

Appendix C

Focused Ecological Risk Assessment Upper Green Bay Portion of the Fox River Site, Green Bay, Wisconsin

FOCUSED ECOLOGICAL RISK ASSESSMENT
UPPER GREEN BAY PORTION OF THE FOX RIVER SITE
GREEN BAY, WI
February 2000

Prepared by:

Mark D. Sprenger, Ph.D.
Environmental Response Team

and

Nancy Beckham
Karen Kracko
Response Engineering and Analytical Contract/
Environmental Response Team

Environmental Response Team Center
Office of Emergency and Remedial Response

TABLE OF CONTENTS

LIST OF TABLES iv

LIST OF FIGURES v

LIST OF ACRONYMS vi

EXECUTIVE SUMMARY vii

1.0 INTRODUCTION AND SITE HISTORY 1

 1.1 Introduction 1

 1.2 Site History 1

2.0 PROBLEM FORMULATION (Steps 3 and 4, U.S. EPA 1997) 2

 2.1 Contaminants of Potential Concern (COPCs) 2

 2.2 Ecological Effects of PCBs 3

 2.3 PCB Fate and Transport 4

 2.4 Ecological Setting 5

 2.5 Complete Exposure Pathways 7

 2.6 Assessment Endpoints 8

 2.6.1 Assessment Endpoint #1: Pelagic Fish Reproduction and Survival 9

 2.6.2 Assessment Endpoint #2: Piscivorous Bird Reproduction and Survival 10

 2.6.3 Assessment Endpoint #3: Piscivorous Mammal Reproduction and Survival 11

 2.7 Conceptual Model 12

 2.8 Selection of Receptor Species 13

 2.8.1 Pelagic Fish 13

 2.8.2 Piscivorous Birds 13

 2.8.3 Piscivorous Mammals 14

 2.9 Testable Hypotheses 14

 2.9.1 Assessment Endpoint #1: Pelagic Fish Reproduction and Survival 14

 2.9.2 Assessment Endpoint #2: Piscivorous Bird Reproduction and Survival 14

 2.9.3 Assessment Endpoint #3: Piscivorous Mammal Reproduction and Survival 14

 2.10 Measurement Endpoints 14

 2.10.1 Measurement Endpoint for Assessment Endpoint #1: Pelagic Fish Reproduction and Survival 15

 2.10.2 Measurement Endpoint for Assessment Endpoint #2: Piscivorous Bird Reproduction and Survival 15

 2.10.3 Measurement Endpoint for Assessment Endpoint #3: Piscivorous Mammal Reproduction and Survival 16

3.0 ASSUMPTIONS 16

4.0 METHODS 18

4.1	Data Compilation (Exposure Characterization)	18
4.1.1	Surface Water PCB Data	19
4.1.2	Sediment PCB Data	19
4.1.3	Fish Whole-Body PCB Data	19
4.1.4	Fish Egg PCB Data	20
4.1.5	Bird Egg PCB Data	21
4.2	Effects Characterization	21
4.3	Methods Used to Evaluate Risk	21
4.3.1	Comparisons of Measured Tissue Concentrations to Literature Values	22
4.3.2	Food Chain Models	23
4.3.3	Nesting Colony Studies	24
5.0	RESULTS OF DATA COMPILATION	24
5.1	Surface Water PCB Data	24
5.2	Sediment PCB Data	24
5.3	Fish Whole-Body PCB Data	24
5.4	Fish Egg PCB Data	25
5.5	Bird Egg PCB Data	25
6.0	RISK CHARACTERIZATION (Step 7)	25
6.1	Assessment Endpoint #1: Pelagic Fish Reproduction and Survival	25
6.1.1	Comparisons of Estimated Fish Egg PCB Concentrations to Literature Values	25
6.1.2	Comparisons of Measured Fish Whole-Body PCB Concentrations to Literature Values	26
6.2	Assessment Endpoint #2: Piscivorous Bird Reproduction and Survival	27
6.2.1	Comparisons of Measured Bird Egg Concentrations to Literature Values	27
6.2.2	Food Chain Models for Piscivorous Birds	28
6.2.2.1	Caspian Tern	28
6.2.2.2	Double-Crested Cormorant	28
6.2.3	Nesting Colony Studies	29
6.2.3.1	Caspian Tern	29
6.2.3.2	Double-Crested Cormorant	31
6.3	Assessment Endpoint #3: Piscivorous Mammal Reproduction and Survival	33
6.3.1	Food Chain Model	33
7.0	UNCERTAINTY ANALYSIS	33
7.1	General Uncertainty Analysis	33
7.2	Site-Specific Uncertainty Analysis	35

7.2.1	Selection of Contaminants of Potential Concern	35
7.2.2	Conceptual Model Limitations	35
7.2.3	Estimates of Exposure Concentration	37
7.2.4	Selection of TRVs	39
8.0	FISH AND WILDLIFE SERVICE INJURY REPORTS	40
8.1	Fish and Wildlife Service Injuries to Fishery Resources Report	40
8.2	Fish and Wildlife Service Avian Injury Report	41
9.0	CONCLUSIONS	42
9.1	Pelagic Fish Reproduction and Survival	42
9.2	Piscivorous Bird Reproduction and Survival	42
9.3	Piscivorous Mammal Reproduction and Survival	44
10.0	LITERATURE CITED	45
APPENDICES		
Appendix A	Toxicity Reference Values	
Appendix B	Life Histories and Exposure Profiles for the Food Chain Models	
Appendix C	PCBs in Walleye from Green Bay and Tributaries	

LIST OF TABLES

Table 1	TRVs Selected for Use in the Upper Green Bay Risk Assessment
Table 2	PCB Concentrations in Surface Water, Upper Green Bay
Table 3	Total PCB Concentrations in Sediment Collected from the Upper Green Bay
Table 4	Total PCB Concentrations in Fish Collected in 1996 from Upper Green Bay
Table 5	Total PCB Concentrations in Fish Collected in 1989 from Upper Green Bay
Table 6	Total PCB Concentrations in Upper Trophic Level Fish Collected from Upper Green Bay
Table 7	Estimated Total PCB Concentrations in Fish Eggs Based on Fish Whole Body PCB Concentrations
Table 8	Total PCB Concentrations in Bird Eggs Collected from Islands In or Near Upper Green Bay
Table 9	Hazard Quotient Calculations for Fish
Table 10	Hazard Quotient Calculations for Bird Eggs
Table 11	Food Chain Model and Chronic Hazard Quotient Calculations for the Caspian Tern
Table 12	Food Chain Model and Chronic Hazard Quotient Calculations for the Double-Crested Cormorant
Table 13	Food Chain Model and Chronic Hazard Quotient Calculations for the Mink
Table 14	Summary of Hazard Quotient Calculation Results

LIST OF FIGURES

Figure 1	Lake Michigan and Green Bay
Figure 2	Green Bay and Associated Islands
Figure 3	Conceptual Model for the Upper Green Bay System
Figure 4	Hazard Quotient Calculation Results by Assessment Endpoint - Maximum
Figure 5	Hazard Quotient Calculation Results by Assessment Endpoint - Mean
Figure 6	Hazard Quotient Calculation Results for Mammals

LIST OF ACRONYMS

AHH	Aryl hydrocarbon hydroxylase
Ah receptor	Aryl hydrocarbon receptor
AUF	Area use factor
BW	Body weight
COPC	Contaminant of Potential Concern
DDD	Dichlorodiphenyl dichloroethane
DDE	Dichlorodiphenyl ethylene
DDT	Dichlorodiphenyl trichloroethane
DME	Drug-metabolizing enzyme
ERA	Ecological risk assessment
EROD	Ethoxyresorufin-O-deethylase
FT	Federal threatened
HQ	Hazard Quotient
kg/day	Kilograms per day
km	Kilometer
K _{ow}	Octanol-water partition coefficient
LD ₅₀	Median lethal dose
LOAEC	Lowest observed adverse effect concentration
LOAEL	Lowest observed adverse effect level
LOD	Limit of detection
LOQ	Limit of quantification
µg/kg µg/kg	Micrograms per kilogram
µg/L	Micrograms per liter
mg/kg	Milligrams per kilogram
mg/kgBW/day	Milligrams per kilogram body weight per day
NRC	Natural Resource Council
NRDA	Natural Resource Damage Assessment
NOAEC	No observed adverse effect concentration
NOAEL	No observed adverse effect level
PCB	Polychlorinated biphenyl
PCDD	Polychlorinated dibenzo- <i>p</i> -dioxins
PCDF	Polychlorinated dibenzofurans
PHHs	Planar halogenated hydrocarbons
QA/QC	Quality assurance/quality control
SE	State endangered
TCDD	2,3,7,8-Tetrachloro- <i>p</i> -dibenzodioxin
TCDD-EQ	2,3,7,8-Tetrachloro- <i>p</i> -dibenzodioxin equivalents
TCDF	2,3,7,8-Tetrachloro- <i>p</i> -dibenzofuran
TEQ	Toxicity equivalents
TRV	Toxicity Reference Value
USGS	United States Geological Survey
UTM	Universal Transverse Mercator

EXECUTIVE SUMMARY

This document presents the results of a focused ecological risk assessment for the upper Green Bay portion of the Fox River site at Green Bay, Wisconsin. This risk assessment serves as an initial ecological risk evaluation leading toward the baseline risk assessment being performed for the Lower Fox River/lower Green Bay (ThermoRetec Consulting Corporation). The objective of this assessment is to determine whether PCBs do not pose a risk to the upper Green Bay system, or whether further risk evaluation is needed. This risk assessment is not the baseline risk assessment for this site, and is not intended to be utilized to derive cleanup levels. This risk assessment was developed based on the eight-step process described in the Ecological Risk Assessment Guidance for Superfund (U.S. EPA 1997).

The baseline risk assessment conducted for the Lower Fox River/lower Green Bay concluded that polychlorinated biphenyls (PCBs) represented the greatest site-related threat to ecological receptors; calculated risks from PCB exposure were 10 to 1,000 times greater than predicted risk from all other chemicals of potential concern (COPCs; ThermoRetec Consulting Corporation). To focus this risk assessment, PCBs were the only contaminant of concern evaluated for the upper Green Bay. Existing PCB data on concentrations in sediment, water, fish tissue, and bird eggs were utilized to evaluate potential risk from PCBs to ecological receptors in the upper Green Bay. No empirical field or laboratory studies were conducted as part of this risk assessment.

Assessment endpoints selected for this risk assessment focused on upper trophic level receptors, based on the ability of PCBs to bioaccumulate in food chains. Direct toxicity of PCBs to benthic organisms was evaluated in the baseline risk assessment conducted for the Lower Fox River/lower Green Bay (ThermoRetec Consulting Corporation) using whole sediment toxicity tests. No acute or chronic toxicity was observed. Based on existing PCB data for lower and upper Green Bay sediment, and because the lower bay is the primary source of PCB-contaminated sediment to the upper bay (Stratus Consulting, Inc. 1999a), PCB concentrations in upper Green Bay are not expected to exceed levels in lower Green Bay. Therefore, assessment endpoints evaluated in the baseline risk assessment for the lower bay which focused on direct toxicity were not evaluated in this risk assessment. The following assessment endpoints were evaluated:

- Pelagic Fish Reproduction and Survival
- Piscivorous Bird Reproduction and Survival
- Piscivorous Mammal Reproduction and Survival

The risk to fish in the upper Green Bay was evaluated using two lines of evidence: 1) measured PCB concentrations in fish tissue; and 2) estimated PCB concentrations in fish eggs were compared with tissue levels published in the literature which have been shown to result in adverse effects to fish. Risk to piscivorous birds was evaluated using three lines of evidence: 1) Measured concentrations in bird eggs were compared to published adverse effect concentrations; 2) Food chain models were employed to calculate dietary exposure concentrations for piscivorous birds. Results of the food chain models were compared with values in the literature (chronic no observed adverse effect levels [NOAELs] or lowest observed adverse effect levels [LOAELs]) which have been associated with toxic effects in birds; 3) Field studies which have evaluated effects of PCBs on birds inhabiting the upper Green Bay were reviewed to supplement conclusions predicted by the first two lines of evidence. Risk to piscivorous mammals was evaluated using a single line of evidence, the results of a food chain model.

The most substantive risk indicated by this assessment are lines of evidence where hazard quotient (HQ) calculations exceeded 1.0 when the LOAEL was used as the effect level and mean PCB concentrations were used as the exposure concentrations. This occurred for the following lines of evidence:

- Caspian tern egg concentration; toxicity reference value (TRV) of 7.6 (decreased hatching success); HQ = 2.1
- Double-crested cormorant egg concentration; TRV of 7.6 (decreased hatching success); HQ = 1.4
- Caspian tern egg concentration; TRV of 8.0 (increased deformity rate); HQ = 1.9
- Double-crested cormorant egg concentration; TRV of 8.0 (increased deformity rate); HQ = 1.3

- Piscivorous mammal food chain model; Natural Resource Damage Assessment (NRDA) data set mean PCB concentration; HQ = 5.3
- Piscivorous mammal food chain model; combined data set mean PCB concentration; HQ = 2.1

Additionally, the food chain model for piscivorous birds utilizing the double-crested cormorant model resulted in a HQ of 1.6 when the LOAEL and maximum fish concentrations from the NRDA data set were used in risk calculations.

The risk characterization for the first assessment endpoint indicated potential risk to pelagic fish reproduction and survival. Hazard quotients calculated using the no observed adverse effect concentration (NOAEC) as the effect level exceeded 1.0 for both egg and whole-body fish tissue PCB concentrations (ranging from 3.9 to 9.5 and 4.0 to 9.4, respectively).

The weight of evidence used to evaluate risk to piscivorous bird reproduction and survival indicates that piscivorous birds are at risk from PCB exposure in the upper Green Bay. Results of the food chain models indicate greater risk using the double-crested cormorant model, which correlates with results observed in field studies (higher deformity rates in Green Bay cormorants than at reference sites).

The food chain model used to evaluate risk to piscivorous mammals indicates they are at risk from PCB exposure at concentrations measured in fish collected in the upper Green Bay. All hazard quotients calculated for the receptor species, mink, exceeded 1.0, and ranged from 2.1 (LOAEL as the effect concentration, mean overall fish PCB concentration) to 397.8 (NOAEL as the effect concentration, maximum fish PCB concentration from the NRDA data set).

1.0 INTRODUCTION AND SITE HISTORY

1.1 Introduction

This ecological risk assessment for the upper Green Bay portion of the Fox River site is a focused evaluation of risk to ecological receptors from PCBs present in the upper section of the bay. This risk assessment serves as an initial ecological risk evaluation leading toward the baseline risk assessment being performed for the Lower Fox River/lower Green Bay (ThermoRetec Consulting Corporation). The objective of this assessment is to determine whether polychlorinated biphenyls (PCBs) do not pose a risk to the upper Green Bay system, or whether further risk evaluation is needed. This risk assessment is not the baseline risk assessment for this site, and is not intended to be utilized to derive cleanup levels. The ecological risk assessment presented here was developed according to the eight step process described in the Ecological Risk Assessment Guidance for Superfund (U.S. EPA 1997).

1.2 Site History

The upper Green Bay portion of the Fox River system is located in Green Bay, Wisconsin (Figure 1). Contamination in the upper Green Bay originated from industrial activities, agricultural activities, and residential surface water runoff along the Lower Fox River, which flows into Green Bay. The Lower Fox River, at the time of this study, was one of the most industrialized rivers in Wisconsin. The Lower Fox River (from Lake Winnebago to Green Bay) had the greatest concentration of pulp and paper mills in the world (ThermoRetec Consulting Corporation). It had received discharges from 15 pulp and/or paper mills, 8 municipal wastewater treatment plants, and one electric generating facility (ThermoRetec Consulting Corporation). As a result of industrial activities, between 190,000 and 375,000 kilograms (kg) of PCBs were discharged into the Lower Fox River from 1954 to the present (ThermoRetec Consulting Corporation). In the mid-1960's, organochlorines were detected in herring gulls nesting on Sister Island in the upper Green Bay (Keith 1966). In the early 1970s, PCBs were detected in water and sediments from the Lower Fox River, and both the Fox River and lower Green Bay were found to contain fish and birds with detectable levels of PCBs in their tissues. The Fox River contributed approximately 92 percent of the PCB loading into the bay in 1989 (DePinto et al. 1994). Other studies have identified up to 362 total contaminants present in sediment, water and biota collected from the Lower Fox River and lower Green Bay (ThermoRetec Consulting Corporation). A more extensive description of the history of the Fox River site is available in the baseline risk assessment for the Lower Fox River and lower Green Bay, which is being prepared through the Wisconsin Department of Natural Resources (ThermoRetec Consulting Corporation).

The risk assessment for the Lower Fox River and lower Green Bay evaluated risks from multiple organic and inorganic contaminants in the Fox River from the outlet of Lake Winnebago to Green Bay and in Green Bay from the outlet of the Fox River to Chambers Island (lower Green Bay). However, contaminants from the Fox River site have been shown to have migrated into upper Green Bay (from Chambers Island up to Lake Michigan, Figures 1 and 2). For example, studies of PCB deposition in bay sediment have shown that PCB-contaminated sediment extends northward along the Door Peninsula for many miles beyond the boundaries of the lower Green Bay (Manchester-Neesvig et al. 1996). Hawley and Niester (1993) estimated that approximately 10 to 33 percent of tributary sediment (most of which is from the Fox River) discharged into the lower bay was transported to the upper bay annually. Based on particle settling velocities in the lower bay and around Chambers Island, Eadie et al. (1991) concluded that PCBs adsorbed to particulate matter can be transported many kilometers (km) before settling. Increased suspended sediment loads from the lower to upper bay were measured during a storm event in September of 1989 (Hawley and Niester 1993). Finally, statistical evaluation of PCB congener patterns in sediment from Lower Fox River, lower Green Bay, upper Green Bay, and Lake Michigan indicates that the

PCB congener pattern in upper Green Bay is more similar to that in lower Green Bay and is unlikely to have been derived from the transport and weathering of Lake Michigan sediment (Stratus Consulting, Inc. 1999a). These studies show that although lower Green Bay is the primary depositional zone for Fox River PCBs, PCBs adsorbed to sediment are being transported from the lower bay to the upper bay.

In addition, available data showed that fish from the upper Green Bay had elevated concentrations of PCBs in their tissue (U.S. EPA 1996; Stratus Consulting, Inc. 1999b and 1999c; Hagler Bailly Services, Inc. 1997). Therefore, since PCBs are one of the primary contaminants associated with the Fox River site, the upper Green Bay is within the extent of contamination of that site. To date, the ecological risks in the upper Green Bay have not been assessed. Therefore, the current risk assessment will evaluate ecological risks in the upper Green Bay from contaminants associated with the Fox River site.

2.0 PROBLEM FORMULATION (Steps 3 and 4, U.S. EPA 1997)

The purpose of problem formulation is to establish the goals, extent, and focus of the baseline ecological risk assessment for the upper Green Bay portion of the Fox River investigation. Problem formulation constitutes Steps 3 and 4 of the U.S. EPA guidance. In the problem formulation phase, the questions and issues that need to be addressed are defined based on potentially complete exposure pathways and ecological effects. Only after these questions and issues are carefully defined should the ecological risk characterization be initiated. The problem formulation presented here is developed according to the guidelines established in the Ecological Risk Assessment Guidance for Superfund (U.S. EPA 1997).

2.1 Contaminants of Potential Concern (COPCs)

A screening level risk assessment was conducted for the Lower Fox River and lower Green Bay (ThermoRetec Consulting Corporation). The contaminants of potential concern (COPCs) were identified based on their concentrations in sediment, surface water, and biota collected from the Fox River and/or lower Green Bay relative to benchmarks; concentration thresholds that represent little or no risk. The volume and spatial extent of each contaminant was also considered when selecting the contaminants to be evaluated. The COPCs selected for further evaluation in the Baseline Risk Assessment for the Lower Fox River/lower Green Bay site were:

- PCBs (total and/or Aroclor 1242)
- 2,3,7,8-Tetrachloro-*p*-dibenzodioxin (2,3,7,8-TCDD or dioxin)
- 2,3,7,8-Tetrachloro-*p*-dibenzofuran (2,3,7,8-TCDF or furan)
- 4,4' Dichlorodiphenyl trichloroethylene (DDT) and its metabolites (dichlorodiphenyl ethylene [DDE], dichlorodiphenyl dichloroethane [DDD])
- Dieldrin
- Arsenic
- Lead
- Mercury

Risk assessment of the above COPCs in the Lower Fox River and lower Green Bay provided a basis for the contaminant selection for the upper Green Bay risk assessment. The baseline risk assessment conducted for the Lower Fox River/lower Green Bay concluded that PCBs represented the greatest threat to ecological receptors. Calculated risks from PCB exposure were 10 to 1,000 times greater than predicted risk from all other COPCs (ThermoRetec Consulting Corporation). It has been well documented that PCBs are the most widespread and dominant contaminant in the Fox River/Green Bay system (ThermoRetec Consulting Corporation 1998); the sources, transport, and fate of PCBs within this system have been extensively characterized. Based on spatial and temporal distributions of PCBs and a statistical analysis of congener patterns in sediment, the Fox

River and the lower Green Bay have been identified as the primary source of PCBs to the upper Green Bay (Stratus Consulting, Inc. 1999a). While the possibility was not discounted that the other contaminants listed above could potentially pose an ecological risk in the upper Green Bay, it was assumed, for the purposes of this risk assessment, that the risk posed by those contaminants was sufficiently evaluated in the ecological risk assessment for the Lower Fox River/lower Green Bay. Therefore, PCBs (as total PCBs) were the only contaminant of concern (COC) evaluated in this risk assessment for the upper Green Bay.

2.2 Ecological Effects of PCBs

The most studied biochemical effect of PCBs in animals is the induction of hepatic mixed function oxidase systems, increasing an organism's capacity to biotransform or detoxify xenobiotic chemicals. Enzymes in this system are sometimes referred to as drug-metabolizing enzymes (DMEs) (Kluwe et al. 1979). Although the increased capacity to detoxify xenobiotic chemicals may appear to benefit an organism, the metabolism of the foreign chemicals can also produce metabolites that are more toxic than the parent compound (Mitchell et al. 1976). In addition, PCB-induced changes in enzyme activity may also alter enzyme substrate concentrations in other metabolic pathways (Montz et al. 1982). Polychlorinated biphenyls also induce microsomal hepatic enzyme systems that metabolize naturally occurring steroid hormones (Peakall 1975). The degree of this enzyme system response has been found to be positively dose-related (Linzey 1987). Polychlorinated biphenyl-induced effects to these hepatic enzyme systems can result in increased liver weight, fatty degeneration, hyalin degeneration, necrosis, hepatocyte formation, and increased hormone metabolism in animals (Batty et al. 1990, Lincer and Peakall 1970, Sanders and Kirkpatrick 1977, Sanders et al. 1974, Stotz and Greichus 1978, Vos 1972, Welsch 1985).

Chlorinated hydrocarbons such as PCBs have been documented as a cause of reproductive dysfunction and mortality in wildlife species (Heaton et al. 1995, Hoffman et al. 1986, Langford 1979). Exposure to PCBs has been found to reduce litter sizes at birth, reduce number of litters, induce longer birthing intervals in mice (Linzey 1987, Merson and Kirkpatrick 1976), and reduce plasma concentrations of estradiol and progesterone in female rats (Johnson et al. 1976). Transplacental movement of PCBs has been reported for humans, rabbits, monkeys, and rats (Storm et al. 1981) causing a dose-dependent reduction in the body weights and survival of exposed mammalian offspring (prenatally as well as postnatally) (Barsotti et al. 1976, Brezner et al. 1984, Fein et al. 1984, Heaton et al. 1995, Wren et al. 1987a,b). Transfer of PCBs to mammalian offspring continues via mother's milk (Wren et al. 1987a). Polychlorinated biphenyls have been implicated as the cause of low embryonic weight in black-crowned night herons (*Nycticorax nycticorax*) (Hoffman et al. 1986). Persistence in courtship behavior was reduced in PCB-fed mourning doves (*Zenaida macroura*) (Tori and Peterle 1983). Reduced sperm concentration, thin-shelled eggs, poor hatching success, and offspring born with teratogenic abnormalities have also been reported (Abrahamson and Allen 1973; Bird et al. 1983; Lowe and Stendell 1991; Scott 1977). Polychlorinated biphenyls have also been shown to transfer from the adult to eggs in fish (Niimi 1982; Mac and Schwartz 1992) and have been implicated in reduced hatching success, larval mortality, and larval growth of fish (Mac and Schwartz 1992; Hendricks et al. 1981; Mac and Edsall 1991; Mac et al. 1993). A more extensive review of the toxic effects caused by PCBs to fish, birds, and mammals can be found in Appendix A.

Much of the toxicity caused by PCBs has been attributed to the planar congeners that resemble TCDD (Geisy et al. 1994). The toxic nature of some prepared PCB mixtures may be associated with trace levels of compounds having four or more chlorine atoms at both the *para* and *meta* positions (Koslowski et al. 1994). This chlorine substitution pattern increases the structural similarity of the congeners to TCDD (Safe 1994). Planar PCBs have affinity for the same cellular receptor (the aryl hydrocarbon or Ah receptor) as TCDD. Dioxin-like PCBs elicit toxic biological responses in animals such as hepatic damage, weight loss, thymic atrophy, dermal disorder,

reproductive toxicity, immunosuppression, teratogenicity, and functional effects to the spleen, adrenal gland and testes (Batty et al. 1990; Sanders et al. 1974).

The specific toxicity reference values (TRVs) selected were based, in part, on the measurement endpoints selected for the risk assessment. A brief discussion on the derivation of TRVs for this risk assessment is provided in Section 4.2; TRV selection is described in detail in Appendix A. The selection of TRVs for the upper Green Bay risk assessment mirrors, as much as possible, the TRVs selected for the Lower Fox River/lower Green Bay risk assessment (ThermoRetec Consulting Corporation).

2.3 PCB Fate and Transport

PCBs are a group of 209 synthetic halogenated aromatic hydrocarbons that are extremely stable, and are resistant to most chemical and biological degradation processes (Eisler 1986; Hornshaw et al. 1983). The persistence of PCBs in the environment is due to their stable carbon-halogen bonds (Risebrough et al. 1968). In general, PCBs have low aqueous solubility (Chou and Griffin 1986), and are lipophilic (Risebrough et al. 1968), allowing them to accumulate in fatty tissues (Hornshaw et al. 1983).

Upon entering an aquatic system, PCBs partition between the water, sediment, particulate matter, and biota (Koslowski et al. 1994). The more lipophilic and hydrophobic a substance, the more concentrated it will be in the sediment and phytoplankton of an aquatic system (Loizeau and Menesguen 1993); PCBs are highly lipophilic and hydrophobic. While it has been shown that transport of PCBs in the dissolved phase can be important during the warmer low flow periods of summer, PCBs generally sorb strongly to sediment particles. It has been shown that PCBs discharged to aquatic environments rapidly sorb to particles and are usually deposited in sediment, often close to the area of discharge (Kalmaz and Kalmaz 1979). After this, dispersal and movement of PCBs in aquatic systems depends largely on the movement of the associated sediment (Connell and Miller 1984).

The fact that sediment transport plays such a significant role in PCB transport in aquatic systems has direct consequences in Green Bay. Studies have indicated that a distinct depositional zone is located northeast of the mouth of the Fox River, along the eastern shore of Green Bay, and extends approximately 27 miles into the bay (Manchester-Neesvig et al. 1996). However, based on measurements of suspended sediment mass flux, Hawley and Niester (1993) estimated that approximately 10 to 33 percent of tributary sediment discharged into the lower bay (the majority of which comes from the Fox River) is transported to the upper Green Bay annually. Similarly, in a study of particle settling velocities in the lower bay and around Chambers Island, Eadie et al. (1991) concluded that PCBs adsorbed to particulate matter can be transported many kilometers within Green Bay before settling. Surface water transport may also be important, as PCBs can be transported in the dissolved phase as well as the particulate phase. Movement of surface water from the lower bay to the upper bay has been documented; most of the flow from the lower to the upper bay occurs along the east side of Chambers Island (Stratus Consulting, Inc. 1999a).

Sedimentation rates in upper Green Bay are generally low, indicating less sediment accumulation than in the lower bay (Manchester-Neesvig et al. 1996). Sediment deposition in the middle of the upper bay may result from events such as storms that move contaminated sediments northward from the lower bay (Manchester-Neesvig et al. 1996). For example, Hawley and Niester (1993) detected an increase in suspended sediment loads from the lower to upper bay during a storm in September of 1989. These studies show that although lower Green Bay is the primary depositional zone for the Fox River, PCB-contaminated sediment is transported with surface water moving from the lower to the upper Green Bay.

Additional potential sources of PCBs to Green Bay include atmospheric deposition and influx from Lake Michigan. Atmospheric deposition has been identified as an important source of PCBs to Lake Superior (Eisenreich et al. 1981) and southern Lake Michigan (Murphy et al. 1981). However, Sweet et al. (1991) estimated that atmospheric deposition of PCBs accounts for less than 10 percent of the total input to Green Bay. The potential contribution from Lake Michigan is unknown; transport of PCBs within the lakes is generally via sediment or biota (Simmons 1984). DePinto et al. (1994) identified the Fox River as the major source of PCBs to Green Bay; they estimated the Fox River contributed 92 percent of the PCB loading to the bay in 1989. To focus this risk assessment, the assumption was made that the primary source of PCBs to Green Bay was the Lower Fox River.

Because PCBs are extremely lipid-soluble, they tend to accumulate in the lipid component, internal organs, and mesenteric fat of organisms (Eisler 1986). Optimum accumulation of PCBs by aquatic biota occurs when planar molecules are substituted with 5 to 7 chlorine atoms (Shaw and Connell 1984). Rapid gill uptake of PCBs has been observed in short-term laboratory experiments with fish (Bruggerman et al. 1981). Generally, when equally exposed, fish accumulate two to three times more PCBs than aquatic invertebrates (Eisler 1986). Once absorbed, PCBs generally partition into the fatty tissues of organisms (Ernst et al. 1976, Phillips 1980, Shaw and Connell 1984). Initially, PCBs concentrate in liver, blood, and muscle; eventually accumulations are highest in adipose tissue and skin. PCB concentrations in a salmonid population were found to be related to fish size as well as fish age (Madenjian et al. 1994).

Controversy exists regarding the relative contribution of food versus direct uptake in determining PCB levels in the tissues of aquatic biota (Rasmussen et al. 1990). Field-collected fish were found to have significantly greater PCB body burdens than laboratory specimens exposed to identical concentrations in water, suggesting that food-chain transfer of PCBs is an important mode of contaminant transfer for top predators (Thomann 1981). Thomann et al. (1992) suggest that PCBs with octanol-water partition coefficients (K_{ow}) greater than 10^5 seem to enter the biota via food-web transfer originating from sediment sources, as opposed to direct uptake from water. Madenjian et al. (1998) indicated that lake trout retain 80 percent of the PCBs that are contained in their food, and concluded that most of the PCB body burden accumulated by lake trout is from their food. Based on the above studies, it was assumed for this risk assessment that dietary uptake was the major route of exposure for upper trophic level organisms.

2.4 Ecological Setting

Green Bay is located in Lake Michigan, in northeastern Wisconsin, within the eastern ridges and lowlands of the state. Green Bay extends 192 km from the mouth of the Fox River northeast to Lake Michigan. Rock Island, Washington Island, and St. Martin's Island mark the separation between Green Bay and Lake Michigan (Figure 2). The largest width of Green Bay is 37 km. The Fox River is the primary tributary to lower Green Bay. Green Bay drains approximately 40,470 square km, which is one-third of the total drainage of Lake Michigan (ThermoRetec Consulting Corporation). The total surface area of Green Bay is 4213 km², of which the upper Green Bay comprises 3260 km² (Gaude 1998). Lower Green Bay is fairly shallow and provides habitat for warm-water fish; half of this area is less than 9.1 m deep. Upper Green Bay is characterized by deeper water, with about 85% of the area more than 9.1 m deep; the upper bay provides mostly deep, cold-water habitat (Stratus Consulting, Inc. 1999b). The deepest part of the bay is 53.6 m deep and is located 6.4 km west of Washington Island.

The benthic community in the bay is expected to consist of a variety of invertebrates, including insects, annelids, molluscs, and crustaceans. A variety of wildlife species are also known or expected to inhabit Green Bay (ThermoRetec Consulting Corporation; Heinz et al. 1984; Ankley et al. 1992). Some of the wildlife species that are expected to use the bay for food or habitat are

listed below (ThermoRetec Consulting Corporation):

Fish

<u>Common name</u>	<u>Scientific Name</u>
Walleye	<i>Stizostedion vitreum</i>
Lake Trout	<i>Salvelinus namaycush</i>
Brown Trout	<i>Salmo trutta</i>
Rainbow Trout	<i>Oncorhynchus mykiss</i>
Brook Trout	<i>Salvelinus fontinalis</i>
Chinook Salmon	<i>Oncorhynchus tshawytscha</i>
Coho Salmon	<i>Oncorhynchus kisutch</i>
White Bass	<i>Morone chrysops</i>
White Sucker	<i>Catostomus commersoni</i>
Carp	<i>Cyprinus carpio</i>
Channel Catfish	<i>Ictalurus punctatus</i>
Brown Bullhead	<i>Ameiurus nebulosus</i>
Shortnose Sturgeon	<i>Acipenser brevirostrum</i>
Lake Sturgeon	<i>Acipenser fulvescens</i>
Rainbow Smelt	<i>Osmerus mordax</i>
Yellow Perch	<i>Perca flavescens</i>
Black Crappie	<i>Pomoxis nigromaculatus</i>
American Gizzard Shad	<i>Dorosoma cepedianum</i>
Emerald Shiner	<i>Notropis atherinoides</i>
Alewife	<i>Alosa pseudoharengus</i>
Minnow spp.	Family Cyprinidae
Darter spp.	<i>Etheostoma spp.</i> , <i>Percina spp.</i> , <i>Ammocrypta spp.</i>

Birds

<u>Common Name</u>	<u>Scientific Name</u>
Mallard	<i>Anas platyrhynchos</i>
Double-crested Cormorant	<i>Phalacrocorax auritus</i>
Forster's Tern	<i>Sterna forsteri</i> (SE)
Common Tern	<i>Sterna hirundo</i> (SE)
Caspian Tern	<i>Sterna caspia</i> (SE)
Black Tern	<i>Chlidonias niger</i>
Herring Gull	<i>Larus argentatus</i>
Little Gull	<i>Larus minutus</i>
Ring-billed Gull	<i>Larus delawarensis</i>
Bald Eagle	<i>Haliaeetus leucocephalus</i> (FT)
Red-breasted Merganser	<i>Mergus serrator</i>
Common Merganser	<i>Mergus merganser</i>
Black-crowned Night Heron	<i>Nycticorax nycticorax</i>
Green Heron	<i>Butorides striatus</i>
Red-winged Blackbird	<i>Agelaius phoeniceus</i>
Tree Swallow	<i>Tachycineta bicolor</i>

Mammals

<u>Common Name</u>	<u>Scientific Name</u>
Mink	<i>Mustela vison</i>
River Otter	<i>Lutra canadensis</i>
Muskrat	<i>Ondatra zibethicus</i>
Beaver	<i>Castor canadensis</i>

SE = Endangered according to the state of Wisconsin
FT = Threatened according to the U.S. Fish and Wildlife Service

2.5 Complete Exposure Pathways

As discussed previously, a large volume of sediment is transported from the Fox River and the inner depositional zone of the lower Green Bay to the upper Green Bay each year. Since PCBs have very low water solubility and a high octanol-water partition coefficient, they are likely to sorb strongly to sediment and thus be transported with the sediment into the upper Green Bay. A variety of organisms reside in and around the upper Green Bay and use the bay for food and/or habitat. It is possible that these organisms are exposed to the contaminants that have been transported into the upper Green Bay from the lower Green Bay.

Benthic invertebrates inhabit upper Green Bay, and are in constant contact with sediment. They are potentially exposed to contaminants via direct contact with sediment and sediment interstitial water. In addition, some benthic invertebrates consume sediment to obtain food. They are also potentially exposed to contaminants by ingesting contaminated food items. Some benthic invertebrates may also be exposed to contaminants via direct contact with surface water since some of these organisms inhabit the top layer of sediment, while others inhabit burrows which are constructed to allow for circulation of surface water throughout the burrow.

Upper Green Bay is inhabited by numerous fish species which occupy different regions of the bay. Benthic fish, such as catfish, feed primarily on the bottom substrate of the bay, ingesting relatively large quantities of sediment, periphyton, and benthic invertebrates. These fish may be exposed to contaminants in the bay via ingestion of contaminated food and water, incidental ingestion of contaminated sediment, and direct contact with contaminated sediment and water. Other species of fish inhabit the open water of the bay and feed on phytoplankton and zooplankton. These fish may be exposed to contaminants via ingestion of contaminated food and water and via direct contact with contaminated water. Since some of these open water fish may also feed on benthic organisms, they may also be exposed to contaminants by ingesting contaminated sediment and benthic invertebrates. Upper trophic level fish, such as walleye, feed on other fish and also inhabit the upper Green Bay. These upper trophic level fish may be exposed to contaminants in the bay by consuming other fish that have accumulated contaminants in their tissues. In addition, these fish may be exposed to contaminants via direct contact with contaminated water or sediment, ingestion of contaminated water, or incidental ingestion of contaminated sediment. Finally, some of these upper trophic level fish may also obtain all or a portion of their diet from benthic invertebrates, and thus may be exposed to contaminants via ingestion of benthic invertebrates or incidental ingestion of sediment. Fish that inhabit the open water of the bay are not expected to spend significant time in contact with the sediment. Therefore, direct contact with contaminated sediment is not expected to be a significant exposure pathway for these types of fish.

Other organisms which utilize the bay for food include a variety of bird species. These birds may potentially be exposed to contaminants by ingesting contaminated food items. They may also be exposed to contaminants via ingestion of contaminated water, incidental ingestion of contaminated sediment, direct contact with contaminated sediment and water, and inhalation.

Mammals, such as mink, also utilize the bay for food. Such mammals may inhabit the islands or shores of the bay and feed on fish or invertebrates. Fish, migrating upstream in the tributaries from Green Bay to spawn, may be consumed by terrestrial mammals utilizing the banks of these rivers. Therefore, mammals inhabiting the banks of Green Bay and its tributaries may potentially be exposed to contaminants by ingesting contaminated food items. They may also be exposed to contaminants by ingestion of contaminated water, incidental ingestion of contaminated sediment, direct contact with contaminated sediment and water, and inhalation.

Particularly lipophilic contaminants are known to adsorb to sediments. Since it has been shown that sediment is transported into the upper Green Bay from the lower Green Bay, lipophilic contaminants in the lower Green Bay are also likely to be transported into the upper Green Bay. Another characteristic of lipophilic contaminants is that they are transported across biological membranes more readily than non-lipophilic contaminants and thus are absorbed by biological organisms readily. Once absorbed, they tend to be stored in fatty tissues, allowing for the accumulation of lipophilic contaminants in these tissues via bioaccumulation. As these contaminants are transferred through the food chain, the concentrations in higher trophic level organisms become greater than concentrations in lower trophic level organisms. This process is known as biomagnification and is of particular importance when evaluating the effects of lipophilic contaminants on upper trophic level receptors. PCBs have high octanol-water partition coefficients and are very lipophilic. Therefore, PCBs are expected to accumulate in receptor tissues and to biomagnify through the food chain. This underscores the significance of the potential exposure pathway through the food chain for upper trophic level fish, birds, and mammals in the upper Green Bay.

It should be noted that the dermal contact and inhalation pathways of exposure were not evaluated. Exposure via these routes is difficult to quantify because little information is available in the literature on exposure rates and contaminant effects via these pathways. For this risk assessment, these exposure pathways were assumed to be insignificant compared to ingestion, due to the ability of PCBs to biomagnify through the food chain.

2.6 Assessment Endpoints

Assessment endpoints are explicit expressions of the actual ecological resources that are to be protected. Valuable ecological resources include those without which ecosystem function would be significantly impaired, or those providing critical resources (e.g., habitat). Appropriate selection and definition of assessment endpoints is critical to the utility of a risk assessment as they focus risk assessment design and analysis. It is not practical or possible to directly evaluate risks to all of the individual components of the ecosystem at the site, so assessment endpoints are used to focus the risk assessment on particular components of the ecosystem that could be adversely affected by the contaminants associated with the site. In general, the assessment endpoints selected for the site were aimed at aquatic and terrestrial organism reproduction and survival.

As discussed in Section 1.1, this risk assessment is an extension of the risk assessment conducted for the Lower Fox River/lower Green Bay (ThermoRetec Consulting Corporation). Therefore, the assessment endpoints for the upper Green Bay stem from those used for the Lower Fox River/lower Green Bay study, which were:

- Functioning Water Column Invertebrate Communities
- Functioning Benthic Invertebrate Communities
- Benthic Fish Reproduction and Survival
- Pelagic Fish Reproduction and Survival
- Insectivorous Bird Reproduction and Survival
- Piscivorous Bird Reproduction and Survival
- Omnivorous Bird Reproduction and Survival
- Piscivorous Mammal Reproduction and Survival

A subset of these assessment endpoints was evaluated for the upper Green Bay. Since the only contaminant to be evaluated in the upper Green Bay risk assessment was PCBs (as discussed in Section 2.1) the assessment endpoints for the upper Green Bay focused on upper trophic level receptors (fish, birds, and mammals). This is because exposure to PCBs in the upper Green Bay is

primarily an issue of bioaccumulation and biomagnification rather than direct toxicity; PCBs are not acutely toxic at levels generally found in the environment. For example, information in the literature indicates that PCBs are not expected to have direct toxic effects on benthic invertebrates at levels found in the upper Green Bay. In one study, sediment from the lower Fox River and lower Green Bay were tested in whole sediment toxicity tests using four different test species. The sediment was aerated first in order to dissipate ammonia. No acute or chronic toxicity was observed for any of the test species in any of the whole sediment toxicity tests (Ankley et al. 1992). PCBs were measured in the sediment and levels as high as 6.57 milligrams per kilogram (mg/kg) total PCBs were detected (Ankley et al. 1992). Since the sediment PCB concentrations in upper Green Bay are not anticipated to exceed those found in the lower Green Bay, any direct threat to the benthic community was assumed to be sufficiently evaluated through the ecological risk assessment (ERA) of the Lower Fox River/lower Green Bay. In the risk assessment for the Lower Fox River/lower Green Bay, two assessment endpoints were evaluated for direct toxic effects to lower trophic level organisms: functioning water column invertebrate communities, and functioning benthic invertebrate communities. As stated above, since direct toxicity is not the primary concern with regard to PCBs and no toxicity was observed in toxicity tests conducted with lower Green Bay sediment, these assessment endpoints were not evaluated in the risk assessment for the upper Green Bay.

