Final (Revised) Baseline Ecological Risk Assessment Allied Paper, Inc. / Portage Creek / Kalamazoo River Superfund Site

Michigan Department of Environmental Quality
Remediation and Redevelopment Division

April 2003

REGULATORY AGENCY NOTICE:

The Michigan Department of Environmental Quality (MDEQ) was the lead agency for the preparation of the Baseline Ecological Risk Assessment (BERA) for the Kalamazoo River. The United States Environmental Protection Agency (EPA) has worked jointly with MDEQ in the development of this BERA and concurs with the results and conclusions presented herein.

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This Executive Summary presents an overview of the Revised **(JANUARY 2003)** Final Baseline Ecological Risk Assessment (BERA) for the Allied Paper, Inc./Portage Creek/Kalamazoo River Superfund Site (API/PC/KR) in Southwestern Michigan. The revisions forming the basis of this **(JANUARY 2003)** revised final document address comments[1] on the

- Revised Final BERA **(JANUARY 2002),**
- Final BERA (June 1999), and
- the Addendum to the BERA (August 15, 2000).

KRSG comments were identified in various letters, data summaries, and technical memorandums received by MDEQ from 1999 through late 2001. A July 19, 1999 letter from KRSG to MDEQ contained comments from Giesy Ecotoxicology, Inc.; A September 11, 2000 letter from M.P. Brown to J. Brian von Gunten summarized similar comments. EPA and FWS concerns and comments were identified in several meetings and telephone conversations throughout summer and fall 2000. The Revised (January 2002) Final BERA addressed all the comments presented in these correspondences. An October 11, 2001 transmittal from M.P. Brown (Blasland, Bouck & Lee, INC.) to J.B. von Gunten (Michigan Department of Environmental Quality-Emergency Response Division (MDEQ-ERD) presented a report of the findings-to-date of Dr. J. Giesy's studies of ecological exposure and risks for the site. Concerns and issues presented in the Giesy report have been addressed with this Revised **(JANUARY 2003)** Final BERA.

The primary purpose of this ERA is to identify and describe actual or potential onsite conditions that can result in unacceptable risks to exposed organisms. Sufficient recent site-specific information indicates that this ERA should focus on the primary chemical stressors present at this site - polychlorinated biphenyls (PCBs). This ERA compares measured or estimated PCB concentrations in different types of exposure media (e.g., surface water, sediment, fish) with predicted biological effects to estimate risks and to preliminarily identify appropriate and protective cleanup levels.

Background and Site Description

Due to the PCB contamination, in August 1990 the site was placed on the Superfund or National Priorities List (NPL). The NPL Study Area (API/KR/PC) includes 3 miles of Portage Creek, from Cork Street to its confluence with the Kalamazoo River, and 80 miles of the Kalamazoo River, from Morrow Lake Dam downstream to Lake Michigan. Also included in the site are five paper residual disposal areas and five paper mill properties.

[1] U.S. Environmental Protection Agency (EPA), the U.S. Fish and Wildlife Service (FWS), and the Kalamazoo River Study Group (KRSG)

The Michigan Department of Community Health has issued a species-specific no consumption fish advisory annually since 1977 for the Kalamazoo River portion of this site due to PCB contamination. The Kalamazoo River and Portage Creek have been designated a site of environmental contamination under Part 201, Environmental Remediation, of the Natural Resources and Environmental Protection Act, 1994 PA 451, as amended (NREPA), due to PCB contamination. The Kalamazoo River and Portage Creek have also been identified as an Area of Concern by the International Joint Commission on the Great Lakes due to the detrimental impact the ongoing release of PCBs has on Lake Michigan.

General Approach to ERA

This ERA follows EPA guidance for conducting ERAs, primarily Ecological Risk Assessment Guidance for Superfund (EPA 1997) and Guidelines for Ecological Risk Assessment (EPA 1998). The major components of the ERA include Problem Formulation, Analysis, and Risk Characterization. The Problem Formulation phase of this ERA establishes the goals and describes the scope and focus of the assessment. In addition, this phase considers site-specific regulatory and policy issues and requirements and preliminarily identifies potential stressors and ecological resources potentially at risk. The outcome of Problem Formulation is the site-specific conceptual model, which describes potential exposure pathways and the relationship between remedial action objectives, assessment endpoints, and measurement endpoints. Uncertainties associated with this phase of the ERA are included at the end of this Section.

The Analysis phase of the ERA describes the nature and extent of contamination (Exposure Assessment) and identifies appropriate and relevant threshold concentrations, standards, or criteria for contaminants of concern (Effects Assessment). Uncertainty analysis related to this phase of the ERA is also included.

The final major component of the ERA, Risk Characterization, considers the information gathered in Problem Formulation and integrates Exposure and Effects data to estimate risks to ecological receptors. Also included in Risk Characterization is a discussion of ecological significance, risk summary, and uncertainty analysis.

This ERA also includes an additional section on Remediation Issues in which preliminary risk-based remediation goals (PRGs) are developed.

This ERA uses several lines of evidence to increase confidence in risk estimates and ERA conclusions. These include the use of simple hazard quotients that compare a single selected exposure concentration to a single selected effects concentration to derive a quotient. This is a common screening level approach for identifying issues of most concern. Supplementing this approach is a comparison of multiple media-specific exposure concentrations for specific site locations to multiple effects concentrations that include site-specific and literature-based values. This approach reduces the uncertainties in relying on single exposure and effects concentrations and contributes to the weight-of-evidence. Also included in this ERA is a food chain

model that estimates PCB dose via ingestion pathways for key receptor species or groups. Finally, this ERA considers field observations and other qualitative data as a check on risk estimates and conclusions.

Representative Receptors

Potential ecological receptors for this study are defined as plants and animals that inhabit or use, or have potential to inhabit or use, the aquatic, riparian/wetland, and terrestrial habitats of the API/PC/KR. The large number of potential receptor species identified for the API/PC/KR obviously precludes an assessment of potential risks for every species listed. Several species or groups of organisms have, therefore, been selected to serve as representative receptors for a detailed evaluation of potential risks. These include aquatic plants, aquatic macroinvertebrates, game fish (e.g., smallmouth bass), forage fish (e.g., sucker), rough fish (e.g., carp), terrestrial invertebrates (e.g., earthworms), small burrowing omnivorous mammals (e.g., deer mouse), semi-aquatic herbivorous mammals (e.g., muskrat), small semi-aquatic carnivorous mammals (e.g., mink), and top mammalian and avian predators (e.g., red fox, great horned owl, bald eagle).

ERA-Related Goals and Objectives

ERA-related remedial action goals and objectives for the API/PC/KR have been determined by MDEQ, and include: (1) the establishment and maintenance of a healthy and diverse aquatic and riparian co-systems in and adjacent to the API/PC/KR, and (2) reductions in PCB concentrations in fish and wildlife such that human consumption restrictions can be lifted.

Site Conceptual Model

The site conceptual exposure model (SCEM) is the primary output of the Problem Formulation phase of the ERA, and is used to develop a series of null hypotheses for the API/PC/KR, primarily those regarding potential exposure scenarios and the relationship between selected assessment and measurement endpoints. The null hypotheses for the API/PC/KR are defined as follows:

1. *The levels of contaminants in water, sediment, and biota are not sufficient to adversely affect the structure or function of the fish populations in the Kalamazoo River and Portage Creek System.*

2. *The levels of contaminants in water, sediment, and biota are not sufficient to adversely affect the survival, growth, and reproduction of plant and animal aquatic receptors utilizing the Kalamazoo River and Portage Creek system.*

3. *The levels of contaminants in water, sediment, soil, and biota are not sufficient to adversely affect the survival, growth, and reproduction of mammalian receptors utilizing the Kalamazoo River and Portage Creek system.*

4. *The levels of contaminants in water, sediment, and biota are not sufficient to adversely affect the survival, growth, and reproduction of avian receptors utilizing the Kalamazoo River and Portage Creek system.*

Summary of Conclusions

Hazard Quotient-based Risks

Hazard quotients based on direct toxicity for aquatic biota and dietary dose for other species reveal that mink are at most risk compared to other representative receptors. This preliminary conclusion is supported by multiple lines of evidence described in the ERA.

Overall Risk Summary

Multiple lines of evidence are used to reach the following conclusions.

- Most aquatic biota such as invertebrates and fish are unlikely to be adversely affected by direct contact with and ingestion of surface water because of relatively low PCB toxicity to most aquatic biota. Bioaccumulation of PCBs is not considered at this stage.

- PCB contamination of surface water and streambed sediment is likely to adversely affect sensitive piscivorous predators, such as mink, through consumption of PCB-contaminated prey, especially fish.

  - Impaired reproduction of mink and ultimately decreases in mink populations are the most likely effects of PCB contamination in aquatic prey. There is qualitative evidence that mink populations are declining or are reduced.

  - Other piscivorous predators, such as bald eagles, are at substantial risk based on assumptions about diet (e.g., fish are the predominant prey item consumed) and exposure (e.g., foraging takes place mostly within contaminated aquatic areas). Preliminary data suggest both these assumptions are valid. Field investigations of bald eagles by U.S. Fish and Wildlife suggest there has been a loss of reproductive capacity and decrease in the populations of bald eagles within the site boundaries.

- Terrestrial and semi-aquatic biota may be at risk from PCB-contaminated floodplain sediment and surface soil, depending on life history (e.g., foraging behavior, diet, mobility) and sensitivity to PCBs. Such risk is in general considered to be low to moderate, depending on species.

  - Omnivorous birds (represented by the robin) that consume a substantial amount of soil invertebrates (e.g., earthworms) would be at significant risk if foraging takes place in mostly contaminated areas.

- Carnivorous terrestrial mammals (represented by the red fox) may be at some risk if foraging is concentrated in riparian areas with contaminated floodplain sediment and diet consists of prey that (1) reside in PCB-contaminated areas, and (2) have taken up substantial amounts of PCBs.

- Carnivorous birds (represented by great horned owl) may be at significant risk, depending on diet. Relatively high risks were calculated in association with high PCB concentrations in eggs, while risk estimates generated as a result of food web modeling were comparatively low. Uncertainties with actual diet of great horned owls in the API/PC/KR area and discrepancies between estimated risks to owls, based on the two different methods mentioned previously, cannot be resolved with available data.

- Omnivorous terrestrial species (represented by mice) are unlikely to be at significant risk unless they reside in the most contaminated areas. PCB uptake in mice appears to be relatively low.

- Semi-aquatic herbivorous mammals (represented by muskrat) may be at risk from PCB contamination because estimated dietary doses exceed recommended threshold values for rats. This conclusion is based on the assumption that laboratory rats and muskrats are equally sensitive to PCBs via ingestion. Muskrats contaminated with PCBs may also cause adverse effects to muskrat predators because some muskrats contain PCBs in excess of recommended dietary limits for PCB-sensitive predators such as mink.

This ERA presents overwhelming evidence that, despite uncertainties identified and discussed in the ERA, two and possibly three of the four proposed null hypotheses can be rejected with little reservation.

The first null hypothesis is accepted because there is no direct evidence that fish communities are being affected by PCB contamination. The impaired fish community of Lake Allegan is comprised primarily of stunted and often malformed carp. The cause of these findings cannot be determined from the available data. It is noted, however, that PCBs cause a wasting syndrome in several mammalian species. There is insufficient site-specific data to determine if fish communities in the Kalamazoo River are being directly affected by PCB contamination.

The second null hypothesis is conditionally rejected. This is based on the finding that at some locations the maximum detected surface water PCB concentrations exceed or closely approach the lowest chronic value for freshwater fish or aquatic plants.

The last two null hypotheses are rejected because risks to mammalian (e.g., mink) and avian predators (e.g., bald eagle), especially those that consume fish, are unacceptable. These conclusions are based primarily on the very high levels of PCB concentrations in fish, other biota, and abiotic media (e.g., floodplain sediments).

The ecosystem associated with the API/PC/KR portion of the Kalamazoo River has been and is currently being adversely affected by PCBs originating from past industrial activities. This evidence by the distribution of PCBs in biota at all trophic levels within the API/PC/KR.

Remediation Issues

The selection of the most appropriate methods for achieving remediation goals is not a risk assessment issue but is a risk management issue to be addressed in the feasibility study (FS) for this API/PC/KR. The application of cleanup values is also considered a risk management decision. This risk assessment derives and recommends threshold PCB concentrations ("cleanup values") for each media type. These values are not necessarily intended to be applied to all locations within the API/PC/KR or within a sub-area of the API/PC/KR. For example, it is probably most appropriate to use cleanup values as average media-specific post-remediation concentration goals within a specific area. Alternatively, a cleanup value can be considered a "never to exceed" value for any onsite sample, but such an application might result in needlessly exceeding remediation goals and costs in most areas within the site. It is most appropriate for risk managers rather than risk assessors to decide how to best apply cleanup values recommended in the risk assessment. The proposed cleanup ranges include no effect levels at the lower end and low but significant effect levels at the upper end. These protective PCB ranges for each media type for the Allied Paper, Inc./Portage Creek/Kalamazoo River Superfund Site are presented below.

- **Range of protective total PCB concentrations in SURFACE WATER is 0.0016 to 0.00197 µg/L (based on mink, the most sensitive of all animals tested to date).**

- **Range of protective total PCB concentrations in INSTREAM SEDIMENT AND FLOODPLAIN SEDIMENT associated with aquatic or semi-aquatic ecosystems is 0.5 to 0.6 mg/kg (based on mink, the most sensitive of all animals tested to date).**

- **Range of protective total PCB concentrations in SURFACE SOILS AND FLOODPLAIN SEDIMENTS associated with terrestrial ecosystems is 6.5 to 8.1 mg/kg (based on omnivorous songbirds such as robin). To protect carnivorous mammals such as red fox, the range is 5.9 to 29.5 mg/kg.**

1 Section One

Section 1
Introduction

This document presents the Revised Final Baseline Ecological Risk Assessment (BERA) for the Allied Paper, Inc./Portage Creek/Kalamazoo River Superfund Site (API/PC/KR) in Southwestern Michigan. The revisions forming the basis of this revised final document address recent comments on the Final (Revised) BERA (January 2002), the Final BERA (June 1999) and the Addendum to the BERA (August 15, 2000); comments were submitted by the U.S. Environmental Protection Agency (EPA), the U.S. Fish and Wildlife Service (FWS), and the Kalamazoo River Study Group (KRSG).

KRSG comments (and those of Giesy Ecotoxicology, Inc.) were presented in letters (July 19, 1999 and September 11, 2000) from KRSG to the Michigan Department of Environmental Quality (MDEQ). In addition, a Technical Memo and letter (October 11, 2001) from KRSG to MDEQ summarized preliminary data obtained by KRSG. Included in this document were preliminary data, evaluations, and conclusions potentially relevant to information presented in the Final (Revised) BERA, dated January 2002. Some of the information presented by the KRSG resulted in a more intensive review of toxicity literature associated with the derivation of appropriate dose-based TRVs for mink and birds. PCB exposure data presented in the October 11, 2001 KRSG document were considered preliminary, and therefore are not included in this revised final BERA. Data such as these may be considered in the future.

EPA and FWS comments were identified in several meetings and telephone conversations throughout summer and fall 2000. Additional comments were received from EPA in spring and summer 2001 through meetings in Benton Harbor and Chicago. The MDEQ has worked closely with EPA from 2001 to 2003 to finalize this risk assessment document.

This assessment uses site-related chemical concentrations, exposure potential, and toxicity information to characterize potential risks to ecological receptors from releases of polychlorinated biphenyls (PCBs) to the Kalamazoo River ecosystem. Risks are estimated assuming no remedial action has occurred at the site, and are intended to assist the risk manager in determining the acceptable clean-up levels to protect ecological receptors.

1.1 Report Objectives

ERAs evaluate the likelihood that adverse ecological effects may occur or are occurring at a site as a result of exposure to single or multiple chemical or physical stressors (EPA 1992a). Risks result from contact between ecological receptors and stressors that are of sufficiently long duration and of sufficient intensity to elicit adverse effects (EPA 1992a). The primary purpose of this ERA is to identify and describe actual or potential onsite conditions that can result in adverse effects to present or future ecological receptors. Sufficient recent site-specific information is

available to allow this ERA to focus on the primary ecological stressors present at this site. These primary stressors have been identified as polychlorinated biphenyls (PCBs). This ERA focuses on comparing measured or estimated PCB exposures with observed or predicted biological effects. This ERA also provides information that can help establish remedial priorities and serve as a scientific basis for regulatory and remedial actions for the API/PC/KR.

1.2 Report Organization

The approach used to conduct this ERA is based on site-specific information and on recent EPA guidance, primarily Ecological Risk Assessment for Superfund: Process for Designing and conducting Ecological Risk Assessments (EPA 1997), supplemented by The Framework for Ecological Risk Assessment (Framework Document, EPA 1992a). EPA (1989, 1992a, 1997) and others (e.g., Barnthouse, et al. 1986) recognize that methods for conducting ERAs must be site-specific, and guidance documents for conducting ERAs are therefore not intended to serve as detailed, specific guidance. As much as practicable, the methods, recommendations, and terminology of the 1997 guidelines for ecological risk are used to conduct this ERA. The organization of this ERA follows the format presented in this document, with some modifications made for site-specific considerations and readability. Following this introduction, a short description of the site is presented in Section 2. The primary components of this ERA are:

- Problem Formulation (Section 3) which describes the goals, scope and focus of the ERA;
- the Analysis Phase (Section 4), which evaluates the data used to assess exposures for local flora and fauna;
- and the Risk Characterization (Section 5), which discusses the risks identified by this ERA. Additionally, Section 5 describes remedial goals for PCBs in sediments, surface water, and floodplain soils associated with the Kalamazoo River.
- References for all sections are provided in Section 6.

Section Two

Section 2
Site Description

The Kalamazoo River drainage basin encompasses approximately 2,000 square miles. The main stem of the Kalamazoo River begins in Albion, Michigan at the confluence of the North and South Branches of the Kalamazoo River, and flows northwesterly for 123 miles through Calhoun, Kalamazoo, and Allegan Counties to Lake Michigan at Saugatuck. The Kalamazoo River is fed by more than 400 miles of tributaries, including Portage Creek. Portage Creek begins in Portage, Michigan and including its west fork, flows a distance of approximately 18.5 miles.

Due to the PCB contamination, in August 1990 the site was placed on the National Priorities List (NPL) in accordance with the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), 1980 PL 96-510 as amended by the Superfund Amendments and Reauthorization Act (SARA) of 1986 also known as Superfund. The NPL Study Area defined (also known as the API/KR/PC) includes three miles of Portage Creek, from Cork Street to its confluence with the Kalamazoo River, and 80 miles of the Kalamazoo River, from Morrow Lake Dam downstream to Lake Michigan (Figure 2-1). Also included in the site are five paper residual disposal areas and five paper mill properties. Paper residuals (residuals) are the waste material produced by the paper mill during the paper making process. The Michigan Department of Community Health has issued a species-specific no consumption fish advisory annually since 1977 for the Kalamazoo River portion of this site due to the PCB contamination. The Kalamazoo River and Portage Creek have been designated a site of environmental contamination under Part 201, Environmental Remediation, of the Natural Resources and Environmental Protection Act, 1994 PA 451, as amended (NREPA), due to PCB contamination. The Kalamazoo River and Portage Creek have also been identified as an Area of Concern by the International Joint Commission on the Great Lakes due to the detrimental impact the release of PCBs have on Lake Michigan.

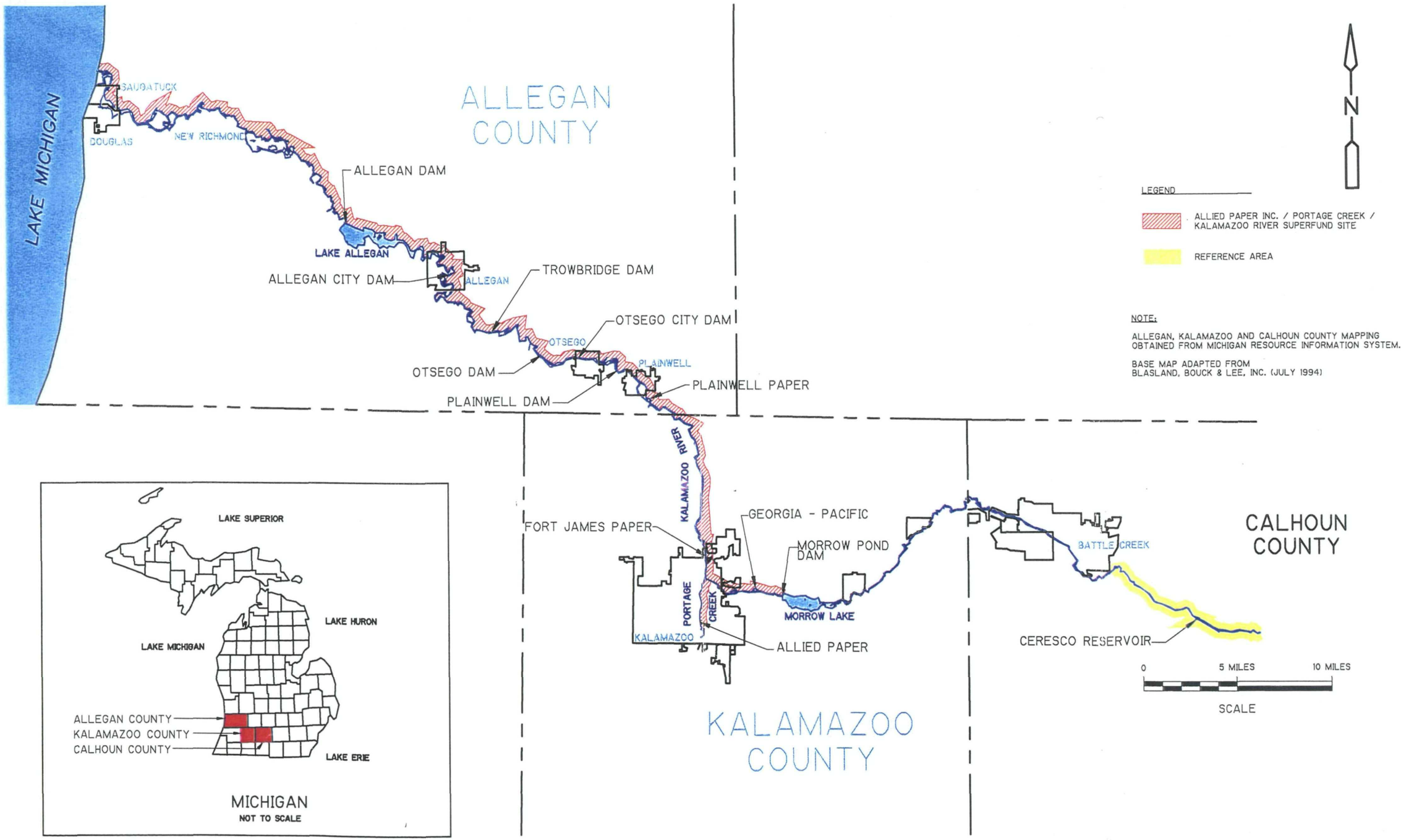
The Kalamazoo River is an alternating series of free flowing sections and impoundments formed by low level dams. The Plainwell, Otsego, and Trowbridge Dams have been removed to their sill levels, exposing approximately 507 acres of former sediments as floodplain soils (Blasland, Bouck & Lee, Inc. 1992). Since these impoundments are all located downstream of the paper mills and landfills, which are the PCB sources, they serve as natural sinks for PCB-contaminated sediments. The former dams continue to impound water but to a lesser extent than when dams were operational. The Michigan Department of Natural Resources (MDNR) owns these three dams and their goal is to remove the remaining structures and return the river to its natural channel. The Otsego City Dam, Allegan City Dam, and the Calkins Dam (Allegan Lake Dam) are still intact. The Calkins dam is used to produce hydroelectric power (Blasland, Bouck & Lee, Inc. 1992).

The NPL identified PCBs as the primary contaminant of concern at the API/PC/KR. PCBs were introduced to the environment as a result of using the river for

discharging of waste. The primary industrial activity associated with PCB releases into the API/PC/KR environment was the recycling of PCB-containing carbonless copy paper at several area paper mills. In the process of de-inking and re-pulping recycled paper, paper mills produce substantial quantities of waste residuals. During the period from 1957 to 1971, carbonless copy paper contained PCBs as an ink solvent. Kalamazoo-area paper mills that de-inked or re-pulped the PCB-containing carbonless copy paper thereby incorporated PCBs in their waste streams. These paper mills disposed of their wastes in several ways that resulted in releases of PCBs to the environment, including direct discharge of wastes to Portage Creek and the Kalamazoo River and placement of wastes in disposal areas (landfills) from which PCBs are leached or eroded. The paper wastes also included kaolinite clays, which can be significant sorbents of PCBs, primarily as a result of surface area. These clays have been deposited in the API/PC/KR and when concentrated, they appear as spongy, light grey clay layers. In addition, PCBs are persistent in the environment and degradation via chemical oxidation, hydrolysis, and photolysis in soil or aquatic systems is generally insignificant (Blasland, Bouck & Lee, Inc. 1992). PCBs are continually being released to the river from erosion of floodplain soils that exist behind the impounded areas and from instream sediments. Therefore, PCBs are a persistent problem at the API/PC/KR. Similar river systems such as the Fox River (WDNR 1993) and the Hudson River (Brown, et al. 1985) have PCB contaminated sediments that are the major supplier of PCBs to the ecosystem once direct discharges have been eliminated.

Figure 2-1A, in *Description of the Current Situation Report* (Blasland, Bouck & Lee, Inc. 1992) provides a more detailed description of the physical settings and characteristics of the API/PC/KR. Much of the abiotic data used in this ERA were obtained from this report.

In 1993, Camp Dresser & McKee (CDM) prepared a Biota Sampling Plan (CDM 1993) that outlined sampling activities for the collection of biotic data within the study area. Sampling of biota was conducted to determine current levels of PCBs in resident biota. Based upon these field studies a site-specific model was developed to evaluate bioaccumulation and risk, upon which remedial activities may be based. Field sampling was conducted by Blasland, Bouck & Lee, Inc., with oversight by CDM and the Michigan Department of Environmental Quality (MDEQ) or by the MDEQ. Biological tissue and corresponding abiotic media data collected in the study area were used in this ecological risk assessment.



