in walleye fillets and 535 ng/g in northern pike fillets. White suckers from Lancaster Brook, a tributary to Duck Creek on the reservation, had an average whole-body total PCB concentration of 1064 ng/g, and suckers from Dutchman Creek had an average whole-body total PCB concentration of 325 ng/g. In addition, a mink captured in March 1999 along Silver Creek, another tributary to Duck Creek, had a liver total PCB concentration of 40.42 μg/g (wet weight), a concentration indicative of elevated PCB exposure (Eisler, 1986). Since fish typically comprise a large portion of mink diet (U.S. EPA, 1993b), the mink exposure to PCBs probably results from consumption of contaminated fish. Therefore, migration data document that fish move between Duck Creek and Green Bay, and PCB concentration data in fish and mink indicate that the fish migration from Green Bay up Duck Creek serves as a transport pathway for PCBs.

In conclusion, PCB pathways to fish were determined using both measures of PCBs in pathway media and the results of transport models. PCB releases from Lower Fox River paper company facilities are the primary source of PCBs to the Lower Fox River and Green Bay. PCBs released from the facilities are transported throughout the river and bay, where fish are exposed to the PCBs through the food chain and through direct contact with surface water and sediment.

3.2 PCB EXPOSURE AND ACCUMULATION IN FISH

Many fish species throughout the Lower Fox River and Green Bay are exposed to and accumulate PCBs released from Fox River paper company facilities. Table 3-1 lists the fish species of the Lower Fox River and Green Bay in which PCBs have been found. PCB

Table 3-1
Lower Fox River/Green Bay Fish Species in Which PCBs Have Been Measured

Species	Measured in Samples from Lower Fox River	Measured in Samples from Green Bay
Alewife	X	X
Black bullhead	X	X
Black crappie	X	X
Bloater chub		X
Bluegill	X	
Bowfin	X	
Brook trout		X
Brown bullhead	X	X
Brown trout		X
Burbot		X
Carp	X	X
Channel catfish	X	X
Chinook salmon	X	X
Cisco/lake herring		X
Coho salmon		X
Common shiner	X	
Emerald shiner	X	
Flathead catfish	X	
Freshwater drum	X	
Gizzard shad	X	X
Golden shiner	X	
Greater redhorse	X	
Green sunfish	X	
Lake sturgeon		X
Lake trout		X
Lake whitefish		X
Largemouth bass		X
Longnose sucker		X
Northern pike	X	X
Pumpkinseed		X
Rainbow smelt		X
Rainbow trout		X
Redhorse sucker	X	

Table 3-1 (cont.)
Lower Fox River/Green Bay Fish Species in Which PCBs Have Been Measured

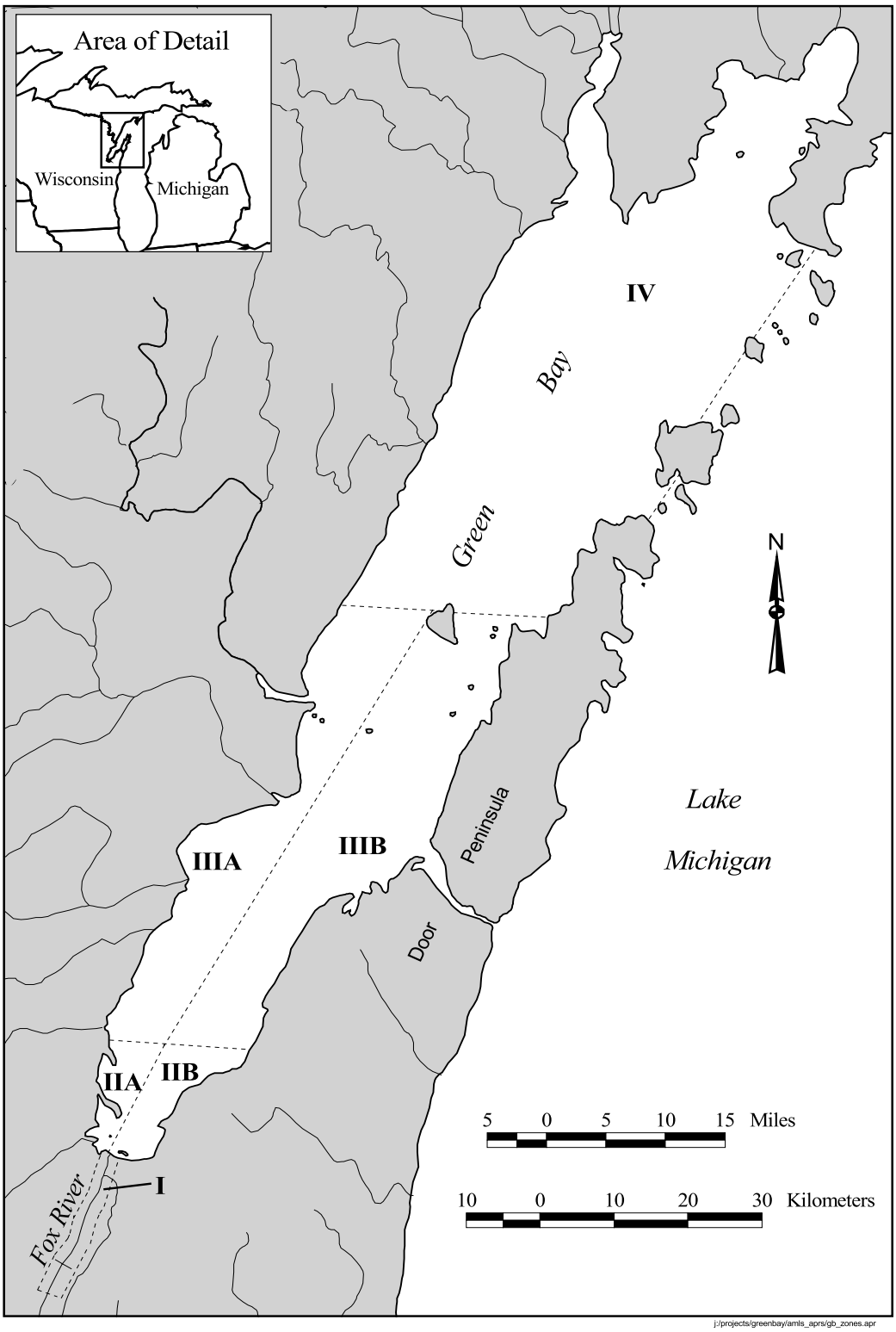
Species	Measured in Samples from Lower Fox River	Measured in Samples from Green Bay
Rock bass	X	
Sauger	X	
Sheepshead/drum	X	
Shorthead redhorse	X	
Smallmouth bass	X	X
Splake		X
Spot-tailed shiner		X
Troutperch		X
Walleye	X	X
White bass	X	X
White perch	X	X
White sucker	X	X
Yellow perch	X	X

Sources: Stratus Consulting (1998); Wisconsin Department of Natural Resources sponsored database at http://www.ecochem.net/FoxRiverDatabaseWeb/default.asp, downloaded July 1999.

accumulation has been documented in all levels of the aquatic food web, such as forage fish (alewives, rainbow smelt), predators (walleye, brown trout, northern pike), and bottom feeders (carp, white sucker), and in fish from a variety of habitats, including coastal wetlands, coastal beaches, near-shore areas, and open water habitat (Connolly et al., 1992; Brazner and DeVita, 1998).

The PCB accumulation data for Fox River/Green Bay fish show several important features. First, fish PCB concentrations are not uniform throughout the river and bay, but vary spatially. For example, as part of the GBMBS, samples of six fish species (alewife, brown trout, carp, gizzard shad, rainbow smelt, and walleye) were collected from numerous locations in the Lower Fox River and Green Bay in 1989-1990 and analyzed for PCB congeners in whole-body samples (Connolly et al., 1992). Sampling locations were grouped into six different zones, shown in Figure 3-2. Zone I is the Lower Fox River downstream of DePere Dam; Zones IIA and IIB are the western and eastern halves, respectively, of the innermost portion of the bay; Zones IIIA and IIIB are the western and eastern halves, respectively, of the rest of the inner bay; and Zone IV is the outer bay (beyond Chambers Island).

Figure 3-2
Fish Sampling Zones Used in Green Bay Mass Balance Study



As an example of the spatial trend of PCBs in Lower Fox River/Green Bay fish, Figure 3-3 plots the total PCB concentrations measured in walleye from the six different zones. Figure 3-3 shows that PCB concentrations are highest in the Lower Fox River and decline with increasing distance into the bay. More detailed analysis of data also shows that PCB concentrations within zones 2 and 3 along the eastern shore of the inner bay (zones 2B and 3B) are generally higher than those along the western shore (zones 2A and 3A) (Stratus Consulting Inc., 1999d). Data collected by the Service in 1996 on walleye and brown trout PCB concentrations show the same spatial pattern (Stratus Consulting Inc., 1999d). This spatial pattern matches the spatial pattern of PCB concentrations in surface water and sediment of the Lower Fox River/Green Bay, indicating the PCB pathway link between surface water/sediment and fish (Stratus Consulting Inc., 1999d).

A second feature of PCB accumulation in assessment area fish is that PCB concentrations in predatory fish, such as walleye and brown trout, tend to be higher than concentrations in the forage fish on which they feed, such as alewives and rainbow smelt. Figure 3-4 compares PCB concentrations in Green Bay walleye and brown trout with those in alewives and rainbow smelt. Since PCBs tend to accumulate in fish lipid, the data are expressed as lipid-normalized PCB concentrations (i.e., wet weight PCB concentration divided by fraction lipid in the sample) to account for any variability in wet weight PCB concentrations between the species that is caused by differences in lipid content alone. The figure shows that within each of the zones, concentrations are consistently higher in walleye and brown trout, indicating biomagnification of PCBs up the food chain. Therefore, top-level predatory fish tend to be the species that are the most highly exposed to PCBs.

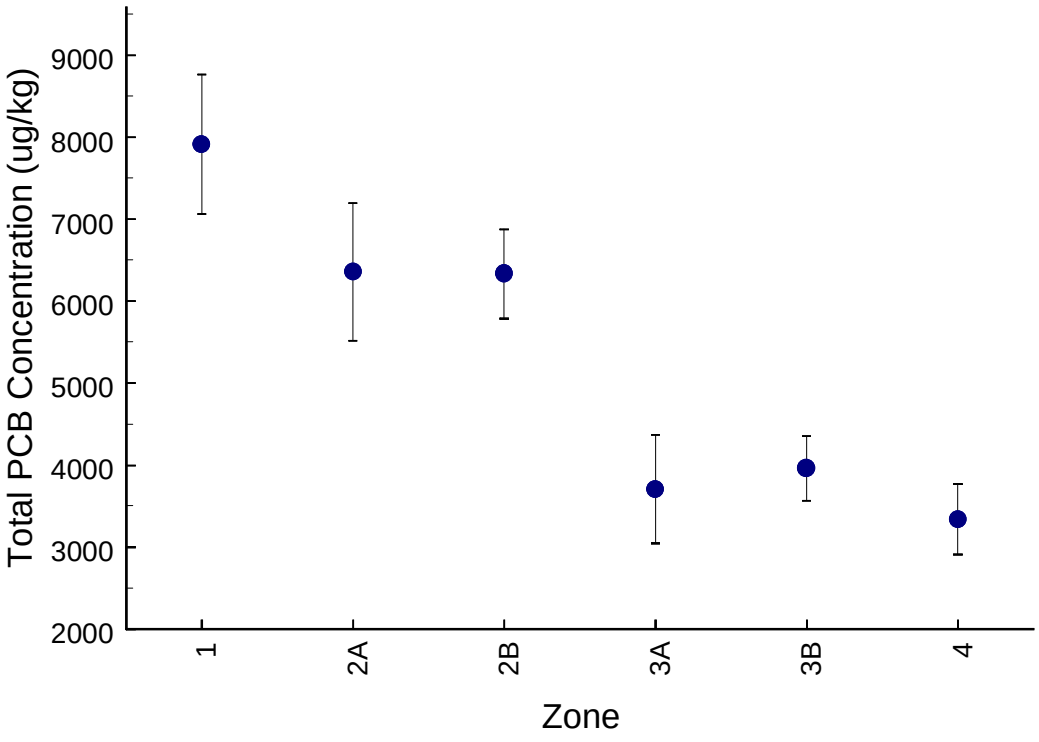
The assessment area data also provide information on the changes in fish PCB accumulation over time. A detailed analysis of temporal trends of PCB concentrations in Green Bay fish is presented in the Trustees' report on PCB pathways (Stratus Consulting Inc., 1999d). The following conclusions were reached:

- In general, PCB concentrations in Green Bay have declined since the 1970s, coinciding with decreases in PCB releases from Lower Fox River paper companies.

- However, the persistent nature of PCBs in the environment and the widespread contamination of the sediment in the river and bay mean that fishery resources will continue to be exposed to PCBs for many years.

- PCB concentrations show a stronger and more consistent decline in forage fish (e.g., yellow perch and perhaps alewife) than in predator fish (walleye and brown trout). Possible explanations for this difference include shifts in walleye and brown trout diet over time, and increased "lag time" for the reductions in PCBs to be detectable in the longer lived predatory species.

Figure 3-3
Total PCB Concentrations in Green Bay Walleye, 1989
(bars equal plus or minus one standard error about the mean;
zone locations are shown in Figure 3-2)

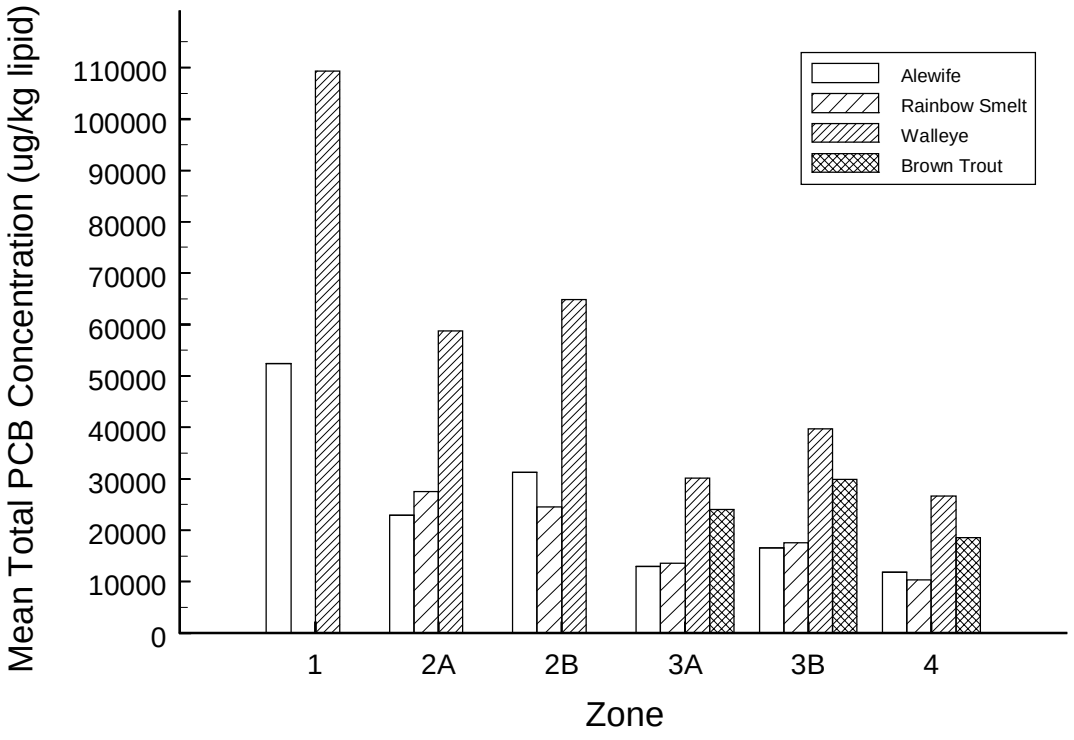


Source: Green Bay Mass Balance Study data from the Wisconsin Department of Natural Resources sponsored database at http://www.ecochem.net/FoxRiverDatabaseWeb/default.asp, downloaded July 1999.

- PCB concentration declines are more prominent in Zone 2 than in zones 3 and 4. A possible explanation for this trend is that the signal of decreased PCB loadings from the Lower Fox River may take longer to reach the portions of the bay that are farther from the river.

- PCB concentrations in fish beyond the innermost portion of the bay do not show a decline between 1989 and 1996.

Figure 3-4
Mean PCB Concentrations in Lower Fox River/Green Bay Forage Fish (alewife and rainbow smelt) and Predatory Fish (walleye and brown trout), by Zone
(zone locations are shown in Figure 3-2)



Source: Green Bay Mass Balance Study data from the Wisconsin Department of Natural Resources sponsored database at http://www.ecochem.net/FoxRiverDatabaseWeb/default.asp, downloaded July 1999**.**

- PCB transport pathways continue in the Lower Fox River/Green Bay system, and Green Bay fish continue to be exposed to PCBs.

In conclusion, the transport of PCBs in surface water and sediments of the Lower Fox River/Green Bay system results in PCB exposure of fish throughout the river and bay. The accumulation of PCBs tends to be highest in predatory species and in areas of the river and bay with the highest sediment and surface water PCB concentrations. Although some PCB concentrations in some fish species from some areas have declined since the 1970s, the PCB transport and exposure pathways continue to result in fish exposure to PCBs.

CHAPTER 4
INJURY ASSESSMENT APPROACH

This chapter describes the regulatory injury definitions used to assess injuries to fishery resources and outlines the overall approach of the injury assessment. A brief description of the types of adverse effects caused by PCBs is also included to provide background for the injury assessment approach used.

4.1 OVERVIEW OF ADVERSE CHANGES IN FISH VIABILITY CAUSED BY PCBS

Exposure to PCBs can cause many different types of adverse effects on fish, including death, cancer, deformities, impairments of the immune system and of the endocrine system, and biochemical changes (Eisler, 1986; Safe, 1994). This section presents a brief overview of the documented adverse effects of PCBs on fish viability. This information provides the background for understanding the Trustees' approach for assessing injuries resulting from adverse changes in viability due to PCB exposure.

Death

Early life stages in fish are more sensitive to mortality effects of PCBs than are adult fish. PCBs cause reduced egg hatchability and fry mortality at concentrations orders of magnitude less than concentrations causing adult mortality (Nebeker et al., 1974; Eisler, 1986). In fish eggs, PCBs accumulate in the lipid-rich yolk to concentrations that are typically much greater than those in the surrounding water (Broyles and Noveck, 1979) and can be greater than those in the maternal fish (Niimi, 1983). Many of the PCB toxic effects to fish embryos occur during absorption of the yolk sac by developing fry (Walker et al., 1994). PCBs have been implicated as causative factors in embryo mortality in Great Lakes fish, leading to reduced reproduction (Mac, 1988; Mac and Schwartz, 1992; Mac et al., 1993).

Cancer

Although PCBs do not initiate the formation of cancerous tumors, it is well documented that they promote or enhance formation of tumors initiated by other factors (Hendricks et al., 1990). For example, rainbow trout that were exposed to PCBs showed a significantly higher incidence of liver tumors induced by aflatoxin B_1 (a tumor initiator) than trout exposed to aflatoxin B_1 alone (Hendricks et al., 1981). The liver is generally the predominant site for fish tumors or pre-tumors that are initiated or promoted by contaminants (Baumann, 1992a; Baumann, 1992b). Higher incidences of liver tumors in fish exposed to contaminants, including PCBs, have been

demonstrated in both field and laboratory studies (Teh et al., 1997a). The results of studies on the tumor promotion of PCBs in fish are consistent with the more extensive body of scientific literature on tumor promotion in mammals (see Silberhorn et al., 1990 for a review).

Deformities

PCB congeners that are structurally similar to 2,3,7,8-tetrachloro-p-dibenzo-dioxin (TCDD) have been shown to cause deformities in developing fish fry (Walker and Peterson, 1992). These deformities, like embryo mortality, occur primarily during the yolk sac absorption stage. They include internal whole organ and soft tissue malformations such as edema of the yolk sac (Walker et al., 1991) and hemorrhaging in various organs (Spitsbergen et al., 1991; Walker and Peterson, 1992), skeletal deformities such as domed skulls and craniofacial deformities (Walker et al., 1994), and overt external malformations such as opercular defects (Helder, 1980; Helder, 1981).

The development of these deformities often precedes embryo mortality during the yolk sac absorption stage (Walker et al., 1994). The occurrence of these deformities has been documented in a variety of fish species, including lake trout, rainbow trout, northern pike, brook trout, Japanese medaka, mummichog, and zebrafish (Walker et al., 1994).

Immune System Impairment

PCBs can also impair the functioning of the immune system in fish. Adverse effects on fish immune systems exposed to PCBs include significantly reduced antibody levels (Thuvander and Carlstein, 1991), reduced immune cell activity (Jones et al., 1979; Arkoosh et al., 1994; Rice and Schlenk, 1995; Rice et al., 1996), and reduced resistance to introduced bacteria (Jones et al., 1979). This suppression of the immune system can result in increased susceptibility of fish to disease, parasitism, and cancer (Khan and Thulin, 1991; Zelikoff, 1994; Anderson and Zeeman, 1995).

Endocrine System Impairment

The disrupting effects of PCBs on animal endocrine systems has been extensively studied and documented in mammals (Safe, 1994). Effects on estrogen production or activity are the most studied endocrine system effects caused by chemicals such as PCBs (Gillesby and Zacharewski, 1998). PCB congeners (or their metabolites) can be estrogen mimics, promoters, or inhibitors, or they can alter the levels of or compete with thyroid hormones in the blood (Hansen, 1994; Gillesby and Zacharewski, 1998). In fish, PCB modulation of estrogen responsiveness can result in inhibition or induction of egg yolk synthesis (Anderson et al., 1998). Field studies on fish have used increased vitellogenin, a protein involved in egg formation, in male carp (Folmar et al., 1996) and rainbow trout (Harries et al., 1996) as indications of exposure to environmental contaminants that disrupt endocrine systems.

These alterations of the endocrine system can affect fish reproduction by affecting sex determination and sex ratios (Matta et al., 1998), delaying maturity (Munkittrick et al., 1997), decreasing fertility and egg production in females (Arcand-Hoy and Benson, 1998), causing gonadal abnormalities (Matta et al., 1998), or reducing testicular growth in males (Jobling et al., 1996). In addition, potentiation of estrogen responsiveness may also enhance tumorigenesis in fish (Teh and Hinton, 1998).

Biochemical Changes

PCB exposure can cause measurable biochemical changes in the liver and other organs of fish. Induction of the enzyme cytochrome P4501A (CYP1A), a protein involved in the metabolism of planar aromatic hydrocarbons, is a hallmark of exposure to PCBs (Safe, 1990; Stegeman and Hahn, 1994). Numerous field studies have documented changes in the level of induction of this enzyme in fish in response to contaminant exposure (Van Der Oost et al., 1991; Sleiderink et al., 1995; Eggens et al., 1996; Schrank et al., 1997). Induction or suppression of the cytochrome P450 activity in fish can have deleterious consequences to fish health. For example, induction of this enzyme system is linked to tumorigenesis via metabolic activation (Stegeman and Hahn, 1994).

4.2 INJURY DEFINITIONS AND MEASURES

4.2.1 Injury Definitions

Injuries to fishery resources were assessed according to the definitions of injury in the Department's NRDA regulations. Specifically, the injury definitions state that fishery resources have been injured as a result of the release of a hazardous substance if the concentration of the substance is sufficient to

- exceed action or tolerance levels established under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342, in edible portions of organisms [43 CFR § 11.62(f)(1)(ii)]; or

- exceed levels for which an appropriate State health agency has issued directives to limit or ban consumption of such organism [43 CFR § 11.62(f)(1)(iii)]; or

- cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction), or physical deformations [43 CFR § 11.62(f)(1)(i)].

4.2.2 Injury Measures

For the injury definition related to exceedences of action or tolerance levels in edible portions, PCB concentrations measured in fish fillets were compared to the PCB tolerance level established by the FDA under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342. For the injury definition of exceedence of levels for which an appropriate State health agency has issued directives to limit or ban consumption, the injury measure is documentation of State fish consumption advisories because of PCB contamination.

The injury definition related to adverse changes in viability can be demonstrated by measuring a biological response that meets the following criteria [43 CFR § 11.62 (f)(2)(i-iv)]:

- The biological response is often the result of exposure to . . . hazardous substances.
- Exposure to . . . hazardous substances is known to cause this biological response in free-ranging organisms.
- Exposure to . . . hazardous substances is known to cause this biological response in controlled experiments.
- The biological response measurement is practical to perform and produces scientifically valid results.

Several biological responses and measures have already been determined in the Department's regulations to meet these four criteria. The following biological responses and measurements are identified in the Department's regulations as meeting the four criteria and were used in the Trustees' injury assessment:

1. Death (mortality) injuries were evaluated through the use of laboratory toxicity testing [43 CFR § 11.62(f)(4)(i)(A)].

2. Cancer injuries were evaluated through measurement of fish neoplasia [43 CFR § 11.62(f)(4)(iv)(E)].

3. Physiological malfunction injuries were evaluated by assessing impacts to fish reproduction [43 CFR § 11.62(f)(4)(v)(E)].

4. Physical deformation injuries were evaluated through examination of:

    - overt external malformations [43 CFR § 11.62(f)(4)(vi)(A)]
    - skeletal deformities [43 CFR § 11.62(f)(4)(vi)(B)]
    - internal whole organ and soft tissue malformations [43 CFR § 11.62(f)(4)(vi)(C)]
    - histopathological lesions [43 CFR § 11.62(f)(4)(vi)(D)].

In addition, three biological responses that meet the four criteria but are not specifically listed in the Department's regulations for conducting NRDA were also used by the Trustees in the fish injury assessment: immune system impairment, endocrine system impairment, and biochemical changes. Documentation that these three biological responses meet the four acceptance criteria for injury determination at 43 CFR § 11.62 (f)(2)(i-iv) is provided below.

Criterion 1: The biological response is often the result of exposure to the hazardous substance.

Although all three of the biological responses, immune system impairment, endocrine system impairment, and biochemical changes, can be caused by environmental factors other than PCBs, their occurrence as a direct result of PCB exposure has been well established in both field and controlled studies, as described below. The injury assessment approach, described in the following section, used measurements of the biological responses in control areas to account for any baseline incidence of the responses potentially caused by other factors.

Criterion 2: Exposure to the hazardous substance causes the biological response in free-ranging organisms.

The occurrence of immune system impairment in fish exposed to PCBs in the field has been well documented (Zelikoff, 1994; Anderson and Zeeman, 1995; Zelikoff et al., 1996). For endocrine system impairment, field studies have documented an increase in vitellogenin in male carp (Folmar et al., 1996) and rainbow trout (Harries et al., 1996) as indications of exposure to environmental contaminants that disrupt estrogen production or activity. For biochemical changes, numerous field studies have documented changes in the level of induction of the P4501A enzyme in response to organic contaminant exposure, including PCBs (Van Der Oost et al., 1991; Sleiderink et al., 1995; Eggens et al., 1996; Schrank et al., 1997).

Criterion 3: Exposure to the hazardous substance causes the biological response in controlled experiments.

Controlled studies have documented numerous adverse effects on the immune system of fish as a result of PCB exposure, including reduced antibody levels (Thuvander and Carlstein, 1991), reduced immune cell activity (Jones et al., 1979; Arkoosh et al., 1994; Rice and Schlenk, 1995; Rice et al., 1996), and reduced resistance to introduced bacteria (Jones et al., 1979). Effects on estrogen production or activity as a manifestation of adverse endocrine system effects resulting from PCB exposure has been well documented (Gillesby and Zacharewski, 1998). Numerous controlled studies have established that the biochemical change of P4501A enzyme induction is a hallmark of PCB exposure (Safe, 1990; Stegeman and Hahn, 1994).

Criterion 4: The biological response measurements is practical to perform and produces scientifically valid results.

The references cited under the first three criteria document both the practicality and the scientific validity of the three responses.

4.3 INJURY ASSESSMENT APPROACH

The fish injury assessment was conducted in accordance with the Assessment Plan [61 FR 43558] and addenda [62 FR 33804; 63 FR 25144]. The injury assessment approach described here is for the assessment of injuries to the fishery resources resulting from exposure to PCBs released from Lower Fox River paper companies. However, the fish injury assessment is closely related to the injury assessment for the surface water/sediment resource of the assessment area. The surface water/sediment resource provides habitat services to fish and is a critical pathway component for PCB exposure to fish, and therefore the surface water/sediment resource can be injured if the habitat service it provides to fish is adversely affected [43 CFR § 11.62(b)(1)(v)]. Injuries to surface water/sediment resources in providing habitat for fish are assessed in a separate Trustee report (Stratus Consulting Inc., 1999c).

4.3.1 Fish Consumption Advisories and FDA Tolerance Level Exceedences

For injuries to fish based on the presence of fish consumption advisories, the Trustee conducted a thorough evaluation of the procedures, methods, and underlying data used by the appropriate agencies of Wisconsin and Michigan in establishing the advisories. The evaluation, which is presented in Chapter 5 of this report, included consideration of:

- fish sampling and analytical chemistry methods
- procedures for proposing, reviewing, and issuing fish consumption advisories within each state
- the history of the fish consumption advisory program in each state
- PCB concentrations used as threshold criteria for establishing advisories and a comparison of measured PCB concentrations to the criteria
- types of advisories issued
- contaminants responsible for the advisories.

The evaluation of fish consumption advisory injuries encompassed the Lower Fox River, the Wisconsin and Michigan waters of Green Bay, and the Wisconsin and Michigan waters of northern Lake Michigan. All fish species were included in the evaluation and injury assessment.

For evaluating injuries related to exceedences of the FDA tolerance level for PCBs in fish tissue, the Service compared available fish tissue PCB concentration data from the Lower Fox River and Green Bay with the FDA tolerance level.

The results of the injury assessment of fish consumption advisories and FDA tolerance level exceedences were published in a report issued to the public in 1998 (Stratus Consulting Inc., 1998). Chapter 5 of this report summarizes the results of that report, and includes an update of fish consumption advisory injuries through 1999.

4.3.2 Overview of Assessment of Adverse Changes to Fish Viability

This section briefly describes the overall approach and process used to assess adverse changes to viability injuries. Two general types of adverse changes to fish viability were assessed: adverse effects on fish health, and adverse effects on fish reproduction. Table 4-1 summarizes the specific injury categories, fish species, injury endpoints, and primary data sources used in the assessment.

The overall approach was based on supplementing available site-specific and literature information with data not previously available. Adverse effects on fish health first involved a preliminary study of walleye, brown trout, and lake trout. In 1996 the Service conducted a small-scale, reconnaissance survey of possible adverse health effects on these species. Walleye were collected from the Lower Fox River downstream of DePere dam and from Green Bay, brown trout were collected from Green Bay, and lake trout were collected from Lake Michigan along the Door Peninsula, near the mouth of Green Bay. Individual fish were tested for various fish health endpoints, as listed in Table 4-1. Details of the 1996 study methods and results are contained in Hagler Bailly Services Inc. (1997a), Teh et al. (1997b), and Stratus Consulting (1999a). The results of the 1996 fish health reconnaissance study indicated that adverse health effects may be occurring to walleye in the assessment area and that additional, more detailed work was warranted. The 1996 data did not indicate adverse effects on the health of brown trout and lake trout, so no further work was conducted on health injuries for these two species.

Therefore, in 1997 the Service conducted a more comprehensive study of health effects on Lower Fox River/Green Bay walleye. The 1997 work included a much larger sample size than the 1996 work, samples from different control areas for comparison, and a broader range of health effect measurements (see Table 4-1).

Table 4-1
Overview of Injury Assessment of Adverse Changes in Fish Viability

General Type of Adverse Effect	Injury Category [43 CFR §11.62(f)(4)]	Species Assessed	Injury Endpoint	Primary Data Sources
Fish health	Cancer	Walleye Brown trout Lake trout	Liver tumors and pre-tumors in adult fish	1996-1997 Service NRDA study
	Disease	Walleye	Incidence of diseases in adult fish	1996-1997 Service NRDA study
	Physiological malfunction	Walleye	Immunosuppression, endocrine disruption, liver biochemistry in adult fish	1996-1997 Service NRDA study
		Brown trout Lake trout	Immunosuppression, liver biochemistry in adult fish	1996-1997 Service NRDA study
	Physical deformation	Walleye Brown trout Lake trout	Lesions in various organs of adult fish	1996-1997 Service NRDA study
Fish reproduction	Death	Lake trout	Embryomortality	1996-1998 USGS study; historical data
	Physical deformation	Lake trout	Deformities in embryos	1996-1998 USGS study; historical data

The results of the 1996-1997 walleye health investigations are presented in a report issued to the public by the Service in 1999 (Stratus Consulting Inc., 1999a). Chapter 6 of this report presents an injury determination based on the results given in that report, and includes an update of measurements of PCB concentrations in liver tissues.

In addition to the fish health studies, adverse changes in reproduction were assessed for lake trout. Among the fish species studied to date, lake trout are the most sensitive to early life stage mortality induced by PCBs and other similar organochlorine compounds (details provided in Chapter 7). The assessment of embryo mortality and embryo deformity injuries to lake trout was based on historical data and other information from the scientific literature, studies of Lake

Michigan lake trout reproduction conducted by the USGS from 1996-1998, and supplemental NRDA laboratory toxicity studies conducted by the USGS in 1996-1998. The injury assessment to lake trout is provided in Chapter 7 of this report.

The Service also considered assessing reproductive injuries to lake sturgeon, a federally threatened species. In 1996 the Service attempted to collect spawning lake sturgeon from the Lower Fox River downstream of DePere Dam, but efforts were not successful. No additional injury assessment work was conducted on lake sturgeon because of these sampling difficulties.

The species selected for assessment of adverse viability injuries, walleye, brown trout, and lake trout, are top-level predatory species and are therefore expected to be among the more highly exposed species to PCBs in the system. Lake trout are the most sensitive species tested to date to the adverse reproductive effects of PCBs. However, the relative sensitivity of these three species selected by the Service for the injury assessment relative to all other species in the Lower Fox River/Green Bay assessment area is unknown, particularly for the injury endpoints related to fish health. Therefore, it is possible that adverse effects on viability could be occurring to fish species in the assessment area that were not selected for focused study.

CHAPTER 5
FISH CONSUMPTION ADVISORY INJURIES

5.1 INTRODUCTION

This chapter determines and summarizes injuries to fish in the Lower Fox River, Green Bay, and northern Lake Michigan resulting from exceedences of: (1) tolerances for PCBs established by the Food and Drug Administration (FDA) under the Food, Drug, and Cosmetic Act [43 CFR 11.62(f)(1)(ii)]; and (2) PCB levels for which Wisconsin and Michigan have issued directives to limit or ban consumption [43 CFR 11.62(f)(1)(iii)]. This chapter begins by summarizing a comprehensive 1998 Service report on fish consumption advisories in the Lower Fox River/Green Bay Assessment Area (Stratus Consulting Inc., 1998) and goes on to update the findings of that report with Wisconsin and Michigan's advisories for 1998 and 1999.

5.2 SUMMARY OF 1998 FISH CONSUMPTION ADVISORY REPORT

In November 1998, the Service released a report entitled "Fish Consumption Advisories in the Lower Fox River/Green Bay Assessment Area." That report presented a detailed examination of the injuries to fishery resources resulting from fish consumption advisories issued by State agencies and from exceedences of FDA tolerance levels for PCBs (Stratus Consulting Inc., 1998).

Tables 5-1 to 5-3, taken from the Service's report, summarize the results of the injury evaluation based on exceedences of the FDA tolerance level for PCBs. This analysis demonstrated that the FDA tolerance was exceeded in multiple fish species in the assessment area: 13 species in the Lower Fox River (Table 5-1); 23 species in Green Bay (Table 5-2); and 6 species in northern Lake Michigan (Table 5-3). Exceedences occurred since the first sample collections in the 1970s and continued through 1995 (the most recent year analyzed in the Service's 1998 report).