Three of the assessment endpoints used for the Lower Fox River/lower Green Bay ecological risk assessment were selected for evaluation in the risk assessment for the upper Green Bay. These assessment endpoints represent upper trophic level receptors that would be expected to be exposed to PCBs which have bioaccumulated and biomagnified in a PCB-contaminated ecosystem. The three assessment endpoints were selected in light of the open, deep water habitat of the upper bay, and were a subset of the assessment endpoints selected for the Lower Fox River/lower Green Bay risk assessment. Assessment endpoints not evaluated in this risk assessment include insectivorous and omnivorous birds. Since the selected assessment endpoint trophic groups feed at higher trophic levels and PCBs bioaccumulate, PCB exposure of the selected trophic groups should be higher. By evaluating and protecting these endpoints which are expected to have the greatest exposure and be most sensitive to potential adverse impacts from exposure to site-related contaminants, the upper bay ecosystem as a whole should also be protected. The specific assessment endpoints that were evaluated in this risk assessment are listed below.

2.6.1 Assessment Endpoint #1: Pelagic Fish Reproduction and Survival

The first assessment endpoint was aimed at pelagic fish reproduction and survival in the upper Green Bay. Fish serve a vital role in nutrient and energy transfer within the bay. Specifically, fish act as a link between aquatic and terrestrial ecosystems and between the benthic and pelagic environments. Fish that consume benthic organisms are consumed by other fish, who are in turn consumed by terrestrial organisms such as mammals and birds. These predator-prey interactions represent a transfer of energy from and within the aquatic ecosystem. Since the number of organisms supported at any position in a food chain depends upon the limits of the energy supply available, the role of energy transfer played by fish is integral to the productivity of an aquatic ecosystem. Furthermore, since energy and nutrient cycles are delicately balanced, even a small decline in the fish population can have detrimental impacts on the balance of energy within an ecosystem.

Fish typically comprise a large proportion of the biomass in an aquatic ecosystem and fill a wide range of trophic positions (e.g., predatory, bottom feeders). Fish serve as predators of zooplankton, periphyton, benthic invertebrates, and other fish. Some fish also serve as food items for predators that inhabit aquatic ecosystems. Also, some fish themselves are piscivorous and consume lower trophic level forage fish. A viable fish population is therefore imperative for the maintenance of viable populations of organisms

that feed on them and upon which they feed.

Fish are also important recreationally and commercially. It has been shown that declines in fish populations associated with chemical contamination have adversely affected commercial and recreational fishing industries in many areas of the country (NRC 1992, Miller et al. 1993). In some areas this has had a major impact on local economies due to losses from decreased tourism and decreased revenues from the commercial sale of fish.

Fish populations are of particular concern due to their role in energy transfer, their role in regulating populations, and their role in maintaining a productive commercial and recreational fishery. Therefore, the first assessment endpoint was aimed at pelagic fish reproduction and survival in the upper Green Bay.

2.6.2 Assessment Endpoint #2: Piscivorous Bird Reproduction and Survival

The second assessment endpoint was aimed at piscivorous bird reproduction and survival in the upper Green Bay. Piscivorous birds are upper trophic level organisms that rely primarily on fish as food. Foraging behavior of piscivorous birds represents a pathway by which nutrients and energy are transferred from aquatic to terrestrial ecosystems. There is a close relationship between terrestrial and aquatic systems due to the nutrient and energy flow between these systems. Nutrients enter lake ecosystems via surface water runoff, input via streams, and water infiltration through the soil. Energy enters lake ecosystems via sunlight and other biological input such as detritus and leaves. Nutrients and energy are transferred from aquatic to terrestrial ecosystems via biological output. An example of a biological output is the act of a piscivorous bird consuming fish. Nutrient and energy cycles between aquatic and terrestrial systems are delicately balanced. Since nutrients and energy are limiting factors in the production of an ecosystem, the transfer of energy from an aquatic to a terrestrial system is essential. Piscivorous birds provide one mechanism by which nutrients and energy are transferred from aquatic to terrestrial ecosystems and are therefore important in the maintenance of balanced nutrient and energy cycles.

Predators are often required to keep prey numbers in check, and impacts on predators could cause detrimental population explosions in prey species. Such population explosions result in an imbalance in the energy and nutrient allocations among the organisms inhabiting the same ecosystem, resulting in declines of affected populations. In an aquatic ecosystem, piscivorous birds help to keep populations of the fish, upon which they feed, in check. By keeping fish populations in check, piscivorous birds indirectly impact population fluctuations of invertebrates and other aquatic organisms. The result is balanced populations of fish and invertebrates, which has commercial, recreational, and ecological benefits.

Piscivorous birds can also be preyed upon by other organisms at even higher trophic levels, such as other birds and mammals. By serving as a food source for these higher trophic level organisms, piscivorous birds also function to maintain the population balance of these higher trophic levels. If the populations of piscivorous bird species declined, the populations of the organisms that prey on piscivorous birds might also decline.

Since piscivorous birds are upper trophic level predators, they are especially susceptible to exposure to contaminants that have accumulated in the organisms upon which they feed. In a freshwater system, birds are common predators of fish. Fish have been shown to accumulate contaminants that are present in aquatic ecosystems. Therefore, birds that

consume fish have the potential to accumulate large concentrations of contaminants in their tissue.

Some birds are resident year-round and some are migratory. The variable mobility of potential avian receptors, the relatively large home range, varied diet, and the often seasonal residency, suggest that the potential for exposure, and the identification of specific exposure routes and concentrations is associated with some uncertainty. Nonetheless, the avian piscivore community is of particular concern due to the potential for exposure and adverse effects in a higher trophic level organism, their role in regulating populations, and their role in energy transfer. Therefore, the second assessment endpoint was aimed at the reproduction and survival of piscivorous birds in the upper Green Bay.

2.6.3 Assessment Endpoint #3: Piscivorous Mammal Reproduction and Survival

The third assessment endpoint was aimed at piscivorous mammal reproduction and survival in the upper Green Bay. Piscivorous mammals are upper trophic level organisms that rely primarily on fish as forage. Foraging behavior of piscivorous mammals represents another pathway by which nutrients and energy are transferred from aquatic to terrestrial ecosystems. As stated above, there is a close relationship between terrestrial and aquatic systems due to the nutrient and energy flow between these systems, and piscivorous mammals provide one mechanism by which nutrients and energy are transferred between ecosystems. Piscivorous mammals can be preyed upon by other organisms at even higher trophic levels, such as other mammals and birds. By serving as a food source for these higher trophic level organisms, piscivorous mammals also function to maintain the population balance of these higher trophic levels. If the populations of piscivorous mammal species declined, the populations of the organisms that prey on piscivorous mammals might also decline.

Since piscivorous mammals are upper trophic level predators, they are especially susceptible to exposure to contaminants that have accumulated in the organisms upon which they feed. In a freshwater system, mammals are common predators of fish. Fish have been shown to accumulate contaminants that are present in aquatic ecosystems. Therefore, mammals that consume fish have the potential to accumulate large concentrations of contaminants in their tissues.

Although the shore area of upper Green Bay is limited relative to the area of the bay itself, piscivorous mammals foraging along the shoreline may be exposed to PCB-contaminated fish. In addition, it is possible that fish migrating upstream in the tributaries of Green Bay to spawn may be consumed by terrestrial mammals utilizing the banks of these rivers. Therefore, mammals inhabiting these upstream areas have the potential to be exposed to significant levels of contaminants originating from the upper Green Bay. The mammalian piscivore community is of particular concern due to the potential for exposure and adverse effects in a higher trophic level organism and their role in energy transfer. Therefore, the third assessment endpoint was aimed at the reproduction and survival of piscivorous mammals in the upper Green Bay.

2.7 Conceptual Model

The conceptual model utilizes contaminant and habitat characteristics to identify critical exposure pathways to the selected assessment endpoints. At the site, contaminants in the water and sediment may come in contact with the aquatic and terrestrial receptors inhabiting the upper Green Bay and its islands and surrounding areas. The potentially complete exposure pathways are described in

detail in Section 2.5. The assessment endpoints selected for this risk assessment are described in section 2.6. The site conceptual model is illustrated in Figure 3.

It should be noted that selection of exposure pathways evaluated in this risk assessment was partially dependent on the availability of existing site-specific information. No site-specific data on PCB concentrations in phytoplankton, aquatic plants, or benthic organisms were available for the upper bay. Exposure pathways not evaluated due to lack of site-specific tissue PCB concentrations include ingestion of phytoplankton; ingestion of sediment, aquatic invertebrates and plants by dabbling ducks; and ingestion of insects by insectivorous birds. However, the selected receptor species feed at higher trophic levels than the receptors in the pathways not being evaluated. Since PCBs bioaccumulate and biomagnify through the food chain, PCB exposure of the selected receptors should be higher than for herbivorous or planktivorous species. Therefore, protection of selected receptor species should be protective of organisms with lower exposure levels.

Exposure pathways that were evaluated in this risk assessment are as follows:

- I. Aquatic Vertebrates (Fish)
 - Direct contact with surface water
 - Direct contact with sediment
 - Ingestion of water
 - Incidental ingestion of sediment
 - Ingestion of fish
- II. Piscivorous Birds
 - Ingestion of surface water
 - Incidental ingestion of sediment
 - Ingestion of fish
- III. Piscivorous Mammals
 - Ingestion of surface water
 - Incidental ingestion of sediment
 - Ingestion of fish

2.8 Selection of Receptor Species

Receptor species were selected as representative of organisms within the complete exposure pathways identified above. Selection was based on potential for exposure to PCBs due to feeding habits or habitat use, sensitivity to adverse effects of PCBs, availability of toxicological data, and consistency with receptors selected for the Lower Fox River/lower Green Bay risk assessment.

2.8.1 Pelagic Fish

Lake trout are top level predators with a high fat content and are therefore likely to accumulate large concentrations of PCBs. Information on the life history of lake trout can be found in Appendix B. Historically, lake trout spawned in Green Bay, utilizing spawning grounds mostly located in the upper bay (Thibodeau 1990). Since the Lake Michigan lake trout population crash in the 1940s and 1950s, lake trout have not spawned in Green Bay, although reproduction is occurring in Lake Michigan. However, although successful reproduction of hatchery-reared trout has occurred in Lake Michigan, sustainable recruitment of lake trout into a fishery has not developed (Holey et al. 1995).

Lake trout have been shown to accumulate PCBs to higher concentrations than any other salmonid species in western Lake Michigan, with mean fillet concentrations approximately two times greater than those in brown trout, chinook salmon, brook trout, rainbow trout, or coho salmon (Miller et al. 1993). Madenjian et al. (1998) indicated that lake trout retain 80 percent of the PCBs that are contained in their food. The authors estimate a net trophic transfer efficiency of 0.73 to 0.89 for lake trout between the ages of 5 and 10 years old. This study also indicated that most of the PCB body burden accumulated by lake trout was from their food. Furthermore, among fish species studied, lake trout have been found to be the most sensitive to PCB-caused fry mortality (Walker et al. 1991).

Given the high degree of accumulation of PCBs in lake trout and their sensitivity to PCB reproductive effects, lake trout are an appropriate receptor species to evaluate pelagic fish reproduction and survival in upper Green Bay.

2.8.2 Piscivorous Birds

Two piscivorous bird species were selected as receptor species representative of piscivorous birds which utilize upper Green Bay: Caspian tern and double-crested cormorant. Information on the life history and an exposure profile for the Caspian tern is provided in Appendix B. Terns may be one of the more sensitive avian species to PCB toxicity (Mineau et al. 1984). Caspian terns generally feed on fish, but will also consume eggs and young of other bird species. In addition, the Caspian tern is currently classified as endangered according to the state of Wisconsin. Based on sensitivity to PCBs and the potential for high exposure to PCBs based on feeding habits, Caspian terns were considered to be an appropriate receptor species representative of piscivorous birds for this risk assessment.

The second species selected as representative of piscivorous birds was the double-crested cormorant. Information on the life history and an exposure profile for the double-crested cormorant is provided in Appendix B. Double-crested cormorants are strict piscivores and have the potential for exposure to PCBs in the upper Green Bay via the consumption of fish. Since they are upper trophic level consumers, they have the potential to accumulate PCBs in their tissues to high concentrations. The concentration of PCBs in eggs of double-crested cormorants has been positively correlated with deformities in hatchlings (Giesy et al. 1994), indicating that a mechanism of toxicity leading to adverse effects from exposure to PCBs may exist in double-crested cormorants. Therefore, double-crested cormorants were also considered to be an appropriate receptor species representative of piscivorous birds for this risk assessment.

2.8.3 Piscivorous Mammals

Mink were selected as receptor species representative of piscivorous mammals which utilize the upper Green Bay area. Information on their life history and an exposure profile for the mink are provided in Appendix B. Life history parameters selected for use in the exposure model are conservative (e.g., highest reported ingestion rate and lowest reported body weight); the objective of this risk assessment is to determine whether no ecological risk is present, or whether further evaluation is needed. The use of conservative assumptions minimizes the possibility of concluding risk is not present when a threat actually does exist.

The habitat of mink includes coastal marshes such as those along the western shore of Green Bay (Chapman and Felhamer 1982). Since a large proportion of the mink's diet is

fish, mink would be expected to accumulate PCBs in their tissues via the consumption of PCB-contaminated fish. Furthermore, of the wildlife species tested, mink are the most sensitive species to the toxicity of PCBs (Eisler 1986). For these reasons, mink were considered to be an appropriate receptor species representative of piscivorous mammals for this risk assessment.

2.9 Testable Hypotheses

Testable hypotheses are specific risk questions that are based upon the assessment endpoints. For this risk assessment, the testable hypotheses were as follows:

2.9.1 Assessment Endpoint #1: Pelagic Fish Reproduction and Survival.

Are levels of site-related contaminants sufficient to cause toxic effects or reproductive impairment in fish that inhabit the upper Green Bay?

2.9.2 Assessment Endpoint #2: Piscivorous Bird Reproduction and Survival.

Are levels of site-related contaminants sufficient to cause toxic effects or reproductive impairment in piscivorous birds that utilize the upper Green Bay?

2.9.3 Assessment Endpoint #3: Piscivorous Mammal Reproduction and Survival.

Are levels of site-related contaminants sufficient to cause toxic effects or reproductive impairment in piscivorous mammals that utilize the upper Green Bay?

2.10 Measurement Endpoints

Each of the testable hypotheses was evaluated using one or more measurement endpoints. The number of measurement endpoints chosen for each assessment endpoint was determined by the type of habitat, the mechanism(s) of toxicity, and the availability of existing data. When more than one measurement endpoint was used to evaluate a single assessment endpoint, a weight-of-evidence approach was employed, whereby the measurement endpoints were treated as lines of evidence. The overall risk to each assessment endpoint was then determined based on the results of the evaluation of each line of evidence, having taken into consideration the degree of importance of each line of evidence.

The measurement endpoints were selected to represent the mechanisms of toxicity and exposure pathways for the assessment endpoints, and to answer questions posed by the testable hypotheses for each assessment endpoint. Similar to the assessment endpoints, the measurement endpoints for this study stemmed from the measurement endpoints selected for the risk assessment of the Lower Fox River/lower Green Bay (ThermoRetec Consulting Corporation). The following measurement endpoints, or lines of evidence, were identified for each of the assessment endpoints in this risk assessment:

2.10.1 Measurement Endpoint for Assessment Endpoint #1: Pelagic Fish Reproduction and Survival.

Two lines of evidence were used to assess whether PCBs are likely to adversely affect survival and reproduction of pelagic fish in the upper Green Bay:

First, data on whole-body concentrations of PCBs in upper trophic level fish collected from the upper Green Bay were obtained from the U.S. Fish and Wildlife Service

database that was used for the Natural Resources Damage Assessment (NRDA), and data collected for the Green Bay Mass Balance Model (Hagler Bailly Services, Inc. 1997; Stratus Consulting, Inc. 1999b; U.S. EPA 1996). These concentrations were assumed to be representative of whole-body tissue concentrations of lake trout inhabiting the upper Green Bay. Measured fish tissue concentrations were compared to values cited in the literature which have been shown to result in toxic effects or reproductive impairment of fish.

Second, estimates of fish egg PCB concentrations were calculated from the whole-body fish tissue concentrations using a ratio calculated based on the data presented in Mac et al. (1993). These estimated fish egg concentrations were then compared with fish egg concentrations of PCBs, derived from the literature, that have been associated with adverse effects in fish.

2.10.2 Measurement Endpoints for Assessment Endpoint #2: Piscivorous Bird Reproduction and Survival

Three lines of evidence were used to evaluate piscivorous bird reproduction and survival in upper Green Bay

First, PCB concentrations measured in bird eggs collected from islands in or near upper Green Bay were ThermoRetec Consulting Corporation concentrations of PCBs in bird eggs cited in the literature which are associated with adverse effects on bird reproduction and survival.

Second, a food chain model for each receptor species was used to estimate daily dietary exposure to PCBs in the upper Green Bay. Data on fish tissue PCB concentrations in the upper Green Bay were obtained from the U.S. Fish and Wildlife Service database used for the NRDA (Hagler Bailly Services, Inc. 1997; Stratus Consulting, Inc. 1999a) and the database compiled for the development of the Green Bay Mass Balance Model (U.S. EPA 1996). Sediment and surface water concentrations to be entered into the food chain models were also obtained from the database compiled for the development of the Green Bay Mass Balance Model. Using the food chain models, a predicted daily PCB dosage was calculated for both receptors. These dosages were then compared with dietary PCB dosages derived from the literature that were associated with toxic effects in birds.

Third, results from published studies in which the effects of PCBs on birds inhabiting the upper Green Bay were evaluated. This information was used to supplement the conclusions drawn from the first two lines of evidence.

2.10.3 Measurement Endpoint for Assessment Endpoint #3: Piscivorous Mammal Reproduction and Survival.

A food chain model for mink was selected as an appropriate measurement endpoint to assess the risk to piscivorous mammal reproduction and survival in the upper Green Bay from exposure to PCBs. Data on fish tissue concentrations of PCBs in the upper Green Bay were obtained from the U.S. Fish and Wildlife Service database used for the NRDA, and the database compiled for the development of the Green Bay Mass Balance Model (U.S. EPA 1996). Sediment and surface water concentrations entered into the food chain models were obtained from the database compiled for the development of the Green Bay Mass Balance Model. Using the food chain model, a predicted daily PCB dosage was calculated for the mink. This dosage was compared with dietary PCB dosages derived from the literature that are associated with toxic effects in mink.

3.0 ASSUMPTIONS

An attempt was made to utilize conservative assumptions throughout this risk assessment due to the uncertainty associated with the risk assessment process. The use of consistently conservative assumptions minimizes the possibility of concluding that risk is not present when a threat actually does exist (i.e., the elimination of false negatives). While there is uncertainty associated with each conservative assumption used, this consistent selection process assures that the uncertainty associated with this type of error will err on the side of a protective outcome. In some cases, there was sufficient information available to justify the use of less conservative assumptions. The assumptions utilized in this risk assessment are described below.

The following conservative assumptions were made to conduct this risk assessment:

- ◆ Maximum contaminant levels measured in tissue and sediment were used in the risk calculations and assumed to be representative of concentrations present site-wide.
- ◆ To calculate total PCB concentrations in fish tissue for the upper Green Bay Mass Balance Model, the concentration of each of the PCB congeners measured for each sample were summed. If a particular congener was not detected in a sample, it was assumed to be present as one-half of either its limit of detection (LOD) or its limit of quantification (LOQ), whichever was reported.
- ◆ Contaminants in food items were assumed to be 100 percent bioavailable and not metabolized and/or excreted during the life of the receptor. Most dietary toxicity reference values (TRVs) are based on administered doses in toxicity tests rather than the resulting absorbed doses. Therefore, this assumption probably does not greatly influence the results of the analysis.
- ◆ For calculations of an area use factor¹ (AUF) for the mink, the minimum reported home range was used.
- ◆ Since most dietary TRVs were derived using dosing intervals shorter than seasonal life history events, it was deemed appropriate to not consider seasonal factors in the life histories of avian receptors for the purposes of this risk assessment. Therefore, breeding territories rather than full migratory ranges were used to calculate AUFs for the Caspian tern and the double-crested cormorant. The portion of the year that these birds have migrated elsewhere and are therefore not utilizing the upper Green Bay was not accounted for in the estimation of their AUFs. It was assumed that these birds are present year round in the upper Green Bay.
- ◆ A literature search was conducted to determine the chronic toxicity of PCBs for use in the food chain models. If no toxicity values could be located for the receptor species, values reported for a closely related species were used. Studies were critically reviewed to determine whether study design and methods were appropriate. If values for chronic toxicity were not available, LD₅₀ (median lethal dose) values were used. For this study, a factor of 100 was used to convert the reported LD₅₀ to a No Observable Adverse Effect Level (NOAEL). A factor of 10 was used to convert a reported Lowest Observable Adverse Effect Level (LOAEL) to a NOAEL. No other safety factors were incorporated into the TRVs selected for this risk assessment. If several toxicity values were reported for a receptor species, the most conservative value was used in the risk calculations as long as the study design, exposure route, mechanism, and species tested were deemed appropriate. For the chronic toxicity endpoints, values obtained from long-term feeding

¹ An area use factor is the ratio of an organism's home range, breeding range, or feeding/foraging range to the area of contamination of a site.

studies were used in preference to those obtained from single dose oral studies.

- ◆ A sediment ingestion rate could not be located for mink; estimated sediment ingestion rates were based on those reported in the literature for a similar species, the raccoon. It was assumed that the sediment ingestion rate of the raccoon, as a percentage of dietary intake, was representative of the sediment ingestion rate for the mink.
- ◆ In the food chain model, the lowest reported body weights and the highest reported ingestion rates for adults were assumed in each case.

The following assumptions were also made to conduct this risk assessment. Some are not conservative (e.g., mean contaminant levels) while others are realistic (e.g., an area use factor of 1.0 for piscivorous birds).

- ◆ Mean contaminant levels measured in tissue and sediment were also used in the risk calculations and assumed to be representative of concentrations present site-wide.
- ◆ PCB concentrations measured in walleye and brown trout were assumed to be representative of concentrations in lake trout. Although lake trout have been found to accumulate the highest concentrations of PCBs found in open-water fish of the Great Lakes (Mac and Schwarz 1992), lake trout data collected under rigorous QA/QC procedures were not available for use in this risk assessment.
- ◆ Dietary composition information was obtained from the literature for the receptor species evaluated using the food chain models. However, simplifications of complex diets were assumed for the receptors. Since fish were the only food items for which PCB residue data existed, the receptors evaluated using the food chain model were assumed to consume 100 percent fish. Fish were assumed to be appropriate surrogates for all other prey species potentially consumed by receptors.
- ◆ It was assumed that Caspian terns and double-crested cormorants could obtain all of their food within the study area.
- ◆ Sediment ingestion rates for the Caspian tern and double-crested cormorant could not be found in the literature. However, due to the open water feeding habits of the Caspian tern and the double-crested cormorant, these receptors were assumed to not incidentally ingest sediment.
- ◆ Numerous studies have documented greater sensitivity of chickens to TCDD-like toxicity compared with other species. Other species tested include pheasants, mallards, goldeneyes, herring gulls, black-headed gulls, common tern and kestrels (Brunstrom 1988, Brunstrom and Reutergardh 1986, Hoffman et al. 1998); all species tested to date have been considerably less sensitive than chickens (Hoffman et al 1998). Dietary LOAELs reported for chickens ranged from 0.0414 to 0.9 milligrams per kilogram body weight per day (mg/kgBW/day), whereas dietary LOAELs reported for other bird species ranged from 1.12 to 36 mg/kgBW/day (Appendix A, Table A-1). We felt a sufficient number of studies had been conducted with other avian species to conclude that effect levels reported for chickens were an anomaly relative to other bird species. Studies in which chickens were the test species were not selected for derivation of the NOAEL and LOAEL in this risk assessment.
- ◆ In some cases, toxicity values in the literature were reported as mg/kg in the diet. These were converted to daily intake (mg/kg BW/day) by using the following formula:

$$\text{Daily Intake (mg/kg BW/day)} = \text{Contaminant Dose (mg/kg diet)} \times \text{Ingestion Rate}$$

$$(\text{kg/day}) \times 1/\text{Bodyweight (kg)}$$

This conversion allowed dietary toxicity levels cited to be converted to a daily dose based on body weight. Contaminant doses are exposure levels utilized in studies which evaluated dietary toxicity of PCBs. All studies evaluated to derive the TRVs utilized in this risk assessment are described in detail, including contaminant dose, in Appendix A, Section A.3.1 and A.4. Life history profiles used to derive exposure parameters (ingestion rates and body weights) for receptor species are presented in Appendix B. Values used for this conversion are summarized in Table A-1 (Appendix A).

4.0 METHODS

4.1 Data Compilation (Exposure Characterization)

Data used in support of the ecological risk assessment was obtained from three original sources. First, fish whole-body PCB concentration data were obtained from a database that was developed for use in the NRDA. Second, additional fish whole-body PCB data as well as surface water and sediment PCB data were obtained from the database developed in support of the Green Bay Mass Balance Model (U.S. EPA 1996). Third, bird egg concentrations as well as information on the success of field populations were obtained from studies in the literature. Of these data sets, the one developed for the NRDA was developed under the most rigorous quality assurance/quality control (QA/QC) procedures. The NRDA data were also the most recent data available and thus was given the most weight in this risk assessment. The data collected in support of the Green Bay Mass Balance Model was older data and has not been fully validated using strict QA/QC procedures. Therefore, these data have a higher level of uncertainty than the NRDA data and were therefore given less weight in the risk assessment. The Mass Balance Model data, however, are the most comprehensive data set available for the upper Green Bay and therefore were considered important supporting data for inclusion in the risk assessment. The QA/QC procedures used to validate the bird data collected from the literature are not known. Therefore, these data were considered the least rigorous data set, but they were also considered to be important information in support of the conclusions of the ecological risk assessment because these types of data do not exist elsewhere. In light of the varying degrees of confidence in the different data sets, the data sets were used both separately and combined in the risk assessment to be able to assign a qualitative level of certainty to each of the conclusions.

4.1.1 Surface Water PCB Data

Surface water data were obtained from the Green Bay Mass Balance data set incorporated into the Fox River Database (<http://www.ecochem.net/FoxRiverDatabaseWeb/default.asp>). This database contains Green Bay Mass Balance data which has been reviewed to eliminate duplicate entries or other anomalies. All data reported for Green Bay Zone IV were utilized in this risk assessment. Green Bay Zone IV, as defined for the Green Bay Mass Balance Model (U.S.EPA 1996), includes the portion of Green Bay north of a line which intersects Chambers Island (Figure 2). Data were reported as “dissolved” and “particulate”; these two fractions were summed to obtain a total PCB concentration for each sample. Any duplicate samples were first averaged to calculate a mean dissolved, particulate and total PCB concentration for that location. Finally, an overall mean and maximum total PCB concentration for surface water in the upper Green Bay was calculated.

Mean and maximum total PCB concentrations in surface water were entered into the food chain models to estimate the expected dosage of PCBs from ingestion of surface water for the Caspian tern, double-crested cormorant, and mink, as described in Section 4.3.2.

4.1.2 Sediment PCB Data

Surface sediment data (0 to 12 inches) were also obtained from the Green Bay Mass Balance data set incorporated into the Fox River Database. All data reported for Green Bay Zone IV were utilized in this risk assessment. Mean and maximum PCB concentrations in surface sediment for the upper Green Bay were calculated. These numbers were used to estimate the expected incidental sediment dosage in the food chain model for the mink.

4.1.3 Fish Whole-Body PCB Data

Fish whole-body PCB data were obtained from two different sources. First, data were available for upper trophic level fish (walleye and brown trout) from the database developed by the U.S. Fish and Wildlife Service and used for the NRDA (Hagler Bailly Services, Inc. 1997; Stratus Consulting, Inc. 1999a). From this database, the total PCB concentration in each sample was calculated by summing the concentrations of each congener and subtracting congener 85 for each sample. Congener 85 was subtracted from the total because the analytical laboratory performing the analysis determined that there was analytical interference with DDE. Due to this interference, it was the opinion of the analytical laboratory, that a sum of all the congeners would have resulted in a gross overestimation of the total PCB concentrations, while the sum of the congener concentrations minus congener 85 was believed to be only a slight underestimation of the total PCB concentrations. Therefore, the congener sum minus congener 85 was determined to be the most appropriate calculation of total PCBs for this data set and was selected for use in this risk assessment.

An overall mean and a maximum total PCB concentration was then calculated for whole-body fish tissue from the NRDA data set. Tissue data from this database are composite samples comprised of three to six fish. The maximum concentration obtained from this data set may underestimate the maximum PCB concentration in individual fish. The resulting concentrations were used both in the food chain models, and in comparisons with fish whole-body PCB concentrations identified in the literature to be associated with adverse effects.

Data for PCB concentrations in fish tissue collected for the Green Bay Mass Balance Model were obtained from a data set extracted from the original Mass Balance Model database and compiled by Stratus Consulting, Inc. in Boulder CO, and from the Mass Balance Model data incorporated into the Fox River Database. Samples from this data set are composite samples comprised of five fish each. In this data set, PCB congener data were available for both upper trophic level fish (walleye and brown trout) as well as forage fish (alewife, carp, and smelt) for the upper Green Bay, corresponding to Region IV of Green Bay for the Mass Balance Model (U.S. EPA 1996). Total PCBs were calculated for each sample by summing the concentrations of each PCB congener detected in each sample. If a particular congener was not detected, it was assumed to be present at one-half of either its LOD or its LOQ, whichever was reported. An overall mean and maximum was calculated separately for forage fish (alewife, carp, and smelt only) and was used in the food chain models. In addition, the data for the upper trophic level fish (walleye and brown trout) were combined with the walleye and brown trout data from the NRDA data set, and an overall mean and maximum total PCB whole-body concentration was calculated from this combined data set. The resulting mean and maximum concentrations were used, as described below, to compare with fish whole-body PCB concentrations that have been associated with adverse effects in the literature.

4.1.4 Fish Egg PCB Data

Estimated concentrations of PCBs in fish eggs were calculated using an egg to whole-body ratio of 0.209 calculated for lake trout using data presented in Mac et al. (1993). Miller (1993) reported mean tissue and egg PCB concentrations in lake trout collected from Lake Michigan; the egg to whole-body ratio calculated from this mean is 0.223. The ratio calculated using the Mac et al. (1993) data was utilized in this risk assessment, as individual fish and egg concentrations were reported rather than means. Similar to the calculation of whole-body data, described in Section 4.1.3, the data obtained from the NRDA database was first taken alone to calculate a mean and a maximum estimated fish egg concentration. To do this, the mean and maximum whole-body PCB concentrations calculated from the NRDA data set were multiplied by 0.209 to obtain the estimated mean and maximum fish egg concentrations for upper trophic level fish from the NRDA data set. A similar calculation (multiplication by 0.209) was performed on upper trophic level fish data from the Mass Balance Model. All estimated fish egg PCB concentrations from the two databases were then combined to obtain an overall mean and maximum estimated PCB concentration in fish eggs. The resulting estimated fish egg PCB concentrations for the NRDA data alone and the combined data were compared with fish egg PCB concentrations in the literature that have been associated with adverse effects in fish, as described in Section 4.3.1.

4.1.5 Bird Egg PCB Data

A variety of published studies have been performed in which bird eggs were collected from islands in and around the upper Green Bay and analyzed for PCBs (e.g., Ewins et al. 1994, Custer et al. in press). These studies were reviewed and data on mean and maximum PCB concentrations reported for bird eggs were compiled. Because these studies were conducted over a broad time span, and since the concentrations of PCBs in bird eggs in the upper Green Bay have generally declined over time (Stratus Consulting, Inc. 1999d), the most recent data available was used to evaluate the present risk from PCBs in the upper Green Bay. Since bird egg PCB data were available for both of the selected receptor species (Caspian tern and double-crested cormorant), data for only these two species were considered.

4.2 Effects Characterization

A comprehensive literature search was conducted to locate studies which evaluated the toxicity of PCBs to ecological receptors. Toxicity reference values (TRVs) were derived based on the results of the literature search. A TRV is a contaminant dose level that is compared with an exposure dose to assess the presence and degree of risk to a receptor or group of receptors from that contaminant. Usually, two TRVs are used to predict ecological risk: a no observable adverse effect level (NOAEL) and a lowest observable adverse effect level (LOAEL). The NOAEL is the highest dose at which adverse effects are not expected to occur, and the LOAEL is the lowest dose at which adverse effects are expected to occur.

Studies located in the literature search were critically evaluated to determine whether they were appropriate to use to derive a TRV. Criteria used to appraise studies included suitability of the test result for evaluating the assessment endpoint, similarity of test organism to selected receptor species, duration of exposure, life stage tested, and ecological relevance of the measured effect. The TRVs selected for this risk assessment were based on high-quality studies which satisfied many or all of the evaluation criteria; they are presented in Table 1. Studies which reported both a LOAEL and NOAEL were selected over studies which reported only one effect level, due to the

uncertainty associated with an unbounded effect level². If only a LOAEL could be identified from the studies, an uncertainty factor of 10 was used to calculate a NOAEL (Dourson and Stara 1983). If a LOAEL could not be located for a receptor, the highest NOAEL was selected, and a factor of 10 was used to calculate a LOAEL. Additional discussion on the TRVs selected for this risk assessment is provided in Section 6.0 (Risk Characterization). The studies used to derive TRVs for this risk assessment are described in detail in Appendix A.

4.3 Methods Used to Evaluate Risk

The hazard quotient (HQ) method (Barnhouse et al. 1986; U.S. EPA 1997) was employed to predict the effects of PCB contamination within the upper Green Bay. This method compares exposure concentrations to ecological endpoints such as mortality, reproductive failure or reduced growth. This is done using chronic toxicity values derived from the literature that are intended to represent a lower dose over a longer duration of exposure, resulting in subtle effects that would be expected to manifest themselves at the population level over the longer term.

The comparisons are expressed as ratios of potential intake values to population effect levels, as follows:

$$\text{Chronic Hazard Quotient} = \frac{\text{Exposure Concentration (Mean or Maximum)}}{\text{Chronic Effect Level (e.g., NOAEL or LOAEL)}}$$

The effect level values for toxicity of PCBs were obtained from published studies, and are summarized in Appendix A. The exposure concentrations and toxicity values were entered into the HQ equation and a HQ was calculated.

If the calculated HQ is greater than one based on a chronic NOAEL, it is an indication that there is a potential chronic risk from that contaminant to the ecological receptor in question. The most significant potential risk is indicated if the HQ exceeds one using mean measured PCB concentrations. It should be noted that the maximum concentration is an actual measured potential exposure concentration; a HQ which exceeds one using the maximum measured PCB concentration is still an indication of potential risk.

A LOAEL is an exposure concentration at which an adverse effect has observed; exposure at this concentration is likely to produce an adverse effect in a receptor. If the HQ is greater than one based on a chronic LOAEL for a particular contaminant, it is an indication that the site levels of that contaminant are likely to produce an adverse effect on survival, reproduction, or growth of the ecological receptor in question. As stated above, the most significant risk is indicated if the HQ exceeds one using mean measured PCB concentrations. In addition, the HQ should be interpreted based on the severity of the effect reported.

4.3.1 Comparisons of Measured Tissue Concentrations to Literature Values

The literature was reviewed to identify fish whole-body, fish egg, and bird egg PCB concentrations that are associated with toxicity. The literature on toxicity-associated tissue levels is summarized in Appendix A. Based on the studies found in the literature,

² A study which reports both a NOAEL and LOAEL (a “bounded” effect level) was considered preferable to studies which reported only one effect level. If an unbounded LOAEL is reported, this does not mean that the concentration is the lowest concentration at which an adverse effect may be observed; it is simply the lowest concentration tested in a particular study.

a no observed adverse effect concentration (NOAEC) and a lowest observed adverse effect concentration (LOAEC) for effects associated with PCB concentrations in fish whole bodies, fish eggs, and bird eggs were developed. The mean and maximum PCB concentrations for each tissue matrix were divided by the toxic threshold tissue concentrations (NOAECs and LOAECs) derived from the literature for each tissue matrix, resulting in a HQ. An HQ greater than 1.0 indicates a potential ecological risk.

Due to the differing degrees of confidence in the two sources of fish tissue data, the fish data were treated in two ways. First, the maximum and mean whole-body PCB concentrations from the database used to support the NRDA was used. Since this database contained whole-body PCB concentrations for two upper trophic level fish species (brown trout and walleye), thus representing the upper trophic level measurement endpoint species (lake trout), and since the data collected for the NRDA were collected under rigorous QA/QC procedures, the resulting HQ is associated with a high level of confidence. However, the NRDA database was comprised of only eight composite samples of fish, and maximum concentrations in individual fish may be underestimated. Therefore, a separate evaluation was conducted in which data from the Green Bay Mass Balance Model database was combined with the NRDA database in order to calculate overall maximum and mean PCB concentrations. To do this, only data for upper trophic level fish (brown trout and walleye) from the Mass Balance Model were used, since this was expected to represent whole-body PCB concentrations in the measurement endpoint species (lake trout) better than whole-body concentrations of forage fish, which were also available in the Mass Balance Model database. Since the Mass Balance Model database was comprised of twelve composite whole-body fish samples, this combined data set decreases the uncertainty derived from having only eight data points upon which to base an evaluation, as would have been the case if only the NRDA data were used.

This information obtained from comparing measured fish tissue and bird egg concentrations and estimated fish egg concentrations to literature values contributed to the risk characterization for the following assessment endpoints:

- ◆ Pelagic fish reproduction and survival
- ◆ Piscivorous bird reproduction and survival

4.3.2 Food Chain Models

Food chain models were used to characterize risk for the following assessment endpoints:

- ◆ Piscivorous bird reproduction and survival
- ◆ Piscivorous mammal reproduction and survival

The effect level values for dietary toxicity of PCBs were based on published studies, and are summarized in Appendix A. The exposure concentrations were estimated by employing a food chain model for each measurement endpoint (e.g., the mink) associated with an assessment endpoint (e.g., piscivorous mammals). In these food chain models, ingestion rates of PCBs for each receptor species were determined based on measured concentrations of PCBs in water, sediment, and food items collected from the upper Green Bay as well as known or estimated water, sediment, and food ingestion rates and body weights of each receptor species (Appendix B).

For this risk assessment, both maximum and mean contaminant exposure scenarios were modeled for each receptor. To model the maximum contaminant exposure scenario, the maximum water, sediment, and fish PCB concentrations were entered into the food chain

models to estimate a maximum contaminant dose for each receptor species. Likewise, to model the mean exposure scenario, the mean measured PCB concentrations in water, sediment, and fish were entered into the food chain models to estimate a mean contaminant dose for each receptor species.

Sediment and fish tissue PCB concentrations were entered into the models as wet weight concentrations to be compared with the toxicity values derived from the literature, which were also entered into the models on a wet weight basis. In addition, the water concentrations entered into the models were for the sum of the dissolved plus particulate PCBs because this represents a more realistic estimate of exposure via oral ingestion of water.

The fish data from the NRDA and the Mass Balance Model databases were treated in two ways in the food chain models. First, the maximum and mean whole-body PCB concentrations from the NRDA database were calculated and entered separately into the food chain models. Since the NRDA database contained whole-body PCB concentrations for only upper trophic level fish species (brown trout and walleye) rather than forage fish species, an overestimation of the PCB dosage from the ingestion of fish is expected since upper trophic level fish are expected to accumulate greater concentrations of PCBs than forage fish. However, since the NRDA database is the only source of data collected using rigorous QA/QC procedures, it was deemed appropriate to use these data in the food chain models. It should be noted that the resulting HQs may be higher than if PCB concentrations for forage fish were used. The Mass Balance Model data set, on the other hand, comprised data for three forage fish species (alewife, carp, and smelt). Therefore, a mean and maximum PCB concentration for forage fish only were calculated from the Mass Balance Model database and were also entered separately in the food chain models. The use of the Mass Balance Model forage fish data helps to address the uncertainty derived from using upper trophic level fish PCB concentrations from the NRDA database to represent forage fish PCB concentrations in the food chain models.

Uncertainty was also associated with the surface water (birds and mink) and sediment (mink only) PCB concentrations that were entered into the food chain models, since these data were also obtained from the Mass Balance Model database. To address this uncertainty in the food chain models, an HQ was calculated for the ingestion of fish alone as well as for the ingestion of fish, sediment, and water together. As a result, the influence of the sediment and water data on the final HQs could be determined, and the uncertainty derived from using the Mass Balance Model sediment and water data in the food chain models could be qualitatively evaluated.

4.3.3 Nesting Colony Studies

The results from published nesting colony studies were used as a third line of evidence to evaluate the risk to piscivorous birds inhabiting the upper Green Bay. Studies that have examined reproductive injuries in bird colonies in the upper Green Bay were summarized and used to support the conclusions regarding risk to the following assessment endpoint:

- ◆ Piscivorous bird reproduction and survival

5.0 RESULTS OF DATA COMPILATION

5.1 Surface Water PCB Data

Fifty-seven surface water samples were collected in the Upper Green Bay study area in support of

the Green Bay Mass Balance Model (U.S. EPA 1996). Total PCB concentrations in surface water ranged from 0.00028 to 0.00311 micrograms per liter ($\mu\text{g/L}$), with a mean concentration of 0.001 $\mu\text{g/L}$ (Table 2).

5.2 Sediment PCB Data

Twenty-eight surface sediment samples were collected in the upper Green Bay Study area. Sediment concentrations ranged from 2.4 to 27.07 micrograms per kilogram ($\mu\text{g/kg}$) wet weight, with a mean PCB concentration of 11.33 $\mu\text{g/kg}$ wet weight (Table 3).

5.3 Fish Whole-Body PCB Data

Fish tissue samples collected for the NRDA data set were composite samples comprised of three to six individual fish. Overall, a total of 10 walleye and 25 brown trout were included in the composite samples. PCB concentrations ranged from 1.17 to 1.98 mg/kg wet weight in brown trout, and 4.61 to 7.26 mg/kg wet weight in walleye (Table 4). Mean and maximum fish tissue concentrations from this data set were 3.23 and 7.26 mg/kg wet weight, respectively. These concentrations were used as the measurement endpoint for Assessment Endpoint #1, and also in food chain models for piscivorous birds and mammals.

PCB concentration ranges measured in fish collected in support of the Mass Balance Model were as follows: 0.11 to 4.20 mg/kg wet weight in forage fish, 1.70 to 3.90 mg/kg wet weight in brown trout, and 0.62 to 5.90 mg/kg wet weight in walleye (Table 5). The mean PCB concentration in forage fish was 1.28 mg/kg wet weight, while mean PCB concentration in upper trophic level fish was 2.98 mg/kg wet weight. Forage fish concentrations were used in food chain models for piscivorous birds and mammals.

To evaluate Assessment Endpoint #1, brown trout and walleye tissue data from both data sets was combined to obtain an overall mean and maximum PCB concentrations in upper trophic level fish of 3.04 and 7.26 mg/kg wet weight, respectively (Table 6).

5.4 Fish Egg PCB Data

Estimated concentrations of PCBs in fish eggs were calculated using an egg to whole-body ratio of 0.209 calculated for lake trout using data presented in Mac et al. (1993). Lake trout whole-body and egg PCB concentrations were reported; the egg concentrations (wet weight) were divided by the whole-body PCB concentrations (wet weight) to calculate the above ratio. Using mean and maximum PCB concentrations from the NRDA data set, a mean and maximum egg PCB concentration of 0.68 and 1.52 mg/kg wet weight was calculated (Table 7). When upper trophic level fish data from the NRDA and Mass Balance data set were combined, the estimated mean egg concentration is 0.64 mg/kg wet weight, and maximum egg concentration is 1.52 mg/kg wet weight.