ALLIED PAPER, INC./PORTAGE CREEK/KALAMAZOO RIVER SUPERFUND SITE

KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT STUDY AREA

CDM
environmental engineers, scientists, planners, & management consultants

Figure No. 2 - 1

Section Three

Section 3
Problem Formulation

The Problem Formulation phase of this ERA establishes the goals and describes the scope and focus of the assessment. In addition, this phase considers site-specific regulatory and policy issues and requirements and preliminarily identifies potential stressors (Section 3.1) and ecological resources potentially at risk (Section 3.2). The outcome of Problem Formulation is the site-specific conceptual exposure model (SCEM), which describes potential exposure pathways and the relationship between remedial action objectives, assessment endpoints, and measurement endpoints. Endpoints are defined and discussed in Section 3.3, and the site conceptual model is described in Section 3.4.

3.1 Stressor Identification

This ERA is focused on the potential ecological effects associated with PCB contamination of surface water, sediment, surface soil, and biota. Current levels of PCB contamination in these media can adversely affect aquatic and terrestrial ecosystems in and adjacent to the API/PC/KR. Other chemical stressors and physical (non-chemical) stressors, such as habitat disturbance, may also contribute to adverse ecological effects at this site. PCB contamination is considered to be the primary focus of this ERA because of the current magnitude and distribution of PCBs throughout the API/PC/KR (Figure 2-1, presented in Section 2). This ERA, therefore, does not consider the additional incremental effects that may be caused by other chemical stressors. Such effects are likely to be relatively minor compared to the actual or potential effects due to PCB exposures.

Dissolved and particulate-sorbed PCBs occur within and adjacent to the API/PC/KR boundaries. Based on extensive data for this site, the primary chemicals or groups of chemicals of potential concern for the API/PC/KR are PCBs. Of most concern are those with higher chlorine (Cl) content such as Aroclor 1016 (40 percent Cl by weight), 1242 (42 percent Cl), 1248 (48 percent Cl), 1254 (54 percent Cl), and 1260 (60 percent Cl). The more highly chlorinated PCBs are environmentally persistent and potentially most hazardous to ecological receptors (Eisler 1986). Most of the measured PCBs at the API/PC/KR are those that are persistent in the environment, such as Aroclors 1242, 1248, 1254, and 1260. Aroclor 1260 is the most commonly found Aroclor in biological tissue. This ERA is focused on the highly chlorinated PCBs observed in biotic and abiotic media.

It should be noted that from a regulatory perspective, all PCBs are regulated in Michigan as total PCBs, not as individual PCB congeners. Also, much of the toxicological literature on PCB effects is based on total PCB exposures. Total PCB concentrations, rather than Aroclor- or congener-specific PCB concentrations, are therefore used in this ERA to represent exposure concentrations. Evaluations of potential risk in this ERA are based on total PCB concentrations in abiotic media (e.g., surface water, sediment, surface soil) and biological tissues. Table 3-1 presents the

primary PCBs detected in abiotic and biological samples. The potential ecological effects associated with total PCBs are summarized in Section 4.2.1.

3.2 Ecological Resources Potentially at Risk

This section identifies and describes the major habitats and organisms, or types of organisms, that may be exposed to the chemical and physical stressors identified at the API/PC/KR site.

3.2.1 Habitat Descriptions

The API/PC/KR ERA is based on data collected from the Kalamazoo River upstream of the City of Battle Creek (upstream reference area) downstream to U.S. Highway 31, east of Lake Michigan (Figure 2-1). The area below Allegan Dam is considered to be impacted by current or past upstream PCB sources. The NPL (Superfund) site is the extent of Portage Creek and the Kalamazoo River including the 100-year floodplain prior to the removal of the Otsego, Plainwell, and Trowbridge Dams down to the sills. The major habitat types within the API/PC/KR site - aquatic habitats, riparian habitats/wetlands, and terrestrial habitats - are qualitatively described below.

Aquatic Habitats

Aquatic habitats within the API/PC/KR site are found within Portage Creek, the Kalamazoo River, and their tributaries. The Kalamazoo River is a large, perennial river that drains a major portion of western Michigan. The API/PC/KR site includes approximately 80 river miles. The character of the Kalamazoo River varies from reach to reach. The Kalamazoo River has been influenced by historic flood events as well as dam construction, operation, and removal. Currently, there are areas impacted by fluvially deposited sediments contaminated with anthropogenic chemicals within and adjacent to the river.

Instream substrates consist of variable proportions of the following:

- Boulders (>256 mm or 10 in)
- Cobble (64 to 256 mm or 2.5 to 10 in)
- Gravel (2 to 64 mm or 0.1 to 2.5 in)
- Sand (0.06 to 2.00 mm)
- Silt (0.004 to 0.06 mm)
- Clay (<0.004 mm)
- Organic matter (e.g., leaves, sticks, etc.)

A complete evaluation of particle size distribution of the API/PC/KR bed sediments has not been performed, but the following generalizations adequately describe the major types of API/PC/KR substrates and habitat conditions:

- Former impoundment sites and areas downstream of those subject to erosion are associated with increased siltation and decreased particle size, potentially increasing contaminant loads in these areas.

- Bottom substrates consist of unconsolidated materials, as well as some submerged and emergent vegetation, which may act as sediment traps.

- The relative abundance of potential fish cover (i.e., undercut banks, overhanging vegetation, deep pools, boulders, logs, aquatic vegetation) varies considerably within the API/PC/KR site. These areas are especially uncommon within certain sections of the broad floodplain where extensive sediment deposition has occurred.

- Stream channel stability varies with the pattern of annual flooding.

- Large areas associated with some of the former impoundments are commonly inundated for several months each year. These events result in seasonally increased habitat for receptors such as mink, muskrat, carp, amphibians, and crayfish.

- Areas of suitable habitat for abundant and diverse macroinvertebrate populations (i.e., cobble or gravel substrates with adequate water flow and depth) are uncommon and unevenly distributed throughout the API/PC/KR site.

To aid in the evaluation of aquatic habitats and chemical exposure for this ERA, the API/PC/KR site is divided into 12 Aquatic Biological Study Areas (ABSAs). Originally, ABSAs defined specific locations from which aquatic biota were collected. To describe aquatic habitats and potential exposure areas, these ABSAs were expanded so that they are contiguous, with ABSA boundaries based on physical features such as dam sites or bridges. This approach results in all reaches within the API/PC/KR site being associated with a specific ABSA. The expanded ABSAs and associated Terrestrial Biological Study Areas (TBSAs) are described in Table 3-2.

Terrestrial samples (e.g., white-footed/deer mice, earthworms, surface soil) were collected from specific areas within selected ABSAs. Soil sampling identified five acceptable terrestrial biological sampling areas (TBSAs 1, 3, 5, 10, and 11) from which terrestrial samples would be collected. In some cases, these soil and biota samples taken from the TBSAs can also be considered semi-aquatic rather than terrestrial because some of the sampling locations are commonly flooded for a significant portion of time each year. These "surface soil" samples collected within the floodplain are therefore not representative of terrestrial exposures in upland areas, and are probably best defined as floodplain sediments. Such sediments are more closely linked to aquatic rather than terrestrial environments from a source (deposition) perspective and from the types of biota inhabiting or utilizing these areas seasonally (e.g., spawning fish, amphibians, and crayfish in wet seasons and more terrestrial biota in dry seasons).

Each of the ABSAs and TBSAs correspond to particular areas of concern for this ERA. The major areas evaluated in this ERA include:

- Reference area (ABSA 1)

- The Portage Creek area (ABSA 12), which influences ABSA 3 and upstream portions of ABSA 4
- The former Plainwell Impoundment area, which influences the lower portion of ABSA 4 and all of ABSA 5
- The Otsego City Dam impoundment area (ABSA 6)
- The former Otsego Dam impoundment area (ABSA 7)
- The former Trowbridge Dam impoundment area (ABSA 8)
- Lake Allegan (ABSA 9)
- Areas immediately downstream of Lake Allegan that may be impacted by upstream areas (ABSA 10)

Impacts to each of these areas of concern are evaluated in this ERA on an ABSA-specific basis. In the Risk Characterization phase of the ERA, the ecological significance of ABSA-specific impacts to each of the major areas of concern is evaluated. Preliminary remedial goals (PRGs) are derived on a site-wide basis for different exposure scenarios and representative receptors.

Riparian Habitats/Wetlands

Riparian habitats exist adjacent to the watercourse of the Kalamazoo River and Portage Creek. Riparian habitats include both upland and wetland habitats within the floodplain of the river. Native floodplain soils are composed of fluvially deposited silts, fine to coarse sands, and gravels of varying sizes. In certain areas, these floodplain soils are covered with contaminated fine-grained sediments.

Numerous wetlands are identified within the API/PC/KR. These include shrub/scrub wetlands, emergent wetlands, and forested wetlands. These provide diverse and abundant vegetation and habitat for a wide variety of aquatic and riparian/terrestrial species dependent on aquatic ecosystems. These areas are, therefore, important for the health and status of several types of terrestrial as well as aquatic biota, and the types of biota supported by these wetlands may vary over season because of periodic flooding.

In general, the wetlands that occur throughout the API/PC/KR are dominated by a large variety of perennial grasses, shrubs, and trees common to western Michigan. See Appendix A for a detailed list of plant species. Outside of industrial or residential areas, there does not appear to be substantial differences in the diversity and abundance of riparian plants from one ABSA to another.

Terrestrial Habitats

Terrestrial habitats beyond the riparian areas and beyond the areas subject to seasonal inundation include relatively flat open areas with varying amounts of vegetative cover, some of which are used for grazing cattle. Also nearby are low rolling hills that are mostly thickly wooded and densely shaded. Terrestrial habitats in the API/PC/KR site are also found in portions of residential and industrial areas and represent ecological islands within urban areas. Finally, upland areas such as those identified in some cases as landfills are also considered terrestrial habitats.

3.2.2 Impacts to Ecological Resources

The API/PC/KR corridor supports a large variety of ecological resources (Section 3.2.3). This ERA is focused on addressing the impacts of PCB contamination to surface water, streambed sediments, floodplain sediments, and surficial soils, as well as biota that are adversely affected by ingestion of PCB-contaminated food items, resulting in increased levels of bioaccumulation of PCBs in higher trophic levels. Figures 3-1 through 3-10 show the results of observed PCB concentrations in various aquatic/semi-aquatic (surface water, fish, mink, muskrat, streambed sediments) and terrestrial (mice, earthworms, surface soils) media that were sampled in the defined ABSAs and TBSAs in accordance with the API/PC/KR Biota Sampling Plan (CDM 1993). Each figure provides the number of samples collected, and the mean, minimum, and maximum PCB concentrations observed in individual media for each ABSA or TBSA. Section 5, Risk Characterization, addresses the risks associated with the observed PCB contamination at the API/PC/KR site.

3.2.3 Identification of Potential Receptors

Potential ecological receptors for this study are defined as plants and animals (i.e., macroinvertebrates, fish, amphibians, reptiles, birds, and mammals) that inhabit or use, or have potential to inhabit or use, the aquatic, riparian/wetland, and terrestrial habitats of the API/PC/KR site. Although other organisms such as bacteria, protozoans, and fungi are essential components of aquatic and terrestrial ecosystems, potential impacts to these organisms are not assessed in this ERA because adequate data are unavailable for such an assessment.

Field surveys conducted by CDM and others revealed a large variety of plant and animal species utilizing all available habitat types in the study area. Studies were not conducted specifically to evaluate relative abundance or diversity of plant and animal species resident to or using the API/PC/KR. In general, however, a large variety of plant and animal species expected in the area were observed during fieldwork conducted in support of the ERA (See Appendix A).

Several plant and animal species of special concern have potential to exist in the study area (Appendix A), including threatened, endangered, and sensitive species such as white false indigo, bald eagle, great blue heron, eastern box turtle, marbled salamander, black redhorse, lake sturgeon, frosted elfin, red-shouldered hawk, and

elktoe mussel. Bald eagles do nest within the lower reaches of the API/PC/KR site, and great blue herons have an established heron rookery along the Kalamazoo River downstream of Lake Allegan. Appendix A also provides lists of invertebrates, fish, amphibians, reptiles, birds, and mammals that are found in this part of Michigan. All of these species have potential to occur within the API/PC/KR site.

Major species, including local subspecies, or types of organisms that have been observed onsite, expected to inhabit or use the API/PC/KR environs, or have potential to inhabit or use the area are described below. The species lists, presented in Appendix A, do not identify every plant or invertebrate that occurs or might occur onsite, but instead include observed species and representatives of major groups of these organisms that may occur onsite. Vertebrate species, including subspecies if applicable, that (1) have been observed onsite, (2) are likely to occur onsite, or (3) have potential to occur onsite, are considered potential receptors and are therefore included in the species lists provided. The potential to inhabit or use the API/PC/KR is based on published geographical ranges, general habitat requirements, comparison to nearby reference areas and, in some cases, the remediation of critical chemical or physical stressors.

The large number of potential receptor species identified for the API/PC/KR obviously precludes an assessment of potential risks for every species listed. Several species or groups of organisms have therefore been selected to serve as representative receptors for a detailed evaluation of potential risks. The selection of these receptors is based on

(1) their perceived importance to local ecosystems (e.g., key prey species),

(2) their population status,

(3) their relationship with human use (e.g., game species),

(4) the size of their home range in relation to the area,

(5) sensitivity to PCBs, and

(6) the availability of data for assessing potential risk.

Using these criteria, the following nine groups of organisms are selected as final ecological receptors for the API/PC/KR.

Aquatic Plants
Primary producers in aquatic ecosystems; can be important food items for zooplankton and other invertebrates which, in turn, are preyed upon by small/young fish and other aquatic life; potentially abundant; potential for high biomass (e.g., algae).

Aquatic and Semi-Aquatic Macroinvertebrates
Important prey species for many game fish; potentially abundant; potential for high biomass (e.g., larval midges, mayflies, stoneflies, caddisflies, and amphipods). Semi-aquatic invertebrates such as crayfish may be important food items for mink and other predators.

Freshwater Game Fish
Potential for high biomass; significant relationship with human use (e.g., smallmouth bass and salmonids).

Freshwater Forage Fish
Potential for high biomass; likely to be significant prey item for piscivorous predators, including game fish (e.g., white sucker).

Freshwater Rough Fish
Potential for high biomass; likely to be significant prey item for piscivorous predators, including mink; intimate contact with potentially contaminated sediment (e.g., common carp).

Terrestrial Invertebrates
Abundant; important prey species for shrews, birds, toads, etc. (e.g., earthworms).

Small Burrowing Terrestrial and Semi-Aquatic Mammals
Abundant; important prey species for certain snakes, birds, and mammals; significant relationship with humans (e.g., white-footed or deer mouse and muskrat).

Small Carnivorous/Omnivorous Mammals
Relatively abundant; relatively small home range; important consumers of aquatic and terrestrial biota; sensitive to PCB exposure; significant relationship with humans (e.g., mink).

Top Predators
At greatest risk for contaminants that bioaccumulate and biomagnify, including PCBs; significant relationship with humans; potentially species of concern (e.g., red fox, great horned owl, peregrine falcon, bald eagle).

3.3 Identification of Endpoints

This section introduces, defines, and discusses appropriate assessment and measurement endpoints for evaluating potential ecological effects.

3.3.1 Assessment Endpoints

Assessment endpoints identify the ecological values to be protected (e.g., abundance and diversity of aquatic macroinvertebrates or fish). Assessment endpoints are directly related to ERA-related remedial action goals and objectives determined for the API/PC/KR site. Appropriate assessment endpoints are developed by risk

assessors and often consider guidance from relevant regulatory agencies. ERA-related remedial action goals and objectives for the API/PC/KR have been determined by MDEQ, and include:

1. The establishment and maintenance of a healthy and diverse aquatic ecosystem in and adjacent to the API/PC/KR site

2. Reductions in PCB concentrations in fish and wildlife such that human consumption restrictions can be lifted

Site-specific remedial action goals and objectives should include:

1. The removal from the environment and isolation of all PCB-contaminated soils, sediments, and groundwater to a level that will achieve state water quality standards in the Kalamazoo River and Portage Creek (0.000026 µg/L for human health and 0.00012 µg/L for wildlife)

2. Remediation until residual levels in the environment are so low that healthy, safe-to-consume (e.g. no fish fillets greater than 2 ppm), self-reproducing, and ecologically diverse fish and wildlife populations can return to and survive in the Kalamazoo River basin

The Michigan Department of Environmental Quality suggests that water, soil, and whole fish cleanup objectives be set at current minimum detectable levels of 0.33 ppm. These are to be achieved while avoiding or minimizing a loss of floodway/floodplain capacity, reductions in river channel length, or loss of wetland values. Assessment endpoints are described as explicit expressions of the environmental variable(s) that are to be protected. The characteristics of the contaminants of concern, toxic mechanisms, and exposure pathways were used to select the following assessment endpoints:

- Preservation of the fish populations (e.g., smallmouth bass and white sucker) and communities utilizing the Kalamazoo River and Portage Creek system

- Preservation of the survival, growth, and reproductive capacity of aquatic receptors (e.g., aquatic plants, benthic macroinvertebrates, fish, larval amphibians) utilizing the Kalamazoo River and Portage Creek system

- Preservation of the survival, growth, and reproductive capacity of mammalian receptors (e.g., mouse, mink, muskrat, red fox) utilizing the Kalamazoo River and Portage Creek system

- Preservation of the survival, growth, and reproductive capacity of avian receptors (e.g., robin, bald eagle, and great-horned owl) utilizing the Kalamazoo River and Portage Creek system

It is assumed that the protection of the aforementioned sensitive aquatic and terrestrial receptors would be associated with the protection of other sensitive organisms or receptors for which toxicity data are lacking such as reptiles, songbirds, etc.

3.3.2 Measurement Endpoints

Assessment endpoints are often difficult to measure or evaluate directly. For example, we cannot predict with certainty the critical concentration of PCBs in surface water and sediment that allows survival and successful reproduction of smallmouth bass or salmonids in the Kalamazoo River. Such critical concentrations are site-specific and depend on innumerable factors. These factors may include the water quality and dietary requirements of prey species consumed by game fish, chemical interactions (i.e., synergistic, antagonistic, or additive), and the physical and chemical characteristics of the API/PC/KR site (e.g., streambed particle size, sediment organic carbon content, dissolved organic carbon concentration in surface water, temperature, dissolved oxygen, streambank and in-stream cover, etc.).

Measurement endpoints are used in cases where assessment endpoints cannot be directly measured or evaluated. Measurement endpoints are *quantitative expressions of observed or measured biological responses to stressors relevant to selected assessment endpoints*. For example, macroinvertebrate abundance (an assessment endpoint) can be evaluated using aquatic toxicity data based on an appropriate measurement endpoint. For example, concentrations of PCBs in API/PC/KR surface water can be compared to concentrations in laboratory test water that resulted in observed ecologically significant effects to sensitive and relevant test species (e.g., smallmouth bass or closely related species).

For this ERA, ecologically significant effects are defined as those affecting survival, growth, or reproduction. Other ecologically significant impacts such as effects on metabolic health were not considered. The example described above expresses the relationship between a relevant measurement endpoint (chronic effects concentration of PCBs in surface water) that is directly related to the assessment endpoints of game fish abundance and reproduction. Measurement endpoints selected for this are based on information from appropriate aquatic ecology/toxicology studies, water quality studies, and terrestrial toxicological studies (e.g., data summarized in EPA 1980 and Eisler 1986) and on site-specific abiotic and biological data.

3.4 Site Conceptual Exposure Model

The site conceptual exposure model (SCEM) is the primary output of the Problem Formulation phase of the ERA, and is used to develop a series of testable null hypotheses for the API/PC/KR, primarily those regarding potential exposure scenarios and the relationship between selected assessment and measurement endpoints. The null hypotheses for the API/PC/KR are defined as follows:

- The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the structure or function of the fish populations in the Kalamazoo River and Portage Creek System

- The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the survival, growth, and reproduction of aquatic plant and animal receptors utilizing the Kalamazoo River and Portage Creek system

- The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the survival, growth, and reproduction of semi-aquatic and terrestrial mammalian receptors utilizing the Kalamazoo River and Portage Creek system

- The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the survival, growth, and reproduction of omnivorous and carnivorous avian receptors utilizing the Kalamazoo River and Portage Creek system

The term "sediment" as used in the aforementioned hypotheses refers to both instream and floodplain sediments. The latter can also be termed "floodplain soils", with the recognition that these apparently terrestrial areas are frequently inundated for long durations (in some cases over half the year).

The conceptual model (Figure 3-11) presents the potential exposure pathways for the primary chemical stressors (PCBs) associated with past industrial activities in or near the API/PC/KR site. These pathways indicate how the ecological resources can co-occur or come in contact with hazardous chemicals or materials such as PCB-contaminated sediments, and include contaminant sources, fate, and transport processes, and exposure routes. Some of the pathways shown in Figure 3-11 are considered to be relatively minor, and not all are fully evaluated in this ERA.

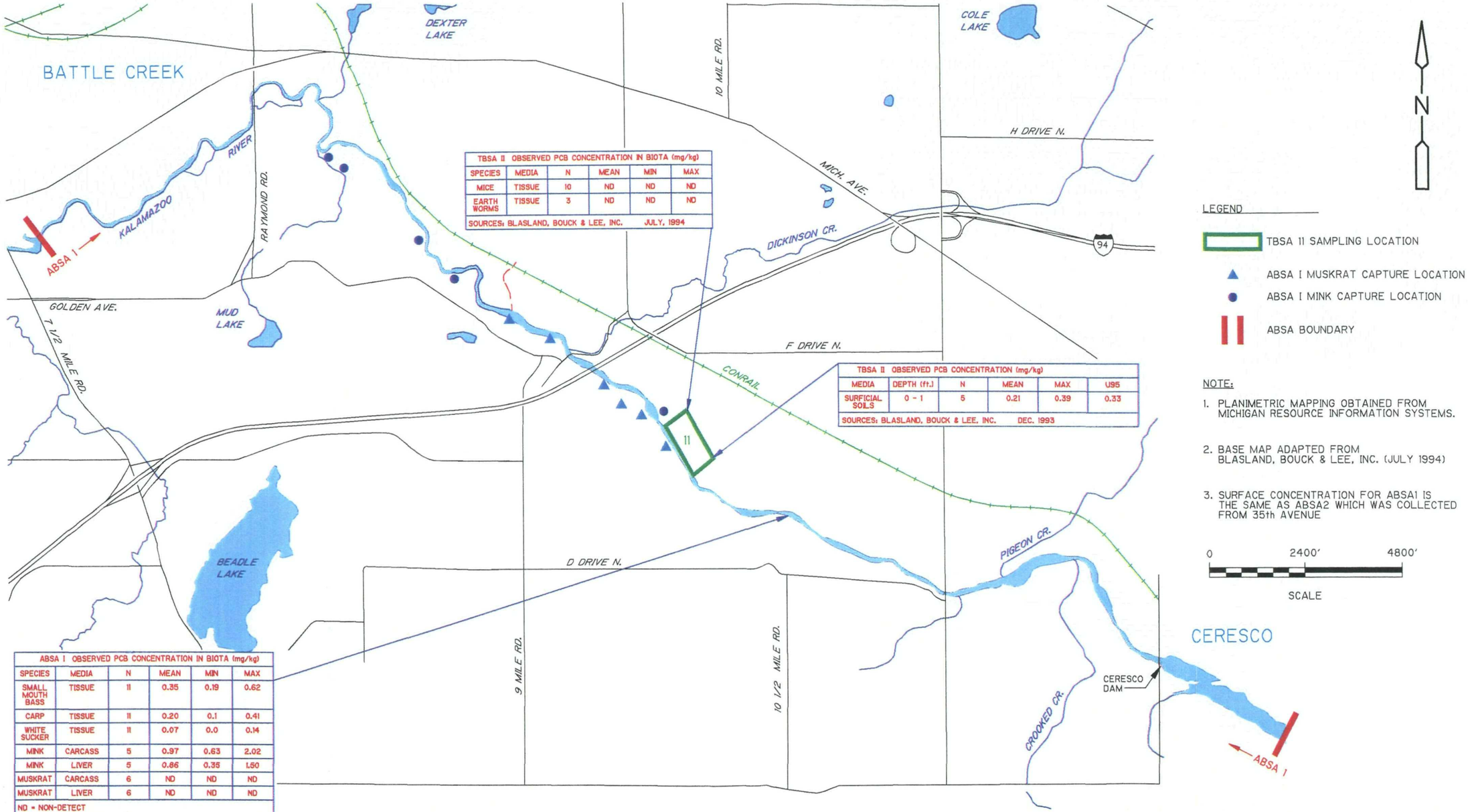
This ERA is focused primarily on assessing population-level risks associated with PCB contamination in abiotic media and biota. Because of the potential for PCBs to accumulate in biological tissues and exert adverse effects in upper trophic level biota, this ERA specifically considers bioaccumulation, food chain effects, and adverse effects in upper trophic level organisms. Reproductive effects in upper trophic level organisms, such as top predators, commonly follow long-term PCB exposure. Since reproductive effects are often observed before other types of effects, protection against reproductive effects should ensure that other adverse effects would not occur. Therefore, reproductive endpoints for top predators are also considered critical to this ERA. Finally, it is assumed in this ERA that population-level effects are most important for most species and that the loss of a single individual is not critical to the population or community. The focus on population-level effects rather than on effects to individual organisms is modified in this ERA for threatened or endangered species. In this case, adverse effects or a loss of even one individual is considered important. Related to the conceptual model are the preliminarily identified remedial action objectives for the API/PC/KR presented in Section 3.3.1. Table 3-3 summarizes the

relationship between assessment endpoints, hypotheses, measurement endpoints, and receptors.

3.5 Uncertainties- Problem Formulation

Uncertainties in Problem Formulation can arise from several sources, most significantly from assumptions used to initially focus the ERA. This ERA is by regulatory direction focused on the primary chemical contaminants identified at this site - PCBs. It is recognized that other chemical stressors have been identified onsite, including some that can be highly toxic and are known to substantially bioaccumulate. It is also recognized that this focused ERA is specifically intended to address PCB contamination at this site.

The major uncertainties in the Problem Formulation phase of the ERA probably stem from the assumptions used to develop the SCEM. The SCEM developed for this ERA is based on a focused ERA in which only key exposure pathways and chemical stressors are fully evaluated. Therefore, uncertainties associated with other minor exposure pathways (e.g., inhalation), or chemical stressors other than PCBs, will not affect the outcome of this focused ERA. All major exposure pathways and pathway components related to PCB contamination at this site have been included in the SCEM. No sources of uncertainty are identified at this stage of the ERA that will substantially affect the outcome of the ERA.