Tables 5-4 to 5-6, taken from the Service's report, summarize Michigan and Wisconsin PCB fish advisories. In the tables, advisories are divided into six time periods that reflect when major changes in advisories took place. Advisories were first issued in 1976 and have continued to the present. Fish consumption advisories have been issued by Wisconsin for 15 species in the Lower Fox River (Table 5-4), and by Wisconsin or Michigan for more than 20 species in Green Bay and Lake Michigan (Tables 5-5 and 5-6).

Table 5-1
Summary of Fish Species in the Lower Fox River
that Have Exceeded the FDA Tolerance[a] for PCBs, 1971-1995

	1971-1974	1975-1979	1980-1984	1985-1989	1990-1995
Brown bullhead		●	○	●	
Carp		●	●	●	●
Channel catfish				○	●
Chinook salmon		●			
Flathead catfish				●	
Gizzard shad			●		
Northern pike		●	○	●	○
Sheepshead/drum			●	●	
Walleye		●	●	●	●
White bass			○	●	●
White perch					●
White sucker		●	○	●	
Yellow perch		●	○		

● = At least one sample exceeded FDA tolerance for PCBs.
○ = No samples exceeded FDA tolerance for PCBs.
A blank cell means that the species was not analyzed for PCBs during that time period.
a. Samples collected through 1984 were compared to the FDA tolerance of 5 mg/kg in edible tissue; samples collected after 1984 were compared to the revised FDA tolerance of 2 mg/kg in edible tissue.

Source: Stratus Consulting Inc. (1998).

Table 5-2
Summary of Fish Species in Green Bay
that Have Exceeded the FDA Tolerance[a] for PCBs, 1971-1995

	1971-1974	1975-1979	1980-1984	1985-1989	1990-1995
Alewife		○	●		
Brook trout			○	●	○
Brown bullhead		●	○		
Brown trout			●	●	●
Carp		●	●	●	●
Channel catfish					●
Chinook salmon			●	●	○
Cisco/lake herring		●		○	
Coho salmon		●			
Gizzard shad		●			
Lake sturgeon				●	
Lake trout	●	●	●		
Longnose sucker				●	●
Northern pike		●	●	○	
Rainbow trout				●	○
Smallmouth bass		○	●	○	○
Splake				●	●
Walleye		○	●	●	●
White bass		●	○		
White perch					●
White sucker		●	○	○	
Whitefish		●	○	○	○
Yellow perch		●	○		○

● = At least one sample exceeded FDA tolerance for PCBs.
○ = No samples exceeded FDA tolerance for PCBs.
A blank cell means that the species was not analyzed for PCBs during that time period.
a. Samples collected through 1984 were compared to the FDA tolerance of 5 mg/kg in edible tissue; samples collected after 1984 were compared to the revised FDA tolerance of 2 mg/kg in edible tissue.

Source: Stratus Consulting Inc. (1998).

Table 5-3
Summary of Fish Species in Northern Lake Michigan that Have Exceeded the FDA Tolerance[a] for PCBs, 1971-1995

	1971-1974	1975-1979	1980-1984	1985-1989	1990-1995
Brook trout			○	●	
Brown trout	●		●	●	●
Chinook salmon	●	●	●	●	●
Coho salmon	●		○		
Lake trout	●	●	●	●	
Whitefish		●	○	●	○

● = At least one sample exceeded FDA tolerance for PCBs.
○ = No samples exceeded FDA tolerance for PCBs.
A blank cell means that the species was not analyzed for PCBs during that time period.
a. Samples collected through 1984 were compared to the FDA tolerance of 5 mg/kg in edible tissue; samples collected after 1984 were compared to the revised FDA tolerance of 2 mg/kg in edible tissue.

Source: Stratus Consulting Inc. (1998).

Table 5-4
Summary of Fish Species in the Lower Fox River for Which PCB Consumption Advisories Have Been Issued by Wisconsin, 1976-1997

	1976-1977	1978-1983[a]	1984[b]-1986	1987-1994	1995-1996	1997
Black crappie		●				●
Bluegill		●				●
Bullhead		●		●	●	
Carp	●	●	●	●	●	●
Channel catfish		●		●	●	●
Drum		●		●	●	
Northern pike		●	●	●	●	●
Rock bass		●				●
Sheepshead		●				●
Smallmouth bass		●				●
Walleye		●	●	●	●	●
White bass		●	●	●	●	●
White perch		●				●
White sucker		●		●	●	●
Yellow perch		●				●

● = Consumption advisory (either "no consumption" or "limit consumption") issued.
A blank cell means no advisory was issued.
a. From 1978 to 1983, a "limit consumption" advisory was issued for all species in the Lower Fox River.
b. For 1984, the advisories are taken from the Health Guide (Wisconsin Department of Natural Resources, 1976-1994).

Source: Stratus Consulting Inc (1998).

Table 5-5
Summary of Fish Species in Green Bay for Which PCB Consumption Advisories Have Been Issued by Wisconsin or Michigan, 1976-1997

	1976-1977	1978-1983	1984[a]-1986	1987-1994	1995-1996	1997
Brook trout	See advisory for trout		●	●	●	●
Brown trout	See advisory for trout		●	●	●	●
Bullheads		●				
Carp	●	●	●	●	●	●
Catfish		●	●			●
Chinook salmon	See advisory for salmon		●	●	●	●
Coho salmon	See advisory for salmon		●			
Lake trout	See advisory for trout		●			●
Northern pike			●	●	●	●
Rainbow trout	See advisory for trout		●	●	●	●
Salmon	●	●	See advisories for coho and chinook salmon			
Smallmouth bass			●			●
Splake				●	●	●
Sturgeon				●	●	●
Trout	●	●	See advisories for lake, brown, brook, and rainbow trout			
Walleye			●	●	●	●
White bass			●	●	●	●
White perch						●
White sucker			●	●		●
Whitefish		●	●			●
Yellow perch						●

● = Consumption advisory (either "no consumption" or "limit consumption") issued.
A blank cell means no advisory was issued.
a. For 1984, the Wisconsin advisories are taken from the Health Guide (Wisconsin Department of Natural Resources, 1976-1994).
The table excludes advisories issued by Michigan for mercury only.

Source: Stratus Consulting Inc. (1998).

Table 5-6
Summary of Fish Species in Lake Michigan (Wisconsin waters and Northern Lake Michigan north of Frankfort, Michigan) for Which PCB Consumption Advisories Have Been Issued by Wisconsin or Michigan, 1976-1997

	1976-1977	1978-1983	1984[a]-1986	1987-1994	1995-1996	1997
Brook trout	See advisory for trout		●			
Brown trout	See advisory for trout		●	●	●	●
Carp	●	●	●	●	●	●
Catfish		●	●	●	●	●
Chinook salmon	See advisory for salmon		●	●	●	●
Chubs						●
Coho salmon	See advisory for salmon		●	●	●	
Lake trout	●	●	●	●	●	●
Longnose suckers[b]				●	●	●
Northern pike			●			
Rainbow trout or steelhead	●	●	●			●
Salmon	●	●	See advisories for chinook and coho salmon			
Smallmouth bass			●			
Smelt						●
Sturgeon						●
Trout	●	●	See advisories for lake, brown, brook, and rainbow trout			
Walleye			●			
White bass			●			
White sucker			●			
Whitefish			●			●
Yellow perch						●

● = Consumption advisory (either "no consumption" or "limit consumption") issued.
A blank cell means no advisory was issued.
a. For 1984, the advisories are taken from the Health Guide (Wisconsin Department of Natural Resources, 1976-1994).
b. Advisory for longnose suckers issued for Little Bay de Noc only.
The table excludes advisories issued by Michigan for mercury only.

Source: Stratus Consulting Inc. (1998).

5.3 FISH CONSUMPTION ADVISORY UPDATE

The Service's 1998 report included analysis of Wisconsin and Michigan fish consumption advisories through 1997. Both states issued advisories in 1998 and 1999, and this section updates the findings of the Service's 1998 report with these more recent fish consumption advisories. Wisconsin's advisories underwent very little change between 1997 and 1999; Michigan, however, introduced a new format for its advisories in 1998 and included more species in 1998 and 1999.

5.3.1 Wisconsin Fish Consumption Advisories

Wisconsin fish consumption advisories for 1998 and 1999 are summarized by location in Tables 5-7 through 5-10 (1997 advisories are included for comparison). The PCB advisory, which is listed in separate charts from the advisory for mercury, underwent only a few changes from 1997 to 1999. White perch was added to the consumption advisory for the Lower Fox River from DePere Dam to Green Bay in 1999 (Table 5-8). In 1998, size classifications for brown trout and chinook salmon changed in the advisory for Wisconsin waters of Green Bay (Table 5-9); these changes made the advisory for chinook salmon more restrictive and the advisory for brown trout less restrictive. In the same year and location, the advisory for white perch also became less restrictive, changing from no consumption to one meal per two months or six meals per year.

In 1999, Wisconsin introduced a new method of communicating fish consumption advisories to the public (M. Young, Wisconsin State Division of Health, pers. comm., 1999). The Wisconsin State Division of Health, concerned that women and people of color were at risk because they were not as likely to know of the advisories, initiated a program to communicate the advisories to these groups of people. As part of this effort, signs conveying the information from the advisories were placed around bodies of water most frequently fished by people of color. The Fox River and Green Bay were specifically targeted, and an estimated 30 signs were posted in this region. Two signs were posted at each location, one conveying the PCB advisory and the other conveying the mercury advisory. The language for the signs was simplified from the printed fish consumption advisory distributed to the public, but advisory information was the same. In addition, posters were printed for display in places such as medical and health offices and nature centers. Although in the first year of the program signs were posted in English only, the Division of Health has plans to expand the program to include signs translated into Spanish and Hmong (M. Young, Wisconsin State Division of Health, pers. comm., 1999).

Table 5-7
Wisconsin Fish Consumption Advisories for the Lower Fox River from Little Lake Butte des Morts to the Dam at DePere

Species	Size	1997	1998	1999
Carp	All	●	●	●
Northern pike	All	1 per mn	1 per mn	1 per mn
Smallmouth bass	All	1 per mn	1 per mn	1 per mn
Walleye	All	1 per mn	1 per mn	1 per mn
White bass	All	1 per mn	1 per mn	1 per mn
White perch	All	1 per mn	1 per mn	1 per mn
Yellow perch	All	1 per wk	1 per wk	1 per wk

● = No consumption.
1 per wk = 1 meal per week or 52 meals per year.
1 per mn = 1 meal per month or 12 meals per year.

Sources: Wisconsin Division of Health and Wisconsin Department of Natural Resources (1997; 1998; 1999).

Table 5-8
Wisconsin Fish Consumption Advisories for the Lower Fox River from the Mouth at Green Bay up to DePere Dam

Species	Size	1997	1998	1999
Black crappie	<9"	1 per mn	1 per mn	1 per mn
	>9"	1 per 2 mn	1 per 2 mn	1 per 2 mn
Bluegill	All	1 per mn	1 per mn	1 per mn
Carp	All	●	●	●
Channel catfish	All	●	●	●
Northern pike	<25"	1 per mn	1 per mn	1 per mn
	>25"	1 per 2 mn	1 per 2 mn	1 per 2 mn
Rock bass	All	1 per mn	1 per mn	1 per mn
Sheepshead	<10"	1 per mn	1 per mn	1 per mn
	10-13"	1 per 2 mn	1 per 2 mn	1 per 2 mn
	>13"	●	●	●
Smallmouth bass	All	1 per 2 mn	1 per 2 mn	1 per 2 mn
Walleye	<16"	1 per mn	1 per mn	1 per mn
	16-22"	1 per 2 mn	1 per 2 mn	1 per 2 mn
	>22"	●	●	●
White bass	All	●	●	●
White perch[a]	All			1 per 2 mn
White sucker	All	1 per 2 mn	1 per 2 mn	1 per 2 mn
Yellow perch	All	1 per mn	1 per mn	1 per mn

● = No consumption.
A blank cell means no advisory was issued.
1 per wk = 1 meal per week or 52 meals per year.
1 per mn = 1 meal per month or 12 meals per year.
1 per 2 mn = 1 meal every 2 months or 6 meals per year.
a. Species added to advisory in 1999.

Sources: Wisconsin Division of Health and Wisconsin Department of Natural Resources (1997; 1998; 1999).

Table 5-9
Wisconsin Fish Consumption Advisories for Wisconsin Waters of Green Bay South of Marinette and its Tributaries (except the Fox River) including the Menominee, Oconto, and Peshtigo Rivers from Their Mouths Up to the First Dam

Species	Size	1997	1998	1999
Brown trout[a]	<14"	1 per mn	NA	NA
	14-21"	1 per 2 mn	NA	NA
	>21"	●	NA	NA
	<17"	NA	1 per mn	1 per mn
	17-28"	NA	1 per 2 mn	1 per 2 mn
	>28"	NA	●	●
Carp	All	●	●	●
Channel catfish	All	1 per 2 mn	1 per 2 mn	1 per 2 mn
Chinook salmon[a]	<29"	1 per wk	NA	NA
	>29"	1 per mn	NA	NA
	<30"	NA	1 per mn	1 per mn
	>30"	NA	1 per 2 mn	1 per 2 mn
Northern pike	<22"	1 per wk	1 per wk	1 per wk
	>22"	1 per mn	1 per mn	1 per mn
Rainbow trout	All	1 per mn	1 per mn	1 per mn
Smallmouth bass	All	1 per mn	1 per mn	1 per mn
Splake	<16"	1 per mn	1 per mn	1 per mn
	16-20"	1 per 2 mn	1 per 2 mn	1 per 2 mn
	>20"	●	●	●
Sturgeon	All	●	●	●
Walleye	<17"	1 per mn	1 per mn	1 per mn
	17-26"	1 per 2 mn	1 per 2 mn	1 per 2 mn
	>26"	●	●	●
White bass	All	●	●	●
Whitefish	All	1 per 2 mn	1 per 2 mn	1 per 2 mn
White perch	All	●	1 per 2 mn	1 per 2 mn
White sucker	All	1 per mn	1 per mn	1 per mn
Yellow perch	All	1 per wk	1 per wk	1 per wk

● = No consumption.
1 per wk = 1 meal per week or 52 meals per year.
1 per mn = 1 meal per month or 12 meals per year.
1 per 2 mn = 1 meal every 2 months or 6 meals per year.
NA = Not applicable, due to change in size criteria.
a. Size criteria changed from 1997 to 1998.

Sources: Wisconsin Division of Health and Wisconsin Department of Natural Resources (1997; 1998; 1999).

Table 5-10
Wisconsin Fish Consumption Advisories for the Wisconsin Waters of Lake Michigan and its Tributaries up to the First Dam, including the Root River, Milwaukee River, Sheboygan River, Manitowoc River, and Kewaunee River

Species	Size	1997	1998	1999
Brown trout	<22"	1 per mn	1 per mn	1 per mn
	>22"	1 per 2 mn	1 per 2 mn	1 per 2 mn
Chinook salmon	<30"	1 per mn	1 per mn	1 per mn
	>30"	1 per 2 mn	1 per 2 mn	1 per 2 mn
Chubs	All	1 per mn	1 per mn	1 per mn
Coho salmon	All	1 per mn	1 per mn	1 per mn
Lake trout	<23"	1 per mn	1 per mn	1 per mn
	23-27"	1 per 2 mn	1 per 2 mn	1 per 2 mn
	>27"	●	●	●
Rainbow trout	<17"	1 per wk	1 per wk	1 per wk
	>17"	1 per mn	1 per mn	1 per mn
Smelt	All	1 per wk	1 per wk	1 per wk
Whitefish	<19"	1 per wk	1 per wk	1 per wk
	19-25"	1 per mn	1 per mn	1 per mn
	>25"	1 per 2 mn	1 per 2 mn	1 per 2 mn
Yellow perch	All	1 per wk	1 per wk	1 per wk

● = No consumption.
1 per wk = 1 meal per week or 52 meals per year.
1 per mn = 1 meal per month or 12 meals per year.
1 per 2 mn = 1 meal every 2 months or 6 meals per year.

Sources: Wisconsin Division of Health and Wisconsin Department of Natural Resources (1997; 1998; 1999).

5.3.2 Michigan Fish Consumption Advisories

Michigan fish consumption advisories for 1998 and 1999 are summarized by location in Tables 5-11 through 5-13 (1997 advisories are included for comparison). Unlike Wisconsin advisories, which contain separate charts for PCBs and mercury, Michigan advisories contain a single chart within which contaminants are specified for each species. Most species in 1997, 1998, and 1999 were cited for PCBs only, with the following exceptions: Green Bay walleye in all three years were cited for PCBs and mercury; Lake Michigan walleye in 1998 and 1999 were cited for PCBs and mercury; Lake Michigan whitefish in 1998 were cited for PCBs and chlordane; Lake Michigan whitefish in 1999 were cited for PCBs, dioxins, and chlordane; Lake Michigan lake trout in 1999 were cited for PCBs and chlordane; and Little Bay de Noc smallmouth bass in 1999 were cited for PCBs and mercury.

In 1998 and 1999, Michigan continued to release its fish consumption advisory to the public in pamphlet format. However, between 1997 and 1998, the state introduced a new format for its consumption guidelines. The new format, implemented in 1998 and continued into 1999, gives more categories of advisory levels and separates consumers into two groups: (1) the general population, including men, boys over the age of 15, and women who are beyond childbearing years; and (2) women and children, including women who are pregnant or breastfeeding, women who intend to have children, girls over the age of 15, and all children under the age of 15. The new format allows for more specificity in application of advisories.

Several species were added to the Michigan advisory in 1998 as follows: (1) burbot, chinook salmon, whitefish, white perch, white sucker, and yellow perch were added to the advisory for Green Bay; (2) burbot and smallmouth bass were added to the advisory for Little Bay de Noc; and (3) chinook salmon, coho salmon, rainbow trout, smelt, whitefish, and yellow perch were added to the advisory for Lake Michigan north of Frankfurt. Brook trout, removed from the Green Bay advisory in 1998, was the only species removed from any location in the 1998 and 1999 advisories.

Additional species were added to the Michigan advisory in 1999 as follows: (1) longnose sucker and smallmouth bass were added to the advisory for Green Bay; and (2) northern pike and white sucker were added to the advisory for Little Bay de Noc. For Green Bay, there were no other changes in the advisory between 1998 and 1999. For Little Bay de Noc, the fish consumption advisory changed for a few species in 1999, becoming slightly more restrictive for consumption of longnose sucker and smallmouth bass in the general population category. For Lake Michigan north of Frankfurt, the consumption advisory became slightly more restrictive for women and children consuming chinook salmon, slightly less restrictive for the general population consuming lake trout, and much more restrictive for both categories consuming whitefish.

Table 5-11
Michigan Fish Consumption Advisories for the Michigan Waters of Green Bay South of Cedar River (applies to Menominee and Cedar rivers below first dam)

Species	Size	1997	1998		1999	
			General Population	Women and Children	General Population	Women and Children
Brook trout	<14"	unlimited				
	>14"	●				
Brown trout	<14"	1 per wk[a]	1 per wk	1 per mn	1 per wk	1 per mn
	14-18"	1 per wk[a]	1 per wk	1 per 2 mn	1 per wk	1 per 2 mn
	>18"	●	●	●	●	●
Burbot[b]	<26"		unlimited	1 per wk	unlimited	1 per wk
	>26"		unlimited	1 per mn	unlimited	1 per mn
Carp[c]	All	●	●	●	●	●
Channel catfish[c]	<12"	●				
	>12"	●	1 per wk	1 per 2 mn	1 per wk	1 per 2 mn
Chinook salmon[b]	All		unlimited	1 per mn	unlimited	1 per mn
Lake trout	<22"	unlimited	unlimited	1 per mn	unlimited	1 per mn
	22-26"	1 per wk[a]	1 per wk	1 per mn	1 per wk	1 per mn
	>26"	1 per wk[a]	1 per wk	1 per 2 mn	1 per wk	1 per 2 mn
Longnose sucker[d]	All				1 per wk	1 per 2 mn
Northern pike	<26"	unlimited	unlimited	1 per mn	unlimited	1 per mn
	>26"	●	unlimited	1 per mn	unlimited	1 per mn
Rainbow trout	<22"	unlimited	unlimited	1 per mn	unlimited	1 per mn
	>22"	●	unlimited	1 per mn	unlimited	1 per mn
Smallmouth Bass[d]	<18"				unlimited	1 per mn
	>18"				1 per wk	1 per mn

Table 5-11 (cont.)
Michigan Fish Consumption Advisories for the Michigan Waters of Green Bay South of Cedar River (applies to Menominee and Cedar rivers below first dam)

Species	Size	1997	1998		1999	
			General Population	Women and Children	General Population	Women and Children
Splake	<14"	1 per wk[a]	1 per wk	1 per mn	1 per wk	1 per mn
	14-18"	1 per wk[a]	1 per wk	1 per 2 mn	1 per wk	1 per 2 mn
	>18"	●	●	●	●	●
Sturgeon	All	●	●	●	●	●
Walleye	<18"	unlimited	unlimited	1 per mn	unlimited	1 per mn
	18-26"	●	1 per wk	1 per 2 mn	1 per wk	1 per 2 mn
	>26"	●	●	●	●	●
White bass	All	●	●	●	●	●
Whitefish[b]	All		unlimited	1 per 2 mn	unlimited	1 per 2 mn
White perch[b]	All		●	●	●	●
White sucker[b]	All		unlimited	1 per mn	unlimited	1 per mn
Yellow perch[b]	All		unlimited	1 per wk	unlimited	1 per wk

● = No consumption.
A blank cell means no advisory was issued.
1 per wk = One meal per week.
1 per mn = One meal per month.
1 per 2 mn = Six meals per year.
a. No consumption for nursing mothers, pregnant women, women who intend to have children, and children under age 15.
b. Species added in 1998.
c. In 1997, carp and channel fish were grouped together in the advisory; this is why all size classes are represented in 1997, but not in 1998 or 1999.
d. Species added in 1999.

Sources: Michigan Department of Natural Resources (1997) and Michigan Department of Community Health (1998; 1999).

Table 5-12
Michigan Fish Consumption Advisory for Little Bay de Noc

Species	Size	1997	1998		1999	
			General Population	Women and Children	General Population	Women and Children
Burbot[a]	<26"		unlimited	1 per wk	unlimited	1 per wk
	>26"		unlimited	1 per mn	unlimited	1 per mn
Longnose sucker	<14"	unlimited	unlimited	1 per mn	1 per wk	1 per mn
	14-18"	1 per wk[b]	1 per wk	1 per 2 mn	1 per wk	1 per 2 mn
	<18"	1 per wk[b]	1 per wk	●	1 per wk	●
Northern pike[c]	<30"				unlimited	1 per mn
	>30"				unlimited	1 per 2 mn
Smallmouth bass[a]	<18"		unlimited	1 per mn	unlimited	1 per mn
	>18"		unlimited	1 per mn	1 per wk	1 per mn
White sucker[c]	All				unlimited	1 per mn

● = No consumption.
A blank cell means no advisory was issued.
1 per wk = One meal per week.
1 per mn = One meal per month.
1 per 2 mn = Six meals per year.
a. Species added in 1998.
b. No consumption for nursing mothers, pregnant women, women who intend to have children, and children under age 15.
c. Species added in 1999.

Sources: Michigan Department of Natural Resources (1997) and Michigan Department of Community Health (1998; 1999).

In 1997, the Environmental Protection Agency (EPA) issued a supplementary fish consumption advisory for Michigan's Great Lakes Waters (U.S. EPA, 1997). The EPA used an advisory system more protective than the one used by Michigan. The EPA supplement listed advisories for PCBs on the following species not included in the Michigan advisory: coho salmon, chinook salmon, rainbow trout, yellow perch, and smelt. All of these species were added to Michigan's advisory in 1998. The supplement also included a more restrictive advisory for brown trout.

Table 5-13
Michigan Fish Consumption Advisories for Lake Michigan North of Frankfurt (includes Big Bay de Noc and parts of northern Green Bay)

Species	Size	1997	1998		1999	
			General Population	Women and Children	General Population	Women and Children
Brown trout	<22"	unlimited	unlimited	1 per mn	unlimited	1 per mn
	>22"	●	●	●	●	●
Carp, catfish	All	●	●	●	●	●
Chinook salmon[a]	<26"		unlimited	1 per mn	unlimited	1 per mn
	>26"		unlimited	1 per mn	unlimited	1 per 2 mn
Coho salmon[a]	<30		unlimited	1 per mn	unlimited	1 per mn
	>30"		unlimited	1 per 2 mn	unlimited	1 per 2 mn
Lake trout	<22"	unlimited	unlimited	1 per mn	unlimited	1 per mn
	22-26"	1 per wk[b]	1 per wk	1 per mn	unlimited	1 per mn
	>26"	1 per wk[b]	1 per wk	1 per 2 mn	1 per wk	1 per 2 mn
Rainbow trout[a]	<18"		unlimited	1 per wk	unlimited	1 per wk
	>18"		unlimited	1 per mn	unlimited	1 per mn
Smelt[a]	All		unlimited	1 per wk	unlimited	1 per wk
Sturgeon	All	●	●	●	●	●
Walleye	<18"		unlimited	1 per wk	unlimited	1 per wk
	18-22"		unlimited	1 per mn	unlimited	1 per mn
	22-26"		1 per wk	1 per mn	1 per wk	1 per mn
	>26"		1 per wk	1 per 2 mn	1 per wk	1 per 2 mn
Whitefish[a]	<14"		unlimited	1 per wk	1 per wk	●
	14-18"		unlimited	1 per wk	●	●
	18-26"		unlimited	1 per mn	●	●
	>26"		unlimited	1 per 2 mn	●	●
Yellow perch[a]	All		unlimited	1 per wk	unlimited	1 per wk

● = No consumption.
A blank cell means no advisory was issued.
1 per wk = One meal per week.
1 per mn = One meal per month.
1 per 2 mn = Six meals per year.
a. Species added in 1998.
b. no consumption for nursing mothers, pregnant women, women who intend to have children, and children under age 15.

Sources: Michigan Department of Natural Resources (1997) and Michigan Department of Community Health (1998; 1999).

5.4 INJURY DETERMINATION

5.4.1 Injury Definitions

Injuries to fishery resources were assessed according to the definitions of injury in the Department's NRDA regulations. Specifically, this chapter assesses injury to fishery resources resulting from PCB exposure according to the following injury definitions:

- the concentration of the released hazardous substance is sufficient to exceed action or tolerance levels established under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342, in edible portions of organisms [43 CFR § 11.62(f)(1)(ii)]

- the concentration of the released hazardous substance is sufficient to exceed levels for which an appropriate State health agency has issued directives to limit or ban consumption of such organism [43 CFR § 11.62(f)(1)(iii)].

Injuries according to the first definition were determined by comparing PCB concentrations measured in fish fillets with the PCB tolerance level established by the FDA under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342. Injuries according to the second definition were determined by evaluating the fish consumption advisories issued by Michigan and Wisconsin in the Lower Fox River/Green Bay area, the state programs and procedures used to determine and issue the advisories, and the role of PCBs in issuing the advisories.

5.4.2 Injury Determination and Spatial/Temporal Extent of Injuries

The information presented in this chapter demonstrates that fishery resources throughout the Lower Fox River/Green Bay area are and have been injured as a result of exposure to PCBs. Injuries result from (1) fish tissue PCB concentrations that exceed the FDA tolerance level, and (2) fish tissue PCB concentrations that have triggered the establishment of fish consumption advisories by the states of Wisconsin and Michigan.

Exceedences of the FDA tolerance level in edible fish tissue have been documented since 1971 in Green Bay and since 1975 in the Lower Fox River. Exceedences most likely occurred prior to these years, but no fish PCB concentration data are available for earlier years. Exceedences continue to occur through 1995, the most recent year of data included in the injury assessment. The spatial extent of the exceedences of the FDA tolerance level in edible fish tissue includes all of the Lower Fox River from Little Lake Butte des Morts to the river mouth and Green Bay. Exceedences have been documented in many fish species throughout the Lower Fox River/Green Bay system.

The fishery resources of the area are also injured by PCBs as a result of extensive fish consumption advisories in place for many fish species. The species, spatial and temporal extent, and degree of fish consumption advisory injuries for Lower Fox River/Green Bay fish are summarized in Tables 5-14 through 5-16. These tables include the tributaries that are noted in the advisories summarized in Tables 5-7 through 5-13. Figure 5-1 depicts the spatial extent of PCB fish consumption advisory injuries, with different colors corresponding to the different areas specified in the advisories. The tables and the figure demonstrate that as a result of PCB contamination, fish consumption advisories have been issued for many fish species in the Lower Fox River by Wisconsin and for many fish species in Green Bay, tributaries to Green Bay, and northern Lake Michigan by Michigan and Wisconsin since 1976, and this continues through the present (1999).

Table 5-14
Summary of Fish Species in the Lower Fox River for Which PCB Consumption Advisories Have Been Issued by Wisconsin, 1976-1999

	1976-1977	1978-1983[a]	1984[b]-1986	1987-1994	1995-1996	1997-1999
Black crappie		●				●
Bluegill		●				●
Bullhead		●		●	●	
Carp	●	●	●	●	●	●
Channel catfish		●		●	●	●
Drum		●		●	●	
Northern pike		●	●	●	●	●
Rock bass		●				●
Sheepshead		●				●
Smallmouth bass		●				●
Walleye		●	●	●	●	●
White bass		●	●	●	●	●
White perch		●				●
White sucker		●		●	●	●
Yellow perch		●				●

● = Consumption advisory (either "no consumption" or "limit consumption") issued.
A blank cell means no advisory was issued.
a. From 1978 to 1983, a "limit consumption" advisory was issued for all species in the Lower Fox River.
b. For 1984, the advisories are taken from the Health Guide (Wisconsin Department of Natural Resources, 1976-1994).

Adapted from Stratus Consulting Inc. (1998), with additional information from Wisconsin Division of Health and Wisconsin Department of Natural Resources (1998; 1999).

Table 5-15
Summary of Fish Species in Green Bay for Which PCB Consumption Advisories Have Been Issued by Wisconsin or Michigan, 1976-1997

	1976-1977	1978-1983	1984[a]-1986	1987-1994	1995-1996	1997	1998	1999
Brook trout	See advisory for trout		●	●	●	●		
Brown trout	See advisory for trout		●	●	●	●	●	●
Bullheads		●						
Burbot							●	●
Carp	●	●	●	●	●	●	●	●
Catfish		●	●			●	●	●
Chinook salmon	See advisory for salmon		●	●	●	●	●	●
Coho salmon	See advisory for salmon		●					
Lake trout	See advisory for trout		●			●	●	●
Lake whitefish			●					
Longnose sucker								●
Northern pike			●	●	●	●	●	●
Rainbow trout	See advisory for trout		●	●	●	●	●	●
Salmon	●	●	See advisories for coho and chinook salmon					
Smallmouth bass			●			●	●	●
Splake				●	●	●	●	●
Sturgeon				●	●	●	●	●
Trout	●	●	See advisories for lake, brown, brook, and rainbow trout					
Walleye			●	●	●	●	●	●
White bass			●	●	●	●	●	●
White perch						●	●	●
White sucker			●	●		●	●	●
Whitefish		●				●	●	●
Yellow perch						●	●	●

● = Consumption advisory (either "no consumption" or "limit consumption") issued.
A blank cell means no advisory was issued.
a. For 1984, the Wisconsin advisories are taken from the Health Guide (Wisconsin Department of Natural Resources, 1976-1994).
The table excludes advisories issued by Michigan for mercury only.

Adapted from Stratus Consulting Inc. (1998), with additional information from Wisconsin Division of Health and Wisconsin Department of Natural Resources (1998; 1999) and Michigan Department of Natural Resources (1998; 1999).

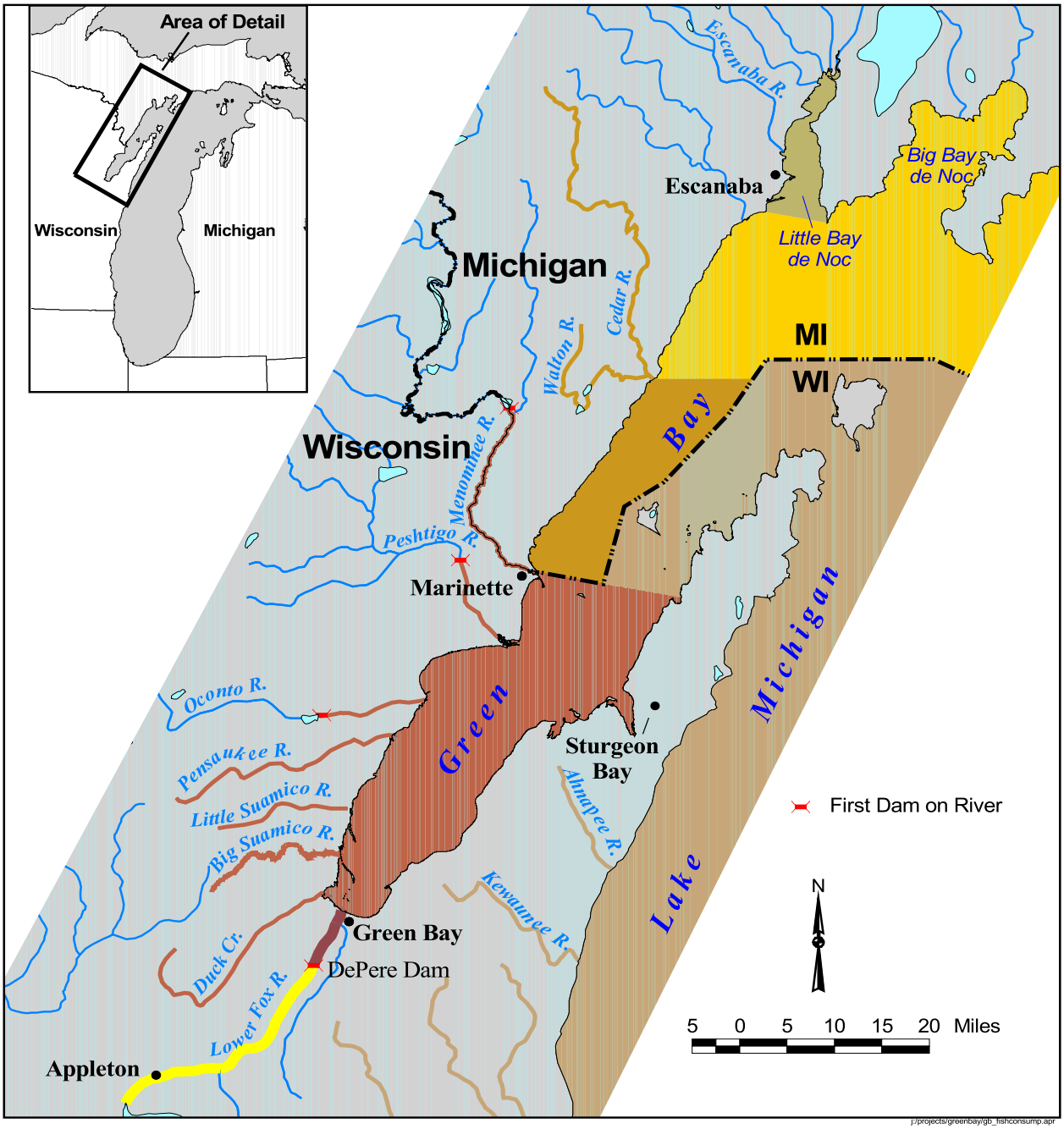
Table 5-16
Summary of Fish Species in Lake Michigan (Wisconsin waters and Northern Lake Michigan north of Frankfort, Michigan) for Which PCB Consumption Advisories Have Been Issued by Wisconsin or Michigan, 1976-1997

	1976-1977	1978-1983	1984[a]-1986	1987-1994	1995-1996	1997	1998-1999
Brook trout	See advisory for trout		●				
Brown trout	See advisory for trout		●	●	●	●	●
Carp	●	●	●	●	●	●	●
Catfish		●	●	●	●	●	●
Chinook salmon	See advisory for salmon		●	●	●	●	●
Chubs						●	●
Coho salmon	See advisory for salmon		●	●	●	●	●
Lake trout	●	●	●	●	●	●	●
Lake whitefish			●				
Longnose suckers[b]				●	●	●	●
Northern pike			●				
Rainbow trout or steelhead	●	●	●			●	●
Salmon	●	●	See advisories for chinook and coho salmon				
Smallmouth bass			●				
Smelt						●	●
Sturgeon						●	●
Trout	●	●	See advisories for lake, brown, brook, and rainbow trout				
Walleye			●				
White bass			●				
White sucker			●				
Whitefish						●	●
Yellow perch						●	●

● = Consumption advisory (either "no consumption" or "limit consumption") issued.
A blank cell means no advisory was issued.
a. For 1984, the advisories are taken from the Health Guide (Wisconsin Department of Natural Resources, 1976-1994).
b. Advisory for longnose suckers issued for Little Bay de Noc only.
The table excludes advisories issued by Michigan for mercury only.