5.5 Bird Egg PCB Data

Several studies were located which reported PCB concentrations measured in Caspian tern and double-crested cormorant eggs from the upper Green Bay study area (Table 8). The most recent data available was selected for use in this risk assessment. Ewins et al. (1994) reported a mean concentration of 15.8 mg/kg wet weight in Caspian tern eggs collected on Gravelly Island in 1991 (Table 8). Maximum and individual egg concentrations were not reported. Custer et al. (in press) reported mean and maximum PCB concentrations of 10.4 and 20.1 mg/kg wet weight, respectively, in double-crested cormorant eggs collected on Spider Island in 1994 and 1995.

6.0 RISK CHARACTERIZATION (Step 7)

6.1 Assessment Endpoint # 1: Pelagic Fish Reproduction and Survival

6.1.1 Comparisons of Estimated Fish Egg PCB Concentrations to Literature Values

Numerous studies have demonstrated that the early life stages of fish are most sensitive to PCB toxicity, and that PCBs are transferred from maternal tissue to eggs (Ankley et al. 1992, Newsted et al. 1995, Larsson et al. 1993). These studies are summarized in Appendix A. Reported NOAEC and LOAEC concentrations ranged from 0.17 to 3.7 mg/kg wet weight, and 0.31 to 5.1 mg/kg wet weight, respectively. Based on study characteristics (e.g., study design, presence of contaminants other than PCBs), a reported LOAEC of 1.6 mg/kg wet weight (Hendricks et al. 1981) and an estimated NOAEC of 0.16 mg/kg wet weight were selected as the most appropriate TRVs for this risk assessment.

Using data from the NRDA database, estimated mean PCB concentrations in eggs were 0.68 mg/kg, wet weight and maximum egg PCB concentrations were 1.52 mg/kg, wet weight. When data from the NRDA database and the Green Bay Mass Balance Model were combined, mean and maximum egg PCB concentrations were 0.64 and 1.52 mg/kg wet weight, respectively. All HQs calculated using the NOAEC exceeded 1.0, and ranged from 4.0 to 9.5 (Table 9). None of the HQs calculated using the LOAEC exceeded 1.0.

Results of risk calculations for fish egg concentrations indicate potential risk to pelagic fish reproduction and survival in the upper Green Bay.

6.1.2 Comparisons of Measured Fish Whole-Body Concentrations to Literature Values

Numerous studies have been conducted with fish in which adverse effects on reproductive endpoints have been observed, and whole-body concentrations of PCBs in adults have been measured. These studies are summarized in Appendix A. Reported NOAEC and LOAEC concentrations ranged from 1.6 to 11.6 mg/kg wet weight, and 9.3 to 429 mg/kg wet weight. No effect concentrations reported in studies in which growth was the measured endpoint ranged from 32 to 645 mg/kg wet weight.

An alternative way to determine whole-body concentrations at which adverse effects would be expected is to estimate a whole-body concentration based on an egg concentration that is associated with adverse effects. This method was derived based on the fact that whole-body concentrations are often available, while fish egg concentrations are not. Early life-stages are most sensitive to adverse effects of PCBs, therefore it is important to identify maternal whole-body concentrations which result in critical egg/fry PCB concentrations. Mac et al. (1993) reported lake trout whole-body and egg concentrations of PCBs; when the egg PCB concentrations (wet weight) were divided by the whole body PCB concentrations (wet weight), a mean ratio of 0.209 was calculated. Using this ratio, an expected lake trout whole-body concentration can be calculated based on a lake trout egg concentration. When the egg LOAEC concentration of 1.6 mg/kg wet weight, cited above, is divided by 0.209, a whole-body concentration that would be expected to elicit adverse effects of 7.7 mg/kg wet weight was calculated. Since this method provided the lowest LOAEC for whole-body fish PCB concentrations, a LOAEC of 7.7 mg/kg wet weight, and a calculated NOAEC of 0.77 mg/kg wet weight were used to evaluate the effects of PCBs on fish survival and reproduction in the upper Green Bay.

Because the LOAEC selected for fish egg concentrations was used to derive a whole-body concentration that would be expected to elicit adverse effects, these two lines of evidence are functionally the same. However, whole-body PCB concentrations are easier to measure than egg concentrations (sample collection is not seasonally limited); therefore use of this method to identify a common measurement (whole-body PCB concentration) that targets the most sensitive life stage was determined to be valid.

Using data from the NRDA database, mean whole-body fish PCB concentrations were 3.23 mg/kg wet weight and maximum whole-body fish PCB concentrations were 7.26 mg/kg wet weight. When data from the NRDA database and the Green Bay Mass Balance Model were combined, mean and maximum whole-body PCB concentrations in upper trophic level fish were 3.04 and 7.26 mg/kg wet weight, respectively. All HQs calculated using the NOAEC exceeded 1.0, and ranged from 3.9 to 9.4 (Table 9). None of the HQs calculated using the LOAEC exceeded 1.0.

Because HQs calculated for fish tissue concentration using the NOAEC as the effect level exceed 1.0, pelagic fish reproduction and survival in the upper Green Bay is potentially at risk from PCB exposure.

6.2 Assessment Endpoint #2: Piscivorous Bird Reproduction and Survival

6.2.1 Comparisons of Measured Bird Egg Concentrations to Literature Values

Field and laboratory studies have been published which correlate concentrations of PCBs in bird eggs with adverse effects on survival, growth, or reproduction. Observed effects include reduction in hatching success, eggshell production and female fertility (Scott 1977, Platonow and Reinhart 1973, McLane and Hughes 1980, Hoffman et al. 1993). These studies are summarized in Appendix A. Reported NOAEC and LOAEC concentrations of PCBs in bird eggs ranged from 0.36 to 39 mg/kg wet weight, and 1.5 to 105 mg/kg wet weight, respectively. The lowest exposure concentrations at which adverse effects were observed were reported in studies conducted with chickens. Numerous studies have documented the greater sensitivity of chickens to TCDD-like toxicity compared with other bird species. Other species tested include pheasants, mallards, goldeneyes, herring gulls, black-headed gulls, common tern and kestrels (Brunstrom 1988, Brunstrom and Reutergardh 1986, Hoffman et al. 1998); all species tested to date have been considerably less sensitive than chickens (Hoffman et al. 1998). A possible explanation for this difference in sensitivity is a difference in concentration of the Ah receptor or its binding affinity for TCDD. This receptor is present in the early stages of chick embryo development but was not found in turkey embryos (Brunstrom and Lund 1988). Because of their greater sensitivity, studies in which chickens were the test species were not selected for derivation of the NOAEC and LOAEC in this risk assessment. The NOAEC of 4.7 mg/kg wet weight and LOAEC of 7.6 mg/kg wet weight reported by Hoffman et al. (1993) for common terns were selected for use in this risk assessment; the adverse effect observed was decreased hatching success.

Measured mean PCB concentrations in Caspian tern and double-crested cormorant eggs were 15.8 mg/kg wet weight, and 10.4 mg/kg wet weight, respectively (Table 10). All HQs calculated for mean PCB concentrations in bird eggs and NOAEC or LOAEC values exceeded 1.0. The maximum concentration measured in cormorant eggs was 20.1 mg/kg wet weight (Custer et al. in press). Hazard quotients calculated using the NOAEC and LOAEC were 4.3 and 2.6, respectively. No maximum concentration was reported by Ewins et al. (1994) for tern eggs, however hazard concentrations calculated using the mean and both effect levels exceeded 1.0, indicating potential risk. Use of the maximum

concentration in risk calculations would only increase the magnitude of the calculated HQ. All HQs calculated for bird egg PCB concentrations exceeded 1.0, indicating that piscivorous bird species utilizing the upper Green Bay are at risk.

Ludwig et al. (1996) reported a NOAEC of 0.8 mg/kg; the adverse effect measured in this study was deformity rate. This concentration was also evaluated in this risk assessment for comparative purposes, however it should be noted that this is an unbounded NOAEC and it was not selected as the sole TRV for this reason. All HQs calculated using this NOAEC exceeded 1.0, and ranged from 13 (mean concentration in double-crested cormorant eggs) to 25.1 (maximum concentration in double-crested cormorant eggs; Table 10). Use of this NOAEC does not change the conclusions of this risk assessment, namely that piscivorous birds utilizing the Upper Green Bay are at risk based on measured egg PCB concentrations.

6.2.2 Food Chain Models for Piscivorous Birds

A literature search was conducted to evaluate dietary toxicity of PCBs to bird species. The results of the literature search are presented in Appendix A. No studies were found in which dietary toxicity of PCBs to either of the selected receptor species (Caspian tern and double-crested cormorant) was tested. Reported NOAEL and LOAEL concentrations for other avian species ranged from 0.0158 to 2.0 mg/kg BW/day, and 0.0414 to 275 mg/kg BW/day, respectively. As before, studies in which chickens were the test species were not selected for derivation of the NOAEL and LOAEL in this risk assessment due to the documented greater sensitivity of this species to adverse effects from PCB exposure. A TRV was selected for this risk assessment based on the ecological significance of the observed adverse effects (reproductive success and behavior), and study design where PCBs were the only dietary contaminant present. A LOAEL of 1.12 mg/kg BW/day reported in studies using ring doves (Peakall and Peakall 1973, Peakall et al. 1972) and mourning doves (Tori and Peterle 1983) was selected as the TRV for this risk assessment. A NOAEL of 0.112 mg/kg BW/day was calculated from this LOAEL using an accepted conversion factor of 10 (Dourson and Stara 1983).

Dietary exposure concentrations for the two piscivorous bird receptor species were calculated using life history parameters summarized in Appendix B. For each species, the following exposure scenarios were evaluated:

- Ingestion of fish with mean and maximum PCB concentrations from the NRDA database (upper trophic level species)
- Ingestion of fish with mean and maximum PCB concentrations from the Green Bay Mass Balance Model database (forage species only)
- Ingestion of fish and ingestion of surface water (water data from the Green Bay Mass Balance Model)

Hazard quotient calculations were done using the NOAEL and LOAEL as the effect level for each of the above scenarios. Results of the food chain model calculations are presented in Table 11 (Caspian Tern) and Table 12 (Double-crested cormorant).

6.2.2.1 Caspian Tern

Hazard quotients calculated using the NOAEL and mean and maximum PCB concentrations in fish from the NRDA database, and maximum concentrations in fish from the Mass Balance Model database exceeded 1.0 (2.0, 4.6 and 2.6, respectively). None of the HQs obtained utilizing the LOAEL in the calculation exceeded 1.0. Adding ingestion of surface water to the exposure calculations had no impact on the results of the HQ calculations (HQs of 2.0, 4.6 and 2.6 for

mean and maximum PCB concentration from the NRDA data set and maximum concentration from the Mass Balance data set), indicating food ingestion is the primary source of contaminant exposure for this species.

Results of the HQ calculations indicate Caspian terns utilizing the upper Green Bay as a foraging area may potentially be at risk from dietary exposure to PCBs.

6.2.2.2 Double-Crested Cormorant

All HQs calculated using the NOAEL as the effect level exceeded 1.0 for this species (Table 12). Ingestion of fish with mean and maximum PCB concentrations from the NRDA database resulted in HQs of 7.2 and 16.2. Calculations using mean and maximum fish PCB concentrations from the Mass Balance Model resulted in HQs of 2.9 and 9.4, respectively. An HQ of 1.6 was calculated using the LOAEL as the effect level and maximum fish concentrations from the NRDA database. None of the other calculations done using the LOAEL resulted in an HQ which exceeded 1.0. As with the Caspian tern, including water ingestion in the exposure scenario had no impact on calculated HQs, indicating that food ingestion is the major exposure route for this species.

Because some HQs calculated for this species exceeded 1.0 when either effect level was evaluated, a food chain exposure using the double-crested cormorant model indicates piscivorous birds utilizing the upper Green Bay are at risk from PCB exposure.

6.2.3 Nesting Colony Studies

6.2.3.1 Caspian Tern

Ludwig and Ludwig (undated report) performed a field study during the 1986 nesting season and looked at rates of deformities and reproductive success in Caspian terns nesting on Gravelly and Gull Islands in upper Green Bay as well as islands in Lake Michigan, Lake Superior, and Lake Huron, the latter of which served as a reference site. The authors found no evidence of developmental defects in Caspian terns nesting in the upper Green Bay. However, they did observe the lowest hatching rate of all the study areas to be in Saginaw Bay and the upper Green Bay, with hatching success on Gravelly and Gull Islands measured to be 72 percent and 71 percent, respectively, compared with a range of 81 to 84 percent in the remaining colonies.

A similar study (Kurita and Ludwig 1988) was performed in 1988 in which Caspian tern eggs were collected from colonies nesting on Gravelly and Gull Islands in the upper Green Bay as well as in Lake Huron, Lake Superior, and Lake Michigan. Eggs were examined for viability and developmental deformities and grouped into four categories: live-normal, dead-normal, infertile, and deformed. The deformed category included both dead- and live-deformed. Unclassifiable and rotten eggs were classified as dead-normal. In the upper Green Bay, 13 Caspian tern eggs were classified as live-normal, 3 as infertile, and 2 as deformed. Organochlorine residues were examined in conjunction with these results, but unlike the cormorants, no trends could be established between PCB residues and rates of deformities in Caspian terns.

In 1990, Mora et al. (1993) examined productivity and colony site tenacity in

relation to PCB concentrations in blood samples collected from Caspian terns nesting in the Great Lakes, including Gravelly and Gull Islands in upper Green Bay. They found that productivity, as measured by the number of eggs laid, hatching success, and fledging success, was not significantly different between the upper Green Bay and the other colonies, even though PCB concentrations measured in the blood samples were greater in Caspian terns collected in upper Green Bay and Saginaw Bay compared with the other colonies. However, the authors report that the hatching success rates observed in this study, which ranged from 74 to 82 percent for all of the colonies studied, were less than the hatching success of Caspian tern colonies nesting in Texas where 85 percent success has been observed and in Finland where 85 to 95 percent success has been noted. Colony site tenacity was exceptionally low in the upper Green Bay colonies (56.5 percent) compared with the other colonies studied (81.2 to 100 percent). The authors explain that Caspian terns are less likely to return to their original breeding area if they experience poor reproduction during the previous year. When natal site tenacity is examined, a correlation is observed with PCB concentrations in blood samples by region, where natal site tenacity decreases with increasing PCB concentrations. However, this correlation is based on a small number of data points. Therefore, more data is needed to confirm this relationship.

Ludwig et al. (1996) summarized a variety of studies conducted from 1987 to 1991, in which field observations of Caspian tern egg death rates and deformity rates were made and either total PCBs or toxicity equivalents (TEQs) were measured in eggs for colonies in the Great Lakes, including Green Bay. The Green Bay colonies had the highest deformity and egg death rates of all the Great Lakes colonies studied except for Saginaw Bay, another region that is known to contain high levels of contamination. However, data specific to the upper Green Bay could not be deciphered from the data presented. Nonetheless, the authors found a significant correlation between TEQs and deformity rates in hatched tern chicks and dead eggs as well as egg death rates, although only egg death rates exhibited a strong correlation ($r^2 = 0.68$). Poor correlations were observed between total PCBs and the observed adverse effects.

Ewins et al. (1994) present the results of a 1991 study on Caspian terns nesting in colonies in the Great Lakes, including two islands (Gravelly and Gull Islands) in the upper Green Bay. Although observations were performed on both islands, eggs were only taken from Gravelly Island. Reproductive output was measured by determining the number of active nests per colony, and by monitoring the nests for numbers of eggs, hatching success, and number of young fledged per nest. Average rates of population change were determined by comparing nest counts for the 1991 study with a count that was conducted in 1980. The results indicated that even though the concentrations of PCBs and dichlorodiphenylethylene (DDE) in the eggs were highest on Gravelly Island and Saginaw Bay, there was no evidence of an overall adverse reproductive effect on Caspian terns in the upper Green Bay, since the number of young per pair was well above the minimum value of 0.6 established by Ludwig (1965) to maintain population stability. Furthermore, a dramatic increase in the number of active Caspian tern nests on Gravelly and Gull Islands in the upper Green Bay was observed from 1980 to 1991. The authors caution in basing definitive conclusions on this study in light of the results of the study by Mora et al. (1993) that indicate that PCBs may be affecting certain reproductive parameters such as natal region fidelity (tendency to return to their original breeding area) in the

upper Green Bay.

The results of the above studies are not conclusive that Caspian terns are at risk from PCBs in the upper Green Bay. The data presented suggest that PCBs are not associated with adverse effects on endpoints such as hatching success and deformities, but one study found a strong negative correlation between Caspian tern site tenacity and PCBs. This indicates that some subtle reproductive effects may be manifesting themselves in the upper Green Bay as a result of exposure to PCB contamination.

6.2.3.2 Double-Crested Cormorant

Ludwig and Ludwig (undated report) performed a field study during the 1986 nesting season and looked at rates of deformities and reproductive success in double-crested cormorants nesting on islands in upper Green Bay (Gravelly and Little Gull Islands) as well as in Lake Michigan, Lake Superior, and Lake Huron; Lake Huron was used as the reference site. They found that the rates of deformities were higher in the upper Green Bay compared with all other sites. Nine cormorants were observed with deformities, including crossed bill, chick edema, unabsorbed yolk sac, dwarfism, and an opaque covering over the eye. It is unclear whether the last deformity is chemically-induced, but the other deformities are similar to those observed in the laboratory as a result of exposure to PCBs (Ludwig et al. 1996). In addition, the lowest hatching rates were also observed in the upper Green Bay, with 63 percent hatchability in upper Green Bay versus 74 percent observed in the reference area (Lake Huron).

A similar study (Kurita and Ludwig 1988) was performed in 1988 in which double-crested cormorant eggs were collected from colonies nesting on Little Gull Island in the upper Green Bay as well as on islands in Lake Huron, Lake Superior, Lake Michigan. Eggs were examined for viability and developmental deformities and grouped into four categories: live-normal, dead-normal, infertile, and deformed. The deformed category included both dead- and live-deformed. Unclassifiable and rotten eggs were classified as dead-normal. In the upper Green Bay, a high rate of reproductive abnormalities was observed. Specifically, 18 cormorant eggs were classified as live-normal, 15 as infertile, and 8 as deformed. Organochlorine residues were examined in conjunction with these results, and it was found that total PCBs were correlated with the numbers of live deformities in cormorant chicks, while rates of dead-normal, dead-deformed, and infertile eggs were better correlated with coplanar PCBs and other chlorinated hydrocarbons.

Fox et al. (1991) performed a review of all studies conducted between 1979 and 1987 in which double-crested cormorants were examined for bill deformities in colonies in the Great Lakes, including Green Bay, as well as four reference areas. They found that the prevalence of chicks with bill defects in Green Bay was markedly greater than all other regions during this time interval. These differences were statistically significant ($p < 0.05$) between Green Bay and the North Channel, Alpena, and Lake Erie, and the difference approached significance ($p < 0.1$) for all other regions. The study also determined that the probability of observing a cormorant chick in Green Bay with a malformed bill was 10 to 32 times greater than for colonies in the reference areas. The incidence of bill defects was significantly greater in Green Bay compared with all other regions studied except for Lake Ontario. Bill defects were observed in

73 percent of the colonies observed in Green Bay, as compared with only 6 percent of the colonies observed in the reference areas. The authors suggest a chemical etiology for the observed bill defects, since an investigation into the cause of similar bill defects in Forster's terns indicated that the defects were associated with increased liver-to-body mass ratios and elevated aryl hydrocarbon hydroxylase (AHH) activity. Furthermore, the authors stated that all three of the more toxic non-ortho PCB congeners have been isolated from tissues of cormorant chicks with crossed bills collected from Green Bay. Two of these congeners are known to cause craniofacial abnormalities in laboratory animals. Although the data presented in this study do not allow one to distinguish between the upper and lower Green Bay colonies, the data presented clearly demonstrate that craniofacial abnormalities were high in double-crested cormorants nesting in Green Bay as a whole between 1979 and 1987 and that these defects may have been caused by exposure to polychlorinated aromatic hydrocarbons such as PCBs.

Tillitt et al. (1992) examined reproductive success of double-crested cormorants from 1986 to 1988 in colonies in and around the Great Lakes. They found that egg mortality was significantly greater in all of the Great Lakes nesting colonies, including the upper Green Bay colonies (Little Gull, Snake, and Gravelly Islands), where egg mortality ranged from 32 to 39 percent. At the reference area (Lake Winnipegosis), egg mortality was only 8 percent. Total PCB concentrations in eggs ranged from 0.05 and 14.8 $\mu\text{g/g}$ wet weight. The authors found a significant correlation between total PCB concentrations in eggs and egg mortality ($p=0.045$). However, the coefficient of determination (r^2) was only 0.319, indicating that much of the variance in egg mortality was not explained by this general linear model. A significant correlation was also observed between egg mortality and the H4IIE rat hepatoma bioassay-derived 2,3,7,8-tetrachloro-*p*-dibenzodioxin equivalents (TCDD-EQ) concentrations ($p \leq 0.0003$, $r^2 = 0.703$). The eggs were analyzed for total PCBs, polychlorinated dibenzo-*p*-dioxins (PCDD), and polychlorinated dibenzofurans (PCDF)-type planar halogenated hydrocarbons (PHHs), and only PCBs were detected. This indicates that PCBs are the main contaminant associated with the observed egg mortality in double-crested cormorants in the Great Lakes colonies, including upper Green Bay.

Ludwig et al. (1996) summarized a variety of studies conducted from 1986 to 1991, in which field observations of double-crested cormorant egg death rates and deformity rates were observed and either total PCBs or TCDD-EQs were measured in eggs for colonies in the Great Lakes, including the upper Green Bay. Deformity rates were higher in all Great Lakes colonies than at a reference colony. Of all the Great Lakes colonies studied, the upper Green Bay had the highest rate of egg deformities (6.14 per thousand for upper Green Bay versus a range of 0.69 to 3.6 per thousand for the other Great Lakes colonies). Similarly, the egg death rate for Green Bay was higher than any other colony studied, although data specific to the upper Green Bay could not be deciphered from the data presented for Green Bay. PCB concentrations ranged from 0.8 mg/kg wet weight at the reference colony to 7.3 mg/kg in eggs collected from Green Bay. The authors found a significant correlation between hatching and deformity rates and both PCBs and TCDD-EQs, indicating that PCBs are playing a large role in the cormorant egg death and deformity rates observed in the upper Green Bay.

The weight of evidence based on the results presented in the studies summarized

above indicate that double-crested cormorants are experiencing adverse reproductive effects in the upper Green Bay. Deformities such as crossed bills, edema, unabsorbed yolk sac, and dwarfism as well as embryo mortality are characteristic of abnormalities observed as a result of exposure to polychlorinated aromatic hydrocarbons such as PCBs. This indicates that double-crested cormorants are at risk from PCBs in the upper Green Bay.

6.3 Assessment Endpoint #3: Piscivorous Mammal Reproduction and Survival

6.3.1 Food Chain Model

A literature search was conducted to evaluate dietary toxicity of PCBs to mammals, and results are presented in Appendix A. Numerous studies were located in which mink were the test species. Because mink are the selected receptor species for this risk assessment, and have been shown to be particularly sensitive to PCBs, these studies were the only mammal studies reviewed to derive the TRV. Reported LOAEL concentrations ranged from 0.055 to 1.1 mg/kg BW/day. The reported effect observed at the 0.055 mg/kgBW/day concentration was decreased kit growth. Reproductive effects (kit survival) were observed at exposure concentrations of 0.5 and 0.72 mg PCB/kg diet (Restum et al. 1998 and Heaton et al. 1995, respectively). Statistically, these two concentrations are effectively the same³. Food consumption was measured in the Heaton et al (1995) study; the reported exposure concentrations of 0.134 and 0.004 mg/kgBW/day was selected as the LOAEL and NOAEL to be utilized in this risk assessment.

The exposure scenarios evaluated for mink were the same as those evaluated for piscivorous birds, except that incidental sediment ingestion was added to the fish and water ingestion scenario. All HQs calculated for mink exceeded 1.0, and ranged from 2.1 (LOAEL as the effect level and mean PCB concentrations from the Mass Balance data set) to 397.8 (NOAEL as the effect level and maximum fish PCB concentrations from the NRDA data set; Table 13). Adding sediment and surface water ingestion to the exposure scenario had almost no effect on calculated HQs, indicating food ingestion is the primary exposure route for this species.

Exposure of mink is limited to feeding along the shoreline of the bay and along tributaries; mink may obtain a significant portion of their diet from tributaries. Although PCB concentrations from bay fish were used to model mink exposure, limited data are available for PCB concentrations in fish collected from tributaries to Green Bay (WI DNR 1999, Appendix C). Whole-body PCB concentrations in walleye collected from the Peshtigo River ranged from 3.25 to 7.3 mg/kg, and from 0.36 to 13.0 mg/kg in walleye collected from the Menominee River. The range of whole-body PCB concentrations in walleye collected from the upper Green Bay (range 0.62 to 7.26 mg/kg) are comparable to those measured in tributary fish, and are a reasonable estimate of mink exposure levels.

The calculated HQs for this species indicates piscivorous mammals utilizing the upper Green Bay area are at risk from exposure to measured PCB concentrations in fish.

³ Based on the reported mean and standard deviation for total PCB concentration in diets used in the two studies.

7.0 UNCERTAINTY ANALYSIS

7.1 General Uncertainty Analysis

There are factors inherent in the risk assessment process that contribute uncertainty and must be considered when interpreting results. Major sources of uncertainty arise from natural variability in biological systems, the introduction of error in the risk assessment process, and the presence of data gaps.

Natural variability is an inherent characteristic of ecological receptors, their stressors, and their combined behavior in the environment. Biotic and abiotic parameters in these systems may vary to such a degree that the exposure of similar ecological receptors within the same system may differ temporally and spatially. Factors that contribute to temporal and spatial variability may be differences in an individual organism's behavior (within the same species), changes in the weather or ambient temperature, unanticipated interference from other stressors, differences between microenvironments, and numerous other factors.

Uncertainty associated with natural variability also arises from the use of literature toxicity values in which a study has examined a single species/single contaminant system under controlled conditions. If conducted in a laboratory, these studies do not take into account the effects of the environmental factors and other stressors that are present in natural systems. These factors may have synergistic, antagonistic, or neutral effects upon the receptor-contaminant interaction.

Point estimates of exposure such as NOAELs, LOAELs, LD₅₀s, and mathematical means that are presented in the literature also have inherent variability, which is incorporated into the risk assessment. Additionally, because these values are statistically determined, they do not represent absolute thresholds; they are reflective of the experimental design. A reported LOAEL may not represent the lowest toxicity threshold for a species simply because lower concentrations were not tested in a study.

In addition, uncertainty associated with variability is introduced from the use of literature values for soil, sediment, water, and food ingestion rates, dietary compositions, and body weights. These values reported in the literature are from studies that may have been conducted at a time of year or in a location that does not necessarily give an accurate representation of the life histories of the receptor species in the upper Green Bay.

Error may be introduced into the risk assessment through the use of invalid assumptions in the conceptual model. Conservative assumptions were made in light of the uncertainty associated with the risk assessment process (e.g., natural variability). Consistent conservative assumptions were used to minimize the possibility of concluding that risk is not present when a threat actually does exist (i.e., the elimination of false negatives). While there is uncertainty associated with each conservative assumption used, this consistent selection process assures that the uncertainty associated with this type of error will err on the side of a protective outcome.

This risk assessment did not examine the contribution of dermal absorption or inhalation exposure as part of the exposure pathway. In contrast to the use of conservative assumptions, the error introduced into this risk assessment by the omission of these routes of exposure may err on the side of a less protective outcome. The relative contribution of this error to alter the outcome of the risk assessment is unknown at this time.

Methodological problems in the literature reviewed for obtaining life history and toxicity information also introduce uncertainty into a risk assessment. Attempts were made to avoid using literature that was questionable. The process used to select appropriate studies on which to base

TRV derivation and life history parameter selection is described in Appendices A and B. However, if limited sources of information existed, potential error due to questionable study design was incorporated into the risk assessment if these data were used.

Data gaps were defined here as the incompleteness of data or information upon which the risk assessment was based. Specifically, these may be an incomplete contaminant data set, missing pieces of life history information, the absence of toxicity-based literature for the receptor of concern, or unknown or questionable QA/QC procedures.

Life history information and literature values for the toxicity of the contaminants of concern were not always available for all of the receptor species. By using closely related species, it was possible to make risk estimates. In reality, however, the information may vary substantially among species, thereby introducing another source of uncertainty.

In cases where a toxicity value has been converted by a factor of 10, the uncertainty associated with the absence of a directly relevant literature value was compounded by the uncertainty associated with a subjective mathematical adjustment.

7.2 Site-Specific Uncertainty Analysis

7.2.1 Selection of Contaminants of Potential Concern

The contaminant of concern evaluated in this risk assessment was selected based on the risk assessment conducted for the Lower Fox River/lower Green Bay (ThermoRetec Consulting Corporation). Of the eight COPCs retained for the above assessment, only PCBs were selected as a COPC for this risk assessment. It is well documented that PCBs are the most widespread contaminant in the Fox River/Green Bay system (ThermoRetec Consulting Corporation 1998). In addition, the above cited risk assessment concluded that risks to ecological receptors from PCB exposure were 10 to 1,000 times greater than predicted risk from the other seven COPCs (ThermoRetec Consulting Corporation). It should be recognized that other contaminants could potentially pose a risk to ecological receptors which utilize the Upper Green Bay. However, to focus this risk assessment, it was assumed that risks from exposure to other contaminants were sufficiently evaluated in the risk assessment conducted for the Lower Fox River/lower Green Bay.

Many of the toxic effects of PCBs are produced by coplanar PCB congeners that have a structure similar to TCDD. Dioxin-like toxic effects include edema, deformities, and early life stage mortality (Safe 1994). One method often used to evaluate toxicity of PCBs is the TCDD toxicity equivalence approach, where the toxic potency of each PCB congener is expressed relative to the potency of TCDD. A reason for utilizing this method is to incorporate the data available for toxicity of TCDD into the data reviewed for TRV derivation. Numerous studies evaluating PCB toxicity to the selected receptors were located in our literature search. Some uncertainty may result from not extending the literature search to include TCDD toxicity, however appropriate TRVs were located for all receptors based on results of the search which was conducted. An underlying assumption of the TEF approach is that toxicity of PCBs is solely related to their TCDD-like toxicity. Theoretically, any NOAEL for PCBs should incorporate dioxin-like toxicity, therefore this method was not utilized in this risk assessment.

7.2.2 Conceptual Model Limitations

Components of the conceptual model which potentially introduce uncertainty into this risk

assessment include transport and fate of PCBs, selected assessment endpoints and receptor species, and identification of complete exposure pathways.

Transport and fate of PCBs was modeled based on contaminant and ecosystem characteristics. Studies have shown that PCBs discharged into aquatic systems rapidly sorb to sediment (Kalmaz and Kalmaz 1979); movement of PCBs in aquatic systems depends mainly on movement of the associated sediment (Connell and Miller 1984). The lower Green Bay is the primary depositional zone for Fox River PCBs, however several studies conducted within Green Bay have documented sediment transport from the lower to upper Bay (Eadie et al. 1991, Manchester-Neesvig et al. 1996, Hawley and Niester 1993). In addition, fish and birds collected from the upper Green Bay have accumulated elevated concentrations of PCBs in their tissues (U.S. EPA 1996; Hagler Bailly Services, Inc. 1997; Stratus Consulting, Inc. 1999a). Although the above studies indicate transport of contaminated sediment from the lower to the upper Green Bay, and transfer of PCBs from sediment to ecological receptors, the magnitude of both transfer processes is uncertain.

An additional source of uncertainty is that other potential sources of PCBs are not considered in this risk assessment. Atmospheric deposition has been identified as an important source of PCBs to Lake Superior (Eisenreich et al. 1981) and southern Lake Michigan (Murphy et al. 1981). However, Sweet et al. (1991) estimated that atmospheric deposition of PCBs accounts for less than 10 percent of the total input to Green Bay. The potential contribution via influx from Lake Michigan is unknown. DePinto et al. (1994) identified the Fox River as the major source of PCBs to Green Bay; they estimated the Fox River contributed 92 percent of the PCB loading to the bay in 1989. To focus this risk assessment, the assumption was made that the primary source of PCBs to Green Bay was the Fox River.

The assessment endpoints selected for this risk assessment are a subset of those evaluated in the risk assessment conducted for the Lower Fox River/lower Green Bay. The only contaminant evaluated in this risk assessment was PCBs; adverse effects from exposure to PCBs are related to bioaccumulation rather than direct toxicity. Therefore, the assessment endpoints selected for this risk assessment focus on bioaccumulation of PCBs and upper trophic level receptors. By evaluating and protecting these assessment endpoints which are most sensitive to potential impacts from exposure to site-related contaminants, the upper bay ecosystem as a whole should also be protected.

Receptor species were selected for this risk assessment based on the complete exposure pathways identified in the conceptual model. The selected receptors act as surrogates for other species which are similar in terms of feeding habits and habitat use, and should be representative of potential risk to other species within the system. Mink and lake trout were selected as receptors based on their sensitivity to PCB effects. Numerous studies have documented the reproductive toxicity of dietary PCBs to mink at low exposure concentrations (Restum et al. 1998, Den Boer 1984, Heaton et al. 1995, Platanow and Karstad 1973). Among fish species studied to date, lake trout have been found to be most sensitive to PCB-caused fry mortality (Walker et al. 1991). In addition, lake trout females from Lake Michigan produce eggs which are deficient in thiamine; some studies have shown that an interaction exists between thiamine and dioxin-like embryo toxicity (Wright et al. 1998, Fisher et al. 1996, Wright and Tillit 1998). Selection of the most sensitive receptors (mink and lake trout) should adequately protect less sensitive species. Bird receptor species were selected based on potential sensitivity (e.g., observed deformities in field studies), complete exposure pathways, and to be consistent with

receptor species selected for the Lower Fox River/lower Green Bay risk assessment. Although there is uncertainty associated with limiting the number of species evaluated, the primary exposure pathway identified (dietary exposure) was sufficiently evaluated in this risk assessment by selecting high trophic level species as receptors.

Some exposure pathways not evaluated in this risk assessment include ingestion of plankton and exposure of dabbling ducks. No site-specific data on PCB concentrations in plankton, aquatic plants, or benthic organisms was available. The receptor species evaluated in this risk assessment feed at higher trophic levels than receptors within pathways not evaluated, therefore PCB exposure of selected receptors should be higher than for herbivorous or planktivorous species. Protection of the selected receptor species should be protective of organisms with lower exposure levels.

7.2.3 Estimates of Exposure Concentration

Uncertainty can be introduced into the risk assessment process by low quality, limited, or missing site-specific data. As discussed in Section 4.1, data utilized in this risk assessment was obtained from three sources. The data set developed for the NRDA was the most recent, and was developed under the most rigorous QA/QC procedures. Data collected in support of the Green Bay Mass Balance Model was older and has not been validated using strict QA/QC procedures. The QA/QC procedures used to validate bird data from published studies are unknown, however all studies cited have been peer-reviewed.

Fish tissue data from both the NRDA and Mass Balance data sets were used for this risk assessment. Although the confidence level in the quality of data from the NRDA model is high, no forage fish were collected. Piscivorous birds and mammals are not likely to consume fish the size of the upper trophic level fish collected, and PCB concentrations tend to increase with increasing fish size. Therefore, use of upper trophic level fish data to estimate dietary exposure of piscivorous birds and mammals may overestimate exposure concentrations. Therefore, forage fish data from the Mass Balance data set was also utilized for this risk assessment, although these data have not been validated at this time. Separate HQ calculations were done for each data source, so that the uncertainty associated with the different data sets could be evaluated.

Another source of uncertainty associated with use of both the NRDA and Mass Balance data sets is that composite samples were analyzed; each sample was comprised of three to six fish, and five fish each, respectively. Maximum PCB concentrations measured may underestimate maximum PCB concentrations for individual fish.

Fish species analyzed for both the NRDA and Mass Balance data set were walleye and brown trout. The selected receptor species, lake trout, tend to accumulate the highest concentrations of PCBs found in open-water fish of the Great Lakes (Mac and Schwartz 1992). Species-specific traits that contribute to this are:

- Lake trout possess a large amount of body fat (average of 12 percent);
- They have a long life span (8 to 10 years), and are exposed to PCBs for a longer period of time than many fish species;
- They grow slowly, leading to a higher PCB body burden (Jensen et al. 1982);
- Alewife, one of their main prey species, contain significant amounts of PCBs (St. Amant et al. 1984).

Because tissue data utilized in this risk assessment are from walleye and brown trout, actual tissue concentrations found in lake trout may be underestimated, therefore potential risk for this receptor species may be underestimated.

Fish collected within Green Bay were used to estimate dietary exposure of mink to PCBs. It should be noted that exposure of mink will be limited to feeding along the shoreline of the bay and along tributaries. Use of fish concentrations from the bay may overestimate fish concentrations in tributaries to the bay. However, comparison of bay fish data (Table 6) with limited data available on PCB concentrations in walleye collected from tributaries to Green Bay (Appendix C) indicate bay fish concentrations may be a reasonable estimate of tributary fish concentrations.

All sediment and water data were obtained from the Mass Balance data set. As stated above, the quality of these data is unknown, as it has not been validated according to strict QA/QC procedures. In addition, only one water sample collected in the Upper Green Bay was located. A separate exposure scenario was evaluated which incorporated sediment and water ingestion into the food chain model for each receptor, so that the uncertainty associated with use of these data could be evaluated. Sediment and water ingestion had no impact on calculated HQs for any receptor (Tables 11, 12, and 13). Therefore the uncertainty associated with use of one data point was not significant within the risk calculations performed for this risk assessment.

The bird egg and tissue data used in this risk assessment was obtained from studies conducted in the Upper Green Bay which were published in peer-reviewed literature. The QA/QC procedures used to evaluate these data and the associated uncertainty are unknown.

A final limitation of the data utilized in this risk assessment is that the most recent samples were collected in 1996. It is recognized that the Upper Green Bay is not a static system, therefore use of old data to characterize present conditions is another source of uncertainty. However, it is known that many of the primary sources of PCBs to this system have been eliminated; the principal current source of PCBs to ecological receptors is a secondary source, the sediment. Several long-term studies have been conducted within this system. The Canadian Wildlife Service has collected herring gull eggs from Big Sister Island almost every year since 1972 (Bishop et al. 1992, Pettit et al. 1994, Pekarik et al. 1998, Hughes et al. 1998). This data set is the most complete data set available to evaluate temporal trends in PCB exposure of birds that utilize the Green Bay system. The highest input of PCBs to the Green Bay system occurred in the early 1970s. After primary sources (e.g., discharges from paper companies related to use of PCB emulsion) were eliminated, PCB concentrations in herring gull eggs declined rapidly until 1982 (mean concentration approximately 142 and 62 mg/kg, wet weight, in 1971 and 1982, respectively). Since 1983 the decline has reached a plateau (mean concentration approximately 27 and 15 mg/kg, wet weight, in 1983 and 1996, respectively), although there is an almost significant negative trend ($r = 0.5$, $P = 0.07$; Stratus Consulting Inc. 1999d). A temporal PCB pattern similar to that seen in herring gulls has been observed in Lake Michigan fish (Stow et al. 1995, Lamon et al. 1998). Based on limited data available for Green Bay fish, the following trends were described: a decline in alewife PCB concentrations from the late 1970s to 1989; a decline consistent with an exponential decrease in yellow perch from 1976 to 1993 in Zone II, and from 1975 to 1984 in Zone III; and a slight linear decline in PCB concentrations in walleye in Zone III from 1976 to 1996 (Stratus Consulting, Inc. 1999a). Based on the above trends, the uncertainty associated with use of old data is that current risk to receptors may be overestimated.

7.2.4 Selection of TRVs

A comprehensive literature search was conducted to locate studies in which the toxicity of PCBs to wildlife receptors was evaluated. These studies were reviewed to evaluate the appropriateness of using a particular study to derive a TRV. Criteria used to evaluate studies are described in Appendix A, Section A.1; two important factors were study design and species tested. Very few toxicological studies have been conducted using wildlife species. Many TRVs were selected from studies in which the test organism was closely related taxonomically to a selected receptor species. It may be more appropriate to select effect levels derived from test organisms which are closely related trophic-wise (e.g., using an effect level for a carnivorous species such as a kestrel to derive a TRV for a piscivorous species). However, an attempt was made to use consistently conservative assumptions where possible in this risk assessment. Conservative assumptions were used to minimize the possibility of concluding that risk is not present when a threat actually does exist (i.e., the elimination of false negatives). If an acceptable study reported an effect level for a dietary exposure route to a taxonomically related species, the lowest reported LOAEL and NOAEL were selected as the TRV.

An exception to this is the selection of LOAELs and NOAELs for bird species. Effect levels reported for chickens were consistently much lower than effect levels reported for other bird species (Appendix A, Table A-1). Numerous studies have documented the greater sensitivity of chickens to TCDD-like toxicity as compared with wild bird species (Eisler and Belisle 1996, Hoffman et al. 1998, Bosveld and van den Berg 1994, Lorenzen et al. 1997). Dietary LOAELs reported for chickens ranged from 0.0414 to 0.9 mg/kgBW/day, whereas dietary LOAELs reported for other bird species ranged from 1.12 to 36 mg/kgBW/day. We felt a sufficient number of studies had been conducted with other avian species to conclude that effect levels reported for chickens were an anomaly relative to other bird species. Therefore, studies in which chickens were the test species were not selected for the derivation of the NOAEC and LOAEC in this risk assessment. However, if any bird species in the Green Bay area have PCB sensitivities similar to that of the chicken, this risk assessment will underestimate potential effects on that species.

In addition to effect levels reported in the literature as critical body concentrations for fish species, an alternative method was used to determine whole-body concentrations at which adverse effects would be expected. This method was derived based on the observation that whole-body concentrations are often measured, while fish egg concentration measurements are rare. Early life-stages are most sensitive to adverse effects of PCBs, therefore it is important to identify maternal whole-body concentrations which result in critical egg/fry PCB concentrations. The TRV for whole-body concentrations was derived using the TRV identified for fish egg concentrations and an egg to whole-body ratio reported by Mac et al. (1993), and resulted in the lowest LOAEC for fish body concentrations. This LOAEC was selected as the TRV for whole-body concentrations because it addresses the sensitivity of early life stages to PCBs. However, the method used to derive this LOAEC results in the loss of two independent lines of evidence to evaluate toxicity of PCBs to fish. Use of a weight-of-evidence approach to evaluate risk reduces uncertainty when all lines lead to similar conclusions about potential risk. However, it was determined that the risk assessment should focus on the most susceptible receptors (early life-stage fish); therefore the most conservative LOAEC was selected as the TRV.