TBSA II OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)

SPECIES	MEDIA	N	MEAN	MIN	MAX
MICE	TISSUE	10	ND	ND	ND
EARTH WORMS	TISSUE	3	ND	ND	ND

SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994

TBSA II OBSERVED PCB CONCENTRATION (mg/kg)

MEDIA	DEPTH (ft.)	N	MEAN	MAX	U95
SURFICIAL SOILS	0 - 1	5	0.21	0.39	0.33

SOURCES: BLASLAND, BOUCK & LEE, INC. DEC. 1993

ABSA I OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)

SPECIES	MEDIA	N	MEAN	MIN	MAX
SMALL MOUTH BASS	TISSUE	11	0.35	0.19	0.62
CARP	TISSUE	11	0.20	0.1	0.41
WHITE SUCKER	TISSUE	11	0.07	0.0	0.14
MINK	CARCASS	5	0.97	0.63	2.02
MINK	LIVER	5	0.86	0.35	1.50
MUSKRAT	CARCASS	6	ND	ND	ND
MUSKRAT	LIVER	6	ND	ND	ND

ND = NON-DETECT

SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994
BLASLAND, BOUCK & LEE, INC. NOV. 1994
USEPA JUNE, 1994

ALLIED PAPER, INC./PORTAGE CREEK/KALAMAZOO RIVER SUPERFUND SITE

KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT
OBSERVED PCB CONCENTRATIONS IN AQUATIC & TERRESTRIAL MEDIA
UPSTREAM REFERENCE AREA NEAR BATTLE CREEK

ABSA 1

Figure No. 3 - 1

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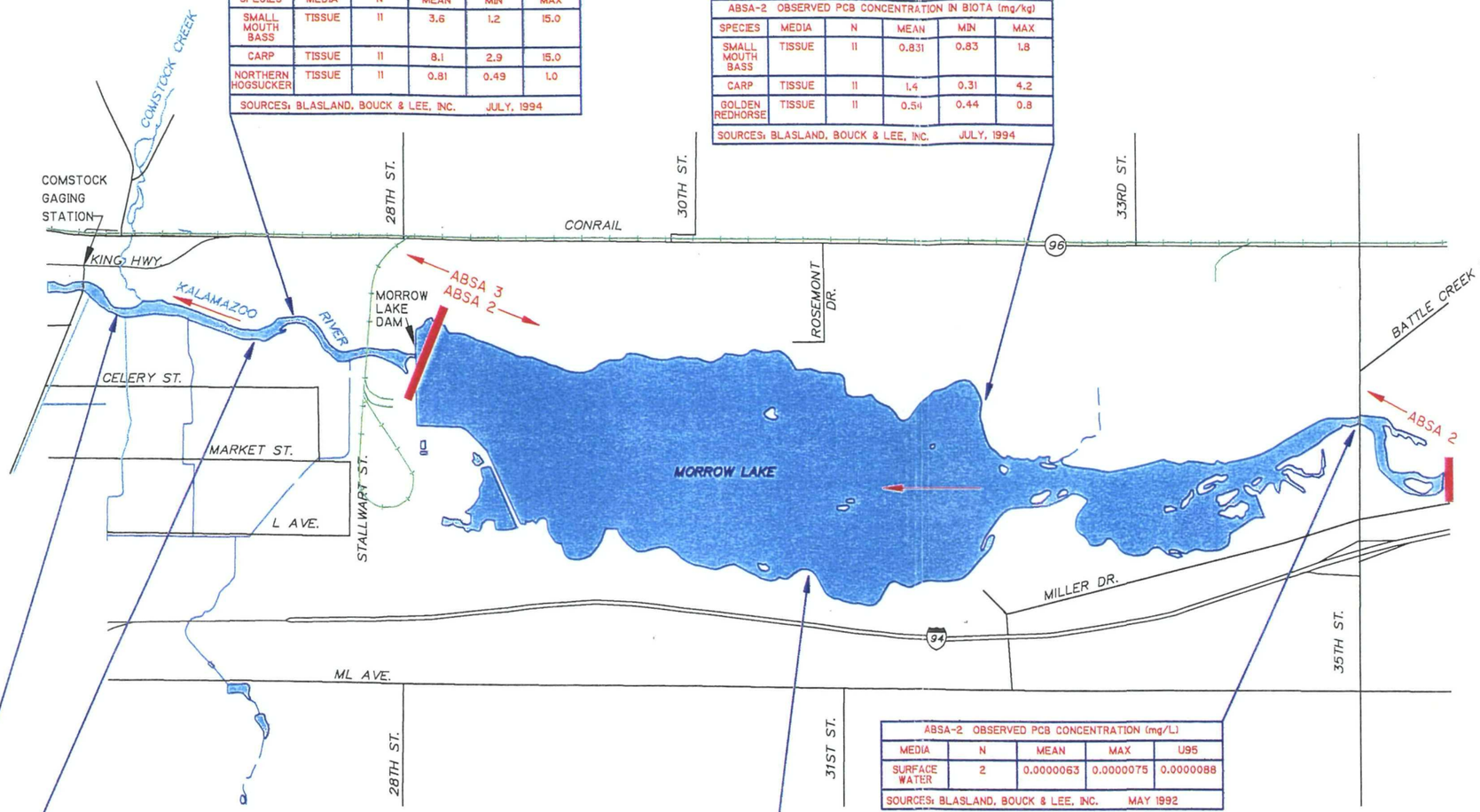
ABSA-3 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)					
SPECIES	MEDIA	N	MEAN	MIN	MAX
SMALL MOUTH BASS	TISSUE	11	3.6	1.2	15.0
CARP	TISSUE	11	8.1	2.9	15.0
NORTHERN HOGSUCKER	TISSUE	11	0.81	0.49	1.0
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994					

ABSA-2 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)					
SPECIES	MEDIA	N	MEAN	MIN	MAX
SMALL MOUTH BASS	TISSUE	11	0.831	0.83	1.8
CARP	TISSUE	11	1.4	0.31	4.2
GOLDEN REDHORSE	TISSUE	11	0.54	0.44	0.8
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994					



ABSA-3 OBSERVED PCB CONCENTRATION (mg/L)				
MEDIA	N	MEAN	MAX	U95
SURFACE WATER	30	0.000015	0.000048	0.000019
SOURCES: BLASLAND, BOUCK & LEE, INC. MARCH 1995				

ABSA-3 OBSERVED PCB CONCENTRATION (mg/kg)				
MEDIA	N	MEAN	MAX	U95
SEDIMENTS	40	2.3	86	6.5
SOURCES: BLASLAND, BOUCK & LEE, INC. APRIL 1994				

ABSA-2 OBSERVED PCB CONCENTRATION (mg/L)				
MEDIA	N	MEAN	MAX	U95
SURFACE WATER	2	0.0000063	0.0000075	0.0000088
SOURCES: BLASLAND, BOUCK & LEE, INC. MAY 1992				

ABSA-2 OBSERVED PCB CONCENTRATION (mg/kg)				
MEDIA	N	MEAN	MAX	U95
SEDIMENTS	20	0.91	2.4	1.2
SOURCES: BLASLAND, BOUCK & LEE, INC. MAY 1995				

N

LEGEND

ABSA BOUNDARIES

NOTE:

1. PLANIMETRIC MAPPING OBTAINED FROM MICHIGAN RESOURCE INFORMATION SYSTEMS.
2. SAMPLING AREAS APPROXIMATED BY BLASLAND, BOUCK & LEE, INC.
3. BASE MAP ADAPTED FROM BLASLAND, BOUCK & LEE, INC. (JULY 1994)

0 2400' 4800'

SCALE:

ALLIED PAPER, INC./PORTAGE CREEK/KALAMAZOO RIVER SUPERFUND SITE

KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT OBSERVED PCB CONCENTRATIONS IN AQUATIC MEDIA MORROW LAKE AREA

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ABSA 2
ABSA 3

Figure No. 3 - 2

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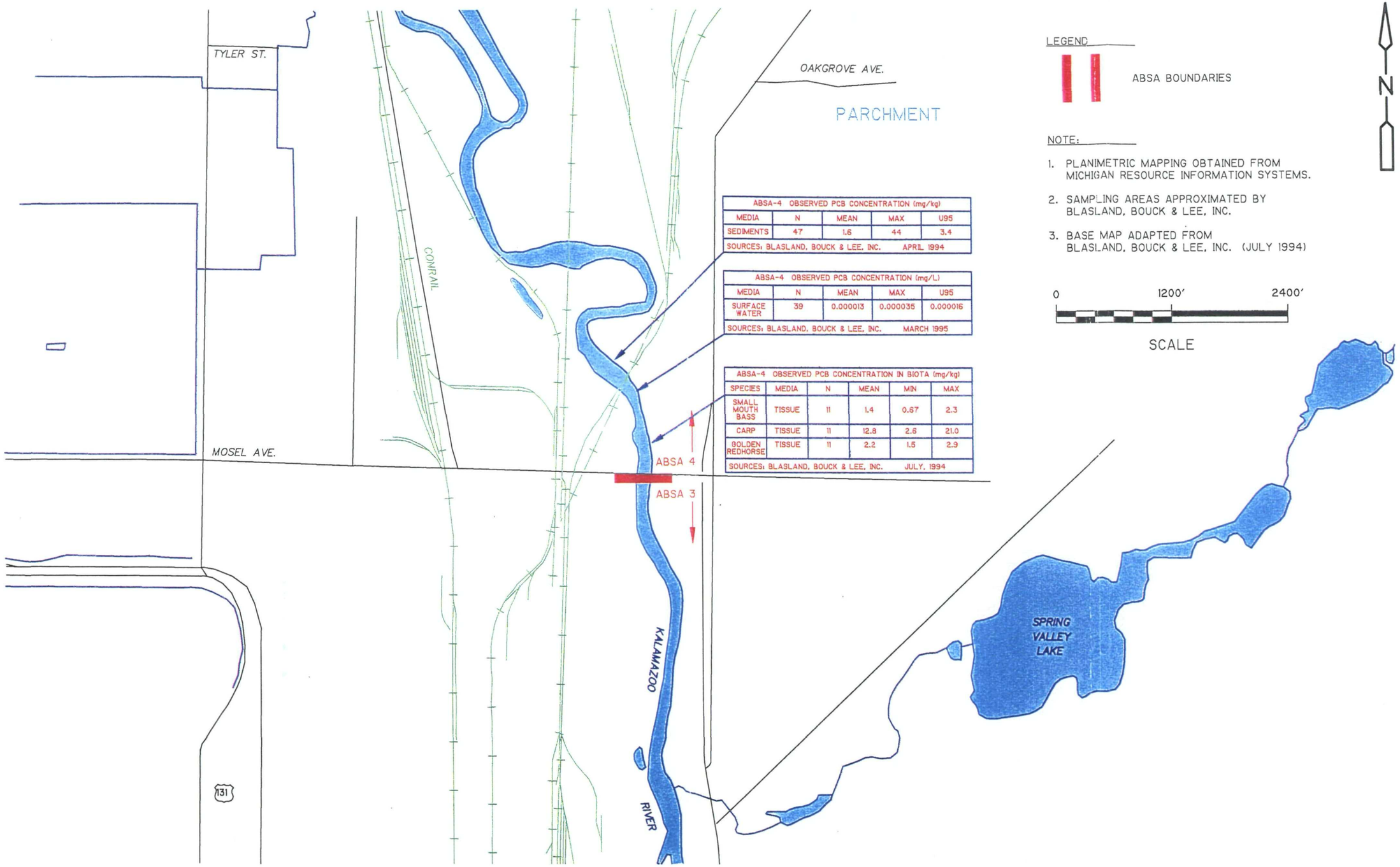

ABSA-4 OBSERVED PCB CONCENTRATION (mg/kg)				
MEDIA	N	MEAN	MAX	U95
SEDIMENTS	47	1.6	44	3.4
SOURCES: BLASLAND, BOUCK & LEE, INC. APRIL 1994				

ABSA-4 OBSERVED PCB CONCENTRATION (mg/L)				
MEDIA	N	MEAN	MAX	U95
SURFACE WATER	39	0.000013	0.000035	0.000016
SOURCES: BLASLAND, BOUCK & LEE, INC. MARCH 1995				

ABSA-4 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)					
SPECIES	MEDIA	N	MEAN	MIN	MAX
SMALL MOUTH BASS	TISSUE	11	1.4	0.67	2.3
CARP	TISSUE	11	12.8	2.6	21.0
GOLDEN REDHORSE	TISSUE	11	2.2	1.5	2.9
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994					

LEGEND

ABSA BOUNDARIES

NOTE:

1. PLANIMETRIC MAPPING OBTAINED FROM MICHIGAN RESOURCE INFORMATION SYSTEMS.
2. SAMPLING AREAS APPROXIMATED BY BLASLAND, BOUCK & LEE, INC.
3. BASE MAP ADAPTED FROM BLASLAND, BOUCK & LEE, INC. (JULY 1994)

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KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT
OBSERVED PCB CONCENTRATIONS IN AQUATIC MEDIA
MOSEL AVENUE AREA

ABSA 4
ABSA 3

Figure No. 3 - 3

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KALAMAZOO RIVER

12TH STREET LANDFILL OU

PLAINWELL DAM

ABSA 6

ABSA 5

BROOKSIDE PARK

131

MAIN ST.

ABSA 5

ABSA 4

PLAINWELL MUNICIPAL WWTP

10

13TH ST.

12TH ST.

PLAINWELL

N

ABSA-5 OBSERVED PCB CONCENTRATION (mg/L)				
MEDIA	N	MEAN	MAX	U95
SURFACE WATER	5	0.000062	0.000091	0.000081
SOURCES: BLASLAND, BOUCK & LEE, INC. MAY 1992				

ABSA-5 OBSERVED PCB CONCENTRATION (mg/kg)				
MEDIA	N	MEAN	MAX	U95
SEDIMENTS	36	6.1	100	41.3
SOURCES: BLASLAND, BOUCK & LEE, INC. APRIL 1994				

ABSA-5 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)					
SPECIES	MEDIA	N	MEAN	MIN	MAX
SMALL MOUTH BASS	TISSUE	11	4.5	2.6	7.9
CARP	TISSUE	11	8.8	4.9	14.0
GOLDEN REDHORSE	TISSUE	11	2.2	1.0	3.1
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY 1994					

TBSA 10 OBSERVED PCB CONCENTRATION (mg/kg)					
MEDIA	DEPTH (ft.)	N	MEAN	MAX	U95
SURFICIAL SOILS	0 - 1	5	6.5	10.2	8.9
SOURCES: BLASLAND, BOUCK & LEE, INC. DEC. 1993					

TBSA-10 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)					
SPECIES	MEDIA	N	MEAN	MIN	MAX
MICE	TISSUE	10	0.09	0.03	0.28
EARTH WORMS	TISSUE	3	0.46	0.13	0.56
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994					

LEGEND

ABSA BOUNDARIES

10 TBSA SAMPLING LOCATION

NOTE:

1. PLANIMETRIC MAPPING OBTAINED FROM MICHIGAN RESOURCE INFORMATION SYSTEMS.
2. SAMPLING AREAS APPROXIMATED BY BLASLAND, BOUCK & LEE, INC.
3. BASE MAP ADAPTED FROM BLASLAND, BOUCK & LEE, INC. (JULY 1994)

0 1200' 2400'

SCALE

ALLIED PAPER, INC./PORTAGE CREEK/KALAMAZOO RIVER SUPERFUND SITE

KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT
OBSERVED PCB CONCENTRATIONS IN AQUATIC AND TERRESTRIAL MEDIA
UPSTREAM OF PLAINWELL DAM

ABSA 4
ABSA 5

Figure No. 3 - 4

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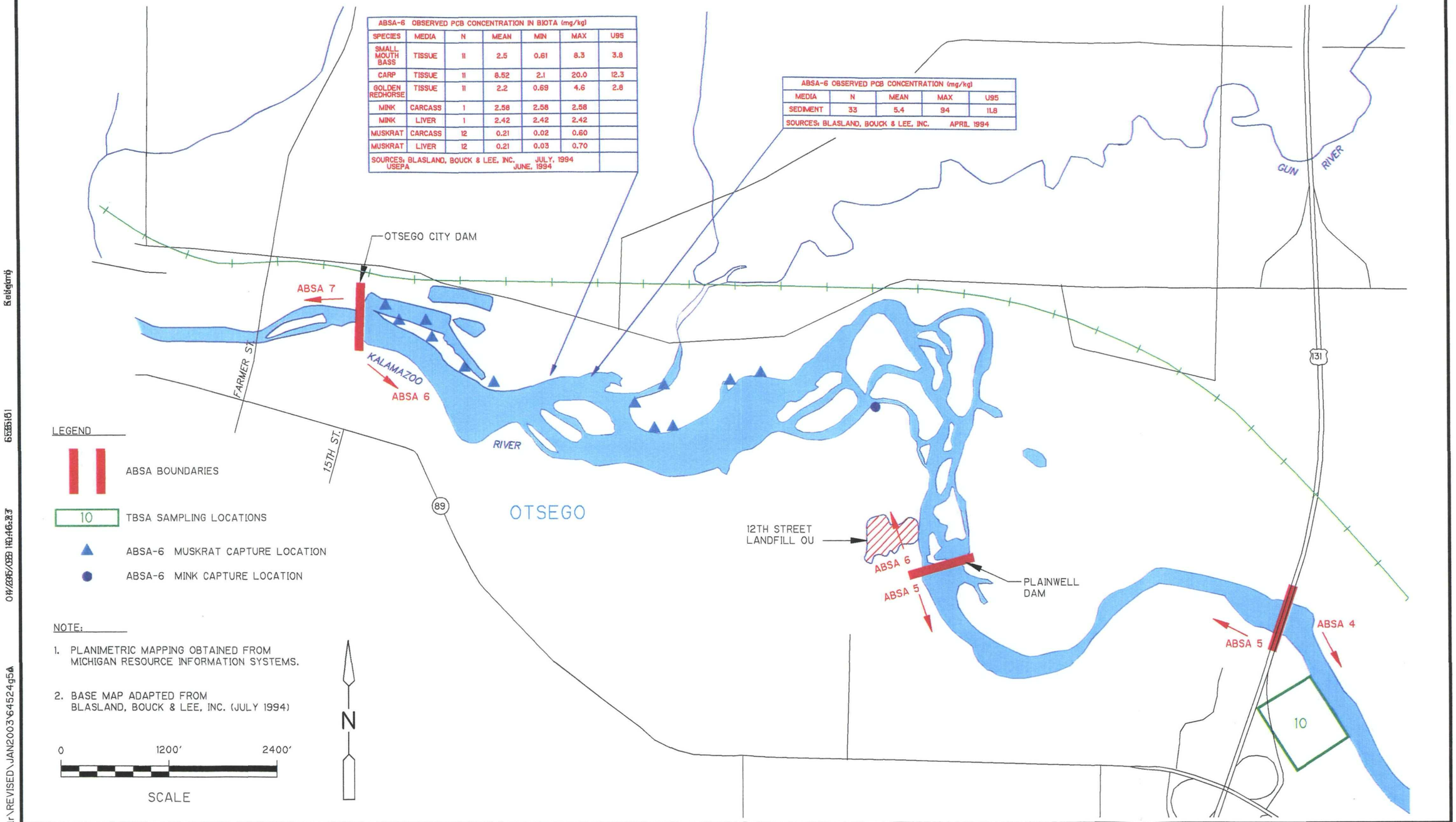
ABSA-6 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)						
SPECIES	MEDIA	N	MEAN	MIN	MAX	U95
SMALL MOUTH BASS	TISSUE	11	2.5	0.61	8.3	3.8
CARP	TISSUE	11	8.52	2.1	20.0	12.3
GOLDEN REDHORSE	TISSUE	11	2.2	0.69	4.6	2.8
MINK	CARCASS	1	2.58	2.58	2.58	
MINK	LIVER	1	2.42	2.42	2.42	
MUSKRAT	CARCASS	12	0.21	0.02	0.60	
MUSKRAT	LIVER	12	0.21	0.03	0.70	
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994 USEPA JUNE, 1994						

ABSA-6 OBSERVED PCB CONCENTRATION (mg/kg)				
MEDIA	N	MEAN	MAX	U95
SEDIMENT	33	5.4	94	11.8
SOURCES: BLASLAND, BOUCK & LEE, INC. APRIL 1994				



ALLIED PAPER, INC./PORTAGE CREEK/KALAMAZOO RIVER SUPERFUND SITE

KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT
OBSERVED PCB CONCENTRATIONS IN AQUATIC & TERRESTRIAL MEDIA
BETWEEN OF OTSEGO CITY DAM AND PLAINWELL DAM

ABSA 5
ABSA 6

Figure No. 3 - 5

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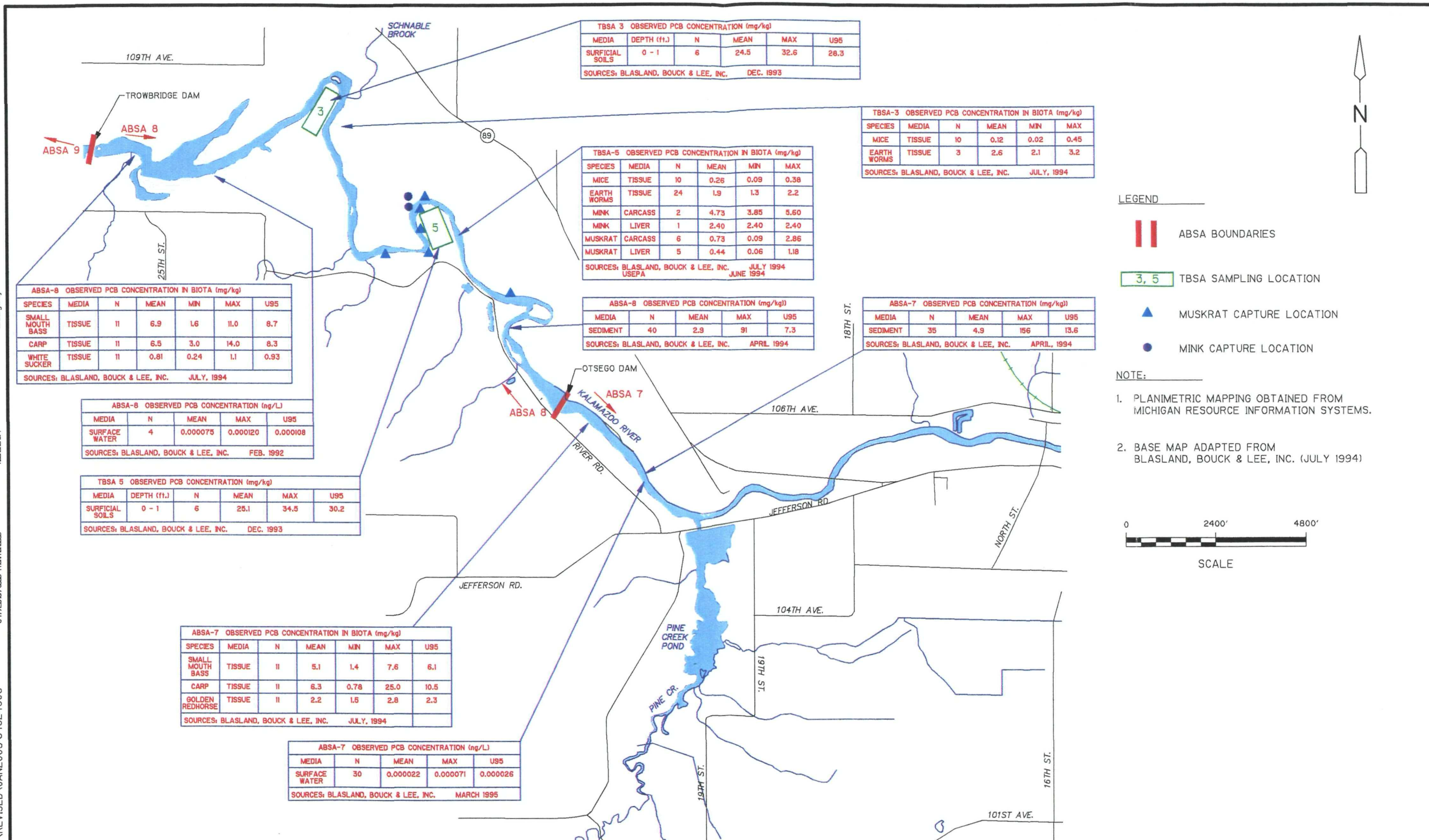

TBSA 3 OBSERVED PCB CONCENTRATION (mg/kg)

MEDIA	DEPTH (ft.)	N	MEAN	MAX	U95
SURFICIAL SOILS	0 - 1	6	24.5	32.6	28.3

SOURCES: BLASLAND, BOUCK & LEE, INC. DEC. 1993

TBSA-3 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)

SPECIES	MEDIA	N	MEAN	MIN	MAX
MICE	TISSUE	10	0.12	0.02	0.45
EARTH WORMS	TISSUE	3	2.6	2.1	3.2

SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994

TBSA-5 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)

SPECIES	MEDIA	N	MEAN	MIN	MAX
MICE	TISSUE	10	0.26	0.09	0.38
EARTH WORMS	TISSUE	24	1.9	1.3	2.2
MINK	CARCASS	2	4.73	3.85	5.60
MINK	LIVER	1	2.40	2.40	2.40
MUSKRAT	CARCASS	6	0.73	0.09	2.86
MUSKRAT	LIVER	5	0.44	0.06	1.18

SOURCES: BLASLAND, BOUCK & LEE, INC. JULY 1994
USEPA JUNE 1994

ABSA-8 OBSERVED PCB CONCENTRATION (mg/kg))

MEDIA	N	MEAN	MAX	U95
SEDIMENT	40	2.9	91	7.3

SOURCES: BLASLAND, BOUCK & LEE, INC. APRIL 1994

ABSA-7 OBSERVED PCB CONCENTRATION (mg/kg))

MEDIA	N	MEAN	MAX	U95
SEDIMENT	35	4.9	156	13.6

SOURCES: BLASLAND, BOUCK & LEE, INC. APRIL, 1994

ABSA-8 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)

SPECIES	MEDIA	N	MEAN	MIN	MAX	U95
SMALL MOUTH BASS	TISSUE	11	6.9	1.6	11.0	8.7
CARP	TISSUE	11	6.5	3.0	14.0	8.3
WHITE SUCKER	TISSUE	11	0.81	0.24	1.1	0.93

SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994

ABSA-8 OBSERVED PCB CONCENTRATION (ng/L)

MEDIA	N	MEAN	MAX	U95
SURFACE WATER	4	0.000075	0.000120	0.000108

SOURCES: BLASLAND, BOUCK & LEE, INC. FEB. 1992

TBSA 5 OBSERVED PCB CONCENTRATION (mg/kg)

MEDIA	DEPTH (ft.)	N	MEAN	MAX	U95
SURFICIAL SOILS	0 - 1	6	25.1	34.5	30.2

SOURCES: BLASLAND, BOUCK & LEE, INC. DEC. 1993

ABSA-7 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)

SPECIES	MEDIA	N	MEAN	MIN	MAX	U95
SMALL MOUTH BASS	TISSUE	11	5.1	1.4	7.6	6.1
CARP	TISSUE	11	6.3	0.78	25.0	10.5
GOLDEN REDHORSE	TISSUE	11	2.2	1.5	2.8	2.3

SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994

ABSA-7 OBSERVED PCB CONCENTRATION (ng/L)

MEDIA	N	MEAN	MAX	U95
SURFACE WATER	30	0.000022	0.000071	0.000026

SOURCES: BLASLAND, BOUCK & LEE, INC. MARCH 1995

NOTE:
1. PLANIMETRIC MAPPING OBTAINED FROM MICHIGAN RESOURCE INFORMATION SYSTEMS.
2. BASE MAP ADAPTED FROM BLASLAND, BOUCK & LEE, INC. (JULY 1994)

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KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT
OBSERVED PCB CONCENTRATIONS IN AQUATIC & TERRESTRIAL MEDIA
TROWBRIDGE DAM AREA UPSTREAM TO OTSEGO

CDM environmental engineers, scientists, planners, & management consultants

ABSA 7
ABSA 8
Figure No. 3 - 6

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ABSA-9 OBSERVED PCB CONCENTRATION (ng/L)				
MEDIA	N	MEAN	MAX	U95
SURFACE WATER	29	0.000020	0.00005	0.000024
SOURCES: BLASLAND, BOUCK & LEE, INC. MARCH 1995				

KALAMAZOO RIVER

ALLEGAN DAM

ABSA 10

ABSA 9

41ST ST.