Adapted from Stratus Consulting Inc. (1998), with additional information from Wisconsin Division of Health and Wisconsin Department of Natural Resources (1998; 1999).

Figure 5-1
Spatial Extent of PCB Fish Consumption Advisories in the Lower Fox River/Green Bay Area. Different colors correspond to the different regions referred to in the advisories (see Tables 5-7 through 5-13 for region descriptions and advisory details). In general, advisories decrease in level of restricted consumption from the Lower Fox River to outer portions of Green Bay.



CHAPTER 6
ADVERSE HEALTH EFFECT INJURIES TO WALLEYE

6.1 INTRODUCTION

This chapter presents an evaluation of adverse health effect injuries to walleye in the Lower Fox River and Green Bay. The chapter begins with a summary of Barron et al. (1999), which describes the results of NRDA studies conducted by the Service that examined the association of PCBs with a comprehensive suite of fish health parameters in Lower Fox River and Green Bay walleye. Additional data on PCBs in walleye are also presented, and then integrated with the results of Barron et al. (1999).

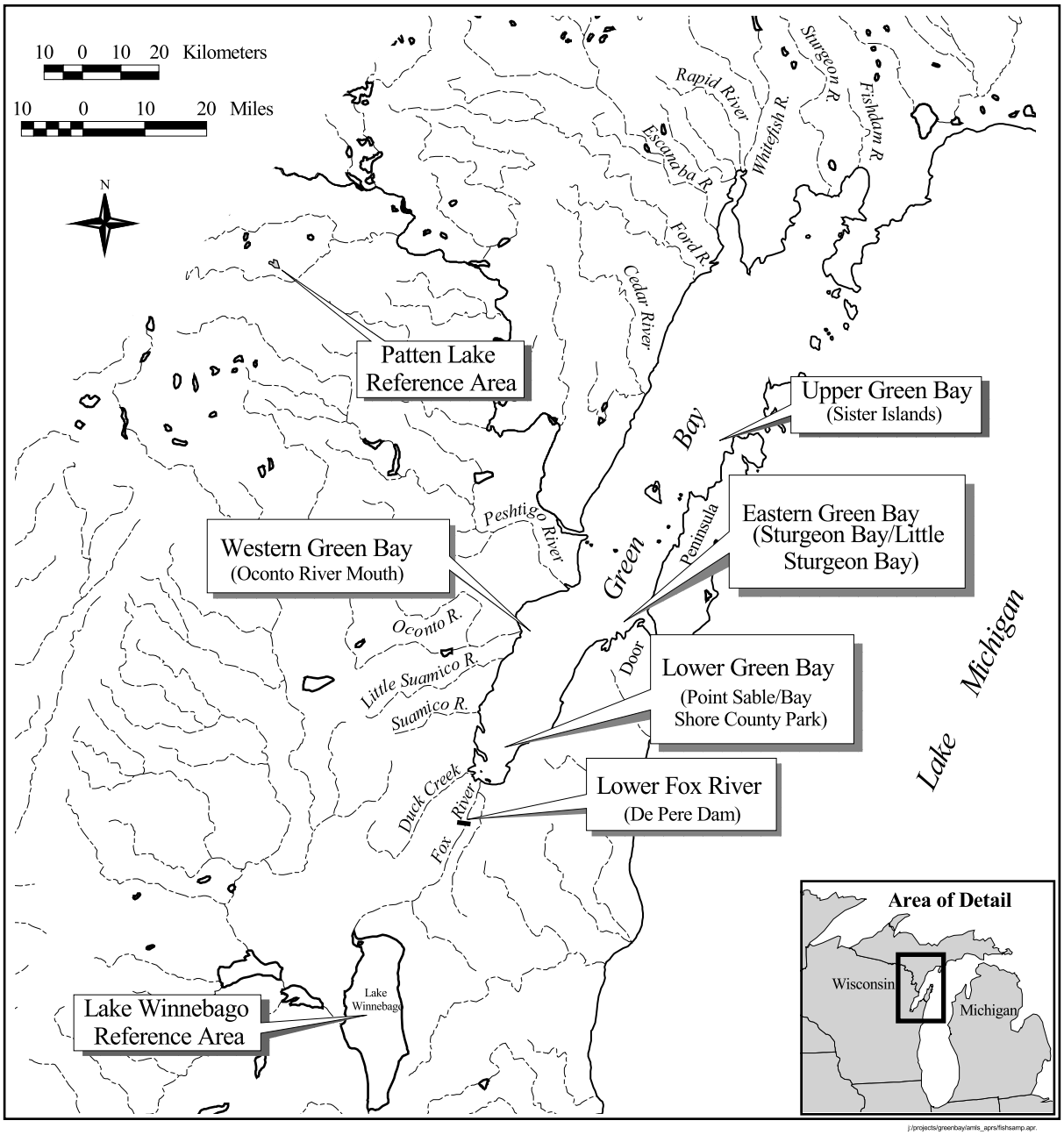
6.2 SUMMARY OF DATA REPORTED IN BARRON ET AL. (1999)

In a series of NRDA studies reported by Barron et al. (1999) [with supporting documentation in Stratus Consulting (1999a)], the Service measured a number of biomarker and physiological responses of adult walleye collected from the Lower Fox River and Green Bay and at two reference sites (Lake Winnebago and Patten Lake) to evaluate the effects of PCBs on walleye health. As discussed below, the most striking differences relating to fish health between assessment and reference area fish were in PCB concentrations and liver tumors and pre-tumors.

Walleye collection locations are shown in Figure 6-1. Assessment area sampling locations from which walleye were collected in 1996 and 1997 are the Lower Fox River, lower Green Bay, western Green Bay, eastern Green Bay, and upper Green Bay (1996 only). These locations were chosen to correspond to zones sampled in the Green Bay Mass Balance Study (Connolly et al., 1992). In 1997, sampling was expanded to include nearby Patten Lake and Lake Winnebago (which is upstream of the assessment area) as reference areas; these reference areas had lower concentrations of PCBs than assessment area sampling locations, but had walleye of similar age as in the assessment area. This sampling procedure was designed to enable comparison between PCB-contaminated fish from Green Bay and uncontaminated fish from similar habitats.

Walleye collected in 1996 and 1997 were evaluated for liver lesions and for several biochemical, physiological, histological, and fish health measurements, including ethoxyresorufin-O-deethylase (EROD) activity, immunological evaluation of kidney and blood samples, measurement of plasma vitellogenin, and examination of tissues for bacterial, viral, and parasitic infections. This suite of tests was designed to evaluate important fish health parameters that can be adversely affected by PCB exposure, as described in Chapter 4.

Figure 6-1
1996-1997 Walleye Collection Locations



Each parameter is described in greater detail below. Walleye samples from 1997 were not yet analyzed for PCB concentrations at the time when the Barron et al. (1999) report was released. The results of the PCB analyses of the 1997 walleye samples are presented in Section 6.3.

Tissue PCB Concentrations

The results of the 1996 PCB analyses in walleye tissue are displayed in Table 6-1. Mean concentrations of PCBs in whole body samples were elevated at all assessment area sampling locations, ranging from 4.6 μg/g (wet weight) in western Green Bay to 8.6 μg/g in eastern Green Bay. Mean concentrations of PCBs in liver samples were similar to whole body concentrations, ranging from 4.1 μg/g in western Green Bay to 7.9 μg/g in eastern Green Bay. PCB congener patterns from walleye collected in 1996 were similar across all assessment area sampling sites, with the coplanar PCBs, including PCB 126 and PCB 77, detected in fish from all assessment areas. The congener pattern in Lower Fox River walleye showed a greater proportion of lower chlorinated congeners than at other assessment area sampling locations. This trend of decreasing proportion of lower chlorinated congeners from the river to the bay is consistent with the Lower Fox River paper companies being the predominant source of PCBs to the river and the bay, with PCBs weathering as they are transported to the bay (Stratus Consulting Inc., 1999d).

Liver Histopathological Lesions

Hepatic foci of cellular alteration (FCA) and hepatic tumors (HT) represent early and late stages, respectively, of the progression of cancer in the liver. FCA are preneoplastic lesions that may develop into tumors, and their presence indicates the initiation of tumor formation. HT can include both malignant or benign tumors.

As reported in Barron et al. (1999), 5-8 year old fish were used to compare lesions between assessment area and reference area walleye because this range of ages overlapped between fish collected at assessment area sampling locations and reference areas. The results of this comparison are summarized here. Table 6-2 shows prevalence of lesions and mean number of lesions per liver sample, by area and by sex. There was a statistically significant ($p = 0.004$ for both sexes combined; $p = 0.003$ for female fish only) elevation of FCA and HT prevalence in assessment area fish over reference area fish. Twenty-six percent of all assessment area fish had at least one FCA or HT, compared to 5.9% of reference area fish. Thirty-four percent of assessment area females had at least one FCA or HT, compared to 6.9% of reference area females.

Immunological Responses

Immunological assessment of walleye collected in 1997 was conducted to identify the potential for immune function impairment in assessment area fish relative to reference area fish. The assessment involved measurement of several blood indices, including hematocrit (red blood

Table 6-1
Mean and Standard Deviation (SD) of
Total PCB Concentrations in Walleye Collected in 1996

Sample Location	Tissue	n	Total PCBs	
			μg/g ww	µg/g Lipid
Lower Fox River	whole[a]	7	6.0 (2.2)	47.5 (13.7)
	liver[b]	1	4.9	32.3
Lower Green Bay	whole[a]	6	5.7 (2.9)	34.0 (15.0)
	liver	4	4.9 (2.2)	37.4 (7.1)
Eastern Green Bay	whole[a]	11	8.6 (3.6)	52.9 (21.8)
	liver	4	7.9 (2.5)	57.4 (17.0)
Western Green Bay	whole[a]	4	4.6 (0.6)	30.8 (6.6)
	liver	4	4.1 (2.5)	28.2 (6.8)
Upper Green Bay	whole[a]	3	5.8 (1.3)	33.3 (6.6)
	liver	4	4.4 (1.4)	32.4 (9.5)

a. Size weighted whole body composites of three to six fish; n is the number of separate sample analyses.
b. Composite of whole livers from four fish; n is the number of separate sample analyses.

Source: Barron et al., 1999.

cell volume), leukocrit (white blood cell volume), and blood differential counts (percentage of white blood cell types). Red blood cells are responsible for transporting oxygen through the body, and white blood cells function as defense against disease. In addition, kidney cells from walleye were used to measure T-cell lymphoproliferation (the production of a type of white blood cell that helps to identify and destroy cells in the body that have become infected by a foreign agent), superoxide anion production, and phagocytosis (the ability of the fish's immune system to attack and consume invading particles).

Table 6-2
Foci of Cellular Alteration and Hepatic Tumors in Male and Female Walleye
(5 to 8 years old; 1996 and 1997 data combined)

Location	Sex	Average Weight (kg)[a]	n	Prevalence: Number of Fish with Lesions (%)				Mean Lesions per Liver Sample[b]	
				FCA and/or HT	FCA	HT	No FCA or HT	FCA	HT
Assessment Area	Male	1.29	23	3 (13%)	3 (13%)	0 (0%)	20 (87%)	2	— [e]
	Female	1.62	41	14 (34%)[c]	11 (27%)[c]	7 (17%)[c]	27 (66%)	> 4.6	2
	Combined	1.48	66[d]	17 (26%)[c]	14 (21%)[c]	7 (11%)[c]	49 (74%)	> 4.1	2
Reference Area	Male	0.81	5	0 (0%)	0 (0%)	0 (0%)	5 (100%)	— [e]	— [e]
	Female	0.86	29	2 (6.9%)	2 (6.9%)	0 (0%)	27 (93%)	1	— [e]
	Combined	0.85	34	2 (5.9%)	2 (5.9%)	0 (0%)	32 (94%)	1	— [e]

a. Computed using available data (weights not recorded on all fish).
b. Only includes fish with an FCA or HT.
c. Significantly different prevalence compared to reference area fish ($p < 0.05$; t-test).
d. Includes fish of undetermined sex.
e. Not applicable (no FCA or HT).

Source: Barron et al. (1999).

No remarkable effects on immunological parameters were found in contaminated walleye, although several statistically significant trends were evident (Barron et al., 1999). In comparing assessment area fish to reference area fish, significant elevations were observed in assessment area hematocrit (red blood cell volume) and in lymphoproliferation of kidney T-cells ($p = 0.007$ and $p = 0.002$, respectively) (Barron et al., 1999).

A significant ($p = 0.04$) reduction was found in number of monocytes (another type of white blood cell that ingests invading particles in the body's tissues). Barron et al. (1999) speculated that this reduction in cells responsible for defense against infectious agents could, by weakening the immune system, contribute to the greater incidence of gill parasites detected in assessment area fish (described below). However, alternative causes (e.g., differences in parasite densities in the waterbodies) are also plausible.

Health Screening

Walleye collected in 1997 were assessed for the incidence and severity of viral, bacterial, and parasitic infections (Barron et al., 1999). No viruses were detected in any samples. A large number of isolates of an unidentified yeast and one identified mold were recovered from assessment area walleye. Assessment area fish also had a high prevalence of the gill parasite *E. luciopercarum*. However, intestinal parasites and bacterial infections were similar in fish collected from assessment area sampling locations and reference areas, and no fish had overt signs of disease.

Hepatic EROD Activity

EROD is an enzyme that is involved in detoxifying pollutants in the body; EROD activity typically is induced when PCBs enter the body. Although fish exposed to PCBs generally exhibit EROD induction, Barron et al. (1999) reported that EROD activity was similar between walleye from assessment area sampling locations and walleye from reference areas, even though PCB concentrations were higher in assessment area fish. As discussed in Barron et al. (1999), laboratory studies have not been conducted to determine the susceptibility of walleye to EROD induction, so it is not known whether walleye normally exhibit EROD induction in response to PCBs. It is also possible that PCBs inhibit EROD activity, that EROD activity is altered during fish capture procedures, or that fish chronically exposed to elevated contaminant levels have a reduced responsiveness toward EROD induction (Barron et al., 1999).

Plasma Vitellogenin

Plasma vitellogenin is a protein produced by females that is used in the formation of eggs. Normally, males do not produce vitellogenin. Plasma vitellogenin levels have been used as an indicator of PCB effects on the endocrine systems of fish (Folmar et al., 1996; Harries et al., 1996). PCBs are potentially endocrine disrupting chemicals and may have estrogenic or anti-estrogenic effects. Plasma vitellogenin was not detected in male fish but was

elevated in all female walleye from eastern Green Bay. Although it is noted in Barron et al. (1999) that this increase in plasma vitellogenin could be the result of a weak estrogenic response to PCBs, it was also noted that any role of PCBs in controlling plasma vitellogenin in male or female walleye has not yet been elucidated.

Walleye Health Assessment Conclusions

A significant increase in the prevalence of liver tumors and pre-tumors was observed in assessment area walleye, with the prevalence being greater in female fish. Assessment area fish also had significantly higher PCB concentrations. Other fish health responses measured (immune system effects, biochemical changes, disease, endocrine system effects) were not notably different in the assessment area.

6.3 SUPPLEMENTAL LIVER PCB ANALYSIS

The walleye livers collected in 1997 reported on by Barron et al. (1999) for fish health indices were analyzed for PCBs subsequent to the release of that report. The samples were analyzed for PCB concentration (as Aroclors) and lipid content. The analytical data were validated by a third party validator and then supplied to the Service (B. Olsiewski, EcoChem, Inc., pers. comm., 1999). Table 6-3 shows liver PCB concentrations for 1997 walleye by location. Mean total PCB concentration in assessment area fish was significantly elevated over reference area fish ($p < 0.001$; t-test), with mean total PCB concentration of 4.35 μg/g across all assessment area fish compared to 0.460 μg/g across all reference area fish. Liver PCB concentrations for individual walleye from 1997 are provided in Appendix A.

6.4 INTEGRATION OF BARRON ET AL. (1999) DATA AND SUPPLEMENTAL PCB ANALYSIS

This section combines the findings presented in Barron et al. (1999) with the supplemental PCB concentration data presented in Section 6.3. Mean total PCB concentrations for all 1996 and 1997 walleye liver samples combined were elevated at assessment area sampling locations compared to reference area locations (Table 6-4) ($p < 0.001$; t-test). Mean total PCB concentration across all assessment area sampling locations was 4.56 μg/g (sd = 2.62), compared to 0.460 μg/g (sd = 0.60) in reference areas. The range of total PCB concentration in the assessment area was 0.857-12.9 μg/g, compared to 0.0-2.22 μg/g in reference areas.

As discussed in Barron et al. (1999), PCBs, even at low concentrations, are known to induce cellular changes, and dietary exposure to PCBs has been documented to increase hepatic tumors in several fish species. Barron et al. (1999) notes that environmental exposure to PCBs has been associated with increased tumor frequencies and other histological lesions in fish, and the lesions observed in walleye are consistent with exposure to carcinogens or tumor promoters. However,

Table 6-3
Mean and Standard Deviation (SD) of Total PCB Concentrations in Walleye Livers Collected in Assessment and Reference Areas in 1997

Sample Location	n	Mean Total PCBs as μg/g wet weight (SD)
Lower Fox River	19	6.42 (1.67)
Lower Green Bay	12	4.07 (3.81)
Eastern Green Bay	17	2.58 (1.36)
Western Green Bay	14	3.92 (1.97)
Upper Green Bay	NS[a]	NS[a]
All assessment area sampling locations	62	4.35 (2.51)
Lake Winnebago	12	0.938 (0.548)
Patten Lake	13	0.0186 (0.0670)
All reference area sampling locations	25	0.460 (0.599)

a. Not sampled.

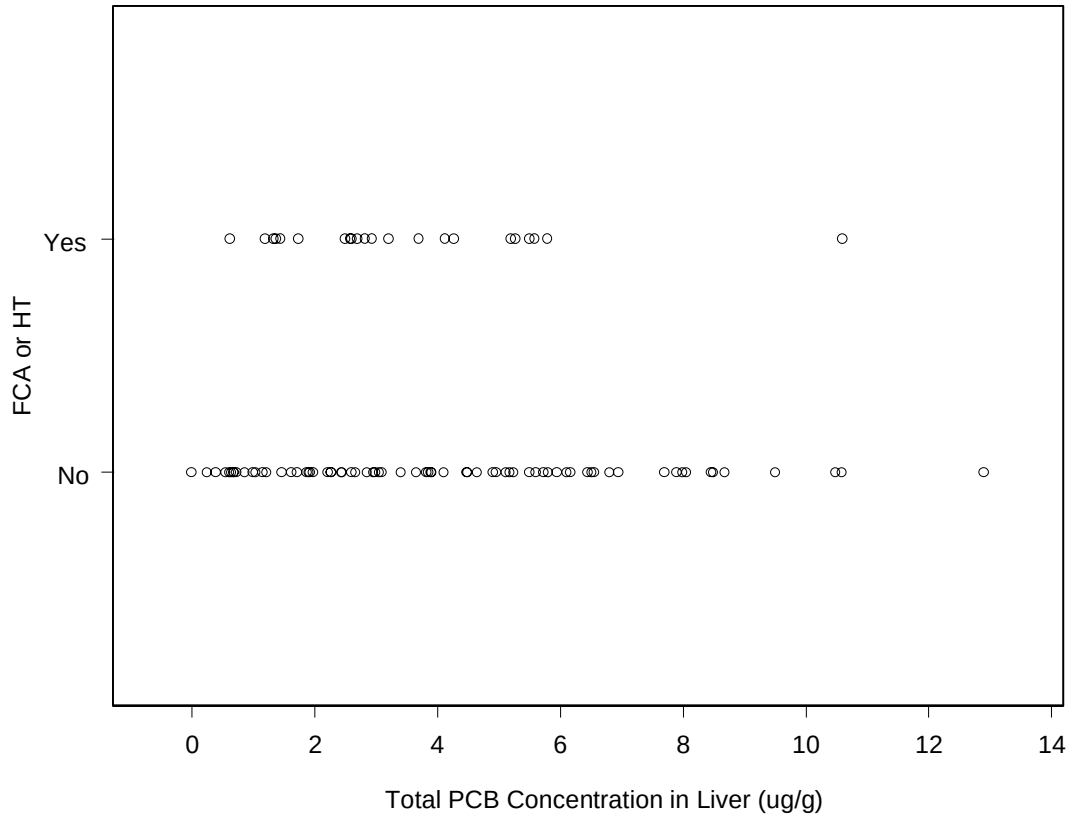
Source: B. Olsiewski, Ecochem, Inc., pers. comm., 1999.

Table 6-4
Mean and Standard Deviation (SD) of Total PCB Concentrations in Walleye Livers Collected in Assessment and Reference Areas in 1996 and 1997 Combined

Sample Location	n	Mean Total PCBs as μg/g wet weight (SD)
Lower Fox River	20	6.35 (1.70)
Lower Green Bay	16	4.29 (3.43)
Eastern Green Bay	21	3.60 (2.65)
Western Green Bay	18	3.96 (2.03)
Upper Green Bay	4	4.40 (1.38)
All assessment area sampling locations	79	4.56 (2.62)
Lake Winnebago	12	0.938 (0.548)
Patten Lake	13	0.0186 (0.067)
All reference area sampling locations	25	0.460 (0.599)

as shown in Figure 6-2, PCB concentration was not correlated with incidence of FCA and/or HT at the level of the individual fish ($p = 0.90$; t-test). This may be because the incidence of FCA and HT is too low in the population to be correlated to PCBs given the sample size, because of non-linearity in dose-response, or because of the confounding influence of unmeasured tumor promoters. Nonetheless, comparison on an area-by-area basis shows that the increased incidence of pre-tumors and tumors in assessment area walleye is associated with elevated exposure of these walleye to PCBs.

Figure 6-2
Individual Fish Liver PCB Concentrations in Fish with and without FCA or HT



6.5 INJURY DETERMINATION

6.5.1 Injury Definition and Measurements

Injuries to walleye were assessed according to the definitions of injury in the Department's NRDA regulations at 43 CFR Part 11. Specifically, injuries were assessed according to the following injury definition:

> the concentration of the released hazardous substance is sufficient to cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction), or physical deformations [43 CFR § 11.62(f)(1)(i)].

Measurements of the following biological responses were used to determine adverse viability injuries to walleye:

- to evaluate cancer injuries: fish neoplasia in livers
- to evaluate disease injuries: viral, bacterial, and pathogenic infections; immune system status (as indicated by various blood and kidney parameters)
- to evaluate physiological malfunctions: endocrine system impairment (as indicated by plasma vitellogenin); biochemical changes (as indicated by liver EROD activity); immune system status (as indicated by various blood and kidney parameters)
- to evaluate physical deformation: histopathological lesions.

As described in Chapter 4, these injury measures meet the criteria in the Department's regulations for biological responses used to determine injuries at 43 CFR § 11.62 (f)(2). The injury determination included an evaluation of whether the biological responses were statistically significantly different between walleye from the Lower Fox River/Green Bay area and walleye from reference areas (Lake Winnebago and Patten Lake), consistent with the Department's regulations [43 CFR §11.62(f)(3)].

6.5.2 Injury Determination

Table 6-5 summarizes the results of the injury determination for walleye. Of the injury endpoints and measurements assessed, only the incidence of walleye liver tumors and pre-tumors was statistically significantly higher in assessment area walleye compared to reference areas (Lake Winnebago and Patten Lake). Of the assessment area fish aged 5-8 years, 26% had liver tumors or pre-tumors compared with 6% of reference area fish. The difference was more dramatic in

Table 6-5
Summary of Injury Determination Results for Walleye

Injury Category [43 CFR §11.62(f)(4)]	Biological Response Measured	Response Meets Department's Criteria?	Statistically Significant Difference between Assessment and Reference Areas?
Cancer	Fish neoplasia (liver tumors and pre-tumors in adult fish)	Yes [43 CFR § 11.62(f)(4)(iv)(E)]	Yes
Disease	Incidence and severity of viral, bacterial, and parasitic infections	Yes (see Section 4.2.2)	No
Physiological malfunction	Immune system impairment (various blood and kidney indices of immune system functioning)	Yes (see Section 4.2.2)	No[a]
	Endocrine system impairment (plasma vitellogenin)	Yes (see Section 4.2.2)	No
	Biochemical changes (liver EROD activity)	Yes (see Section 4.2.2)	No
Physical deformation	Histopathological lesions (not including liver tumors and pre-tumors)	Yes [43 CFR § 11.62(f)(4)(vi)(D)].	No

a. Although several individual blood parameters were significantly different, it was concluded that these differences did not indicate overall immune system impairment.

females, with 34% of assessment area fish having liver tumors or pre-tumors versus 7% of reference area fish. Assessment area fish also had statistically significantly higher concentrations of PCBs in the liver. The other injury endpoints and measurements do not show consistent differences between the assessment area and reference area. Therefore, it is concluded that walleye have not suffered disease or physiological malfunction injuries.

Although the causal link between PCB exposure and liver tumor formation in assessment area walleye is not conclusively established by these data, it is well documented that PCBs promote or enhance liver tumor formation (Hendricks et al., 1990). For example, rainbow trout that were exposed to PCBs showed a significantly higher incidence of liver tumors induced by aflatoxin B_1 (a tumor initiator) than trout exposed to aflatoxin B_1 alone (Hendricks et al., 1981). The liver is generally the predominant site for fish tumors or pre-tumors that are initiated or promoted by contaminants (Baumann, 1992a; Baumann, 1992b). Higher incidences of liver tumors in fish

exposed to contaminants, including PCBs, have been demonstrated in both field and laboratory studies (Teh et al., 1997a).

A statistically significant difference in fish neoplasia between the assessment and reference areas is specified in the Department's regulations as constituting injury to fish:

> Injury has occurred when a statistically significant difference can be measured in the frequency of occurrence of the fish neoplasia when comparing population samples from the assessment area and a control area [43 CFR §11.62(4)(iv)(A)].

Therefore, the higher incidence of liver tumors and pre-tumors in assessment area walleye constitutes an injury according to the Department's regulations at 43 CFR Part 11.

CHAPTER 7
REPRODUCTIVE INJURIES TO LAKE TROUT

7.1 INTRODUCTION

This chapter presents an evaluation of injuries to lake trout (*Salvelinus namaycush*) in Green Bay and northern Lake Michigan resulting from PCB exposure. Lake trout are the native top predator in the aquatic ecosystem of Lake Michigan, and have provided valuable ecological, cultural, recreational fishing, and commercial fishing services. Lake trout populations were decimated in the 1940s following the invasion of the sea lamprey into the Great Lakes. Despite subsequent control of sea lamprey populations, extensive stocking efforts and limits on fishing have failed to result in a naturally reproducing lake trout population in Lake Michigan or Green Bay.

Environmental contaminants, including PCBs, have been implicated in the failure of lake trout to reproduce naturally in Lake Michigan (Willford et al., 1981). Lake trout are sensitive to PCB toxicity, and studies have linked lake trout reproductive failure in Lake Michigan with PCB exposure (Mac et al., 1993). Therefore, the Service conducted this injury assessment on lake trout.

This chapter is organized as follows:

- Section 7.2 describes the lake trout resource of Green Bay and Lake Michigan, including historical population trends and efforts to re-establish naturally reproducing populations.
- Section 7.3 describes PCB toxicity to early life stages of lake trout, the life stages that have been found to be most sensitive to PCB toxicity.
- Section 7.4 summarizes historical studies of the possible link between PCBs and reproductive failure of lake trout in Lake Michigan.
- Section 7.5 describes the methods and results of studies conducted by the United States Geological Survey (USGS) in 1996-1998 on reproductive failure of Lake Michigan lake trout.
- Section 7.6 describes the methods and results of supplemental NRDA and USGS studies in 1996-1998 on the possible interaction of PCB toxicity with thiamine deficiency in lake trout.

- Section 7.7 models PCB concentrations in Lake Michigan lake trout eggs in the past, and compares the model concentrations to injury thresholds.

- Section 7.8 summarizes the available information and presents the determination of injury to lake trout.

As described in Section 7.8, based on the information and analyses presented in this chapter, the Trustees conclude that there is indication that PCBs caused lake trout reproductive failure in the 1970s. However, given that evidence suggests little to no reproductive effects of PCBs since 1980 and that other factors appear to be substantially more important to the survival of lake trout fry, the Trustees determine that current data do not support the conclusion that lake trout in Green Bay and Lake Michigan are injured by the PCBs released from Fox River paper companies.

7.2 DESCRIPTION OF THE LAKE TROUT RESOURCE

Lake trout in Green Bay and Lake Michigan represent a nationally significant resource. The national significance of lake trout in the Great Lakes, the largest area of contiguous fresh waters in the world, results from its prominence as the keystone native predator in all five Great Lakes. Because of its prominence in the endemic Great Lakes fish communities, lake trout are considered to be a surrogate of ecosystem health for the basin (Edwards et al., 1990). The significance of the lake trout resource is reflected in the extensive state, federal, and international efforts over the years to maintain and restore the lake trout, as described below.

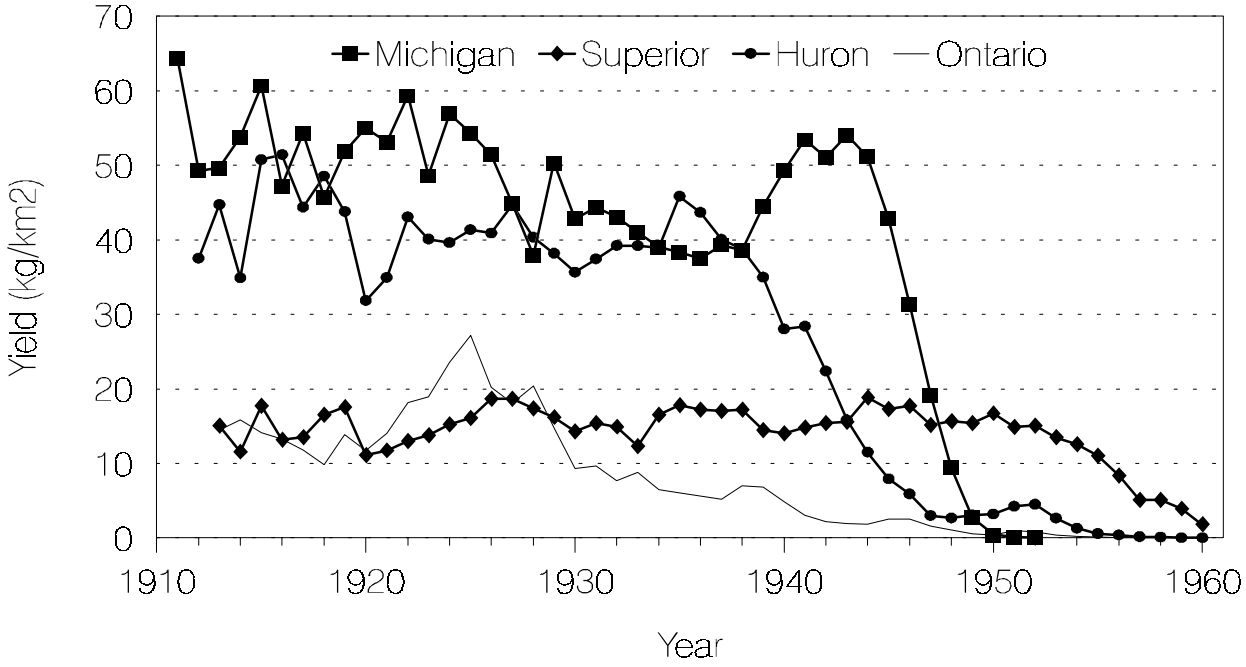
Lake trout are a large, long-lived species, with life spans of up to 20 years (Burnham-Curtis and Bronte, 1996; Schram and Fabrizio, 1998). Lake trout prefer cool water, about 10°C, and therefore spend most of their lives in the deeper waters of lakes (Scott and Grossman, 1973; Becker, 1983). Lake trout prey primarily on aquatic invertebrates and forage fish, with diets varying by seasonal abundance of dietary items (Becker, 1983). Three distinct forms or strains of lake trout were present in Lake Michigan in the early 1900s and probably persisted until the extirpation of the native lake trout in the mid-1950s (Brown et al., 1981). One of the forms, described as the “salmon” or “bay trout,” was unique to Green Bay. Bay trout were small, lean, and very red-fleshed and spawned later than other forms.

An understanding of the early life stages of lake trout is important in an evaluation of the effects of PCB toxicity on lake trout reproduction. Lake trout spawn during the fall from mid-October to early December, when females deposit their eggs on rocky substrate on the lake bottom (Martin and Oliver, 1980; Marsden, 1995). During egg formation in the female, large amounts of yolk protein and lipids are produced in the liver and translocated to the developing eggs (Tyler et al., 1990). It is during this stage when contaminants in the maternal fish, including PCBs, can be transferred to and deposited in the developing eggs (Walker et al., 1994). Newly hatched salmonid larva are termed “sac fry,” in reference to the yolk sac that serves as the primary source

of nutrition to the developing larvae (Heming and Buddington, 1988). Approximately one month after hatching, the yolk sac is completely absorbed and the larva begins exogenous feeding (Becker, 1983). This stage is termed the "swim-up" stage. The egg, sac fry, and swim-up development stages have been the focus of much of the work on PCB toxicity to lake trout, as they are more sensitive to PCB toxic effects than adults (U.S. EPA, 1993a).

Historically, Lake Michigan consistently produced more native lake trout per surface area and sustained higher annual catches than any of the other Great Lakes (Figure 7-1). The lake trout was an important sustenance species for native Americans (Kinietz, 1940) and was the most valuable commercial fish species in the lake from 1890 until the mid-1940s (Wells and Mclain, 1972). During the early 1900s, commercial harvest in the Wisconsin waters of Lake Michigan was 50% of the lakewide harvest (Baldwin et al., 1979).