8.0 FISH AND WILDLIFE SERVICE INJURY REPORTS

The assessment area defined for the Natural Resource Damage Assessment (NRDA) conducted by the U.S. Fish and Wildlife Service includes the Lower Fox River and all of Green Bay. Several NRDA reports have been released that assess injuries to natural resources of the Lower Fox River/Green Bay system that have resulted from releases of PCBs to the Lower Fox River. The injury reports for fishery resources (Stratus Consulting, Inc. 1999b) and avian resources (Stratus Consulting, Inc. 1999d) are summarized below so that the conclusions of the NRDA can be compared with the results of this risk assessment.

8.1 Fish and Wildlife Service Injuries to Fishery Resources Report

As part of the larger Great Lakes ecosystem, Green Bay provides important fish habitat and supports a diverse and productive fishery. Although the historic fish community composition has changed due to overfishing and the introduction of exotic species, the fishery resource continues to provide valuable ecological services. The injury report describes PCB transport and exposure pathways in the assessment area.

Pathways by which the fishery resources of Green Bay have been exposed to PCBs released from Lower Fox River paper companies were described based on transport processes (water circulation patterns and sediment transport and deposition patterns) and the spatial and temporal distribution of PCBs in sediment, water and biota in relation to the primary source. Elevated concentrations of PCBs have been documented in surface water, sediment, plankton, and fish within the assessment area.

Laboratory and field studies have shown that exposure of fish to PCBs results in adverse effects which meet the NRDA definition of injury. Effects include mortality, promotion or enhanced formation of tumors initiated by other factors, deformities, and impairment of immune and endocrine systems. Early life stages in fish are more sensitive to PCB-related mortality than adult fish (Eisler 1986).

Impacts to fish in the assessment area were evaluated based on measured concentrations of PCBs in fish tissue, and presence of adverse effects associated with PCB exposure. Two general types of changes to fish viability were assessed: adverse effects on fish health, and adverse effects on fish reproduction.

Fish health was evaluated using a suite of tests designed to measure parameters that can be adversely affected by PCB exposure. These included examination of tissues for bacterial, viral and parasitic infections, immunological evaluation of kidney and blood samples, evaluation of liver lesions, and measurement of ethoxyresorufin-O-deethylase (EROD) activity and tissue PCB concentration. Walleye were collected from five locations within the assessment area and two reference locations. Tissue PCB concentrations were significantly higher in assessment area walleye than in fish collected from the reference areas. Assessment area fish also had a significantly higher incidence of liver tumors and pre-tumors. It has been documented that PCBs promote or enhance liver tumor formation (Hendricks et al. 1990); therefore the injury report concluded walleye health has been adversely impacted by PCB exposure.

Adverse effects on reproduction were assessed for lake trout based on historical data, information from the scientific literature, and reproduction and laboratory toxicity studies conducted for the NRDA by the United State Geological Survey (USGS). The toxicity equivalence approach was used to compare historic PCB concentrations in lake trout eggs with toxicity thresholds for embryomortality. Mean egg total PCB concentrations over time were modeled and compared with LD₁₀ and LD₅₀ concentrations. The analysis concluded that in the mid-1970s egg PCB

concentrations were sufficient to cause sac fry mortality to some Green Bay lake trout eggs; by 1980, concentrations in less than one percent of Lake Michigan lake trout eggs are estimated to have been sufficient to cause mortality. Limited PCB data were available for Green Bay and western Lake Michigan lake trout; analysis of these data suggest PCB concentrations were higher in Green Bay lake trout.

Results of the toxicity studies conducted by the USGS for the NRDA indicate that thiamine deficiency rather than exposure to PCBs or other TCDD-like compounds is currently the primary causal factor for fry mortality in Lake Michigan lake trout. The Trustees concluded that current data do not support concluding that lake trout in Green Bay and Lake Michigan are injured by the PCBs released from Fox River paper companies.

The report concluded that the most significant injury to fishery resources in the Lower Fox River and Green Bay is the presence of extensive fish consumption advisories. Walleye within the assessment area are experiencing increased liver tumors compared with fish from reference areas. Available information does not support concluding that other PCB-related injuries assessed (brown trout and lake trout health, lake trout reproduction) are currently occurring, although they may have in the past.

8.2 Fish and Wildlife Service Avian Injury Report

The Lower Fox River/Green Bay area is an important site within the Great Lakes Ecoregion for breeding and migratory birds (Robbins 1991, Jacobs 1991). The assessment area, due to its comparatively undisturbed nature and the quality and extent of habitats it provides, supports bird populations and communities more diverse than those found in many other areas of the Great Lakes. Because the majority of the PCBs released into the assessment area are concentrated in the aquatic systems of the Fox River and Green Bay (Connolly et al. 1992), the NRDA focused on bird species which utilize aquatic habitats. Critical habitats identified in the NRDA were wetlands and small uninhabited islands in Green Bay that provide nesting sites for colonial waterbirds.

Exposure to a hazardous substance can be characterized by direct measurement of that substance in biota tissue [43 CFR § 11.63(f)(4)(I)]. Numerous studies have been conducted which evaluate PCB concentrations in assessment area birds. For all species and studies where a statistical comparison was made between PCB concentrations in assessment and reference area tissues, PCB concentrations were significantly higher in tissues from the assessment area. Based on evaluation of foraging areas and analysis of PCB concentrations in prey species, the NRDA report concluded that the primary route of exposure for most assessment area bird species is dietary.

Laboratory and field studies have shown that exposure of birds to PCBs results in numerous adverse effects that meet the NRDA definition of injury. These effects include death, behavioral abnormalities, physiological malfunctions and physical deformities. Avian embryos are the life stage most sensitive to PCB toxicity, followed by nestlings, then adults (Hoffman et al. 1998).

Two lines of evidence were used to evaluate injury to avian species utilizing the Fox River/Green Bay assessment area: comparison of egg PCB concentrations to concentrations of PCBs in bird eggs cited in the literature associated with adverse effects on bird reproduction and survival; and field studies conducted in Green Bay which evaluated PCB effects on bird populations.

Based on a literature search, PCB concentrations in eggs ranging from 3 to 20 mg/kg wet weight were identified as a toxic effect concentration range. Mean total PCB concentrations measured in eggs of five assessment area species (double-crested cormorants, Caspian terns, common terns, red-breasted mergansers, and Forster's terns) from 1983 to 1996 were within or exceeded the range where adverse reproductive effects have been shown to occur.

Field studies conducted with eight species (Forster's, common, and Caspian tern; double-crested cormorant; bald eagle; black-crowned night heron; tree swallow; and red-breasted merganser) were evaluated to determine whether sufficient evidence existed to conclude that birds in the assessment area have been injured by exposure to PCBs. Observed effects (decreased hatching success, deformities, edema) and their relationship to measured egg PCB concentrations provide strong evidence that Green Bay Forster's terns have been adversely affected by PCB exposure. Contaminants other than PCBs measured in eggs did not appear to be significant contributors to the observed toxicity. In a single field study conducted with common tern, observed effects were consistent with those observed in Forster's tern and with those caused by PCBs. Available studies do not provide strong evidence that reproductive success of Caspian terns has been adversely affected by PCB exposure, however there is some evidence of increased deformity rates. Two studies concluded that hatch success rates in Green Bay cormorant nests were significantly lower than in control areas; one found no difference between Green Bay and reference site nests. All studies that have compared bill deformity rates in embryos and nestlings between Green Bay and reference sites have found higher rates in Green Bay cormorants. Two studies conducted on bald eagles have found that productivity of Green Bay eagles is significantly lower than at inland sites where eagles are not exposed to point source releases of PCBs. Although studies conducted with black-crowned night heron, tree swallow, and red-breasted merganser conclude that these species have been exposed to PCBs at levels that exceed background concentrations, no significant adverse effects were observed. The conclusion from this evaluation was that sufficient evidence exists to conclude that Forster's, common and Caspian terns, double-crested cormorants, and bald eagles have been injured by PCBs, and that the occurrence of PCB-induced injuries has been widespread throughout the assessment area.

9.0 CONCLUSIONS

A LOAEL is an exposure concentration at which an adverse effect has been observed in a toxicological study; therefore a HQ greater than 1.0 based on a chronic LOAEL indicates that site levels of that contaminant may produce an adverse effect on the ecological receptor in question. The most substantive risk indicated by this risk assessment are lines of evidence where HQ calculations exceeded 1.0 when the LOAEL was used as the effect level and mean PCB concentrations were used as the exposure concentration. This occurred for the bird egg concentrations and the mink food chain model (Table 14).

Although the most substantive risk is indicated if the HQ exceeds one using mean measured PCB concentrations, the maximum concentration is an actual site-specific measured potential exposure concentration. A HQ which exceeds 1.0 using the maximum measured PCB concentration is still an indication of potential risk. The food chain model utilizing double-crested cormorant exposure parameters resulted in a HQ greater than 1.0 when the LOAEL and maximum fish concentrations from the NRDA data set were used in risk calculations.

A calculated HQ greater than 1.0 based on a chronic NOAEL indicates there is a potential chronic risk from that contaminant to the ecological receptor in question. Because concentrations of a contaminant on-site exceed the observed no-effect level for that contaminant, it can not be concluded that there is not risk associated with measured on-site concentrations. This occurred for the fish egg and tissue concentrations, and the food chain models for piscivorous birds.

Lines of evidence evaluated for this risk assessment for each individual assessment endpoint and conclusions based on the risk characterization for each are discussed below.

9.1 Pelagic Fish Reproduction and Survival

Two lines of evidence were used to estimate risk to pelagic fish reproduction and survival in the upper Green Bay: comparison of fish tissue and egg concentrations to adverse effect levels cited in

published studies. Hazard quotients calculated using the NOAEC exceeded 1.0 for both egg and upper trophic level fish tissue PCB concentrations (ranging from 3.9 to 9.5 and 4.0 to 9.4, respectively), indicating potential risk to pelagic fish reproduction and survival.

Although the conclusion of this risk assessment is potential risk to the selected receptor species, (lake trout) and the NRDA fish injury assessment concluded no actual adverse effects to lake trout reproduction are currently occurring, the two reports are not inconsistent. Lake trout were utilized in this risk assessment as a representative pelagic fish species; the risk characterization indicated potential risk to pelagic fish based on contaminant concentrations in eggs and fish tissue which exceed concentrations at which no adverse impacts have been documented. Although the reported LOAEC was not exceeded, a LOAEC derived from the literature is not necessarily the lowest concentration at which an adverse effect will occur, it is simply the lowest concentration that has been tested. Because concentrations of a contaminant on-site exceed the observed no-effect level for that contaminant, it can not be concluded that there is not risk associated with measured on-site concentrations. The NRDA fish injury assessment did find actual adverse effects which are consistent with effects observed after PCB exposure in another pelagic fish, walleye (increased incidence of liver tumors).

9.2 Piscivorous Bird Reproduction and Survival

Three lines of evidence were used to evaluate risk to piscivorous birds utilizing the upper Green Bay area: comparison of bird egg concentrations to adverse effect levels published in the literature; modeled food chain exposure and comparison of estimated dietary exposure concentrations to published adverse effect levels; and published studies on birds utilizing the upper Green Bay.

Comparison of bird egg concentrations to adverse effect levels cited in the literature indicates that piscivorous birds utilizing the upper Green Bay area are at risk from exposure to PCBs. Measured concentrations of PCBs in Caspian tern and double-crested cormorant eggs exceed levels shown to cause adverse reproductive effects (hazard quotients ranging from 1.3 to 25.1).

Food chain exposure models indicate that piscivorous birds are potentially at risk from dietary PCB exposure levels; all except one HQ calculated using the NOAEL as the effect level exceeded 1.0 (range between 0.8 and 16.2). The HQ calculated using a double-crested cormorant exposure model exceeded 1.0 (HQ = 1.6) when the LOAEL was used as the effect level and maximum fish concentrations from the NRDA data set were used.

Published studies in which the effects of PCBs on birds inhabiting the upper Green Bay were reviewed as the third line of evidence for this assessment endpoint. This line of evidence also indicates that piscivorous birds may be at risk from PCB exposure. Adverse effects associated with PCB exposure (decreased hatching success, embryo deformities) were not observed in studies conducted with Caspian terns. One study found a strong negative correlation between nest site tenacity and PCB concentrations, however population-level implications of subtle behavioral changes are not known. Studies conducted on double-crested cormorants in the upper Green Bay indicate this species has experienced adverse reproductive effects. Hatch success rates were lower and physical deformity rates were higher in Green Bay cormorants than at reference sites. PCBs have been shown in laboratory experiments to cause deformities in avian embryos similar to those seen in Green Bay cormorants (crossed bills, edema, dwarfism).

The weight of evidence used to evaluate risk to piscivorous birds indicates these species are potentially at risk from PCB exposure. Results from food chain exposure models indicate greater risk using the double-crested cormorant model, which correlates with results observed in field studies.

Double-crested cormorants were utilized in this risk assessment as a model for piscivorous birds. Although cormorant populations in the Great Lakes are doing well, risk calculations indicate that other species within this feeding guild may be at risk for experiencing adverse effects. Factors contributing to the cormorant population increase observed in the Great Lakes since 1973 include a rise in the numbers of prey fish, decreased levels of toxic chemicals, a decrease in commercial fishing, and legislation which protects cormorants (Environment Canada 1995). An additional point which should be noted is that the decline in PCB concentrations measured in bird eggs in the late 1970s reached a plateau in the mid-1980s; relatively little decline has occurred since. Although the primary source of PCBs to the upper bay has been eliminated, exposure concentrations for birds appear to have remained similar for the last decade.

9.3 Piscivorous Mammal Reproduction and Survival

The food chain model used to evaluate risk to piscivorous mammals indicates mink are at risk from PCB exposure in the upper Green Bay area. All HQs calculated for this species exceeded 1.0, and ranged from 2.1 (LOAEL as the effect concentration, mean overall fish PCB concentration) to 397.8 (NOAEL as the effect concentration, maximum fish PCB concentration from the NRDA data set).

10.0 LITERATURE CITED

- Abrahamson, L.J. and J.R. Allen. 1973. "The Biological Response of Infant Nonhuman Primates to a Polychlorinated Biphenyl." *Environ. Health Persp.* June:81-86.
- Ankley, G.T., K. Lodge, D.J. Call, M.D. Balcer, L.T. Brooke, P.M. Cook, R.G. Kreis,, Jr., A.R. Carlson, R.D. Johnson, G.J. Niemi, R.A. Hoke, C.W. West, J.P. Giesy, P.D. Jones, and Z.C. Fuying. 1992. "Integrated Assessment of Contaminated Sediments in the Lower Fox River and Green Bay, Wisconsin." *Ecotoxicology and Environmental Safety*, 23:46-63.
- Barnhouse, L.W., G.W. Suter, S.M. Bartell, J.J. Beauchamp, R.H. Gardner, E. Linder, R.V. O'Neill and A.E. Rosen. 1986. *User's Manual for Ecological Risk Assessment*. Publication Number 2679, ORNL-6251. Environmental Services Division, Oak Ridge National Laboratory, Oak Ridge, TN.
- Barsotti, D.A., R.J. Marlor, and J.R. Allen. 1976. "Reproductive Dysfunction in Rhesus Monkeys Exposed to Low Levels of Polychlorinated Biphenyls (Aroclor® 1248)." *Fd. Cosmet. Toxicol.*, 14:99-103. In: Heaton, S.N., S.J. Bursian, J.P. Giesy, D.E. Tillitt, J.A. Render, P.D. Jones, D.A. Verbrugge, T.J. Kubiak, and R.J. Aulerich. 1995. "Dietary Exposure of Mink to Carp from Saginaw Bay, Michigan. 1. Effects on Reproduction and Survival, and the Potential Risks to Wild Mink Populations." *Arch. Environ. Contam. Toxicol.*, 28:334-343.
- Batty, J., R.A. Leavitt, N. Biondo, and D. Polin. 1990. "An Ecotoxicological Study of a Population of the White Footed Mouse (*Peromyscus leucopus*) Inhabiting a Polychlorinated Biphenyls-Contaminated Area." *Arch. Environ. Contam. Toxicol.* 19:283-290.
- Bird, D.M., P.H. Tucker, G.A. Fox, and P.C. Lague. 1983. "Synergistic Effects of Aroclor 1254 and Mirex on the Semen Characteristics of American Kestrels." *Arch. Environ. Contam. Toxicol.* 12:633-640.
- Bishop, C.A., D.V. Weseloh, N.M. Burgess, J.Struger, R.J. Norstrom, R. Turle and K.A. Logan. 1992. *Atals of Contaminants in Eggs of Fish-Eating Colonial Birds of the Great Lakes (1970-1988)*. Vols. I and II. Environment Canada. Canadian Wildlife Service. Technical Report Series No. 152.
- Bosveld, A.T.C. and M. Van den Berg. 1994. "Effects of polychlorinated biphenyls, dibenzo-p-dioxins, and dibenzofurans on fish-eating birds." *Environmental Reviews*, 2:147-166.
- Brezner, S., J. Terkel, and A.S. Perry. 1984. "The Effect of Aroclor® 1254 (PCB) on the Physiology of Reproduction in the Female Rat - I." *Comp. Biochem. Physiol.*, 77C(1):65-70. In: Heaton, S.N., S.J. Bursian, J.P. Giesy, D.E. Tillitt, J.A. Render, P.D. Jones, D.A. Verbrugge, T.J. Kubiak, and R.J. Aulerich. 1995. "Dietary Exposure of Mink to Carp from Saginaw Bay, Michigan. 1. Effects on Reproduction and Survival, and the Potential Risks to Wild Mink Populations." *Arch. Environ. Contam. Toxicol.* 28:334-343.
- Bruggerman, W.A., L.B.J.M. Martron, D. Kooiman, and O. Hutzinger. 1981. "Accumulation and Elimination Kinetics of Di-, Tri-, and Tetrachlorobiphenyls by Goldfish After Dietary and Aqueous Exposure." *Chemosphere* 10:811-815. In: Rasmussen, J.B., D.J. Rowan, D.R.S. Lean, and J.H. Carey. 1990. "Food Chain Structure in Ontario Lakes Determines PCB Levels in Lake Trout (*Salvelinus namaycush*) and Other Pelagic Fish." *Can. J. Aquat. Sci.* 47:2030-2038.
- Brunstrom, B. 1988. Sensitivity of Embryos from Duck, Goose, Herring Gull and Various Chicken Breeds to 3,3',4,4'-Tetrachlorobiphenyl. *Poult. Sci.* 67:52-57.
- Brunstrom, B. and L. Reutergardh. 1986. Differences in Sensitivity of some Avian Species to the Embryotoxicity of a PCB, 3,3',4,4'-tetrachlorobiphenyl, Injected into the Eggs. *Environ. Pollut.* 42:37-45.

Brunstrom, B. and J. Lund. 1988. Differences between chick and turkey embryos in sensitivity to 3,3',4,4'-Tetrachlorobiphenyl and in Concentration/Affinity of the Hepatic Receptor for 2,3,7,8-Tetrachlorodibenzo-p-dioxin. *Comp. Biochem. Physiol.* 91C(2):507-512.

Chapman, J.A. and G.A. Felhamer. 1982. *Wild Mammals of North America: Biology, Management, Economics*. Baltimore, MD: Johns Hopkins University Press. p. 1147.

Chou, S.F.J. and R.A. Griffin. 1986. "Solubility and Soil Mobility of Polychlorinated Biphenyls." Pages 101-120 in "PCBs and the Environment, Volume I." (J.S. Waid, ed.). CRC Press, Boca Raton, Florida. 228p.

Connell, D.W. and G.J. Miller. 1984. *Chemistry and Ecotoxicology of Pollution*. New York, NY: John Wiley & Sons, Inc. 444 p.

Connolly, J.F., T.F. Parkerton, J.D. Quadrine, 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: 200. Report Prepared for the U.S. EPA Large Lakes Research Station, Grosse Ile, MI.

Custer, T.W., C.M. Custer, R.K. Hines, S. Gutreuter, K.L. Stromborg, P.D. Allen and M.J. Melancon. In press. Organochlorine Contaminant Effects on Double-Crested Cormorants Nesting in Green Bay, Wisconsin. *Environ. Toxicol. Chem.*

Den Boer, M.H. 1984. "Reproduction Decline of Harbour Seals: PCBs in the Food and their Effect on Mink." *Netherlands Research Institute for Nature Management Annual Report*, p. 77-86.

DePinto, J.V., R. Raghunathan, P. Sierzenga, X. Zhang, V.J. Bierman, P.W. Rodgers and T.C. Young. 1994. Recalibration of GBTOX: An Integrated Exposure Model for Toxic Chemicals in Green Bay, Lake Michigan. Draft Final Report. Prepared for the U.S. EPA, Large Lakes and Rivers Research Branch, Grosse Ile, MI.

Dourson, M.L. and J.F. Stara. 1983. Regulatory History and Experimental Support of Uncertainty (Safety) Factors. *Regulatory Toxicology and Pharmacology*. 3:224-238.

Eadie, B.J., G.L. Bell and N. Hawley. 1991. Sediment Trap Study in the Green Bay Mass Balance Program: Mass and Organic Carbon Fluxes, Resuspension, and Particle Settling Velocities. NOAA Technical Memorandum ERL GLERL-75. Great Lakes Environmental Research Laboratory, Ann Arbor, MI. July 1991. 29 pp.

Environment Canada. 1995. The Rise of the Double-crested Cormorant on the Great Lakes. Available at <http://www.cciw.ca/glimr/data/cormorant-fact-sheet/intro.html>

Eisenreich, S.J., B.B. Looney, M. Holdrinet, D.P. Dodge and S.J. Nepszy. 1981. Airborne organic contaminants in the Great Lakes ecosystem. *Environ. Sci. Technol.* 15:30-38. *As cited in Simmons 1984.*

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). 72 pp.

Eisler, R. and A.A. Belisle. 1996. Planar PCB Hazards to Fish, Wildlife and Invertebrates: A Synoptic Review. *National Biological Service Biological Report 31*. 75 pp.

Ernst, W., H. Goerke, G. Eder, and R.G. Schaefer. 1976. "Residues of chlorinated hydrocarbons in marine organisms in relation to size and ecological parameters. I. PCB, DDT, DDE and DDD in fishes and

molluscs from the English Channel." *Bull. Environ. Contam. Toxicol.* 15:55.

Ewins, P.J., C.V. Weseloh, R.J. Norstrom, K. Legierse, H.J. Auman and J.P. Ludwig. 1994. Caspian Terns on the Great Lakes: Organochlorine Contamination, Reproduction, diet and Population Changes, 1972-91. *Canadian Wildlife Service Occasional Paper Number 85*.

Fein, C.G., J.L. Jacobsen, S.W. Jacobsen, P.M. Schwartz, and J.K. Dowler. 1984. "Prenatal Exposure to Polychlorinated Biphenyls: Effects on Birth Size and Gestational Age." *J. Ped.*, 105(2):315-320. In: Heaton, S.N., S.J. Bursian, J.P. Giesy, D.E. Tillitt, J.A. Render, P.D. Jones, D.A. Verbrugge, T.J. Kubiak, and R.J. Aulerich. 1995. "Dietary Exposure of Mink to Carp from Saginaw Bay, Michigan. 1. Effects on Reproduction and Survival, and the Potential Risks to Wild Mink Populations." *Arch. Environ. Contam. Toxicol.*, 28:334-343.

Fisher, J.P., J.D. Fitzsimons, G.F. Combs and J.M. Spotsbergen. 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(2):167-178.

Fox, G.A., B. Collins, E. Hayakawa, D.V. Weseloh, J.P. Ludwig, T.J. Kubiak and T.C. Erdman. 1991. Reproductive Outcomes in Colonial Fish-Eating Birds: A Biomarker for Developmental Toxicants in the Great Lakes Food Chains. II. Spatial Variation in the Occurrence and Prevalence of Bill Defects in Young Double-Crested Cormorants in the Great Lakes, 1979-1987. *J. Great Lakes Res.* 17:158-167.

Fox River Database. Available at <<http://www.ecochem.net/FoxRiverDatabaseWeb/default.asp>>

Gaude, P. 1998. "Completely-mixed Embayment Model." Available at <<http://www.eng.buffalo.edu/~pgaude/embayment.htm>>.

Giesy, J.P., D.A. Verbrugge, R.A. Othout, W.W. Bowerman, M.A. Mora, P.D. Jones, J.L. Newsted, C. Vandervoort, S.N. Heaton, R.J. Aulerich, S.J. Bursian, J.P. Ludwig, G.A. Dawson, T.J. Kubiak, D.A. Best, and D.E. Tillitt. 1994. "Contaminants in Fishes from Great Lakes-Influenced Sections and Above Dams of Three Michigan Rivers. II: Implications for Health of Mink." *Arch. Environ. Contam. Toxicol.* 27:213-223.

Hagler Bailly Services, Inc. 1997. 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.

Hawley, N. and J. Niester. 1993. Measurement of horizontal sediment transport in Green Bay, May-October, 1989. *Journal of Great Lakes Research.* 19(2): 368-378.

Heaton, S.N., S.J. Bursian, J.P. Giesy, D.E. Tillitt, J.A. Render, P.D. Jones, D.A. Verbrugge, T.J. Kubiak, and R.J. Aulerich. 1995. "Dietary Exposure of Mink to Carp from Saginaw Bay, Michigan. 1. Effects on Reproduction and Survival, and the Potential Risks to Wild Mink Populations." *Arch. Environ. Contam. Toxicol.* 28:334-343.

Heinz, G.H., D.M. Swineford, and D.E. Katsma. 1984. "High PCB Residues in Birds From the Sheboygan River, WI." *Environ. Monitor. Assess.* 4:155-161.

Hendricks, J.D., W.T. Scott, T.P. Putnam, and R.O. Sinnhuber. 1981. Enhancement of aflatoxin B1 hepatocarcinogenesis in rainbow trout (*Salmo gairdneri*) embryos by prior exposure of gravid females to dietary Aroclor 1254. p. 203-214. In: D.R. Branson and K.L. Dickson (Eds.). Aquatic toxicology and hazard assessment: fourth conference. ASTM STP 737. American Society for Testing and Materials.

- 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. Abstract, Proceedings of the American Association for Cancer Research Volume 31, March 1990. 1 pp.
- Hoffman, D.J., B.A. Rattner, C.M. Bunck, H.M. Ohlendorf, and R.W. Lowe. 1986. "Association Between PCBs and Lower Embryonic Weight in Black-Crowned Night Herons in San Francisco Bay." *J. Toxicol. Environ. Health.* 19:383-391.
- Hoffman, D.J., G.J. Smith and B.A. Rattner. 1993. Biomarkers of Contaminant Exposure in Common Terns and Black-Crowned Night Herons in the Great Lakes. *Environ. Toxicol. Chem.* 17:1095-1103.
- Hoffman, D.J., M.J. Melancon, J.D. Eisemann, P.N. Klein and J.W. Spann. 1998. Comparative developmental Toxicity of Planar PCB Congeners in Chickens, American Kestrels and Common Terns. *Environ. Toxicol. Chem.* 17:747-757.
- Holey, M.E., R.W. Rybicki, G.W. Eck, E.H. Brown, Jr., J.E. Marsden, D.S. Lavis, M.L. Toney, T.N. Trudeau and R.M. Horrall. 1995. Progress Toward Lake Trout Restoration in Lake Michigan. *J. Great Lakes Res.* 21 (Supplement 1):128-151.
- Horn, E.G., L.J. Hetling, and T.J. Tofflemire. 1979. "The Problem of PCBs in the Hudson River System." *New York Acad. Sci.* 320:591-609.
- Hornshaw, T.C., R.J. Aulerich, H.E. Johnson. 1983. "Feeding Great Lakes Fish to Mink: Effects on Mink and Accumulation and Elimination of PCBs by Mink." *J. Toxicol. Environ. Health.* 11:933-946.
- Hughes, K.D., D.V. Weseloh and B.M. Braune. 1998. The Ratio of DDE to PCB Concentrations in Great Lakes Herring Gull Eggs and Its Use in Interpreting Contaminants Data. *J. Great Lakes Res.* 24(1):12-31.
- Ingersoll, C.G., P.S. Haverland, E.L. Brunson, T.J. Canfield, F.J. Dwyer, C.E. Henke, N.E. Kemble, D.R. Mount, and R.G. Fox. 1996. "Calculation and evaluation of sediment effect concentrations for the amphipod *Hyaella azteca* and the Midge *Chironomus tentans*." *J. Great Lakes Res.* 22:602-623.
- Jensen, A.L., S.A. Spigarelli and M.M. Thommes. 1982. PCB uptake by five species of fish in Lake Michigan, Green Bay of Lake Michigan, and Cayuga Lake, New York. *Can. J. Fish. And Aquat. Sci.* 39:700-709.
- Johnson, H.T., J.E. Keil, R.G. Gaddy, C.B. Loadholt, G.R. Henigar, and E.M. Walker. 1976. "Prolonged Ingestion of Commercial DDT and PCB: Effects on Progesterone Levels and Reproduction in the Mature Female Rat." *Arch. Environ. Contam. Toxicol.* 3:479-490. In: Linzey, A.V. 1987. "Effects of Chronic Polychlorinated Biphenyls Exposure on Reproductive Success of White-footed Mice (*Peromyscus leucopus*)." *Arch. Environ. Contam. Toxicol.* 16:455-460.
- Kalmaz, E.V. and Kalmaz, G.D. 1979. "Transport, Distribution, and Toxic Effects of Polychlorinated Biphenyls in Ecosystems: Review." *Ecological Modelling.* 6:223.
- Keith, J.A. 1966. "Reproduction in a Population of Herring Gulls (*Larus argentatus*) Contaminated by DDT." *J. Appl. Ecol.* 3 (Suppl.):57-70.
- Kluwe, W.M., C.L. Herrmann, and J.B. Hook. 1979. Effects of Dietary Polychlorinated Biphenyls and Polybrominated Biphenyls on the Renal and Hepatic Toxicities of Several Chlorinated Hydrocarbon Solvents in Mice." *J. Toxicol. Environ. Health.* 5:605-615.
- Koslowski, S.E., C.D. Metcalfe, R. Lazar, and G.D. Haffner. 1994. "The Distribution of 42 PCBs,

Including Three Coplanar Congeners, in the Food Web of the Western Basin of Lake Erie." *J. Great Lakes Res.* 20:260-270.

Kurita, H. and J.P. Ludwig. 1988. "Embryonic teratologies and abnormalities assessed in naturally-incubated eggs of double-crested cormorants (*Phalacrocorax auritus*) and Caspian terns (*Hydroprogne caspia*) from Michigan Great Lakes colonies in 1988." Reports to the Michigan Audubon Society on the 1986-1988 Findings of the Michigan Colonial Waterbird Monitoring Project. Dec. 15, 1988, Ecological Research Services. Cited in: Yamashita, N., S. Tanabe, J.P. Ludwig, H. Kurita, M.E. Ludwig, and R. Tatsukawa. 1993. "Embryonic abnormalities and organochlorine contamination in double-crested cormorants (*Phalacrocorax auritus*) and Caspian terns (*Hydroprogne caspia*) from the upper Great Lakes in 1988." *Environmental Pollution*. 79:163-173.

Lamon, E.C. III, S.R. Carpenter and C.A. Stow. 1998. Forecasting PCB concentrations in Lake Michigan salmonids: A dynamic linear model approach. *Ecol. Appl.* 8(3):659-668.

Langford, H.D. 1979. "Looking at Polychlorinated Biphenyls as Environmental Object Lesson." *News Rep.* 29(9):1-5. In: Heaton, S.N., S.J. Bursian, J.P. Giesy, D.E. Tillitt, J.A. Render, P.D. Jones, D.A. Verbrugge, T.J. Kubiak, and R.J. Aulerich. 1995. "Dietary Exposure of Mink to Carp from Saginaw Bay, Michigan. 1. Effects on Reproduction and Survival, and the Potential Risks to Wild Mink Populations." *Arch. Environ. Contam. Toxicol.* 28:334-343.

Larson, J.M., W.H. Karasov, L. Sileo, K.L. Stromborg, B.A. Hanbidge, J.P. Giesy, D.E. Tillitt and D. Verbrugge. 1996. Reproductive Success, Developmental Anomalies, and Environmental Contaminants in Double-Crested Cormorants (*Phalacrocorax auritus*). *Environ. Toxicol. Chem.* 15:553-559.

Larsson, P., L. Orla, and L. Collvin. 1993. "Reproductive status and lipid content as factors in PCB, DDT and HCH contamination of a population of Pike (*Esox lucius* L.)." *Environ. Toxicol. Chem.*, 12:855-861.

Lincer, J.L. and D.B. Peakall. 1970. "Metabolic Effects of Polychlorinated Biphenyls in American Kestrel." *Nature* 228:783. In: Vos, J.G. 1972. "Toxicology of PCBs for Mammals and for Birds." *Environ. Health Persp.* April:105-117.

Linzey, A.V. 1987. "Effects of Chronic Polychlorinated Biphenyls Exposure on Reproductive Success of White-footed Mice (*Peromyscus leucopus*)." *Arch. Environ. Contam. Toxicol.* 16:455-460.

Loizeau, V. and A. Menesguen. 1993. "A Steady-state Model of PCB Accumulation in Dab Food Web." *Oceanologica Acta.* 16:633-640.

Lorenzen, A., J.L. Shutt and S.W. Kennedy. 1997. Sensitivity of Common Tern (*Sterna hirundo*) Embryo Hepatocyte Cultures to CYP1A Induction and Porphyrin Accumulation by Halogenated Aromatic Hydrocarbons and Common Tern Egg Extracts. *Arch. Environ. Contam. Toxicol.* 32:126-134.

Lowe, P.T. and R.C. Stendell. 1991. "Eggshell Modifications in Captive American Kestrels Resulting from Aroclor 1248 in the Diet." *Arch. Environ. Contam. Toxicol.* 20:519-522.

Ludwig, J.P. 1965. "Biology and structure of the Caspian tern (*Hydroprogne caspia*) population of the Great Lakes from 1896-1964." *Bird-Banding.* 13:1-9.

Ludwig, J.P., H. Kurita-Matsuba, H.J. Auman, M.E. Ludwig, C.L. Summer, J.P. Giesy, D.E. Tillitt, and P.D. Jones. 1996. "Deformities, PCBs, and TCDD-equivalents in double-crested cormorants (*Phalacrocorax auritus*) and Caspian terns (*Hydroprogne caspia*) of the upper Great Lakes 1986-1991: Testing a cause-effect hypothesis." *Great Lakes Res.* 22:172-197.

- Ludwig, J.P. and M.P. Ludwig. Undated report. "Productivity and deformities in chicks of the Michigan Great Lakes' colonies of double-crested cormorants (*Phalacrocorax auritus*) and Caspian terns (*Hydroprogne caspia*) in 1986." Ecological Research Services, Inc., Bay City, MI.
- Mac, M.J. and C.C. Edsall. 1991. "Environmental Contaminants and the Reproductive Success of Lake Trout in the Great Lakes: An Epidemiological Approach." *Journal of Toxicology and Environmental Health*. 33:375-394.
- 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." *J. Great Lakes Res.* 19:752-765.
- 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*. 25:189-192.
- Madenjian, C.P., S.R. Carpenter, and P.S. Rand. 1994. "Why Are the PCB Concentrations of Salmonine Individuals from the Same Lake So Highly Variable." *Can. J. Fish. Aquat. Sci.* 51:800-807.
- Madenjian, C.P., R.J. Hesselberg, T.J. DeSorcie, L.J. Schmidt, R.M. Stedman, R.T. Quintal, L.J. Begnoche, D.R. Passino-Reader. 1998. Estimate of net trophic transfer efficiency of PCBs to Lake Michigan lake trout from their prey. *Env. Sci. Tech.* 32(7) 886-891.
- Manchester-Neesvig, J.B., Andren A.W. and D.N. Edgington. 1996. Patterns of mass sedimentation and of deposition of sediment contaminated by PCBs in Green Bay. *J. Great Lakes Res.* 22(2): 444-462.
- McLane, A.R. and Hughes, D.L. 1980. "Reproductive success of screech owls fed Aroclor 1248." *Arch. Environ. Contam. Toxicol.*, 9:661-665.
- Merson, M.H. and R.L. Kirkpatrick. 1976. "Reproductive Performance of Captive White-footed Mice Fed a PCB." *Bull. Environ. Contam. Toxicol.* 16:392-398.
- Miller, M.A. 1993. Maternal Transfer of Organochlorine Compounds in Salmonines to their Eggs. *Can. J. Fish. Aquat. Sci.* 50:1405-1413.
- Miller M.A., N.M. Kassulke, and M.D. Walkowski. 1993. "Organochlorine concentrations in Laurentian Great Lakes salmonines: Implications for fisheries management." *Arch. Environ. Contam. Toxicol.* 25:212-219.
- Mineau, P., G.A. Fox, R.J. Norstrom, D.V. Weseloh, D.J. Hallett, and L. A. Ellenton. 1984. "Using the Herring Gull to Monitor Levels and Effects of Organochlorine Contamination in the Canadian Great Lakes." In: *Toxic Contaminants in the Great Lakes, Advances in Environmental Sciences and Technology*. Eds. J.O. Nriagu and M.S. Simmons. New York: Wiley, p. 425-452.
- Mitchell, J.R., W.R. Snodgrass, and J.R. Gillette. 1976. "The Role of Biotransformation in Chemical-Induced Liver Injury." *Environ Health Persp.* 15:27-38. In: Kluwe, W.M., C.L. Herrmann, and J.B. Hook. 1979. Effects of Dietary Polychlorinated Biphenyls and Polybrominated Biphenyls on the Renal and Hepatic Toxicities of Several Chlorinated Hydrocarbon Solvents in Mice." *J. Toxicol. Environ. Health.* 5:605-615.
- Montz, W.E., W.C. Card, and R.L. Kirkpatrick. 1982. "Effects of Polychlorinated Biphenyls and Nutritional Restriction on Barbiturate-Induced Sleeping Times and Selected Blood Characteristics in Raccoons (*Procyon lotor*)." *Bull. Environ. Contam. Toxicol.* 28:578-583.

- Mora, M.A., H.J. Auman, J.P. Ludwig, J.P. Giesy, D.A. Verbrugge and M.E. Ludwig. 1993. Polychlorinated Biphenyls and Chlorinated Insecticides in Plasma of Caspian Terns: Relationships with Age, Productivity, and Colony Site Tenacity in the Great Lakes. *Arch. Environ. Contam. Toxicol.* 24:320-331.
- Murphy, T.J., A. Schinsky, G. Paolucci and C.P. Rzeszutko. 1981. *Atmospheric Inputs of Pollutants to Natural Waters*. S.J. Eisenreich, ed. Ann Arbor Science Publishers. As cited in Simmons 1984.
- Natural Resource Council (NRC). 1992. *Restoration of Aquatic Ecosystems. Science, Technology and Public Policy*. National Academy Press.
- Newsted, J.L., J.P. Giesy, G.T. Ankley, D.E. Tillitt, R.A. Crawford, J.W. Gooch, P.D. Jones, and M.S. Denison. 1995. "Development of toxic equivalency factors for PCB congeners and the assessment of TCDD and PCB mixtures in rainbow trout." *Environ. Toxicol. Chem.*, 14:861-871.
- Niimi, A.J. 1982. "Biological and Toxicological Effects of Environmental Contaminants in Fish and Their Eggs." *Can. J. Fish. Aquat. Sci.* 40:306-312.
- Peakall, D.B., J.L. Lincer, and S.E. Bloom. 1972. "Embryonic mortality and chromosomal alterations caused by Aroclor 1254 in ring doves." *Environ. Health Perspec.*, 1:103-104.
- Peakall, D.B. and M.L. Peakall. 1973. Effect of a polychlorinated biphenyl on the reproduction of artificially and naturally incubated dove eggs. *J. Appl. Ecol.* 10:863-868.
- Peakall, D.B. 1975. "PCBs and Their Environmental Effects." *CRC Critical Rev. Environ. Control.* 5:469-508.
- Pekarik, C., D.V. Weseloh, G.C. Barrett, M. Simon, C.A. Bishop and K.E. Pettit. 1998. *An Atlas of Contaminants in Eggs of Fish-Eating Colonial Birds of the Great Lakes (1993-1997). Vol. I. Accounts by Location*. Environment Canada. Canadian Wildlife Service. Technical Report Series No. 321.
- Pettit, K.E., C.A. Bishop, D.V. Weseloh and R.J. Norstrom. 1994. *An Atlas of Contaminants in Eggs of Fish-Eating Colonial Birds of the Great Lakes (1989-1992)*. Environment Canada. Canadian Wildlife Service. Technical Report Series No. 193.
- Phillips, D.J.H. 1980. *Quantitative Aquatic Biological Indicators: Their Use to Monitor Trace Metal and Organochlorine Pollution*. London: Applied Science Publishers. p. 488.
- Platanow, N.S. and L.H. Karstad. 1973. "Dietary Effects of Polychlorinated Biphenyls on Mink." *Can. J. Comp. Med.* 37:391-400.
- Platanow, N.S. and B.S. Reinhart. 1973. The effects of polychlorinated biphenyls (Aroclor 1254) on chicken egg production, fertility and hatchability. *Can. J. Comp. Med.* 37:341-346.
- Rasmussen, J.B., D.J. Rowan, D.R.S. Lean, and J.H. Carey. 1990. "Food Chain Structure in Ontario Lakes Determines PCB Levels in Lake Trout (*Salvelinus namaycush*) and Other Pelagic Fish." *Can. J. Aquat. Sci.* 47:2030-2038.
- Restum, J.C., S.J. Bursian, J.P. Giesy, J.A. Render, W.G. Helferich, E.B. Shipp, D.A. Verbrugge, and R.J. Aulerich. 1998. "Multigenerational Study of the Effects of Consumption of PCB-Contaminated Carp from Saginaw Bay, Lake Huron, on Mink. 1: Effects on Mink Reproduction, Kit Growth, and Survival, and

Selected Biological Parameters." *J. Toxicol. Env. Health*, 54:343-375.

Risebrough, R.W., P. Rieche, D.B. Peakall, S.G. Herman, and M.N. Kirven. 1968. "Polychlorinated Biphenyls in the Global Ecosystem." *Nature (Lond.)* 220:1098-1102. In: Kluwe, W.M., C.L. Herrmann, and J.B. Hook. 1979. Effects of Dietary Polychlorinated Biphenyls and Polybrominated Biphenyls on the Renal and Hepatic Toxicities of Several Chlorinated Hydrocarbon Solvents in Mice." *J. Toxicol. Environ. Health*. 5:605-615.

Robbins, S.D. 1991. *Wisconsin Birdlife. Population and Distribution Past and Present*. The University of Wisconsin Press, Madison.

Safe, S. 1994. Polychlorinated biphenyls (PCBs): Environmental Impact, Biochemical and Toxic Responses, and Implications for Risk Assessment. *Crit. Rev. Toxicol.* 24:87-149.

Sanders, O.T. and R.L. Kirkpatrick. 1977. "Reproductive Characteristics and Corticoid Levels of Female White-footed Mice Fed *ad Libitum* and Restricted Diets Containing a Polychlorinated Biphenyl." *Environ. Res.* 13:358-363.

Sanders, O.T., R.L. Zepp, and R.L. Kirkpatrick. 1974. "Effect of PCB Ingestion on Sleeping Times, Organ Weights, Food Consumption, Serum Corticosterone and Survival of Albino Mice." *Bull. Environ. Contam. Toxicol.* 12:394-398.