ALLEGAN DAM RD.

LAKE ALLEGAN

DUMONT CR.

BABYLON RD.

42ND ST.

34TH ST.

39TH ST.

MONROE RD.

36TH ST.

DELAND ST.

40

89

116TH AVE.

N

LEGEND

ABSA BOUNDARIES

NOTE:

1. PLANIMETRIC MAPPING OBTAINED FROM MICHIGAN RESOURCE INFORMATION SYSTEMS.
2. SAMPLING AREAS APPROXIMATED BY BLASLAND, BOUCK & LEE, INC.
3. BASE MAP ADAPTED FROM BLASLAND, BOUCK & LEE, INC. (JULY 1994)

0 2400' 4800'

SCALE

ABSA-9 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)						
SPECIES	MEDIA	N	MEAN	MIN	MAX	U95
SMALL MOUTH BASS	TISSUE	11	6.5	2.7	12.0	8.2
CARP	TISSUE	11	5.6	0.16	21.0	9.0
WHITE SUCKER	TISSUE	11	0.81	0.5	1.7	1.0
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994						

ABSA-9 OBSERVED SEDIMENT CONCENTRATION (mg/kg)				
MEDIA	N	MEAN	MAX	U95
SEDIMENT	36	2.4	7.2	3.1
SOURCES: BLASLAND, BOUCK & LEE, INC. APRIL 1994				

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KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT OBSERVED PCB CONCENTRATIONS IN AQUATIC MEDIA LAKE ALLEGAN

ABSA 9

Figure No. 3 - 7

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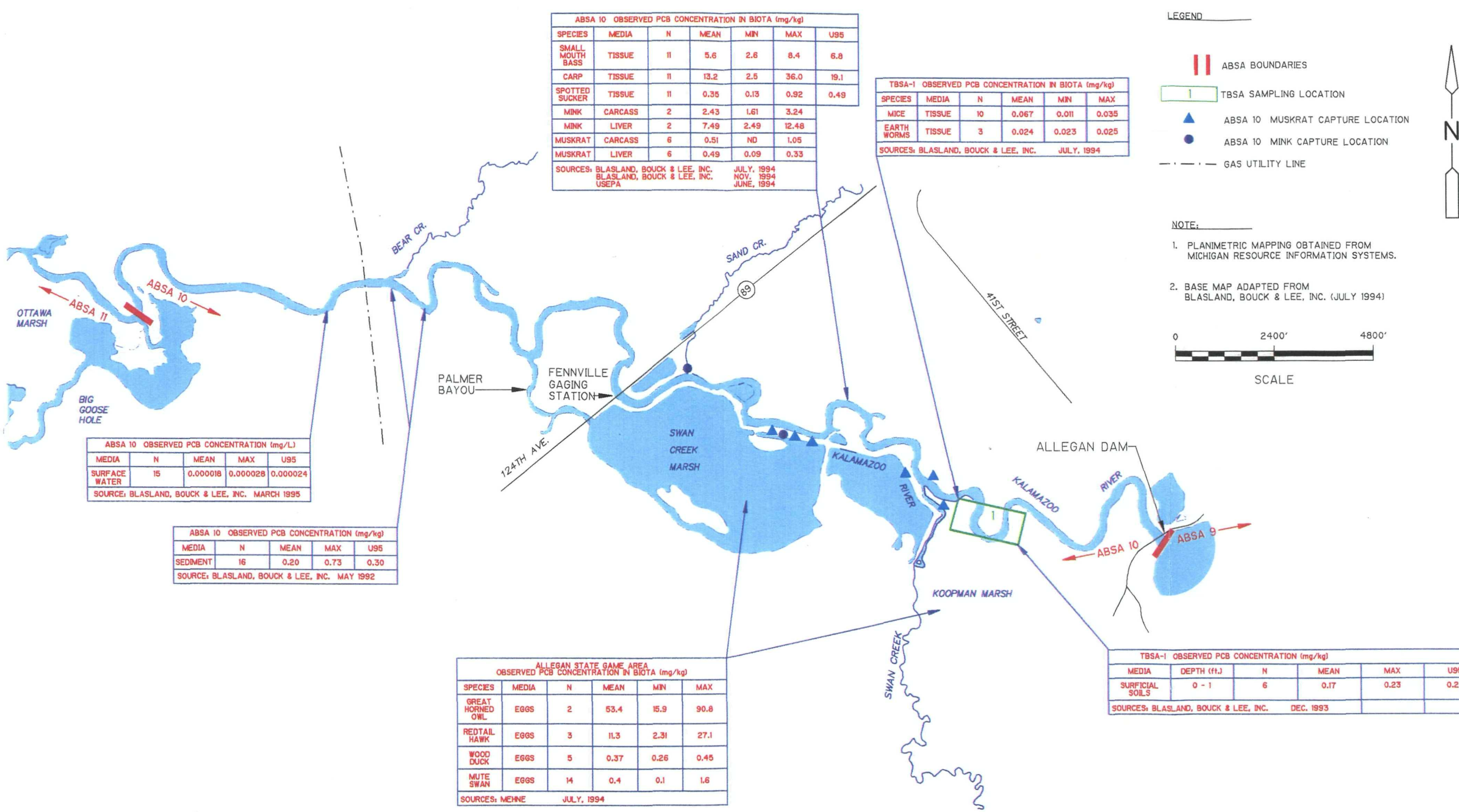

ABSA 10 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)						
SPECIES	MEDIA	N	MEAN	MIN	MAX	U95
SMALL MOUTH BASS	TISSUE	11	5.6	2.6	8.4	6.8
CARP	TISSUE	11	13.2	2.5	36.0	19.1
SPOTTED SUCKER	TISSUE	11	0.35	0.13	0.92	0.49
MINK	CARCASS	2	2.43	1.61	3.24	
MINK	LIVER	2	7.49	2.49	12.48	
MUSKRAT	CARCASS	6	0.51	ND	1.05	
MUSKRAT	LIVER	6	0.49	0.09	0.33	
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994 BLASLAND, BOUCK & LEE, INC. NOV. 1994 USEPA JUNE, 1994						

TBSA-1 OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)					
SPECIES	MEDIA	N	MEAN	MIN	MAX
MICE	TISSUE	10	0.067	0.011	0.035
EARTH WORMS	TISSUE	3	0.024	0.023	0.025
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994					

ABSA 10 OBSERVED PCB CONCENTRATION (mg/L)				
MEDIA	N	MEAN	MAX	U95
SURFACE WATER	15	0.000018	0.000028	0.000024
SOURCE: BLASLAND, BOUCK & LEE, INC. MARCH 1995				

ABSA 10 OBSERVED PCB CONCENTRATION (mg/kg)				
MEDIA	N	MEAN	MAX	U95
SEDIMENT	16	0.20	0.73	0.30
SOURCE: BLASLAND, BOUCK & LEE, INC. MAY 1992				

ALLEGAN STATE GAME AREA OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)					
SPECIES	MEDIA	N	MEAN	MIN	MAX
GREAT HORNED OWL	EGGS	2	53.4	15.9	90.8
REDTAIL HAWK	EGGS	3	11.3	2.31	27.1
WOOD DUCK	EGGS	5	0.37	0.26	0.45
MUTE SWAN	EGGS	14	0.4	0.1	1.6
SOURCES: MEHNE JULY, 1994					

TBSA-1 OBSERVED PCB CONCENTRATION (mg/kg)					
MEDIA	DEPTH (ft.)	N	MEAN	MAX	U95
SURFICIAL SOILS	0 - 1	6	0.17	0.23	0.23
SOURCES: BLASLAND, BOUCK & LEE, INC. DEC. 1993					

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KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT OBSERVED PCB CONCENTRATIONS IN AQUATIC AND TERRESTRIAL MEDIA DOWNSTREAM OF ALLEGAN DAM

ABSA 10

Figure No. 3 - 8

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62ND ST.

ABSA II OBSERVED PCB CONCENTRATION (mg/kg)				
MEDIA	N	MEAN	MAX	U95
SEDIMENT	10	0.27	1.4	0.53
SOURCES: BLASLAND, BOUCK & LEE, INC. MAY 1992				

POTTOWATAMIE MARSH

ABSA II OBSERVED PCB CONCENTRATION (mg/L)				
MEDIA	N	MEAN	MAX	U95
SURFACE WATER	15	0.000059	0.00012	0.000077
SOURCES: BLASLAND, BOUCK & LEE, INC. MAY 1992				

MORRISON BAYOU

KALAMAZOO RIVER

ABSA II OBSERVED PCB CONCENTRATION IN BIOTA (mg/kg)						
SPECIES	MEDIA	N	MEAN	MIN	MAX	U95
SMALL MOUTH BASS	TISSUE	11	2.6	0.87	5.0	3.3
CARP	TISSUE	11	9.0	2.0	32	13.9
WHITE SUCKER	TISSUE	11	1.1	0.78	1.6	1.2
SOURCES: BLASLAND, BOUCK & LEE, INC. JULY, 1994						

58TH ST.

ABSA 11

ABSA 10

MANN CR.

LEGEND

ABSA BOUNDARIES

NOTE:

1. PLANIMETRIC MAPPING OBTAINED FROM MICHIGAN RESOURCE INFORMATION SYSTEMS.
2. SAMPLING AREAS APPROXIMATED BY BLASLAND, BOUCK & LEE, INC.
3. BASE MAP ADAPTED FROM BLASLAND, BOUCK & LEE, INC. (JULY 1994)

0 1200' 2400'

SCALE

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ALLIED PAPER, INC./PORTAGE CREEK/KALAMAZOO RIVER SUPERFUND SITE

KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT OBSERVED PCB CONCENTRATIONS IN AQUATIC MEDIA POTTOWATAMIE MARSH AREA

ABSA 11

Figure No. 3 - 9

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MICHIGAN AVENUE PORTAGE CREEK	
DATE	PCBs (mg/kg)
8/76	0.50
11/72	85
7/83	15
7/83	15
7/83	15

PORTAGE STREET	
DATE	PCBs (mg/kg)
7/83	10
7/83	11

VINE STREET	
DATE	PCBs (mg/kg)
8/76	56
1976	56

LAKE STREET	
DATE	PCBs (mg/kg)
7/72	25
7/72	12
1972	120

STOCKBRIDGE AVENUE	
DATE	PCBs (mg/kg)
7/72	19

REED AVENUE	
DATE	PCBs (mg/kg)
7/72	25

ALCOTT STREET	
DATE	PCBs (mg/kg)
7/72	26

MICHIGAN AVENUE KALAMAZOO RIVER	
DATE	PCBs (mg/kg)
8/76	7.7
7/82	< 0.05

KING HIGHWAY	
DATE	PCBs (mg/kg)
7/82	< 0.05

BRYANT STREET	
DATE	PCBs (mg/kg)
7/72	12

ABSA 12 OBSERVED PCB CONCENTRATION (mg/kg)						
SPECIES	MEDIA	N	MEAN	MIN	MAX	U95
CARP	FILLET	11	3.42	1.5	8.80	4.68
CARP	CARCASS	6	8.13	2.4	12.0	11.27
WHITE SUCKER	WHOLE BODY	7	1.42	0.23	2.40	1.88
SOURCES: BLASLAND, BOUCK & LEE, INC. MAY 1998						

CONFLUENCE
MICHIGAN AVE.
RED ARROW PARK
RIVERVIEW PARK
KING HY.
PKWY
VINE ST.
LAKE ST.
STOCKBRIDGE
TOWN
CROSS
S. MILLS ST.
REED ST.
BRYANT ST.
ALCOTT ST.
MILLER ST.
CORK ST.
PORTAGE PAPER MILL
PORTAGE CREEK
WESTNEDGE AVE.
BURDICK ST.
PORTAGE ST.
MONARCH MILL POND
CHARLES AVE.
SPRINKLER D.
COMSTOCK CREEK
COMSTOCK GAGING STATION
KING HWY.
KALAMAZOO RIVER
AVE.
COMSTOCK
CELERY ST.
RIVER ST.
MARKET ST.
DAVIS CREEK
KING HIGHWAY LANDFILL
FORMER KING MILL PROPERTY
WILLOW BOULEVARD / A-SITE
GEORGIA-PACIFIC CORPORATION KALAMAZOO MILL
FORMER BRYANT MILL POND AREA
ALLIED PAPER INC.
FORMER MONARCH MILL PROPERTY

1" = 2000'
1000 0 2000

LEGEND

- WATER COLUMN SAMPLE
- SURFACE SEDIMENT SAMPLE (mg/kg) (CONCENTRATION)

NOTE:

1. PLANIMETRIC MAPPING OBTAINED FROM MICHIGAN RESOURCE INFORMATION SYSTEMS.
2. BASE MAP ADAPTED FROM BLASLAND, BOUCK & LEE, INC. (FEB, 1991)

ALLIED PAPER, INC./PORTAGE CREEK/KALAMAZOO RIVER SUPERFUND SITE

KALAMAZOO RIVER ECOLOGICAL RISK ASSESSMENT
OBSERVED PCB CONCENTRATIONS IN AQUATIC & TERRESTRIAL MEDIA
PORTAGE CREEK AREA

ABSA 12

Figure No. 3 - 10

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FIGURE 3-11
POTENTIAL EXPOSURE SCENARIOS
SITE CONCEPTUAL MODEL
API/PC/KR

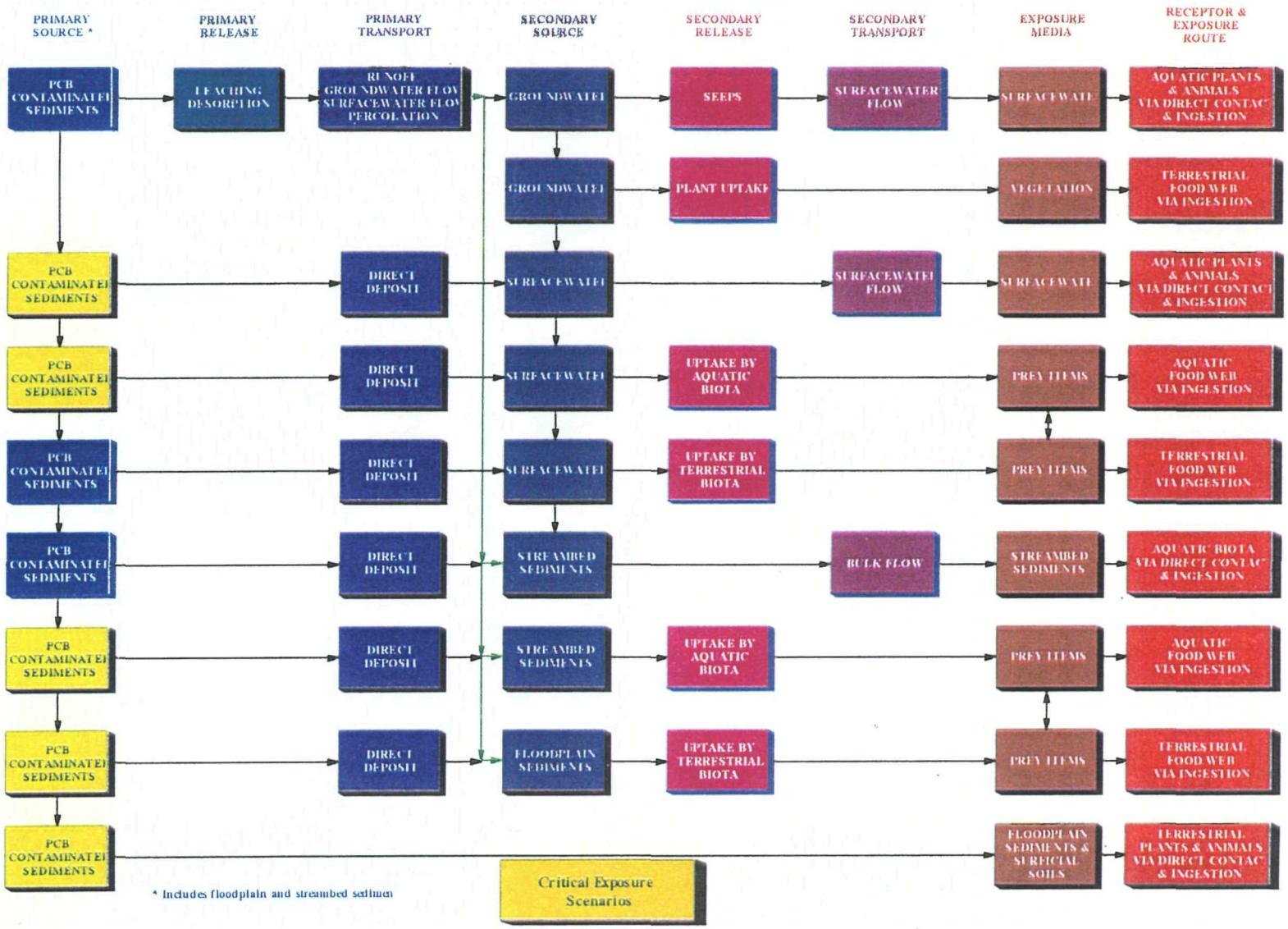


Table 3-1
Polychlorinated Biphenyls (PCBs) Detected in Abiotic and Biological Samples
API/PC/KR

PCBs	Media of Concern
Aroclor 1260	SW, SED, FP SED, SS, BIO
Aroclor 1254	SW, SED, FP SED, SS, BIO
Aroclor 1248	SW, SED, FP SED, SS, BIO
Aroclor 1242	SW, SED, FP SED, SS, BIO
Aroclor 1232	SW, SED, FP SED, SS
Aroclor 1221	SW, SED, FP SED, SS
Aroclor 1016	SW, SED, FP SED, SS, BIO

SW	Surface Water
SED	Streambed Sediment
FP SED/SS	Floodplain Sediment/Surface Soil (sediments deposited within 100-ear floodplain)
SS	Surface Soil (from soil samples taken from terrestrial biological study areas (TBSAs))
BIO	Biological tissue

Table 3-2
Biological Study Areas
API/PC/KR

ABSA 1	Kalamazoo River upstream of the city of Battle Creek (upstream reference site). Aquatic biota were collected near the I-94 junction with the Kalamazoo River. Includes TBSA 11. (See Figure 3-1).
ABSA 2	Kalamazoo River from the downstream boundary of ABSA 1 to Morrow Lake Dam. Aquatic biota were collected from Morrow Lake. (See Figure 3-2).
ABSA 3	Kalamazoo River from Morrow Dam to Mosel Ave., Kalamazoo. Aquatic biota were collected just downstream of Morrow Dam. (See Figure 3-2).
ABSA 4	Kalamazoo River at Mosel Ave. to Hwy. 131 bridge. Aquatic biota were collected from the Kalamazoo River near Mosel Avenue. (See Figure 3-3).
ABSA 5	Kalamazoo River near Hwy 131 bridge and Plainwell Dam. Aquatic biota were collected from the Kalamazoo River upstream of Plainwell Dam. Includes TBSAs 8, 9 and 10. (See Figures 3-4).
ABSA 6	Kalamazoo River from Plainwell Dam to Otsego City Dam. Aquatic biota were collected from the Kalamazoo River upstream of Otsego City Dam. Includes TBSA 10. (See Figures 3-5).
ABSA 7	Kalamazoo River from Otsego City Dam to Otsego Dam. Aquatic biota were collected just upstream of Otsego Dam. (See Figure 3-6).
ABSA 8	Kalamazoo River from Otsego Dam to Trowbridge Dam. Aquatic biota were collected upstream of Trowbridge Dam. Includes TBSAs 3 and 5. (See Figures 3-6).
ABSA 9	Kalamazoo River from Trowbridge Dam to Lake Allegan Dam. Aquatic biota were collected from Lake Allegan. (See Figure 3-7).
ABSA 10	Kalamazoo River from Lake Allegan Dam to Ottawa Marsh. Aquatic biota were collected downstream of Allegan Dam. Includes TBSA 1. (See Figure 3-8).
ABSA 11	Kalamazoo River from Ottawa Marsh to US 31. Aquatic biota were collected near Saugatuck. (See Figure 3-9).
ABSA 12	Portage Creek (See Figure 3-10).

Table 3-3
Assessment and Measurement Endpoints and ERA Null Hypotheses
API/PC/KR

Assessment Endpoint	ERA Null Hypotheses	Measurement Endpoints	Representative Receptor / Group
Preservation of the fish populations (e.g., smallmouth bass, white sucker, and carp) and communities utilizing the Kalamazoo River and Portage Creek system	*The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the structure or function of the fish populations in the Kalamazoo River and Portage Creek System.*	*Toxicity data* - Surface water and sediment total PCB concentrations affecting the survival, growth, or reproduction of fish	Carp Smallmouth bass Sucker
Preservation of the survival, growth, and reproductive capacity of aquatic receptors (e.g., benthic macroinvertebrates, fish, larval amphibians) utilizing the Kalamazoo River and Portage Creek system	*The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the survival, growth, or reproduction of plant and animal aquatic receptors utilizing the Kalamazoo River and Portage Creek system.*	*Toxicity data* - Surface water and sediment total PCB concentrations affecting the survival, growth, or reproduction of aquatic plants, fish, aquatic invertebrates, or larval amphibians	Aquatic plants Benthic invertebrates Fish Larval amphibians
Preservation of the survival, growth, and reproductive capacity of mammalian receptors (e.g., mouse, mink, muskrat, red fox) utilizing the Kalamazoo River and Portage Creek system	*The levels of PCBs in water, sediment, soil, and biota are not sufficient to adversely affect the survival, growth, or reproduction of mammalian receptors utilizing the Kalamazoo River and Portage Creek system.*	*Toxicity data and biota PCB concentrations* - Sediment, surface soil, and dietary item total PCB concentrations affecting the survival, growth, or reproduction of omnivorous and carnivorous mammals	Earthworm (dietary item) White-footed / deer mouse Muskrat Mink Red fox
Preservation of the survival, growth, and reproductive capacity of avian receptors (e.g., bald eagle and great-horned owl) utilizing the Kalamazoo River and Portage Creek system	*The levels of PCBs in water, sediment, and biota are not sufficient to adverselyl affect the survival, growth, or reproduction of avian receptors utilizing the Kalamazoo River and Portage Creek system.*	*Toxicity data and biota PCB concentrations* - Sediment, surface soil, and dietary item total PCB concentrations affecting the survival, growth, or reproduction of omnivorous and carnivorous birds	American robin Great horned owl Bald eagle

Section Four

Section 4
Analysis Phase

This phase of the ERA analyzes exposure data (Exposure Assessment) and effects data (Effects Assessment) for the major stressors (PCBs) and representative receptors previously identified in Problem Formulation.

4.1 Ecological Exposure Assessment

Exposure Assessment evaluates and summarizes available exposure data, including exposure-related data on potential ecological receptors. The primary output of exposure assessment is an exposure profile that presents the magnitude (e.g., concentration) and distribution (e.g., surface water, sediment) of stressors to which ecological receptors may be exposed. For this ERA, the primary chemical stressors are PCBs because of the magnitude and extent of PCB contamination onsite. This focused ERA recognizes that other potential chemical stressors have been identified in the environment, but considers these other chemical stressors to be of much less ecological concern (i.e., much lower risk) than PCBs. Exposure profiles serve as input into the final stage of risk assessment, Risk Characterization.

4.1.1 Exposure Profiles - PCBs

Exposure Profiles describe the magnitude and distribution of stressors identified in the Problem Formulation phase. Exposure profiles for PCBs are summarized in Tables 4-1 and 4-2. Table 4-1 includes the sitewide range of total PCB concentrations and identifies the individual Aroclors for which abiotic media were sampled. Table 4-2 includes summary data on important chemical properties (i.e., environmental persistence, bioavailability, and bioconcentration potential) for PCBs. Non-chemical stressors are discussed in Section 4.1.2.