Figure 7-1
Historical Lake Trout Yield/Area for Each of the Great Lakes

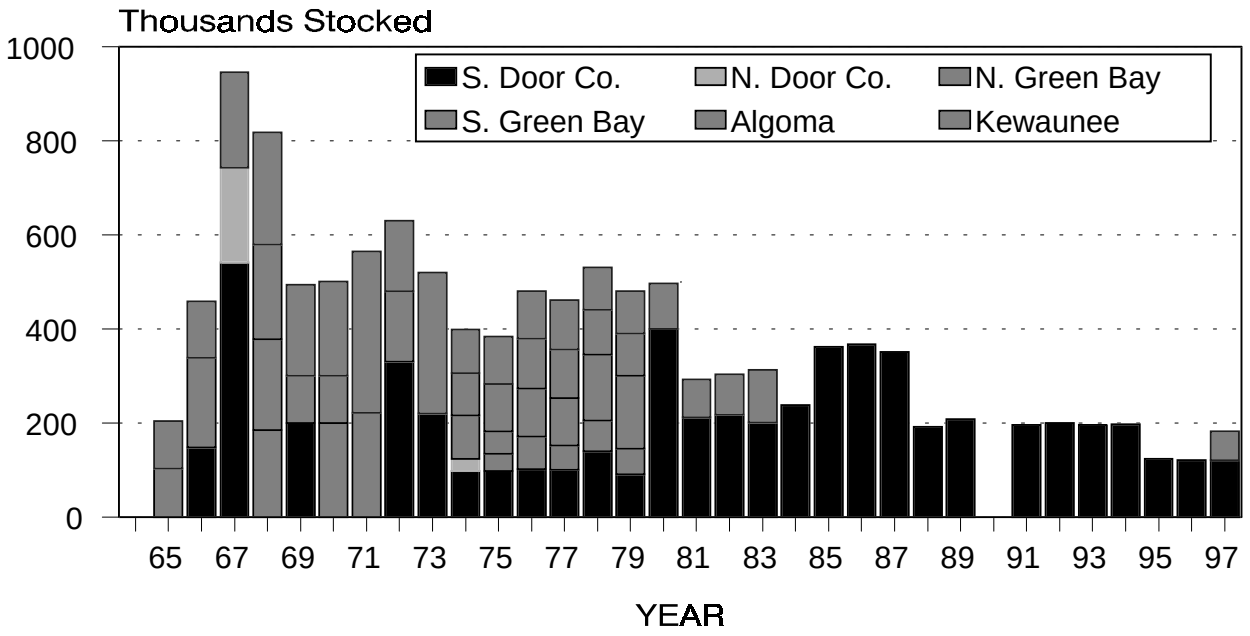


The Lake Michigan and Green Bay lake trout fishery abruptly collapsed in the late 1940s following the invasion of the sea lamprey (Figure 7-1). The last wild lake trout caught in Lake Michigan were recorded from assessment nets fished near the Sheboygan Reef in 1957 (Eschmeyer, 1957).

Several joint national and international initiatives have been enacted to restore Lake Michigan lake trout populations. The primary focus of fishery management efforts has been to increase their

abundance through stocking to a level where natural reproduction can be successful. Lake trout stocking from the Service's national fish hatcheries began in 1965, following successful chemical treatments to control lamprey numbers in Lake Michigan (Smith and Tibbles, 1980; Holey et al., 1995). Figure 7-2 shows the number of lake trout stocked in Green Bay and the Wisconsin waters of Lake Michigan near Green Bay. The peak year of stocking in these areas was 1967, when close to 1 million lake trout were stocked. Lake trout were stocked in Green Bay from 1965 to 1982, but Green Bay stocking was ceased because of poor success. Since lake trout stocking in Green Bay ceased, the only lake trout recruitment to the bay is from movement of lake trout from northern Lake Michigan. Figure 7-2 shows that during the last decade, approximately 200,000 lake trout have been stocked annually in the Wisconsin waters of Lake Michigan.

Figure 7-2
Annual Lake Trout Stocking in Green Bay and Wisconsin Waters of Lake Michigan near Green Bay



In 1985, a lakewide plan for lake trout restoration in Lake Michigan was implemented by fishery agencies (LMLTTC, 1985; Holey et al., 1995). Major features of the 1985 plan were to focus trout stocking efforts in habitat that historically was the most productive, establish total mortality goals and provide greater protection from exploitation through creation of refuges and regulations to further limit harvest, increase the genetic diversity of the trout stocked by developing new strains of hatchery broodstock, and continue efforts to monitor the impacts of contaminants and sea lamprey mortality on trout survival (Holey et al., 1995).

Despite a density of spawning lake trout in some areas of Lake Michigan that is considered adequate for successful reproduction (Holey et al., 1995; Selgeby et al., 1995), the lake trout population remains unable to sustain itself because of low natural reproduction. A number of causes for this low reproduction have been cited, including over-exploitation, insufficient numbers of lake trout stocked, poor egg survival due to contaminants, lack of genetic diversity in the strains of lake trout stocked, inadequate homing ability in hatchery lake trout, stocking of fish in poor habitat, and continued mortality due to sea lamprey predation (LMLTTC, 1985; Holey et al., 1995). Other possible factors include predation on lake trout fry by alewife (Jones et al., 1995; Krueger et al., 1995b) and early mortality syndrome (EMS), also known as swim-up syndrome, thought to be caused by thiamine deficiencies in lake trout eggs (McDonald et al., 1998).

Given these possible causal factors, the Service recognizes that contaminants alone, including PCBs, are not solely responsible for the failure of Green Bay and Lake Michigan lake trout to reproduce naturally. However, given the sensitivity of lake trout to PCB toxicity, as part of the NRDA the Service evaluated the potential for PCBs to be a contributing factor in the reproductive failure of Green Bay and Lake Michigan lake trout.

7.3 PCB TOXICITY TO LAKE TROUT

This section provides an overview of PCB toxicity to lake trout, focusing on early life stage toxicity that occurs to developing embryos and fry, since this life stage is the most sensitive to PCB toxicity (Peterson et al., 1993; Cook et al., 1993; Walker and Peterson, 1994a; U.S. EPA, 1995; ASTDR 1996; Eisler and Belisle, 1996). Many of the toxic effects of PCBs on developing lake trout are produced by specific coplanar PCB congeners that have a structure similar to that of 2,3,7,8-tetrachloro-*p*-dibenzodioxin (TCDD) and cause toxicity through a similar mechanism, as discussed below. Selected polychlorinated dibenzofuran (PCDF) and polychlorinated dibenzodioxin (PCDD) congeners also cause the same toxic effects through the same mechanism. Much of the laboratory and field work on the toxicity of these compounds has been conducted using TCDD as the model compound, since TCDD is the most potent of these compounds in causing the effects. Therefore, much of our discussion of the toxicity of the coplanar PCBs is based on work conducted with TCDD.

This section presents a summary of the toxic effects caused by coplanar PCBs to developing lake trout, a description of the TCDD toxicity equivalence approach to expressing PCB doses in terms of equivalent TCDD doses, and a discussion of TCDD injury thresholds.

7.3.1 Description of Early Life Stage Toxicity of Coplanar PCBs and TCDD

The toxic effects of coplanar PCBs, PCDFs, and PCDDs are considered to occur by the same mechanism, referred to as aryl hydrocarbon receptor (AhR)-mediated toxicity (Safe, 1990, 1994;

Whitlock, 1990, 1993). The AhR is a cellular protein that binds to TCDD and the TCDD-like PCBs, PCDDs, and PCDFs. The binding of the TCDD-like compounds with AhR initiates a series of events within the cell that eventually lead to the range of biological responses produced by the compounds (Sutter et al., 1991; Whitlock, 1993).

The cardiovascular system is a key site of action for TCDD-like chemicals (Spitsbergen et al., 1991; Mizell et al., 1996; Guiney et al., 1997; Henry et al., 1997; Cantrell et al., 1996, 1997). Toxicity in TCDD-exposed lake trout sac fry results from cessation of blood flow. This reduction in blood flow has been observed in the caudal vein and vascular beds of the head, trunk, and gills (Hornung and Peterson, unpublished results; Henry et al., 1997). Subsequent to the reduction in blood flow there is an increase in yolk sac and pericardial edema, development of craniofacial deformities, and failure of the swim bladder to inflate (Harris et al., 1994; Henry et al., 1997). Death usually follows the development of these signs of TCDD toxicity (Spitsbergen et al., 1991). Other signs of TCDD toxicity in developing trout embryos include necrotic lesions in the retina, brain, liver, and spinal cord; lethargy and arrested soft tissue and skeletal development in half-hatched embryos and sac fry; decreased access to yolk sac nutrients, which contributes to reduced growth of the sac fry and to mortality; and hemorrhage (Spitsbergen et al., 1991).

Lake trout embryos exposed to TCDD or coplanar PCBs as fertilized eggs show stage-specific periods of toxicity (Spitsbergen et al., 1991). The most sensitive stage is the sac fry stage, and contaminant effects appear generally from the middle to the end of the sac fry stage. Toxicity beyond the sac fry stage is typically very low at exposure concentrations that cause toxicity to sac fry (U.S. EPA, 1993a). Toxicity also can occur earlier, at the time of egg hatching, although at greater concentrations than cause toxicity to sac fry (Walker et al., 1991). The affected lake trout embryos are typically incompletely hatched, have significant yolk sac edema, and have high mortality rates. Thus, the critical period for early life stage mortality in lake trout is from about one week prior to hatching until the end of the sac fry stage.

The time course and symptoms of TCDD-like toxicity in lake trout sac fry are similar to blue-sac disease in hatchery-reared salmonids (Spitsbergen et al., 1991). The blue-sac disease that occurs in hatcheries is thought to be triggered by physical or chemical stressors such as elevated ammonia, temperature shock, or hypoxia (Wolf, 1969 as cited in Spitsbergen, 1991; Burkhalter and Kaya, 1977; Lasee, 1995). Since the TCDD-like PCBs, PCDDs, and PCDFs cause a symptomology essentially identical to that of blue-sac disease, the toxicity caused by PCBs, PCDDs, and PCDFs in lake trout and other fish species has been termed “blue-sac syndrome” (Walker and Peterson, 1994a).

Blue-sac syndrome is distinct from “swim-up syndrome” (also referred to as “early mortality syndrome,” or EMS), a type of early life stage mortality observed in Great Lakes lake trout that does not appear to be associated with TCDD-like toxicity (Fitzsimons, 1995a; Marcquenski and Brown, 1997). Swim-up syndrome occurs later than blue-sac syndrome, typically just before the fry begin feeding, and has a distinct set of symptoms, including lethargy, loss of equilibrium, anorexia, hyper-excitability, and finally death (Fitzsimons et al., 1995; Marcquenski and Brown,

1997). The distinction between blue-sac syndrome and swim-up syndrome is important in evaluating the evidence for and against the current and recent lake trout reproductive failure of Lake Michigan lake trout being caused by PCBs, and is discussed in subsequent sections of this chapter.

There are wide differences in susceptibility of freshwater fish species to the early life stage mortality caused by TCDD and TCDD-like compounds. Lake trout is the most sensitive fish species tested to date. Other salmonids (e.g., brook and rainbow trout) are also more sensitive than other, nonsalmonid species; brook trout and rainbow trout are about 3-6 times less sensitive than lake trout, whereas the other species are 8-38 times less sensitive (Helder, 1981; Walker et al., 1991; Walker and Peterson, 1994b; Henry et al., 1997; Elonen et al., 1998).

The reason for the greater sensitivity of lake trout to TCDD and TCDD-like PCBs, PCDDs, and PCDFs is not known. Elonen et al. (1998) suggested that the ability of nonsalmonid fish species to tolerate higher egg concentrations of TCDD might be related to their shorter development time. The time from hatch to first feeding ranged from 1 to 18 days for the nonsalmonid species they tested compared to 30-70 days for trout, with lake trout having the longest development time. Comparison of post swim-up TCDD elimination rates between the nonsalmonid species and lake trout suggests that lake trout with a long development time may retain TCDD longer than species with short development times. However, species differences in cellular responses to TCDD may also be involved (Henry et al., 1997).

Using sexually mature female lake trout, Walker et al. (1994) demonstrated that there was not a significant difference in the potency of TCDD in causing lake trout sac fry mortality when exposure of eggs to TCDD occurred via maternal transfer, waterborne exposure, or egg injection. Similarly, the concentration of TCDD in brook trout eggs that resulted in dose-related increases in sac fry mortality was similar following waterborne exposure and maternal transfer (Walker and Peterson, 1994b). Therefore, these studies indicate that it is the egg dose of TCDD that determines toxicity, not the route of exposure.

When Lake Ontario lake trout eggs were exposed to graded doses of TCDD, blue sac syndrome associated mortality was observed at the same doses as for lake trout eggs from Lake Superior or a hatchery, which were much less contaminated than the Lake Ontario eggs (Guiney et al., 1996). The similar TCDD dose related responsiveness of the Lake Ontario lake trout sac fry to TCDD exposure indicated that chronic sub lethal exposure of lake trout to TCDD-like compounds does not provide resistance to toxicity from subsequent exposure to TCDD, as was reported for *Fundulus heteroclitus* collected from a TCDD contaminated bay (Prince and Cooper, 1995).

7.3.2 The TCDD Toxicity Equivalence Approach to Expressing Toxic Doses

The TCDD toxicity equivalence approach has been developed as a way of expressing the dose of TCDD-like PCB, PCDD, and PCDF congeners in terms of an equivalent concentration of TCDD.

Since TCDD and coplanar PCB toxicity occurs through a similar mechanism, a PCB dose can be converted to a TCDD dose that would cause the same level of effects. This approach standardizes the expression of the dose based on the relative toxic "potency" of different chemicals, and allows for comparison of PCB exposure concentrations with the extensive literature on TCDD toxicity to salmonids.

The toxic potencies of the AhR-active PCB, PCDF, and PCDD congeners vary by several orders of magnitude. The PCB congeners that are most potent have chlorine substitutions in the meta- and para- positions that allow them to maintain a planar conformation. These non-ortho-substituted, coplanar PCB congeners include PCB 77, 81, 126 and 169. Mono-ortho-substituted PCB congeners are essentially inactive in causing early life stage mortality in rainbow trout (Walker and Peterson, 1991; Zabel et al., 1995a), although such congeners do show AhR-mediated toxicity in mammals and birds (Van den Berg et al., 1998).

In the toxicity equivalence approach, the toxic potency of each congener is expressed relative to the potency of TCDD, which is recognized as the most potent AhR agonist in vertebrates (Safe, 1994). The relative potency of a congener is termed its toxic equivalency factor, or TEF.[1] TEFs are derived as the ratio of the TCDD concentration causing a specific effect (e.g., mortality to 50% of exposed embryos) to the congener concentration causing the same effect. Zabel et al. (1995a) tested 15 PCB congeners and found that only the coplanar PCBs 77, 81, 126, and 169 caused early life stage mortality to trout and hence have TEFs > 0. While the early life stage toxicity to trout of the remaining 194 PCB congeners has yet to be tested, knowledge of the structure activity relationship for such toxicity between the non-ortho-, mono-ortho, and di-ortho-substituted PCB congeners already tested suggests that the coplanar PCB congeners (e.g., PCB 77, 81, 126, and 169) will be the most significant contributors to TCDD-like developmental toxicity (Zabel et al., 1995a; Walker et al., 1996; Cook et al., 1997).

TEFs are used to convert congener concentrations measured in an environmental sample to equivalent TCDD concentrations by multiplying the measured congener concentration by its respective TEF. For a mixture of PCBs or other TCDD-like compounds, the TCDD equivalents of each congener are summed to calculate a TCDD concentration that has equivalent toxic potency to the mixture of PCBs present. In this way, mixtures of PCBs and other AhR-active compounds can be expressed in terms of TCDD toxic equivalents, or TEQ. Specifically,

1. The term TEF has been used to refer to consensus estimates of the potency of an individual PCB, PCDD, or PCDF congener relative to TCDD, as determined by a panel of international experts chosen by the World Health Organization (Van den Berg, 1998). The term relative potency (REP) refers to the potency of an individual PCB, PCDD, or PCDF congener relative to TCDD for a specific toxic endpoint in a single study (Van den Berg, 1998). Throughout this report we use the term TEF to refer to both REPs and TEFs to avoid confusion with the historical literature and to reflect the fact that fish TEFs are based largely on REPs for trout early life stage mortality (Van den Berg, 1998).

$$TEQ = \sum_{i=1}^{n} [C_i \cdot TEF_i]$$

where:

TEQ	=	total TEQ from all AhR-active compounds
C_i	=	concentration of AhR-active compound *i* in the sample
TEF_i	=	TEF of compound *i*.

There are two major assumptions in applying the TCDD toxicity equivalence approach to predicting the toxicity of PCBs and related compounds. The first is that congeners interact additively to produce AhR-mediated toxicity. Hence, toxicity may be predicted by summation of the TEQs calculated for each TCDD-like AhR agonist present in fish tissue. This additivity assumption has been validated for salmonid early life stage toxicity (Zabel et al., 1995b; Hornung et al., 1996; Walker et al., 1996).

The second major assumption in applying the TCDD toxicity equivalence approach is that the toxicity of PCBs, PCDDs, and PCDFs is solely related to their TCDD-like toxicity. This assumption generally holds for predicting lethality in salmonid embryos and larvae (Zabel et al., 1995a; Cook et al., 1997). However, other toxic effects of PCBs observed in mammals, such as carcinogenesis, neurotoxicity, or endocrine disruption, are, for certain mono-ortho- and di-ortho-substituted PCB congeners, not AhR-mediated (Safe, 1994). Thus, non-AhR-mediated toxicity attributable to PCBs is not assessed by the TCDD toxicity equivalence approach.

7.3.3 Thresholds for Early Life Stage Toxicity to Lake Trout

Several detailed laboratory investigations have been conducted on the toxicity of TCDD to developing lake trout from which toxicity thresholds can be derived. Table 7-1 lists egg LD50 values for TCDD-caused mortality to lake trout sac fry determined in different studies. An LD50 value is the concentration of TCDD in lake trout eggs at which 50% of the sac fry die from TCDD exposure. The LD50 values shown in Table 7-1 range from 42 to 80 pg/g egg. The range of LD50 values is probably a reflection in part of the steep dose-response curve for TCDD-caused mortality in lake trout sac fry, which makes determining an LD50 value with precision difficult (as also reflected in the wide 95% fiducial limits shown in Table 7-1). Other reasons for the variability may include differences in lake trout strains tested, differences in dosing and handling procedures across laboratories, and analytical and experimental variation.

We used the geometric mean of the values shown in Table 7-1, 61.2 pg/g egg, as a central estimate of the LD50 values published in the literature. Given the range of LD50s shown in Table 7-1, this mean LD50 may underestimate or overestimate the actual LD50 concentration for Green Bay and Lake Michigan lake trout. For example, studies have suggested that lake trout eggs with low thiamine levels, such as occur in Lake Michigan, may be more susceptible to TCDD toxicity than those with high thiamine levels (such as those commonly used in laboratory

Table 7-1
TCDD LD50s for Lake Trout Embryomortality

Source	TCDD LD50 (95% fiducial limits) (pg/g egg)	Notes
Walker et al. (1991)	65 (not reported)	Waterborne egg exposure
Walker et al. (1996)	74 (70-80)	Waterborne egg exposure
Walker et al. (1994)	58 (36-90)	Waterborne egg exposure
	69 (64-75)	Egg exposure via maternal transfer
	80 (68-91)	Egg injection
Guiney et al. (1996)	69 (58-80)	Waterborne egg exposure
	44 (36-52)	Waterborne egg exposure
	65 (60-71)	Waterborne egg exposure
	53 (41-55)	Waterborne egg exposure
	72 (65-78)	Waterborne egg exposure
	57 (50-61)	Waterborne egg exposure
	42 (33-52)	Waterborne egg exposure
Geometric Mean	61.2	

toxicity tests) (see Section 7.6). In this case, using the mean LD50 value of 61.2 pg/g egg could underestimate embryomortality caused by TCDD-like compounds. Nevertheless, the geometric mean of the published LD50s from the laboratory toxicity studies was used as a best estimate for use in comparison to exposure concentrations.

We also used the results of the published TCDD toxicity tests on lake trout sac fry to obtain a central estimate of an LD10, which is the concentration that causes mortality to 10% of the exposed population. Only two of the published papers listed in Table 7-1 provide information sufficient to determine LD10s: Walker et al. (1991) provide a graph from which we visually estimated an LD10 of 35 pg/g egg, and Walker et al. (1996) report an LD10 of 55 pg/g egg. Using the LD50 values that accompany these two values, we estimated an average dose-response curve by plotting the points on log concentration/probit scales and taking the average slope as the average dose-response relationship. Using this average slope, we then estimated an LD10 for each of the LD50 values shown in Table 7-1, and used the geometric mean of these LD10 values as the central estimate. The geometric mean of the LD10 values corresponding to the LD50 values shown in Table 7-1 is 40.9 pg/g egg.

Using the toxicity equivalence approach, these LD50 and LD10 concentrations can be compared to PCB congener concentrations measured in Lake Michigan lake trout eggs to evaluate the potential for PCBs to cause lake trout embryomortality. This comparison is shown in the following sections for both recent and historical lake trout PCB TEQs.

Several researchers have observed sublethal effects on trout fry at TCDD concentrations below those that cause mortality. The studies conducted by the USGS and reported in Section 7.6 suggest the occurrence of craniofacial deformities, hemorrhaging, and yolk sac edema at TCDD concentrations below those that caused lethality. Although quantitative dose-response curves could not be established from the studies, the data indicate that TCDD exposure can cause gross deformities at concentrations below those that are lethal. Spitsbergen et al. (1991) found that at an egg injection dose of 40 pg/g egg lake trout, which did not cause an increase in sac fry mortality, 30% of the sac fry experienced mild yolk sac edema, a rate higher than in control fry. The authors reported that most of the sac fry with mild yolk sac edema survived through the sac fry stage and showed no gross lesions at the time of swim-up. Other sublethal effects of TCDD include behavioral alteration and reduced growth in rainbow trout (Helder, 1981; Merhle et al., 1988) and reduced growth in pike (Helder, 1980). However, available data are not sufficient to develop quantitative dose-response relationships between TCDD exposure and sublethal effects in lake trout. It is also not known to what extent the sublethal effects, such as reduced growth and behavioral alterations, could adversely affect fry survival in the wild. Therefore, in this injury assessment the sublethal effects of TCDD are treated only qualitatively.

7.4 PREVIOUS STUDIES ON FACTORS ASSOCIATED WITH LAKE MICHIGAN LAKE TROUT EMBRYOMORTALITY

As noted previously, EMS, or swim-up syndrome, has been identified as a potentially important contributing factor to the overall reproductive failure of lake trout (Mac, 1988). Most studies on the causes of EMS have focused on the potential role of contaminants, including PCBs, on egg survival, egg hatchability, and fry mortality. More recent work has focused on thiamine deficiency, possibly in conjunction with contaminants such as PCBs, as a possible factor in causing the observed swim-up syndrome. This section describes and summarizes the results and conclusions of studies that have investigated the possible causes of the reduced fry and egg viability that has been and continues to be observed in Lake Michigan lake trout.

7.4.1 Contaminant Exposure

The Service began conducting studies on the potential relationship between Lake Michigan lake trout egg and fry viability and egg contaminant concentrations in the early 1970s (Table 7-2). In 1972 and 1975, Mac et al. (1981) compared the survivability of lake trout eggs from northeastern and southeastern Lake Michigan with the survivability of hatchery eggs when reared under hatchery conditions. The mortality of eggs and sac fry was monitored, but the tests were terminated prior to fry swim-up. No relationship was found between egg or sac fry mortality and egg PCB or DDE concentration.

Table 7-2
Investigations of the Relationship Between Contaminants and Egg and Fry Viability of Lake Michigan Lake Trout

Study	Egg Sampling		Endpoint Assessed	Results Summary
	Year	Location		
Mac et al. (1981)	1972 and 1975	L. MI; hatchery	Egg and fry mortality	▸ No relationship between PCBs and egg or fry mortality.
Stauffer (1979)	1973-1974 and 1974-1975	L. MI; hatchery	Egg and fry mortality	▸ Higher egg mortality in L. MI eggs than hatchery eggs. ▸ No linear correlation of egg or fry mortality with PCBs or DDT.
Berlin (1981)	1975	L. MI	Fry mortality	▸ Higher mortality in fry exposed to PCBs in water and food than control fry. ▸ Water and food exposure concentrations similar to L. MI conditions increased mortality over controls.
Mac et al. (1985)	1980	L. MI; L. Huron; L. Superior; hatchery	Egg and fry mortality	▸ Higher mortality of L. MI eggs than of hatchery, L. Superior, or L. Huron eggs. ▸ Higher mortality of fry from L. MI than from other sources; mortality associated with swim-up syndrome. ▸ Higher contaminant concentrations in L. MI eggs and fry than from other sources (no correlational analysis conducted).
Mac and Bergstedt (1981)	1975	L. MI	Fry temperature preference	▸Fry exposed to PCBs at 25x L. MI concentrations in water and food preferred cooler temperatures than control fry.
Rottiers and Bergstedt (1981)	1975	L. MI	Fry swimming performance	▸ No effects of fry exposure to PCBs in water and food on swimming performance.
Mac (1981)	1975	L. MI	Fry vulnerability to predation	▸ No effects of fry exposure to PCBs in water and food on vulnerability to predation.
Mac et al. (1993)	1979-1988	L. MI; L. Superior; L. Huron; L. Ontario	Egg and fry mortality; blue sac syndrome; swim-up syndrome	▸ Significant correlation between egg mortality and PCB concentration in eggs. ▸ Significant correlation between blue-sac syndrome and PCB concentration in eggs. ▸ No relationship between swim-up syndrome and PCB concentration in eggs. ▸ Blue-sac syndrome a small component of overall fry mortality.

In a related study conducted by the Michigan DNR from 1973 to 1976, Stauffer (1979) studied the mortality of hatchery and Lake Michigan eggs and fry reared under hatchery conditions. Fry were observed through six weeks after swim-up. A significantly higher percentage of Lake Michigan eggs than hatchery eggs died during incubation, although the egg mortality was not linearly correlated with egg PCB concentrations. Stauffer (1979) surmised that the higher mortality of Lake Michigan eggs could be caused by factors such as differences in egg fertilization, egg handling, or incubation. There was no significant difference in mortality between hatchery and Lake Michigan fry, and the observed variation in fry mortality was not linearly correlated to egg PCB concentrations.

The Service expanded its research in 1975-1976 to test the effects of chronic exposure of Lake Michigan lake trout fry to contaminants in their food and water (Willford et al., 1981). In these studies, newly hatched fry from eggs of Lake Michigan lake trout were exposed for six months to PCB and/or DDE concentrations in food and water similar to those in Lake Michigan (1x treatment) and to concentrations about 5 and 25 times greater (5x and 25x treatments, respectively). Control treatments were fry from the same Lake Michigan egg source exposed only to hatchery background levels of PCBs and/or DDE in food and water. Endpoints assessed for these studies included fry survival and growth (Berlin et al., 1981), temperature preference (Mac and Bergstedt, 1981), swimming performance (Rottiers and Bergstedt, 1981), and vulnerability to predation (Mac, 1981).

Berlin et al. (1981) found that fry exposed to the 1x, 5x, and 25x PCB treatments all had higher mortality (46.4%, 36.3%, and 56.8%, respectively) than control fry (21.7%). Since the 1x treatment represented food and water concentrations similar to ambient conditions in Lake Michigan, these results demonstrate that exposure to environmental PCBs caused increased fry mortality. However, fry mortality did not follow a monotonic dose-response relationship with PCBs, as the 1x treatment had higher mortality (46.4%) than the 5x treatment (36.3%). Nevertheless, this study led researchers to conclude that PCBs were responsible in part for reduced lake trout fry survival in Lake Michigan in the mid-1970s (Willford et al., 1981).

Of the other endpoints assessed in fry in these 1975 studies (temperature preference, swimming performance, and vulnerability to predation), only temperature preference was adversely affected (Willford et al., 1981). Mac and Bergstedt (1981) found that exposed fry, including fry exposed to the 1x treatment, exhibited a lowering of the preferred temperature, with frequency distributions of residence temperatures significantly different among all treatments. They concluded that such changes in the preferred temperature could reduce the energetic efficiency of a fish and thereby reduce growth and survival in fish exposed to PCBs.

In 1980, Mac et al. (1985) measured the survival of lake trout eggs and fry from four different sources reared under different environmental conditions. Eggs and fry collected from Lake Michigan, Lake Huron, Lake Superior, and from hatchery brood stock were incubated and raised in water from each of the three sites and in well water. Mac et al. (1985) found that Lake Michigan egg and fry mortality was higher than for eggs and fry from the other sources,

regardless of rearing conditions. The symptoms associated with the fry mortality (e.g., loss of equilibrium, swimming in a corkscrew fashion, lying on their sides, and emaciation) are consistent with swim-up syndrome and not blue-sac syndrome (Fitzsimons et al., 1995; Marcquenski and Brown, 1997). PCB concentrations were highest in Lake Michigan eggs, although potential correlations between PCB concentrations and egg or fry mortality were not evaluated.

A multiyear study by Mac et al. (1993) provides the strongest evidence for an association between PCBs and reduced reproductive success of Lake Michigan lake trout. Eggs were collected from lake trout throughout the Great Lakes for multiple years from 1979 to 1988, and egg mortality, fry mortality, and incidence of blue-sac and swim-up syndromes in Lake Michigan lake trout were compared with those from Lake Superior, Lake Huron, and Lake Ontario. PCB concentrations (as PCB congeners) were measured in the egg samples.

Mac et al. (1993) detected several linear correlations between egg PCB concentration and biological parameters. The strongest correlation was between total egg mortality and total PCB concentration in the eggs ($r = 0.701$, $p < 0.001$, $N = 23$). A relationship was also found between egg PCB concentration and blue-sac disease, although the extent of blue-sac disease (0.7% to 5.8%) was low. No relationship was found between overall fry mortality or swim-up syndrome and PCBs.

The results of Mac et al. (1993), who used the most comprehensive dataset of any of the historical studies of Lake Michigan lake trout egg and fry mortality in relation to PCBs, are important for several reasons. The finding of a positive correlation between egg PCB concentrations and blue-sac disease in sac fry is consistent with the symptoms of blue-sac disease being very similar to those caused by PCB toxicity (as described in Section 7.3). Thus, the blue sac disease reported by Mac et al. (1993) could very likely have actually been the toxic effects of PCBs on sac fry. The incidence of blue-sac disease in the fry was relatively low (0.7% to 5.8%), and much lower than the incidence of swim-up syndrome, which is not known to be caused by PCB toxicity. The minor contribution of blue-sac disease relative to swim-up syndrome in causing the observed overall fry mortality is reflected in the fact that overall fry mortality was not correlated with PCBs, even though blue-sac disease was. The studies of Stauffer (1979) and Mac et al. (1981), discussed earlier, did not differentiate between blue-sac disease and swim-up syndrome, and would not have been able to detect a low incidence of sac fry mortality caused by PCBs.

Another important finding of the Mac et al. (1993) study is that egg mortality was strongly correlated with egg PCB concentration. This finding is not consistent with the higher tolerance of lake trout eggs to AhR-mediated PCB toxicity compared with sac fry (as discussed in Section 7.3), since the incidence of PCB-caused sac fry mortality (as manifested in blue-sac disease) appears to be low. Mac et al. (1985) and Stauffer (1979) both found higher egg mortality of Lake Michigan eggs compared with eggs from other sources (hatchery, Lake Superior, or Lake

Huron), although Stauffer did not see a correlation between egg mortality and egg PCB concentration as Mac et al. (1993) did.

There are several possible explanations for the egg mortality/PCB relationship observed by Mac et al. (1993). One is that the observed correlation is spurious, for example if the egg mortality is caused by some other factor with which PCBs happened to be correlated. The higher incidence of mortality in Lake Michigan eggs versus eggs from other sources suggests that some property inherent to Lake Michigan eggs other than PCBs may have made them more susceptible to mortality. The higher mortality of Lake Michigan eggs could have been related to experimental design, for example, if Lake Michigan eggs had to be transported farther or under different conditions to the hatchery for rearing than eggs from other sources.

Another possible explanation for the egg mortality/PCB relationship observed by Mac et al. (1993) is that the egg stage is more susceptible to environmental contaminants, including PCBs, than the sac fry stage under the exposure conditions actually experienced by lake trout in Lake Michigan. Most of the laboratory tests documenting the lower sensitivity of the egg stage have focused on the AhR-active congeners of the PCBs, PCDDs, and PCDFs, whereas Lake Michigan lake trout are exposed to a mixture of congeners and other compounds.

In conclusion, although the results of historical studies on the relationship between egg and fry viability and PCB contamination in Lake Michigan lake trout are mixed, the studies provide some evidence that PCBs have caused fry mortality (as blue sac disease) in the past. The relationship between egg mortality and PCBs in Lake Michigan is less clear, with some studies indicating a PCB effect in the past and others not. The historical studies also provide evidence that fry reared in Lake Michigan PCB exposure conditions (in water and food) experienced increased mortality.

7.4.2 Thiamine Deficiency

Recent work by several investigators has suggested a link between swim-up syndrome and thiamine deficiency in Lake Michigan lake trout. This relatively recent hypothesis has not undergone as much testing as the possible role of contaminant and so relatively few data exist. However, the existing evidence points to a strong link between swim-up syndrome and thiamine deficiency.

The inability to link swim-up syndrome mortality with contaminant burdens led Fitzsimons (1995b) to study an alternative hypothesis that the syndrome is the result of nutritional factors. Fitzsimons (1995b) tested the ameliorative effects of a number of B-vitamins (thiamine, riboflavin, folic acid, nicotinic acid, pyridoxine hydrochloride), administered by either water immersion or injection, on swim-up syndrome using eggs collected from Lake Ontario female lake trout between 1990 and 1993. Fitzsimons (1995b) found that thiamine, but none of the other vitamins, was effective both in reversal and prevention of the clinical signs and mortality associated with the syndrome. Injection of sac fry with thiamine before swim-up resulted in a significant improvement

in 1-month post swim-up survival relative to controls, with average mortality in the thiamine-treated group at 9.4% compared to 75.8% mortality in the control group. Recovery of fry affected with the symptoms of the syndrome was assessed by immersing the afflicted fry in a thiamine solution. After 30 days of immersion, mean cumulative mortality in the thiamine solution group, at 42.2%, was significantly lower than in the control group, where 100% mortality was observed (Fitzsimons, 1995b).

Similar syndromes have been reported for Atlantic salmon from the Finger Lakes in New York (called Cayuga syndrome) and from the Baltic Sea (called M74) (McDonald et al., 1998). These mortality syndromes all affect fry with very low thiamine levels, and sac fry mortality resulting from these syndromes has been shown to be dramatically reduced by therapeutic thiamine treatments of eggs or sac fry (McDonald et al., 1998). Immersion of eggs and fry in thiamine is now routine practice at hatcheries that use feral broodstock to stock salmon in Lake Michigan and the Baltic Sea (McDonald et al., 1998).

Low thiamine levels in the eggs of Lake Michigan lake trout may be related to the diet of the maternal fish. The primary food source for adult lake trout in Lake Michigan is alewife (Madenjian et al., 1998), which contains thiaminase, a group of enzymes capable of destroying thiamine (Fitzsimons, 1995b). It has been hypothesized that the presence of the non-native alewive in the Lake Michigan food chain has resulted in low thiamine levels in lake trout eggs, which has resulted in swim-up syndrome and fry mortality (Fitzsimons, 1995b).

However, there may be interactions, not detected by previous investigations, between low thiamine levels and contaminants that make fish more susceptible to thiamine deficiency and/or the toxic effects of TCDD-like compounds. For example, Wright et al. (1998) found that treatment of rainbow trout and Japanese medaka eggs with thiamine made them less sensitive to TCDD toxicity. Similarly, the results of supplemental NRDA studies described in Section 7.6 provide evidence that lake trout eggs low in thiamine are more susceptible to the toxic effects of TCDD and PCB 126 than are eggs high in thiamine. In addition, swim-up syndrome is more prevalent in contaminated systems such as Lake Michigan and Lake Ontario, and it has been suggested that contaminants may increase the thiamine requirements of early life stages (McDonald et al., 1998). For example, contaminants such as PCBs and DDT are known to reduce thiamine levels in laboratory rats and may be an important factor when thiamine concentrations are very low (McDonald et al., 1998). Further research is required before possible interactions of contaminants such as PCBs with thiamine deficiency can be excluded (McDonald et al., 1998).

7.5 USGS STUDIES ON LAKE MICHIGAN LAKE TROUT REPRODUCTION: 1996-1998

The USGS carried out studies in 1996 through 1998 to investigate further the possible relationships between egg contaminant concentrations, egg thiamine levels, and egg/fry viability in

Lake Michigan lake trout. These studies provide useful information for assessing current injuries to Lake Michigan lake trout resulting from PCB exposure.