Scott, M.L. 1977. "Effects of PCBs, DDT, and Mercury Compounds in Chickens and Japanese Quail." *Federation Proc.* 36:1888-1893.

Shaw, G.R. and D.W. Connell. 1984. "Physicochemical Properties Controlling Polychlorinated Biphenyl (PCB) Concentrations in Aquatic Organisms." *Environ. Sci. Technol.* 18:23-31. In: Koslowski, S.E., C.D. Metcalfe, R. Lazar, and G.D. Haffner. 1994. "The Distribution of 42 PCBs, Including Three Coplanar Congeners, in the Food Web of the Western Basin of Lake Erie." *J. Great Lakes Res.* 20:260-270.

Simmons, M.S. 1984. PCB Contamination in the Great Lakes. Pages 287-309 in: *Toxic Contaminants in the Great Lakes*. Nriagu, J.O. and M.S. Simmons (eds.). John Wiley and Sons.

Smith, S.L., D.D. MacDonald, K.A. Keenleyside, C.G. Ingersoll, and L.J. Field. 1996. "A preliminary evaluation of sediment quality assessment values for freshwater ecosystems." *J. Great Lakes Res.* 22:624-638.

St. Amant, J.R., M.E. Pariso and T.B. Sheffy. 1984. Polychlorinated Biphenyls in Seven Species of Lake Michigan Fish, 1971-1981. Pages 311-319 in: *Toxic Contaminants in the Great Lakes*. Nriagu, J.O. and M.S. Simmons (eds.). John Wiley and Sons.

Storm, J.E., J.L. Hart and R.F. Smith. 1981. Behavior of Mice After Pre- and Postnatal Exposure to Aroclor 1254. *Neurobehav. Toxicol. Teratol.* 3:5-9.

Stotz, I.J. and Y.A. Greichus. 1978. "The Effects of a Polychlorinated Biphenyl Aroclor 1254 on the White Pelican: Ultrastructure of hepatocytes." *Bull. Environ. Contam. Toxicol.*, 19(3):319-325, In: National Academy of Sciences (NAS). 1979. "Polychlorinated Biphenyls." Rep. Comm. Assess. PCBs in Environ., Environ. Stud. Bd., Comm. Nat. Resour., Nat. Res. Coun., Nat. Acad. Sci., Washington, D.C. 182pp.

Stowe, 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 Application.* 5:248-260.

Stratus Consulting, Inc. 1999a. PCB Pathway Determination for the Lower Fox River/Green Bay Natural Resource Damage Assessment. Available at <<http://www.fws.gov/r3pao/NRDA/index.html>>.

Stratus Consulting, Inc. 1999b. Injuries to Fishery Resources, Lower Fox River/Green Bay Natural Resource Damage Assessment. Available at <<http://www.fws.gov/r3pao/NRDA/index.html>>.

Stratus Consulting, Inc. 1999c. Association Between PCBs, Liver Lesions, and Biomarker Responses in Adult Walleye (*Stizostedium vitreum vitreum*) Collected from Green Bay, Wisconsin. Prepared for U.S. Fish and Wildlife Service. April 13.

Stratus Consulting, Inc. 1999d. Injuries to Avian Resources, Lower Fox River/Green Bay Natural Resource Damage Assessment. Available at <<http://www.fws.gov/r3pao/NRDA/index.html>>.

Struger J. and D.V. Weseloh. 1985. "Great Lakes Caspian Terns: Egg Contaminants and Biological Implications." *Colonial Waterbirds*, 8:142-149.

Sweet, C.W., T.J. Murphy, J.H. Barnasch, C.A. Kelsey and J. Hong. 1991. *Atmospheric Deposition of PCBs into Green Bay*. Annual Meeting of the Air and Waste Management Association. Vancouver, British Columbia, Canada, June 16-21, 1991. P. 116 (abstract only). As cited in *ThermoRetec Consulting Corporation. 1999*

ThermoRetec Consulting Corporation. 1998. *Remedial Investigation, Lower Fox River, Wisconsin*. Prepared for Wisconsin Department of Natural Resources by ThermoRetec, St. Paul, Minnesota and Natural Resources Technology, Inc. Peauaukee, Wisconsin.

ThermoRetec Consulting Corporation. *Baseline Human Health and Ecological Risk Assessment: Lower Fox River, Wisconsin*. Prepared for Wisconsin Department of Natural Resources, Madison, WI. ThermoRetec Project No.: 3-3584-435, February 24, 1999, Seattle, WA.

Thomann, R.V. 1981. "Equilibrium Model of Fate of Microcontaminants in Diverse Aquatic Food Chains." *Can. J. Fish Aquat. Sci.* 38:280-296. In: Koslowski, S.E., C.D. Metcalfe, R. Lazar, and G.D. Haffner. 1994. "The Distribution of 42 PCBs, Including Three Coplanar Congeners, in the Food Web of the Western Basin of Lake Erie." *J. Great Lakes Res.* 20:260-270.

Thomann, R.V., J.P. Connolly, and T.F. Parkerton. 1992. "An Equilibrium Model of Organic Chemical Accumulation in Aquatic Food Webs with Sediment Interaction." *Environ. Toxicol. Chem.* 11:615-629. In: Koslowski, S.E., C.D. Metcalfe, R. Lazar, and G.D. Haffner. 1994. "The Distribution of 42 PCBs, Including Three Coplanar Congeners, in the Food Web of the Western Basin of Lake Erie." *J. Great Lakes Res.* 20:260-270.

Tillitt, D.E., G.T. Ankley, J.P. Giesy, J.P. Ludwig, H. Kurita-Matsuba, D.V. Weseloh, P.S. Ross, C.A. Bishop, L. Sileo, K.L. Stromborg, J. Larson, and T.J. Kubiak. 1992. "Polychlorinated biphenyl residues and egg mortality in double-crested cormorants from the Great Lakes." *Environmental Toxicology and Chemistry*. 11:1281-1288.

Tori, G.M. and T.J. Peterle. 1983. "Effects of PCBs on Mourning Dove Courtship Behavior." *Bull. Environ. Contam. Toxicol.* 30:44-49.

U.S. EPA (U.S. Environmental Protection Agency). 1996. "Great Lakes National Program Office (GLNPO) Green Bay Data Sets." Available at: <<http://www.epa.gov/grtlakes/gbdata/gbay2.html>>.

U.S. EPA (U.S. Environmental Protection Agency). 1997. *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments*. EPA 540-R-97-006.

- Vos, J.G. 1972. "Toxicology of PCBs for Mammals and for Birds." *Environ. Health Perspect.* April:105-117.
- Walker, M.K., J.M. Spitsbergen, J.R. Olson, R.E. Peterson. 1991. "2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity during early life stage development of lake trout (*Salvelinus namaycush*)." *Can. J. Fish. Aquat. Sci.* 48:875-883.
- Welsch, F. 1985. "Effects of Acute or Chronic Polychlorinated Biphenyl Ingestion on Maternal Metabolic Homeostasis and on the Manifestations of Embryotoxicity Caused by Cyclophosphamide in Mice." *Arch. Toxicol.* 57:104-113.
- Williams, L.L., J.P. Giesy, D.Verbrugge, S. Jurzystra and K.L. Stromborg. 1995. Polychlorinated Biphenyl and 2,3,7,8-Tetrachloro-*p*-dioxin Equivalents in Eggs of Double-Crested Cormorants from a Colony near Green Bay, Wisconsin, USA. *Arch. Environ. Contam. Toxicol.* 29:327-333.
- Wren, C.D., D.B. Hunter, J.F. Leatherland, and P.M. Stokes. 1987a. "The Effects of Polychlorinated Biphenyls and Methylmercury, Singly and in Combination on Mink. I: Uptake and Toxic Responses." *Arch. Environ. Contam. Toxicol.* 16:441-447.
- Wren, C.D., D.B. Hunter, J.F. Leatherland, and P.M. Stokes. 1987b. "The Effects of Polychlorinated Biphenyls and Methylmercury, Singly and in Combination on Mink. II: Reproduction and Kit Development." *Arch. Environ. Contam. Toxicol.* 16:449-454.
- Wright, P.J. and D.E. Tillitt. 1998. Dioxin-like Toxicity of Lake Michigan Lake Trout Extract to Developing Lake Trout. Society of Environmental Toxicology and Chemistry 19th Annual Meeting, 15-19 November 1998, Charlotte, NC. Page 264 (abstract only).
- Wright, P.J., D.E. Tillitt and J. Zajicek. 1998. Dioxin Toxicity in Lake Trout Embryos with low Thiamine Levels. Society of Environmental Toxicology and Chemistry 19th Annual Meeting, 15-19 November 1998, Charlotte, NC. Page 265 (abstract only).
- Yamashita, N., S. Tanabe, J.P. Ludwig, H.Kurita, M.E. Ludwig and R. Tatsukawa. 1993. Embryonic Abnormalities and Organochlorine Contamination in Double-Crested Cormorants (*Phalacrocorax auritus*) and Caspian Terns (*Hydroprogne caspia*) from the Upper Great Lakes in 1988. *Environ. Pollut.* 79:163-173.

Table 1. TRVs Selected for Use in the Upper Green Bay Risk Assessment
 Upper Green Bay Portion of the Fox River Site
 Green Bay, WI
 February 2000

RECEPTOR	MEDIA	TRV		UNITS	EFFECT	REFERENCE
		NOAEL	LOAEL			
Fish	Egg	0.16	1.6	mg/kg, ww	Decreased fry growth	Hendricks et al. 1981
Fish	Whole-body	0.77	7.7	mg/kg, ww	Estimated based on egg LOAEL, egg:body ratio	Mac et al. 1993
Piscivorous Bird	Egg	4.7	7.6	mg/kg, ww	Decreased hatching success	Hoffman et al. 1993
Piscivorous Bird	Egg	0.8	8	mg/kg, ww	Decreased hatching success, increased deformity rate	Ludwig et al. 1996
Piscivorous Bird	Diet	0.112	1.12	mg/kgBW/day	Reproductive success and nesting behavior	Tori and Peterle 1983, Peakall and Peakall 1973, Peakall et al. 1972
Piscivorous Mammal	Diet	0.004	0.13	mg/kgBW/day	Kit survival	Heaton et al. 1995

TRV = Toxicity reference value

NOAEL = No observed adverse effect level

LOAEL = Lowest observed adverse effect level

mg/kg, ww = milligrams per kilogram, wet weight

mg/kgBW/day = milligrams per kilogram body weight per day

Table 2. PCB Concentrations in Surface Water, Upper Green Bay
Green Bay Mass Balance Model Data Set
Green Bay, WI
February 2000

Sample Number	SampleDate	PCB Concentrations (µg/L)		
		Dissolved	Particulate	Total
89GG20S23	4/30/89	0.00095	0.00099	0.00195
89GG20S43	5/1/89	0.00073	0.00238	0.00311
89GG20S63	5/1/89	0.00081	0.00044	0.00125
89GG20S83	4/30/89	0.00083	0.00057	0.00140
89GG21S03	4/30/89	0.00070	0.00207	0.00277
89GG21S23	5/1/89	0.00101	0.00131	0.00232
89GG32S63	6/9/89	0.00097	0.00040	0.00137
89GG32S65	6/9/89	0.00051	0.00024	0.00074
89GG32S83	6/8/89	0.00080	0.00019	0.00098
89GG33S03	6/9/89	0.00066	0.00035	0.00101
89GG33S05	6/10/89	0.00049	0.00025	0.00074
89GG33S23	6/10/89	0.00045	0.00027	0.00072
89GG33S43	6/10/89	0.00049	0.00027	0.00076
89GG33S63	6/10/89	0.00057	0.00037	0.00094
89GG33S65	6/10/89	0.00057	0.00047	0.00104
89GG33S83	6/11/89	0.00053	0.00035	0.00088
89GG33S85	6/11/89	0.00058	0.00038	0.00096
89GG42S63	7/28/89	0.00044	0.00024	0.00068
89GG42S65	7/28/89	0.00039	0.00024	0.00062
89GG42S83	7/28/89	0.00048	0.00035	0.00083
89GG43S03	7/29/89	0.00045	0.00029	0.00074
89GG43S05	7/29/89	0.00054	0.00029	0.00083
89GG43S23	7/29/89	0.00040	0.00021	0.00061
89GG43S25	7/29/89	0.00038	0.00030	0.00068
89GG43S43	7/29/89	0.00035	0.00022	0.00057
89GG43S45	7/29/89	0.00051	0.00030	0.00081
89GG43S63	7/29/89	0.00041	0.00023	0.00064
89GG43S65	7/29/89	0.00000 U	0.00028	0.00028
89GG43S83	7/30/89	0.00050	0.00000 U	0.00050
89GG50S43	9/13/89	0.00045	0.00017	0.00062
89GG50S45	9/13/89	0.00069	0.00012	0.00081
89GG50S63	9/13/89	0.00048	0.00016	0.00064
89GG50S83	9/14/89	0.00055	0.00018	0.00073
89GG50S85	9/14/89	0.00052	0.00034	0.00085
89GG51S03	9/14/89	0.00038	0.00019	0.00056
89GG51S05	9/14/89	0.00048	0.00019	0.00067
89GG51S23	9/14/89	0.00076	0.00021	0.00096
89GG51S25	9/14/89	0.00066	0.00038	0.00104
89GG51S43	9/15/89	0.00063	0.00020	0.00083
89GG51S45	9/15/89	0.00061	0.00033	0.00094
89GG51S63	9/15/89	0.00055	0.00021	0.00076
89GG51S65	9/15/89	0.00049	0.00024	0.00072
90GG02S63	10/20/89	0.00073	0.00037	0.00110
90GG02S83	10/21/89	0.00062	0.00075	0.00136
90GG03S03	10/21/89	0.00067	0.00038	0.00105
90GG03S23	10/21/89	0.00065	0.00043	0.00108
90GG03S43	10/21/89	0.00088	0.00051	0.00140
90GG03S63	10/21/89	0.00089	0.00059	0.00148
90GG03S83	10/22/89	0.00083	0.00123	0.00206
90GG10S63	2/17/90	0.00083	0.00052	0.00135
90GG20S43	4/26/90	0.00045	0.00025	0.00070
90GG20S63	4/27/90	0.00042	0.00032	0.00074
90GG20S83	4/27/90	0.00052	0.00032	0.00085
90GG21S03	4/27/90	0.00041	0.00046	0.00087
90GG21S23	4/27/90	0.00038	0.00029	0.00067
90GG21S43	4/27/90	0.00056	0.00040	0.00096
90GG21S63	4/28/90	0.00035	0.00040	0.00075
Mean Water Concentration, Total PCBs				0.00100
Maximum Water Concentration, Total PCBs				0.00311

µg/L = micrograms per liter

U = Not detected

Table 3. Total PCB Concentrations in Sediment Collected from the Upper Green Bay
Green Bay Mass Balance Model Data Set
Green Bay, WI
February 2000

Sample #	Year Collected	Total PCB Concentrations (µg/kg, ww)
E052B09A	1987	18.61
E326B07A	1988	20.13
E309B11A	1988	19.54
E339B02A	1988	23.86
D342B08A	1989	18.68
D344B02A	1989	5.07
D342B02A	1989	12.83
D344B05A	1989	4.63
D344B08A	1989	3.24
E054B08A	1989	6.53
E063B07A	1989	11.47
E071B03A	1989	13.74
E148B02A	1989	16.92
D342B05A	1989	5.78
E184B02A	1989	27.07
E054B02A	1989	10.5
E054B05A	1989	7.98
E304B18A	1989	18.86
E148B07A	1989	19.18
E197B09A	1990	5.22
E204B02A	1990	9.73
E284B08A	1990	3.95
E204B05A	1990	9.31
E284B03A	1990	5.08
E191B05A	1990	4.65
E319B17A	1990	8.74
E191B02A	1990	2.4
E284B17A	1990	3.67
Mean Sediment Concentration:		11.33
Maximum Sediment Concentration:		27.07

µg/kg, ww = micrograms per kilogram, wet weight

Table 4. Total PCB Concentrations in Fish Collected in 1996 from Upper Green Bay
 Natural Resource Damage Assessment Data Set
 Upper Green Bay
 Green Bay, WI
 February 2000

Sample #	Species	Number of Individual Fish per Sample	Total PCBs (mg/kg,ww)
UPPER TROPHIC LEVEL FISH:			
BTUG02CP	Brown Trout	5	1.75
BTUG04CP	Brown Trout	5	1.75
BTUG03CP	Brown Trout	6	1.17
BTUG05CP	Brown Trout	4	1.98
BTUG01CP	Brown Trout	5	1.70
WEUG02CP	Walleye	3	4.61
WEUG03CP	Walleye	3	7.26
WEUG01CP	Walleye	4	5.65
Mean:			3.23
Maximum:			7.26

mg/kg, ww = milligrams per kilogram, wet weight

Table 5. Total PCB Concentrations in Fish Collected in 1989 from Upper Green Bay
Green Bay Mass Balance Model Data Set
Upper Green Bay
Green Bay, WI
February 2000

Sample Number	Date Collected	Species	Total PCBs
			(mg/kg, ww)
FORAGE FISH			
WDI119001BC1	09/11/89	Alewife	0.11
WDJ049008BC1	10/04/89	Alewife	2.00
WDF199001BC1	06/19/89	Alewife	0.25
WDG189001BC1	07/18/89	Alewife	0.98
WDG189002BC1	07/18/89	Alewife	0.85
WDG189003BC1	07/18/89	Alewife	0.90
WDJ049009BC1	10/04/89	Alewife	1.80
WDJ049010BC1	10/04/89	Alewife	1.40
WDF199027BC1	06/19/89	Carp	3.70
WDI129011BC1	09/12/89	Carp	2.90
WDF199025BC1	06/19/89	Carp	4.10
WDK099005BC1	11/09/89	Carp	2.40
WDJ039031BC1	10/03/89	Carp	1.70
WDJ039028BC1	10/03/89	Carp	3.20
WDK089003BC1	11/08/89	Carp	1.90
WDI129015BC1	09/12/89	Carp	4.20
WDI129014BC1	09/12/89	Carp	1.80
WDJ039026BC1	10/03/89	Carp	2.50
WDI069008BC1	09/06/89	Rainbow Smelt	0.84
WDI069006BC1	09/06/89	Rainbow Smelt	0.26
WDI069005BC1	09/06/89	Rainbow Smelt	0.52
WDI069001BC1	09/06/89	Rainbow Smelt	0.78
WDJ049018BC1	10/04/89	Rainbow Smelt	1.60
WDI069004BC1	09/06/89	Rainbow Smelt	0.29
WDI069003BC1	09/06/89	Rainbow Smelt	0.33
WDF199007BC1	06/19/89	Rainbow Smelt	0.47
WDF199006BC1	06/19/89	Rainbow Smelt	0.53
WDF199005BC1	06/19/89	Rainbow Smelt	0.44
WDF199004BC1	06/19/89	Rainbow Smelt	0.43
WDJ049024BC1	10/04/89	Rainbow Smelt	0.22
WDJ049021BC1	10/04/89	Rainbow Smelt	0.19
WDJ049022BC1	10/04/89	Rainbow Smelt	0.81
WDJ049023BC1	10/04/89	Rainbow Smelt	1.10
WDF199003BC1	06/19/89	Rainbow Smelt	0.15
WDJ049025BC1	10/04/89	Rainbow Smelt	0.34
WDF199002BC1	06/19/89	Rainbow Smelt	0.16
Mean for Forage Fish:			1.28
Maximum for Forage Fish:			4.20

Table 5. Total PCB Concentrations in Fish Collected in 1989 from Upper Green Bay
Green Bay Mass Balance Model Data Set
Upper Green Bay
Green Bay, WI
February 2000

Sample Number	Date Collected	Species	Total PCBs
			(mg/kg, ww)
UPPER TROPHIC LEVEL FISH			
WDF079018BC1	06/07/89	Brown Trout	1.80
WDJ099001BC1	12/30/99	Brown Trout	2.20
WDJ099003BC1	12/30/99	Brown Trout	3.90
WDJ189006BC1	12/30/99	Brown Trout	2.80
WDJ189007BC1	12/30/99	Brown Trout	2.40
WDJ189010BC1	12/30/99	Brown Trout	2.70
WDJ099002BC1	12/30/99	Brown Trout	2.30
WDF069003BC1	06/06/89	Brown Trout	1.70
WDG079001BC1	07/07/89	Brown Trout	2.30
WDG209001BC1	07/20/89	Brown Trout	2.30
WDF069002BC1	06/06/89	Brown Trout	2.90
WDG209003BC1	07/20/89	Brown Trout	3.70
WDG209002BC1	07/20/89	Brown Trout	3.10
WDI219001BC1	12/30/99	Walleye	3.80
WDF139004BC1	06/13/89	Walleye	4.80
WDJ149001BC1	12/30/99	Walleye	5.90
WDF139003BC1	06/13/89	Walleye	3.30
WDF139002BC1	06/13/89	Walleye	3.20
WDF139001BC1	06/13/89	Walleye	2.50
WDJ229002BC1	12/30/99	Walleye	0.62
WDJ229003BC1	12/30/99	Walleye	5.70
WDJ319001BC1	12/30/99	Walleye	2.10
WDK089001BC1	12/30/99	Walleye	3.70
WDI209002BC1	12/30/99	Walleye	3.20
WDG209004BC1	07/20/89	Walleye	3.30
WDK149001BC1	12/30/99	Walleye	1.30
Mean for Upper Trophic Level Fish:			2.98
Maximum for Upper Trophic Level Fish:			5.90

mg/kg, ww = milligrams per kilogram, wet weight

Table 6. Total PCB Concentrations in Upper Trophic Level Fish Collected from Upper Green Bay
 NRDA and Mass Balance Data Sets Combined
 Upper Green Bay
 Green Bay, WI
 February 2000

Sample #	Database	Species	Total PCBs (mg/kg, ww)
BTUG02CP	NRDA	Brown Trout	1.75
BTUG05CP	NRDA	Brown Trout	1.98
BTUG01CP	NRDA	Brown Trout	1.70
BTUG03CP	NRDA	Brown Trout	1.17
BTUG04CP	NRDA	Brown Trout	1.75
WDF079018BC1	Mass Balance Model	Brown Trout	1.80
WDJ099001BC1	Mass Balance Model	Brown Trout	2.20
WDJ099003BC1	Mass Balance Model	Brown Trout	3.90
WDJ189006BC1	Mass Balance Model	Brown Trout	2.80
WDJ189007BC1	Mass Balance Model	Brown Trout	2.40
WDJ189010BC1	Mass Balance Model	Brown Trout	2.70
WDJ099002BC1	Mass Balance Model	Brown Trout	2.30
WDF069003BC1	Mass Balance Model	Brown Trout	1.70
WDG079001BC1	Mass Balance Model	Brown Trout	2.30
WDG209001BC1	Mass Balance Model	Brown Trout	2.30
WDF069002BC1	Mass Balance Model	Brown Trout	2.90
WDG209003BC1	Mass Balance Model	Brown Trout	3.70
WDG209002BC1	Mass Balance Model	Brown Trout	3.10
WEUG02CP	NRDA	Walleye	4.61
WEUG03CP	NRDA	Walleye	7.26
WEUG01CP	NRDA	Walleye	5.65
WDI219001BC1	Mass Balance Model	Walleye	3.80
WDF139004BC1	Mass Balance Model	Walleye	4.80
WDJ149001BC1	Mass Balance Model	Walleye	5.90
WDF139003BC1	Mass Balance Model	Walleye	3.30
WDF139002BC1	Mass Balance Model	Walleye	3.20
WDF139001BC1	Mass Balance Model	Walleye	2.50
WDJ229002BC1	Mass Balance Model	Walleye	0.62
WDJ229003BC1	Mass Balance Model	Walleye	5.70
WDJ319001BC1	Mass Balance Model	Walleye	2.10
WDK089001BC1	Mass Balance Model	Walleye	3.70
WDI209002BC1	Mass Balance Model	Walleye	3.20
WDG209004BC1	Mass Balance Model	Walleye	3.30
WDK149001BC1	Mass Balance Model	Walleye	1.30
Overall Mean:			3.04
Overall Maximum:			7.26

NRDA = Natural Resource Damage Assessment Data Set

Mass Balance Model = Green Bay Mass Balance Model Data Set

mg/kg, ww = milligrams per kilogram, wet weight

Table 7. Estimated Total PCB Concentrations in Fish Eggs
Based on Fish Whole Body PCB Concentrations
Upper Green Bay
Green Bay, WI
February 2000

Whole Body Sample #	Database	Species	Estimated Total PCBs in Fish Eggs (mg/kg, ww)
BTUG02CP	NRDA	Brown Trout	0.37
BTUG05CP	NRDA	Brown Trout	0.41
BTUG01CP	NRDA	Brown Trout	0.36
BTUG03CP	NRDA	Brown Trout	0.24
BTUG04CP	NRDA	Brown Trout	0.37
WEUG02CP	NRDA	Walleye	0.96
WEUG01CP	NRDA	Walleye	1.18
WEUG03CP	NRDA	Walleye	1.52
NRDA Database Mean:			0.68
NRDA Database Maximum:			1.52
WDF079018BC1	Mass Balance Model	Brown Trout	0.38
WDJ099001BC1	Mass Balance Model	Brown Trout	0.46
WDJ099003BC1	Mass Balance Model	Brown Trout	0.82
WDJ189006BC1	Mass Balance Model	Brown Trout	0.59
WDJ189007BC1	Mass Balance Model	Brown Trout	0.50
WDJ189010BC1	Mass Balance Model	Brown Trout	0.56
WDJ099002BC1	Mass Balance Model	Brown Trout	0.48
WDF069003BC1	Mass Balance Model	Brown Trout	0.36
WDG079001BC1	Mass Balance Model	Brown Trout	0.48
WDG209001BC1	Mass Balance Model	Brown Trout	0.48
WDF069002BC1	Mass Balance Model	Brown Trout	0.61
WDG209003BC1	Mass Balance Model	Brown Trout	0.77
WDG209002BC1	Mass Balance Model	Brown Trout	0.65
WDI219001BC1	Mass Balance Model	Walleye	0.79
WDF139004BC1	Mass Balance Model	Walleye	1.00
WDJ149001BC1	Mass Balance Model	Walleye	1.23
WDF139003BC1	Mass Balance Model	Walleye	0.69
WDF139002BC1	Mass Balance Model	Walleye	0.67
WDF139001BC1	Mass Balance Model	Walleye	0.52
WDJ229002BC1	Mass Balance Model	Walleye	0.13
WDJ229003BC1	Mass Balance Model	Walleye	1.19
WDJ319001BC1	Mass Balance Model	Walleye	0.44
WDK089001BC1	Mass Balance Model	Walleye	0.77
WDI209002BC1	Mass Balance Model	Walleye	0.67
WDG209004BC1	Mass Balance Model	Walleye	0.69
WDK149001BC1	Mass Balance Model	Walleye	0.27
Overall Mean:			0.64
Overall Maximum:			1.52

mg/kg, ww = milligrams per kilogram, wet weight

Egg concentration = Whole-body PCB concentration times 0.209

0.209 = Egg to whole body ratio calculated for lake trout; Mac et al. 1993

NRDA = Natural Resource Damage Assessment Data Set

Table 8. Total PCB Concentrations in Bird Eggs Collected from Islands In or Near Upper Green Bay
 Upper Green Bay
 Green Bay, WI
 February 2000

Collection Location	Collection Year	Species	Mean Total PCBs (mg/kg, ww)	Standard Deviation	# of eggs	Reference
Gravelly Island	1980	Caspian tern	36.2	9.2	10	Struger and Weseloh 1985
Gravelly/Gull Islands	1988	Caspian tern	11	nd	18	Yamashita et al. 1993
Gravelly Island	1991	Caspian tern	15.8	nd	10	Ewins et al. 1994
Little Gull Island	1986	Double-crested cormorant	14.8	0.1	nd	Tillett et al. 1992
Gravelly/Little Gull Islands	1987	Double-crested cormorant	12.3	0.6	nd	Tillett et al. 1992
Spider Island	1988	Double-crested cormorant	5.3	0.3	nd	Tillett et al. 1992
Little Gull Island	1988	Double-crested cormorant	7.2	nd	41	Yamashita et al. 1993
Spider/Hog/Fish Islands	1988	Double-crested cormorant	14.2	nd	38	Dale and Stromborg 1993
Spider Island	1989	Double-crested cormorant	15.5	8.04	27	Williams et al. 1995
Spider Island	1989 - 1990	Double-crested cormorant	7.8	3.3	26	Larson et al. 1996
Spider Island	1994 - 1995	Double-crested cormorant	10.4*	4.6	10	Custer et al. in press

mg/kg, ww = milligrams per kilogram, wet weight

* wet weight vs. dry weight not specified

nd = no data available

Table 9. Hazard Quotient Calculations for Fish
 Upper Green Bay
 Green Bay, WI
 February 2000

EGGS:

	Estimated PCB Conc. (mg/kg, ww)	Fish Egg NOAEC ($\mu\text{g}/\text{kg}$, ww)	Fish Egg LOAEC ($\mu\text{g}/\text{kg}$, ww)	Fish Egg HQ using the NOAEC	Fish Egg HQ using the LOAEC
NRDA Database Mean	0.68	0.16	1.6	4.2	0.4
NRDA Database Max.	1.52	0.16	1.6	9.5	0.9
Overall Mean	0.64	0.16	1.6	4.0	0.4
Overall Max.	1.52	0.16	1.6	9.5	0.9

WHOLE BODY:

	PCB Conc. (mg/kg, ww)	Whole Body NOAEC (mg/kg, ww)	Whole Body LOAEC (mg/kg, ww)	Whole Body HQ using the NOAEC	Whole Body HQ using the LOAEC
NRDA Database Mean	3.23	0.77	7.7	4.2	0.4
NRDA Database Max.	7.26	0.77	7.7	9.4	0.9
Overall Mean	3.04	0.77	7.7	3.9	0.4
Overall Max.	7.26	0.77	7.7	9.4	0.9

mg/kg, ww = milligrams per kilogram, wet weight

$\mu\text{g}/\text{kg}$, ww = micrograms per kilogram, wet weight

NOAEC = No observable adverse effect concentration

LOAEC = Lowest observed adverse effect concentration

HQ = Hazard quotient

Table 10. Hazard Quotient Calculations for Bird Eggs
 Upper Green Bay
 Green Bay, WI
 February 2000

	Bird Egg PCB Conc. (mg/kg, ww)	Bird Egg NOAEC ^a (mg/kg, ww)	Bird Egg LOAEC ^a (mg/kg, ww)	Bird Egg HQ using the NOAEC	Bird Egg HQ using the LOAEC
Caspian tern	15.8 (mean)	4.7	7.6	3.4	2.1
Double-crested cormorant	10.4 (mean)	4.7	7.6	2.2	1.4
Double-crested cormorant	20.1(max)	4.7	7.6	4.3	2.6

	Bird Egg PCB Conc. (mg/kg, ww)	Bird Egg NOAEC ^b (mg/kg, ww)	Bird Egg LOAEC ^b (mg/kg, ww)	Bird Egg HQ using the NOAEC	Bird Egg HQ using the LOAEC
Caspian tern	15.8 (mean)	0.8	8	19.8	1.9
Double-crested cormorant	10.4 (mean)	0.8	8	13.0	1.3
Double-crested cormorant	20.1(max)	0.8	8	25.1	2.5

^a TRV from Hoffman et al. 1993. Effect observed was decreased hatching success.

^b TRV from Ludwig et al. 1996. Effect observed was increased deformity rate.

mg/kg, ww = milligrams per kilogram, wet weight

NOAEC = No observable adverse effect concentration

LOAEC = Lowest observed adverse effect concentration

HQ = Hazard quotient

Table 11. Food Chain Model and Chronic Hazard Quotient Calculations for the Caspian Tern
 Upper Green Bay
 Green Bay, WI
 February 2000

Using the NOAEL and the Maximum PCB Concentrations:

Maximum Water Conc. (mg/L)	Source of Fish Data	Maximum Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	NOAEL (mg/kg BW/day)	HQ Without Water Ingestion	HQ With Water Ingestion
0.00000311	NRDA Database	7.26	0.04	0.0405	1	0.574	0.51	0.112	4.6	4.6
0.00000311	Mass Balance Model*	4.20	0.04	0.0405	1	0.574	0.30	0.112	2.6	2.6

Using the NOAEL and the Mean PCB Concentrations:

Mean Water Conc. (mg/L)	Source of Fish Data	Mean Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	NOAEL (mg/kg BW/day)	HQ Without Water Ingestion	HQ With Water Ingestion
0.00000100	NRDA Database	3.23	0.04	0.0405	1	0.574	0.23	0.112	2.0	2.0
0.00000100	Mass Balance Model*	1.28	0.04	0.0405	1	0.574	0.09	0.112	0.8	0.8

Using the LOAEL and the Maximum PCB Concentrations:

Maximum Water Conc. (mg/L)	Source of Fish Data	Maximum Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	LOAEL (mg/kg BW/day)	HQ Without Water Ingestion	HQ With Water Ingestion
0.00000311	NRDA Database	7.26	0.04	0.0405	1	0.574	0.51	1.12	0.5	0.5
0.00000311	Mass Balance Model*	4.20	0.04	0.0405	1	0.574	0.30	1.12	0.3	0.3

Using the LOAEL and the Mean PCB Concentrations:

Mean Water Conc. (mg/L)	Source of Fish Data	Mean Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	LOAEL (mg/kg BW/day)	HQ Without Water Ingestion	HQ With Water Ingestion
0.00000100	NRDA Database	3.23	0.04	0.0405	1	0.574	0.23	1.12	0.2	0.2
0.00000100	Mass Balance Model*	1.28	0.04	0.0405	1	0.574	0.09	1.12	0.1	0.1

NRDA = Natural Resource Damage Assessment Data Set

* Data from the Mass Balance Model is for forage fish only.

mg/kg, ww = milligrams per kilogram, wet weight

mg/L = milligrams per liter

L/day = liters per day

kg/day = kilograms per day

AUF = area use factor

mg/kgBW/day = milligrams per kilogram body weight per day

NOAEL = No observable adverse effect level

LOAEL = Lowest observed adverse effect level

HQ = Hazard quotient

Table 12. Food Chain Model and Chronic Hazard Quotient Calculations for the Double-Crested Cormorant
 Upper Green Bay
 Green Bay, WI
 February 2000

Using the NOAEL and the Maximum PCB Concentrations:

Maximum Water Conc. (mg/L)	Source of Fish Data	Maximum Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	NOAEL (mg/kg BW/day)	HQ Without Water Ingestion	HQ With Water Ingestion
0.00000311	NRDA Database	7.26	0.079	0.475	1	1.9	1.81	0.112	16.2	16.2
0.00000311	Mass Balance Model*	4.20	0.079	0.475	1	1.9	1.05	0.112	9.4	9.4

Using the NOAEL and the Mean PCB Concentrations:

Mean Water Conc. (mg/L)	Source of Fish Data	Mean Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	NOAEL (mg/kg BW/day)	HQ Without Water Ingestion	HQ With Water Ingestion
0.00000100	NRDA Database	3.23	0.079	0.475	1	1.9	0.81	0.112	7.2	7.2
0.00000100	Mass Balance Model*	1.28	0.079	0.475	1	1.9	0.32	0.112	2.9	2.9

Using the LOAEL and the Maximum PCB Concentrations:

Maximum Water Conc. (mg/L)	Source of Fish Data	Maximum Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	LOAEL (mg/kg BW/day)	HQ Without Water Ingestion	HQ With Water Ingestion
0.00000311	NRDA Database	7.26	0.079	0.475	1	1.9	1.81	1.12	1.6	1.6
0.00000311	Mass Balance Model*	4.20	0.079	0.475	1	1.9	1.05	1.12	0.9	0.9

Using the LOAEL and the Mean PCB Concentrations:

Mean Water Conc. (mg/L)	Source of Fish Data	Mean Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	LOAEL (mg/kg BW/day)	HQ Without Water Ingestion	HQ With Water Ingestion
0.00000100	NRDA Database	3.23	0.079	0.475	1	1.9	0.81	1.12	0.7	0.7
0.00000100	Mass Balance Model*	1.28	0.079	0.475	1	1.9	0.32	1.12	0.3	0.3

NRDA = Natural Resource Damage Assessment Data Set

* Data from the Mass Balance Model is for forage fish only.

mg/kg, ww = milligrams per kilogram, wet weight

mg/L = milligrams per liter

L/day = liters per day

kg/day = kilograms per day

AUF = area use factor

mg/kgBW/day = milligrams per kilogram body weight per day

NOAEL = No observable adverse effect level

LOAEL = Lowest observed adverse effect level

HQ = Hazard quotient

Table 13. Food Chain Model and Chronic Hazard Quotient Calculations for the Mink
Upper Green Bay
Green Bay, WI
February 2000

Using the NOAEL and the Maximum PCB Concentrations:

Maximum Water Conc. (mg/L)	Maximum Sediment Conc. (mg/kg, ww)	Source of Fish Data	Maximum Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Sediment Ing. Rate (kg/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	NOAEL (mg/kg BW/day)	HQ Without Sed. or Water Ingestion	HQ with Sed and Water Ingestion
0.00000311	0.027	NRDA Database	7.26	0.0572	0.0103	0.114	1	0.52	1.59	0.004	397.6	397.8
0.00000311	0.027	Mass Balance Model*	4.20	0.0572	0.0103	0.114	1	0.52	0.92	0.004	230.2	230.3

Using the NOAEL and the Mean PCB Concentrations:

Mean Water Conc. (mg/L)	Mean Sediment Conc. (mg/kg, ww)	Source of Fish Data	Mean Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Sediment Ing. Rate (kg/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	NOAEL (mg/kg BW/day)	HQ Without Sed. or Water Ingestion	HQ with Sed and Water Ingestion
0.00000100	0.011	NRDA Database	3.23	0.0572	0.0103	0.114	1	0.52	0.71	0.004	177.2	177.3
0.00000100	0.011	Mass Balance Model*	1.28	0.0572	0.0103	0.114	1	0.52	0.28	0.004	70.2	70.2

Using the LOAEL and the Maximum PCB Concentrations:

Maximum Water Conc. (mg/L)	Maximum Sediment Conc. (mg/kg, ww)	Source of Fish Data	Maximum Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Sediment Ing. Rate (kg/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	LOAEL (mg/kg BW/day)	HQ Without Sed. or Water Ingestion	HQ with Sed and Water Ingestion
0.00000311	0.027	NRDA Database	7.26	0.0572	0.0103	0.114	1	0.52	1.59	0.134	11.9	11.9
0.00000311	0.027	Mass Balance Model*	4.20	0.0572	0.0103	0.114	1	0.52	0.92	0.134	6.9	6.9

Using the LOAEL and the Mean PCB Concentrations:

Mean Water Conc. (mg/L)	Mean Sediment Conc. (mg/kg, ww)	Source of Fish Data	Mean Fish Conc. (mg/kg, ww)	Water Ing. Rate (L/day)	Sediment Ing. Rate (kg/day)	Food Ing. Rate (kg/day)	AUF	Body Weight (kg)	Dose (mg/kg BW/day)	LOAEL (mg/kg BW/day)	HQ Without Sed. or Water Ingestion	HQ with Sed and Water Ingestion
0.00000100	0.011	NRDA Database	3.23	0.0572	0.0103	0.114	1	0.52	0.71	0.134	5.3	5.3
0.00000100	0.011	Mass Balance Model*	1.28	0.0572	0.0103	0.114	1	0.52	0.28	0.134	2.1	2.1

NRDA = Natural Resource Damage Assessment Data Set

* Data from the Mass Balance Model is for forage fish only.

mg/kg, ww = milligrams per kilogram, wet weight

mg/L = milligrams per liter

L/day = liters per day

kg/day = kilograms per day

AUF = area use factor

mg/kgBW/day = milligrams per kilogram body weight per day

NOAEL = No observable adverse effect level

LOAEL = Lowest observed adverse effect level

HQ = Hazard quotient

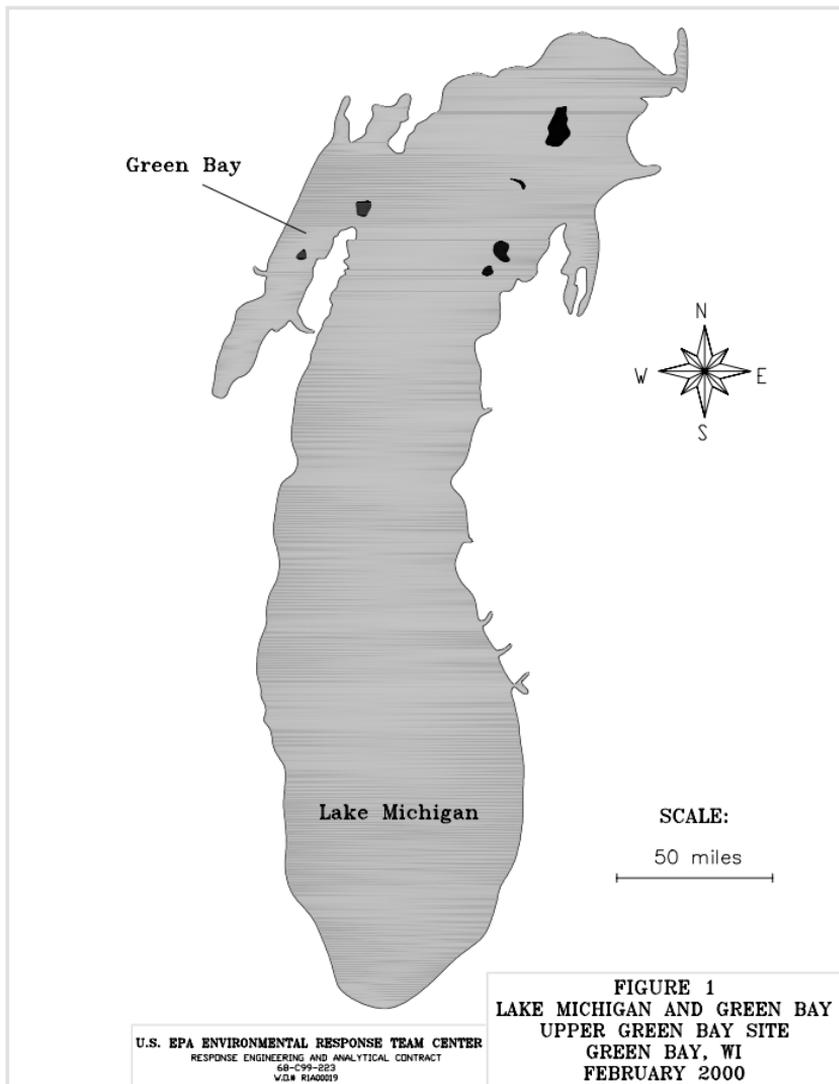
Table 14. Summary of Hazard Quotient Calculation Results
 Upper Green Bay Portion of the Fox River Site
 Green Bay, WI
 February 2000