Recently collected data considered useable for risk assessment purposes are used to describe the magnitude and distribution of PCBs in the API/PC/KR environment. The majority of the abiotic (i.e., sediment, water, surface soil) data used in this ERA are from 1993 and 1994, when most of the biological sampling was conducted. Some floodplain sediment/soil samples collected during this time period were achieved under stable conditions and analyzed in 1997. The floodplain sediment/soil database used in this ERA is based on data from samples collected in 1993 and 1994, including those analyzed in 1997. Where data gaps have been identified, they have been addressed with data collected before 1993 and rarely after 1994. For example, data on PCB concentrations in plants were collected in 2000. In nearly all cases where pre-1993 were used, they were taken from the Description of the Current Situation (BBL 1992). With the exception noted above, data collected since 1994 are not included in the ERA because it is important to compare abiotic and biological data from the same time period to the extent possible. The extensive aquatic and terrestrial biological sampling conducted in 1993 serves as the basis for this ERA. Abiotic data collected in 1993 and 1994 are therefore considered most useful for comparison purposes. Such data are used in this ERA except where important data gaps are identified. The

relationships between biological data and abiotic data are established or estimated only for those ABSAs associated with 1993/1994 data. Where such data are lacking for a location or an abiotic media type, relationships are not established. These relationships include the derivation of soil/water partition factors, bioconcentration factors (BCFs), and biota-sediment accumulation factors (BSAFs).

Although no single concentration value can truly represent the variability of chemical concentrations measured in each medium of concern, the arithmetic mean value best represents the average concentration to which API/PC/KR receptors may be exposed. Where sufficient data have been collected, the arithmetic mean represents the average exposure concentration and the upper 95th confidence limit of the arithmetic mean (U95) is often used to represent a reasonable maximum exposure. Support for using U95 values is found in EPA guidance (1992b) for calculating values that are most representative of the higher end of actual chemical concentrations in environmental media to which human or ecological receptors may be exposed. This guidance states, however, that calculation of U95 values is appropriate only when sufficient data are available. In some cases, insufficient data have been collected from each individual sampling location to allow for complete confidence in U95 values. In cases where data are minimal, calculated U95 values sometimes exceed maximum detected concentrations.

Sufficient data for calculating U95 values have been collected for most abiotic and some biological media (e.g., fish). U95 values are therefore used to represent exposure concentrations in abiotic media and for those biological data associated with sufficient data. The latter category includes whole body fish data. Arithmetic mean and maximum PCB concentrations in most media are also presented in this section for comparison purposes. Arithmetic means include non-detect (ND) data using two accepted methods based on the source of the data. Means of abiotic data collected in 1994 are derived using a randomly selected number between zero and the laboratory reported detection limit to represent non-detects. In the few cases where older abiotic data are used, means are derived using the EPA-recommended method where half the detection limit is used to represent non-detects.

In cases where data are insufficient for deriving confident U95 values (e.g., mink, earthworms, mouse, and muskrat), maximum detected values are used because they probably best represent reasonable maximum exposures. This is especially true where, because data are limited, the true maximum exposure concentrations are unlikely to have been measured. This approach is scientifically defensible considering data limitations, and in fact follows guidance provided by state and federal regulatory agencies. For the most part, however, U95 values are considered representative of reasonable maximum exposure concentrations and are preferred where data quantity allows confidence in the derived values.

Finally, because this ERA is not based on a single line of evidence or single exposure point concentrations, the distribution of potential exposure concentrations associated

with abiotic media is also considered important. For this reason, the arithmetic mean, U95, and maximum concentration of PCBs in abiotic media are also compared to relevant effects concentrations to additionally describe risks. These descriptions are presented graphically in Section 5 (Risk Characterization) for PCBs in surface water, streambed and floodplain sediment, and surface soil for each of the defined sampling areas. These graphical presentations (Figures 5-1 to 5-4) present total PCB concentrations for each abiotic media type overlaid with relevant media-specific effects concentrations, criteria, or thresholds.

Table 4-1 presents the sitewide (non-reference) and reference area ranges of total PCB concentrations detected in abiotic media. Table 4-2 presents important chemical properties for the PCBs identified at the API/PC/KR. Each of these properties is discussed below.

Environmental Persistence

Environmental persistence indicates whether a chemical is likely to be long-lasting in the environment or, alternatively, be degraded by natural processes. Higher chlorinated PCBs, i.e., those with five or more chlorine atoms, are more persistent in the environment than those with three or less chlorine atoms (Eisler 1986). PCBs in sediments (including floodplain sediments) at the API/PC/KR site are the higher chlorinated Aroclors.

Bioconcentration Potential

Bioconcentration potential indicates whether a chemical is likely to be retained in biological tissues after it is taken in by ingestion or other means. Retention of chemicals is not in itself an appropriate measurement endpoint unless it is associated with adverse ecological effects. Retention is, however, useful for verifying exposure and for evaluating bioavailability and the potential for food chain/food web effects. BCFs, derived under equilibrium conditions, are often used as screening-level data to evaluate bioconcentration potential. BCFs are based on the ratio of contaminant concentration in aquatic biota to contaminant concentration in water. Because BCFs are derived under equilibrium conditions and under relatively long exposure durations, they consider both uptake and elimination (depuration) rates. Chemicals with BCFs greater than 300 generally indicate a potential to bioconcentrate (EPA 1991). Chemicals with log BCFs above 3 (BCFs above 1,000) are considered to have significant potential to bioaccumulate (EPA 1992b). For this ERA, available freshwater BCFs for invertebrates and fish that have potential to occur in the API/PC/KR site, or those that are closely related to indigenous species, are used to evaluate bioconcentration potential. In addition, degree of chlorination for individual Aroclors is commonly used to estimate bioconcentration potential.

Bioavailability

For this ERA, bioavailable chemicals are defined as those that exist in a form that has the ability to cause adverse ecological effects or bioaccumulate. As stated previously, bioaccumulation may not in itself constitute a significant ecological effect, but

provides important evidence of both exposure and potential for causing adverse effects to multiple trophic levels under certain conditions. For example, some lipophilic chemicals, such as PCBs, are taken up by biota and are stored in fatty tissues with no apparent ill effects. However, under stressful conditions, such as during winter when only poor quality foods are available, these fats are metabolized and the contaminants can then cause adverse effects.

Chemical properties (e.g., degree of chlorination) or environmental conditions (e.g., high levels of dissolved and particulate organic carbon) can affect the potential bioavailability and toxicity of many chemicals, including PCBs. The bioavailability and, therefore, toxicity of some PCBs in surface water can be influenced by the concentration of dissolved organic carbon. In addition, sediment organic carbon content, measured as total organic carbon (TOC), apparently affects bioavailability and toxicity of some PCBs. For some chemicals, chemical form and thus toxicity can change rather rapidly under changing environmental conditions (e.g., fluctuations in pH, temperature, or surface water flow). Seasonal conditions such as snowmelt and rainfall are likely to affect bioavailability of PCBs in the API/PC/KR. For the most part, however, PCB bioavailability (and potential toxicity) is expected to remain fairly stable because PCBs bind strongly to organic particulate matter. Once taken up by animals, PCBs are likely to be stored predominately in fatty tissues. PCB analyses of biological tissues generally measure Aroclor 1254 and (especially) Aroclor 1260. This finding is supported by studies that show biological conversion of one Aroclor to another after uptake. The chemical mixtures found in abiotic exposure media show little resemblance to Aroclors measured in biological tissues (Eisler 1986). The finding that PCBs have been detected in the tissues of all sampled biota comprising multiple trophic levels at concentrations exceeding important thresholds supports the preliminary assumption that PCBs at this site are indeed bioavailable.

4.1.2 Exposure Profiles - Non-chemical Stressors

Although not the focus of Superfund risk assessments, non-chemical stressors such as disturbed habitats can also affect ecological receptors. Such stressors can therefore be important components of exposure profiles. Non-chemical stressors identified for the API/PC/KR include multiple impacts due to urbanized settings, and may include siltation of instream substrates, historical damming of Portage Creek and the Kalamazoo River, and disturbed riparian/terrestrial habitats adjacent to both the creek and the river. These physical stressors occur throughout the API/PC/KR site to limited degrees, but the extent and severity of such impacts are expected to minimal when compared to the wide ranging impacts of exposure to PCBs. The potential effects of these non-chemical stressors are discussed in Effects Characterization (Section 4.2) of the ERA.

4.1.3 Exposure Scenarios

Exposure-related information for each of the representative groups of organisms previously identified as potential receptors for this ERA is described in this section.

These descriptions are based on likely exposure scenarios preliminarily identified in the SCEM developed in the Problem Formulation phase of the ERA. These preliminary exposure scenarios are refined for the major representative receptors or receptor groups previously identified.

The receptor groups are represented by organisms identified in Section 3.2.3, and include those that are presently being exposed or have potential to be exposed under current conditions. Exposure scenarios, summarized in Table 4-3, are simplified descriptions of how potential receptors or representative receptor groups may come in contact with previously identified stressors.

As presented in Table 4-3, some organisms or representative groups of organisms can be exposed to contaminants by direct uptake (through or on roots of plants) or by ingestion of contaminated media and/or prey. Estimates of plant uptake are most appropriately based on site-specific soil-to-plant transfer factors for the specific plant species and tissues (e.g., fruits) likely to be consumed. Species-specific plant data are limited, however, and do not include a wide variety of plant species or tissues likely to be eaten by representative receptors such as mouse, muskrat, or fox. Daily intake rates for representative animals are most appropriately calculated using site-specific data (e.g., contaminant concentrations in food items and dietary composition). Site-specific data related to diet of consumers and certain other critical input parameters are, however, unavailable for this ERA. Daily intake rates for terrestrial animals are therefore based on literature values for dietary intake and site-specific tissue data where such data exist.

Although several potential exposure scenarios can be identified for ecological receptors, it is most appropriate to focus the assessment on critical exposure scenarios. This ERA is focused on the most critical exposure scenarios identified in the SCM (Figure 3-11). Critical exposure scenarios are discussed below.

Aquatic Exposures

The primary PCB-related risks for aquatic organisms are likely to be from direct contact with and ingestion of contaminated surface water (including suspended sediments) in areas where surface water PCB concentrations are elevated. In addition, ingestion of bottom sediment and sediment pore (interstitial) water with elevated PCBs poses risks to benthic invertebrates, bottom-dwelling fish, and to varying extents, other aquatic biota.

Finally, aquatic organisms that occupy upper trophic levels can be adversely affected by ingesting PCB-contaminated prey. The relative contribution from each exposure source (surface water, sediment, interstitial water, prey) to overall aquatic exposure to PCBs cannot, however, be reliably determined for most aquatic organisms because data describing the variability in factors that can affect total exposure are lacking. These factors can include intraspecific and interspecific differences in life stage, season, diet, ingestion rate, specific habitat, etc. This assessment evaluates potential

risks posed to aquatic biota primarily by comparing ambient PCB concentrations in surface water and streambed sediment to media-specific criteria, such as chronic ambient water quality criteria (AWQC) and critical effects concentrations (e.g., no or low observed adverse effects concentrations) for appropriate species.

Semi-Aquatic and Terrestrial Exposures

Because PCBs tend to bioconcentrate to a high degree and biomagnify, ingestion of contaminated surface water and surface soil by terrestrial animals is expected to be less significant than ingestion of contaminated food. The uptake of chemical contaminants by terrestrial plants can also be important if the contaminants of concern are easily taken up, phytotoxic, or can cause food chain effects to herbivorous consumers. The importance of the food-ingestion pathway and uptake by terrestrial plants depends, however, on the types and abundance of plant and animal receptors as well as on the types and concentrations of chemical contaminants present. Terrestrial/riparian wildlife are common along the API/PC/KR, even though riparian and terrestrial habitats have been visibly degraded in some areas. Significant potential, therefore, exists for terrestrial and riparian species to be exposed to PCB contamination.

Terrestrial/riparian plant communities along the API/PC/KR have been affected by past industrial activities and other human-induced stresses. In some areas containing PCB residual material (e.g., A-Site) the effects are sufficiently limiting to preclude the existence of vegetation, and in other areas existing plant communities are dominated by "weedy" type forbs and shrubs. The causes of observed stress on certain plant communities has not been determined, but may be the result of physical (e.g., habitat alteration) or chemical (contamination/toxicity) stress.

Most herbivorous wildlife species are unlikely to frequent the few barren areas observed; however, those areas dominated by weedy forbs may be an attraction to certain receptors within the API/PC/KR area. Several terrestrial/riparian vertebrate species common in western Michigan that require suitable vegetative cover and other specific habitat requirements (e.g., muskrat and white-footed mouse) are commonly observed within all or most portions of the API/PC/KR area. Although suitable habitat for mink is available throughout most of the API/PC/KR area, populations appear depressed based on mink trapping results.

Because vegetation is only rarely absent or visibly stressed within the API/PC/KR area, and because herbivorous wildlife are common, plant consumers can be exposed to site-related contaminants (e.g., PCBs) under present conditions. Similarly, most predators or consumers of herbivorous species can also be exposed to site-related contaminants because adequate cover and prey are generally available.

Although a large variety of commonly observed terrestrial animal species including resident and migratory birds have been reported onsite, certain other local types of animals species that are not easily observed or often reported probably also occur

regularly or permanently within the API/PC/KR area. These include macroinvertebrates (e.g., insects, spiders, centipedes, millipedes), amphibians (e.g., toads, Ranid frogs, tree frogs, salamanders, newts), reptiles (e.g., lizards, snakes, turtles), and mammals (e.g., shrews, raccoons, voles, skunks, weasels, etc.) and are summarized in the tables in Appendix A. Although for the most part data are lacking, risks to these organisms could occur as a result of direct contact with or ingestion of contaminants via surface water, sediment, soil, and food items. For many terrestrial ecological receptors exposed to PCBs, the most important pathway involves ingestion of PCB-contaminated prey. Finally, PCB exposures are likely to be limited in areas with insufficient cover and prey because such areas are probably avoided by most terrestrial species.

Portions of the API/PC/KR riparian habitat have been reduced by commercial, industrial, and residential development. Many resident species have apparently adapted to the encroachment of humans and these species can therefore be found in close proximity of the landfills and abandoned industrial facilities along the Kalamazoo River and Portage Creek.

Exposures via Food Chain Transfer

PCBs detected onsite have been in the environment for some time, and as a result are considered weathered. Weathered PCBs are comprised of various combinations of different PCB congeners that differ in their environmental persistence and toxicity. Most of the PCB data used in this ERA are based on Aroclor analyses, and exposures are described using total PCB data. PCBs are known to bioaccumulate as a result of ingestion of PCB-contaminated surface water, sediment, soil, vegetation, and prey. BCFs or bioaccumulation factors (BAFs) are often used to evaluate the bioaccumulation potential of chemicals in the environment. As stated previously, chemicals with BCFs less than 300 are considered to have low bioaccumulation potential, while those with BCF between 300 and 1,000 have moderate potential to bioaccumulate. Chemicals with BCFs greater than 1,000 are of most concern with regard to potential bioaccumulation. Table 4-2 lists literature-based freshwater BCFs for the PCBs detected onsite.

Upper trophic level predators, such as mink or bald eagle, are likely to be most exposed to PCBs via consumption of contaminated prey. Food webs for such species can be based on PCBs in surface soil, instream sediment, or floodplain sediment/soils. Bald eagles, for example, are most closely associated with PCBs in fish, which in turn are exposed to PCBs in the water column, instream sediments, and prey. For other species such as mink, dietary exposures are likely to be based on a variety of abiotic media, including surface water, instream sediment, floodplain sediment, and possibly surface soils in more upland areas. Food chain modeling requires that the relationships between source media and prey be known. Food chain modeling is used to calculate PCB doses and dose-based hazard quotients.

Media-specific preliminary remedial goals (PRGs) are also calculated using food chain modeling for most upper trophic level receptors except mink. PRGs for mink are based on the site-specific relationships between PCBs in fish, water, and sediment instead of on food chain modeling for the reasons discussed below.

(1) The inclusion of mixed terrestrial and aquatic prey means that two PRGs (soil and sediment) need to be solved simultaneously, which results in an array of possible combinations of protective soil and sediment PRGs.

(2) Since the experimental species and receptor species are the same, a simplified approach is permissible – (i.e., back-calculating PRGs from dietary PCB concentrations protective of mink, instead of the body weight normalized approach required for extrapolating toxicity information between species).

(3) The modeled terrestrial component of riverine mink diet is minimal (~15% of total diet), and the central question is *what level of sediment PCBs would be protective of mink predominately feeding on aquatic resources.*

4.1.4 Exposure Analysis

Information on distributions of stressors and receptors are combined and summarized in this section, and potential for exposure is discussed. For PCBs, such discussions consider important chemical properties summarized in Table 4-2 (i.e., environmental persistence, bioavailability, and bioconcentration potential). For identified receptors or representative groups of receptors, estimates of potential exposure consider the important ecological parameters that can increase or in other ways modify exposure, such as habitat use and foraging behavior. Exposure-related information for key organisms or representative receptors is summarized in Appendix B.

Samples of several representative organisms, including some of those discussed above, were collected and analyzed for whole body PCB analyses. The U95 (fish) and maximum (terrestrial biota) whole body PCB concentration for each of these organisms or groups of organisms is used to evaluate PCB exposure in representative biota, and support food chain modeling.

The concentrations and ABSA-wide distributions of PCBs in sampled biota and abiotic media are presented in Tables 4-5a and 4-5b.

Table 4-5a presents all other biological and abiotic concentration data. These data are presented on an area-by-area basis. This presentation is, for Table 4-5a, based on previously defined spatial units for sampling aquatic biota (ABSAs) and terrestrial biota (TBSAs) (Figures 3-1 to 3-10). As discussed previously, boundaries of ABSAs are defined so that all areas of the API/PC/KR site are associated with an ABSA. This expansion of ABSAs beyond sampled areas is not intended to suggest that the abiotic (i.e., sediment, soil, and water) samples collected are representative of non-sampled areas within the ABSA. The variability of such samples precludes having much

confidence in such assumptions. Instead, the ABSAs are expanded in consideration of mobile receptors such as fish and mink. The PCB concentrations of mobile receptors collected within an ABSA are assumed to be (1) representative of concentrations in mobile biota found in the expanded ABSA, and (2) the result of exposures from within the entire ABSA.

Table 4-5b presents total PCB concentrations measured in bird eggs collected onsite. In most cases these egg data include total PCB concentrations in individual eggs taken from the same nest. Where this is the case, these data cannot be considered completely independent samples because the eggs were laid by the same parent bird. Multiple eggs were taken from nests of most bird species listed in Table 4-5b.

Figure 4-1 graphically presents the relationships between PCBs in surface water, sediment, and whole body fish collected onsite, on an ABSA-specific basis. This figure reveals that PCB concentrations in fish and abiotic media are generally related but the relationship is not linear. This finding is not unexpected since fish receive PCBs from multiple sources and via several exposure pathways. PCB concentrations in fish tissue are therefore not expected to be completely correlated to PCB concentrations in surface water, sediment, or prey. More importantly, it is expected and confirmed that elevated fish tissue PCB concentrations are associated with elevated PCB concentrations in abiotic media. In addition, low fish tissue PCB concentrations are associated with low PCB concentrations in abiotic media.

4.1.5 Food Web/Food Chain Modeling

The PCB Food Web Model (Figure 4-2) is described below and food web-related data are presented in Appendices C-1 and C-2. Appendix C-1 presents the input parameters and concentration data for abiotic and biotic media. Appendix C-2 is a spreadsheet used to calculate doses and PRGs for representative semi-aquatic and terrestrial receptors.

This food web model is an important component of the ERA because it describes important characteristics of key receptors and associated exposures to PCBs. These key species were selected because they are common or potential inhabitants of the API/PC/KR corridor and most likely obtain their food from the river and/or associated terrestrial habitats. EPA Region 5 Biological Technical Advisory Group (BTAG) has approved these key species for this ERA. Section 5.1.4 provides a discussion on the estimated average potential daily dosage (APDD) and threshold effects values for "key" species. This is a simplified model utilizing measured and estimated input parameters and established mathematical relationships between input parameters. Models such as these are used to estimate the average potential dietary exposure for upper trophic level organisms from ingestion of contaminated prey. For this ERA, the risks posed to lower trophic level organisms and all aquatic organisms are assessed by comparing exposure point concentrations in exposure media to concentrations that can cause ecologically significant effects. For this ERA, ecologically significant effects are defined as those adversely affecting survival,

growth, or reproduction. Survival or mortality can be determined in acute toxicity tests (i.e., tests of short duration and generally high exposure concentrations) or chronic toxicity tests (i.e., tests of long duration and comparatively lower exposure concentrations). Growth and reproductive effects are usually measured by chronic testing.

PCBs are not acutely toxic to many species, yet long-term exposures can have adverse effects on individuals, populations, and communities. The presence of detectable PCB concentrations in biological tissues is not in itself considered ecologically significant unless such concentrations can be correlated to adverse effects. For example, common snapping turtles (*Chelydra serpentina*) are known to accumulate and retain substantial amounts of PCBs in fatty tissues with no observed ill effects (Olafsson, et al. 1983 in Eisler 1986). Consumers of snapping turtles, however, may be at significant risk if dietary intake is of sufficient quantity, frequency, and duration to result in exposure to PCB concentrations similar to those measured at the API/PC/KR site.

As previously stated, it is most appropriate to focus the ERA on critical exposure scenarios. This ERA, and specifically the food web model, is focused on the most critical exposure scenarios for ecological receptors. For terrestrial species, and for nearly all identified carnivores, the potential exposure from ingestion of PCB-contaminated surface water is considered insignificant relative to the potential risks from ingestion of PCB-contaminated prey. This assumption is based on relatively low surface water PCB concentrations and total potential PCB intake compared to prey concentrations and total potential intake via ingestion of contaminated prey. The risks to carnivores and all terrestrial species from the ingestion of PCB-contaminated surface water are, therefore, not included in this assessment.

The primary PCB-related risks for aquatic organisms, especially those occupying lower trophic levels, are likely to be from direct contact with and ingestion of contaminated surface water, sediment, and pore or interstitial water. Certain aquatic organisms such as predatory game fish can also be significantly exposed to PCBs through ingestion of contaminated prey. The relative contribution to overall PCB exposure from each exposure pathway and exposure source (e.g., water, sediment, prey) cannot, however, be reliably determined for most aquatic organisms because of the variability in factors that can affect total exposure.

These factors can include intraspecific and interspecific differences in life stage, season, diet, ingestion rate, specific habitat, etc. This assessment evaluates potential risks posed to aquatic biota primarily by comparing ambient PCB concentrations in surface water and sediment to media-specific and, where appropriate, site-specific criteria, standards, or critical effects concentrations (e.g., no or low observed adverse effects concentrations).

A primary output of the PCB Food Web Model is an estimation of the average potential daily dose (APDD mg PCB/kg body weight-day) from ingestion of

PCB-contaminated prey for upper trophic level organisms. This estimation is based on the following formula from EPA (1993):

$$ADD_{pot} = \sum_{K=1}^{n} (C_k * FR_k * NIR_k)$$

Where:
- ADD_{pot} = Potential average daily dose (mg PCB/kg BW-day)
- C_k = Average PCB concentration in the k^{th} food type (mg/kg)
- FR_k = Dietary fraction of intake of the k^{th} food type (range 0 to 1.0)
- NIR_k = Normalized ingestion rate of the k^{th} food type (wet weight of prey ingested per day, kg/d)
- n = Number of contaminated food types

Normalized ingestion rate is the ingestion rate normalized for body weight:

$$NIR_k = IR_k / BW$$

Where IR_k is the ingestion rate (kg/d) of the predator and BW is the body weight (kg) of the predator. As stated above, this term is expressed as wet weight, or NIR_{ww}.

For species for which incidental sediment or soil ingestion is significant, an additional term is added to the equation presented above, as shown below.

$$ADD_{pot} = \sum_{K=1}^{n} (C_k * FR_k * NIR_k) + (NIR_{dw} * PCB_{Soil} * DF_{Soil})$$

The combination of both NIR_{ww} and NIR_{dw} is required because PCB concentrations in biota serving as prey are expressed as wet weight and sediment and soil PCB concentrations are expressed as dry weight.

The site foraging factor or SFF is commonly added to the above equation (multiplied in the numerator) to account for the fact that some animals forage over a wide range. Ingestion of contaminated prey may therefore be adjusted by the portion of time foraging takes place in contaminated areas. This adjustment is most appropriate where predators with large foraging ranges are evaluated at small sites.

$$SFF = Site\ Foraging\ Factor$$

(Site area, hectares/home or foraging range, hectares) (Range = 0 to 1.0)

This ERA does not adjust the SFF and retains the SFF at 1.0, assuming that the foraging range is less than or equal to the site area. This assumption appears conservative or overly protective until one considers that nearly the entire site provides suitable habitat and food for most predators. There is no reason to believe, and there is no evidence that predators such as mink will leave the site and obtain food beyond site boundaries. All known bald eagles nests are along the Kalamazoo River and it is assumed that eagles will obtain all of their food from the Kalamazoo

River corridor. This is critical, because if a breeding pair is capable of producing fledglings, they will most likely be fed contaminated prey from the Kalamazoo River corridor. Section 5 discusses some additional evidence that supports this preliminary assumption.

Each of these input parameters, in addition to other parameters used to support the ERA (e.g., bioconcentration factors), is discussed below. Finally, for readability, the potential average daily dose (ADD_{pot}) is referred to in subsequent sections of the ERA as the APDD or average potential daily dose.

Representative Species

For assessing potential risks to ecological receptors, certain local species are selected to represent important trophic levels in aquatic and terrestrial food chains for this site. Important trophic levels for each identified food chain include primary producers (plants), primary consumers (herbivores), secondary consumers (carnivores), and top predators (carnivores at the top of a food chain). Some organisms can occupy more than one trophic position in a food web. For example, raccoons consume both plants and animals and, in some food webs, can also be considered top predators. For this assessment, forage and rough fish include both herbivorous and carnivorous species, and detritivores are included with herbivores and omnivores.

Primary Trophic Levels and Categories of Representative Organisms

Primary Producers

General categories of organisms identified as primary producers include:

- Algae
- Aquatic macrophytes
- Terrestrial macrophytes

Primary Consumers

General categories of organisms identified as being predominantly herbivorous, omnivorous, or detritivorous, include:

- Aquatic invertebrates (benthic and water column)
- Forage fish
- Rough fish
- Terrestrial invertebrates
- Small terrestrial omnivorous rodents
- Omnivorous songbirds
- Semi-aquatic herbivorous mammals

Secondary Consumers

General categories of organisms identified as being predominantly carnivorous include:

- Game fish
- Small terrestrial/semi-aquatic carnivorous mammals
- Birds of prey
- Large terrestrial carnivorous mammals

Top Predators

Secondary consumers or carnivores specifically identified as top predators for this assessment include red fox, great horned owl, bald eagle, and mink.