7.5.1 Methods

From 1996 through 1998, eggs were collected each fall from spawning female lake trout from western Lake Michigan near Sturgeon Bay, Wisconsin (near Green Bay)[2]. A total of 73 lots of eggs from individual spawning females were collected, with 13 lots collected in 1996, 28 in 1997, and 32 in 1998. Eggs were fertilized with pooled milt of up to six males collected near Sturgeon Bay at the same time as the eggs. Each female's lot of eggs was divided into subsamples for monitoring of egg and fry viability and measurement of thiamine, PCBs, PCDDs, and PCDFs.

Samples for the egg and fry mortality studies were transported on ice to the Great Lakes Science Center in Ann Arbor via car or overnight mail shipment. Each container of eggs was transferred to a Heath incubator tray that received a continuous flow of laboratory well water.[3] Water temperatures were recorded for each tray daily. Three times a week dead eggs were counted and removed. For the first 13 days, dead eggs were discarded; beginning on day 14, dead eggs were put in Stockard's solution to determine fertilization status. After all the remaining live eggs had hatched, subsamples of normal fry from each female were transferred to separate 38 L glass tanks. Normal and abnormal fry that were not transferred were counted to determine hatching rate, and abnormal fry were removed and euthanized. Tanks were checked daily for air flow, water flow and temperature, and dead fry.

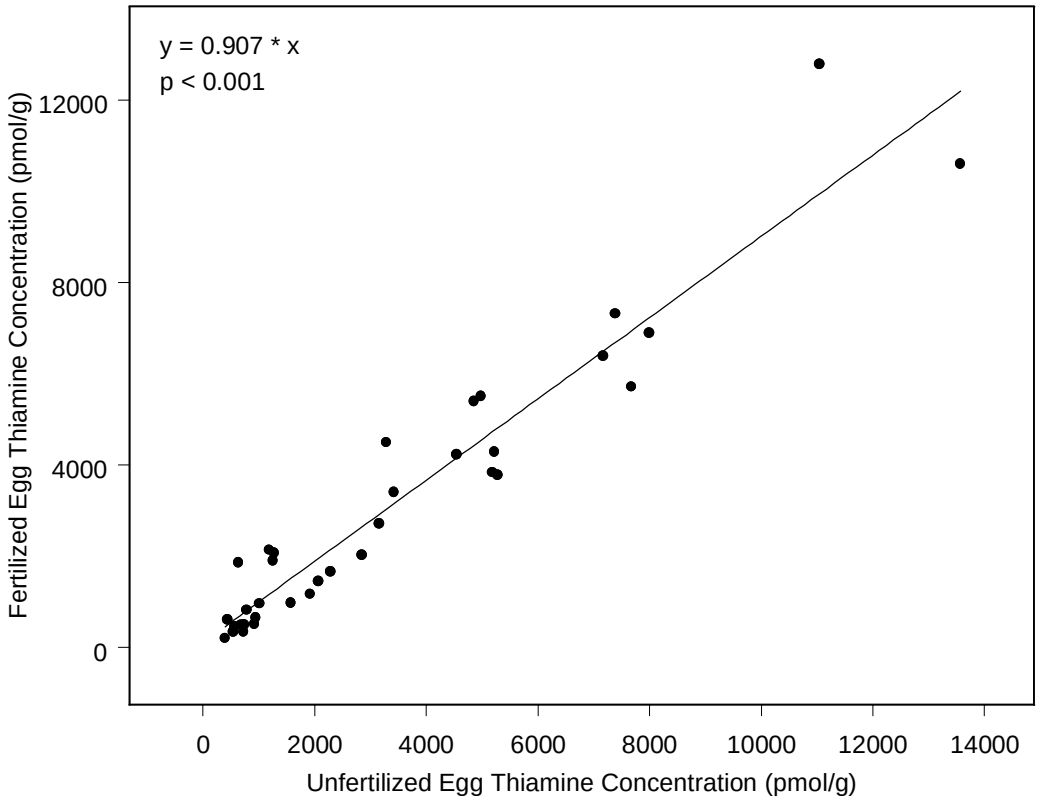
Subsamples designated for thiamine concentration analysis were sent to the USGS Research and Development Laboratory (Leetown Science Center) in Wellsboro, Pennsylvania. Methods for the thiamine analysis are given in Brown et al. (1998).[4] Thiamine analysis was performed on fertilized eggs from all 12 lots in 1996, and 3 of the lots were also analyzed for thiamine in unfertilized eggs. In 1997, all 28 egg lots were analyzed for thiamine concentration in unfertilized eggs only, and in 1998, both fertilized and unfertilized eggs in all 32 lots were analyzed. From these results, a linear regression was fit to predict thiamine concentrations in unfertilized eggs from fertilized egg concentrations ($p<0.001$, $r^2= 0.9615$) (Figure 7-3).Thiamine concentrations were thereafter expressed as thiamine concentration in unfertilized eggs.

2. Field sampling procedures, field notebooks, and field measurements are documented in Hagler Bailly Services (1997b) and Green Bay Fishery Resources Office (1999a; 1999b). These documents are available at the Service's reading room in the Green Bay Field Office.

3. Full details on egg and fry rearing method are described in forthcoming USGS reports on the studies.

4. Full documentation and complete results are given in Holey and Honeyfield (1999), available in the Service's reading room in the Green Bay Field Office.

Figure 7-3
Regression for Estimating Thiamine Concentrations in Fertilized versus Unfertilized Eggs



Subsamples designated for contaminant analysis were sent to Battelle in Duxbury, Massachusetts where they were analyzed for concentrations of PCBs as Aroclors or as individual congeners (including coplanar congeners on selected samples). Selected samples were also analyzed for PCDD and PCDF congeners. Analytical laboratory documentation is provided in Battelle Ocean Sciences (1997b; 1997a) and Battelle (1999) (available in the Service's reading room in the Green Bay Field Office).

The TEF approach was used to express contaminant concentrations in terms of TCDD equivalent concentrations, or TEQs. TEFs used for this analysis are given in Table 7-4. Since only a subset of the eggs was analyzed for the coplanar congeners 77, 81, 126, and 169, a regression was fit between total PCB concentration and TEQ from PCBs in these samples (n = 10) and applied to the rest of the samples to predict PCB TEQ for samples with total PCB measurements only. The results of the regression between total PCB concentration and PCB TEQ were significant ($p<0.001$; $r^2= 0.80$) (Figure 7-4). For samples analyzed for PCDD and PCDF congeners, no

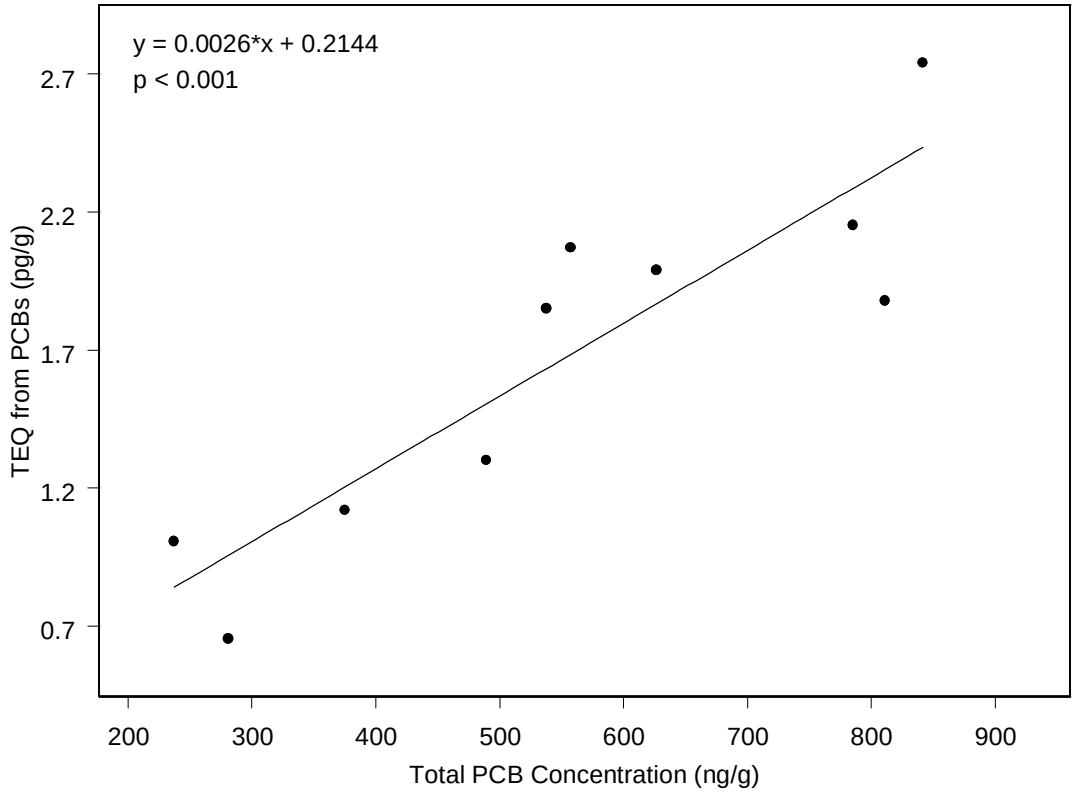
Table 7-4
Toxic Equivalency Factors for PCDD, PCDF, and PCB Congeners

Class	Congener	TEF
PCDDs	2,3,7,8-TCDD	1.0[a]
	1,2,3,7,8-PCDD	0.659[a]
	1,2,3,4,7,8-HxCDD	0.263[a]
	1,2,3,6,7,8-HxCDD	0.020[b]
	1,2,3,7,8,9-HxCDD	0.01[c]
	1,2,3,4,6,7,8-HpCDD	0.0015[b]
	OCDD	-
PCDFs	2,3,7,8-TCDF	0.030[a]
	1,2,3,7,8-PeCDF	0.032[a]
	2,3,4,7,8-PeCDF	0.339[a]
	1,2,3,4,7,8-HxCDF	0.240[a]
	1,2,3,6,7,8-HxCDF	0.1[c]
	1,2,3,7,8,9-HxCDF	0.1[c]
	2,3,4,6,7,8-HxCDF	0.1[c]
	1,2,3,4,6,7,8-HpCDF	0.01[c]
	1,2,3,4,7,8,9-HpCDF	0.01[c]
	OCDF	0.0008[d]
PCBs	77	0.00018[a]
	81	0.00062[b]
	126	0.0049[a]
	169	0.00004[b]

a. Original source: Zabel et al. (1995), adjusted to mass equivalence ratios from molar equivalence ratios by Cook et al. (1997).
b. Original source: Walker and Peterson (1991), adjusted to mass equivalence ratios from molar equivalence ratios by Cook et al. (1997).
c. World Health Organization consensus TEF for fish, as reported by Van den Berg et al. (1998)
d. From description in Van den Berg et al. (1998).

relationship was found between total PCB concentration and the TEQ calculated from the PCDD and PCDF congener measurements. Therefore, total TEQs (i.e., TEQs based on PCB, PCDD, and PCDF congeners) could be calculated only for those samples in which PCDD and PCDF congeners were measured (n = 20) and could not be predicted for those samples in which only PCBs were measured.

Figure 7-4
Regression for Estimating TEQ Contributed by PCBs from Total PCB Concentration



We performed Pearson and Spearman correlation tests to determine whether any of the measured egg and fry rearing endpoints were correlated with egg thiamine concentration, total PCB concentration, or total TEQ (for only those samples measured for PCDD and PCDF congeners). Endpoints assessed were the percentages of eggs that died during incubation (egg mortality), fry that died during rearing (fry mortality), eggs unhatched, abnormal fry hatched, and normal fry hatched. Hatching rates were calculated both as percentages of the total number of eggs and as percentages of the number of fertilized eggs. All correlation tests were conducted after an angular transformation of the percent response data (Snedecor and Cochran, 1980).

7.5.2 Results

Figure 7-5 shows each egg or fry viability endpoint plotted against egg thiamine concentration, Figure 7-6 shows each endpoint against egg total PCB concentration, and Figure 7-7 shows each endpoint against egg total TEQ concentration (from PCBs, PCDFs, and PCDDs).[5] Only one relationship was significant ($p < 0.05$ in both Spearman and Pearson correlation tests): egg thiamine concentration versus fry mortality (the upper left hand graph in Figure 7-6). This relationship is shown in more detail in Figure 7-8, which shows that egg batches with less than approximately 1000 pmol thiamine/g egg had high fry mortality, whereas egg batches with more than this concentration had low mortality. The 1000 pmol/g concentration equates to a mass-based concentration of approximately 300 ng thiamine (as thiamine hydrochloride)/g egg, which is consistent with the dose-response curve reported by Fisher et al. (1996) for thiamine deficiency-induced swim-up syndrome in lake trout eggs. Thiamine concentrations were not correlated with any other endpoint, and PCB and TEQ concentrations were not correlated with any of the endpoints.

To investigate whether low levels of thiamine appeared to make eggs or fry more susceptible to PCB or TCDD-like toxicity, we conducted two different analyses: (1) tests for correlation between contaminants and egg/fry viability for only those eggs with low thiamine content (less than 1000 pmol/g); and (2) partial correlation tests with thiamine as the partial variable to determine if PCBs or TEQs explained the variation in the data remaining after the thiamine correlation is accounted for.

The egg and fry viability endpoints are plotted against PCB and total TEQ concentrations for eggs with less than 1000 pmol/g thiamine in Figure 7-9 and Figure 7-10, respectively. Egg total PCB concentrations and total TEQs were not significantly correlated with any of the egg or fry viability endpoints in only those eggs with less than 1000 pmol/g ($p > 0.05$, Pearson and Spearman correlation tests). Similarly, the partial correlation tests revealed no significant relationship between either egg total PCB or TEQ concentration and any of the egg/fry viability measurements after thiamine effects were partialed out.

7.5.3 Conclusions

The results of the 1996-1998 USGS studies provide clear evidence that thiamine deficiency, rather than PCBs or other TCDD-like compounds, currently is the primary causal factor for the fry mortality in Lake Michigan lake trout. In addition, there is no evidence in the data that PCBs or total TEQs explain any of the variability in the fry mortality data. Other viability measures (egg mortality, fry abnormalities, unhatched eggs) are not explained by egg thiamine, PCB, or TCDD-like compound concentrations.

5. Full tables of results are included in Appendix B.

Figure 7-5
Thiamine Concentration in Lake Trout Eggs vs Measures of Egg and Fry Viability

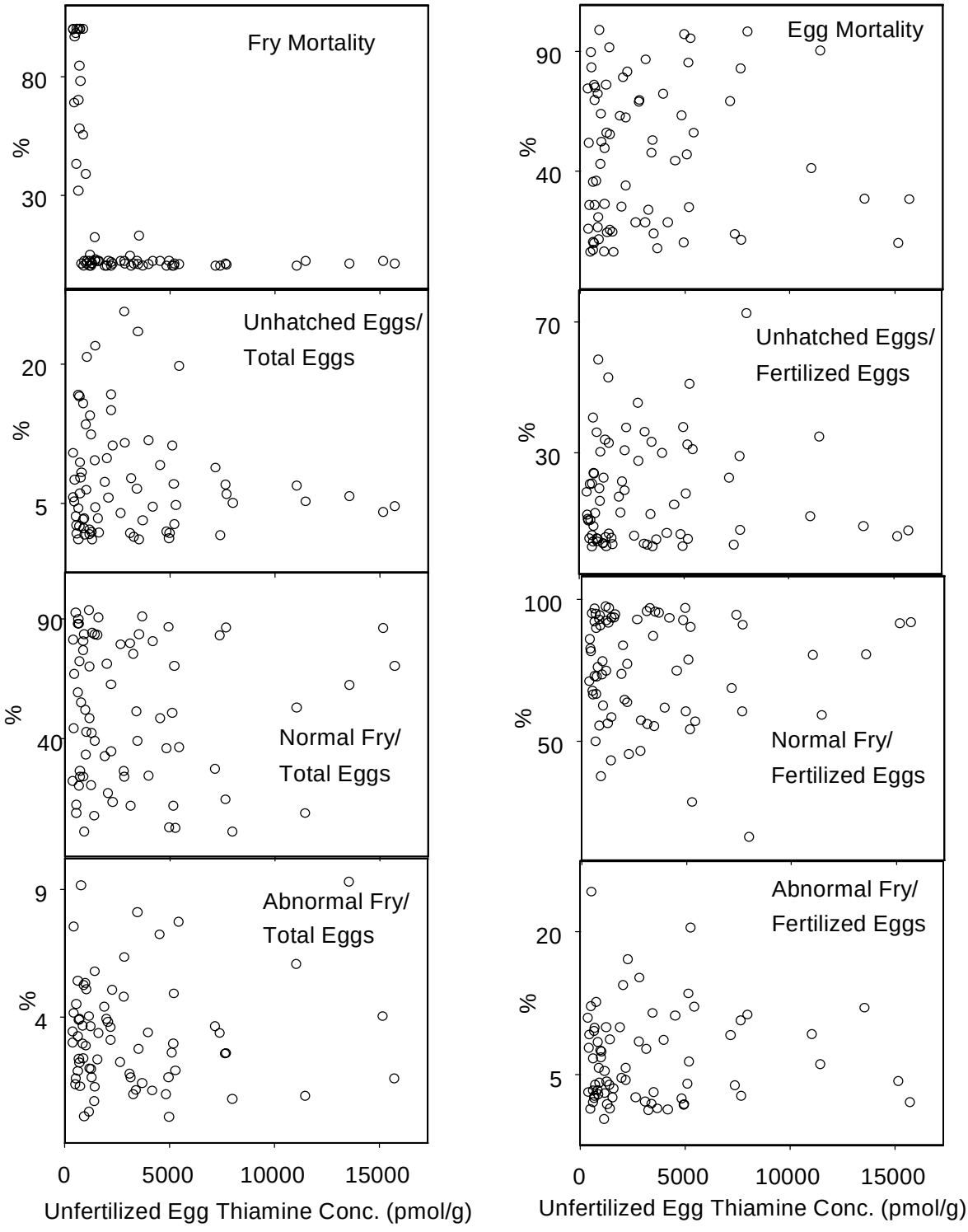


Figure 7-6
PCB Concentration in Lake Trout Eggs vs Measures of Egg and Fry Viability

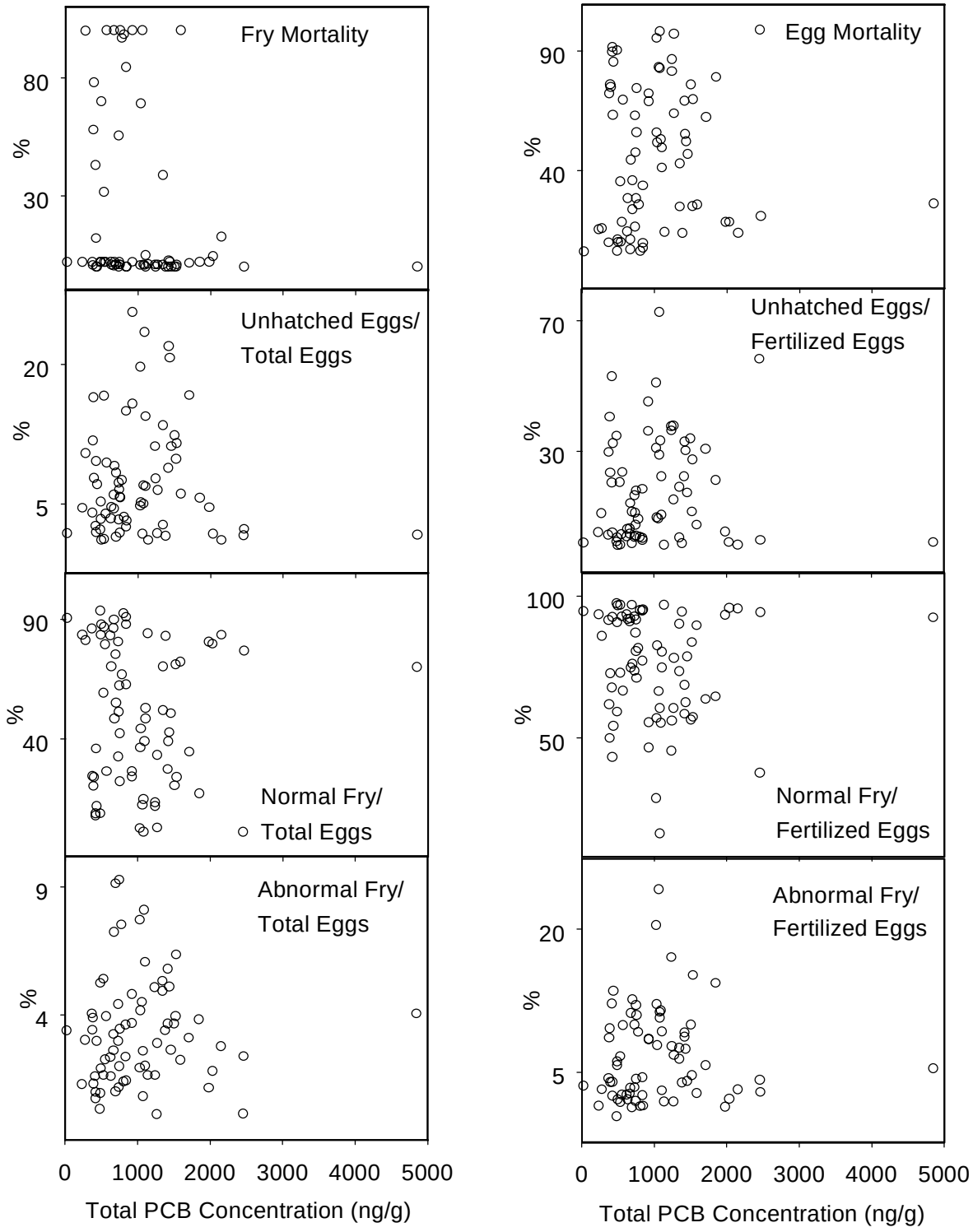


Figure 7-7
Total TEQ Concentration (from PCBs, PCDDs, and PCDFs) in Lake Trout Eggs vs Measures of Egg and Fry Viability

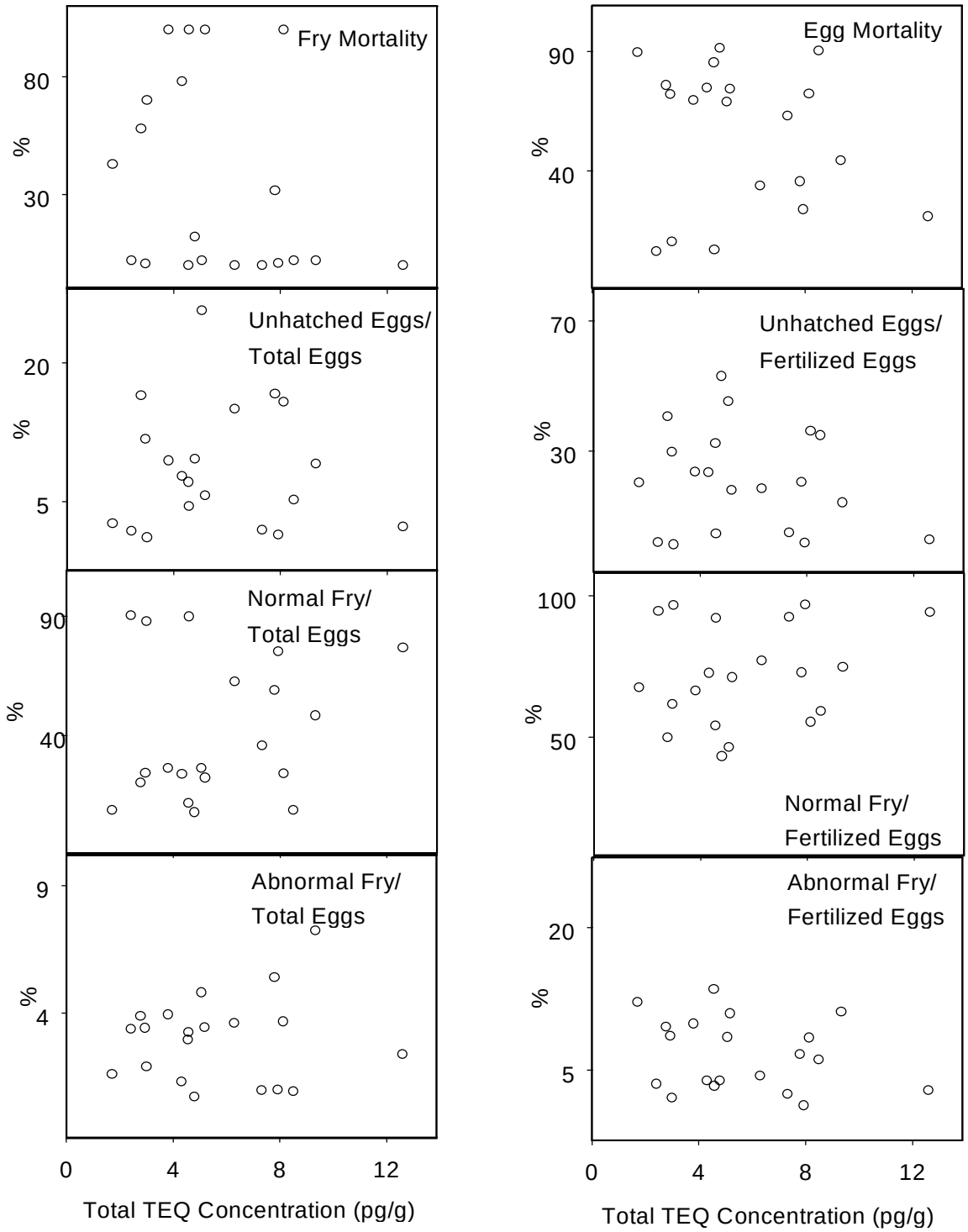


Figure 7-8
Thiamine Concentration in Lake Trout Eggs vs Fry Mortality

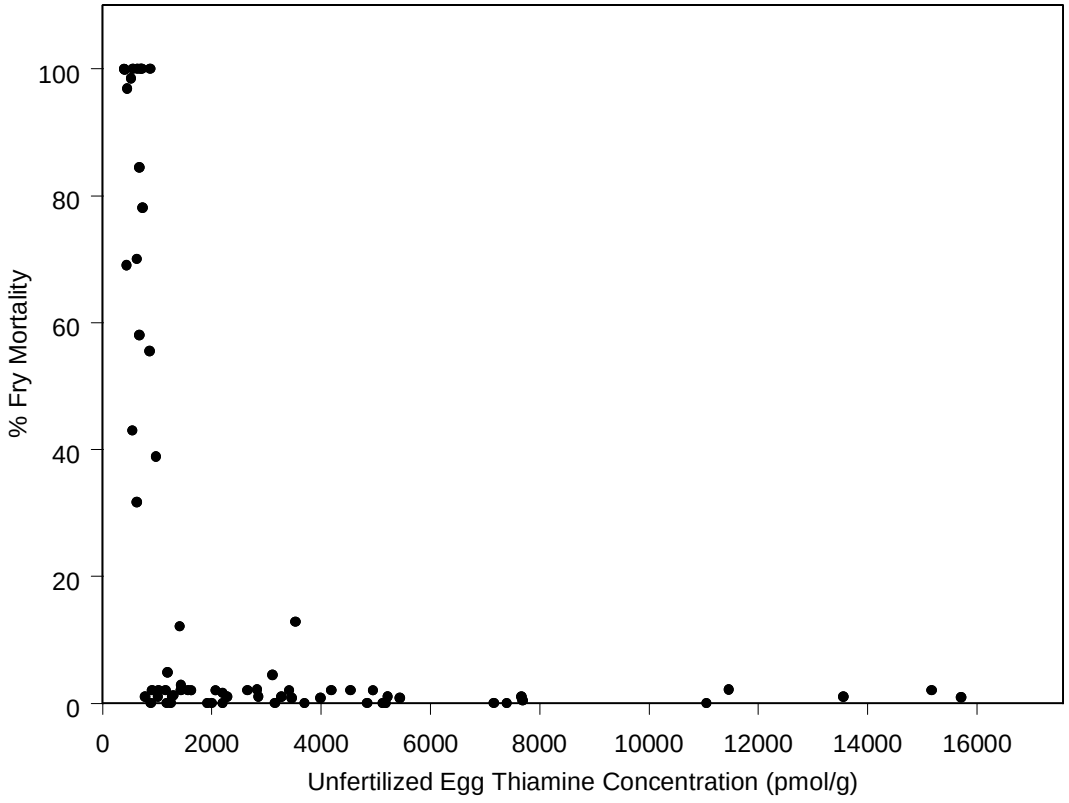


Figure 7-9
PCB Concentration in Lake Trout Eggs vs Measures of Egg and Fry Viability for Samples with Thiamine Concentrations of Less than 1000 pmol/g

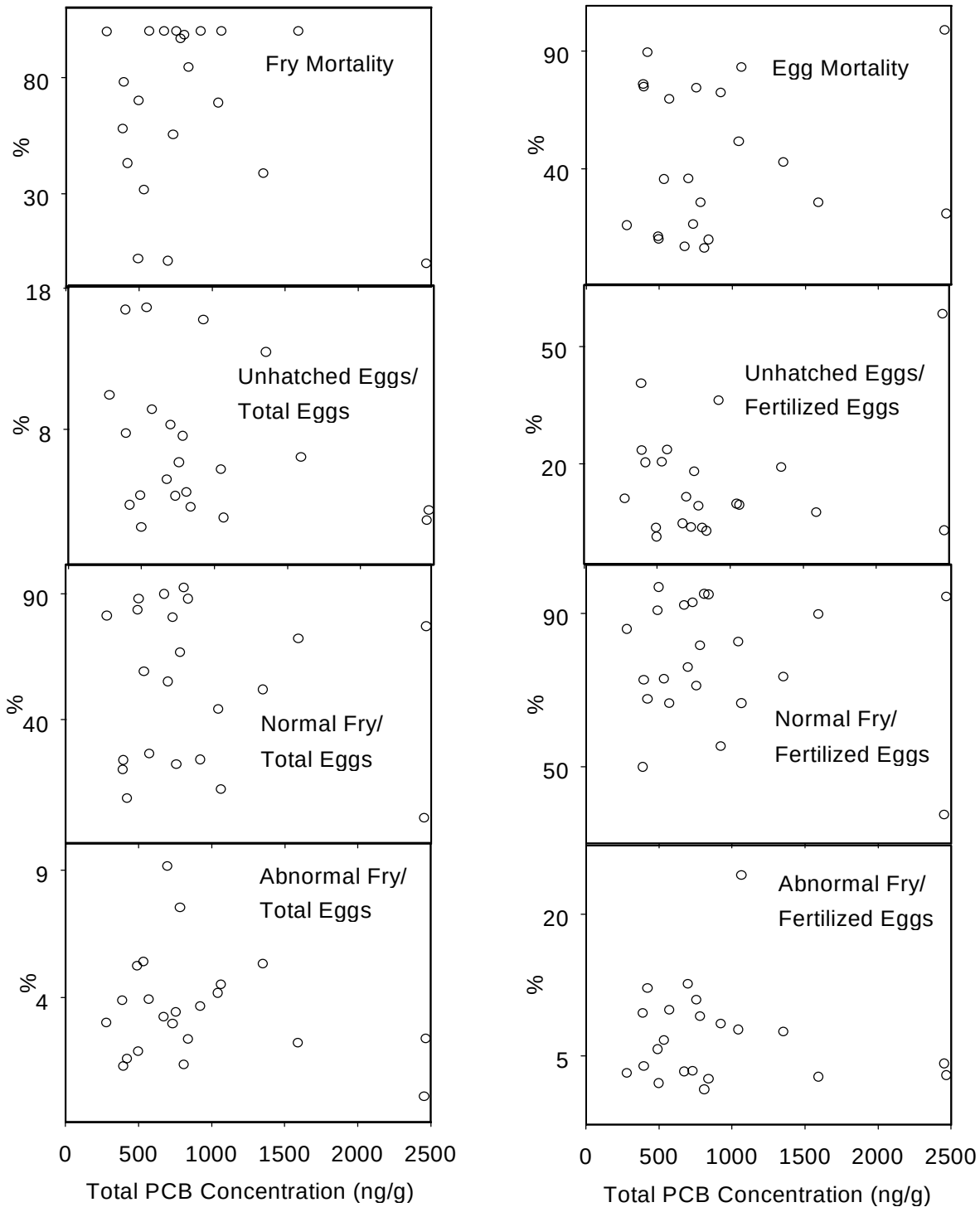
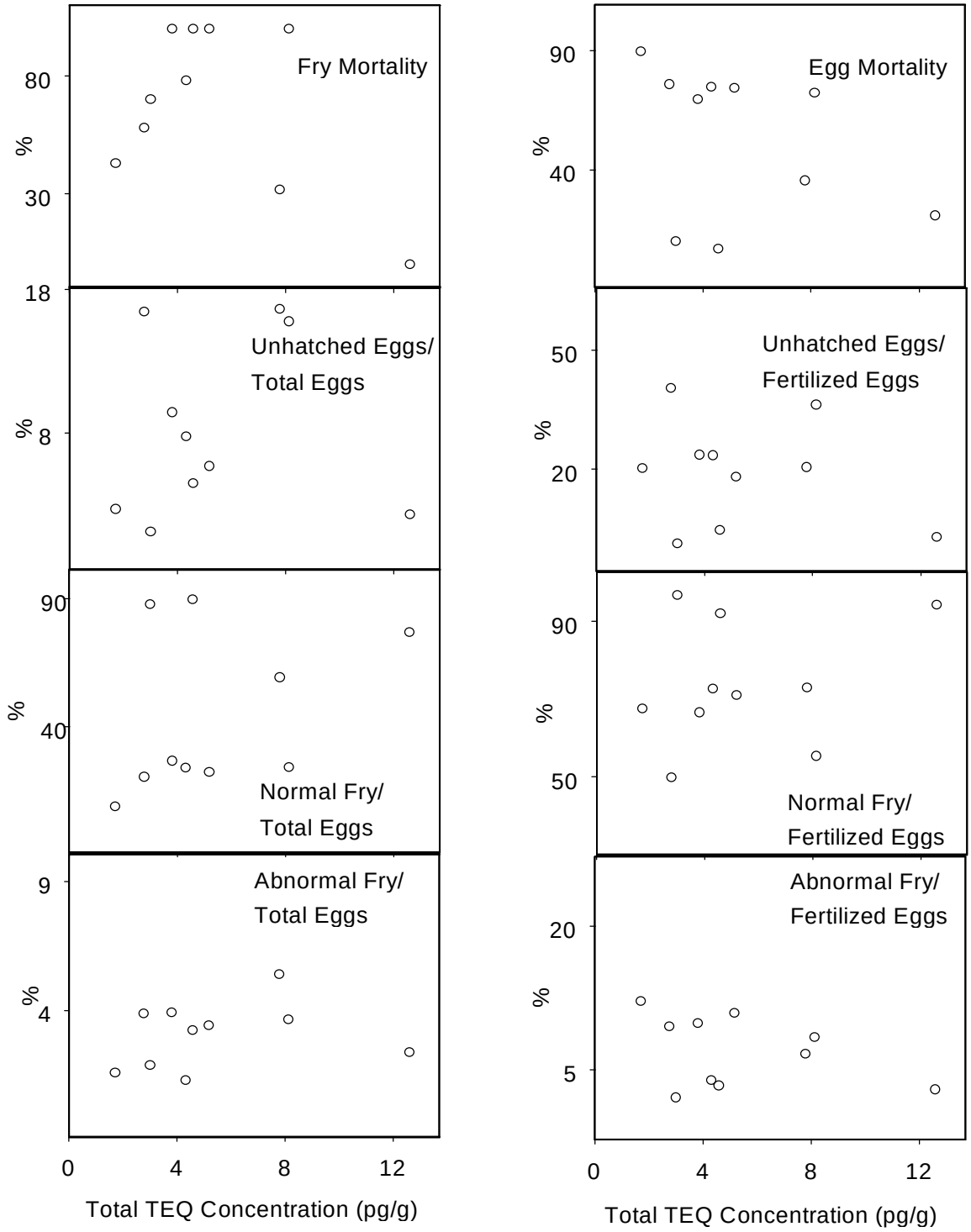


Figure 7-10
Total TEQ Concentration (from PCBs, PCDDs and PCDFs) vs Measures of Egg and Fry Viability for Samples with Thiamine Concentrations of Less than 1000 pmol/g



7.6 SUPPLEMENTAL NRDA AND USGS STUDIES ON PCB/THIAMINE INTERACTION

In 1996-1998, the Service and the USGS conducted a study on the possible interaction of low thiamine levels in lake trout eggs with PCB toxicity in Lake Michigan lake trout. The study, which was described in the NRDA Assessment Plan [61 Fed. Reg. 43558], was designed to evaluate whether lake trout eggs low in thiamine are more susceptible to the toxic effects of coplanar PCBs and other AhR-active compounds, including TCDD, than eggs high in thiamine. Several previous researchers have postulated that such an interaction could exist (McDonald et al., 1998). Full study methods and results are presented in USGS (1999a; 1999b), which are available in the Service's reading room in the Green Bay Field Office.