ASSESSMENT ENDPOINT	LINES OF EVIDENCE	NOAEL HQ	LOAEL HQ	PREDICTED RISK	SECTION NUMBER
Pelagic Fish Reproduction and Survival	Egg Concentration				
	NRDA data mean	4.2	0.4	Potential	6.1.1
	NRDA data maximum	9.5	0.9	Potential	6.1.1
	Overall mean	3.9	0.4	Potential	6.1.1
	Overall maximum	9.5	0.9	Potential	6.1.1
	Adult Tissue Concentration				
	NRDA data mean	4.2	0.4	Potential	6.1.2
	NRDA data maximum	9.4	0.9	Potential	6.1.2
Piscivorous Bird Reproduction and Survival	Overall mean	4	0.4	Potential	6.1.2
	Overall maximum	9.4	0.9	Potential	6.1.2
	Egg Concentration (TRV = 4.7, 7.6)				
	Caspian tern	3.4	2.1	Yes	6.2.1
	Double-crested cormorant				
	Mean	2.2	1.4	Yes	6.2.1
	Maximum	4.3	2.6	Yes	6.2.1
	Egg Concentration (TRV = 0.8, 8.0)				
	Caspian tern	19.8	1.9	Yes	6.2.1
	Double-crested cormorant				
	Mean	13	1.3	Yes	6.2.1
	Maximum	25.1	2.5	Yes	6.2.1
	Food Chain Model				
	Caspian tern				
	NRDA data mean	2	0.2	Potential	6.2.2.1
	NRDA data maximum	4.6	0.5	Potential	6.2.2.1
Overall mean	0.8	0.1	No	6.2.2.1	
Overall maximum	2.6	0.3	Potential	6.2.2.1	
Double-crested cormorant					
NRDA data mean	7.2	0.7	Potential	6.2.2.2	
NRDA data maximum	16.2	1.6	Yes	6.2.2.2	
Overall mean	2.9	0.3	Potential	6.2.2.2	
Overall maximum	9.4	0.9	Potential	6.2.2.2	
Field Studies					
Caspian tern	NA	NA	Not Conclusive	6.2.3.1	
Double-crested cormorant	NA	NA	Yes	6.2.3.2	
Piscivorous Mammal Reproduction and Survival	Food Chain Model				
	Mink				
	NRDA data mean	177.3	5.3	Yes	6.3.1
	NRDA data maximum	397.8	11.9	Yes	6.3.1
	Overall mean	70.2	2.1	Yes	6.3.1
Overall maximum	230.3	6.9	Yes	6.3.1	

HQ = Hazard quotient

NRDA = Natural Resource Damage Assessment Data Set

NA = Data not applicable to hazard quotient method



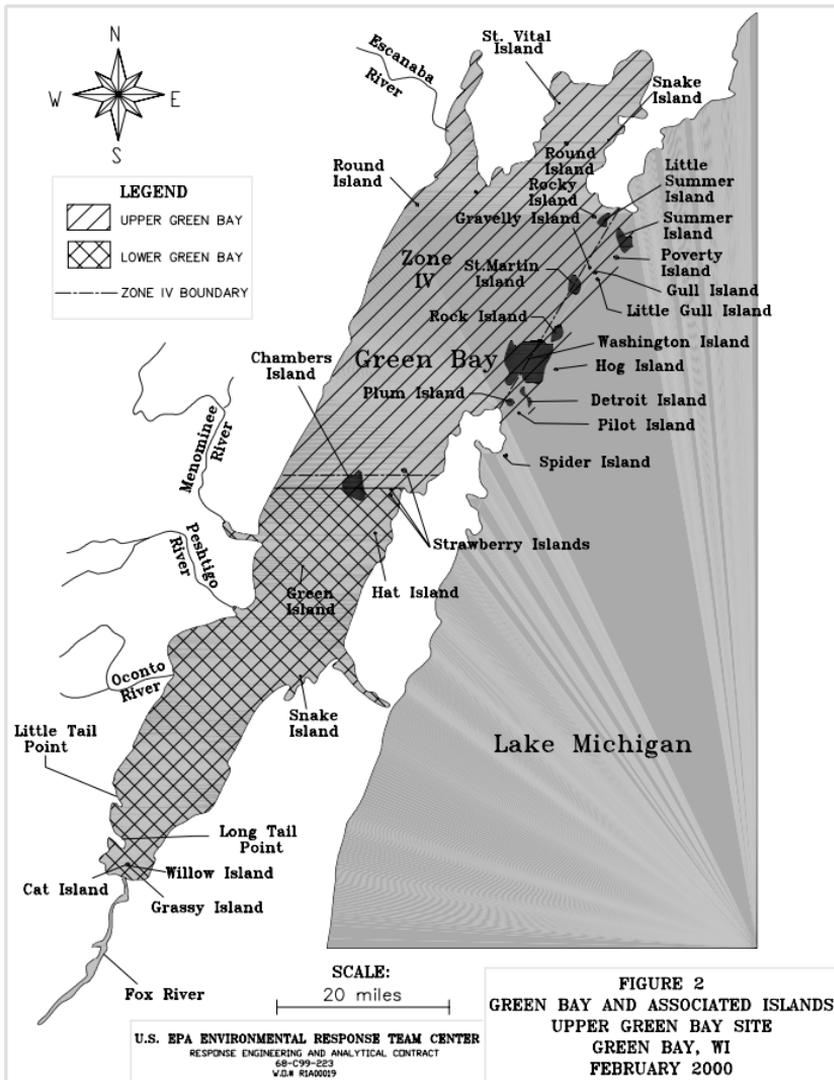
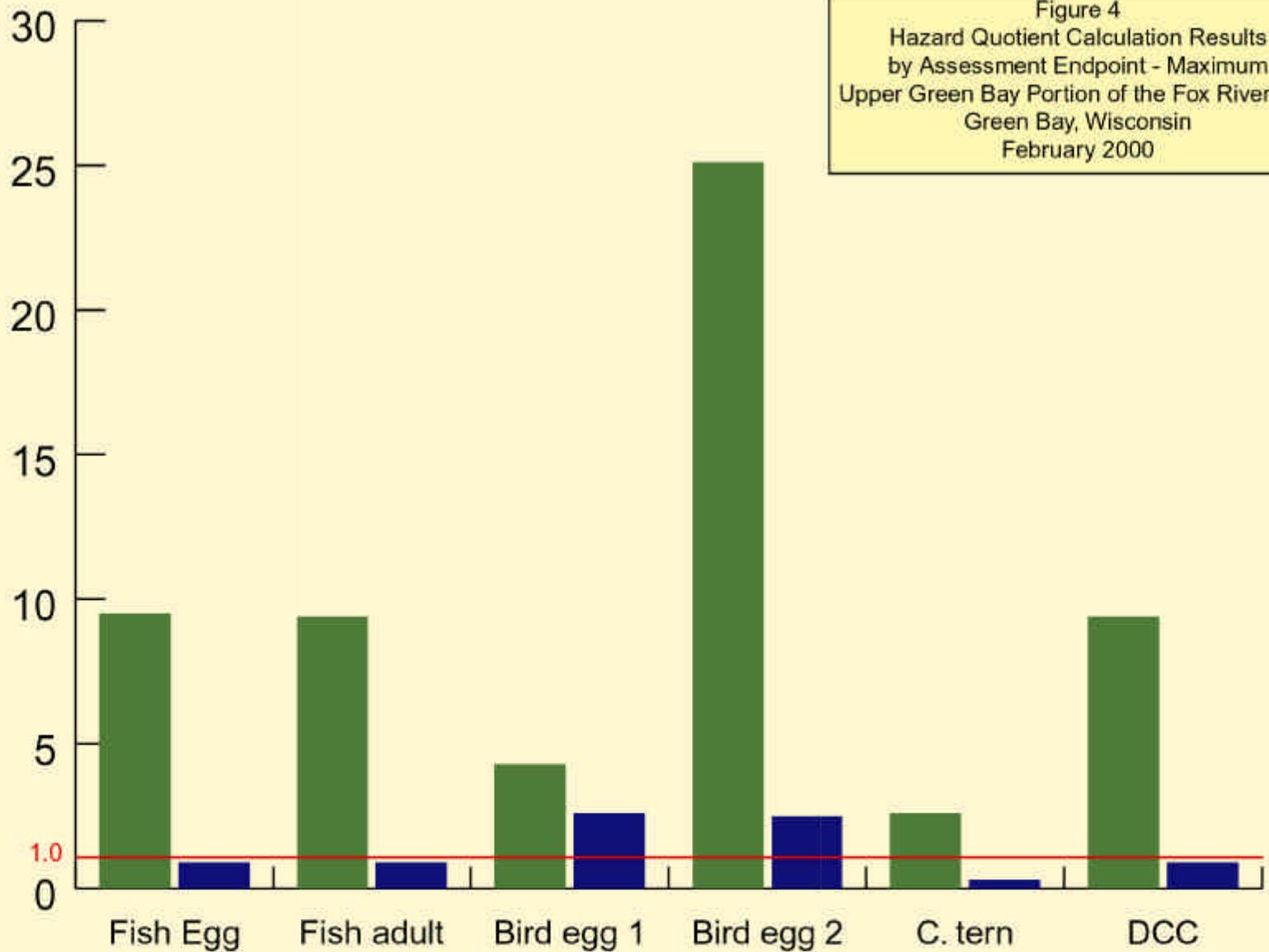
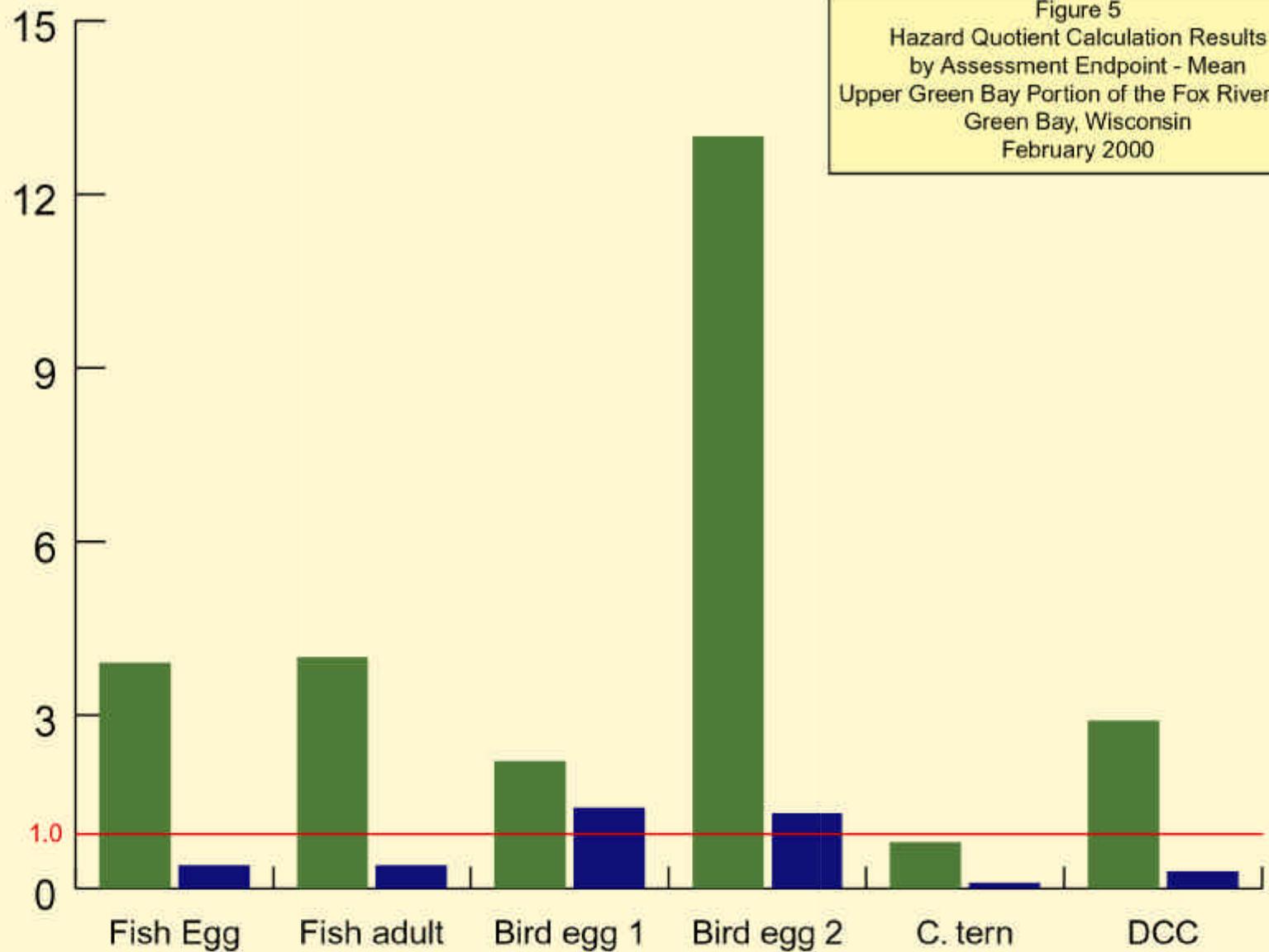


Figure 4
 Hazard Quotient Calculation Results
 by Assessment Endpoint - Maximum
 Upper Green Bay Portion of the Fox River Site
 Green Bay, Wisconsin
 February 2000



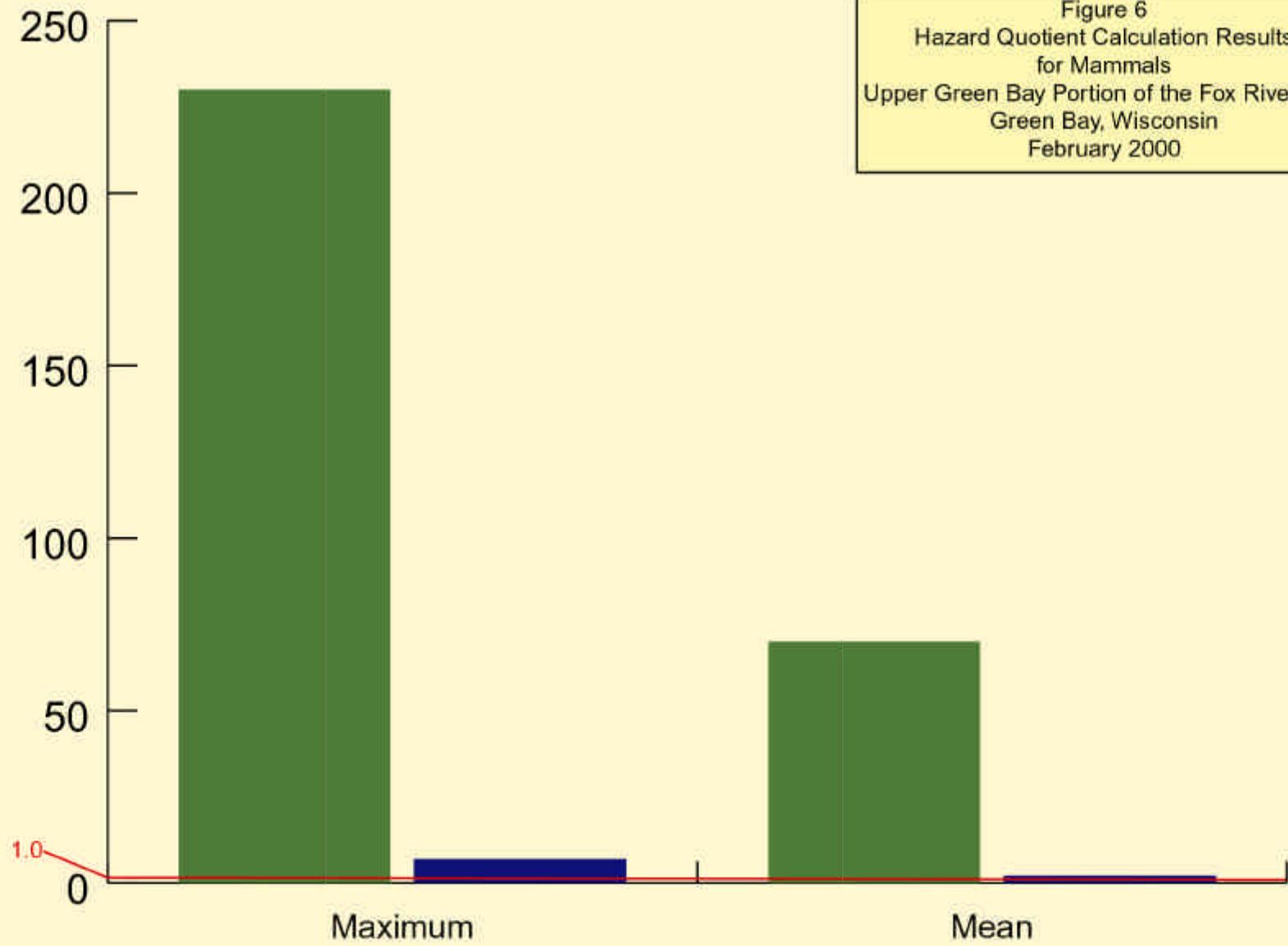
Key	 LOAEL	Bird egg 1: TRV = 4.7, 7.6
	 NOAEL	Bird egg 2: TRV = 0.8, 8.0
		DCC = double crested cormorant
		C. tern = Caspian tern

Figure 5
 Hazard Quotient Calculation Results
 by Assessment Endpoint - Mean
 Upper Green Bay Portion of the Fox River Site
 Green Bay, Wisconsin
 February 2000



Key	 LOAEL	Bird egg 1: TRV = 4.7, 7.6
	 NOAEL	Bird egg 2: TRV = 0.8, 8.0
		DCC = double crested cormorant
		C. tern = Caspian tern

Figure 6
Hazard Quotient Calculation Results
for Mammals
Upper Green Bay Portion of the Fox River Site
Green Bay, Wisconsin
February 2000



Key	 LOAEL
	 NOAEL

APPENDIX A

Toxicity Reference Values
Upper Green Bay Portion of the Fox River Site
Green Bay, WI
February 2000

APPENDIX A

TOXICITY REFERENCE VALUES

A.1 Derivation of Toxicity Reference Values (TRVs)

A toxicity reference value (TRV) is a contaminant dose level that is compared with a predicted exposure dose level, calculated based on site-specific data, in order to assess the presence and degree of risk to a receptor or group of receptors from that contaminant. A TRV is based on data from laboratory toxicological evaluations. Usually, two TRVs are used in order to predict ecological risk, a no observable adverse effect level (NOAEL) and a lowest observable adverse effect level (LOAEL). The NOAEL is the highest dose at which adverse effects are not expected to occur, and the LOAEL is the lowest dose at which adverse effects are expected to occur.

In order to derive TRVs, a comprehensive literature search was performed in which studies on the toxicity of PCBs to ecological receptors were located. A variety of databases were available to be searched for literature references containing toxicological information. Some of these literature sources included Biological Abstracts, Applied Ecology Abstracts, Chemical Abstract Services, Medline, Toxline, BIOSIS, ENVIROLINE, Current Contents, Hazardous Substances Data Bank (HSDB), Registry of Toxic Effects of Chemical Substances (RTECS), Integrated Risk Information System (IRIS), and the Aquatic Information Retrieval Database (ACQUIRE).

In addition, a number of secondary literature sources provided summaries or reviews of the toxicological literature related to a variety of contaminants. These documents were not used directly to derive TRVs because they do not capture the details of the toxicological methods which are imperative to the selection of technically defensible TRVs. However, these summary documents provided an excellent source of original studies that may have been overlooked in the database searches. Examples of such summary documents include Agency for Toxic Substances Disease Registry (ATSDR) documents, U.S. Fish and Wildlife Service Contaminant Hazard Reviews, U.S. EPA Great Lakes Water Quality Initiative documents, and U.S. EPA Ambient Water Quality Criteria documents.

Studies that were obviously not useful or appropriate for deriving a TRV were eliminated. A number of criteria were considered when evaluating the appropriateness of using a particular study for deriving a TRV. The most important consideration was the suitability of the test result for evaluating the assessment endpoint. A number of additional criteria were also considered. For example, studies were selected in which the test organism was in as similar a taxonomic grouping as possible to the measurement endpoint species. Doses had to be quantified and effects measured and reported. The exposure duration was preferably either chronic, sub-chronic, or involved a sensitive life stage, and multigenerational studies were also deemed appropriate. For laboratory studies, the likelihood that a similar result would be obtained if the test were repeated was an additional consideration. Sample sizes had to be adequate and the treatment groups must have been compared to appropriate control groups. At the very least, a negative control should have been included in the study design. In addition, the measured endpoints of the study had to be ecologically relevant. For the purposes of deriving a TRV for an ecological risk assessment, an ecologically relevant endpoint is one which is closely tied to the survival of a population in the field. Usually, the endpoints that are measured for this purpose are survival, growth, and reproduction. In addition, appropriate statistical analyses must have been performed and the statistical significance reported. Finally, the study design preferably included at least three treatments in addition to any controls which may have been selected.

The selected TRVs were based preferably on high-quality studies which satisfy many or all of the requirements above. From these high quality studies, the lowest concentration that was associated with

adverse ecological effects on the test organism was selected as the LOAEL. Studies which reported both a LOAEL and NOAEL were selected over studies which reported only one effect level, due to the uncertainty associated with an unbounded effect level. If a LOAEL could not be located for a receptor, the highest concentration that was associated with no adverse effects was selected as the NOAEL. If only a LOAEL or a NOAEL could be identified from the studies, an uncertainty factor of 10 was used to convert from one to the other (U.S. EPA 1989; Sample et al. 1996; Amdur et al. 1996). Professional judgement was used in some cases to select the most appropriate TRV.

The studies which were used to derive toxicity reference values for this risk assessment are described below. In addition, these studies are also summarized in Table A1.

A.2 Toxicity of PCBs to Fish

A.2.1 Toxicity of PCBs in Fish Eggs

A number of studies indicate that the early life stages of fish are the most sensitive to PCB toxicity and that PCBs are transferred from maternal tissue to eggs (Ankley et al. 1991; Newsted et al. 1995; Larsson et al. 1993). Lake trout eggs have been shown to be particularly sensitive to PCB toxicity (Mac et al. 1985; Mac 1988; Zabel et al. 1995). Therefore, a literature review was conducted to determine toxicity reference values for PCBs in fish eggs. Ankley et al. (1991) collected 10 female Lake Michigan chinook salmon, sampled their eggs, and measured hatching success and fry survival to swim-up. Total PCBs in the eggs were negatively correlated with hatching success. Concentrations of approximately 3.7 and 4.2 mg/kg ww in the egg were identified as the NOAEC and LOAEC, respectively. Mac and Schwartz (1992) found a decrease in hatching of eggs from lake trout collected from the Great Lakes at a PCB concentration of approximately 3 mg/kg,ww, and observed no effects at an egg concentration of approximately 2.8 mg/kg, ww. When 2-year old female rainbow trout were exposed to Aroclor 1254 in the diet for two months and then spawned, fry growth was decreased at a corresponding egg concentration of 1.6 mg/kg, ww (Hendricks et al. 1981). In another study, rainbow trout eggs containing 2.7 mg/kg PCBs, ww, exhibited 75% mortality, and 60 to 70% had deformities after 30 days posthatch (Hogan and Brauhn 1975). Mac and Edsall (1991) collected lake trout from southeastern Lake Michigan, reared them in the laboratory, and measured egg hatchability and fry survival. They found a significant decrease in hatchability and fry survival in eggs with a concentration of 0.314 mg/kg total PCBs. No adverse effects on hatchability and fry survival were noted in eggs with a concentration of total PCBs of 0.173 mg/kg, ww.

Studies conducted using fish collected from the Great Lakes were not utilized to derive TRVs for PCBs in this risk assessment due to the presence of measurable concentrations of other contaminants due to their exposure in Lake Michigan. The study by Hendricks et al. (1981) was used to derive the fish egg toxicity reference values for this risk assessment. This is because of the low LOAEC observed in this study, and the fact that the test species used in this study (rainbow trout) is taxonomically similar to the measurement endpoint species (lake trout). Therefore, a fish egg concentration of 1.6 mg/kg, wet weight, was used as a LOAEC to evaluate the toxicity of PCBs to fish in the upper Green Bay. This value was converted to a NOAEC of 0.16 mg/kg, wet weight, using an accepted conversion factor of 10.

A.2.2 Toxicity of PCBs in Fish Whole Body Tissues

A variety of additional studies have been performed on fish in which reproductive endpoints have been adversely affected and whole body concentrations of PCBs were measured. Lethal body burden concentrations have been estimated at greater than 100 mg/kg for young fish and greater than 250 mg/kg for older fish (Niimi 1996). When fathead minnows were exposed to Aroclor 1254 at 1.8 ug/L, spawning was reduced. Corresponding male and female mean tissue

concentrations were 196 and 429 mg/kg PCBs, respectively (Nebeker et al. 1974). Freeman and Idler (1975) exposed brook trout to 0.2 mg/L Aroclor 1254 in water, and exposed the resulting eggs to either control water or water containing 0.2 mg/L Aroclor 1254. They found that egg hatch was only 78% (compared to 100% in the control) when the eggs were exposed to control water. When the eggs were exposed to water containing Aroclor 1254, none of the eggs hatched. The corresponding adult muscle tissue contained 32.8 mg/kg PCBs. In another study, when fingerling channel catfish were exposed to four Aroclors in the diet for 193 days, no effects on growth were observed, and PCB tissue concentrations were 14 to 32 mg/kg. In the same study, growth in salmon was not affected after exposure to Aroclor 1254 in the diet for 260 days. The salmon tissue concentrations were from 0.4 to 645 mg/kg (Mayer et al. 1977). In another study, adult fathead minnows were exposed for 16 weeks in aquaria containing a 2 to 4 cm layer of sediment contaminated with three different concentrations of PCBs, and tissue PCB concentrations were measured at 7 and 16 weeks. Reproduction was significantly less than the controls in fish exposed to the two highest concentrations. Corresponding mean tissue PCB concentrations ranged from 13.7 to 47.2 mg/kg, wet weight (wet weight). No significant adverse effects were noted in fish exposed to the lowest concentration, corresponding to tissue concentrations ranging from 5.25 mg/kg, wet weight, at 7 weeks to 11.6 mg/kg, wet weight, at 16 weeks (U.S. ACOE 1988). When Mayer et al. (1985) exposed rainbow trout to 2.9 ug/L of an Aroclor mixture (1:2 ratio of 1254:1260) for 90 days, growth was reduced by ten percent. The corresponding PCB tissue concentration was 120 mg/kg. In fish exposed to 0.2 to 5 ug/L of the Aroclor mixture, growth was not affected, and fish tissue concentrations were 6 to 70 mg/kg. Hansen et al. (1976) exposed catfish to 20 mg/kg Aroclor in the diet for 140 days, after which PCB administration was suspended for 56 days, followed by another 56 days with 20 mg/kg PCBs in the diet again. By day 130, growth rates in the PCB-fed fish were significantly lower than those in the control. However, from day 140 to day 252, during which PCBs were fed only during the last 56 days, the growth rate of the PCB-fed fish was greater than that in the controls. By the end of the study, the mean whole body fish concentration in the treated group was 10.86 mg/kg PCBs. When Aroclor 1254 was fed to trout at 15 mg/kg in the diet for 224 days, growth and liver histology were not affected at corresponding tissue concentrations of 8 mg/kg PCBs (Lieb et al. 1974). When brook trout were exposed to 3.1 to 13 ug/L Aroclor 1248 for 118 days, 21-100% mortality was observed, and concentrations of PCBs in dead fry were greater than 125 mg/kg (Mauck et al. 1978). When cyprinid minnows were exposed to Clophen A50 in the diet, premature hatching and death of fry were observed, with corresponding whole body concentrations of 15 and 170 mg/kg, wet weight. No significant adverse effects were noted in fish with corresponding whole body concentrations of 1.6 mg/kg, wet weight (Bengtsson 1980). Mac et al. (1993) found a correlation between embryo mortality and PCB concentrations in lake trout whole body tissues at concentrations ranging from approximately 3 to 14 mg/kg, wet weight. However, since the lake trout in this study were not compared to appropriate controls, a NOAEC and a LOAEC could not be determined from this study.

Another method to determine whole body concentrations at which adverse effects would be expected is to estimate a whole body concentration based on an egg concentration that is associated with adverse effects. This method was derived based on the fact that whole body concentrations are often available, while fish concentrations are not. Early life stages are most sensitive to adverse effects of PCBs, therefore it is important to identify maternal whole-body concentrations that result in critical egg/fry PCB concentrations. In a study by Mac et al. (1993), lake trout whole body and egg concentrations of PCBs were measured in seven lake trout collected from various Great Lakes. When the egg PCB concentrations (wet weight) were divided by the whole body PCB concentrations (wet weight), a mean ratio of 0.209 was calculated. Using this ratio, one can calculate an expected lake trout whole body concentration based on a lake trout egg concentration. Therefore, a whole body concentration that would be expected to elicit adverse effects can be calculated from an egg concentration that has been shown to elicit adverse effects. When the egg LOAEC concentration of 1.6 mg/kg, wet weight, derived above (Section A.2.1), is

divided by 0.209, the resulting whole body concentration is 7.7 mg/kg, wet weight.

Since the latter method provided the lowest LOAEC for whole body fish PCB concentrations, a LOAEC of 7.7 mg/kg, wet weight was selected to evaluate the effects of PCBs on fish survival and reproduction in the upper Green Bay using whole body concentrations. This LOAEC was converted to a NOAEC of 0.77 mg/kg, wet weight, in whole body fish tissue using an accepted conversion factor of 10.

A.3 Toxicity of PCBs to Birds

There is a great degree of variability among different bird species in response to PCBs. In sensitive species, normal patterns of growth, behavior, reproduction, and metabolism may be altered. Liver concentrations of PCBs are generally highest in piscivorous birds, followed by birds that feed on other small birds and mammals, birds that feed on worms and insects, and herbivorous or seed eating birds, respectively (NAS 1979).

A.3.1 Dietary Toxicity of PCBs to Birds

No studies were found in which the toxicity of PCBs to either of the two measurement endpoint species (Forster's tern and double-crested cormorant) was examined. Therefore, literature pertaining to the toxicity of PCBs to other bird species was reviewed and is summarized below. It should be noted that due to the fact that the test species used in the studies summarized below are different from the measurement endpoint species, the dosages calculated in these studies had to be normalized to account for differences in food ingestion rates and body weights between the test species and the measurement endpoint species. To do this, the concentrations of PCBs in food reported in the literature were multiplied by the food ingestion rate and divided by the body weight of the test species. If the food ingestion rate and/or the body weight of the test organisms were not reported in the study, then a food ingestion and/or body weight reported elsewhere in the literature was used. If this information was not available elsewhere in the literature, then body weights were obtained from Dunning (1993) and converted into food ingestion rates using an allometric equation developed by Nagy (1987).

A dietary concentration of 1500 mg/kg (dry weight) was administered to red-winged blackbirds for six days, by which time 50 percent of the birds had died (Stickel et al. 1984). Due to the acute nature of this study (short duration and high mortality), it was not used to assess the chronic effects of PCBs to birds in this risk assessment. In another study, robins, *Erithacus rubecula*, fed a diet containing 5 mg Clophen A50 per day for a period of 11 to 13 days displayed abnormal nocturnal behavior and activity patterns compared to control birds (Ulfstrand and Sondergrund 1971). The average body weight of this robin is reported to be 18.2 grams (Dunning 1993). Subsequently, the daily dose would equal 275 mg Clophen A50/kg/day.

Mallard ducklings, over 9 weeks of age, were fed a PCB-treated diet for 5 days, followed by 3 days of an untreated diet. The 8-day LC50s ranged from 1,975 mg/kg for Aroclor 1260 to 3,182 mg/kg for Aroclor 1242 (Heath et al. 1972). The lowest LC50 value was converted to a LOAEL of 197.5 mg/kg using an accepted conversion factor of 10. In order to express this value in units of mg/kg BW/day, 197.5 mg/kg was multiplied by a food ingestion rate of 0.15 kg/day and the inverse of the lowest reported body weight of 1 kg, both reported for juvenile mallard ducks (Szaro et al. 1981). This yielded an exposure concentration of 29.63 mg/kg BW/day. In another study, a dietary concentration of 150 mg/kg Aroclor 1242 resulted in egg shell thinning of 8.9% in mallard ducks (Haseltine and Prouty 1980). To convert this dosage to units of mg/kg BW/day, the dose was first multiplied by the food ingestion rate for the mallard duck of 0.25 kg/day (Newell et al. 1987), and then divided by the lowest reported adult body weight of 1.043 kg (U.S. EPA 1993) to yield a dose of approximately 36 mg/kg BW/day.

When Aroclor 1254 was fed to 9 month-old mallard hens at a concentration of 25 mg/kg, dry weight, in the diet for at least one month prior to egg laying, no detrimental effects on reproduction or nest attentiveness were observed (Custer and Heinz 1980). Assuming that the diet was one-third solids, this equates to a wet weight concentration of approximately 8.3 mg/kg. To convert this dosage to units of mg/kg BW/day, the dose was first multiplied by the food ingestion rate for the mallard duck of 0.25 kg/day (Newell et al. 1987), and then divided by the lowest reported adult body weight of 1.043 kg (U.S. EPA 1993) to yield a dose of approximately 2.0 mg/kg BW/day.

When screech owls were fed Arclor 1248 in their diet at a concentration of 3 mg/kg for two breeding seasons, the number of eggs per clutch, hatchability, chick malformations, survival, and eggshell thickness were not affected (McLane and Hughes 1980). To convert to units of mg/kg BW/day, this value was divided by the reported mean body weight of 0.185 kg for screech owls (Dunning 1993) and multiplied by a food ingestion rate of 0.019 kg/day that was calculated using an allometric equation (Nagy 1987). This resulted in a dietary dosage of 0.3 mg/kg BW/day.

Nestling white pelicans captured from the wild received 100 mg of Aroclor 1254 as daily oral doses for 10 weeks in addition to a controlled diet. Following the 10 week exposure period, the birds were stressed for an additional 2 weeks by reducing their food consumption in half. The initial mean body weight of the birds prior to the treatment was 6.2 kg. The mean body weight at the end of the 12 week experimental period was 4.8 kg. Micrograph examination of the livers from the birds in the treatment group indicated a 22 percent increase in hepatocyte size, a significant 25 percent increase in the number of mitochondria, a significant 20 percent fewer cristae per mitochondria, and a 22 percent increase in the number of lysosomes, microbodies, and other membrane-bounded vacuoles (Stotz and Greichus 1978). For this risk assessment, the dose (100 mg/day) was multiplied by the inverse of the lower mean body weight (from the end of the experimental period) to yield an exposure concentration of 20.8 mg/kg BW/day.

Peakall and Peakall (1973) maintained ring doves on a diet that contained 10 mg/kg Aroclor 1254. They found that reproductive success was dependent on exposure of the female to the PCB compound. Females fed PCB-spiked food were less attentive to their nest and had erratic nesting behaviors which interfered with egg development. Artificial incubation greatly increased the breeding success for these birds. The food concentration of 10 mg/kg was converted to 1.12 mg Aroclor 1254 /kg/day in chicken feed using 11.2 gm/day as the ingestion rate, and 100 grams as a body mass estimate (data based on mourning dove; Kenaga 1973). Similar values were obtained by Peakall et al. (1972) for the ringed turtle dove, in which a dietary Aroclor 1254 concentration of 10 mg/kg adversely affected hatching success due to heavy embryonic mortality. Another study investigated the behavioral component of reproduction in mourning doves given dietary supplements of 0, 10, or 40 mg/kg Aroclor 1254 (Tori and Peterle 1983). Using the ingestion rate and body weight specified previously (Kenaga 1973), these doses correspond to 0, 1.12 mg/kg BW/day, and 4.48 mg/kg BW/day. Control doves displayed normal courtship behaviors and patterns. Doves that were fed at the 10 ppm (1.12 mg/kg BW/day) level spent twice as much time in the courtship phase as the control birds, with only 50% completing courtship and nesting. Of the 50% that did nest and incubate eggs, nest initiation was significantly delayed, resulting in a delay in egg laying as well. None of the doves on the 40 ppm dietary supplement completed the nesting process (Tori and Peterle 1983). It was hypothesized that the decline of reproductive activity was induced by the degradation of estrogen and androgen present in the birds which is presumably a result of increased hepatic microsomal enzyme activity due to the presence of PCBs (Tori and Peterle 1983).

Hatchability of chicken eggs was reduced in hens fed a diet which was supplemented with 20 mg/kg of total PCBs; reproductive impairment was observed at supplemental dietary levels as low as 5 mg/kg (Heinz et al. 1984). The lower dose was converted to 0.9 mg/kg BW/day using a reported body weight of 0.8 kg and an ingestion rate of 0.14 kg/day for adult chickens (RTECS

1986). When Lillie et al. (1975) exposed chickens to diets containing either Aroclor 1016, 1232, 1242, 1248, or 1254 for 8 weeks, hatching success was significantly reduced at a concentration as low as 10 mg/kg (for Aroclor 1232 and Aroclor 1242), while no effects were noted at a concentration of 5 mg/kg. These values were converted to 1.75 and 0.875 mg/kg BW/day, respectively, using the reported body weight and ingestion rate for chickens indicated above. Similar results were described in Britton and Huston (1973), in which eggs from chickens fed diets containing 10 mg/kg Aroclor 1242 also exhibited reduced hatching success. Again, no effects were observed at a dietary concentration of 5 mg/kg. Similar results were also obtained by Scott (1977), in which hatching success was also decreased in chickens fed a diet containing 10 mg/kg Aroclor 1248. In this study, no effects were observed at 1 mg/kg. The value of 1 mg/kg was converted to 0.175 mg/kg BW/day using a reported body weight of 0.8 kg and an ingestion rate of 0.14 kg/day for adult chickens (RTECS 1986). When Platanow and Reinhart (1973) exposed chickens to Aroclor 1254 in the diet, a concentration of 5 mg/kg resulted in a decrease in both egg production and female fertility. This concentration was converted to a dietary dosage of 0.875 mg/kg BW/day using the reported body weight and ingestion rate indicated above. Finally, when Lillie et al. (1974) exposed chickens to diets containing either Aroclor 1221, 1232, 1242, 1248, 1254, or 1268, chick growth was significantly reduced at a concentration as low as 2 mg/kg (for Aroclors 1248 and 1254). To convert this concentration to units of mg/kg BW/day, the body weight and ingestion rate indicated above were used, yielding a dietary dosage of 0.35 mg/kg BW/day.

Yearling male American kestrels were fed prey items (day-old cockerels) containing approximately 33 mg/kg, wet weight, of Aroclor 1254 for 62 to 69 days. This dose was converted by the investigators to a daily exposure concentration of 9 to 10 mg/kg BW/day. Kestrels receiving the treated diet exhibited a significant 22 to 27 percent reduction in sperm concentrations. This response was associated with a muscle PCB concentration of 107 mg/kg, lipid normalized, and a testes concentration of 128 mg/kg, lipid normalized (Bird et al. 1983).

Male and female pairs of American kestrels were fed diets containing 3 mg/kg, wet weight, of Aroclor 1248 incorporated into a commercial diet for approximately 20 weeks. Eggs were collected from the pairs 2 to 4 days after egg-laying was complete. The eggs collected from the treated pairs of birds exhibited a significant 5 percent reduction in eggshell thickness. This response was associated with a parent muscle tissue PCB concentration of 18.5 ± 5.1 mg/kg, wet weight (Lowe and Stendell 1991). Neither the body weights nor the food ingestion rates were reported in this study; therefore, values from a different study were used to convert the 3 mg/kg dose into an exposure concentration to be used in this risk assessment. The 3 mg/kg dose was multiplied by the inverse of an adult American kestrel body weight of 0.200 kg and a food ingestion rate of 0.0154 kg/day (Nice 1938) to yield an exposure concentration of 0.231 mg/kg BW/day. However, a more recent summary paper by Peakall and Lincer (1996) indicates that PCBs do not cause eggshell thinning except at very high doses that are likely to cause other reproductive toxicological effects as well. Therefore, the LOAEL based on the Lowe and Stendall (1991) study was not used in this risk assessment to evaluate the dietary toxicity of PCBs in birds.

Summer et al. (1996a) exposed white Leghorn hens for eight weeks with commercial diets mixed with contaminated carp from Saginaw Bay, Lake Huron. The concentrations of PCBs in the resulting diets, measured as the sum of Aroclors 1242, 1248, 1254, and 1260, were 0.3 mg/kg (control), 0.8 mg/kg, and 6.6 mg/kg, wet weight. Hens were artificially inseminated weekly, and food consumption, body weights, and egg production were monitored daily. Food consumption initially declined in all the treatment groups but was greatest in the high dose group by the end of the study. Body weights were greater in the control and the low dose groups by the end of the study. Finally, egg production initially decreased during the acclimation period prior to the study, but egg production in the high dose group returned to pre-trial levels by the end of the study while egg production in the control and the low dose group remained significantly lower. The decreased

egg production, as well as the increased body weights, in the control and the low dose group were explained by the authors as effects of fatty liver hemorrhagic syndrome (FLHS), with which the necropsy results were consistent. It was hypothesized that the PCBs in the high dose group provided a protective mechanism against FLHS, thus resulting in the higher egg production, since this protective mechanism had been observed in other studies. In a second phase of this experiment (Summer et al. 1996b), eggs were allowed to develop through day 25 of incubation, and hatching and deformity rates were observed and noted. Rates of deformities correlated with concentrations of PCBs in food, and both treatments (0.8 and 6.6 mg/kg, wet weight, in the diet) produced significantly higher rates of deformities (24% and 40%, respectively) compared to the control (17%). To convert the lower PCB treatment concentration (0.8 mg/kg, wet weight) to units of mg/kg BW/day, the average daily PCB consumption of hens in this treatment group reported by the authors (Summer et al. 1996a) for the 8-week duration of the study (67.1 ug/day) was divided by the corresponding average body weight (1620 g) to obtain a dietary dosage of 0.0414 mg/kg BW/day. To convert the control PCB concentration (0.3 mg/kg, wet weight) to units of mg/kg BW/day, the average daily PCB consumption of hens in this treatment group reported by the authors (Summer et al. 1996a) for the 8-week duration of the study (26.75 ug/day) was divided by the corresponding average body weight (1690 g) to obtain a dietary dosage of 0.0158 mg/kg BW/day. Although this study provided the lowest LOAEL and NOAEL of the studies presented here, these values were not selected for use in this risk assessment because the food source for the study came from an area that is known to contain a variety of pollutants in addition to PCBs, and the contribution of these other contaminants to the effects observed in this study are unknown.

The results of the Tori and Peterle (1983), Peakall and Peakall (1973), and Peakall et al. (1972) studies were selected for use in this risk assessment due to the significance of the endpoints (reproductive success and behavior) and the specificity of the test chemical (PCBs only). Therefore, a LOAEL of 1.12 mg/kg BW/day will be used in this risk assessment to evaluate the risk from PCBs to the Forster's tern and the double-crested cormorant. A NOAEL of 0.112 mg/kg BW/day was calculated from this LOAEL using an accepted conversion factor of 10.