Local species are selected to represent general categories of organisms and important trophic levels in identified food chains. Several of these species or categories of organisms have been sampled to determine whole body PCB concentrations. Whole body (where applicable) PCB concentrations are estimated for other non-sampled species or categories of organisms. These estimates are based on species-specific BCFs or BAFs as much as possible, and on measured PCB concentrations in exposure media. For example, the PCB concentration in algae (mg/kg) is estimated by multiplying the measured surface water PCB concentration (mg/L) by an appropriately derived BCF for freshwater algae.

PCB concentrations in whole body (wet weight) or specific tissue (wet weight) are *measured* in several selected species, as summarized in Tables 4-5a and 4-5b. These species, and the associated trophic category, include:

- Terrestrial macrophytes - Based on bioaccumulation of PCBs in terrestrial plants, from data collected from onsite garden plot in 2000
- White sucker (*Catostomus commersoni*) or equivalent - forage fish
- Common carp (*Cyprinus carpio*) - rough fish
- Smallmouth bass (*Micropterus dolomieui*) - game fish
- Earthworm (*Lumbricus terrestris*) or equivalent - terrestrial invertebrate
- Deer mouse or white-footed mouse (*Peromyscus maniculatus* or *P. leucopus*) - small omnivorous terrestrial mammal
- Muskrat (*Ondatra zibethica*) - semi-aquatic herbivorous mammal
- Mink (*Mustela vison*) - terrestrial/semi-aquatic carnivorous mammal
- Bird Eggs (multiple species) - omnivorous, carnivorous, piscivorous avian receptors

PCB concentrations are **estimated** for:

- Algae and aquatic macrophytes - Based on bioconcentration of PCBs in diatoms and *Hydrilla*, respectively

- Aquatic invertebrates (benthic) - Based on bioconcentration of PCBs in scuds (*Gammarus*) and midge (*Chaoborus*) larvae determined in laboratory experiments

- Aquatic invertebrates (water column) - Based on bioconcentration of PCBs in cladocerans (*Daphnia*) and mosquito larvae (*Culex*)

- American robin (*Turdus migratorius*) - Whole body estimates based on estimated diet (using site-specific and modeled data) and diet-to-carcass BAF (alewife to herring gull) as determined by Braune and Norstrom (1989).

- PCB tissue concentrations are neither measured nor estimated for the three remaining representative top predator species: great horned owl (*Bubo virginianus*), red fox (*Vulpes fulva*), and bald eagle (*Haliaeetus leucocephalus*). This is not considered a critical data gap for three reasons:

  1. The primary purpose of determining PCB concentrations in selected organisms is to estimate potential dose through dietary exposure for consumers of contaminated prey. Top predators, by definition, are unlikely to be regularly consumed by other organisms.

  2. Data are unavailable to adequately interpret whole body or tissue PCB concentrations for these or closely related species. Contaminant body burdens are not in themselves appropriate assessment endpoints and, in general, are not useful without comparison to appropriately derived toxicity data (i.e., effects related to body burden concentrations).

  3. The primary risks associated with PCB contamination to top predators are through ingestion of PCB-contaminated prey, and available toxicity data primarily relate toxic effects to dietary dose rather than to PCB concentrations in whole body or specific tissue type.

For these reasons, estimations of the average potential daily dose (APDD) from ingestion of contaminated prey are used to assess potential PCB-related risks for the great horned owl, red fox, and bald eagle.

Input Parameters and Assumptions

The following subsections show the model input parameters, as well as assumptions made for each. Appendix C-1 includes all input parameters, thresholds or criteria, and associated assumptions for all media and receptors. Appendix C-2 shows the calculations for PCB doses, hazard quotients (HQs), and PRGs for terrestrial and semi-aquatic receptors. Appendix C-2 consists of two parts. C-2-A is a spreadsheet used to calculate doses, HQs, and PRGs for terrestrial receptors, and C-2-B is a similar spreadsheet for semi-aquatic receptors.

PCB Concentration

Where data quantity allow, PCB concentrations are based on the U95 concentration of PCBs in abiotic media (surface water, streambed and floodplain sediment, and surface soil) of concern. These values are based on specific terrestrial and aquatic biota sampling areas (TBSAs and ABSAs), as described in the *Biota Sampling Plan* (CDM 1993). U95 values are also used to describe PCB concentrations in biological tissues if sufficient data have been collected to allow for U95 calculations. Where data are more limited (e.g., terrestrial biota), maximum detected values are used for the reasons discussed previously. Values are in mg PCB/L for surface water and mg PCB/kg (dry weight) for sediments, surface soil (from TBSAs), and biological tissue.

PCB concentrations in surface water (mg/L), streambed and floodplain sediment (mg/kg), and surface soil (mg/kg) are based on measured values. PCB concentrations in biological tissue (mg/kg, wet weight) are estimated for aquatic organisms considered representative of lower trophic levels. These organisms include algae, aquatic macrophytes, and aquatic (benthic and water column) macroinvertebrates. In addition, PCB concentrations are estimated for birds, represented by American robin, from calculated PCB concentration in robin diet, using literature-based diet to whole body (carcass) data for birds. PCB concentrations for earthworms (depurated), all fish species, muskrat, mink, and mice are based on the ABSA- or TBSA-specific maximum measured whole body (and liver for mink and muskrat) PCB concentration for these organisms. Terrestrial plant PCB concentrations are based on measured garden plot data for several crop species from ABSA 8, collected in 2000. For species likely to eat fruits or berries (e.g., robin and fox), the BAF determined for tomatoes at this location was used to estimate PCB concentrations in fruits and berries. PCB concentrations were neither measured nor estimated in the remaining three species (great horned owl, red fox, bald eagle) for the reasons cited previously.

Exposure Media

Exposure media represent the primary media to which specific receptors or categories of receptors may be exposed. These media include surface water, streambed and floodplain sediment, and surface soil. Streambed sediments are bottom sediments covered with surface water. Floodplain sediments are those sediments deposited behind former impoundments, and may or may not be dry depending on specific location and season. Floodplain sediments that are inundated for several months each year are best viewed as streambed sediments for the purposes of food chain modeling and derivation of preliminary remedial goals (PRGs). Floodplain sediments that are never inundated or only rarely wet should be viewed as surface soils. Media identified as *surface soils* specifically refer to those soils collected within TBSAs. TBSA soil samples may include samples taken from perennially dry areas representing true terrestrial exposures as well as samples taken from seasonally inundated areas. The latter are more appropriately considered floodplain sediments, and are more closely associated with aquatic exposures. Surface soils are also assumed to best describe those solid media found in upland areas, including areas associated with elevated landfills. Finally, floodplain sediments for ABSA 11 (Ottawa and Potawamie Marshes)

are identified as wetland/marsh sediments that differ from sediments associated with the former impoundments.

Bioconcentration or Bioaccumulation Factor

BCFs/BAFs (Aquatic)

BCFs are based on the ratio of tissue contaminant concentrations in species of concern (mg/kg) to contaminant concentrations in surface water (mg/L). Bioconcentration considers only direct uptake from water, and does not include uptake from food. In general, BCFs are used for aquatic plants, aquatic invertebrates, and fish, and are based on laboratory tests in which sediments and contaminated prey are absent. Some BCFs presented in Appendix C-1 are derived from literature-based values and are applicable where specific biota such as algae, aquatic macrophytes, and aquatic invertebrates were not sampled. Laboratory-derived BCFs may not reflect bioconcentration potential under field (i.e., natural) conditions. For this study, the uptake of PCBs by algae, aquatic macrophytes, and aquatic invertebrates is estimated from appropriately-derived (i.e., following EPA guidelines) geometric mean BCFs in the literature, while BCFs (actually BAFs) for fish are calculated from site-specific measured U95 PCB concentrations in surface water and fish. There is greater confidence in the calculated BAFs for fish compared to BCFs for algae, aquatic macrophytes, and aquatic invertebrates. Confidence in the field or site-specific BCFs is increased because these data reflect uptake from all sources, not just water. Confidence in these same values is decreased to some degree because the fish and surface water data were not collected at exactly the same times and locations. These relationships are, however, considered useable because the surface water and fish data were collected within approximately the same time period and are ABSA-specific.

BAFs (Terrestrial)

BAFs are similar to BCFs except that they reflect uptake from both food and water. The uptake of contaminants by fish and other aquatic organisms exposed to contaminated surface water, sediment, and prey in the field is best described using BAFs rather than BCFs.

BAFs can also be used to describe the soil-to-plant transfer of contaminants in terrestrial systems. For this assessment, BAFs for terrestrial macrophytes are based on one of two values.

- For diets composed of multiple types of plant tissues (e.g., roots, stems, leaves, fruits, and seeds, estimated plant PCB concentrations are based on the upper 95th confidence limit of the arithmetic mean measured co-located soil and plant PCB concentrations from a garden plot in ABSA 8 or

- For diets composed primarily of fruits or berries, estimated plant PCB concentrations are based on measured co-located soil and tomato PCB concentrations from a garden plot in ABSA 8.

These data were collected in part in response to KRSG comments (September 11, 2000 letter) on the lack of site-specific soil-to-plant bioaccumulation factors. These data were obtained in 2000, and are based on eight crop species. These soil and plant PCB concentrations, along with calculated BAFs for co-located samples, are presented below in Table 4-6. This ERA uses the site-specific U95 BAF of 0.037 to estimate general plant uptake and PCB doses for herbivorous receptors likely to consume a variety of plant tissues such as leaves, stems, and seeds. Calculated PCB doses for herbivorous or omnivorous receptors expected to consume primarily fruits (e.g., robin) are based on the soil to tomato BAF of <0.0008 (set to 0.0008). It is recognized that these BAFs may overestimate or underestimate PCB uptake for terrestrial plant species because of uncertainties related to sample size and PCB uptake in plant species and tissue types (e.g., seeds) likely to be consumed by certain representative herbivorous or omnivorous receptors.

To provide other lines of evidence regarding plant uptake of PCBs, Table 4-7 presents other literature-based values for PCB transfer from surface soil to terrestrial plants. The soil-to-plant transfer factors or BAFs presented on Table 4-7 are ranked from lowest to highest. The site-specific BAF of approximately 0.04, from the garden plot data, is also included on this table and is identified in bold type. It can be seen that the selected site-specific soil-to-plant BAF of 0.04 falls approximately at the mid-point of the ranked literature-based data. These literature-based data include experimental and modeled BAFs, and are believed to encompass the range of values that may be observed in the field with a variety of plant species and tissue types. It is noted that species and plant tissue types (e.g., seeds) that are likely to be consumed by herbivorous or omnivorous consumers such as deer mice are not included in this list of literature-based plant BAFs. Although this is an area of uncertainty, the garden plot data and resulting BAFs (0.037 and 0.0008) are considered adequately representative of soil-to-plant PCB transfer at this site.

The results of some studies presented in Table 4-7 indicate that certain terrestrial plants can accumulate PCBs from soil to a concentration greater than the original soil concentration (i.e., BAF>1). Trapp, et al. (1990) presents the results of two experiments in which the average plant PCB concentration was approximately 1.3 times that of the soil in which the plant was grown. Pal, et al. (1980) described biomagnification factors (BMFs) for several plant species. As expected, most terrestrial species accumulated PCBs from the soil at a BAF (or BMF) of less than 1.0. However, included in this list of BMFs for several plant species are two results that support a higher BAF for some species. Carrots, for example, accumulated PCBs from the soil at a factor of about 0.25, while weeds exposed in the same study accumulated up to a factor of 0.96 times the soil concentration (i.e., BAF = 0.96). Weeds exposed in a study focused on sugarbeet accumulation of PCBs took up PCBs from the soil at a factor of 0.80 (BAF = 0.80).

Much higher BAFs are described by Pal, et al. (1980) for aquatic and riparian plants that occur in wet soils or soils that are frequently flooded.

BAFs are also calculated from measured PCB concentrations for most of the remaining aquatic, semi-aquatic, and terrestrial species. In cases where more than one media type is identified as a potential source of PCB contamination, BAFs are based on the primary exposure media. For example, mink feed on a wide variety of aquatic, semi-aquatic, and terrestrial animals. PCB contamination in surface water, streambed and floodplain sediment, and surface soil can all contribute to PCB accumulation in mink through ingestion. For this reason, it is inappropriate to calculate BAFs or PRGs based on multiple, often uncertain exposure scenarios. Food chain modeling for mink is limited in this ERA to calculation of doses used to derive hazard quotients. Calculated aquatic (surface water) and terrestrial (surface soil) BAFs are based on TBSA/ABSA-specific PCB concentrations measured in abiotic exposure media and biota (Table 4-8), where these data are available. In addition, Table 4-8 presents BSAFs for ABSAs where streambed sediment and fish were collected over approximately the same time period. BSAFs reflect the potential transfer of a contaminant in sediment to biological tissues. The confidence in the ABSA-specific BSAFs is increased by the relatively large amount of fish and sediment data collected over approximately the same time period from the same ABSA. Contributing to decreased confidence in these BSAFs is the fact that the fish and sediment data were not collected at exactly the same location and time. The latter is not considered a critical data gap because of the mobility of fish and the variability in sediment PCB concentrations within an ABSA.

Diet-to-Bird BMF

Site-specific data are lacking for PCB concentrations in whole body birds. Whole body bird PCB concentrations must therefore be estimated from available site-specific data (e.g., PCB concentrations in worms and plants) and literature-based data (e.g., biological multiplication factor (BMF) that relates PCBs in diet to whole body burden). Literature-based BMFs have been reviewed for use in this ERA for estimating total PCB concentrations in whole body birds from bird diets. The selection of the most appropriate BMF is important because the consumption of whole body birds contributes to modeled total PCB dietary doses (and risks) for great horned owl, red fox, bald eagle, and mink.

The diet-to-bird BMF selected for food chain modeling in this ERA is 93, taken from Braune and Norstrom (1989). This BMF is based on PCB-contaminated fish (alewife) consumed by herring gulls. The BMF (93) from Braun and Norstrom was also used for total PCBs in the Great Lakes Initiative (rounded to 90) for estimating risk to bald eagle (USEPA 1995b). This peer-reviewed EPA document is used for regulatory purposes. The BMF of 93 is also consistent with a caged juvenile herring gull feeding study that resulted in a diet-to-bird BMF of 97 (quantified as A1254 and described as "apparent PCBs", Anderson and Hickey 1976).

Additional supporting information is used to confirm the consistency of the Braun and Norstrom study with other similar studies. This included a comparison of diet-to-egg BMFs. Diet-to-egg BMFs are not used directly in this ERA but data from two separate studies are compared here to provide additional support for using the Braun and Norstrom BMF data.

Lipid-normalized diet-to-egg BMFs for individual PCB congeners in the Braun and Norstrom study are consistent with (and actually lower than) the lipid-normalized fish-to-egg geometric mean congener BMFs calculated by Blankenship and Giesy (2002) from multiple studies. Lipid-normalization is based on the following lipid contents reported by Braun and Norstrom (1989): herring gull whole body - 10.3 percent, gull egg - 7.7 percent, and alewife - 2.8 percent.

Congener-specific lipid normalized diet-to-egg BMFs are presented below for both the Braun and Norstrom (B&N) study and the geometric means calculated by Blankenship and Giesy (B&G). The Braun and Norstrom (B&N) data presented below include additional congener data not included in the original paper (1989) but subsequently reported by Hoffman, Rice, and Kubiak (1996).

PCB Congener	77	101	105	110	118	126	138	153	169
B&G	0.89	4.52	7.95	5.4	26.15	29.74	27.74	32.57	31.25
B&N + H,R&K	0.7	2.9	7.3	2.5	11.3	10.5	17	17.3	16.7

The total PCB lipid-normalized diet-to-egg BMF from the Braun and Norstrom study is 11.5. This is comparable to the total PCB geometric mean lipid-normalized diet-to-egg BMF of 18.1 (range 10.4-36.8) reported by Koslowski, et al. 1994 for Lake Erie gulls--one of the studies relied on by Blankenship and Giesy (2002). Blankenship and Giesy (2002) did not, however, report total PCBs.

As discussed above, the Braun and Norstrom BMFs are supported by the results of several studies. However, substantially lower diet-to-bird BMFs of 10 or less for total PCBs have also been reported in the literature. This leads to uncertainty with the diet-to-bird BMF expected in the field. The more conservative (higher) BMF determined by Braun and Norstrom is selected for this ERA because regulatory guidance recommends using a conservative or more protective approach where uncertainty exists.

Finally, the value assigned to the diet-to-bird BMF affects food chain modeling for only the great horned owl, red fox, bald eagle, and mink, in decreasing order of importance. The order of importance is based on the estimated dietary fraction comprised of birds for each of these receptors. The estimated dietary fraction of birds is 47 percent for great horned owl, 19 percent for red fox, 17 percent for bald eagle, and 5 percent for mink. The diet-to-bird BMF influences to a small degree the risk estimates (i.e., hazard quotients) for mink, but does not affect the PRGs established for protection of mink, which are not based on food chain modeling.

Home Range

An animal's home range can greatly affect its degree of exposure. For example, animals with home ranges entirely within a contaminated area will have greater exposure potential than animals with home ranges that substantially exceed the area of a contaminated site. This assumption may not always hold true, however, because home range values are often only estimates of the average area used by a particular species. It is not unreasonable to assume that an animal with a large home range will, at times, remain within a smaller area if that area provides adequate food and cover. In addition, models that estimate dietary exposures, including this model, are very sensitive to variability in home range estimates. Average home ranges for adult animals are presented in the model.

Site Foraging Frequency

Standard practice in assessing dietary exposures for wildlife includes the derivation of site foraging frequency (SFF). This term is used to describe the ratio of the site area to the average home range for the species of concern. As commonly used, SFF values range from 0 to 1.0. It is apparent that animals with large home ranges are less likely to be significantly exposed to site-related contamination than animals that live entirely within site boundaries. However, as stated above, the use of home ranges for estimating exposure likelihood has certain critical limitations. First, home range estimates are based on overall use, yet certain individuals or populations may use smaller areas for foraging and cover if conditions are suitable. Also, dietary exposure models are extremely sensitive to variability in the input parameter identified here as SFF. It is not uncommon for dietary exposure models to predict zero or nearly no risk for species associated with highly contaminated sites solely because their average home range is very large. The API/PC/KR area is large, and areas of PCB contamination are not evenly distributed in size or location. Thus, accurately correlating home range to site area is difficult at this site for species with large home ranges. However, this ERA focuses on those species that would primarily spend all or most of their time within the Kalamazoo River corridor.

Finally, the methods for determining home ranges are not intended to support the specific needs of ecological risk assessment. Home range sizes, which are presented in Appendix C, are often determined by locating nests, dens, or spawning areas for species of concern and then recording the locations of individual organisms observed in the area of the nest or den. Locations of individual organisms observed are then plotted on a map and connected by lines forming a polygon, with the nest or den located within the polygon.

The area of the resulting polygon is considered to be a home range. This method does not consider frequency and size of foraging areas within the estimated home range, and therefore may be inappropriate for ecological risk assessment use. For the reasons cited, this assessment sets the SFF to 1.0 for all species for which dietary exposure is calculated. Although this adds conservatism to the model, it is considered prudent to prevent gross under-estimations of potential risks for some ecological receptors.

Dietary Fraction
Dietary fraction is an estimate of the fraction of total diet contributed by each prey type. For this study, estimates of dietary fraction are based on values reported in the literature. Where more than one literature source of dietary information is available, estimates are based on the average of all relevant literature sources (primarily EPA 1993) or the values most relevant to Western Michigan. The fraction of soil or sediment incidentally ingested is also included if such ingestion is deemed appropriate. For example, muskrat are assumed to incidentally ingest a substantial amount of sediment while feeding and grooming, while bald eagles feeding in a riverine environment on fish probably ingest little or no sediment.

Average Ingestion Rate
Average ingestion rates (kg/d) are determined for species of concern from values in the literature. Most data are taken from EPA's *Exposure Factor Handbook, Volume I* (1993). Ingestion rates are presented as both wet weight and dry weight—the latter is used where ingestion of sediment or soil is significant. Sediment and soil PCB concentrations are expressed as mg/kg dry weight, while plant and animal dietary items are expressed as mg/kg wet weight.

Average Body Weight
Average body weights (kg) for representative adult organisms are based on values presented in literature sources. Where more than one source was consulted, the value used is based on the average of all species-specific adult body weights presented. In some cases, average body weights can be substantially different for males and females of the same species. Where this is the case, values used are based on the average of values reported for adult males and females.

Model Output
As stated above, the primary model output is an estimate of the average potential daily dose (APDD, mg PCB/kg BW-d) for upper trophic level organisms from ingestion of contaminated prey. This value is not determined for lower trophic level organisms (e.g., algae, macroinvertebrates, earthworm, forage fish) or game and rough fish because either it is not applicable (e.g., algae) or input parameters (e.g., ingestion rates) are generally unknown or associated with a high degree of uncertainty. APDD values may over- or underestimate actual PCB doses because of site-specific diet or foraging habits. Also, actual PCB doses probably vary seasonally and spatially.

For organisms for which APDD is not calculated, risk estimations are based on comparisons of exposure point concentrations of PCBs (e.g., PCB concentration is surface water) to NOAECs, LOAECs, criteria, or recommended limits.

Average Potential Daily Dose, APDD, (mg PCB/kg BW-d) is calculated from the equation described previously, and serves as the primary output of the PCB Food Web Model. This value is used to estimate potential risk to upper trophic level

organisms from ingestion of contaminated prey by comparison with critical dietary concentrations.

Toxicity Assessment
The potential toxicity of PCBs to representative organisms is evaluated by comparing measured or estimated PCB concentrations in abiotic media or prey to

- appropriate media-specific criteria (e.g., AWQC),
- safe levels not associated with adverse effects (e.g., NOAECs or EC_{10}/ED_{10}), or
- species-specific concentrations at which adverse effects begin to be observed (e.g., LOAECs or EC_{25}/ED_{25}).

Although considered part of the food web model as a preliminary evaluation, these data are further discussed in the Effects Assessment portion of the ERA. The effects assessment also discusses other effects data used in the Risk Characterization phase of the ERA, including site-specific values with which overall risks to ecological receptors are evaluated.

No Observed Adverse Effects Concentration (NOAEC)
NOAECs are obtained from the literature for species of concern or for closely related species that are expected to exhibit toxicologically similar responses to PCB exposures. Species-specific NOAECs are compared to measured or estimated PCB concentrations from similar routes of exposure (e.g., direct contact or ingestion of food items) for selected species. Specific NOAECs selected for this study include the highest concentrations associated with no adverse effect from toxicity tests conducted with species of concern. Also consulted are primary data sources referenced in EPA contaminant-specific criteria documents (aquatic organisms) and FWS contaminant hazard review documents (terrestrial organisms). NOAECs are not associated with adverse effects; therefore, PCB concentrations at or near the relevant NOAECs are assumed to be associated with no risk. NOAECs are commonly estimated by (LOAEC/10). Based on the comparison of two studies performed with field-contaminated fish, Giesy, et al. (1994) recommended the use of LOAEC/3 for estimating NOAECs for mink exposed to PCBs through diet. A review of available data for certain species of birds and mammals supports the recommendation of Giesy, et al. This ERA uses LOAEC/3 to estimate NOAEC for mouse and muskrat and uses NOAEC * 3 to estimate LOAEC for great horned owl. The phrase No Observed Adverse Effects Level or NOAEL is used when exposure is expressed as dose (i.e., mg/kg-d). A different (ED_x or EC_x) approach, discussed below, is used to derive the no effect and low effect toxicity reference values (TRVs) for mink and non-raptor birds.

Lowest Observed Adverse Effects Concentration (LOAEC)
LOAECs are also obtained from the literature for species of concern or for closely related species that are expected to exhibit toxicologically similar responses to PCB

exposures. Similar to NOAECs, species-specific LOAECs are compared to measured or estimated PCB concentrations from similar routes of exposure (e.g., direct contact or ingestion of food items) for selected species. LOAECs are by definition associated with adverse effects; therefore, PCB concentrations at or near the relevant LOAECs are associated with some, possibly unacceptable risk. LOAECs based on dose are termed Lowest Observed Adverse Effects Levels or LOAELs. As mentioned above, a different approach is used to derive the no and low effect TRVs for mink, American robin and bald eagle. Owl-specific toxicity data are used to assess risks to great horned owls. A summary of this approach follows.

Effect Concentration (EC_x) / Effect Dose (ED_x)

It can be difficult to determine the most appropriate no effect and low effect TRVs for mink and non-raptor birds exposed to PCBs based on reported NOAELs and LOAELs. Such difficulties arise because of significant differences in the methodologies and designs of studies in which mink and non-raptor birds are fed PCB-contaminated food. Important differences include test endpoints, chemical form of PCBs fed, test duration, and potential confounding effects of other contaminants present in food items. These differences result in varying degrees of confidence in reported or calculated doses defined as NOAELs. For this reason, there are often disagreements on the appropriateness of any given NOAEL or LOAEL defined as a preferred TRV. As an alternative to selecting a single NOAEL or LOAEL, this ERA uses a more detailed analysis of toxicity data to derive the no effect and low effect TRVs for mink and non-raptor birds. The approach is introduced below for mink and birds, with a more detailed discussion of these TRVs in Section 4.2, Effects Assessment.

MINK - The no and low effect TRVs for mink are based on a detailed analysis of the literature on the effects of PCBs on mink. The TRVs for mink, which form the basis of the surface water and sediment preliminary remedial goals (PRGs) for this site, are discussed in detail in Section 4.2. The calculated dietary PCB low effect TRV for mink is 0.6 mg/kg wet weight (diet). The estimated no effect TRV is 0.5 mg/kg wet weight (diet) for mink. In addition to the discussion of mink TRVs in Section 4.2, Appendix D provides a complete and detailed discussion of the method used to derive these TRVs.

BIRDS - The no and low effect TRVs for birds (i.e., American robin and bald eagle) are based on a detailed analysis of the effects of PCBs on chicken, one of the best-studied and most sensitive avian receptors of the few species investigated to date. The TRVs for non-raptor birds are discussed in detail in Section 4.2, Effects Assessment. The calculated low effect TRV for birds is 0.5 mg/kg_{BW}-d, based on Aroclor 1248, the predominant Aroclor detected in earthworms in the Kalamazoo River floodplain. The calculated no effect TRV for birds is 0.4 mg/kg_{BW}-d, also based on Aroclor 1248. Appendix D presents a detailed summary of the ED_x/EC_x method used to derive TRVs for birds other than great horned owl, and Section 4.2 presents a more detailed analysis of the final TRVs selected for these birds.