The specific objective of the study was to determine TCDD, PCB 77, and PCB 126 dose-response curves for lake trout eggs with different levels of thiamine to determine if egg thiamine content affects susceptibility to the toxic effects of TCDD and coplanar PCB congeners. An extract of Lower Fox River walleye that contained a mixture of the PCB, PCDD, and PCDF congeners found in the walleye was also used as a toxicant. Lake trout eggs were collected from various sources to obtain eggs of varying thiamine content: Lake Michigan (at Sturgeon Bay, Wisconsin, near Green Bay), to represent ambient thiamine content of assessment area fish; Charleston Lake, a relatively uncontaminated lake in Ontario that has lake trout which are low in thiamine; and the USGS Research and Development Laboratory (Leetown Science Center) in Wellsboro, Pennsylvania. Adult lake trout at the Wellsboro laboratory were fed different diets in an attempt to manipulate the thiamine content of the eggs.

Eggs from a given female were divided into two lots prior to fertilization. Immediately after fertilization, the two lots of eggs were water-hardened in solutions of different thiamine concentrations to obtain eggs from the same female that differed in thiamine content. Eggs from these lots were then exposed to TCDD, PCB 77, PCB 126, or walleye extract at six different concentrations to obtain dose-response curves. Eggs were dosed either via egg injection or through waterborne exposure. Endpoints measured included mortality, craniofacial deformities, yolk sac edema, hemorrhaging, and pathological lesions. Dose-response curves for lake trout egg batches of varying thiamine content were then compared to determine if eggs low in thiamine are more susceptible to the toxic effects of TCDD, PCB 77, PCB 126, and the walleye extract.

Several technical problems occurred during the study [as described in USGS (1999a; 1999b)] that limit the ability of the study to definitively answer the study objectives. However, the study did produce several valuable results:

- Mortality in eggs with low thiamine levels occurred at PCB 126 concentrations that were lower than concentrations in eggs with high thiamine levels. A similar result was indicated for sublethal endpoints with TCDD or the walleye extract as the toxicant. These results support the hypothesis of an interaction between low thiamine levels in lake trout eggs and increased susceptibility to TCDD and PCB toxicity. However, paired tests on egg batches

with different thiamine levels using TCDD or the walleye extract as the toxicant did not produce clear dose-response curves for mortality and thus are difficult to interpret.

- Sublethal effects, including craniofacial deformities, yolk sac edema, and hemorrhaging, occurred at TCDD, PCB 126, and walleye extract concentrations below those that caused fry mortality. Although the long-term effect of these sublethal effects is not known, these data indicate that injury thresholds based on mortality alone may underestimate injury to lake trout.

- Mortality significantly higher than control mortality occurred at egg concentrations of the walleye extract that are approximately 20 times higher than present-day concentrations of the similar mixture that is present in Lake Michigan lake trout eggs (when expressed as TCDD equivalents). Thus, these data substantiate the 1996-1998 USGS egg rearing studies that found that PCBs are not primary causative factors in present-day lake trout fry mortality.

7.7 COMPARISON OF ESTIMATED 1975-1995 PCB CONCENTRATIONS IN LAKE TROUT EGGS WITH INJURY THRESHOLDS

Although the data above indicate that current PCB concentrations are not causing reproductive injuries to lake trout, PCB concentration in Green Bay and Lake Michigan lake trout have declined from their measured historical highs in the 1970s (Stow et al., 1999). This section presents an estimate of historical concentrations of PCBs and TEQs in Lake Michigan lake trout eggs and compares these estimated concentrations with toxicity thresholds. The purpose of this analysis is to evaluate the potential for PCBs to have caused or contributed to the sac fry mortality observed historically in Lake Michigan lake trout.

7.7.1 Methods

Models Describing Temporal PCB Declines in Lake Michigan Lake Trout

The only historical data on PCB concentrations in lake trout eggs from Green Bay or Lake Michigan near Green Bay are a limited number of samples from 1982 (Wisconsin Department of Natural Resources, 1995) and from 1987 (Wisconsin Department of Natural Resources, 1987). Therefore, to estimate historical egg PCB concentrations, we used the models of Stow et al. (1999) that were developed for Lake Michigan lake trout fillet PCB concentrations. The Stow et al. models were based on lake trout fillet PCB concentration data from 1974 to 1994 obtained from the Wisconsin and Michigan Departments of Natural Resources [see Stow et al. (1995) for a detailed description of the data sources]. These data, which were collected as part of the State's fish contaminant monitoring programs, are the best data available for evaluating historical PCB

concentrations in Green Bay or Lake Michigan lake trout.[6] The fillet PCB data used by Stow et al. (1999) in constructing their models are from all of Lake Michigan, and thus their models represent the lake-wide average decline in lake trout PCB concentrations.

Stow et al. (1999) determined that two models of PCB decline in lake trout provided the best fit to the data: first-order exponential decay with a nonzero asymptote and mixed-order decline. The formula for the first-order model with a nonzero asymptote is as follows:

$$C_t = C_o e^{-kt} + C_a$$

where C is PCB concentration, t is time, k is the rate constant, and C_a represents the nonzero asymptote. This model approximates the temporal decline in PCB concentrations as a first-order exponential decay that approaches an asymptote greater than zero. Both the decay rate and the assymptote were determined by Stow et al. (1999) from the available data on historical lake trout fillet PCB concentrations.

The mixed-order model described by Stow et al. (1999) is as follows:

$$C_t = [C_o^{(1-\theta)} - kt(1-\theta)]^{1/(1-\theta)}$$

where C is the PCB concentration, t is time, k is the rate constant, and θ is a pseudo-order parameter. This pseudo-order parameter allows the model to estimate declines using a mixed-order parameter, which allows for concentrations to change over time at a variable rate (Stow et al., 1999).

We used both the first-order with nonzero asymptote and the mixed-order models derived by Stow et al. (1999) to estimate historical concentrations of PCBs in lake trout eggs. The curves defined by the models were calibrated through the 1996-1998 mean concentration measured in lake trout eggs from near Sturgeon Bay, Wisconsin (near Green Bay on the eastern side of the Door Peninsula). In this way, the shape of the lake trout egg concentration curve over time is identical to that determined by Stow et al. (1999) for lake trout fillets, but the position of the curve on the y-axis is scaled to egg PCB concentrations measured in lake trout from near Green Bay rather than to lake-wide fillet PCB concentrations.

Estimates of historical concentrations of TEQs from PCBs were derived from the estimated total PCB concentrations using the regression between 1996-1998 egg total PCBs and TEQs from PCBs described in Section 7.5.2. Estimates of historical TEQs from PCDD and PCDF congeners were obtained by fitting the Stow et al. (1999) models through the mean TEQ from PCDD plus PCDF congeners in the 1996-1998 lake trout eggs (3.8 pg/g). TEQs from PCDD and PCDF congeners were calculated using the TEFs listed in Table 7-4. Estimated historical total TEQs

6. Extensive dated sediment core data collected as part of the ongoing Lake Michigan Mass Balance Study may also be useful for estimating historical concentrations, but the data were not available at the time of this report.

(from PCBs, PCDDs, and PCDFs) were obtained by summing the estimated PCB TEQs with the estimated PCDD+PCDF TEQs for each year.

Historical Exceedence of Injury Thresholds

We used the central estimates of the TCDD LD10 (40.9 pg/g) and LD50 (61.2 pg/g), described in Section 7.3.3, as thresholds against which the historical estimated PCB and total TEQs are compared.

The mixed-order and nonzero asymptote models can be used to estimate the mean egg PCB and TEQ concentrations in each year but do not estimate the population distribution about the mean. Therefore, we applied the population distribution of the 1996-1998 egg PCB data to each of the previous year's estimated mean to derive an estimated population distribution of egg PCB concentrations for each year. This approach allows for an estimation of the percent of the population that exceeds a given injury threshold in each year.

The 1996-1998 egg PCB concentrations were determined to be log-normally distributed ($p > 0.05$ using the Kolmogorov-Smirnov goodness-of-fit test). To apply the standard deviation from these data to the yearly means estimated by the models, an assumption is required as to how (if at all) the standard deviation changes as the population mean increases. To examine the relationship between standard deviation and mean PCB concentrations in Green Bay fish, we used available data on historical walleye and carp PCB concentrations from Green Bay, the species for which the most historical data are available. Figure 7-11 plots two possible assumptions: the standard deviation is constant over all yearly mean PCB concentrations, or the ratio of the standard deviation to the yearly mean is constant. As Figure 7-11 shows, for carp and walleye the standard deviation increases with increasing yearly mean, but the ratio of the standard deviation to the yearly mean (shown as the solid circles and solid line) is approximately constant. Therefore, we applied these results for Green Bay carp and walleye to lake trout and assumed that the ratio of the standard deviation to mean in the 1996-1998 data (on a natural log scale) was constant through time. In this way, a standard deviation could be estimated for each year based on that year's estimated mean value.

The estimated population distributions were compared to the TCDD LD10 and LD50 values using the procedure depicted in Figure 7-12. The percent of the population exceeding the LD10 and LD50 was estimated for each year as the area under the curve where the estimated total TEQ is greater than each LD value.

Assumptions

To estimate historical injury to Green Bay lake trout eggs using these models, several key assumptions are required. One important assumption is that the models developed by Stow et al. (1999) for PCBs in lake trout fillets are also applicable to PCBs in eggs (once calibrated to egg concentrations). This assumption essentially amounts to whether the concentration of PCBs in

Figure 7-11
Relationship between the Mean and Standard Deviation and between the Mean and the Mean/Standard Deviation Ratio of Total PCB Concentration Measurements for Walleye Collected from the Fox River and Green Bay between 1978 and 1996 and for Carp Collected from Green Bay between 1977 and 1992. Each point represents a different year of data.

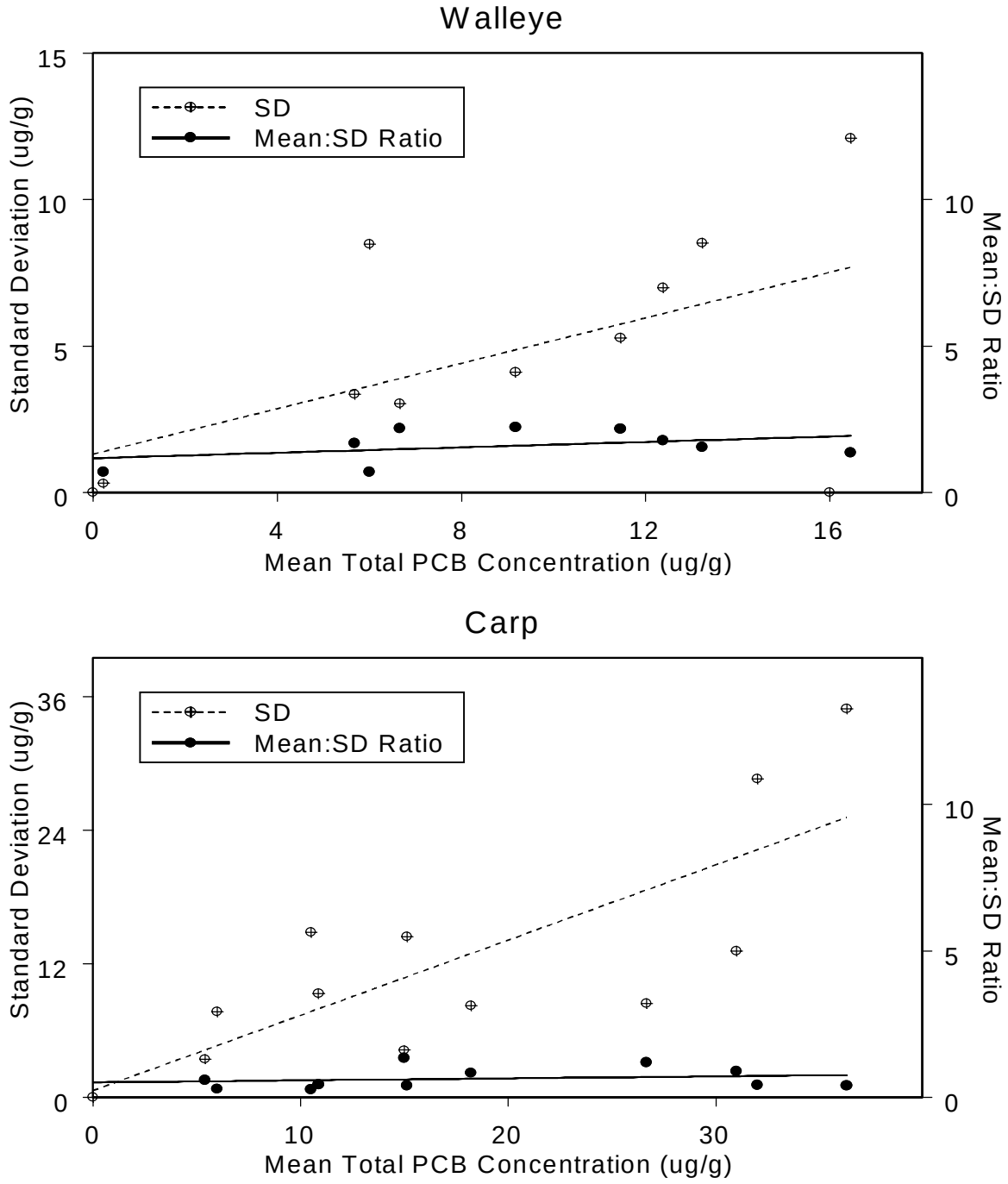
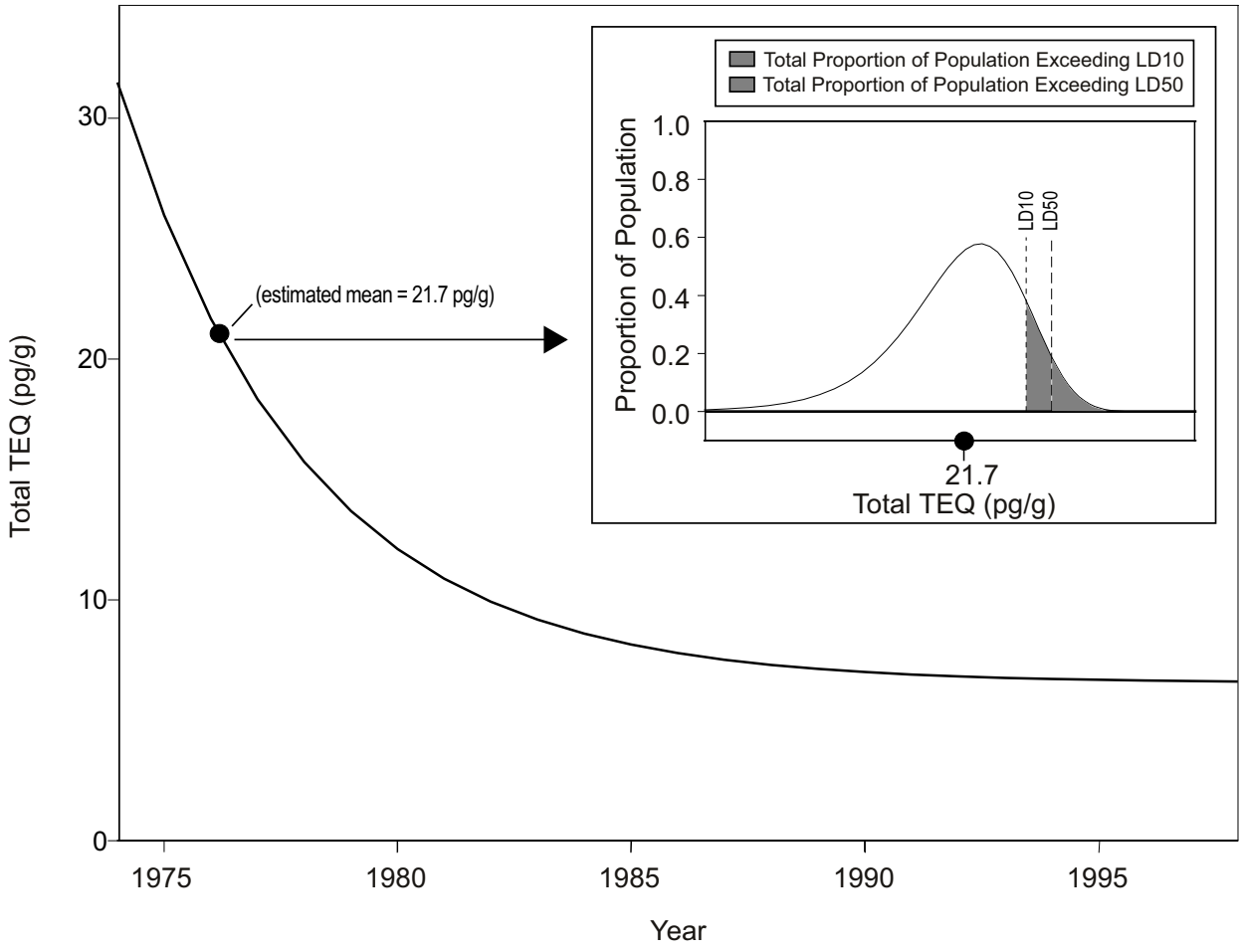


Figure 7-12
Graphical Depiction of Method for Estimating Percent Exceedence of the LD10 and LD50 for Each Year (1976 is shown)



lake trout eggs is related to that in fillets, or whether they are independent of each other. Miller (1993) investigated the relationship between concentrations of PCBs and DDE in Lake Michigan and Lake Superior salmonids and their eggs and found that the concentrations of these compounds in the muscle tissue of the parent lake trout were significantly correlated with the concentrations in their eggs. Furthermore, Miller (1993) found that the total concentration of PCBs in the muscle tissue of Lake Michigan lake trout accurately predicted the total concentration of all organochlorine compounds, including PCBs, in their eggs. Therefore, fillet and egg PCB concentrations are strongly related to each other, and a model based on fillet concentrations can be used to estimate egg concentrations.

Another assumption is that the temporal trend curves derived by Stow et al. (1999) for lake-wide Lake Michigan lake trout are applicable to Green Bay and northern Lake Michigan lake trout. Stow et al. (1999) used data from the entire lake to construct their models, which thus represent

lake-wide average trends. Although we calibrated the Stow et al. (1999) curves to egg PCB concentrations measured in lake trout from near Green Bay, the data are insufficient to determine whether the lake-wide average temporal trend is appropriate for lake trout in and near Green Bay. Nevertheless, there are no compelling reasons to assume that the temporal trend of PCBs in Green Bay and northern Lake Michigan lake trout would be substantially different from the lake-wide average trend.

We also assumed that congener patterns have not changed over time in Green Bay lake trout, so that the relationship between total PCB concentration and PCB TEQ in 1997 would be constant over time. Mac et al. (1993) analyzed lake trout eggs collected from Lake Michigan between 1979 and 1988, and found no significant change in PCB congener composition over the sampling period, demonstrating that individual congeners are declining at similar rates in lake trout eggs. Hebert et al. (1999) examined patterns of PCB congener bioaccumulation in herring gull eggs between 1971 and 1982, comparing eggs from Lake Ontario to eggs from Green Bay, and observed minor, but no large, shifts in congener composition in Green Bay. These studies suggest that PCB congener patterns in Green Bay and Lake Michigan biota have been relatively consistent over time, so that the relationship between total PCBs and TEQ from PCBs has also been constant over time.

Finally, we assumed that the temporal model for PCBs can also be applied to PCDDs and PCDFs. Although this assumption is not required for estimating PCB TEQ concentrations, it is required for estimating total TEQ concentrations from all AhR-active compounds. The 1996-1998 data for Lake Michigan lake trout eggs show no relationship between total PCBs and TEQs from PCDDs and PCDFs, suggesting that PCB and PCDD/PCDF concentrations are independent of each other in these data. Therefore a temporal model for PCBs may not be appropriate for PCDDs and PCDFs. However, the lack of a relationship within eggs at one point in time does not necessarily indicate a lack of a relationship over time. Models developed for PCBs, PCDDs, and PCDFs for Lake Ontario indicate a similar rate of decline for all three classes of compounds (Cook et al., 1994). Therefore, there is a reasonable basis to assume that the model developed for PCBs in Lake Michigan lake trout may also be useful for estimating historical PCDD and PCDF concentrations.

7.7.2 Results

The model parameters for estimating the changes in mean egg total PCB concentrations over time are provided in Table 7-5, and the model results are shown in Figure 7-13. Included in Figure 7-13 are the 1996-1998 data and the only historical data on total PCB concentrations in western Lake Michigan lake trout available, collected in 1982 and 1987 from lake trout captured near Sturgeon Bay, Wisconsin. Figure 7-13 shows that both models are consistent with the 1982 and 1987 measurements, although both models appear to slightly overestimate concentrations in 1987 and underestimate concentrations in 1982. Table 7-6 gives the estimated mean values for

Table 7-5
Model Parameters for Estimating Historical Egg Total PCB Concentrations

Constant	First-Order Model with a Nonzero Asymptote	Mixed-Order Model
k^a (1/t)	0.25	0.010
C_o^b (µg/g)	3.89	7.18
C_a^b (µg/g)	1.02	NA
θ^a	NA	2.95

a. From Stow et al. (1999).
b. Calibrated to the mean of the 1996-1998 egg PCB concentration data.

PCB concentration, TEQ from PCB, and total TEQ for each year estimated by the model, and Figure 7-14 shows the relative contributions to the total TEQ by PCBs and by PCDDs and PCDFs.

Comparison of Estimated Historical Concentrations with Injury Thresholds

The yearly results of the percent exceedences of the LD10 and LD50 for the each model are illustrated in Figure 7-15. The two models gave slightly different results. The mixed-order model estimated higher percent exceedence in 1974 and 1975, but then allowed for more rapid decline in total TEQ, and therefore in exceedence, through the late 1970s. After 1979, the rate of decline estimated by the mixed-order model slows down, and the model again estimates higher exceedences than the nonzero asymptote model. The nonzero asymptote, since it is a first-order model, estimated a more steady rate of decline in exceedence of the LD10 and LD50.

The LD10 and LD50 are the lethal doses of TCDD which cause 10% and 50% mortality, respectively, in exposed lake trout. Therefore, approximately 10% of the lake trout eggs at the LD10 and 50% of the lake trout eggs at the LD50 would be expected to experience mortality, and greater percentages of eggs with concentrations above the LD10 and LD50 would experience mortality. Nevertheless, the models estimate that by 1980, concentrations in less than 1% of Lake Michigan lake trout eggs were high enough to cause mortality.

Table 7-6
Mean Concentrations of Lake Trout Total PCBs, TEQ from PCBs, and Total TEQ Estimated by the Mixed-Order Model and the Nonzero Asymptote Model

	Total PCB Conc. (µg/g)		TEQ Conc. from PCBs (pg/g)		Total TEQ Conc. (pg/g)	
Year	Mixed-Order	Nonzero Asymptote	Mixed-Order	Nonzero Asymptote	Mixed-Order	Nonzero Asymptote
1974	7.18	4.91	19.92	13.63	46.30	31.49
1975	4.19	4.05	11.62	11.25	27.02	25.97
1976	3.24	3.38	8.99	9.39	20.90	21.67
1977	2.73	2.86	7.58	7.94	17.63	18.33
1978	2.41	2.45	6.67	6.81	15.51	15.72
1979	2.17	2.14	6.03	5.93	14.01	13.69
1980	2.00	1.89	5.53	5.25	12.86	12.11
1981	1.86	1.70	5.14	4.71	11.96	10.88
1982	1.74	1.55	4.83	4.30	11.22	9.92
1983	1.64	1.43	4.56	3.97	10.60	9.18
1984	1.56	1.34	4.33	3.72	10.07	8.60
1985	1.49	1.27	4.14	3.53	9.61	8.14
1986	1.43	1.22	3.96	3.37	9.21	7.79
1987	1.37	1.17	3.81	3.25	8.86	7.52
1988	1.32	1.14	3.67	3.16	8.54	7.30
1989	1.28	1.11	3.55	3.09	8.25	7.13
1990	1.24	1.09	3.44	3.03	7.99	7.00
1991	1.20	1.08	3.34	2.99	7.76	6.90
1992	1.17	1.07	3.24	2.96	7.54	6.83
1993	1.14	1.06	3.16	2.93	7.34	6.76
1994	1.11	1.05	3.08	2.91	7.15	6.72
1995	1.08	1.04	3.00	2.89	6.98	6.68
1996	1.06	1.04	2.93	2.88	6.82	6.65
1997	1.03	1.03	2.87	2.87	6.67	6.63
1998	1.01	1.03	2.81	2.86	6.53	6.61

Figure 7-13
Estimated Mean Total PCB Concentrations in Lake Michigan Lake Trout Eggs, 1974-1998. Included are measured total PCB concentrations in 1982 from near Sturgeon Bay, Wisconsin, and in 1996-1998 from near Sturgeon Bay, Wisconsin.

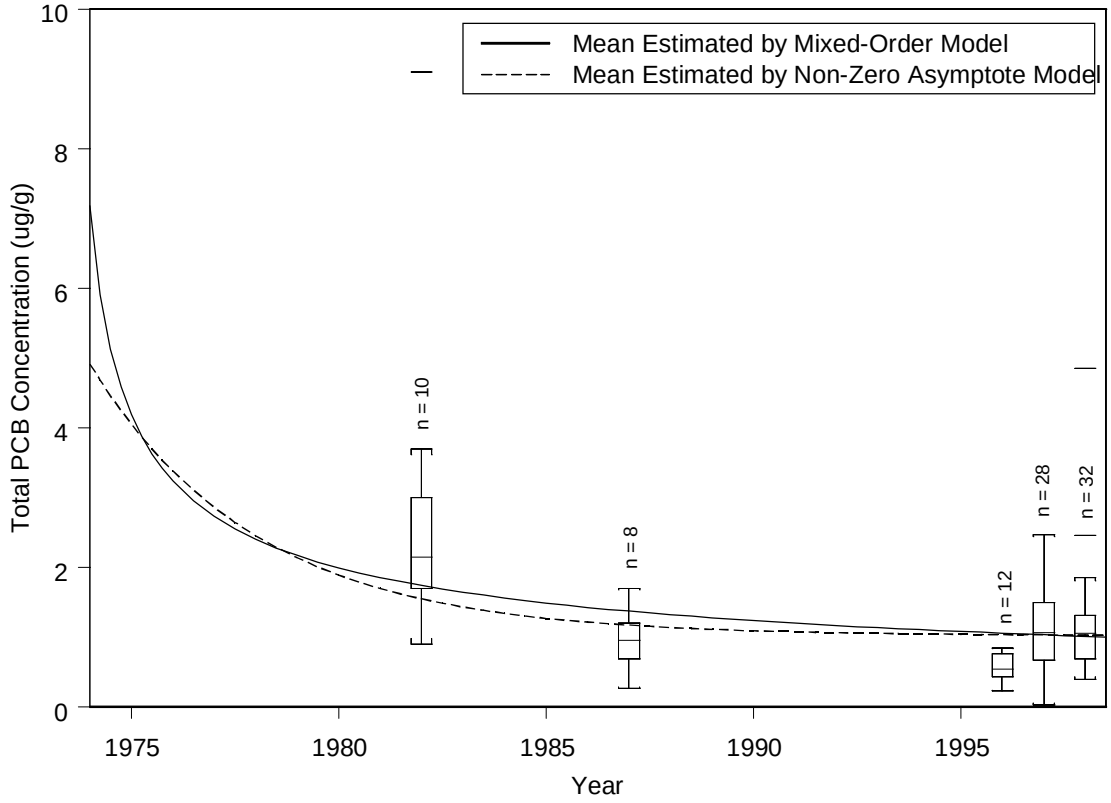


Figure 7-14
The Relative Contributions to the Total TEQ by PCBs and by PCDDs and PCDFs

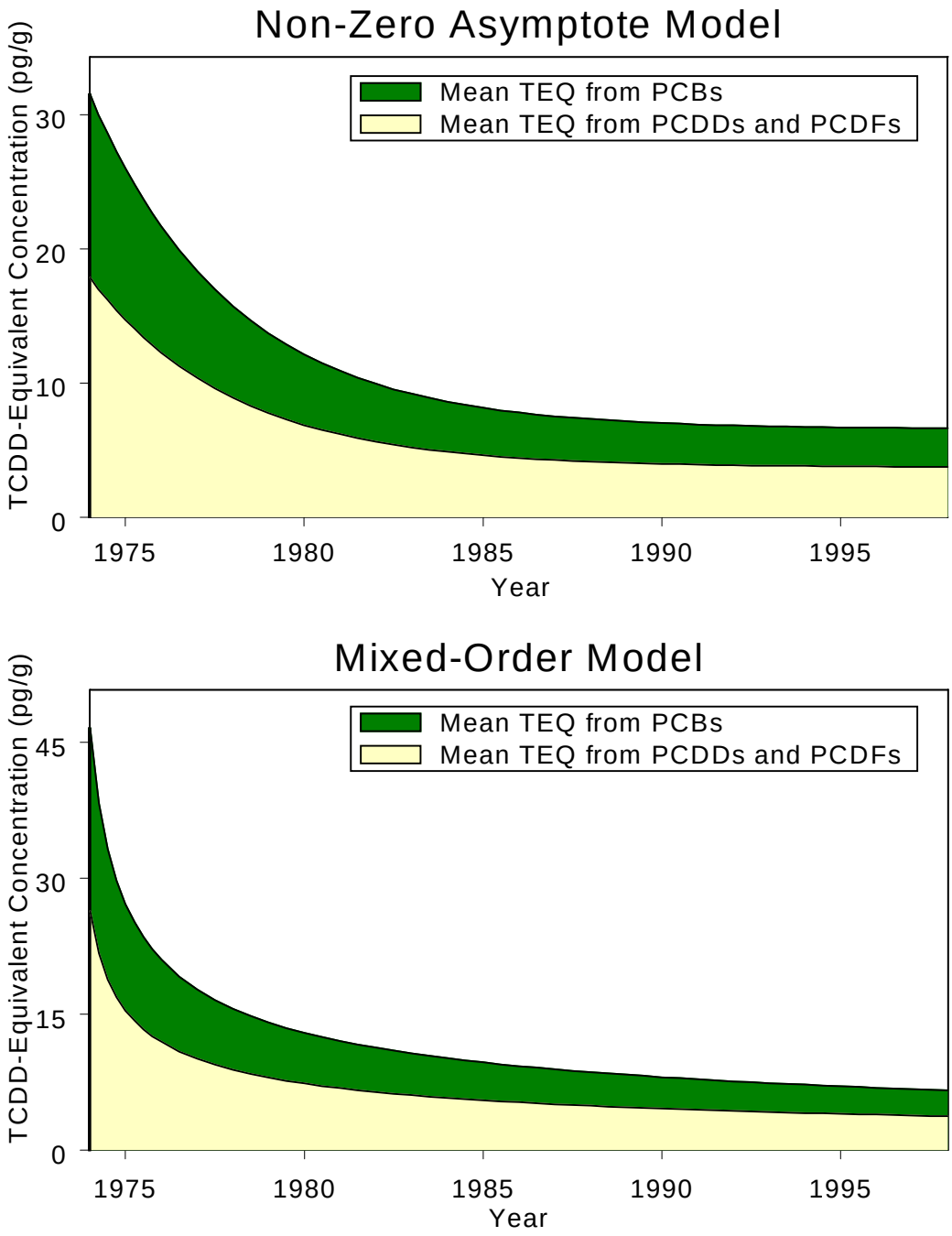
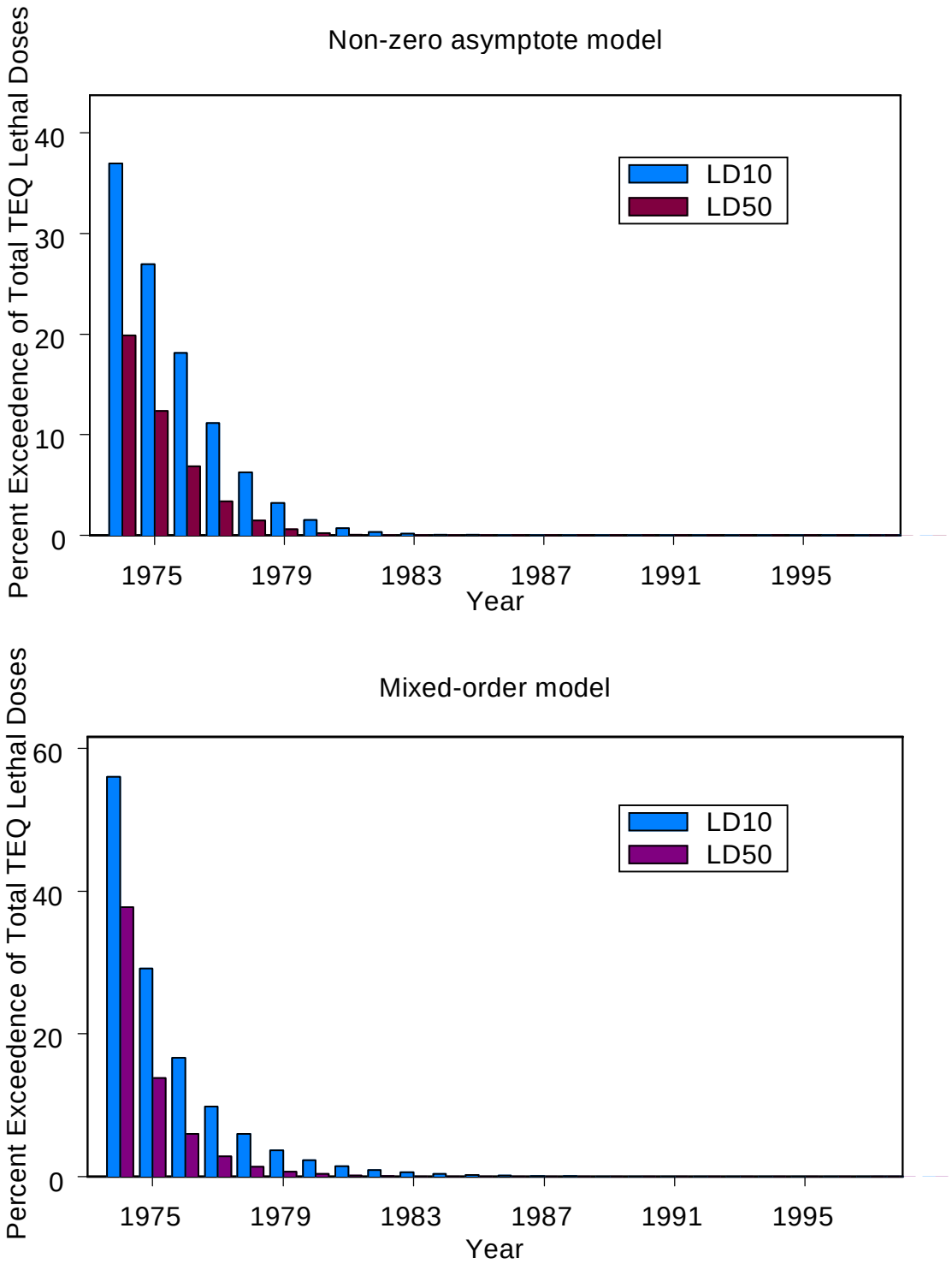


Figure 7-15
Estimated exceedences of the LD10 and LD50 for PCB, PCDD, and PCDF exposure (as total TEQ) since 1974.