A.3.2 Toxicity of PCBs in Bird Eggs

A variety of field and laboratory studies have been performed in which concentrations of PCBs in bird eggs have been correlated with adverse effects on survival, growth, or reproduction. No apparent adverse reproductive effects were observed in nine colonies of great blue herons, of which the highest mean egg PCB concentration was 7.8 mg/kg, wet weight (Boily et al. 1994). Similarly, no adverse reproductive effects were observed in a field population of black-crowned night herons with mean egg PCB concentrations of up to 10.9 mg/kg, wet weight (Tremblay and Ellison 1980). Mallard ducks fed Aroclor 1254 did not exhibit any adverse effects on reproductive success or nest attentiveness at corresponding egg PCB concentrations of 23.3 mg/kg, wet weight (Custer and Heinz 1980). Haseltine and Prouty (1980) observed 8.9% egg shell thinning at a corresponding mean egg concentration of 105 mg/kg, wet weight, in mallard ducks fed 150 ppm Aroclor 1242. No effects on the number of eggs laid, eggs hatched, number of young fledged, and eggshell thickness were observed in screech owls fed 3 ppm Aroclor 1248, resulting in a mean egg PCB concentration of 7.1 mg/kg, wet weight (McLane and Hughes 1980). In bald eagles, the mean egg PCB concentration in successful nests (defined as having one or more young produced in the year of sample egg collection) was 7.2 mg/kg, wet weight, and in unsuccessful nests, the mean egg PCB concentration was 13 mg/kg, wet weight (Wiemeyer et al. 1984). Similar results were obtained for bald eagles by Wiemeyer et al. (1993), in which a significant reduction in the number of young raised were noted at a corresponding mean egg PCB concentration of 13 mg/kg, although the authors indicate that DDE may have contributed more to the decreased production than PCBs. Wiemeyer (1990) later reports that eagle egg PCB concentrations of 4.0 mg/kg should be adequate to ensure normal reproduction. These studies, however, are confounded by the presence of DDE in the eggs, and controversy exists over the contribution of DDE versus PCBs causing the

observed effects (Bosveld and Van den Berg 1994). Bosveld and Van den Berg (1994) also report adverse effects on hatching success in the Forster's tern and common tern at egg PCB concentrations of 19 mg/kg and 8 mg/kg, respectively, with a corresponding NOAEL for both bird species of 7 mg/kg (Bosveld and Van den Berg 1994).

Struger and Weseloh (1985) did not observe any adverse effects on eggshell thickness or reproductive success in caspian terns from the Great Lakes with egg PCB concentrations as high as approximately 39 mg/kg PCBs, wet weight. Based on data presented in Kubiak et al. (1989), a NOAEC and a LOAEC of 4.5 mg/kg, wet weight, and 22.2 mg/kg, wet weight, respectively, can be derived for hatching success in the Forster's tern. Hoffman et al. (1993) did not observe any apparent adverse effects in a field population of common terns with corresponding egg PCB concentrations of 4.7 mg/kg, wet weight, but a decrease in hatching success and increase in embryo deformities was observed at corresponding egg PCB concentrations of 7.6 mg/kg, wet weight. Peakall et al. (1972) observed a decrease in hatching success due to heavy embryonic mortality at a corresponding mean egg concentration of 50 mg/kg, dry weight, in turtle doves fed 10 ppm Aroclor 1254. Assuming a percent solids composition of 33% for chicken eggs, this corresponds to a wet weight concentration of approximately 16 mg/kg.

Ludwig et al. (1996) reviewed available data on concentrations of contaminants in eggs and observed deformities in embryos and chicks of Double-crested cormorants and Caspian terns. Between 1986 and 1991, hatched chicks and live and dead eggs from 37 colonies in the upper Great Lakes were evaluated annually for gross anatomical deformities. Deformity rates were higher in all Great Lakes areas evaluated (including Green Bay) than at a reference colony. Hatching and deformity rates were correlated with concentrations of planar PCBs and TCDD-EQs. PC concentrations ranged from 3.6 mg/kg in eggs collected from Lake Superior to 7.3 mg/kg in eggs collected from Green Bay; PCB concentration in eggs from the reference colony was 0.8 mg/kg. The authors concluded that the weight of evidence was sufficient to conclude there is a causal relationship between the incidence of deformities in cormorants and terns and exposure to planar halogenated compounds measured as TCDD-EQs or total PCBs in the Great Lakes.

Tillitt et al. (1992) monitored 11 double-crested cormorant colonies around the Great Lakes as well as a reference site outside of the Great Lakes for hatching success in 1986, 1987, and 1988. A significant correlation was found between total egg PCB concentrations and egg mortality. A NOAEC and LOAEC could not be derived from this study because 21% egg mortality was observed in a colony whose mean egg PCB concentration was 0.1 mg/kg, wet weight, whereas the reference area exhibited 8% egg mortality with a corresponding mean egg PCB concentration of 0.8 mg/kg, wet weight. The next highest mean egg PCB concentration was 4.4 mg/kg, wet weight, for another colony, where 26% egg mortality was observed.

When Britton and Huston (1973) exposed laying hens to a dietary concentration 10 ppm Aroclor 1242 in the lab, no effects on hatching success were noted at a corresponding mean egg yolk PCB concentration of 0.95 mg/kg, wet weight, but hatching success was significantly reduced at a corresponding mean egg yolk PCB concentration of 1.5 mg/kg, wet weight. In another study, a drastic reduction in the hatchability of chicks was observed at a corresponding mean egg PCB concentration of 2.5 mg/kg, but no adverse effects on eggshell quality, egg production, or hatchability were noted at a mean egg PCB concentration of 0.36 mg/kg in chickens (Scott 1977). In another study, chickens fed 5 ppm Aroclor 1254 exhibited a significant reduction in egg production and female fertility, with a corresponding egg PCB concentration of 5 mg/kg (Platanow and Reinhart 1973). The same study states that no adverse effects were noted at egg PCB concentrations less than 5 mg/kg, wet weight.

These studies indicate that the chicken is the most sensitive species to PCB toxicity. Indeed, numerous studies have documented the greater sensitivity of chickens to TCDD-like toxicity as

compared to bird species in the wild (Eisler and Belisle 1996). Therefore, studies in which chickens were used as the test subject were not selected for derivation of the NOAEC and LOAEC in this risk assessment, since doing so would overestimate the risk posed to the bird species inhabiting the upper Green Bay.

Based on the Hoffman et al. (1993) study, a LOAEC of 7.6 mg/kg, wet weight, and a NOAEC of 4.7 mg/kg, wet weight, for PCBs in bird eggs were selected for use in the ecological risk assessment. Ludwig et al. (1996) reported a NOAEC of 0.8 mg/kg. This concentration will also be evaluated in this risk assessment for comparative purposes, however it should be noted that this is an unbounded NOAEL and it was not selected as the sole TRV for this reason.

A.4 PCB Toxicity to Mammals

A variety of PCB-induced toxic effects have been observed in mammals. Mink are particularly sensitive to dietary PCB levels (Aulerich et al. 1985; Giesy et al. 1994). Anorexia, weight loss, lethargy, enlarged livers, and intestinal discharge of blood have been noted in exposed mink (Eisler 1986). Placental and mammary transfer of PCBs have been shown to be direct routes of transfer of PCBs between mother and young. PCB exposure can lead to behavioral disorders, specifically in sleep/wake cycles, and in animals that hibernate or aestivate (Montz et al. 1982; Sanders and Kirkpatrick 1977). Negative effects of PCBs on metabolism, thyroid control, ATPase activity, oxidative phosphorylation, steroid hormone activity, immunity, and vitamin A pathways have been noted (Safe 1984; U.S.EPA 1980).

PCB toxicity in mammals is highly variable. While some PCBs are extremely toxic, and can produce death and cause reproductive failure in very low levels, others appear to produce few, if any, toxic responses (Eisler 1986). Toxic responses to PCBs are also highly species specific. Mink are highly susceptible to PCB toxicity, while closely related mammals, such as the European ferret, are more resistant (Eisler 1986). Younger mammals appear to be more susceptible to PCB poisoning than adults (Eisler 1986). Mutagenic, carcinogenic, and teratogenic effects of PCB exposure have been observed, with mutagenic activity appearing to increase with increasing chlorination of the PCB molecule (Eisler 1986).

Several studies were found pertaining to the dietary toxicity of PCBs to mink, most of which examined effects on reproduction, growth and survival. Since the mink is the measurement endpoint receptor to be evaluated in this risk assessment, these mink studies were the only studies that were reviewed to derive a TRV for piscivorous mammals.

In a preliminary study to determine the cause of reproductive complications in mink fed Great Lakes fish, adult breeder mink were fed a basal diet supplemented with 30 mg/kg of PCBs for six months (181 days). However, all of the mink died, emaciated, by the end of the experimental period (Aulerich and Ringer 1977). As a result of the preliminary study, a long-term study was conducted to ascertain the effects of long-term, low-level consumption of PCBs on growth. Mink were fed a basal diet supplemented with 5 and 10 mg/kg of PCBs for a period of approximately 8.5 months. The basal diet plus 10 mg/kg of PCBs resulted in a significant 56 percent decrease in body weight gain after a period of 4 months. Body weight gain was reduced by 39 percent in the 5 mg/kg treatment group, but this reduction was not significant. Both the 5 and 10 mg/kg treatment groups failed to produce offspring; the control group produced 17 live and 8 dead kits. Various degrees of embryotoxicity were observed during necropsy of the treated animals (Aulerich and Ringer 1977). The 5 and 10 mg/kg doses were converted to daily exposure concentrations by multiplying them by the food ingestion rate of 0.114 kg/day [calculated by multiplying the highest reported food ingestion rate for mink of 0.22 g/g BW/day (U.S. EPA 1993) by the lowest reported body weight of 520 g (Merritt 1987), and dividing by 1000] and dividing by the lowest body weight (0.923 kg) reported by the investigators for this treatment group. This yielded exposure concentrations of 1.1 and 2.2 mg/kg BW/day for the 5 and 10 mg/kg treatment groups, respectively.

Based on the results of this experiment, another experiment was conducted to determine the effects of long-

term consumption of low-level PCBs on reproduction. Fifteen mg/kg of Aroclor 1254 in the diet resulted in a complete inhibition of reproduction and 31 percent adult mortality, compared to 6 percent mortality in the controls. Five mg/kg of Aroclor 1254 resulted in a 95 percent reduction in the number of kits born live; the ratio of live kits to female adults was reduced by 87 percent. However, in an effort to determine the persistence of the impaired reproductive condition, 11 adult females that received 5 mg/kg of Aroclor 1254 for a period of six months were placed on a control diet for one year. The results indicate that the impaired reproductive performance of these females was not a permanent condition (Aulerich and Ringer 1977). The 5 and 15 mg/kg doses were converted to daily exposure dosages by multiplying them by the food ingestion rate (0.114 kg/day) for the mink and dividing them by the lowest reported body weight for the mink (0.52 kg) to yield exposure dosages of 1.1 and 3.3 mg/kg BW/day, respectively.

Eight month old mink fed a basal diet containing 1.0 mg/kg of Aroclor 1254 for a period of approximately six months exhibited no mortality or any significant changes in the thyroid, pituitary, adrenal glands, or serum T3 and T4 levels (Wren et al 1987a). Reproduction and kit development was evaluated under the same test conditions in a separate study (Wren et al. 1987b) by the same investigators. Male fertility and female offspring production were not affected by the 1.0 mg/kg Aroclor 1254 diet. However, growth rate of kits nursed by exposed mothers was significantly reduced. The investigators estimated the daily exposure concentrations to be 0.10 mg/kg BW/day for males and 0.18 mg/kg BW/day for females.

When Kubiak and Best (1991) fed mink a liver diet contaminated with PCBs, a concentration of 1.0 mg/kg PCBs resulted in reproductive impairment and a concentration of 5 mg/kg resulted in mortality. This dose was converted to a daily exposure concentration by multiplying it by the food ingestion rate of the mink (0.114 kg/day) and dividing by the lowest reported body weight of mink (0.52 kg). This yielded an exposure concentration of 0.22 mg/kg BW/day.

In another study, one-year-old mink were fed a diet of beef and cereal prepared from cows which had been given 10 consecutive daily oral doses of 1 and 10 mg/kg of Aroclor 1254 dissolved in an olive oil and dairy concentrate (Platanow and Karstad 1973). The cows did not exhibit any clinical, gross, or histopathological signs of PCB toxicity. The cows were killed 24 hours following the last dose, and the musculature, liver, and kidneys ground and mixed with commercial mink food cereal at a level of 24 percent cereal. The resulting rations containing 0.64 and 3.57 mg/kg of total PCB were fed to mink for a period of 160 days. The mink were fed this diet *ad libitum* 2 months prior to the breeding season and continued for 160 days. All 16 mink that were fed 3.57 mg/kg of PCBs died by day 105. Two of the 16 mink that were fed 0.64 mg/kg died by days 122 and 129. The mink exhibited poor appetites, lethargy, and weakness before dying. Some passed tarry feces, indicating gastrointestinal hemorrhaging. At both treatment levels, males survived longer than females. These doses were converted to daily exposure concentrations by multiplying them by the food ingestion rate of the mink (0.114 kg/day) and dividing by the lowest reported body weight of mink (0.52 kg). This yielded exposure concentrations of 0.14 and 0.78 mg/kg BW/day for the 0.64 and 3.57 mg/kg doses, respectively.

In another study, male and female ranch-bred mink were acclimated to a diet consisting of ocean fish scraps, commercial mink cereal, and meat by-products. Ocean fish scraps made up 40 percent of this diet. Dietary treatment levels were prepared by substituting 10, 20, and 40 percent of the ocean fish scraps with PCB-contaminated carp from Saginaw Bay, Lake Huron. The mean dietary PCB concentrations were 0.015 mg/kg (control), 0.72 mg/kg (10 percent carp), 1.53 mg/kg (20 percent carp), and 2.56 mg/kg (40 percent carp). Groups of 15 mink (3 males, 12 females) were assigned to one of the four treatment groups for a period of 12 weeks. Mink receiving the highest PCB-containing diet (40 percent carp or 0.32 mg/kg BW/day, as reported by the investigators) exhibited a 42 percent reduction in mean litter size, 86 percent fewer live kits at birth, and no kits surviving beyond 24-hours post-partum. Even mink receiving the 10 percent carp diet (or 0.13 mg/kg BW/day, as reported by the investigators) exhibited a 67 percent reduction in kits surviving three to six weeks relative to the control (Heaton et al. 1995).

In a related study on multigenerational effects in mink fed the same Saginaw Bay PCB-contaminated carp,

Restum et al.(1998) observed a significant reduction in kit body weights after parental exposure to 0.25 mg/kg, wet weight (0.05 mg/kg BW/day, as reported by the authors) of PCBs in fish. A significant reduction in kit survival was observed at a parental exposure concentration of 0.5 mg/kg wet weight. Of note in their study was that adverse effects on kit survival were observed even several months after the parents had been placed on the control diet. The inference was that long-term effects on mink can be observed even after short exposure periods to a PCB-contaminated diet. Some uncertainty is associated with using this study to derive the LOAEL because the mink in these studies were fed carp from Saginaw Bay, an area known to contain contaminants in addition to PCBs. However, the authors purport that the results of other studies on the effects of DDT, dieldrin, and heptachlor on mink indicate that at least these contaminants are not likely to have contributed to the toxicity observed in their study.

The LOAEL and NOAEL observed in the Heaton et al (1995) study (0.72 and 0.015 mg/kg diet) were selected as the TRVs for this risk assessment. The LOAELs cited by Heaton et al. (1995; 0.72 mg/kg) and Restum et al. (1998; 0.5 mg/kg) are effectively the same, and probably fall within the margin of error of the two studies. The daily exposure levels of 0.134 and 0.004 mg/kgBW/day reported by Heaton et al. (1995) were used in risk calculations.

REFERENCES

- Amdur, M.O., J. Doull, and C.D. Klaasen. 1996. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 5th Ed. New York: McGraw-Hill. p. 80-81.
- Ankley, G.T., D.E. Tillitt, J.P. Giesy, P.D. Jones, and D.A. Verbrugge. 1991. "Bioassay-Derived 2,3,7,8-Tetrachlorodibenzo-p-dioxin Equivalents in PCB-containing Extracts from the Flesh and Eggs of Lake Michigan Chinook Salmon (*Oncorhynchus tshawytscha*) and Possible Implications for Reproduction." *Can. J. Fish. Aquat. Sci.*, 48:1685-1690.
- Aulerich, R. J., S.J. Bursian, W.J. Breslin, B.A. Olson, and R.K. Ringer. 1985. "Toxicological manifestations of 2,4,5,2',4',5'-, 2,3,6,2'3'6'-, and 3,4,5,3'4'5'-hexachlorobiphenyl and Aroclor 1254 in Mink." *J. Toxicol. Environ. Health.*, 15:63-79.
- Aulerich, R.J. and R.K. Ringer. 1977. "Current Status of PCB Toxicity to Mink, and Effect on Their Reproduction." *Arch. Environ. Contam. Toxicol.*, 6:279-292.
- Bengtsson, B.E. 1980. "Long-Term Effects of Polychlorinated Biphenyl Clophen A-50 on Growth, Reproduction, and Swimming Performance in the Minnow *Phoxinus phoxinus*." *Water Res.*, 14:681-688.
- Bird, D.M., P.H. Tucker, G.A. Fox, and P.C. Lague. 1983. "Synergistic Effects of Aroclor 1254 and Mirex on the Semen Characteristics of American Kestrels." *Arch. Environ. Contam. Toxicol.*, 12:633-640.
- Boily, M.H., L. Champoux, D.H. Bourbonnais, J.L. Des Granges, J. Rodrigue and P.A. Spear. 1994. "β-carotene and retinoids in eggs of great blue herons (*Ardea herodias*) in relation to St. Lawrence River contamination." *Ecotoxicology* 3:271-286.
- Bosveld, A.T.C. and M. Van den Berg. 1994. "Effects of polychlorinated biphenyls, dibenzo-p-dioxins, and dibenzofurans on fish-eating birds." *Environmental Reviews*, 2:147-166.
- Britton, W.M. and T.M. Huston. 1973. "Influence of polychlorinated biphenyls in the laying hen." *Poult. Sci.*, 32:1620-1624.
- Custer, T.W. and G.H. Heinz. 1980. "Reproductive success and nest attentiveness of mallard ducks fed Aroclor 1254." *Environ. Pollut.*, 21:313-318.
- Den Boer, M.H. 1984. "Reproduction Decline of Harbour Seals: PCBs in the Food and their Effect on Mink." *Netherlands Research Institute for Nature Management Annual Report*, p. 77-86.
- Dunning, J.B. 1993. *Avian Body Masses*. Boca Raton: CRC Press.
- 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). 72 pp.
- Eisler, R. and A.A. Belisle. 1996. Planar PCB Hazards to Fish, Wildlife and Invertebrates: A Synoptic Review. *National Biological Service Biological Report* 31. 75 pp.
- Freeman, H.C. and D.R. Idler. 1975. "The Effect of Polychlorinated Biphenyl on Steroidogenesis and Reproduction of Brook Trout (*Salvelinus fontinalis*)." *Can. J. Biochem.*, 53:666-670.
- Giesy, J.P., D.A. Verbrugge, R.A. Othout, W.W. Bowerma, M.A. Mora, P.D. Jones, J.L. Newsetd, C. Vandervoot, S.N. Heaton, R.J. Aulerich, S.J. Bursian, J.P. Ludwig, G.W. Dawson, T.J. Kubiak, D.A. Best,

- D.E. Tillitt. 1994. "Contaminants in Fishes from Great Lakes-Influenced Sections and Above Dams of Three Michigan Rivers: II. Implications for Health of Mink." *Arch. Contam. Toxicol.*, 60:1282-1288.
- Hansen, D.J., S.C. Schimmel, and J. Forester. 1974. "Aroclor 1254 in Eggs of Sheepshead Minnows: Effect on Fertilization Success and Survival of Embryos and Fry." *Proc. Southeastern Assoc. Game Fish. Comm.*, p. 805-812.
- Hansen, D.J., Schimmel, S.C., and J. Forester. 1975. "Effects of Aroclor 1016 on Embryos, Fry, Juveniles and Adults of Sheepshead Minnows (*Cyrinodon variegatus*)." *Trans. Amer. Fish. Soc.*, 104:584-588.
- Hansen, L.G., W.B. Wiekhotst, and J. Simon. 1976. "Effects of Dietary Aroclor 1242 on Channel Catfish (*Ictalurus punctatus*) and the Selective Accumulation of PCB Components." *J. Fish. Res. Board Can.*, 33:1343-1352.
- Haseltine, S.D. and R.M. Prouty. 1980. "Aroclor 1242 and reproductive success of adult mallards (*Anas platyrhynchos*)." *Environ. Res.*, 23:29-34.
- Heath, R.G., J.W. Spann, E.F. Hill, and J.F. Kreitzer. 1972. "Comparative Dietary Toxicities of Pesticides to Birds." U.S. Fish and Wildl. Service, Special Scientific Report - Wildlife No. 152. Washington, D.C.
- Heaton, S.N., S.J. Bursian, J.P. Giesy, D.E. Tillitt, J.A. Render, P.D. Jones, D.A. Verbrugge, T.J. Kubiak, and R.J. Aulerich. 1995. "Dietary Exposure of Mink to Carp from Saginaw Bay, Michigan. 1. Effects on Reproduction and Survival, and the Potential Risks to Wild Mink Populations." *Arch. Environ. Contam. Toxicol.*, 28:334-343.
- Heinz, G.H., D.M. Swineford, and D.E. Katsma. 1984. "High PCB Residues in Birds From the Sheboygan River, WI." *Environ. Monitor. Assess.*, 4:155-161.
- Hendricks, J.D., W.T. Scott, T.P. Putnam, and R.O. Sinnhuber. 1981. Enhancement of aflatoxin B1 hepatocarcinogenesis in rainbow trout (*Salmo gairdneri*) embryos by prior exposure of gravid females to dietary Aroclor 1254. p. 203-214. In: D.R. Branson and K.L. Dickson (Eds.). Aquatic toxicology and hazard assessment: fourth conference. ASTM STP 737. American Society for Testing and Materials, Philadelphia, PA.
- Hoffman D.J., G.J. Smith, and B.A. Rattner. 1993. "Biomarkers of contaminant exposure in common terns and black-crowned night herons in the Great Lakes." *Environ. Toxicol Chem.*, 12:1095-1103.
- Hogan, J.W. and J.L. Brauhn. 1975. "Abnormal Rainbow Trout Fry from Eggs Containing High Residues of a PCB (Aroclor 1242)." *Prog. Fish-Cult.*, 37:229.
- Holm G., L. Norrgren, T. Andersson, and A. Thuren. 1993. "Effects of Exposure to Food Contaminated with PBDE, PCN or PCB on Reproduction, Liver Morphology and Cytochrome P450 Activity in the Three-spined Stickleback, *Gasterosteus aculeatus*." *Aquatic Toxicology (Amsterdam)*, 27:33-50.
- Kenaga, E.E. 1973. Factors to be considered in the evaluation of the toxicity of pesticides to birds in their environment. Pages 166-181 in : Environmental quality and safety, global aspects of chemistry, toxicology and technology as applied to the environment. Vol. II Coulston, F. And F. Korte, eds. Academic Press, Inc. New York.
- Kubiak T.J., H.J. Harris, L.M. Smith, T.R. Schwartz, D.L. Stalling, J.A. Trick, L. Sileo, D.E. Docherty and T.C. Erdman. 1989. "Microcontaminants and reproductive impairment of the Forster's Tern on Green Bay, Lake Michigan-1983." *Arch. Environ. Contam. Toxicol.*, 18:706-727.

- Kubiak, T.J. and D.A. Best. 1991. "Wildlife risks associated with passage of contaminated anadromous fish at federal energy regulatory commission licensed dams in Michigan." United States Fish and Wildlife Service White Paper. Contaminants Program, Division of Ecological Services, USFWS. East Lansing, Michigan.
- Larsson, P., L. Orla, and L. Collvin. 1993. "Reproductive status and lipid content as factors in PCB, DDT and HCH contamination of a population of Pike (*Esox lucius* L.)." *Environ. Toxicol. Chem.*, 12:855-861.
- Lieb, A.J., D.D. Bills, and R.O. Sinnhuber. 1974. "Accumulation of Dietary Polychlorinated Biphenyls (Aroclor 1254) by Rainbow Trout (*Salmo gairdneri*)." *J. Agric. Food Chem.*, 22:638-642.
- Lillie, R.J., H.C. Cecil, J. Bitman, G.F. Fries, and J. Verrett. 1974. "Differences in response of caged white leghorn layers to various polychlorinated biphenyls (PCBs) in the diet." *Poult. Sci.*, 53:726-732.
- Lillie, R.J., H.C. Cecil, J. Bitman, G.F. Fries, and J. Verrett. 1975. "Toxicity of certain polychlorinated and polybrominated biphenyls on reproductive efficiency of caged chickens." *Poult. Sci.*, 54:1550-1555.
- Lowe, P.T. and R.C. Stendell. 1991. "Eggshell Modifications in Captive American Kestrels Resulting from Aroclor 1248 in the Diet." *Arch. Environ. Contam. Toxicol.*, 20:519-522.
- Ludwig, J.P., H. Kurita-Matsuba, H.J. Auman, M.E. Ludwig, C.L. Summer, J.P. Giesy, D.E. Tillitt, and P.D. Jones. 1996. "Deformities, PCBs, and TCDD-equivalents in double-crested cormorants (*Phalacrocorax auritus*) and Caspian terns (*Hydroprogne caspia*) of the upper Great Lakes 1986-1991: Testing a cause-effect hypothesis." *Great Lakes Res.* 22:172-197.
- Mac, M.J. 1988. "Toxic Substances and Survival of Lake Michigan Salmonids: Field and Laboratory Approaches." In: *Toxic Contaminants and Ecosystem Health: A Great lakes Focus*, Ed. M.S. Evans, New York, NY: John Wiley & Sons.
- Mac, M.J. and C.C. Edsall. 1991. "Environmental Contaminants and the Reproductive Success of Lake Trout in the Great Lakes: An Epidemiological Approach." *Journal of Toxicology and Environmental Health*, 33:375-394.
- 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." *J. Great Lakes Res.* 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*, 25:189-192.
- 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." *J. Great Lakes Res.*, 19:752-765.
- Mauck, W.L., P.M. Mehrle, and F.L. Mayer. 1978. "Effects of the Polychlorinated Biphenyl Aroclor 1254 on Growth, Survival, and Bone Development in Brook Trout (*Salvelinus fontinalis*)." *J. Fish. Res. Board Can.*, 35:1084-1088.
- Mayer, F.L., P.M. Mehrle, and H.O. Sanders. 1977. "Residue Dynamics and Biological Effects of Polychlorinated Biphenyls in Aquatic Organisms." *Arch. Environ. Contam. Toxicol.*, 5:501-511.
- Mayer, K.S., F.L. Mehrle, and A. Witt, Jr. 1985. "Waste Transformer Oil and PCB Toxicity to Rainbow Trout." *Trans. Am. Fish. Soc.* 114:869-886.

- McLane, A.R. and Hughes, D.L. 1980. "Reproductive success of screech owls fed Aroclor 1248." *Arch. Environ. Contam. Toxicol.*, 9:661-665.
- Merritt, J.F. 1987. *Guide to the Mammals of Pennsylvania*. Pittsburgh, PA: University of Pittsburgh Press. 408 p.
- Montz, W.E., W.C. Card, and R.L. Kirkpatrick. 1982. "Effects of Polychlorinated Biphenyls and Nutritional Restriction on Barbiturate-Induced Sleeping Times and Selected Blood Characteristics in Raccoons (*Procyon lotor*)." *Bull. Environ. Contam. Toxicol.* 28:578-583.
- Nagy, K.A. 1987. "Field Metabolic Rate and Food Requirement Scaling in Mammals and Birds." *Ecol. Mongr.* 57:111-128.
- NAS (National Academy of Sciences). 1979. "Polychlorinated Biphenyls." Rep. Comm. Assess. PCBs in Environ., Environ. Stud. Bd., Comm. Nat. Resour., Nat. Res. Coun., Nat. Acad. Sci., Washington, D.C. 182pp.
- 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." *Trans. Amer. Fish. Soc.*, 103:562-568.
- Newell, A., D.W. Johnson, and L. Allen. 1987. "Niagra River Biota Contamination Project: Fish Flesh Criteria for Piscivorous Wildlife." NYS DEC Technical Report 87-3.
- Newsted, J.L., J.P. Giesy, G.T. Ankley, D.E. Tillitt, R.A. Crawford, J.W. Gooch, P.D. Jones, and M.S. Denison. 1995. "Development of toxic equivalency factors for PCB congeners and the assessment of TCDD and PCB mixtures in rainbow trout." *Environ. Toxicol. Chem.*, 14:861-871.
- Nice, M.M. 1938. "The Biological Significance of Bird Weights." *Bird Banding*, 9(1):1-11. In: Pages 166-181, Kenaga, E.E. 1973. "Factors to Be Considered in the Evaluation of the Toxicity of Pesticides to Birds in their Environment." Environmental Quality and Safety. Academic Press, New York.
- Niimi, A.J. 1996. "PCBs in aquatic organisms." In: *Environmental Contaminants in Wildlife: Interpreting Tissue Concentrations*. Eds. W.N. Beyer, G.H. Heinz, and A. W. Redmon-Norwood. Boca Raton, FL: Lewis Publishers. p. 117-152.
- Peakall, D.B., J.L. Lincer, and S.E. Bloom. 1972. "Embryonic mortality and chromosomal alterations caused by Aroclor 1254 in ring doves." *Environ. Health Perspec.*, 1:103-104.
- Peakall, D.B. and J.L. Lincer. 1996. "Do PCBs cause eggshell thinning?" *Environmental Pollution*, 97:127-129.
- Peakall, D.B. and M.L. Peakall. 1973. Effect of a polychlorinated biphenyl on the reproduction of artificially and naturally incubated dove eggs. *J. Appl. Ecol.* 10:863-868.
- Platanow, N.S. and L.H. Karstad. 1973. "Dietary Effects of Polychlorinated Biphenyls on Mink." *Can. J. Comp. Med.*, 37:391-400.
- Platanow, N.S. and B.S. Reinhart. 1973. "The effects of polychlorinated biphenyls (Aroclor 1254) on chicken egg production, fertility and hatchability." *Can. J. Comp. Med.*, 37:341-346.
- Restum, J.C., S.J. Bursian, J.P. Giesy, J.A. Render, W.G. Helferich, E.B. Shipp, D.A. Verbrugge, and R.J. Aulerich. 1998. "Multigenerational Study of the Effects of Consumption of PCB-Contaminated Carp from Saginaw Bay, Lake Huron, on Mink. 1: Effects on Mink Reproduction, Kit Growth, and Survival, and

Selected Biological Parameters." *J. Toxicol. Env. Health*, 54:343-375.

RTECS. 1986. Registry of Toxic Effects of Chemical Substances Database. Published by the National Institute for Occupational Safety and Health (NIOSH).

Safe, S. 1984. "Polychlorinated biphenyls (PCBs) and polybromated biphenyls (PBBs): Biochemistry, toxicology, and mechanisms of action." *CRC Crit. Rev. Toxicol.*, 13:319-393.

Sample, B.E., D.M. Opresko, and G.W. Suter. 1996. Toxicological Benchmarks for Wildlife: 1996 Revision. Prepared for the U.S. Department of Energy. ES/ER/TM-86/R3. p. 5-6.

Sanders, O.T. and R.L. Kirkpatrick. 1977. "Reproductive Characteristics and Corticoid Levels of Female White-footed Mice Fed *ad Libitum* and Restricted Diets Containing a Polychlorinated Biphenyl." *Environ. Res.* 13:358-363.

Scott, M.L. 1977. "Effects of PCBs, DDT, and mercury compounds in chickens and Japanese quail." *Federation Proceed.*, 36:1888-1893.

Stickel, W.H., L.F. Stickel, R.A. Dyrland, and D.L. Hughes. 1984. "Aroclor 1254 Residues in Birds: Lethal Levels and Loss Rates." *Arch. Environ. Contam. Toxicol.*, 13:7-13.

Stotz, I.J. and Y.A. Greichus. 1978. "The Effects of a Polychlorinated Biphenyl Aroclor 1254 on the White Pelican: Ultrastructure of hepatocytes." *Bull. Environ. Contam. Toxicol.*, 19(3):319-325, In: National Academy of Sciences (NAS). 1979. "Polychlorinated Biphenyls." Rep. Comm. Assess. PCBs in Environ., Environ. Stud. Bd., Comm. Nat. Resour., Nat. Res. Coun., Nat. Acad. Sci., Washington, D.C. 182pp.

Struger J. and D.V. Weseloh. 1985. "Great Lakes caspian terns: egg contaminants and biological implications." *Colonial Waterbirds*, 8:142-149.

Summer, C.L., J.P. Giesy, S.J. Bursian, J.A. Render, T.J. Kubiak, P.D. Jones, D.A. Verbrugge, and R.J. Aulerich. 1996a. "Effects induced by feeding organochlorine-contaminated carp from Saginaw Bay, Lake Huron, to laying White Leghorn hens. I. Effects on health of adult hens, egg production, and fertility." *J. of Toxicol. and Environ. Health*, 49:389-407.

Summer, C.L., J.P. Giesy, S.J. Bursian, J.A. Render, T.J. Kubiak, P.D. Jones, D.A. Verbrugge, R.J. Aulerich. 1996b. "Effects induced by feeding organochlorine-contaminated carp from Saginaw Bay, Lake Huron, to laying White Leghorn hens. II. Embryotoxic and teratogenic effects." *J. of Toxicol. and Environ. Health*, 49:409-438.

Szaro, R.C., G. Hensler, G.H. Heinz. 1981. "Effects of chronic ingestion of No. 2 fuel oil on Mallard ducklings." *J. of Toxicol. and Environ. Health*, 7:789-799.

Tillitt, D.E., G.T. Ankley, J.P. Giesy, J.P. Ludwig, H. Kurita-Matsuba, D.V. Weseloh, P.S. Ross, C.A. Bishop, L Sileo, K.L. Stromborg, J. Larson, T.J. Kubiak. 1992. "Polychlorinated biphenyl residues and egg mortality in double-crested cormorants from the Great Lakes." *Environ. Toxicol. Chem.*, 11:1281-1288.

Tori, G.M. and T.J. Peterle. 1983. "Effects of PCBs on Mourning Dove Courtship Behavior." *Bull. Environ. Contam. Toxicol.* 30:44-49.

Tremblay J. and L.N. Ellison L.N. 1980. "Breeding success of the black-crowned night heron in the St. Lawrence estuary." *Can. J. Zool.* 58:1259-1263.

Ulfstrand, S. and A. Södergren. 1971. Effect of PCB on nocturnal activity in caged robins, *Erithacus rubecula* L. *Nature*. 231:467.

U.S. ACOE (U.S. Army Corps of Engineers). 1988. "Relationship between PCB tissue residues and reproductive success of fathead minnows." Environmental Effects of Dredging - Technical Notes. U.S. Army Corps of Engineers Waterways Experiment Station. Technical Note EEDP-01-13. 9 pp.

U.S. EPA (U.S. Environmental Protection Agency). 1980. *Ambient Water Quality Criteria for Polychlorinated biphenyls*. U.S. Environmental Protection Agency. Rep. 440/5-80-068. 211 pp.

U.S. EPA (U.S. Environmental Protection Agency). 1989. "Risk Assessment Guidance for Superfund. Volume I. Human Health Evaluation Manual (Part A). Interim Final." Office of Emergency and Remedial Response. EPA/540/1-89/002.

U.S. EPA. 1993. "Wildlife Exposure Factors Handbook, Volume I of II." United States Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/R-93/187a.

Walker, M.K. and R.E. Peterson. 1991. "Potencies of Polychlorinated Dibenzo-p-dioxin, Dibenzofuran, and Biphenyl Congeners, Relative to 2,3,7,8-Tetrachlorodibenzo-p-dioxin, for Producing Early Life Stage Mortality in Rainbow Trout (*Oncorhynchus mykiss*)." *Aquat. Toxicol. (Amst.)*, 21:219-238.

Wiemeyer, S.N., T.G. Lamont, C.M. Bunck, C.R. Sindelar, F.J. Gramlich, J.D. Fraser and M.A. Byrd. 1984. "Organochlorine pesticide, polychlorobiphenyl, and mercury residues in bald eagle eggs-1969-79-and their relationships to shell thinning and reproduction." *Arch. Environ. Contam. Toxicol.*, 13:529-549.

Wiemeyer, S.N. 1990. "Organochlorines and mercury residues in bald eagle eggs 1968-1984: Trends and relationships to productivity and shell thickness." In: *Proceedings of the Expert Consultation Meeting on Bald Eagles*. Eds. Best, D.A., M. Gilbertson, and H. Hudson. International Joint Commission. February 12 - 13, 1990.

Wiemeyer, S.N., C.M. Bunck, and C.J. Stafford. 1993. "Environmental contaminants in bald eagle eggs - 1980-84 - and further interpretations of relationships to productivity and shell thickness." *Arch. Environ. Contam. Toxicol.*, 24:13-227.

Wren, C.D., D.B. Hunter, J.F. Leatherland, and P.M. Stokes. 1987a. "The Effects of Polychlorinated Biphenyls and Methylmercury, Singly and in Combination on Mink. I: Uptake and Toxic Responses." *Arch. Environ. Contam. Toxicol.*, 16:441-447.

Wren, C.D., D.B. Hunter, J.F. Leatherland, and P.M. Stokes. 1987b. "The Effects of Polychlorinated Biphenyls and Methylmercury, Singly and in Combination on Mink. II: Reproduction and Kit Development." *Arch. Environ. Contam. Toxicol.*, 16:449-454.

Zabel, E.W., P.M. Cook, and R.E. Peterson. 1995. "Potency of 3,3',4,4',5-Pentachlorobiphenyl (PCB 126), Alone and in Combination with 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD), to Produce Lake Trout Early Life Stage Mortality." *Environ. Toxicol. Chem.*, 14:2175-2179.

APPENDIX B

Life Histories and Exposure Profiles for the Food Chain Models
Upper Green Bay Portion of the Fox River Site
Green Bay, WI
February 2000

APPENDIX B

LIFE HISTORIES AND EXPOSURE PROFILES FOR THE FOOD CHAIN MODELS

B.1 Lake Trout (*Salvelinus namaycush*)

B.1.1 Life History

Lake trout are large, torpedo-shaped fish similar to the brook trout. Their body coloring consists of white spots on a silvery-gray background, shading to white on the belly. Lake trout range over much of the glaciated North America and are usually found near the bottom of well-oxygenated lakes. They usually occur in water about 50 degrees Fahrenheit (Smith 1985). The average lake trout in Lake Michigan weighs 7 pounds (with a range of 3 to 9 pounds) and adults range in length from 17 to 27 inches. Lake trout are long lived and do not reach sexual maturity until 6 to 8 years old (University of Wisconsin 1998).

Lake trout spawn between September and December over rock and rubble. The eggs drop into crevices and are protected from predation in the crevices. Newly hatched lake trout feed on small zooplankton, but as they grow the diet shifts to insects and small fish. The diet of adult lake trout is 100 percent fish and may consist of chubs, sculpin, smelt, and alewife. Madenjian et al. (1998a) also found that the diet of lake trout greater than 600 millimeters total length in both the near shore and off shore waters of Lake Michigan was dominated by alewife.

Habitat deterioration causing lowered dissolved oxygen levels and reduced spawning grounds, over fishing, sea lamprey infestation, and pesticides caused a severe decline in the population of lake trout. Lake trout were once the most valuable commercial fish in the Upper Great Lakes. Lakes Erie and Ontario formerly supported a commercial fishery for this species but the native stock is now considered extinct (Smith 1985). There are many active programs to restock the population, improve habitat, reduce pesticide levels, and control sea lamprey which have increased the size of the population. Currently, there is still a commercial fishing ban for this species (Smith 1985).

B.2 Caspian Tern (*Sterna caspia*)

B.2.1 Life History

Caspian tern (*Sterna caspia*) is one of about fifty species of terns worldwide. It is 19-23" and is the largest tern in North America and the world. It is often mistaken for a gull due to its large size and gull-like characteristics. It is largely white, with a black cap, pale gray back and wings, and a heavy bright red bill and dusky underwing. Its legs and feet are black and has a slightly forked tail. In winter, the adult has white streaks on the crown. Young Caspian terns resemble their parents but they have a mottled plumage and an orange bill. A Caspian terns large sized, thicker bill, and low pitched harsh calls makes them easily distinguishable from other tern species. (WDNR 1998; Cassidy 1990; Bull and Farrand 1977; NRC 1998).

Caspian terns inhabit sandy or pebble shores of lakes and large rivers along seacoasts (Bull and Farrand 1977). The Caspian tern breeds on sea coasts, estuaries, or shores of inland lakes and seas and occasionally on rocky islands. These terns return to their breeding grounds in April, May and June (Richards 1990). They nest in colonies but may join Common tern or Ring-billed Gull colonies and they have been known to nest in isolated pairs (Environment Canada 1999). The eggs are either laid in a shallow depression in the ground or in nests lined with grasses, seaweeds, or mosses (WDNR 1998). Eggs are laid from May to July, two to three at a time and are buff

colored, blotched and spotted with dark brown. The shell lacks gloss and is rough to the touch (Richards 1990). They incubate 20-27 days, and chicks remain near the nest after hatching. The fledgling stage lasts 28-35 days and typically one young fledges from a successful nest (WDNR 1998). Caspian terns of the Great Lakes, disperse along the Atlantic Coast, in fall. They winter on the shores of the Gulf of Mexico and the Caribbean Islands (Environment Canada 1999).

Caspian terns typically plunge dive for small fish but also feed on the surface sometimes eating eggs or young of other birds. Alewives and rainbow smelt are the main prey for Caspian terns in and around the Great Lakes area but they have also been known to take Yellow Perch and Rock Bass (WDNR 1998; NRC 1998).

B.2.2 Exposure Profile for the Food Chain Model

The body weight of the Caspian tern ranges from 574 to 782 g (Dunning 1993). Data on food ingestion rates were not available, so the food ingestion rate for the Caspian tern was calculated using an allometric equation for food ingestion for birds (Nagy 1987). The food ingestion rate (FI) was calculated as $0.648 * (\text{weight in grams})^{0.651}$ (U.S. EPA 1993). Using the lowest reported body weight of 574 g, a food ingestion rate of 40.5 g/day was calculated.

No data for water ingestion rates were available. Therefore, an allometric equation was used to calculate the water ingestion rate (WI) for Caspian terns as well. The rate was calculated in liters per day as $0.059 * (\text{weight in kilograms})^{0.67}$ (U.S. EPA 1993). Using the lowest reported body weight of 574 g (0.574 kg), the water ingestion rate was determined to be 0.04 L/day (Nagy 1987).

An incidental sediment ingestion rate could not be located for Caspian tern. However, due to the open water feeding habits of this bird, it was assumed that the Caspian tern does not ingest sediment directly. Based on the probable feeding habits of Caspian tern prey items, it is also unlikely that the birds ingest sediment indirectly through their prey items.

A feeding radius for the Caspian tern could not be located in the literature. However, given the large size of the upper Green Bay, it was assumed that a Caspian tern could obtain 100 percent of its food from the upper Green Bay. Therefore, an area use factor of one will be assumed for this receptor.

Since Caspian terns consume fish, and given the habitat of the upper Green Bay, it will be assumed that 100 percent of the diet of the Caspian tern is comprised of fish for the purposes of the food chain model in this study.