Criteria or Recommended Limits

In some cases, criteria (e.g., AWQC) or maximum allowable limits (e.g., those recommended for the protection of sensitive birds or mammals) have been established for species or other taxa of concern. Where such values are available, they are presented in the food web model for comparison to measured or estimated PCB concentrations determined in this study. Criteria and limits presented in Appendix C are not site-specific but are instead based on general toxicological data. The comparisons between toxicological data from the literature and exposure data for this site are used to evaluate reasonable maximum exposures for the API/PC/KR site, based on U95 PCB concentrations in abiotic and most biological media.

A comparison of arithmetic average PCB exposure data to toxicological data may also be useful, but is considered less appropriate for a large and diverse site like the API/PC/KR. The API/PC/KR site is associated with highly variable abiotic PCB concentrations from one area to another, and average measured concentrations of PCBs are not likely to represent the true average or especially the reasonable worst-case exposure. U95 and, in cases where sample size is small, maximum ABSA- and/or TBSA-specific exposure concentrations are therefore preferred for evaluating potential effects in ecological receptors.

Preliminary Remedial Goals (PRGs)

This ERA develops a range (i.e., no effect to low effect) of site-specific PRGs to be considered as remedial goals associated with the protection of key receptors or habitat types. Where data allow, these site-specific PRGs are based on measured PCB concentrations in exposure media and food items as well as site-specific bioaccumulation in sampled biota. The equations used to calculate terrestrial and aquatic PRGs are presented below. PRGs are presented in the risk characterization phase of the ERA, and the derivation of receptor-specific PRGs is presented in Appendix C-2. The first example is for terrestrial receptors that are assumed to ingest soil along with prey.

Terrestrial SED/SOIL PRG =

$$\textit{(No Effect or Low Effect TRV / SUM (NIRww * } BAF_{Prey}1...x \textit{ *}^{DFPrey}1...x\textit{) + (}NIR_{dw} * DF_{Soil}\textit{))}$$

Where:

No Effect or Low Effect TRV = Species-specific dose (mg PCB/kg BW per day)

NIR_{ww} = normalized daily ingestion rate (IR / BW), mg/kg-d, wet weight

BAF_{Prey} = bioaccumulation factor for PCBs in prey item

DF_{Prey} = dietary fraction of prey ingested

NIR_{dw} = normalized daily ingestion rate (IR / BW), mg/kg, dry weight

DF_{Soil} = dietary fraction of soil/sediment ingested

PRGs for mink exposed to aquatic and semi-aquatic (seasonally inundated) sediments are based on surface water PCB thresholds derived to protect fish tissue from accumulating critical levels of PCBs. These PRGs also consider the site-specific relationships between PCBs in surface water and sediments. The general equation for deriving aquatic PRGs is presented below. Two different ways of viewing this derivation are presented.

Aquatic SED PRG for Mink Protection

= SW threshold * SW-to-SED Partition Factor

or

Aquatic SED PRG for Mink Protection

= Fish Tissue Threshold/BSAF

Where:

BSAF = biota sediment accumulation factor

The fish tissue threshold is based on the surface water threshold and site-specific bioaccumulation of PCBs into fish tissue. The surface water to sediment partition factor is the mean site-specific value for co-located surface water and sediment PCB concentrations. These two equations are therefore mathematically related and are not different. Section 4.2.1 shows these PRG derivations in greater detail.

Site-specific effects data are presented in Section 4.2, Ecological Effects Assessment, and are further discussed in Section 5, Risk Characterization, where risk estimates and proposed cleanup goals or PRGs are presented. An interpretation of the output of the food web model Appendices C-1 and C-2 is presented in the Risk Characterization section of the ERA. The Risk Characterization section discusses the results of the food web model and integrates exposure and effects data to estimate risks to ecological receptors of the API/PC/KR. Effects assessment follows an analysis of uncertainties associated with exposure analysis and the food web model.

4.1.6 Uncertainty Evaluation - Exposure Assessment

Sources of uncertainty in the exposure assessment include the values used to represent the magnitude and distribution of media-specific contamination. Obviously, all media cannot be sampled at all locations, and data interpolation and/or extrapolation are necessary. It is expected that the samples collected have been appropriately analyzed to adequately describe the nature and extent of PCB contamination at the API/PC/KR site. Uncertainty in this assessment is decreased by

the biological sampling specifically designed to support food web modeling and to support descriptions of the magnitude and distribution of PCB contamination at the API/PC/KR site. Because ABSA and TBSA-specific sampling was relatively complete for abiotic media, the use of U95 concentrations of PCBs in SW, SED, FP SED, SS, and most biota minimize the chance that risk estimations based on the selected exposure concentrations have been greatly under- or over-estimated.

Another major source of potential uncertainty in the ERA is the food web model. All models, including simplified models such as the one described herein, are associated with uncertainty. In general, more complex bioenergetic-type models have greater potential to accurately estimate contaminant transfer between environmental compartments but also have greater potential to introduce unacceptable levels of uncertainty unless critical information on site-specific input parameters are available.

For example, aquatic food web models based on bioenergetics have been established that calculate biomagnification factors (BMFs) for organic contaminants from exposure media through all major trophic levels to top predators. These models often require the use and evaluation of input parameters that are currently unknown, such as contaminant depuration rates for a particular species. Values for other species or even other chemicals are sometimes used to represent the required input parameter.

Models may also be sensitive to slight differences in input parameter values, and results can, therefore, be highly uncertain. The uncertainty in resulting BMF estimations for higher trophic level organisms are also magnified because the model is based on addition and multiplication of values from lower trophic levels. For these reasons, complex computer-based food chain models are not considered appropriate for this assessment.

Although every caution was taken in this assessment to limit uncertainty as much as possible, simple models can also be associated with uncertainty. Where potential levels of uncertainty could adversely affect the results of the assessment, conservative approaches were taken that may result in over-protection of some local species. For example, many simple food chain models commonly predict, largely as a result of home range estimates, little or no risk to top predators from ingestion of contaminated prey. The SFF calculated from large home range estimates can therefore "drive" the model output (i.e., the APDD) for certain potentially important species. As discussed above, the foraging behavior of individual organisms and even populations are sufficiently unknown to warrant a more conservative or protective approach. To err on the side of over-protection is considered prudent and, in fact, follows regulatory guidance.

The most likely causes of uncertainty in this assessment are the variability of values associated with certain input parameters, especially values used to describe the distribution of PCB contamination in various media and biota. There is greater uncertainty in PCB concentrations estimated for certain prey items. For example, PCB

concentrations are estimated (using a literature-based BMF) for whole body birds that serve as prey for certain representative receptors (great horned owl, red fox, bald eagle, and mink). These estimated whole body PCB concentrations in birds are based on modeled PCB concentrations for robin using the literature-based BMF and site-specific data for plants and worms comprising robin diet. PCB concentrations in robin diet include a significant exposure via consumption of earthworms. Birds that consume mostly seeds or fruits are likely to have lower PCB exposures than those that eat mostly earthworms. Also, the selected diet-to-bird BMF (93, from Braun and Norstrom 1989) exceeds the diet-to-bird BMF determined in some other studies. The combined impacts of using a vermivore to represent songbirds and using a high diet-to-bird BMF probably overestimates risks to predators of songbirds. On the other hand, risks may be underestimated for predators of piscivorous birds such as mergansers, herons, and kingfishers.

Using U95 values for the larger abiotic and biological media data set and maximum values for the smaller biological data sets is expected to limit uncertainty and risk under-estimation to an acceptable degree. Literature values for BCFs and, to a lesser degree dietary fractions, are also critical with regard to potential for uncertainty due to uncertainties associated with laboratory to field extrapolations. There is more confidence in values used to represent species-specific ingestion rates and body weights because, in most cases, there is reasonable concurrence by investigators. Finally, NOAECs, LOAECs, EC_{10}, ED_{10}, EC_{25}, ED_{25}, criteria, and recommended limits are often based on literature values derived under controlled conditions that may not be fully relevant to natural field conditions. Also, certain criteria or recommended limits are usually intended to protect large and diverse groups of organisms (i.e., aquatic life, mammals, etc.). These values may therefore be over- or under-protective of certain local species and/or populations.

Uncertainty in this assessment regarding field-generated data is likely to be limited mostly to uncertainties in the representativeness of biological samples. Such samples are expected to be highly variable even within a species because of differences in individual behavior and activities. Even these factors are expected to vary from season to season and from one location to another. These types of uncertainties provide one basis for using maximum detected concentrations of PCBs in biological tissues for risk estimations. It is therefore more unlikely that this assessment underestimates risk because conservative approaches such as these are used where appropriate, and any uncertainties are probably biased towards over-protection.

4.2 Ecological Effects Assessment

Effects Assessment includes an evaluation of data sources and data types, and presents media-specific and stressor-specific ecological effects concentrations for PCBs, the primary chemical stressors identified at the API/PC/KR. These data serve as major components of stressor-response profiles, which describe the relationship between ecological stressors and effects. Certain types of effects data, such as NOAELs/No Effect Levels and LOAELs/Low Effect Levels, form the basis for the PRGs developed to protect key receptors representative of particular exposure scenarios and receptor groups.

4.2.1 Evaluation of Effects Data

This section of the ERA describes and provides support for the sources and types of effects data (e.g., toxicity data) selected for use in the ERA. Data sources and types are described on a media-specific basis. Selected measurement endpoints or effects data are based on relevance to the API/PC/KR site, and site-related stressors and receptors are considered in this selection. These data are directly applicable to assessment endpoints and remedial action objectives determined for the API/PC/KR site which include:

1. The preservation of the survival, growth, and reproduction of wildlife

2. The establishment and maintenance of a healthy and diverse aquatic ecosystem in and adjacent to the API/PC/KR site

3. Reductions in PCB concentrations through removal and destruction of contaminated media

4. Reductions in PCB concentrations in fish and wildlife such that human consumption restrictions can be lifted

Some effects data are more relevant and useful than others. For example, effects data are unavailable for certain receptors or receptor groups associated with the API/PC/KR. In these cases, the effects assessment is based on more general effects data available in the literature. Finally, site-specific data, such as bioconcentration and bioaccumulation factors determined by recent sampling and analysis of media and biota, are used to support estimations of risks for ecological receptors. The effects assessment provides multiple lines of evidence using numerous data sources to evaluate risks. This approach is especially important where relevant site-specific data are limited. The availability of effects data is media specific, and relevant data sources for each media of concern are presented below.

Effects Data Sources (Surface Water)

Acceptable and relevant effects data for PCBs in surface water are generally available. More general (i.e., not site specific) surface water toxicity data used in this ERA are from the EPA *Ambient Water Quality Criteria (AWQC) document for Polychlorinated*

Biphenyls (EPA 1980) and Polychlorinated Biphenyl Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review (Eisler 1986). The chronic AWQC derived by EPA is based on protection of mink (the most sensitive wildlife species tested) and considers fish ingestion by mink.

Site-specific surface water total PCB concentrations are also derived to protect mink, under the assumption that protection of mink results in protection of all other less sensitive receptors. These protective values are based on limiting total PCBs in mink diet to levels associated with no effects and low levels of adverse effects. These two values, No Effect and Low Effect dietary toxicity reference values (TRVs), form the basis for the surface water total PCB thresholds designed to protect mink at this site.

As discussed in Section 4.1, it can be difficult to determine the most appropriate no effect and low effect TRVs for mink exposed to PCBs based on reported NOAELs and LOAELs. This ERA therefore uses a different (EC_x) approach to derive the no effect and low effect TRVs for mink. The no and low effect TRVs for mink are based on a detailed analysis of the effects of PCBs on mink. The TRVs are derived from exposure-response curves by interpolation of the effective dietary concentration (EC_x) to female mink that corresponds to specific relative responses (calculated as the treatment response divided by the control response). The low effect level is defined as 0.75 of the control response for a toxicological endpoint (EC_{25}, which represents a 25% decrease in response) and the no effect level is equal to 0.90 of the control response (EC_{10}, which represents a 10% decrease in response). Appendix D provides a more detailed analysis of this approach.

The calculated dietary PCB low effect TRV for mink is 0.6 mg/kg wet weight (diet) based on the effects of Aroclor 1254 on the number of live kits per mated female and kit body weight, adjusted for continuous exposure through two breeding seasons or generations; and the no effect TRV is 0.5 mg/kg based on the effects of Aroclor 1254 on the number of live kits per mated female, adjusted for continuous exposure through two breedings seasons or generations.

The 0.5 and 0.6 mg PCB/kg dietary thresholds for mink are used to calculate a threshold surface water concentration that is protective of mink that consume PCB-contaminated fish. The mean of the average BAF for carp, smallmouth bass, and sucker is used to estimate PCB uptake in fish. This mean BAF is 305,000, as presented on Table 4-8. This BAF and the dietary No Effect TRV 0.5 mg/kg is used to calculate the surface water (SW) threshold associated with no adverse effects.

The SW threshold presented below is based on the average water-to-fish BAF (mean of the mean BAF for all three species) and the assumption that mink diet is comprised of 100 percent fish, with each of the three fish species representing one third of the diet. This conservative approach is based on the need to maintain PCB concentrations in the primary food of mink (fish) at levels that are protective of mink.

No Effect SW threshold

$$= \frac{0.5 \text{ mg PCB/kg fresh weight diet}}{305{,}000}$$

$$= 0.0000016 \text{ mg PCB/L water}$$

$$= \mathbf{0.0016\ \mu g\ PCB/L\ water}$$

The surface water threshold calculated to prevent whole body fish from containing more than 0.5 mg PCB/kg wet weight is 0.0016 µg/L.

Similarly, a Low Effect SW threshold is calculated using the same mean BAF and the Low Effect dietary threshold of 0.6 mg/kg.

Low Effect SW threshold

$$= \frac{0.6 \text{ mg PCB/kg fresh weight diet}}{305{,}000}$$

$$= 0.00000197 \text{ mg PCB/L water}$$

$$= \mathbf{0.00197\ \mu g\ PCB/L\ water}$$

The surface water threshold calculated to prevent whole body fish from containing more than 0.6 mg PCB/kg wet weight is 0.00197 µg/L.

Effects Data Sources (Sediment)

Universally accepted biological effects concentrations for most sediment contaminants have not been developed for ecological receptors. In general, the most useful data on potential sediment toxicity is obtained from site-specific studies using site sediments and resident or representative test species.

Site-specific sediment toxicity data are unavailable for this ERA. The evaluation of the potential toxicity associated with PCB contamination of onsite streambed sediments is based on the comparison of PCB concentrations in API/PC/KR streambed sediments to various relevant data. These include background concentrations, EPA-recommended and site-specific sediment concentrations based on the equilibrium partitioning (EP) approach (EPA 1988b) using both literature-based and measured (site-specific) input parameters (e.g., sediment/water partition coefficients or K_ds), and other relevant data from sources such as Long and Morgan (1991) and Persaud, et al. (1993). Databases such as that of Long and Morgan (1991) have been established that describe the co-occurrence of chemical contaminants and apparent biological effects, and others (e.g., Persaud, et al. 1993) include interim criteria for contaminants in sediment. Although the data presented in these more general (i.e., non-site-specific) databases are associated with certain limitations and uncertainties, they can contribute useful information to the overall evaluation of potential sediment toxicity

using a weight-of -evidence approach. Such an approach is used in the risk characterization phase of this ERA. There, sediment toxicity data are supplemented with comparisons between onsite PCB concentrations in API/PC/KR sediments and concentrations that either co-occur with observed adverse biological effects (Long and Morgan 1991) or have been established as interim sediment quality criteria by Ontario, Canada (Persaud, et al. 1993). The same mink dietary studies used to derive SW thresholds are used to derive site-specific thresholds for PCBs in sediment that protect mink.

The calculated site-specific surface water thresholds of 0.0016 and 0.00197 µg/L are used along with the mean site-specific sediment/surface water partition factor of 301,712 (rounded to 302,000) to derive site-specific sediment thresholds. Again, these sediment thresholds conservatively assume that mink diet is comprised of 100 percent fish and that the primary abiotic source of PCBs in mink prey is instream sediment. These mink-based PRGs are considered protective of riverine mink that consume fish. This approach for deriving mink-based sediment PRGs is justified for the following reasons:

- the terrestrial components of mink diet are minimal compared to aquatic components, represented by fish
- PRG calculation from dietary concentrations (as performed below) rather than dose is appropriate because the receptor species (mink) and the test species (mink) used to derive dietary thresholds is the same
- PRGs based on a diet comprised of both aquatic and terrestrial prey species requires that both sediment and soil PRGs be calculated simultaneously, resulting in an array of results.

The derivation of these sediment PRGs follow:

No Effect SED PRG

= No Effect SW threshold * SW-to-SED Partition Factor

= 0.0016 µg PCB/L * 302,000

= 483 µg PCB/kg sediment

= **0.5 mg PCB/kg sediment**

Low Effect SED PRG

= Low Effect SW threshold * SW-to-SED Partition Factor

= 0.00197 µg PCB/L * 302,000

= 595 µg PCB/kg sediment

= **0.6 mg PCB/kg sediment**

The calculated site-specific PRGs for PCBs in sediment, based on preventing fish tissue from containing more than 0.5 and 0.6 mg PCB/kg wet weight and site-derived BAFs from surface water, are 0.5 and 0.6 mg PCB/kg sediment.

These sediment PRGs can also be viewed using the BSAF approach. This is not an independent derivation because it is based on the same water-sediment-fish relationships described above. As presented on Table 4-8, the average site-specific BSAF, based on all fish species collected onsite, is 1.02. This alternative method of viewing this derivation is as follows:

No Effect SED PRG

= No Effect Fish Tissue Threshold/BSAF

= 0.5 mg PCB/kg wet weight whole body fish/1.02

= **0.5 mg PCB/kg sediment**

Low Effect SED PRG

= Low Effect Fish Tissue Threshold/BSAF

= 0.6 mg PCB/kg wet weight whole body fish/1.02

= **0.6 mg PCB/kg sediment**

Viewing these derivations using the BSAF approach allows simple estimations of whole body fish PCB concentrations from sediment PCB concentrations. Because the mean BSAF is nearly one (1.02), whole body fish PCB concentrations can be approximated by total PCB concentrations in sediment (SED * 1.02 = Fish).

Effects Data Sources (Surface Soil and Floodplain Sediments)

Similarly, accepted critical effects concentrations for chemicals in surface soils and floodplain sediments have not been developed solely for the protection of ecological receptors. As for sediment (streambed) contaminants, site-specific data are considered to be the most useful and appropriate for evaluating the potential toxicity of API/PC/KR surface soils and floodplain sediments. Such data are not, however, available, and three other approaches are used in the risk characterization phase of this ERA.

First, PCB concentrations in onsite surface soil and floodplain sediments are compared to background concentrations based on relevant and available data. Second, more general data sources on the potential hazards of contaminated surface soil and floodplain sediments are used to additionally evaluate the potential toxicity of API/PC/KR surface soil and floodplain sediment. Critical threshold levels for chemicals in surface soils, based on several soil functions including the protection of

wildlife, have been derived by and used in various countries (e.g., Norway; The Netherlands; West Germany; England; Ontario and Quebec, Canada) for several years (Siegrist 1989). The most appropriate critical threshold levels from sources such as these, based on general acceptance and data quality and quantity, are used to evaluate the potential toxicity of PCBs in surface soil and floodplain sediment. Evaluation of these alternative data sources suggests that the Ontario and Quebec (Siegrist 1989) values are the most appropriate and useful for this ERA. Preferred data (e.g., site-specific soil toxicity data) are unavailable, but the comparisons of PCB concentrations in onsite surface soil to threshold values (e.g., those derived by Ontario and Quebec) contribute to the weight-of-evidence regarding the potential toxicity of API/PC/KR surface soils and floodplain sediments. Because the soil threshold values presented in Siegrist (1989) and the sediment toxicity database of Long and Morgan (1991) are general and not site-specific, they can only contribute to multiple lines of evidence concerning the potential toxicity of surface soil or sediment. They are not, therefore, used alone to definitively describe API/PC/KR surface soil or floodplain sediment as toxic.

Media- and Receptor-Specific Dose-based TRVs

Media-specific and receptor-specific TRVs are calculated for a subset of representative receptors. These are dose-based NOAELs/No Effect Levels and LOAELs/Low Effect Levels for terrestrial species.

NOAELs and LOAELs are used as TRVs for red fox, great horned owl, muskrat, mouse, and mink. These TRVs form the basis for calculating hazard quotients and PRGs. Appendices C-2-A and C-2-B present the receptor-specific TRVs for all terrestrial and semi-aquatic receptors. As for mink, TRVs for non-raptor birds are based on the ED_x/EC_x approach introduced in Section 4.1 and discussed above (for mink). A discussion of the specific TRVs for non-raptor birds follows.

The no and low effect TRVs for non-raptor birds are based on a detailed analysis of the effects of PCBs on chicken, one of the best-studied and most sensitive avian receptors of the few species investigated to date. The TRVs are derived from exposure-response curves by interpolation of the effective dose to hens (ED_x) that corresponds to specific relative responses (calculated as the treatment response divided by the control response). The low effect dose is defined as 0.75 of the control response for a toxicological endpoint (ED_{25}, which represents a 25% decrease in response) and the no effect dose is equal to 90% of the control response (ED_{10}, which represents a 10% decrease in response).

The calculated low effect TRV for birds is 0.5 mg/kg_{BW}-d, based on Aroclor 1248, the predominant Aroclor detected in earthworms in the Kalamazoo River floodplain. The calculated no effect TRV for birds is 0.4 mg/kg_{BW}-d, also based on Aroclor 1248. TRVs calculated from exposure to commercial PCB products may underestimate the toxicity of PCBs in the field because of weathering and selective retention in biota. Effects may also be underestimated due to the relatively short-term exposure

durations of the majority of chicken studies (6 to 9 weeks). A single study continued exposure for 39 weeks in a single treatment, and this study showed increased adverse effects in the final weeks (Platonow and Reinhart 1973). However, since chickens are the most sensitive avian species tested to date with PCBs, application of uncertainty factors is not recommended for interspecific or subchronic-to-chronic extrapolations.

Appendix D presents a detailed summary of the ED_x/EC_x method used to derive TRVs for mink and non-raptor birds, and Appendices C-2-A and C-2-B present all the receptor-specific TRVs used to derive hazard quotients and PRGs.

Effects Data Sources (Bird Egg Data)

Bird egg data (Table 4-5b) are compared to egg-based thresholds for adverse effects (Table 4-9).

These effects data are based on relevant endpoints such as hatching success and survival of newly hatched young. Table 4-9 presents the selected bird egg toxicity or effects data used to estimate risks to bird eggs from PCB-contamination.

4.2.2 Stressor-Response Profiles

Stressor-response profiles (Table 4-10) present critical effects data for relevant ecological receptors or appropriate surrogate species that may be exposed to PCBs at the API/PC/KR site. The information presented in Table 4-10 includes relevant toxicity data from literature sources and includes site-specific information to the extent possible. For example, site-specific toxicity values for surface soil are included, along with a threshold streambed sediment PCB concentration, based on site-specific sediment/surface water partitioning, that is protective of aquatic species and piscivorous wildlife. These profiles include information on the lethal and sublethal effects that may be exhibited by exposed organisms correlated to media-specific PCB concentrations. Because effects and other relevant data are sparse for individual Aroclors, and because concentrations of detected PCBs (e.g., Aroclor 1260) approach concentrations of total PCBs measured, all effects data are based on total PCB concentrations. Likely responses to non-chemical stressors are not included in these profiles, but are qualitatively discussed below.

Siltation of Instream Substrate

Siltation, particularly as it contributes to the transport and deposition of PCB-containing residuals waste, may be contributing to ecological stress in the API/PC/KR area. Siltation can result in decreased dissolved oxygen concentrations, greater concentrations of contaminants sorbed onto fine grained sediments and other fine particulate matter, and shifts in macroinvertebrate community structure. For example, certain worm species and midge larvae are better adapted to silt than are stoneflies, caddisflies, and mayflies. Areas of siltation are likely to be characterized by lower species diversity than that found in areas of gravel/cobble. Siltation can directly (by smothering) and indirectly (by changing prey availability and community structure) affect survival of benthic macroinvertebrates. Siltation can adversely affect

fish reproduction and survival by smothering eggs and immature (prior to swim-up) fish. The paper waste residuals are very fine-grained particles which are easily suspended in the water column and when deposited concentrate PCBs in the sediments.

Impoundment Structures/Dams

Impoundment structures or dams can affect the movement of fish in the river, the distribution of PCBs and the exposure potential for aquatic receptors. Although impoundment structures present barriers to fish migration, the greatest threat from these structures is that they form a sink for the PCB residual materials. PCB residuals behind the formerly impounded areas are constantly being eroded into the Kalamazoo River and Portage Creek, and some of which will become bioavailable to aquatic receptors.

The impounded waters behind these structures provide excellent habitat for many game species and it is common to observe anglers at these locations. The exposure potential can be greater for both human and aquatic/terrestrial receptors at these sites.

Disturbed Terrestrial/Riparian Habitat

Most soil-dwelling animals, especially those that have limited mobility, are likely to avoid some terrestrial areas because preferred natural soils are no longer available when covered with significant amounts of contaminated sediments. While the potential toxicity of contaminated soils and streambank sediments cannot be ignored, it is likely that the physical presence of waste soils also affects habitat suitability for certain terrestrial organisms. Where terrestrial vegetation has either not been affected or has been re-established, a variety of terrestrial animals can find cover and food. Additionally, these disturbed areas are attractive sites for the development of "weedy" type plants, which can provide a food source for avian and terrestrial receptors.