Application to Green Bay

The application of these models to Green Bay is uncertain. The models derived by Stow et al. (1999) are based on trends in Lake Michigan lake trout, and the extent to which trends in PCB concentrations in Lake Michigan lake trout would be similar to those in Green Bay lake trout (if they were there) is difficult to determine. For example, historical exposure of lake trout to PCBs may have been different in Green Bay and Lake Michigan, and concentrations may have declined at different rates. In addition, the migration of lake trout within and between Green Bay and Lake Michigan could influence how concentrations in Green Bay lake trout relate to those in Lake Michigan. Differences in the diet may also alter the pattern of PCB accumulation in lake trout from the two regions.

To address this uncertainty, we compared historical PCB concentrations in Green Bay and northern Lake Michigan lake trout during two time periods for which data were available from both locations: 1975-1978 and 1983-1984 (both periods are during the time when lake trout were stocked in Green Bay) (Wisconsin Department of Natural Resources, 1995). For both time periods, the average wet weight and lipid-normalized concentrations were higher in Green Bay than in Lake Michigan, though significant differences were found only for the 1975-1978 time period. From 1975-1978, wet weight PCB concentration in Green Bay averaged 9.98 mg/kg compared to 7.37 mg/kg in Lake Michigan ($p<0.005$, Wilcoxon rank-sum), while lipid-normalized PCB concentration in Green Bay averaged 89.6 mg/kg compared to 68.8 mg/kg in Lake Michigan ($p<0.001$, Wilcoxon rank-sum test). From 1983-1984, wet weight PCB concentration in Green Bay averaged 3.96 mg/kg compared to 3.38 mg/kg in Lake Michigan, while lipid-normalized PCB concentration averaged 27.8 mg/kg in Green Bay versus 27.3 mg/kg in Lake Michigan. Thus, the limited data available suggest that estimates of egg PCB concentrations for Lake Michigan lake trout may undersestimate concentrations for Green Bay lake trout. Therefore, mortality to Green Bay lake trout from PCB exposure may have been somewhat higher than that estimated for Lake Michigan lake trout, but most likely was still not substantial by 1980.

7.7.3 Conclusions

The analysis presented in this section estimates that historical exceedences of the LD10 and LD50 injury thresholds by PCB concentrations in Green Bay and Lake Michigan lake trout eggs were relatively low. Since 1980, the percent mortality to lake trout eggs from PCB exposure is estimated to be less than 1%. These results suggest that, although lake trout sac fry were most likely adversely affected by PCB toxicity in the past, PCB contamination is currently below the threshold that would contribute significantly to mortality and has been since the early 1980s.

7.8 SUMMARY AND INJURY DETERMINATION

That Lake Michigan lake trout have failed to re-establish naturally reproducing populations, and that egg and/or fry mortality has contributed to that failure, is clear. For the Trustees' injury determination, the issue is the degree to which the reproductive failure has resulted from PCBs released from Lower Fox River paper companies. This section presents injury determination conclusions for lake trout based on the information presented in the chapter.

7.8.1 Injury Definition and Measurements

Injuries to lake trout were assessed according to the definitions of injury in the Department's NRDA regulations. Specifically, the injury definitions state that fishery resources have been injured as a result of the release of a hazardous substance if the concentration of the substance is sufficient to

> cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction), or physical deformations [43 CFR § 11.62(f)(1)(i)].

Adverse changes in viability to lake trout are assessed using the following measurements:

- death, as documented in laboratory toxicity testing [43 CFR § 11.62(f)(4)(i)(A)]
- reduced fish reproduction [43 CFR § 11.62(f)(4)(v)(E)]
- overt external malformations [43 CFR § 11.62(f)(4)(vi)(A)] such as edema
- skeletal deformities [43 CFR § 11.62(f)(4)(vi)(B)] such as craniofacial deformities
- internal whole organ and soft tissue malformations [43 CFR § 11.62(f)(4)(vi)(C)] such as edema.

7.8.2 Weight of Evidence Evaluation

The results of historical studies on the relationship between egg and fry mortality and PCBs are mixed. Some studies documented a statistically significant relationship between PCBs and mortality, whereas others did not. However, most of the studies did not differentiate between the symptoms and timing of blue-sac syndrome (caused by PCBs and other TCDD-like compounds) and swim-up syndrome (caused by thiamine deficiencies). Therefore, the endpoints measured in many of these studies may not be specific to PCB toxicity, decreasing the studies' power to detect any relationship between PCBs and egg or fry mortality, particularly if the contribution of PCBs to the observed mortality is small.

Our results from modeling PCB concentrations in lake trout eggs from 1974 to 1995 is consistent both with the limited amount of historical lake trout egg PCB data and with the results of the studies that have examined potential relationships between PCBs and egg/fry mortality in Lake Michigan lake trout. The hindcast model, coupled with TCDD toxic thresholds obtained from available toxicity studies on lake trout, suggests that in the mid-1970s concentrations of PCBs were sufficient to cause sac fry mortality to some Green Bay and Lake Michigan lake trout eggs, and that the incidence of PCB-caused mortality declined rapidly to near zero since then. By 1980, the hindcast model estimates that less than 1% of the eggs had PCB concentrations sufficient to cause toxicity. This percentage is consistent with the 0.7% to 5.8% incidence of mortality attributed to blue sac disease, whose symptoms are very similar to PCB toxic effects, by Mac et al. (1993) in Lake Michigan lake trout eggs from the late 1970s and early 1980s. Given this low percentage of PCB-caused mortality, it would be difficult to detect PCB-mortality relationships in studies of mortality in eggs and fry, particularly if the mortality endpoints assessed are not specific to PCB toxic effects. Thus, the estimates of the model, although subject to several sources of uncertainty, are consistent with the historical observations on PCB-mortality relationships and suggest little or no mortality since the 1970s.

Studies in 1996-1998 indicate that thiamine deficiency, not PCBs, is a primary causative factor in current lake trout reproductive failure. Sac fry mortality was strongly correlated with low egg thiamine concentration and had no relationship with PCB concentration. This conclusion is consistent with the fact that the measured PCB concentrations are approximately an order of magnitude less than those determined to be toxic in lake trout laboratory toxicity studies. Thiamine deficiency causes swim-up syndrome, which has a set of symptoms and timing distinct from the toxic effects of PCBs, which are similar to blue-sac disease. Although many historical studies do not specify whether observed fry mortality was based on symptoms similar to swim-up syndrome or blue-sac disease, in those that do most (if not all) of the observed fry mortality was related to swim-up syndrome rather than blue-sac disease. Finally, thiamine deficiency as the cause for fry mortality in Lake Michigan lake trout is consistent with the results of studies from other areas, including the Finger Lakes region of New York and the Baltic Sea.

Therefore, the available information supports a conclusion that PCBs currently play little or no role in Lake Michigan lake trout egg/fry mortality, and that historically they may have played a small role in the 1970s.

7.8.3 Uncertainties

There are several uncertainties in this injury assessment that may lead to an underestimate of injuries. One uncertainty is the degree to which any PCB/thiamine interaction makes Lake Michigan lake trout eggs more susceptible to EMS or other reproductive effects than predicted based on available field and laboratory toxicity data. Such an interaction could make lake trout eggs that contain PCBs more susceptible to EMS, which is consistent with the higher incidence of EMS observed in contaminated Great Lakes (McDonald et al., 1998). A similar interaction could

be involved in studies that have suggested that eggs low in thiamine, but high enough to survive, are more susceptible to PCB toxicity than eggs high in thiamine, such as those typically used in laboratory toxicity studies (Section 7.6; Wright et al., 1998). Such an interaction could make some Lake Michigan lake trout eggs more susceptible to PCB toxicity. Available information is not sufficient to quantitatively assess the potential interaction of low thiamine levels and PCB exposure. Nevertheless, the 1996-1998 Lake Michigan lake trout egg rearing studies conducted by the USGS (described in Section 7.5) show that if such a PCB/thiamine interaction is occurring, it is of very small magnitude under current conditions relative to the effects of thiamine deficiency.

Another issue is the potential for sublethal effects of PCBs on developing lake trout, such as behavioral alterations, reduced growth, or deformities. Some available information suggests the potential for sublethal effects to occur at exposure concentrations below those that cause mortality and below currently available toxicity thresholds (described in Sections 7.3 and 7.6). These sublethal effects could decrease the viability of lake trout fry in the wild, effectively decreasing the injury threshold concentrations for PCBs in lake trout. Some studies have indicated that at least some of these sublethal effects may be reversible (Spitsbergen et al., 1991). Again, however, the information available currently is insufficient to address these potential injuries quantitatively.

Finally, another uncertainty is the potential for the laboratory-derived thresholds for TCDD toxicity to lake trout sac fry to underestimate actual toxicity in the field. For example, the results of Mac et al. (1993) suggest that egg mortality is related to PCB concentrations in lake trout eggs from the Great Lakes (including Lake Michigan), a result that cannot be explained within the context of the current laboratory studies on TCDD toxicity to lake trout eggs and fry. Although the current information from laboratory studies is compelling, it may be that the current laboratory studies do not fully capture the contaminants, exposure regimes, mechanisms of toxicity, and interactions with other factors that occur in the wild. With the information currently available, these potentially mitigating factors can only be treated qualitatively as uncertainties in the injury assessment for lake trout.

7.8.4 Injury Determination Conclusion

Based on the available information, the Trustees conclude the following regarding injuries to lake trout in Green Bay and Lake Michigan resulting from PCB exposure:

- PCB concentrations in the mid-1970s were sufficient to cause sac fry mortality in some lake trout eggs.

- PCB concentrations since the 1970s have not been sufficient to cause sac fry mortality to lake trout. By 1980, concentrations in less than 1% of Lake Michigan lake trout eggs are estimated to have been sufficient to cause mortality.

- PCB concentrations since the 1970s may be sufficient to cause sublethal effects or interact with low thiamine levels, but the presence and magnitude of these possible effects cannot be determined from the available information.

Given that evidence suggests little to no reproductive effects of PCBs since 1980 and that other factors appear to be substantially more important to the survival of lake trout fry, the Trustees determine that current data do not support the conclusion that lake trout in Green Bay and Lake Michigan are injured by the PCBs released from Fox River paper companies.

CHAPTER 8
FISH INJURY ASSESSMENT SUMMARY

The Trustees have conducted an assessment of injuries to fishery resources of the Lower Fox River/Green Bay environment that result from releases of PCBs from Fox River paper company facilities. The injury assessment included determination of PCB transport pathways from paper company facilities to fishery resources of the river and bay, injury determination, and injury quantification. The injury assessment was conducted consistent with the Department's NRDA regulations at 43 CFR Part 11, and included assessment of injuries associated with state fish consumption advisories because of PCBs, exceedences of the Food and Drug Administration's PCB tolerance level, and adverse effects on fish viability.

The most significant injury to fishery resources of the Lower Fox River and Green Bay that results from paper company PCB releases is the presence of extensive fish consumption advisories. The advisories, ranging from limited to no fish consumption, are in place for dozens of fish species throughout the Lower Fox River, Green Bay, and northern Lake Michigan. The advisories have been in place since the 1970s and continue to the present (1999). The quantification of the losses to the public as a result of the PCB fish consumption advisories is presented in the Trustees' report on recreational fishing damages (Stratus Consulting, 1999e).

Consistent with the fish consumption advisories are injuries resulting from exceedences of the Food and Drug Administration's tolerance level for PCBs in fish tissue. The tolerance level is exceeded in many fish species throughout the assessment area. This injury is indicative of the extensive PCB contamination of Lower Fox River and Green Bay fish.

Walleye in the Lower Fox River and Green Bay suffer from the injury of increased liver tumors. The injury is most pronounced in female walleye, in which 34% of fish from the river and bay had liver tumors or pre-tumors compared with 7% of fish from reference areas. The Trustees assessed other adverse viability injuries, including brown trout and lake trout health and lake trout reproduction, and concluded that available information does not support a conclusion that these fish currently are suffering from PCB-caused injuries, although they may have in the past.

CHAPTER 9
REFERENCES

Allen, P., J. Sullivan and L. Persson. 1987. Toxic Substances Management Technical Advisory Committee Report: Lower Green Bay Remedial Action Plan. Published by Wisconsin Department of Natural Resources. PUBL-WR-166 87.

Anderson, D.P. and M.G. Zeeman. 1995. Immunotoxicology in Fish. In *Fundamentals of Aquatic Toxicology: Effects, Environmental Fate, and Risk Assessment*, G.M. Rand, ed. Washington D.C.: Taylor & Francis. pp. 371-402.

Anderson, M.J., J. Zelikoff, S.J. Teh, M.S. Okihiro, D.E. Hinton, N. Denslow and D. Beltman. 1998. Biochemical Indices, Immune Status, and Toxicopathologic Lesions Assessed in Smallmouth Bass Inhabiting an Organochlorine River (abstract). p. 47.

Arcand-Hoy, L.D. and W.H. Benson. 1998. Fish reproduction: An ecologically relevant indicator of endocrine disruption. *Environmental Toxicology and Chemistry* 17(1): 49-57.

Arkoosh, M.R., E. Clemons, M. Myers and E. Casillas. 1994. Suppression of b-cell mediated immunity in juvenile chinook salmon (*Oncorhynchus tshawytscha*) after exposure to either a polycyclic aromatic hydrocarbon or to polychlorinated biphenyls. *Immunopharmacology and Immunotoxicology*. 16(2): 293-314.

ATSDR. 1996. Public Health Implications of PCB Exposures. U.S. Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA. 21 pp.

Baldwin, N. S., R. W. Saalfeld, M. A. Ross, and H. J. Buettner. 1979. Commercial fish production in the Great Lakes 1867-1977. Great Lakes Fisheries Commission Technical Report 3. 187 pp.

Barron, M.G., M.J. Anderson, D. Beltman, T. Podrabsky, W. Walsh, D. Cacela, J. Lipton, S.J. Teh, D.E. Hinton, J.T. Zelikoff, A.L. Dikkeboom, B.A. Lasee, S.K. Woolley, D.E. Tillitt, M. Holey, P. Bouchard and N. Denslow. 1999. Association between PCBs, liver lesions, and biomarker responses in adult walleye *(Stizostedium vitreum vitreum)* collected from Green Bay, Wisconsin. *Journal of Great Lakes Research* (in review).

Battelle. 1999. Final Data Report for Fox River, WI RI/FS Sample Analysis. Prepared for U. S. EPA Region 5, July 22.

Battelle Ocean Sciences. 1997a. Tissue Samples for PCB Analysis: PCB Congener Analysis by GC/ECD Bird Egg and Fish Egg. Prepared for Hagler Bailly, U. S. Fish and Wildlife Service, Lower Fox River/Green Bay NRDA Project. Battelle Batch #97-126, 97-129. Project Number G003264.

Battelle Ocean Sciences. 1997b. Tissue Samples for PCB Analysis: PCB Congener Analysis by GC/MS Bird Egg and Fish Egg. Prepared for Hagler Bailly, U. S. Fish and Wildlife Service, Lower Fox River/Green Bay NRDA Project. Battelle Batch #97-126, 97-129. Project Number G003264.

Baumann, P.C. 1992a. Methodological considerations for conducting tumor surveys of fishes. *Journal of Aquatic Ecosystem Health* 1: 127-133.

Baumann, P.C. 1992b. The use of tumors in wild populations of fish to assess ecosystem health. *Journal of Aquatic Ecosystem Health* 1: 135-146.

Becker, G.C. 1983. Fishes of Wisconsin. University of Wisconsin Press. 1052 pp.

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. In *Chlorinated Hydrocarbons as a Factor in the Reproduction and Survival of Lake Trout (Salvelinus namaycush) in Lake Michigan. Technical Paper No. 105,* U.S. Fish and Wildlife Service, ed. Washington, DC: U.S. Government Printing Office. pp. 11-22.

Bertrand, G., J. Lang and J. Ross. 1976. The Green Bay Watershed: Past/Present/Future. University of Wisconsin Sea Grant College Program Technical Report #229. January. 300 pp.

Brazner, J. and W. DeVita. 1998. PCBs, DDE, and mercury in young-of-the-year littoral fishes from Green Bay, Lake Michigan. *Journal of Great Lakes Research* 24(1): 83-92.

Brazner, J.C. and J.J. Magnuson. 1994. Patterns of fish species richness and abundance in coastal marshes and other nearshore habitats in Green Bay, Lake Michigan. *Verh. Internat. Verein. Limnol* 25: 2098-2104.

Brown, E. H., Jr., G.W. Eck, N.R. Foster, R.M Horrall and C.E. Coberly. 1981. Historical evidence for discrete stocks of lake trout (*Salvelinus namaycush*) in Lake Michigan. *Canadian Journal of Fisheries and Aquatic Sciences* 38:1747-1758.

Brown, S.B., D.C. Honeyfield and L. Vandenbyllaardt. 1998. Thiamine Analysis in Fish Tissues. In *Early Life Stage Mortality Syndrome in Fishes of the Great Lakes and Baltic Sea,* G. McDonald, J.D. Fitzsimons and D.C. Honeyfield, eds. Bethesda, MD: American Fisheries Society. pp. 73-81.

Broyles, R.H. and M.I. Noveck. 1979. Uptake and distribution of 2,4,5,2',4',5'-hexachlorobiphenyl in fry of lake trout and chinook salmon and its effects on viability. *Toxicology and Applied Pharmacology.* 50: 299-308.

Burkett, D. P., W. N. Busch, J. R. McClain, M. E. Holey, T. R. Busiahn and M. C. Fabrizio. 1995. Great Lakes Fishery Resources Study: Report to Congress. Department of Interior, U.S. Fish and Wildlife Service, Washington D.C. 198 pp.

Burkhalter, D.E. and C.M. Kaya. 1977. Effects of prolonged exposure to ammonia on fertilized eggs and sac fry of rainbow trout (*Salmo gairdneri*). *Transactions of the American Fisheries Society* 106:470-475.

Burnham-Curtis, M.K. and C.R. Bronte. 1996. Otoliths reveal a diverse age structure for humper lake trout in Lake *Superior. Transactions of the American Fisheries Society* 125:844-851.

Cantrell, S.M., L.H. Lutz, D.E. Tillitt and M. Hannink. 1996. Embryotoxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD): The embryonic vasculature is a physiological target for TCDD-induced DNA damage and apoptotic cell death in medaka (*Orizias latipes*). *Toxicology and Applied Pharmacology* 141:23-34.

Cantrell, S., J. Joy-Schlezinger, J.J. Stegeman, D.E. Tillitt and M. Hannink. 1997. Quantitation of programmed cell death in TCDD-treated medaka (*Oryzias latipes*). *Toxicology and Applied Pharmacology* (Submitted).

Cogswell, S. 1998. Fishery Surveys on Seven Streams in the Oneida Indian Reservation, Wisconsin. 1998 Stream Assessments and Contaminant Collections. Green Bay Fishery Resources Office.

Cogswell, S. and D. Bougie. 1998. Duck Creek Fisheries Assessment: Final Report. Green Bay Fishery Resources Office Report 98-1. October 30.

Colborn, T.E., A. Davidson, S.N. Green, R.A. Hodge, I.C. Jackson and R.A. Liroff. 1990. *Great Lakes: Great Legacy?,* Washington, DC.: The Conservation Foundation. 301 pp.

Connolly, J.F., T.F. Parkerton, J.D. Quadrini, S.T. Taylor and A.J. Thumann. 1992. Development and Application of a Model of PCBs in the Green Bay, Lake Michigan Walleye and Brown Trout and their Food Webs. Prepared for the U.S. EPA, Large Lakes Research Station, Grosse Ile, Michigan. October 2. 200 pp.

Cook, P.M., R.J. Erickson, R.L. Spehar, S.P. Bradbury and G.T. Ankley. 1993. Interim report on data and methods for assessment of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin risks to aquatic life and

associated wildlife. U.S. Environmental Protection Agency, Office of Research and Development Report EPA/600/R-63/055, Washington, DC.

Cook, P.M., B.C. Butterworth, W.K. .Walker, M.W. Hornung, E.W. Zabel and R.E. Peterson. 1994. Lake trout recruitment in the Great Lakes: relative risks for chemical-induced early life stage mortality. 16 pp.

Cook, P.M., E.W. Zabel and R.E. Peterson. 1997. The TCDD toxicity equivalence approach for characterizing risks for early life stage mortality in trout. R. Rolland, M. Gilbertson and R. Peterson, eds., In *Chemically-Induced Alterations in the Functional Development and Reproduction of Fishes*. Pensacola, FL: SETAC Press. pp. 9-27.

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.

Eggens, M.L., A. Opperhuizen and J.P. Boon. 1996. Temporal variation of CYP1A indices, PCB and 1-OH pyrene concentration in flounder, *Platichthys flesus,* from the Dutch Wadden Sea. *Chemosphere* 33(8): 1579-1596.

Eggold, B.T. 1995. Wisconsin's 1995 open water sportfishing effort and catch from lake Michigan and Green Bay. Wisconsin DNR.

Eisler, R. 1986. Polychlorinated biphenyl hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildlife Service Biological Report 85(1.7), Contaminant Hazard Reviews Report No. 7. April. 72 pp.

Eisler, R. and A.A. Belisle. 1996. Planar PCB Hazards to Fish Wildlife, and Invertebrates: A Synoptic Review. National Biological Service Contaminant Hazard Reviews Report 31. U.S. Department of the Interior, Washington, DC. 75 pp.

Elonen, G.E., R.L Spehar, G.W. Holcombe, R.D. Johnson, J.D. Fernandez, R.J. Erickson, J.E. Tietge, and P.M. Cook. 1998. Comparative toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin to Seven Freshwater Fish Species During Early Life-Stage Development. *Environmental Toxicology and Chemistry* 17: 472-483.

Eschmeyer, P.H. 1957. The near extinction of lake trout in Lake Michigan. *Transactions of the American Fisheries Society* 85(1955):102-119.

Exponent. 1999. Draft Technical Memorandum 7c: Model Evaluation Workgroup Recommendations for Modeling PCB Bioaccumulation in Fish Tissue in the Fox River and Green Bay. Prepared for the Fox River Model Evaluation Workgroup. July.

Fisher, J.P., J.D. Fitzsimons, G.F. Combs. Jr. and J.M. Spitsbergen. 1996. Naturally occurring thiamine deficiency causing reproductive failure in Finger Lakes Atlantic salmon and Great Lakes lake trout. *Transactions of the American Fisheries Society.* 125: 167-178.

Fitzsimons, J.D. 1995a. A Critical Review of the Effects of Contaminants on Early Life Stage (ELS) Mortality of Lake Trout in the Great Lakes. *Journal of Great Lakes Research* 21(Supplement 1): 267-276.

Fitzsimons, J.D. 1995b. The effect of B-vitamins on a swim-up syndrome in Lake Ontario lake trout. *Journal of Great Lakes Research* 21(Supplement 1): 286-289.

Fitzsimons, J.D., S. Huestis and B. Williston. 1995. Occurrence of a Swim-up Syndrome in Lake Ontario Lake Trout in Relation to Contaminants and Cultural Practices. *Journal of Great Lakes Research* 21(Supplement 1): 277-285.

Folmar, L.C., N.D. Denslow, V. Rao, M. Chow, D.A. Crain, J. Enblom, J. Marcino and L.J Guillette Jr. 1996. Vitellogenin induction and reduced serum testosterone concentration in feral male carp (*Cyprinus carpio*) captured near a major metropolitan sewer plant. *Environmental Health Prospectives* 104: 1096-1101.

Gillesby, B.E. and T.R. Zacharewski. 1998. Exoestrogens: Mechanisms of action and strategies for identification and assessment. *Environmental Toxicology and Chemistry* 17(1): 3-14.

Great Lakes Fishery Commission. 1980. A Joint Strategic Plan for Management of Great Lakes Fisheries. Ann Arbor, MI.

Great Lakes Fishery Commission. 1997. A Joint Strategic Plan for Management of Great Lakes Fisheries. Ann Arbor, MI.

Green Bay Fishery Resources Office. 1998. Great Lakes Native Fish Restoration: Lake Sturgeon, *Acipenser fulvescens.* Annual Fisheries Stewardship Progress Report, Lake Michigan. Prepared by U.S. Fish and Wildlife Service. December 4.

Green Bay Fishery Resources Office. 1999a. Field Data and Documentation for the 1997 Study: Collection of Lake Trout Eggs for Thiamine Supplementation and Halogenated Aromatic Hydrocarbon Exposure Studies. Prepared by U.S. Fish and Wildlife Service.

Green Bay Fishery Resources Office. 1999b Field Data and Documentation for the 1998 Study: Collection of Lake Trout Eggs for Thiamine Supplementation and Halogenated Aromatic Hydrocarbon Exposure Studies. Prepared by U.S. Fish and Wildlife Service.

Guiney, P.D., P.M. Cook, J.M. Casselman, J.D. Fitzsimmons, H.A. Simonin, E.W. Zabel and R.E. Peterson. 1996. Assessment of 2,3,7,8-tetrachlorodibenzo-p-dioxin induced sac fry mortality in lake trout *(Salvelinus namaycush)* from different regions of the Great Lakes. *Canadian Journal of Fisheries and Aquatic Sciences* 53: 2080-2092.

Guiney, P.D., R.M. Smolowitz, R.E. Peterson and J.J. Stegeman. 1997. Correlation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin induction of cytochrome P4501A in vascular endothelium with toxicity in early life stages of lake trout. *Toxicology and Applied Pharmacology* 143:256-273.

Hagler Bailly Services Inc. 1997a. Field Data and Documentation for 1996 Phase III Task 5 Studies: Collection of Walleye, Brown Trout, and Lake Trout for Contaminant Analysis and Preliminary Determination of Physiological and Deformative Injuries: Lower Fox River/Green Bay NRDA. Prepared for U.S. Fish and Wildlife Service, March 28.

Hagler Bailly Services Inc. 1997b. Summary Documentation: 1996 Collections of Lake Trout Eggs for the Lake Trout Egg Injection and Thiamine Supplementation Studies. Submitted to U.S. Fish and Wildlife Service, Green Bay Ecological Services Field Office. March 28.

Hansen, L.G. 1994. Halogenated Aromatic Compounds. In *Basic Environmental Toxicology,* L.G. Cockerham and B.S. Shane, eds. Boca Raton, FL: CRC Press. pp. 627.

Hansen, M.J., P.T. Schultz and B.A. Lasee. 1990. Changes in Wisconsin's Lake Michigan Salmonid Sport Fishery, 1969-1985. *North American Journal of Fisheries Management* 10: 442-457.

Harries, J.E., D.A. Sheahan, S. Jobling, P. Matthiessen, P. Neall, E.J. Routledge, R. Rycroct, J.P. Sumpter and T. Taylor. 1996. A survey of estrogenic activity in United Kingdom inland waters. *Environmental Toxicology and Chemistry* 15(11): 1993-2002.

Harris, G.E., Y. Kiparissis, and C.D. Metcalfe. 1994. Assessment of the toxic potential of PCB congener 81 (3,4,4′,5-tetrachlorobiphenyl) to fish in relation to other non-ortho-substituted PCB congeners. *Environmental Toxicology and Chemistry* 13: 1405-1413.

Hebert, C.E., R.J. Norstrom, J. Zhu and C.R. Macdonald. 1999. Historical changes in PCB patterns in Lake Ontario and Green Bay, Lake Michigan, 1971 to 1982, from herring gull egg monitoring data. *Journal of Great Lakes Research* 25(1): 220-233.

Helder, T. 1980. Effects of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) on Early Life Stages of the Pike (*Esox lucius L.*). *The Science of the Total Environment* 14: 255-264.

Helder, T. 1981. Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) on early life stages of rainbow trout (*Salmo Gairdneri, Richardson*). *Toxicology* 19: 101-112.

Heming, T. A., and R. K. Buddington. 1988. Yolk absorption in embryonic and larval fishes. In Hoar, W. S., and D. J. Randall, eds. Fish Physiology. New York Academic Press Volume XIA.

Hendricks, J.D., D.N. Arbogast and G.S. Bailey. 1990. Aroclor 1254 (PCB) Enhancement of 7,12-Dimethylbenz(A)-Anthracene (DMBA) Hepatocarcinogenesis in Rainbow Trout. An Abstract from the Proceedings of the American Association for Cancer Research Volume 31, March 1990. 1 pp.

Hendricks, J.D., W.T. Stott, T.P. Putnam and R.O. Sinnhuber. 1981. Enhancement of Aflatoxin B_1 hepatocarcinogenesis in rainbow trout (*Salmo gairdneri*) embryos by prior exposure of gravid females to dietary Aroclor 1254. 203-214 pp.

Henry, T. R., J.M. Spitsbergen, M.W. Hornung, C.C. Abnet, and R.E. Peterson. 1997. Early life stage toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in zebrafish (*Danio rerio*). *Toxicology and. Applied Pharmacology* 142: 56-68.

Holey, M.E., R.W. Rybicki, G.W. Eck, E.H.B. Jr., J.E. Marsden, D.S. Lavis, M. L. Toneys, T.N. Trudeau and R.M. Horrall. 1995. Progress toward lake trout restoration in Lake Michigan. *Journal of Great Lakes Research* 21 (Supplement 1): 128-151.

Holey, M.E. and D.C. Honeyfield. 1999. Documentation and Results: Thiamine Analysis of Lake Trout Eggs and Lake Trout and Brown Trout Livers Collected from Green Bay and Northern Lake Michigan, 1996-1998.

Hornung, M.W., E.W. Zabel and R.E. Peterson. 1996. Additive Interaction between Pairs of Polybrominated Dibenzo-*p*-dioxin, Dibenzofuran, and Biphenyl Congeners in a Rainbow Trout Early Life Stage Mortality Bioassay. *Toxicology and Applied Pharmacology* 140: 345-355.

Jobling, S., D. Sheahan, J.A. Osborne, P. Matthiessen and J.P. Sumpter. 1996. Inhibition of testicular growth in rainbow trout (*Oncorhynchus Mykiss*) exposed to estrogenic alkylphenolic chemicals. *Environmental Toxicology and Chemistry* 15(2): 194-202.

Jones, D.H., D.H. Lewins, T.E. Eurell and M.S. Cannon. 1979. Alteration of the immune response of Channel catfish (*Ictalurus punctatus*) by polychlorinated biphenyls. Abstract from a Symposium on Pathobiology of Environmental Pollutants: Animal Models and Wildlife as Monitors. pp. 385-386.

Jones, M. L., G.W. Eck, D.O. Evans, M.C. Fabrizio, M.H. Hoff, P.L. Hudson, J. Janssen, D. Jude, R. O'Gorman and J.F. Savino. 1995. Limitations to lake trout (*Salvelinus namaycush*) rehabilitation in the Great Lakes imposed by biotic interactions occurring at early life stages. *Journal of Great Lakes Research* 21(Supplement 1): 505-517.

Khan, R.A. and J. Thulin. 1991. Influence of pollution on parasites of aquatic animals. *Advances in Parasitology.* 30: 201-237.

Kinietz, V. W. 1940. The Indians of the Western Great Lakes, 1615-1760. University of Michigan Press, Ann Arbor, MI.

Krueger, C. C., D. L. Perkins, E. L. Mills, and J. E. Marsden. 1995b. Predation by alewives on lake trout fry in Lake Ontario: role of an exotic species in preventing restoration of a native species. *Journal of Great Lakes Research* 21(Supplement 1):458-469.

Lasee, B.A. 1995. Introduction to Fish Health Management. U.S. Fish and Wildlife Service. Onalaska, Wisconsin. 139 pp.

Linscombe, G., N. Kinler and R.J. Aulerich. 1982. Mink (*Mustela vison*). In *Wild Mammals of North America: Biology, Management, Economics*, J.A. Chapman and G.A. Feldhamer, eds. Baltimore, MD: The Johns Hopkins University Press. pp. 629-643.

LMLTTC (Lake Michigan Lake Trout Technical Committee). 1985. *A draft lakewide plan for lake trout restoration in Lake Michigan.* In *Minutes Lake Michigan Committee (1985 Annual Meeting)*, Ann Arbor, MI, Great Lakes Fisheries Commission, March 19, 1985.

Mac, M.J. 1981. Vulnerability of Young Lake Trout to Predation After Chronic Exposure to PCB's and DDE. U.S. Fish and Wildlife Service, ed., In *Chlorinated Hydrocarbons as a Factor in the Reproduction and Survival of Lake Trout (Salvelinus namaycush) in Lake Michigan. Technical Paper No. 105.* Washington, DC: U.S. Government Printing Office. pp. 29-32.

Mac, M.J. 1988. Toxic Substances and Survival of Lake Michigan Salmonids: Field and Laboratory Approaches. M.S. Evans, ed., In *Toxic Contaminants and Ecosystem Health: A Great Lakes Focus.* New York, NY: John Wiley and Sons. pp. 389-401.

Mac, M.J. and R.A. Bergstedt. 1981. Temperature Selection by Young Lake Trout After Chronic Exposure to PCB's and DDT. In *Chlorinated Hydrocarbons as a Factor in the Reproduction and Survival of Lake Trout (Salvelinus namaycush) in Lake Michigan. Technical Paper No. 105,* U.S. Fish and Wildlife Service, ed. Washington, DC: U.S. Government Printing Office. pp. 33-35.

Mac, M.J., W.H. Berlin and D.V. Rottiers. 1981. Comparative Hatchability of Lake Trout Eggs Differing in Contaminant Burden and Incubation Conditions. In *Chlorinated Hydrocarbons as a Factor in the Reproduction and Survival of Lake Trout (Salvelinus namaycush) in Lake Michigan. Technical Paper No. 105,* U.S. Fish and Wildlife Service, ed. Washington, DC: U.S. Government Printing Office. pp. 8-10.

Mac, M.J., C.C. Edsall and J.G. Seelye. 1985. Survival of lake trout eggs and fry reared in water from the upper Great Lakes. *Journal of Great Lakes Research.* 11(4): 520-529.

Mac, M.J. and T.R. Schwartz. 1992. Investigations into the effects of PCB congeners on reproduction in lake trout from the Great Lakes. *Chemosphere* 25(1-2): 189-192.

Mac, M.J., T.R. Schwartz, C.C. Edsall and A.M. Frank. 1993. Polychlorinated biphenyls in Great Lakes lake trout and their eggs: Relations to survival and congener composition 1979-1988. *Journal of Great Lakes Research* 19(4): 752-765.

Madenjian, C.P., T.J. DeSorcie and R.M. Stedman. 1998. Ontogenic and spatial patterns in diet and growth of lake trout in Lake Michigan. *Transactions of the American Fisheries Society* 127: 236-252.

Marcquenski, S.V. and S.B. Brown. 1997. Early Mortality Syndrome in Salmonid Fishes From the Great Lakes. In *Chemically-Induced Alterations in the Functional Development and Reproduction of Fishes,* R. Rolland, M. Gilbertson and R. Peterson, eds. Pensacola, FL: SETAC Press. pp. 135-144.

Marsden, J.E. 1995. Lake trout spawning habitat in the Great Lakes - a review of current knowledge. *Journal of Great Lakes Research* 21 (Supplement 1):487-197.

Martin, N.V. and C.H. Oliver. 1980. The lake charr, *Salvelinus namaycush*. In Charrs: Salmonid Fishes of the Genus Salvelinus. E.K. Balon, ed. The Hague.

Matta, M.B., C. Cairncross and R.M. Kocan. 1998. Possible effects of polychlorinated biphenyls on sex determination in rainbow trout. *Environmental Toxicology and Chemistry* 17(1): 26-29.