B.3 Double-crested cormorant (*Phalacrocorax auritus*)

B.3.1 Life History

Cormorant is the common name for any of several web-footed water birds of the family Phalacrocoracidae, in the order Pelecaniformes. These fish-eating birds nest in colonies on the seacoasts of temperate and tropical regions of the world. A few species also live on large island lakes and rivers. They have slender, hooked beaks, long flexible necks, a patch of bare skin under the mouth, and a stiff tail. Their plumage is usually a glossy black, but some have white areas and many have brightly colored featherless rings around the eyes. They dive and swim deeply underwater in pursuit of fish (Environmental Advocates 1998).

The most widely distributed North American species is the double-crested cormorant, *P. auritus*, of both the Atlantic and Pacific coasts; it is the only species likely to be seen in the interior of the

continent (Environmental Advocates 1998). The double-crested cormorant is a black duck-like bird with an orange beak with a hook at the tip and orange at the jowls or cheeks. When paddling, the beak is held angled higher than parallel to water. It has an expandable throat pouch that is orange colored, and its wing span is four feet (Environmental Advocates 1998). The length of the body ranges from 74 to 89 cm. Tufts of narrow and curved black feathers found on its head during breeding season are referred to in the bird's name. Immature birds are more gray and brown (Nova Scotia Museum of Natural History 1998).

The double-crested cormorant breeds from southwestern Alaska and the interior of North America to the Gulf of St. Lawrence and southern Newfoundland, south to the southern United States and the Bahamas. Most of the birds in Atlantic Canada breed in the western Gulf of St. Lawrence and on the Atlantic coast of mainland Nova Scotia. The bird winters from the southern parts of its summer range south to Florida and the Gulf of Mexico (Nova Scotia Museum of Natural History 1998). In some of the mid-western United States cormorants are listed as endangered or threatened (SCCF 1995).

Double-crested cormorants nest in both salt and fresh water areas. In the south they nest in trees and in the north they nest in rocky ledge areas. They can be found nesting among the heron rookeries (SCCF 1995). Their nests are made from seaweed and other coarse vegetable matter placed on a rude foundation of small sticks. They usually nest in colonies, but sometimes in smaller groups, and the sites commonly chosen are of three types: on projecting shelves on the sides of steep cliffs; on level surfaces above the sea wall and preferably near its edge; and in trees 2-10 m or more in height. The trees chosen are usually on islands with low shores without cliffs and quickly die from exposure to the cormorants' excreta. The double-crested cormorant lays from three to six eggs (usually 4 to 5). The eggs are bluish white with an overlay of a chalk-like substance (Nova Scotia Museum of Natural History 1998). Both mother and father share in the child care. Babies are blind and helpless at the time of hatching. The young eat semi-digested foods from the parent's beak. Fledging occurs at 8 weeks (SCCF 1995).

The double-crested cormorant eats almost entirely fish and for the most part species of fish not important to commercial fisheries. They chase fish underwater using both their powerful webbed feet and their wings in a sort of breast stroke to propel them through the water. Cormorants appear clumsy trying to get airborne after feeding. They generally always leave the water faced into the wind and use their feet to help them build speed for take off. Cormorants often sit on posts or wires to dry out with their wings outstretched (SCCF 1995).

B.3.2 Exposure Profile for the Food Chain Model

The body weight of an adult double-crested cormorant has been reported to be 1.9 kg (Environment Canada 1996). The double-crested cormorant has been estimated to consume approximately 25% of its body weight in fish per day (Environment Canada 1996), which equates to 0.475 kg/day. A water ingestion rate of 0.079 L/day was calculated using an allometric equation for water ingestion for birds (Nagy 1987).

An incidental sediment ingestion rate could not be located for the double-crested cormorant. However, due to the open water feeding habits of this bird, it was assumed that the double-crested cormorant does not incidentally ingest sediment.

A feeding radius for the double-crested cormorant could not be located in the literature. However, given the large size of the upper Green Bay, it was assumed that the double-crested cormorant could obtain 100 percent of its food from the upper Green Bay. Therefore, an area use factor of one will be assumed for this receptor.

Since double-crested cormorants are primarily piscivorous, it will be assumed that 100 percent of the diet of the double-crested cormorant is comprised of fish for the purposes of the food chain model in this study.

B.4 Mink (*Mustela vison*)

B.4.1 Life History

Mink are distributed over much of boreal North America, southward throughout the eastern United States and in the west to California, New Mexico, and Texas (Jones and Birney 1988). They are brown, weasel-like animals that can be found in virtually any habitat containing permanent water and are not commonly found in upland areas (Jones and Birney 1988). Although primarily nocturnal, their activity often extends into midday (Hoffmeister 1989).

Dens are always near water, and they are usually an old muskrat burrow or constructed by the mink itself (Jones and Birney 1988). Males tend to live in their own burrows which are less elaborate than ones occupied by females (Barbour and Davis 1974). Home ranges tend to be linear since mink often follow a shoreline (Jones and Birney 1988). Mink are solitary and mark their territories by spraying (Merritt 1987).

Seasonal food availability governs the dietary composition (Barbour and Davis 1974). Their diets may consist of crayfish, frogs, fish, snakes, rodents, rabbits, and plants among other items (Jones and Birney 1988; Schwartz and Schwartz 1981). Crayfish are a major portion of the summer diet in many regions of North America (Barbour and Davis 1974; Jones and Birney 1988; Merritt 1987).

Breeding occurs from January to early April with highly variable gestation periods ranging from 40 to 75 days (Merritt 1987; Schwartz and Schwartz 1981). A highly variable single litter of 1 to 17 young may be produced (Schwartz and Schwartz 1981). Average litter sizes vary among regions (Barbour and Davis 1974; Hoffmeister 1989; Jones and Birney 1988; Merritt 1987; Schwartz and Schwartz 1981). Young are weaned at about five to six weeks of age and are sexually mature by ten months (Merritt 1987; Schwartz and Schwartz 1981). Occasionally great horned owls, foxes, coyotes, bobcats, and dogs will prey on mink (Merritt 1987; Schwartz and Schwartz 1981). Although some individuals have lived up to six years, mink seldom exceed two years of age in the wild (Schwartz and Schwartz 1981).

B.4.2 Exposure Profile for the Food Chain Model

Adult mink weigh from 520 to 1,730 g (Merritt 1987; U.S. EPA 1993). Home ranges vary from 19 to 1,900 acres (U.S. EPA 1993).

A year-round food ingestion rate of 0.22 g/g BW/day has been estimated for both male and female mink (U.S. EPA 1993). To express this value in units of g/day, the food ingestion rate was multiplied by the lowest reported body weight (520 g) to yield a food ingestion rate of 114 g/day.

An estimated water ingestion rate of 0.11 g/g BW/day was reported for farm-raised females (U.S. EPA 1993). To express this value in units of g/day, this water ingestion rate was multiplied by the lowest reported body weight of 520 g to yield a water ingestion rate of 57.2 g/day (57.2 ml/day).

An incidental soil or sediment ingestion rate was not available from the literature for the mink. Therefore, an incidental soil or sediment ingestion rate for another mammalian species with similar feeding habits will be used to represent the incidental sediment ingestion rate for a mink. The raccoon was selected as a mammal with similar feeding habits as the mink because both species are

omnivorous, opportunistic feeders and will consume mammals, but also hunt aquatic prey such as fish, crayfish, and amphibians (U.S. EPA 1993). Beyer et al. (1994) reported a soil ingestion rate of 9 percent of the diet for raccoons. Therefore, it will be assumed for the purposes of this risk assessment that the sediment ingestion rate of the mink is also 9 percent of the diet. Using a food ingestion rate of 114 g/day, the incidental sediment ingestion rate is calculated to be 10.3 g/day for a mink.

REFERENCES

- Barbour, R.W. and W.H. Davis. 1974. *Mammals of Kentucky*. Lexington, KY: University Press. 322 p.
- Beyer, W.N., E.E. Conner, and S. Gerould. 1994. "Estimates of Soil Ingestion by Wildlife." *J. Wildl. Manage.*, 58(2):375-382.
- Bull, J. and J. Farrand, Jr. 1977. *The Audubon Society Field Guide to North American Birds-Eastern Region*. New York: Alfred A. Knopf, Inc. p. 385-386.
- Dunning, J.B., Jr. 1993. *CRC Handbook of Avian Body Masses*. Boca Raton, FL: CRC Press. p. 69.
- Environment Canada. 1996. "The Rise of the Double-crested Cormorant on the Great Lakes." Available at <http://www.cciw.ca/glimr/data/cormorant-fact-sheet.htm> .
- Environment Canada. 1999. "Threatened bird species in Quebec: trends and distribution." Available at: http://www.qc.ec.gc.ca/faune/oiseaux_de_mer/html/Epp640.html.
- Environmental Advocates. 1998. "Cormorant." Available at <http://www.cnet.windsor.ns.ca/Environment/Advocates/Anim> .
- Hoffmeister, D.F. 1989. *Mammals of Illinois*. Urbana, IL: University of Illinois Press. 348 p.
- Jones, Jr., J.K.J. and E.C. Birney. 1988. *Handbook of Mammals of the North Central States*. Minneapolis, MN: University of Minnesota Press. 346 p.
- Madenjian, C.P., T.J. DeSorcie, and R.M. Stedman. 1998a. "Ontogenic and Spatial Patterns in Diet and Growth of Lake Trout in Michigan." *Trans. Amer. Fish. Soc.*, 127(2):236-252.
- Madenjian, C.P., R.J. Hesselberg, T.J. DeSorcie, L.J. Schmidt, R.M. Stedman, R.T. Quintal, L.J. Begnoche, D.R. Passino-Reader. 1998b. "Estimate of Net Trophic Transfer Efficiency of PCBs to Lake Michigan Lake Trout from their Prey." *Env. Sci. Tech.*, 32(7) 886-891.
- Merritt, J.F. 1987. *Guide to the Mammals of Pennsylvania*. Pittsburgh, PA: University of Pittsburgh Press. 408 p.
- Nagy, K.A. 1987. "Field Metabolic Rate and Food Requirement Scaling in Mammals and Birds." *Ecol. Mongr.* 57:111-128.
- National Geographic Society. 1987. *Field Guide to the Birds of North America, Second Edition*. Washington, DC: National Geographic Society. p. 164.
- NRC (Natural Resources Canada). 1998. "Caspian Tern (*Sterna caspia*)." Available at: <http://www-nais.ccm.emr.ca/schoolnet/issues/risk/birds/ebirds/casptrn.html>.
- Nova Scotia Museum of Natural History. 1998. "Double-crested Cormorant." Available at <http://www.museum.ednet.ns.ca/mnh/nature/nsbirds/bns0028.htm> .
- Peterson, R.T. 1980. *A Field Guide to the Birds: A Completely New Guide to All the Birds of Eastern and Central North America*. 4th ed.. Boston, MA: Houghton Mifflin Company. 384 p.

Richards, A. 1990. *Seabirds of the Northern Hemisphere*. New York, NY: Gallery Books. p. 133-136.

Sanibel-Captiva Conservation Foundation (SCCF). 1995. "Double Crested Cormorant *Phalacrocorax auritus*." Available at <http://www.naples.com/sanibel/birdcorm.htm> .

Schwartz, C.W. and E.R. Schwartz. 1981. *The Wild Mammals of Missouri, Revised Edition*. Columbia, MO: University of Missouri Press and Missouri Dept. Conserv. 356 p.

Smith, C. Lavett. 1985. *The Inland Fishes of New York State*. The New York State Department of Environmental Conservation, Albany, NY.

University of Wisconsin. 1998. "Fish of the Great Lakes/lake Trout." Sea Grant Institute Web Site. Available at <http://www.seagrant.wisc.edu/communications/publications/fish/laketrout1.htm>.

U.S. EPA. 1993. "Wildlife Exposure Factors Handbook, Volume I of II." United States Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/R-93/187a.

WDNR (Wisconsin Department of Natural Resources). 1998. "Caspian Tern (*Sterna caspia*)." Available at: <http://www.dnr.state.wi.us/org/land/er/birds/caster.htm>.

APPENDIX C

PCBs in Walleye from Green Bay and Tributaries
Wisconsin Department of Natural Resources Data, December 1999
Upper Green Bay Portion of the Fox River Site
Green Bay, WI
February 2000

FISH RESULTS BY SITE NAME - COLUMN STYLE
PCBS IN WALLEYE FROM CREEK BAY AND TRIBUTARIES

----- SITE=POK RIVER BELOW DEPERE LOCATION CCDE=055008 COUNTY=BROWN -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7705	23 20E 15	05/06/1977	WALLEYE	SKIN ON FILLET	1	15.00		4.5 - UG/G
7714	23 20E 15	05/06/1977	WALLEYE	SKIN ON FILLET	1	17.80		6.8 - UG/G
8304	23 20E 15	06/13/1983	WALLEYE	WHOLE FISH	2	19.70	1.30	16. - UG/G
8405	23 20E 15	01/01/1984	WALLEYE	SKIN ON FILLET	3	15.80	0.40	8.1 - UG/G
8406	23 20E 15	01/01/1984	WALLEYE	SKIN ON FILLET	3	16.57	0.63	2.4 - UG/G
8601	23 20E 15	10/06/1986	WALLEYE	WHOLE FISH	3	17.00	0.78	12. - UG/G
8603	23 20E 15	10/06/1986	WALLEYE	SKIN ON FILLET	1	19.00	1.06	2.1 - UG/G
8604	23 20E 15	10/06/1986	WALLEYE	SKIN ON FILLET	1	21.00	1.45	2.1 - UG/G
8605	23 20E 15	10/06/1986	WALLEYE	SKIN ON FILLET	1	20.50	2.39	1.6 - UG/G
8727	23 20E 15	05/22/1987	WALLEYE	SKIN ON FILLET	1	15.50	0.65	0.88 - UG/G
8728	23 20E 15	05/22/1987	WALLEYE	SKIN ON FILLET	2	13.00	0.34	0.76 - UG/G
8729	23 20E 15	05/22/1987	WALLEYE	SKIN ON FILLET	3	8.60	0.08	0.51 - UG/G
8848	23 20E 15	04/28/1988	WALLEYE	SKIN ON FILLET	1	19.88	1.29	1.2 - UG/G
8849	23 20E 15	04/28/1988	WALLEYE	SKIN ON FILLET	1	21.88	1.57	1.9 - UG/G
8850	23 20E 15	04/28/1988	WALLEYE	SKIN ON FILLET	1	22.88	2.14	2.3 - UG/G
8906	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	14.00		2.0 - UG/G
8907	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	16.60		1.25 - UG/G
8908	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	16.70		1.47 - UG/G
8909	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	17.30		1.56 - UG/G
8910	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	17.60		1.53 - UG/G
8911	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	17.80		0.8 - UG/G
8912	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	18.00		2.04 - UG/G
8913	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	19.00		1.5 - UG/G
8914	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	19.80		1.58 - UG/G
8915	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	24.10		1.7 - UG/G
8916	23 20E 15	10/25/1989	WALLEYE	SKIN ON FILLET	5	21.50		1.63 - UG/G
9232	23 20E 15	04/13/1992	WALLEYE	SKIN ON FILLET	1	18.30	0.16	0.26 - UG/G
9233	23 20E 15	04/18/1992	WALLEYE	SKIN ON FILLET	1	19.50	0.16	0.25 - UG/G
9234	23 20E 15	04/23/1992	WALLEYE	SKIN ON FILLET	1	13.20	0.33	0.75 - UG/G
9235	23 20E 15	04/23/1992	WALLEYE	SKIN ON FILLET	1	13.20	0.35	0.78 - UG/G
9236	23 20E 15	04/23/1992	WALLEYE	SKIN ON FILLET	1	14.50	0.41	1.1 - UG/G
9237	23 20E 15	04/23/1992	WALLEYE	SKIN ON FILLET	1	14.50	0.45	0.71 - UG/G
9238	23 20E 15	04/23/1992	WALLEYE	SKIN ON FILLET	1	15.00	0.47	0.47 - UG/G
9239	23 20E 15	04/23/1992	WALLEYE	SKIN ON FILLET	1	15.80	0.57	0.88 - UG/G
9240	23 20E 15	04/23/1992	WALLEYE	SKIN ON FILLET	1	17.00	0.84	0.35 - UG/G
9241	23 20E 15	04/23/1992	WALLEYE	SKIN ON FILLET	1	17.50	0.82	0.2 - UG/G
9242	23 20E 15	03/29/1992	WALLEYE	SKIN ON FILLET	1	17.80	0.93	1.1 - UG/G
9243	23 20E 15	04/23/1992	WALLEYE	SKIN ON FILLET	1	13.20	1.01	0.2 - UG/G
9244	23 20E 15	03/29/1992	WALLEYE	SKIN ON FILLET	1	13.50	1.39	4.6 - UG/G
9245	23 20E 15	03/29/1992	WALLEYE	SKIN ON FILLET	1	13.80	1.74	1. - UG/G
9246	23 20E 15	03/29/1992	WALLEYE	SKIN ON FILLET	1	20.20	1.48	2.3 - UG/G
9247	23 20E 15	03/29/1992	WALLEYE	SKIN ON FILLET	1	21.50	1.85	2.6 - UG/G
9248	23 20E 15	03/29/1992	WALLEYE	SKIN ON FILLET	1	21.50	1.84	3.4 - UG/G
9249	23 20E 15	03/29/1992	WALLEYE	SKIN ON FILLET	1	23.00	2.22	3.8 - UG/G
9250	23 20E 15	03/29/1992	WALLEYE	SKIN ON FILLET	1	23.50	2.30	1.7 - UG/G
9251	23 20E 15	03/29/1992	WALLEYE	SKIN ON FILLET	1	24.00	2.44	2.7 - UG/G
9401	23 20E 15	10/06/1994	WALLEYE	SKIN ON FILLET	3	16.10	0.61	0.78 - UG/G
9645	23 20E 15	08/20/1996	WALLEYE	SKIN ON FILLET	1	15.70		0.45 - UG/G

FISH / SEDIMENT CONTAMINANTS SYSTEM JOB ID: 657

10:03 Monday, December 27, 1999

FISH RESULTS BY SITE NAME - COLUMB STYLE

PCBS IN WALLEYE FROM GREEN BAY AND TRIBUTARIES

----- SITE-FOX RIVER BELOW DEPREE LOCATION CODE=015008 COUNTY=BRONX -----
 (continued)

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
9646	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	16.60	0.12	1.6 - MG/KG
9647	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	15.90	0.14	1.4 - MG/KG
9648	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	16.50	0.15	0.85 - MG/KG
9649	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	17.20	0.15	2.7 - MG/KG
9650	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	15.90		1.0 - MG/KG
9651	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	16.90	0.15	0.83 - MG/KG
9652	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	15.70		0.93 - MG/KG
9653	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	18.00	0.16	2.9 - MG/KG
9654	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	15.30		0.85 - MG/KG
9655	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	16.00	0.12	0.95 - MG/KG
9656	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	16.50	0.69	0.95 - MG/KG
9657	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	14.80	0.50	0.75 - MG/KG
9658	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	15.50	0.56	0.73 - MG/KG
9659	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	15.50		0.67 - MG/KG
9660	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	15.00		0.75 - MG/KG
9661	23 20C 15	08/20/1956	WALLEYE	SKIN ON FILLET	1	21.00		1.5 - MG/KG
9662	23 20C 15	08/20/1956	WALLEYE	WHOLE FISH	1	16.00		8. - MG/KG
9663	23 20C 15	08/20/1956	WALLEYE	WHOLE FISH	1	16.60	0.53	8.1 - MG/KG
9664	23 20C 15	08/20/1956	WALLEYE	WHOLE FISH	1	18.00	0.86	18. - MG/KG
9665	23 20C 15	08/20/1956	WALLEYE	WHOLE FISH	1	16.90	0.73	7.5 - MG/KG
9666	23 20C 15	08/20/1956	WALLEYE	WHOLE FISH	1	15.70	0.65	7.1 - MG/KG
9667	23 20C 15	08/20/1956	WALLEYE	WHOLE FISH	1	15.90	0.65	14. - MG/KG
9668	23 20C 15	08/20/1956	WALLEYE	WHOLE FISH	1	16.50	0.71	7. - MG/KG
9669	23 20C 15	08/20/1956	WALLEYE	WHOLE FISH	1	17.20	0.75	11. - MG/KG
9670	23 20C 15	04/15/1956	WALLEYE	SKIN ON FILLET	1	20.00		0.96 - MG/KG
9671	23 20C 15	04/15/1956	WALLEYE	SKIN ON FILLET	1	21.00		1.5 - MG/KG
9672	23 20C 15	05/03/1956	WALLEYE	SKIN ON FILLET	1	21.00		1.6 - MG/KG
9673	23 20C 15	04/29/1956	WALLEYE	SKIN ON FILLET	1	22.40		1.4 - MG/KG
9674	23 20C 15	04/29/1956	WALLEYE	SKIN ON FILLET	1	23.90		4. - MG/KG
9675	23 20C 15	04/15/1956	WALLEYE	SKIN ON FILLET	1	20.50		2. - MG/KG
9676	23 20C 15	04/15/1956	WALLEYE	SKIN ON FILLET	1	20.50		1.1 - MG/KG
9837	23 20C 15	04/20/1958	WALLEYE	SKIN ON FILLET	1	11.50	0.20	0.19 - UG/G
9838	23 20C 15	04/20/1958	WALLEYE	SKIN ON FILLET	1	11.75	0.22	0.15 - UG/G
9839	23 20C 15	04/22/1958	WALLEYE	SKIN ON FILLET	1	12.75	0.27	0.15 - UG/G
9840	23 20C 15	04/22/1958	WALLEYE	SKIN ON FILLET	1	12.83	0.29	0.21 - UG/G
9841	23 20C 15	04/17/1958	WALLEYE	SKIN ON FILLET	1	13.18	0.31	0.11 - UG/G
9842	23 20C 15	04/22/1958	WALLEYE	SKIN ON FILLET	1	15.63	0.54	0.36 - UG/G
9843	23 20C 15	04/23/1958	WALLEYE	SKIN ON FILLET	1	17.75	0.76	1.1 - UG/G
9844	23 20C 15	04/23/1958	WALLEYE	SKIN ON FILLET	1	17.83	0.76	1.4 - UG/G
9845	23 20C 15	07/08/1958	WALLEYE	SKIN ON FILLET	1	18.25	1.11	0.54 - UG/G
9850	23 20C 15	04/17/1958	WALLEYE	SKIN ON FILLET	1	22.00	1.69	1.2 - UG/G
9851	23 20C 15	04/17/1958	WALLEYE	SKIN ON FILLET	1	22.50	1.63	1.8 - UG/G
9852	23 20C 15	04/17/1958	WALLEYE	SKIN ON FILLET	1	24.25	2.53	0.79 - UG/G

FISH / SEDIMENT CONTAMINANTS SYSTEM JOB ID: 657
 FISH RESULTS BY SITE NAME - COLUMB' STIAS
 PCBs IN WALLEYE FROM GREEN BAY AND TRIBUTARIES

10:03 Monday, December 27, 1999

SITE-FOX RIVER DEPERE FAIRGROUNDS LOCATION CODE=055016 COUNTY-BROWN

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7805	23 201 22	04/11/1978	WALLEYE	WHOLE FISH	5	18.00	25.	- UG/G
7901	23 201 22	04/04/1979	WALLEYE	WHOLE FISH	2	15.80	16.	- UG/G
7904	23 201 22	04/04/1979	WALLEYE	SKIN ON FILLET	1	15.20	3.7	- UG/G
7905	23 201 22	04/04/1979	WALLEYE	SKIN ON FILLET	1	17.10	3.3	- UG/G
7906	23 201 22	04/04/1979	WALLEYE	SKIN ON FILLET	3	11.40	3.3	- UG/G
7907	23 201 22	04/04/1979	WALLEYE	SKIN ON FILLET	6	9.40	1.5	- UG/G
7908	23 201 22	04/04/1979	WALLEYE	SKIN ON FILLET	2	12.00	3.2	- UG/G
8001	23 201 22	10/02/1980	WALLEYE	WHOLE FISH	5	15.10	0.40	9.1 - UG/G
8101	23 201 22	03/13/1981	WALLEYE	WHOLE FISH	5	19.20	1.10	10.7 - UG/G
8102	23 201 22	03/13/1981	WALLEYE	WHOLE FISH	4	20.40	1.60	13.7 - UG/G
8104	23 201 22	09/28/1981	WALLEYE	WHOLE FISH	5	17.00	0.70	22. - UG/G
8104	23 201 22	09/28/1981	WALLEYE	WHOLE FISH	5	17.30	0.70	13. - UG/G
8201	23 201 22	08/03/1982	WALLEYE	WHOLE FISH	5	16.20	0.67	16.3 - UG/G
8501	23 203 22	08/31/1985	WALLEYE	SKIN ON FILLET	1	14.50	0.50	1.6 - UG/G
8502	23 203 22	08/31/1985	WALLEYE	SKIN ON FILLET	1	15.00	0.46	2. - UG/G
8503	23 203 22	08/31/1985	WALLEYE	SKIN ON FILLET	1	15.75	0.58	1.6 - UG/G
8504	23 203 22	08/31/1985	WALLEYE	SKIN ON FILLET	1	17.25	0.91	2.9 - UG/G
8505	23 203 22	08/31/1985	WALLEYE	SKIN ON FILLET	1	18.00	0.96	1.2 - UG/G
8506	23 203 22	08/31/1985	WALLEYE	SKIN ON FILLET	1	19.50	1.17	1.2 - UG/G
8508	23 203 22	08/31/1985	WALLEYE	LIVER	4	18.00	12.	- UG/G
8701	23 203 22	05/11/1987	WALLEYE	SKIN ON FILLET	2	9.90	0.13	0.47 - UG/G

SITE-FOX RIVER HIGHWAY 172 BRIDGE LOCATION CODE=055012 COUNTY-BROWN

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8703	23 201 14	05/11/1987	WALLEYE	SKIN ON FILLET	2	12.23	0.26	0.59 - UG/G
8704	23 201 14	05/11/1987	WALLEYE	SKIN ON FILLET	1	13.50	0.36	0.4 - UG/G
8705	23 201 14	05/11/1987	WALLEYE	SKIN ON FILLET	1	15.30	0.54	1.7 - UG/G

SITE-FOX RIVER HIGHWAY 29 LOCATION CODE=055006 COUNTY-BROWN

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8702	23 201 01	09/30/1980	WALLEYE	WHOLE FISH	5	13.5	0.45	15. - UG/G

SITE-FOX RIVER HIGHWAY 143 BRIDGE LOCATION CODE=055014 COUNTY-BROWN

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
873B	24 201 01	05/11/1987	WALLEYE	SKIN ON FILLET	1	19.3	1.14	2.3 - UG/G
873C	24 201 01	05/11/1987	WALLEYE	SKIN ON FILLET	1	20.5	1.41	3.1 - UG/G

FISH / SEDIMENT CONTAMINANTS SYSTEM JOB ID: 657

10:03 Monday, December 27, 1999

FISH RESULTS BY SITE NAME - COLUMN STYLE
PCBS IN WALLEYE FROM GREEN BAY AND TRIBUTARIES

----- SITE-FOX RIVER JONES POINT LOCATION CODE=055007 COUNTY-BROWN -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8002	23 208 14	10/02/1980	WALLEYE	WHOLE FISH	4	9.3	0.11	6. - UG/G

----- SITE-FOX RIVER MOULT LOCATION CODE=055004 COUNTY-BROWN -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7906	24 208 24	10/17/1979	WALLEYE	WHOLE FISH	5	15.0	0.70	6. - UG/G
8202	24 208 24	09/23/1982	WALLEYE	WHOLE FISH	4	17.1	0.71	19.8 - UG/G

----- SITE-FOX RIVER RAILROAD AT FORT HOWARD LOCATION CODE=055015 COUNTY-BROWN -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8702	24 218 11	05/11/1987	WALLEYE	SKIN ON FILLET	1	21.3	1.25	2.3 - UG/G
8706	24 218 11	05/11/1987	WALLEYE	SKIN ON FILLET	1	22.3	2.16	1.9 - UG/G

----- SITE-GREEN BAY GRID 1001 LOCATION CODE=055011 COUNTY-BROWN -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8904	24 218 11	04/21/1989	WALLEYE	SKIN ON FILLET	5	19.2	1.17	2.383 - UG/G
8905	24 218 11	04/21/1989	WALLEYE	SKIN ON FILLET	5	17.2	0.85	1.96 - UG/G
8906	24 218 11	04/24/1989	WALLEYE	SKIN ON FILLET	5	17.6	0.88	1.32 - UG/G
8907	24 218 11	08/27/1989	WALLEYE	SKIN ON FILLET	5	18.0	1.01	1.48 - UG/G
8908	24 218 11	09/13/1989	WALLEYE	SKIN ON FILLET	5	21.1	1.66	1.56 - UG/G
8909	24 218 11	09/13/1989	WALLEYE	SKIN ON FILLET	5	21.1	1.76	2.059 - UG/G
8910	24 218 11	08/24/1989	WALLEYE	SKIN ON FILLET	5	18.4	1.06	1.3 - UG/G
8911	24 218 11	08/21/1989	WALLEYE	SKIN ON FILLET	5	20.0	1.35	1.44 - UG/G
8912	24 218 11	08/24/1989	WALLEYE	SKIN ON FILLET	5	18.4	1.07	1.285 - UG/G
8913	24 218 11	08/24/1989	WALLEYE	SKIN ON FILLET	5	19.9	1.37	2.064 - UG/G
8914	24 218 11	08/24/1989	WALLEYE	SKIN ON FILLET	5	18.0	0.95	1.499 - UG/G
8915	24 218 11	11/13/1989	WALLEYE	SKIN ON FILLET	5	20.8	1.74	0.971 - UG/G
8916	24 218 11	09/28/1989	WALLEYE	SKIN ON FILLET	5	18.0	1.22	0.055 - UG/G
8911	24 218 11	04/24/1989	WALLEYE	SKIN ON FILLET	5	16.8	0.78	1.457 - UG/G
8912	24 218 11	05/01/1989	WALLEYE	SKIN ON FILLET	5	18.1	0.97	1.492 - UG/G
8913	24 218 11	05/01/1989	WALLEYE	SKIN ON FILLET	5	18.6	1.05	1.14 - UG/G
8914	24 218 11	11/01/1989	WALLEYE	SKIN ON FILLET	5	21.3	1.88	1.664 - UG/G

FISH RESULTS BY SITE NAME - COLUMN STYLE
PCBS IN WALL-EYE FROM GREEN BAY AND TRIBUTARIES

----- SITE-GREEN BAY GRID 605 LOCATION CODE=155014 COUNTY=DOOR -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
3906		06/13/1989	WALLEYE	SKIN ON FILLET	5	21.1	1.89	1.22 - UG/G
3907		06/13/1989	WALLEYE	SKIN ON FILLET	5	20.0	1.61	1.25 - UG/G
3908		06/13/1989	WALLEYE	SKIN ON FILLET	5	21.4	1.94	1.456 - UG/G
3909		07/21/1989	WALLEYE	SKIN ON FILLET	4	20.3	1.76	1.37 - UG/G
3910		09/23/1989	WALLEYE	SKIN ON FILLET	5	21.1	1.75	9.884 - UG/G
3911		07/19/1989	WALLEYE	SKIN ON FILLET	5	21.7	1.90	1.51 - UG/G

----- SITE-GREEN BAY GRID 703 LOCATION CODE=155030 COUNTY=DOOR -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8919	30 21E 09	11/10/1989	WALLEYE	SKIN ON FILLET	5	21.3	1.82	1.072 - UG/G
8920	30 21E 09	09/11/1989	WALLEYE	SKIN ON FILLET	4	20.4	1.57	0.5E - UG/G
8921	30 21E 09	11/09/1989	WALLEYE	SKIN ON FILLET	5	21.2	1.61	1.427 - UG/G
8922	30 21E 09	08/31/1989	WALLEYE	SKIN ON FILLET	5	20.7	1.66	0.789 - UG/G

----- SITE-GREEN BAY GRID 802 LOCATION CODE=435011 COUNTY=COONTO -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8995	20 21E 07	06/19/1989	WALLEYE	SKIN ON FILLET	3	19.9	1.31	1.02 - UG/G

----- SITE-GREEN BAY GRID 803 LOCATION CODE=155008 COUNTY=DOOR -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7601	28 253 30	06/23/1976	WALLEYE	SKIN ON FILLET	5	12.8		0.7 - UG/G
7602	28 253 30	06/23/1976	WALLEYE	SKIN ON FILLET	5	14.8		0.6 - UG/G
7603	28 253 30	06/23/1976	WALLEYE	SKIN ON FILLET	5	16.5		0.5 - UG/G
8005	28 253 30	07/27/1980	WALLEYE	SKIN ON FILLET	7	15.9	0.95	8.1 - UG/G
8101	28 253 30	05/20/1981	WALLEYE	SKIN ON FILLET	1	18.3	1.01	3.4 - UG/G
8102	28 253 30	05/20/1981	WALLEYE	SKIN ON FILLET	1	19.5	1.35	2.3 - UG/G
8103	28 253 30	05/20/1981	WALLEYE	SKIN ON FILLET	1	21.3	1.71	4.2 - UG/G
8104	28 253 30	05/20/1981	WALLEYE	SKIN ON FILLET	1	21.5	1.93	2.5 - UG/G
8105	28 253 30	05/20/1981	WALLEYE	SKIN ON FILLET	1	21.8	1.80	2.3 - UG/G
8106	28 253 30	05/20/1981	WALLEYE	SKIN ON FILLET	1	23.4	2.20	4. - UG/G
8107	28 253 30	05/20/1981	WALLEYE	ABDOMINAL FAT	1	25.6	2.90	94. - UG/G
8108	28 253 30	05/20/1981	WALLEYE	SKIN ON FILLET	1	25.8	2.90	5.1 - UG/G
8601	28 253 30	05/28/1986	WALLEYE	SKIN ON FILLET	1	17.5	0.88	0.92 - UG/G
8602	28 253 30	05/28/1986	WALLEYE	SKIN ON FILLET	1	22.3	1.85	2.3 - UG/G
8603	28 253 30	05/28/1986	WALLEYE	SKIN ON FILLET	1	24.5	1.04	4.7 - UG/G
8901	28 253 30	10/23/1989	WALLEYE	SKIN ON FILLET	5	17.7	1.02	0.856 - UG/G

FISH / SEDIMENT CONTAMINANTS SYS. JOB ID: 657
 FISH RESULTS BY SITE NAME - COLUMB SITE
 PCBs IN WALLEYE FROM GREEN BAY AND TRIBUTARIES

10:03 Monday, December 27, 1999

----- SITE-GREEN BAY GRID #03 LOCATION CODE=155008 COUNTY=DOOR -----
 (continued)

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8902	28 258 30	10/22/1989	WALLEYE	SKIN ON FILLET	5	19.2	1.43	0.822 - WG/G
8903	28 258 30	10/21/1989	WALLEYE	SKIN ON FILLET	5	19.7	1.48	1.37 - WG/G
8906	28 253 30	10/21/1989	WALLEYE	SKIN ON FILLET	5	19.4	1.35	1.304 WG/G

----- SITE-GREEN BAY GRID #04 LOCATION CODE=155005 COUNTY=DOOR -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7901	28 253 25	04/30/1979	WALLEYE	WHOLE FISH	5	17.1		6.3 - WG/G
7903	28 253 25	05/30/1979	WALLEYE	WHOLE FISH	5	17.9	1.10	7.5 - WG/G
8901	28 253 25	05/35/1989	WALLEYE	SKIN ON FILLET	4	15.9	0.70	0.802 - WG/G
8903	28 253 25	05/32/1989	WALLEYE	SKIN ON FILLET	5	18.7	1.11	1.11 - WG/G
8904	28 253 25	05/32/1989	WALLEYE	SKIN ON FILLET	5	17.6	0.92	0.924 - WG/G
8905	28 253 25	07/12/1989	WALLEYE	SKIN ON FILLET	5	17.2	0.89	0.593 - WG/G
8909	28 253 25	08/22/1989	WALLEYE	SKIN ON FILLET	5	18.3	1.09	1.227 - WG/G

----- SITE-GREEN BAY LITTLE STURGEON BAY LOCATION CODE=155004 COUNTY=DOOR -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8301	27 248 11	05/19/1983	WALLEYE	SKIN ON FILLET	5	15.3	0.58	0.92 - OG/G
8302	27 248 11	05/19/1983	WALLEYE	SKIN ON FILLET	5	22.6	2.10	4. - OG/G
8405	27 248 11	05/30/1984	WALLEYE	SKIN ON FILLET	5	15.5	0.70	0.84 - OG/G
8405	27 248 11	05/30/1984	WALLEYE	SKIN ON FILLET	5	19.0	1.20	1.3 - OG/G
8407	27 248 11	05/30/1984	WALLEYE	SKIN ON FILLET	3	23.0	2.60	4.7 - OG/G

----- SITE-GREEN BAY RED ARROW POINT LOCATION CODE=385011 COUNTY=MARINETTE -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7601	30 248 10	08/03/1976	WALLEYE	SKIN ON FILLET	3	14.8		0.52 - OG/G

----- SITE-MENOMINEE RIVER ANSUL CHEMICAL LOCATION CODE=385025 COUNTY=MARINETTE -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7604	10 248 08	08/18/1976	WALLEYE	SKIN ON FILLET	3	16.7		3.3 - OG/G
8303	10 248 08	05/31/1983	WALLEYE	SKIN ON FILLET	6	15.2	0.32	< 0.2 - OG/G
8304	10 248 08	05/31/1983	WALLEYE	SKIN ON FILLET	1	17.6	1.15	0.31 - OG/G

FISH RESULTS BY SITE NAME - COLLECTOR STYLE
PCB IN WALLEYS FROM GREEN BAY AND TRIBUTARIES

----- SITE-MENOMONIEE RIVER BELOW ANGOL CHEMICAL LOCATION CODE-J85007 COUNTY-MARINETTE -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8701	30 24E 08	06/04/1987	WALLEYE	SKIN ON FILLET	1	14.7	0.44	0.34 - UG/G
8702	30 24E 08	06/04/1987	WALLEYE	SKIN ON FILLET	1	18.3	1.06	1.1 - UG/G
8703	30 24E 08	06/04/1987	WALLEYE	SKIN ON FILLET	1	19.4	1.20	3. - UG/G
8704	30 24E 08	06/04/1987	WALLEYE	SKIN ON FILLET	1	21.4	1.29	2.9 - UG/G
8705	30 24E 08	06/04/1987	WALLEYE	SKIN ON FILLET	1	24.4	2.26	1.3 - UG/G
8802	30 24E 08	11/11/1988	WALLEYE	SKIN ON FILLET	2	7.7	0.06	< 0.2 - UG/G
8803	30 24E 08	11/11/1988	WALLEYE	SKIN ON FILLET	1	13.6	0.39	< 0.2 - UG/G
8804	30 24E 08	11/11/1988	WALLEYE	SKIN ON FILLET	1	15.3	0.49	< 0.2 - UG/G
8805	30 24E 08	11/11/1988	WALLEYE	SKIN ON FILLET	1	16.6	0.70	< 0.2 - UG/G

----- SITE-MENOMONIEE RIVER HATTIE STREET LOCATION CODE-J85021 COUNTY-MARINETTE -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7782	30 24E 08	06/08/1977	WALLEYE	SKIN ON FILLET	4	19.9		0.2 - UG/G

----- SITE-MENOMONIEE RIVER MARINETTE LOCATION CODE-185018 COUNTY-MARINETTE -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7701	30 24E 08	09/18/1977	WALLEYE	WHOLE FISH	5	17.5		13. - UG/G
9314	30 24E 08	04/26/1953	WALLEYE	SKIN ON FILLET	1	19.5	1.1	0.12 - UG/G
9316	30 24E 08	04/26/1953	WALLEYE	SKIN ON FILLET	1	22.0	1.8	1.9 - UG/G

----- SITE-MENOMONIEE RIVER MOVIN LOCATION CODE-38904 COUNTY-MARINETTE -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
7904	30 24E 05	07/12/1979	WALLEYE	WHOLE FISH	5	16		3.36 - UG/G

----- SITE-OCONTO RIVER BELOW STILES DAM LOCATION CODE-435014 COUNTY-OCONTO -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
9324	05 21E 18	04/07/1993	WALLEYE	SKIN ON FILLET	1	23.3	2.28	3.3 - UG/G
9325	05 21E 18	04/07/1993	WALLEYE	SKIN ON FILLET	1	23.5	2.40	2.2 - UG/G
9315	05 21E 18	06/08/1993	WALLEYE	SKIN ON FILLET	1	21.0	1.33	1.7 - UG/G

FISH / SEDIMENT CONTAMINANTS SYSTEM JOB ID: 657
 FISH RESULTS BY SITE NAME - COLUMB STYLE
 PCBs IN WALLEYE FROM GREEN BAY AND TRIBUTARIES

0539R105
 10:03 Monday, December 27, 1999

----- SITE-OCONTO RIVER OCONTO LOCATION CODE-435004 COUNTY-OCONTO -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8466	28 25E 19	09/19/1984	WALLEYE	SKIN ON FILLET	5	16.85	0.85	1.1 - UG/G

----- SITE-PESHIGO RIVER BELOW BADGER MILL LOCATION CODE-385022 COUNTY-MARINETTE -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8401	30 24E 06	05/31/1984	WALLEYE	SKIN ON FILLET	5	11.9	0.24	0.27 - UG/G
8402	30 24E 06	05/31/1984	WALLEYE	SKIN ON FILLET	4	13.2	0.39	0.29 - UG/G
9302	30 24E 06	04/09/1993	WALLEYE	SKIN ON FILLET	1	20.9	1.56	1.1 - UG/G
9304	30 24E 06	04/09/1993	WALLEYE	SKIN ON FILLET	1	21.6	1.96	0.62 - UG/G
9332	30 24E 06	06/02/1993	WALLEYE	SKIN ON FILLET	1	27.0	3.15	1.6 - UG/G

----- SITE-PESHIGO RIVER BELOW HIGHWAY 41 LOCATION CODE-385023 COUNTY-MARINETTE -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8301	30 23E 30	06/16/1983	WALLEYE	SKIN ON FILLET	2	22.9	2.42	3. - UG/G
8303	30 23E 30	06/21/1983	WALLEYE	SKIN ON FILLET	3	20.8	1.70	4.6 - UG/G

----- SITE-PESHIGO RIVER PESHIGO LOCATION CODE-385024 COUNTY-MARINETTE -----

FIELD NUMBER	T/R/S	COLLECTION DATE	SAMPLE TYPE	SAMPLE FORM	NUMBER OF FISH	AVERAGE LENGTH (IN.)	AVERAGE WEIGHT (KG.)	PCB
8001	30 23E 19	09/04/1980	WALLEYE	WHOLE FISH	2	16.6	0.80	7.3 - UG/G
8102	30 23E 19	08/14/1981	WALLEYE	WHOLE FISH	2		0.32	3.5 - UG/G
8105	30 23E 19	08/24/1981	WALLEYE	WHOLE FISH	4	16.1	0.75	3.25 - UG/G
8201	30 23E 19	08/19/1982	WALLEYE	WHOLE FISH	4	11.7	0.22	3.71 - UG/G