McDonald, G., J.D. Fitzsimons and D.C. Honeyfield, eds. 1998. *Early Life Stage Mortality Syndrome in Fishes of the Great Lakes and Baltic Sea*. *American Fisheries Society Symposium 21*. Bethesda, MD: American Fisheries Society. pp. 177.

Mehrle, P.M., D.R. Buckler, E.E. Little, L.M. Smith, J.D. Petty, P.H. Peterman, D.L. Stalling, G.M. DeGraeve, J.J. Coyle, J.J. and W.J. Adams. 1988. Toxicity and bioconcentration of 2,3,7,8-tetrachlorodibenzo-p-dioxin and 2,3,7,8-tetrachlorodibenzofuran in rainbow trout. *Environmental Toxicology and Chemistry* 7: 47-62.

Michigan Department of Community Health. 1998. Michigan Fish Advisory: Important Facts to Know if You Eat Michigan Fish. 59 pp.

Michigan Department of Community Health. 1999. Michigan State Fish Consumption Advisory Charts for Green Bay, Little Bay de Noc, Lake Michigan. www.mdch.state.mi.us/pha/fish/.

Michigan Department of Natural Resources. 1997. 1997 Michigan Fishing Guide. Rules Apply April 1, 1997 to March 31, 1998. 64 pp.

Miller, M.A. 1993. Maternal transfer of organochlorine compounds in salmonines to their Eggs. *Canadian Journal of Fisheries and Aquatic Sciences* 50: 1405-1413.

Mizell, M., J.J. Stegeman, E. Romig, R. Smolowitz, J. Schlezinger, R. Katayani, B. Woodin and M. Mortensen. 1996. Chemically induced cardiovascular defects in developmental stages of vertebrates: Dose-response and phenotypic comparisons in medaka and zebrafish exposed to aryl hydrocarbon receptor agonists. *Biology Bulletin* 191:294-295.

Munkittrick, K.R., M.R. Servos, J.H. Carey and G.J.V.D. Kraak. 1997. Environmental impacts of pulp and paper wastewater: evidence for a reduction in environmental effects at North American pulp mills since 1992. *Water Science & Technology* 35(2-3): 329-338.

Nebeker, A.V., F.A. Puglisi and D.L. DeFoe. 1974. Effect of polychlorinated biphenyl compounds on survival and reproduction of the fathead minnow and flagfish. *Transactions of the American Fisheries Society* 3: 562-568.

Niimi, A.J. 1983. Biological and toxicological effects of environmental contaminants in fish and their eggs. *Canadian Journal of Fisheries and Aquatic Sciences* 40: 306-312.

Peterson, R.E., H.M. Theobald and G.L. Kimmel. 1993. Developmental and reproductive toxicity of dioxins and related compounds: Cross-species comparisons. *Critical Reviews in Toxicology* 23:283-335.

Prince, R. and K.R. Cooper. 1995. Comparisons of the effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin on chemically impacted and nonimpacted subpopulations of *Fundulus heteroclitus*: I. TCDD toxicity. *Environmental Toxicology and Chemistry* 14:579-587.

Rice, C.D., D.H. Kergosien and S.M. Adams. 1996. Innate immune function as a bioindicator of a pollution stress in fish. *Ecotoxicology and Environmental Safety* 33: 186-192.

Rice, C.D. and D. Schlenk. 1995. Immune function and cytochrome P4501A activity after acute exposure to 3,3',4,4',5-pentachlorobiphenal (PCB 126) in channel catfish. *Journal of Aquatic Animal Health* 7: 195-204.

Rottiers, D.V. and R.A. Bergstedt. 1981. Swimming Performance of Young Lake Trout After Chronic Exposure to PCB's and DDE. In *Chlorinated Hydrocarbons as a Factor in the Reproduction and Survival of Lake Trout (Salvelinus namaycush) in Lake Michigan. Technical Paper No. 105,* U.S. Fish and Wildlife Service, ed. Washington, DC: U.S. Government Printing Office. pp. 23-28.

Safe, S. 1990. Polychlorinated biphenyls (PCBs), dibenzofurans (PCDFs), and related compounds: Environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). *Critical Reviews in Toxicology* 21: 51-88.

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.

Schram, S.T. and M.C. Fabrizio. 1998. Longevity of the Lake Superior lake trout. *North American Journal of Fisheries Management* 18:700-703.

Schrank, C.S., S.M. Cormier and V.S. Blazer. 1997. Contaminant exposure, biochemical, and histopathological biomarkers in White Suckers from contaminated and reference sites in the Sheboygan River, Wisconsin. *Journal of Great Lakes Research* 23(2): 119-130.

Scott, W.B. and E.J. Grossman. 1973. *Freshwater Fishes of Canada.* Ottawa, Canada: Canadian Government Publishing Centre, Ministry of Supply and Services Canada, Fisheries Research Board of Canada, Bryant Press Limited. 966 pp.

Selgeby, J. H., B. R. Bronte, E. H. Brown, Jr., M. J. Hansen, M. E. Holey, J. P. Van Amberg, K. M. Muth, D. B. Makaukas, P. C. McKee, D. M. Anderson, C. P. Ferreri, and S. T. Schram. 1995. Lake trout restoration in the Great Lakes: stock-size criteria for natural reproduction. *Journal of Great Lakes Research* 21(Supplement 1): 498-504.

Silberhorn, E.M., H.P. Glauert and L.W. Robertson. 1990. Carcinogenicity of polyhalogenated biphenyls: PCBs and PBBs. *Critical Reviews in Toxicology* 20(6): 440-496.

Sleiderink, H.M., I. Oostingh, A. Goksoyr and J.P. Boon. 1995. Sensitivity of cytochrome p450 induction in dab (*Limanda limanda*) of different age and sex as a biomarker for environmental contaminants in the southern North Sea. *Archives of Environmental Contamination and Toxicology* 28: 423-430.

Smith, B.R. and Tibbles, J.J. 1980. Sea Lamprey (*Petromyzon marinus)* in Lakes Huron, Michigan, and Superior: history of invasion and control, 1936-78. *Canadian Journal of Fisheries and Aquatic Sciences* 37:1780-1801.

Snedecor, G.W., and W.G. Cochran. 1980. Statistical Methods. Ames, Iowa: The Iowa State University Press. 507pp.

Spitsbergen, J.M., M.K. Walker, J.R. Olson and R.E. Peterson. 1991. Pathologic alterations in early life stages of lake trout, *Salvelinus namaycush*, exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin as fertilized eggs. *Aquatic Toxicology* 19: 41-72.

Stauffer, T.M. 1979. Effects of DDT and PCB's on survival of lake trout eggs and fry in a hatchery and in Lake Michigan, 1973 - 1976. *Transactions of the American Fisheries Society* 108: 176-186.

Stegeman, J.J. and M.E. Hahn. 1994. Biochemistry and molecular biology of monooxygenases: current perspectives on forms, functions, and regulation of cytochrome P450 in aquatic species. In *Aquatic Toxicology*, D.C. Malins and G.K. Ostrander, eds. Boca Raton: CRC Press, Inc. pp. 87-206.

Stow, C.A., S.R. Carpenter, L.A. Eby, J.F. Amrhein and R.J. Hesselberg. 1995. Evidence that PCBs are approaching stable concentrations in Lake Michigan fishes. *Ecological Applications* 5(1): 248-260.

Stow, C.A., L.J. Jackson and S.R. Carpenter. 1999. A mixed-order model to assess contaminant declines. *Environmental Monitoring and Assessment* 55: 435-444.

Stratus Consulting Inc. 1998. Fish Consumption Advisories in the Lower Fox River/Green Bay Assessment Area: Final Report. Prepared for U. S. Fish and Wildlife Service, U. S. Department of Justice, U. S. Department of the Interior. November 24.

Stratus Consulting Inc. 1999a. Association Between PCBs, Liver Lesions, and Biomarker Responses in Adult Walleye *(Stizostedium vitreum vitreum)* Collected from Green Bay, Wisconsin. April 13.

Stratus Consulting Inc. 1999b. Injuries to Avian Resources, Lower Fox River/Green Bay Natural Resource Damage Assessment. Prepared for U. S. Fish and Wildlife Service, U. S. Department of the Interior, U. S. Department of Justice. May 7.

Stratus Consulting Inc. 1999c. Injuries to Surface Water Resources, Lower Fox River/Green Bay Natural Resource Damage Assessment. Prepared for U.S. Fish and Wildlife Service, U.S. Department of Interior, U.S. Department of Justice. November 8.

Stratus Consulting Inc. 1999d. PCB Pathway Determination for the Lower Fox River/Green Bay Natural Resource Damage Assessment. Prepared for U. S. Fish and Wildlife Service, U. S. Department of the Interior, U. S. Department of Justice. August 30.

Stratus Consulting Inc. 1999e. Recreational Fishing Damages From Fish Consumption Advisories in the Waters of Green Bay. Prepared for U.S. Fish and Wildlife Service, U. S. Department of Justice, U. S. Department of Interior. November 1.

Sutter, T.R., K. Guzman, K.M. Dold and W.F. Greenlee. 1991. Targets for dioxins: Genes for plasminogen activator inhibitor-2 and interleukin-1ß. *Science* 254:415-418.

Teh, S.J., S.M. Adams and D.E. Hinton. 1997a. Histopathologic biomarkers in feral freshwater fish populations exposed to different types of contaminant stress. *Aquatic Toxicology* 37: 51-70.

Teh, S.J. and D.E. Hinton. 1998. Gender-specific growth and hepatic neoplasia in medaka (*Oryzias latipes*). *Aquatic Toxicology* 41(1-2): 141-159.

Teh, S.J., M.S. Okihiro and D.E. Hinton. 1997b. Final Pathology Report: Histopathological Analysis of Brown Trout *(Salmo trutta),* Lake Trout *(Salvelinus namaycush),* and Walleye *(Stizostedion vitreum vitreum)*: A Data Report for Hagler Bailly Consulting Inc. in Support of the Lower Fox River and Green Bay Natural Resource Damage Assessment Project. University of California at Davis. September 1.

Thuvander, A. and M. Carlstein. 1991. Sublethal exposure of rainbow trout (*Oncorhynchus mykiss*) to polychlorinated biphenyls: effect on the humoral immune response to *Vibrio anguillarum*. *Fish & Shellfish Immunology* 1: 77-86.

Toweill, D.E. and J.E. Tabor. 1982. River Otter *(Lutra canadensis)*. In *Wild Mammals of North America: Biology, Management, Economics*, J.A. Chapman and G.A. Feldhamer, eds. Baltimore, MD: The Johns Hopkins University Press. pp. 668-703.

Tyler, C. R., J. P. Sumpter, and P. R. Witthames. 1990. The dynamics of oocyte growth during vitellogenisis in rainbow trout (*Oncorhynchus mykiss*). *Biology of Reproduction* 43:202-209.

U.S. EPA. 1993a. Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife. Prepared by Environmental Research Laboratory, Office of Research and Development, Duluth, MN. March. EPA/600/R-93/055.

U.S. EPA. 1993b. Wildlife Exposure Factors Handbook: Volume II of II. U.S. Environmental Protection Agency Report #EPA/600/R-93/187b.

U.S. EPA. 1997. 1997 Supplementary Fish Consumption Advisory for Michigan's Great Lakes Waters. One folded page.

U.S. EPA. 1995. The Effects of Great Lakes Contaminants on Human Health. Report to Congress. EPA-905-R-95-017. Great Lakes National Program Office, Chicago, IL.

U.S. EPA and Environment Canada. 1995. *The Great Lakes: An Environmental Atlas and Resource Book*. Chicago, IL: U.S. Environmental Protection Agency. 46 pp.

U.S. Fish and Wildlife Service. 1998. Maps showing locations of walleye tag returns from fish tagged in Michigan waters of Green Bay. 5 pp.

USGS. 1999a. Lake Trout Early Life Stage Mortality: Interactions of the Nutrient Thiamine and Dioxin-like PCBs and Their Mixtures Found in Green Bay. Final report of interagency agreement 14-48-0009-98-978. Prepared for U.S. Fish and Wildlife Service. October 31.

USGS. 1999b. Lake Trout Early Life Stage Mortality: Preliminary Studies into the Interactions of the Nutrient Thiamine and Dioxin-like PCBs and Their Mixtures Found in Green Bay. Final report of interagency agreement 14-48-0009-96-1023. Prepared for U.S. Fish and Wildlife Service. October 31.

University of Wisconsin-Green Bay. 1993. The State of the Bay: A Watershed Perspective. Produced by the UWGB Institute for Land and Water Studies.

Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy, A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, R.X.R.v. Leeuwen, A.K.D. Liem, C. Nolt, R.E. Peterson, L. Poelinger, S. Safe, D. Schrenk, D. Tillett, 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* 106(12): 775-792.

Van Der Oost, R., H. Heida, A. Opperhuizen and N.P.E. Vermeulen. 1991. Bioaccumulation of organic micropollutants in different aquatic organisms: Sublethal toxic effects of fish. *Aquatic Toxicology and Risk Assessment: ASTM STP 1124.* 14: 166-180.

Walker, M.K. and R.E. Peterson. 1994a. Aquatic Toxicity of Dioxins and Related Chemicals. Chapter 11 in *Dioxins and Health*, A. Schecter, ed. New York and London: Plenum Press. pp. 347-387.

Walker, M.K. and R.E. Peterson. 1994b. Toxicity of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin to brook trout (*Salvelinus fontinalis*) during early development. *Environmental Toxicology and Chemistry* 13:817-820.

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. *Canadian Journal of Fisheries and Aquatic Sciences* 51: 1410-1419.

Walker, M.K., P.M. Cook, B.C. Butterworth, E.W. Zabel and R.E. Peterson. 1996. Potency of a complex mixture of polychlorinated dibenzo-p-dioxin, dibenzofuran, and biphenyl congeners compared to 2,3,7,8-tetrachlorodibenzo-p-dioxin in causing fish early life stage mortality. *Fundamental and Applied Toxicology* 30: 178-186.

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. *Aquatic Toxicology* 21: 219-238.

Walker, M.K. and R.E. Peterson. 1992. Toxicity of Polychlorinated Dibenzo-p-Dioxins, Dibenzofurans, and Biphenyls During Early Development in Fish. In *Chemically-Induced Alterations in Sexual and Functional Development: The Human/Wildlife Connection.* Princeton, NJ: Princeton Scientific Publishing Co. pp. 195-202.

Walker, M.K., J.M. Spitsbergen, J.R. Olson and R.E. Peterson. 1991. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity during early life stage development of lake trout (*Salvelinus namaycush*). *Canadian Journal of Fisheries and Aquatic Sciences* 48: 875-883.

Wells, L., and A. L. McLain. 1972. Lake Michigan: effects of exploitation, introductions, and eutrophication on the salmonid community. *Journal of the Fisheries Resource Board of Canada* 29:889-989.

Whitlock, Jr., J.P. 1990. Genetic and molecular aspects of 2,3,7,8-tetrachlorodibenzo-p-dioxin action. *Annual Review of Pharmacology and Toxicology* 30: 251-277.

Whitlock, J.P. 1993. Mechanistic Aspects of Dioxin Action. *Chemical Research in Toxicology* 6:754-763.

Willford, W.A., R.A. Bergstedt, W.H. Berlin, N.R. Foster, R.J. Hesselberg, M.J. Mac, D.R.M. Passino, R.E. Reinert and D.V. Rottiers. 1981. Introduction and Summary. In *Chlorinated Hydrocarbons as a Factor in the Reproduction and Survival of Lake Trout (Salvelinus namaycush) in Lake Michigan. Technical Paper No. 105*, U.S. Fish and Wildlife Service, ed. Washington, DC: U.S. Government Printing Office. pp. 1-7.

Wisconsin Department of Natural Resources. 1976-1994. Health Guide for People Who Eat Sport Fish from Wisconsin Waters. PUBL-IE-019. April.

Wisconsin Department of Natural Resources. 1987. 1987 PCB Lake Trout Egg Concentrations From Sturgeon Bay. 15 pp.

Wisconsin Department of Natural Resources. 1988. Lower Green Bay Remedial Action Plan for the Lower Fox River and Lower Green Bay Area of Concern. 319 pp.

Wisconsin Department of Natural Resources. 1995. Fish Contaminant Monitoring Database, 1971-1995. Provided to Hagler Bailly in electronic format by J. Amrhein, WDNR.

Wisconsin Department of Natural Resources. 1996. Fish movement data, Green Bay and Lake Michigan. Data by WDNR.

Wisconsin Department of Natural Resources. 1999. Lake Michigan Management Reports (Fisheries Data). Great Lakes Fishery Commission, Lake Michigan Committee 1999 Annual Meeting. March 25.

Wisconsin Division of Health and Wisconsin Department of Natural Resources. 1998. Important Health Information for People Eating Fish from Wisconsin Waters. PUB No FH824 98 Rev.

Wisconsin Division of Health and Wisconsin Department of Natural Resources. 1999. Important Health Information for People Eating Fish from Wisconsin Waters. PUB No FH824 99 Rev.

Wisconsin Division of Health and Wisconsin DNR. 1997. Important Health Information for People Eating Fish from Wisconsin Waters. Prepared by the Natural Resources Board, with acknowledgments to the Bureau of Public Health, Great Lakes Fish Advisory Task Force, and Wisconsin Department of Natural Resources staff, PUB No FH824 97.

Wright, P.H., D.E. Tillitt, J. Zajicek, M. Anderson, J. Fitzsimons, M. Holey and D. Honeyfield. 1998. Dioxin toxicity to lake trout embryos with low thiamine levels. Abstract presented at the Society of Environmental Toxicology and Chemistry Annual Meeting, Charlotte, North Carolina. 1 pp.

Zabel, E.W., P.M. Cook and R.E. Peterson. 1995a. Toxic equivalency factors of polychlorinated dibenzo-*p*-dioxin, dibenzofuran and biphenyl congeners based on early life stage mortality in rainbow trout (*Oncorhynchus mykiss*). *Aquatic Toxicology* 31: 315-328.

Zabel, E.W., M.K. Walker, M.W. Hornung, M.K. Clayton and R.E. Peterson. 1995b. Interactions of polychlorinated dibenzo-*p*-dioxin, dibenzofuran, and biphenyl congeners for producing rainbow trout early life stage mortality. *Toxicology and Applied Pharmacology* 134:204-213.

Zelikoff, J.T. 1994. Fish Immunotoxicology. In *Immunotoxicology and Immunopharmacology, Second Edition*, J.H. Dean, A.E. Munson and I. Kimber, eds. New York: Raven Press. pp. 71-94.

Zelikoff, J.T., W. Wang, N. Islam, L.E. Twerdok, D.C. Finch and R.A. Finch. 1996. Applicability of laboratory-developed immunotoxicology assays for predicting pollutant-induced effects in feral populations. 76 pp.

APPENDIX A
PCB CONCENTRATIONS IN WALLEYE LIVERS COLLECTED IN 1997

Table A-1
Fish Identification and Liver PCB Concentration for Walleye Collected in 1997

Sample Location	Fish Identification[a]	Total PCB Concentration (mg/kg wet weight) in Liver
Eastern Green Bay	weeg08/2701	1.33
	weeg08/2702	3.82
	weeg08/2703	2.27
	weeg08/2704	3.86
	weeg08/2805	2.66
	weeg08/2806	3.04
	weeg08/2907	2.93
	weeg08/2908	0.86
	weeg08/2909	1.90
	weeg08/2910	1.47
	weeg08/2911	1.62
	weeg08/2912	1.03
	weeg08/2913	2.43
	weeg09/1514	2.58
	weeg09/1515	2.27
	weeg09/1516	6.55
	weeg09/1517	3.21
Lower Fox River	wefr08/1201	8.68
	wefr08/1202	-[b]
	wefr08/1203	5.79
	wefr08/1204	6.51
	wefr08/1205	4.49
	wefr08/1206	6.80
	wefr08/1207	4.64
	wefr08/1208	7.99
	wefr08/1209	8.49
	wefr08/1210	5.95
	wefr08/1211	5.61
	wefr08/1212	4.91

Table A-1 (cont.)
Fish Identification and Liver PCB Concentration for Walleye Collected in 1997

Sample Location	Fish Identification[a]	Total PCB Concentration (mg/kg wet weight) in Liver
Lower Fox River	wefr08/1213	8.05
	wefr08/1214	5.11
	wefr08/1215	10.6
	wefr08/1216	4.48
	wefr08/1217	5.58
	wefr08/1218	6.17
	wefr08/1219	6.95
	wefr08/1220	5.23
	welg08/2601	1.15
Lower Green Bay	welg08/2602	1.87
	welg08/2603	1.98
	welg08/2604	10.5
	welg08/2605	1.00
	welg08/2606	2.85
	welg08/2607	12.9
	welg08/2608	3.65
	welg08/2609	2.45
	welg08/2610	5.27
	welg08/2611	3.90
	welg08/2612	1.38
Western Green Bay	wewg08/1001	5.17
	wewg08/1002	1.44
	wewg08/1003	2.95
	wewg08/1004	1.93
	wewg08/1005	5.73
	wewg08/1006	3.10
	wewg08/1007	6.45
	wewg08/1008	1.74
	wewg08/1009	3.69
	wewg08/1010	4.12
	wewg08/1311	8.46
	wewg08/1312	2.82
	wewg08/1313	4.27
	wewg08/1314	2.99

Table A-1 (cont.)
Fish Identification and Liver PCB Concentration for Walleye Collected in 1997

Sample Location	Fish Identification[a]	Total PCB Concentration (mg/kg wet weight) in Liver
Lake Patten	welp09/1601	0.24
	welp09/1602	0.00
	welp09/1603	0.00
	welp09/1604	0.00
	welp09/1605	0.00
	welp09/1606	0.00
	welp09/1607	0.00
	welp09/1608	0.00
	welp09/1609	0.00
	welp09/1610	0.00
	welp09/1611	0.00
	welp09/1612	0.00
	welp09/1613	0.00
Lake Winnebago	welw08/1101	0.40
	welw08/1102	0.62
	welw08/1103	0.61
	welw08/1104	0.68
	welw08/1105	0.73
	welw08/1106	0.68
	welw08/1107	0.64
	welw08/1108	1.21
	welw08/1109	0.54
	welw08/1110	1.72
	welw08/1111	2.22
	welw08/1112	1.20
	welw08/601	-[b]
	welw08/602	-[b]
	welw08/603	-[b]
	welw08/604	-[b]
	welw08/605	-[b]
	welw08/606	-[b]
	welw08/607	-[b]
	welw08/608	-[b]
	welw08/609	-[b]

a. Identification code used in field collections.
b. -: not determined.

APPENDIX B
EGG AND FRY VIABILITY AND CONTAMINANT DATA IN 1996-1998 USGS STUDIES

Table B-1
Data from Egg and Fry Rearing Studies Conducted by USGS

	Eggs Collected		Eggs that Died		Number of Eggs Hatched			
Egg Lot I.D.	Total	Fertilized	Total	Fertilized	Normal Fry	Abnormal Fry	Total	Fry Mortality Percent[a]
stbay01.1996	3216	1896	1531	211	1648	37	1685	2.0
stbay02.1996	1592	1414	232	54	1323	37	1360	2.0
stbay03.1996	2044	1822	315	93	1703	26	1729	2.0
stbay05.1996	679	651	43	15	634	2	636	2.0
stbay06.1996	588	572	37	21	543	8	551	98.4
stbay09.1996	246	220	25	3	213	4	217	2.0
stbay10.1996	179	153	33	7	142	4	146	2.0
stbay11.1996	955	879	108	32	797	50	847	2.0
stbay12.1996	903	739	232	68	603	68	671	96.8
stbay13.1996	1275	1182	125	32	1120	30	1150	84.4
stbay14.1996	1199	1134	190	125	973	36	1009	99.8
stbay15.1996	371	349	37	15	319	15	334	2.0
stbay01.1997	965	661	504	200	412	49	461	2.0
stbay02.1997	742	523	393	174	289	60	349	0.8
stbay03.1997	853	553	423	123	413	17	430	4.8
stbay04.1997	1071	756	459	144	555	57	612	38.8
stbay05.1997	1147	454	828	135	280	39	319	0.8
stbay06.1997	1097	164	990	57	97	10	107	2.1
stbay07.1997	730	414	503	187	192	35	227	2.1
stbay08.1997	856	546	479	169	311	66	377	0.8
stbay09.1997	1296	864	716	284	505	75	580	2.8
stbay10.1997	844	153	772	81	66	6	72	12.1
stbay11.1997	971	528	605	162	336	30	366	1.6
stbay12.1997	981	796	348	163	580	53	633	31.6
stbay13.1997	500	322	234	56	253	13	266	0.0

Table B-1 (cont.)
Data from Egg and Fry Rearing Studies Conducted by USGS

	Eggs Collected		Eggs that Died		Number of Eggs Hatched			
Egg Lot I.D.	Total	Fertilized	Total	Fertilized	Normal Fry	Abnormal Fry	Total	Fry Mortality Percent[a]
stbay14.1997	958	934	67	43	860	31	891	100.0
stbay15.1997	527	428	178	79	330	19	349	0.0
stbay16.1997	711	309	514	112	171	26	197	100.0
stbay17.1997	1390	1133	289	32	1068	33	1101	0.0
stbay18.1997	763	647	191	75	542	30	572	0.0
stbay19.1997	1185	1029	195	39	955	35	990	55.4
stbay20.1997	772	732	86	46	666	20	686	0.4
stbay21.1997	1720	1492	247	19	1445	28	1473	1.2
stbay22.1997	771	737	48	14	697	26	723	2.0
stbay23.1997	1173	943	301	71	846	26	872	100.0
stbay24.1997	1072	1023	83	34	974	15	989	0.0
stbay25.1997	909	793	126	10	758	25	783	12.8
stbay26.1997	1060	914	195	49	853	12	865	2.0
stbay27.1997	1408	1172	261	25	1122	25	1147	4.4
stbay28.1997	1132	865	320	53	794	18	812	0.9
stbay01.1998	876	24	866	14	9	1	10	NA[b]
stbay02.1998	800	149	665	14	99	36	135	100.0
stbay03.1998	660	292	416	48	215	29	244	0.0
stbay04.1998	634	252	438	56	173	23	196	0.0
stbay05.1998	678	148	578	48	80	20	100	0.0
stbay06.1998	751	223	612	84	101	38	139	1.0
stbay07.1998	585	233	407	55	155	23	178	100.0
stbay08.1998	504	211	351	58	121	32	153	1.0
stbay09.1998	522	49	498	25	14	10	24	NA[b]
stbay10.1998	947	251	749	53	162	36	198	2.0
stbay11.1998	1133	148	1015	30	100	18	118	43.0
stbay12.1998	853	281	638	66	204	11	215	78.0
stbay13.1998	899	62	882	45	10	7	17	NA[b]
stbay14.1998	1161	471	881	191	235	45	280	58.0
stbay15.1998	992	311	737	56	221	34	255	100.0
stbay16.1998	1418	300	1227	109	168	23	191	0.0
stbay17.1998	1116	53	1083	20	32	1	33	NA[b]
stbay18.1998	1056	822	263	29	741	52	793	1.0

Table B-1 (cont.)
Data from Egg and Fry Rearing Studies Conducted by USGS

	Eggs Collected		Eggs that Died		Number of Eggs Hatched			
Egg Lot I.D.	Total	Fertilized	Total	Fertilized	Normal Fry	Abnormal Fry	Total	Fry Mortality Percent[a]
stbay19.1998	931	225	771	65	136	24	160	1.0
stbay20.1998	1225	888	439	102	674	112	786	1.0
stbay21.1998	819	633	233	47	510	76	586	1.0
stbay22.1998	1081	579	558	56	478	45	523	69.0
stbay23.1998	857	563	353	59	452	52	504	0.0
stbay24.1998	867	369	554	56	288	25	313	1.0
stbay25.1998	882	322	669	109	181	32	213	0.0
stbay26.1998	802	519	356	73	388	58	446	2.0
stbay27.1998	900	818	92	10	791	17	808	70.0
stbay28.1998	711	624	98	11	589	24	613	0.0
stbay29.1998	724	281	457	14	260	7	267	0.0
stbay30.1998	544	411	142	9	380	22	402	0.0
stbay31.1998	717	557	170	10	540	7	547	1.0
stbay32.1998	1275	587	712	24	538	25	563	1.0

a. Measured from normal fry that were transferred and reared for approximately 75 days post-hatch.
b. NA = No measurements made for fry mortality.

Table B-2
Data for Thiamine and Contaminant Analysis on Eggs Collected for USGS Studies

	Thiamine Concentration (pmol/g)		Contaminant Concentrations			
Egg Lot I.D.	Fertilized Eggs	Unfertilized Eggs	Total PCB Conc. (ng/g)	TEQ Conc. from TCDDs and TCDFs (pg/g)	TEQ Conc. from PCBs (pg.g)[b]	Percent Lipid
stbay01.1996	3393.6	3418.5	744.7	NA	0.49	2.14
stbay02.1996	962.4	1572.0	626.6	NA	1.99	3.22
stbay03.1996	1307.0	1441.3[a]	237.0	NA	1.01	2.20
stbay05.1996	1055.0	1163.4[a]	489.0	NA	1.30	2.70
stbay06.1996	475.0	523.8[a]	811.1	NA	1.88	2.81
stbay09.1996	4490.0	4951.5[a]	537.9	NA	1.85	3.74
stbay10.1996	2413.0	2661.0[a]	557.5	NA	2.07	3.24
stbay11.1996	508.5	918.8	494.6	NA	0.39	3.39
stbay12.1996	409.0	451.0[a]	785.2	NA	2.15	2.77
stbay13.1996	621.0	684.8[a]	841.4	NA	2.74	3.91
stbay14.1996	373.0	411.3[a]	281.3	NA	0.65	2.38
stbay15.1996	13765.0	15179.8[a]	374.8	NA	1.12	2.86
stbay01.1997	937.1[a]	1033.4	1440.4	NA	3.96	4.33
stbay02.1997	3143.8[a]	3466.9	1095.3	NA	3.06	2.28
stbay03.1997	1083.9[a]	1195.3	1110.0	NA	3.10	3.58
stbay04.1997	891.5[a]	983.1	1353.4	NA	3.73	2.96
stbay05.1997	3622.6[a]	3994.9	379.8	1.75	1.20	3.68
stbay06.1997	10393.6[a]	11461.8	491.7	7.02	1.49	3.41
stbay07.1997	2564.46[a]	2827.9	926.5	2.45	2.62	3.73
stbay08.1997	4941.5[a]	5449.4	1038.3	NA	2.91	2.54
stbay09.1997	1304.4[a]	1438.5	1425.4	NA	3.92	3.00
stbay10.1997	1284.3[a]	1416.3	424.4	3.48	1.32	3.38
stbay11.1997	1992.9[a]	2197.8	1715.6	NA	4.67	3.65
stbay12.1997	576.3[a]	635.6	535.9	6.19	1.61	3.92
stbay13.1997	4645.6[a]	5123.0	1461.4	NA	4.01	2.84
stbay14.1997	584.0[a]	644.1	675.2	2.62	1.97	4.83
stbay15.1997	1995.3[a]	2200.4	841.4	3.91	2.40	4.10
stbay16.1997	796.6[a]	878.5	925.7	5.52	2.62	3.49
stbay17.1997	809.0[a]	892.2	2469.3	5.97	6.63	3.51
stbay18.1997	1818.2[a]	2005.1	1528.5	NA	4.19	3.00
stbay19.1997	786.9[a]	867.8	734.6	NA	2.12	3.19

Table B-2 (cont.)
Data for Thiamine and Contaminant Analysis on Eggs Collected for USGS Studies

	Thiamine Concentration (pmol/g)		Contaminant Concentrations			
Egg Lot I.D.	Fertilized Eggs	Unfertilized Eggs	Total PCB Conc. (ng/g)	TEQ Conc. from TCDDs and TCDFs (pg/g)	TEQ Conc. from PCBs (pg.g)[b]	Percent Lipid
stbay20.1997	6981.0[a]	7698.5	670.0	NA	1.96	2.20
stbay21.1997	1183.6[a]	1305.2	1143.3	NA	3.19	2.52
stbay22.1997	1474.7[a]	1626.2	28.4	2.14	0.29	3.64
stbay23.1997	640.6[a]	706.5	1593.1	NA	4.36	2.46
stbay24.1997	3350.9[a]	3695.3	846.8	NA	2.42	3.08
stbay25.1997	3204.9[a]	3534.3	2158.2	NA	5.83	3.71
stbay26.1997	3804.5[a]	4195.5	1985.2	NA	5.38	3.33
stbay27.1997	2826.2[a]	3116.7	2036.2	NA	5.51	2.76
stbay28.1997	14253.6[a]	15718.6	636.6	NA	1.87	2.83
stbay01.1998	646.3	942.2	2456.9	NA	6.60	4.43
stbay02.1998	449.7	562.9	1065.9	NA	2.99	4.03
stbay03.1998	1172.8	1918.1	734.8	NA	2.12	3.77
stbay04.1998	6382.9	7173.7	1420.3	NA	3.91	4.87
stbay05.1998	3836.1	5184.8	438.3	3.21	1.35	4.62
stbay06.1998	1656.7	2285.4	1241.1	NA	3.44	4.36
stbay07.1998	335.8	725.7	572.4	2.12	1.70	3.88
stbay08.1998	2019.3	2852.0	1538.4	NA	4.21	3.91
stbay09.1998	3776.6	5284.4	1032.9	NA	2.90	4.45
stbay10.1998	1443.3	2069.8	1854.5	NA	5.04	4.02
stbay11.1998	337.6	546.4	422.9	0.40	1.31	4.16
stbay12.1998	489.7	739.9	398.0	3.07	1.25	3.37
stbay13.1998	6887.5	7998.5	1079.3	NA	3.02	4.47
stbay14.1998	494.8	678.1	391.1	1.55	1.23	4.14
stbay15.1998	202.7	393.6	758.7	3.00	2.19	3.85
stbay16.1998	2709.6	3160.4	1245.7	NA	3.45	4.74
stbay17.1998	5502.6	4976.7	1272.6	NA	3.52	4.14
stbay18.1998	4279.5	5218.6	1352.0	NA	3.73	4.64
stbay19.1998	5715.3	7668.6	1079.1	NA	3.02	4.72
stbay20.1998	819.5	788.6	701.9	NA	2.04	3.69
stbay21.1998	10592.4	13566.8	750.0	NA	2.16	4.38
stbay22.1998	603.8	441.7	1045.2	NA	2.93	4.48

Table B-2 (cont.)
Data for Thiamine and Contaminant Analysis on Eggs Collected for USGS Studies

Egg Lot I.D.	Thiamine Concentration (pmol/g)		Contaminant Concentrations			
	Fertilized Eggs	Unfertilized Eggs	Total PCB Conc. (ng/g)	TEQ Conc. from TCDDs and TCDFs (pg/g)	TEQ Conc. from PCBs (pg.g)[b]	Percent Lipid
stbay23.1998	12784.6	11051.0	1110.0	NA	3.10	4.14
stbay24.1998	958.1	1019.2	1274.4	NA	3.53	4.04
stbay25.1998	1893.7	1252.8	1509.8	NA	4.14	3.59
stbay26.1998	4227.3	4548.9	681.6	7.35	1.99	4.10
stbay27.1998	1850.2	631.3	500.4	1.50	1.52	4.04
stbay28.1998	7318.1	7395.3	1387.9	NA	3.82	3.78
stbay29.1998	5390.3	4849.3	428.2	6.01	1.33	3.89
stbay30.1998	2137.1	1183.4	4852.4	NA	12.83	1.70
stbay31.1998	4498.8	3281.2	701.3	5.90	2.04	3.96
stbay32.1998	2061.4	1273.7	754.0	NA	2.17	3.06

a. Estimated by regression between fertilized egg and unfertilized egg thiamine concentrations.
b. Analyzed only for eggs collected in 1996 (refer to Egg Lot I.D. for collection year). For lots collected in 1997 and 1998, estimated by regression between total PCB concentration and TEQ concentration from PCBs using data for lots collected in 1996.
NA = not analyzed.