4.2.3 Uncertainty Evaluation – Effects Assessment

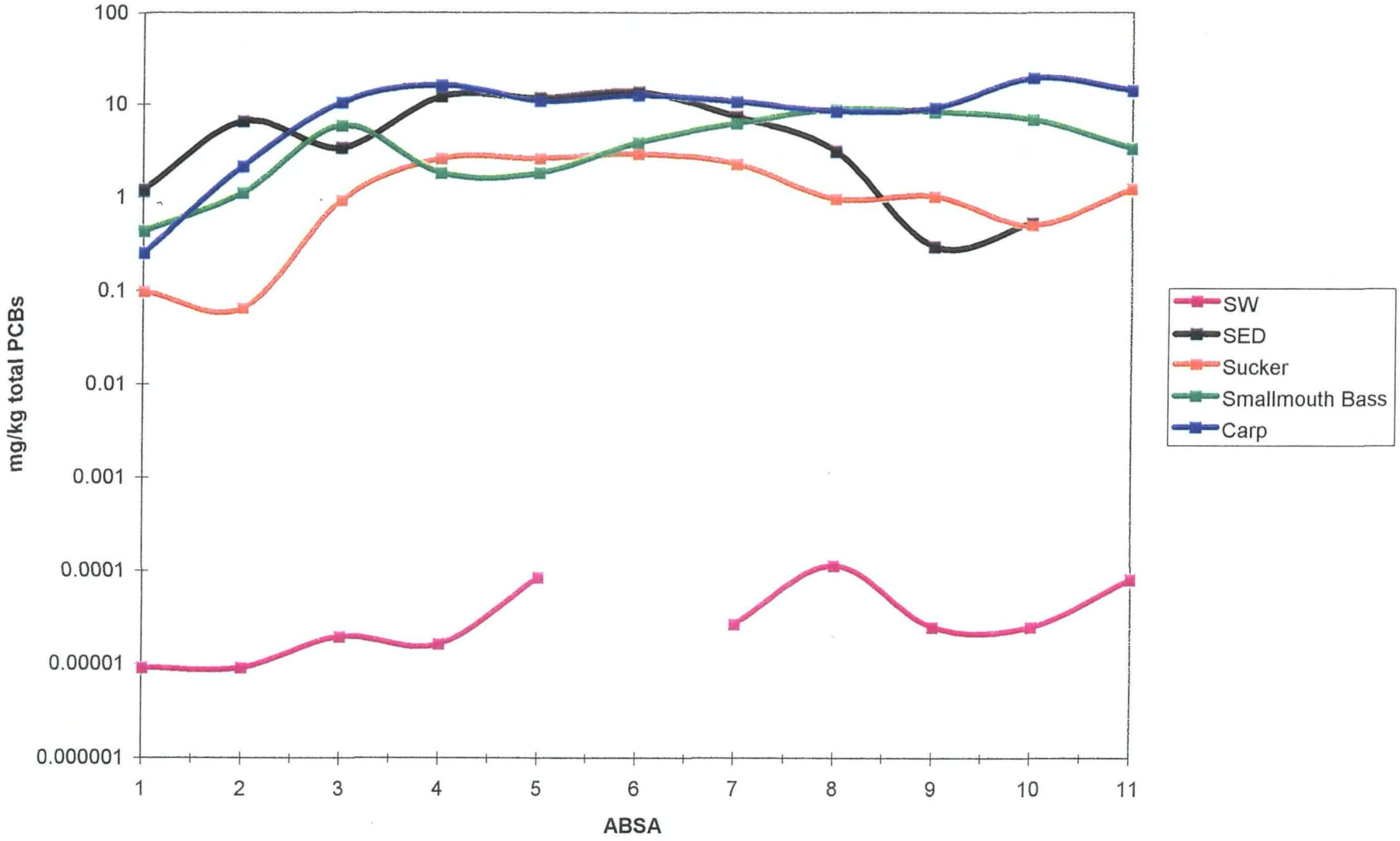
In this section, the major sources of uncertainty in the effects analysis are identified and their potential impact on the ERA is evaluated. Media-specific toxicity data used in this ERA to describe the potential effects to ecological receptors are probably the primary source of uncertainty in the effects analysis.

Extrapolations are often used to relate measurement endpoints (e.g., lethal concentration) to assessment endpoints (e.g., macroinvertebrate abundance) or to relate one measurement endpoint (lethal concentration) to another (sublethal effects concentration). Extrapolations between taxa (e.g., species to species) or between responses (e.g., lethal to sublethal) are commonly used where specific data are limited. The use of these types of extrapolation is a commonly accepted practice but may increase uncertainty in risk assessment. The use of extrapolated data is, therefore, limited as much as possible in this ERA.

Data based on studies specific to the API/PC/KR area are preferred and are, therefore, used as much as possible in this ERA to minimize the uncertainties commonly associated with extrapolating toxicity or other data. Effects data for surface water and sediment contaminants are considered to be associated with low to moderate uncertainty, respectively. The unavailability of relevant site-specific surface water, sediment, and surface soil toxicity data increases uncertainty somewhat, but the availability of site-specific PCB concentrations in exposure media and resident biota helps minimize these uncertainties. There is considerably more uncertainty in the data used to evaluate the potential toxicity of contaminated surface soils because ecotoxicity data for terrestrial biota exposed to PCBs in surface soil are not as abundant as are data for evaluating PCBs in surface water and sediment.

As stated above, where possible, site-specific effects data are used to minimize uncertainty in the effects analysis. Because site-specific data are for the most part limited (to PCB tissue concentrations) or are unavailable (toxicity data), multiple lines of evidence are used to assess potential for ecological effects. This relies on ecological effects data from a large variety of appropriate and relevant data sources, and thus decreases the overall uncertainty compared to assessments based on only one or a few data sources. Several of the values used to quantitatively estimate critical threshold contaminant concentrations (e.g., AWQC, LOAECs, ED_{25}, site-specific tissue concentrations, Co-Occurrence Analysis (COA), Effects Range-Median (ER-M), and others) are often relatively similar in magnitude. These similarities allow greater acceptance of and support for each individual value, and in turn provide justification for using multiple lines of evidence in this ERA.

Figure 4-1
U95 Total PCB Concentrations
in Fish, SW, and SED



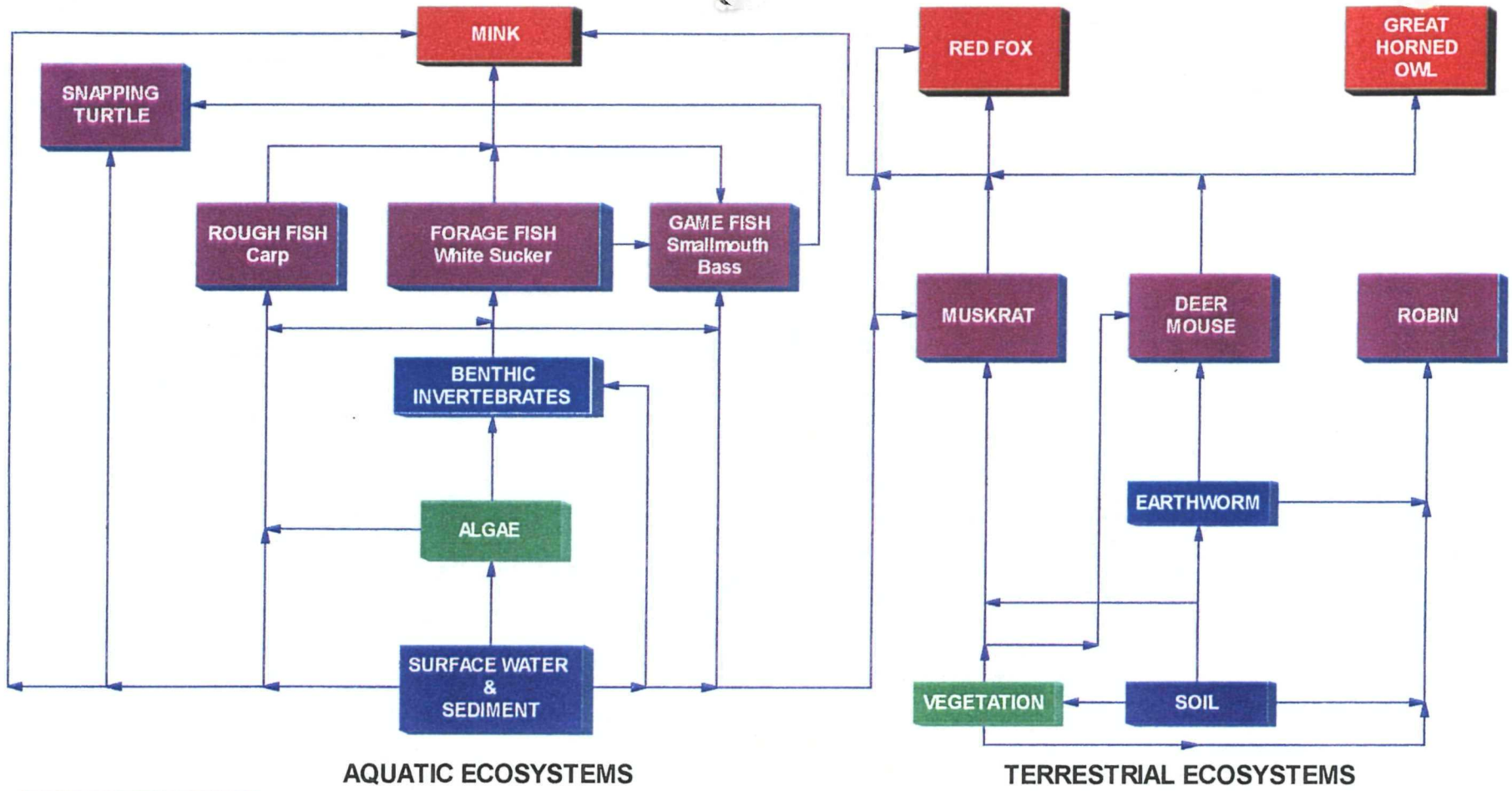


Figure 4-2
PRIMARY PATHWAYS FOR AQUATIC AND TERRESTRIAL ORGANISMS
API/PC/KR

Table 4-1
Sitewide Concentrations in Abiotic Media
API/PC/KR

Chemical	Abiotic Media	Concentration Range	
		Sitewide[1]	(reference area[2])
Aroclor 1016 Aroclor 1221 Aroclor 1232 Aroclor 1242 Aroclor 1248 Aroclor 1254 Aroclor 1260	The following media types were analyzed for individual Aroclors and Total PCBs: Surface Water (SW) Streambed Sediment (SED) Floodplain Sediment (FP SED) Surface Soil (SS)	Concentration range for individual Aroclors not applicable - ERA is focused on distribution and magnitude of Total PCBs	
Total PCBs	Groundwater (GW, µg/L) Surface Water (SW, µg/L) Streambed Sediment (SED, mg/kg) Floodplain Sediment (FP SED, mg/kg) Surface Soil (SS, mg/kg)	ND - 3 ND - 0.23 ND - 156 ND - 85 0.065 - 34.5	(NA) (ND) (NA) (NA) (ND) - 0.39

1 Sitewide: API/PC/KR except upstream reference area (ABSA 1)
2 Reference Area: ABSA 1
ND Non-detect
NA Data Not Available
Surface soil and FP SED data based on 0-6 inch depth

Table 4-2
Exposure Profile for PCBs - Chemical Properties
API/PC/KR

PCBs	Environmental Persistence	Bioconcentration Potential and Bioavailability
General	All PCBs are environmentally persistent, but less chlorinated Aroclors (e.g., 1016, 1221) are more easily degraded by bacteria than more chlorinated Aroclors such as Aroclors 1254 and 1260 (Eisler 1986).	Influenced by N-octanol/water partition coefficient (K_{ow}) which relates to solubility, and by stearic factors relating to chlorine substitution patterns (Eisler 1986). Bioaccumulation potential directly related to log Kow and stearic effects (Shaw and Connell 1982 in Eisler 1986). Generally, less chlorinated Aroclors are taken up to a lower degree than highly chlorinated Aroclors. An exception is found with Aroclor 1254, which apparently is taken up to a greater degree than all other Aroclors studied, including Aroclor 1260 (Eisler 1986). PCBs concentrate in liver, blood, and muscle in mammals. Generally, PCBs are lipophilic, and are most highly accumulated in fatty tissues. The pattern of Aroclor distribution in biological tissues, especially those of warm-blooded animals, only vaguely resemble the mixtures from which they originated (Hansen, et al. 1983 in Eisler 1986). Most commonly, PCBs measured in tissues are identified as Aroclor 1260. PCB metabolism and bioaccumulation is species-specific, and similar exposures result in different bioaccumulation rates.
Aroclor 1221	Persistent	Low to Moderate Bioaccumulation Potential/Bioavailability[1]
Aroclor 1232	Persistent	Moderate Bioaccumulation Potential/Bioavailability[1] Freshwater bioconcentration factor (BCF) for white sucker (*Catostomus commersoni*) equals 5,500 (Frederick 1975 in EPA 1980).
Aroclor 1016	Persistent	Moderate Bioaccumulation Potential/Bioavailability[1]
Aroclor 1242	Persistent	Moderate to High Bioaccumulation Potential/Bioavailability[1] Freshwater BCFs range from 36,000 (scud, *Gammarus pseudolimnaeus*, Nebeker and Pugilsi, 1974 in EPA, 1980) to 274,000 (fathead minnow, *Pimephales promelas*, Nebeker, et al. 1974 in EPA 1980).
Aroclor 1248	Persistent	High Bioaccumulation Potential/Bioavailability[1] Freshwater BCFs range from 52,000 (bluegill, *Lepomis macrochirus*, Stalling 1971 in EPA 1980) to 120,000 (fathead minnow, DeFoe, et al. 1978 in EPA 1980).
Aroclor 1254	Persistent	High Bioaccumulation Potential/Bioavailability[1] Freshwater BCFs range from 2,700 (phantom midge larvae, *Chaoborus punctipennis*, Mayer, et al. 1977 in EPA 1980) to 238,000 (fathead minnow, Nebeker, et al. 1974 in EPA 1980).
Aroclor 1260	Persistent	High Bioaccumulation Potential/ Bioavailability[1] BCF for fathead minnow equals 270,000 (DeFoe, et al. 1978 in EPA 1980)

[1] Estimated from degree of chlorination and available freshwater BCFs

Table 4-3
Exposure Information for Representative Ecological Receptors
API/PC/KR

Representative Receptor Group	Primary Stressor	Primary Potential Exposure Routes /Processes
Aquatic Plants (e.g., floating and rooted macrophytes and algae)	SW PCBs SED PCBs	SW Contact and Uptake SED/IWContact and IW Uptake
Aquatic Macroinvertebrates (e.g., mayfly larvae)	SW PCBs SED PCBS	SW Contact and Ingestion, Ingestion of PCB-contaminated Prey SED/IW Contact and Ingestion, Ingestion of PCB-contaminated Prey
Freshwater Game Fish (e.g., smallmouth bass)	SW PCBs SED PCBs	SW Contact and Ingestion, Ingestion of PCB-contaminated Prey SED/IW Contact and Ingestion, Ingestion of PCB-contaminated Prey
Freshwater Forage Fish (e.g., white sucker)	SW PCBS SED PCBs	SW Contact and Ingestion, Ingestion of PCB-contaminated Prey SED/IW Contact and Ingestion, Ingestion of PCB-contaminated Prey
Freshwater Rough Fish (e.g., common carp)	SW PCBs SED PCBs	SW Contact and Ingestion, Ingestion of PCB-contaminated Prey SED/IW Contact and Ingestion, Ingestion of PCB-contaminated Prey
Terrestrial Invertebrates (e.g., earthworms)	SS/FP SED PCBs	SS/FP SED Contact and Ingestion
Small Burrowing Terrestrial and Semi-aquatic Mammals (e.g., deer and white-footed mouse, muskrat)	SED/FP SED/SS PCBs	SED/FP SED/SS Contact and Ingestion, Ingestion of PCB-contaminated Vegetation/Prey
Small Omnivorous/Carnivorous Mammals (e.g., mink)	SW/SED/FP SED PCBs	Ingestion of PCB-contaminated Aquatic and Terrestrial Prey
Top Predators (e.g., red fox, great horned owl, bald eagle)	SW/SED/FP SED/SS PCBS	Ingestion of PCB-contaminated aquatic and terrestrial prey

SW	Surface Water
FP SED	Floodplain Sediment/Soil
IW	Interstitial Water
SED	Instream Sediment
SS	Surface Soil

Table 4-4
Potential Exposure via Contaminant Ingestion Pathway for Representative Aquatic and Terrestrial Organisms
API/PC/KR

Representative Receptor Group	Primary PCB Exposure Media	Discussion of Uptake/Ingestion Pathway
Aquatic Plants (e.g., floating and rooted macrophytes and algae)	SW SED	Hydrophobic PCBs in the water column are physically adsorbed on particulate matter, including algal cells (Eisler 1986). In addition, PCBs can be transferred from aqueous solution into algal lipids. These PCBs then can cause direct toxic effects to algae by inhibiting photosynthesis and motility. Finally, PCBs accumulated by algae are readily introduced into aquatic food chains (Rohrer, et al. 1982 in Eisler 1986).
Aquatic Macroinvertebrates (e.g., mayfly larvae)	SW SED	PCBs can be taken up by aquatic macroinvertebrates via ingestion of surface water, sediment, sediment pore water, and PCB-contaminated prey such as algae. Uptaken PCBs can cause direct toxic effects in macroinvertebrates, and can also be passed on to upper trophic level organisms through ingestion of PCB-contaminated macroinvertebrates. In addition, certain types of macroinvertebrates, such as mysid crustaceans in Lake Michigan, have a low assimilation efficiency for PCBs and a high efficiency for fecal excretion of ingested PCBs (Evans, et al. 1982 in Eisler 1986). PCB uptake from sediment by chironomids (midge larvae) can be correlated to sediment PCB concentration (Larsson 1984 in Eisler 1986). PCBs can be transported from aquatic to terrestrial environments via aquatic midge larvae to terrestrial midge adults (Larsson 1984 in Eisler 1986). Terrestrial consumers of adult midges can therefore be indirectly exposed to sediment-source PCBs.
Freshwater Game Fish (e.g., smallmouth bass)	SW SED PREY	More persistent and highly chlorinated PCBs can be found in trace amounts in fish from almost every major river in the United States (Schmitt, et al. 1985 in Eisler 1986). PCB-contaminated sediments and atmospheric deposition are the most important sources of PCBs in fish (Eisler 1986). Several studies reveal downward trends in PCB concentrations in whole body fish from throughout the U.S., especially for less chlorinated PCBs such as Aroclor 1242 (Eisler 1986). Total PCBs in fish measure environmental PCB contamination more reliably than do measurements for specific commercial mixtures such as Aroclor PCBs (Schmitt, et al. 1985 in Eisler 1986). Diet is major route of PCB uptake in most fish, but water can be a major source of PCB uptake in certain species under certain conditions (Greig, et al. 1983 in Eisler 1986). Although lipophilic, PCBs can also be deposited in gonads, eggs, muscle, and skin to varying degrees, depending on fish species (Eisler 1986).
Freshwater Forage Fish (e.g., white sucker)	SW SED	As above, but ingestion of prey less important because of omnivorous diet. Uptake of PCBs expected to be lower than for piscivorous gamefish or bottom dwelling rough fish.
Freshwater Rough Fish (e.g., common carp)	SW SED	As above, but ingestion of prey less important because of mostly herbivorous diet. Incidental ingestion of sediment may be important exposure route for bottom dwelling rough fish such as common carp. Direct contact with and ingestion of PCB-contaminated pore (interstitial) water may greatly increase exposure potential for benthic rough fish such as common carp.
Terrestrial Invertebrates (e.g., earthworm)	SS FP SED	Little data exist on PCB transfer from surface soil and floodplain sediments to earthworms. Earthworms have depurated ingested surface soil (i.e., "empty" earthworms) are expected to have higher whole body PCB concentrations than surface soils from which they were collected because of bioaccumulation.

Table 4-4
Potential Exposure via Contaminant Ingestion Pathway for Representative Aquatic and Terrestrial Organisms
API/PC/KR

Representative Receptor Group	Primary PCB Exposure Media	Discussion of Uptake/Ingestion Pathway
Small Burrowing Terrestrial and Semi-Aquatic Mammals (e.g., deer and white-footed mouse, muskrat)	SED FP SED PREY	Terrestrial burrowing rodents such as the white-footed deer mouse, are likely to ingest PCBs primarily through ingestion of invertebrate prey and plants. Vegetation portion of the diet is expected to contribute only small amounts of PCBs compared to contribution from animal prey. Semi-aquatic burrowing mammals like muskrats that are primarily herbivorous are most likely to take in PCBs through incidental ingestion of PCB-contaminated streambed and floodplain sediments. Omnivorous and herbivorous small mammals are expected to have lower PCB exposures than carnivorous species, especially those that consume substantial amounts of aquatic prey (e.g., mink).
Small Omnivorous/ Carnivorous Mammals (e.g., mink)	PREY	Mink are especially sensitive to PCBs, and their diet includes organisms that are most likely to be highly contaminated with PCBs (rough fish, benthic invertebrates such as crayfish, etc.). Several studies suggest that more highly chlorinated PCBs are eliminated more slowly than lower chlorinated PCBs in semi-aquatic carnivorous mammals studied (Eisler 1986). May be exposed via riverine diet, based predominately on fish, or via wetland diet, consisting of crayfish, muskrat, birds, and amphibians.
Top Predators (e.g., red fox, great horned owl, bald eagle)	PREY	PCB contamination most important to top predators (upper level carnivores) compared to lower trophic level organisms (Shaw and Connell 1982; Malins, et al. 1980 in Eisler 1986). Consumers of PCB-contaminated fish are likely to be at most risk because elevated PCB concentrations are expected in fish and other aquatic biota. Exposure through ingestion of prey must consider exposure frequency and duration as well as diet, and foraging range of top predators is critical to this evaluation.

Table 4-5a
Concentration and Distribution of Total PCBs in Sampled Biota and Abiotic Media
API/PC/KR

Media (ppm ww biota, dw abiotic)	TBSA 11 ABSA 1 reference	ABSA 2	Portage Creek	ABSA 3	TBSA 10 ABSA 4	ABSA 5	ABSA 6 Plainwell	ABSA 7 Otsego	TBSA 3, 5 ABSA 8 Trowbridge	ABSA 9	TBSA 1 ABSA 10 Allegan	ABSA 11
Smallmouth Bass [1]												
(max)	0.62	1.8		15	2.3	7.9	8.3	7.6	11	12	8.4	5.0
(mean)	0.35	0.83		3.6	1.4	4.6	2.5	5.1	6.9	6.5	5.6	2.6
(U95)	0.43	1.1		5.8	1.8	1.8	3.8	6.1	8.7	8.2	6.8	3.3
Sucker [1]												
(max)	0.14	0.8	2.4	1.0	2.9	3.1	4.6	2.8	1.1	1.7	0.92	1.6
(mean)	0.074	0.054	1.4	0.081	2.2	2.2	2.2	2.1	0.78	0.81	0.35	1.1
(U95)	0.096	0.063	1.9	0.90	2.5	2.5	2.8	2.3	0.93	1.0	0.49	1.2
Carp [1]												
(max)	0.41	4.2	10.8*	15	21	14	20	25	14	21	36	32
(mean)	0.20	1.4		8.1	12.8	8.8	8.5	6.3	6.5	5.6	13.2	8.9
(U95)	0.25	2.1		10.4	16.1	10.7	12.3	10.5	8.3	9.0	19.1	13.9
Terrestrial Plants												
(max)									0.069			
(mean)									0.023			
Earthworm [1]												
(WB max)	ND				0.66				3.2 (TBSA 3) 2.2 (TBSA 5)			
White-footed/ *Deer Mouse* [1]												
(WB max)	ND				0.28				0.45 (TBSA 3) 0.38 (TBSA 5)		0.35	
Muskrat [2]												
(WB max)	ND						0.6	0.2	2.9		1.1	
(liver max)	ND						0.7	0.3	1.2		0.5	
Mink [2]												
(WB max)	2.0						2.6	none	5.6		3.2	
(liver max)	1.5						2.4	collected	2.4		12.5	
Surface Water [3]												
(max)	0.0000075	0.0000075	0.000230	0.000048	0.000035	0.000091	no data	0.000071	0.000120	0.000052	0.000028	0.00012
(mean)	0.0000063	0.0000063	0.000058	0.000015	0.000013	0.000062	no data	0.000022	0.000075	0.000020	0.000018	0.000059
(U95)	0.0000088 (ABSA 1-2)	0.0000088 (ABSA 1-2)	0.000059	0.000019	0.000016	0.000081	no data	0.000026	0.000108	0.000024	0.000024	0.000077
Streambed SED [3]												
(max)	no data	2.4	120	86	44	100	94	156	91	7.2	0.73	1.4
(0-6□) (mean)	no data	0.91	31.3	2.3	1.6	6.1	5.4	4.9	2.9	2.4	0.20	0.27
(U95)	no data	1.2	47.1	6.5	3.4	12.2	11.8	13.6	7.3	3.1	0.30	0.53

Table 4-5a
Concentration and Distribution of Total PCBs in Sampled Biota and Abiotic Media
API/PC/KR

Media (ppm ww biota, dw abiotic)	TBSA 11 ABSA 1 reference	ABSA 2	Portage Creek	ABSA 3	TBSA 10 ABSA 4	ABSA 5	ABSA 6 Plainwell	ABSA 7 Otsego	TBSA 3, 5 ABSA 8 Trowbridge		ABSA 9	TBSA 1 ABSA 10 Allegan	ABSA 11
FP SED [4]													Ottawa Marsh
(max)	no data	no data	no data	no data	no data	85	no data	36	81		no data	no data	0.04 - 2.8
(mean)	no data	no data	no data	no data	no data	10.9	no data	8.4	12.3		no data	no data	(x = 0.77)
(U95)	no data	no data	no data	no data	no data	16.2	no data	11.7	15.9		no data	no data	Potaw. Marsh
													0.04 - 1.97
													(x = 0.37)
									TBSA 3	TBSA 5			
Surface Soil [5]													
(max)	0.39	no data	no data	no data	10.2	no data	no data	no data	32.6	34.5	no data	0.23	no data
(mean)	0.21	no data	no data	no data	6.5	no data	no data	no data	24.5	25.1	no data	0.17	no data
(U95)	0.33	no data	no data	no data	8.9	no data	no data	no data	28.3	30.2	no data	0.23	no data
Mean Streambed SED/SW Partition Factor (Kd) [6]	301,712			342,105	212,500			523,077			129,167		

ND PCBs Not Detected
no data no recent data available for location or media type
NA Not applicable
* Estimated from filet and remaining carcass PCB concentrations (0.90 * PCB conc of remaining carcass: 0.90*12 mg/kg)

Footnotes:
1) Blasland, Bouck & Lee, Biota Investigation, July 1994.
2) MDNR, June 1994
3) Blasland, Bouck & Lee TM16, March 1995 (SW PC, ABSA 3,4,7,9,10) and TM10, April 1994 (SED ABSA 3,4,5,6,7,8,9)
Blasland, Bouck & Lee Description of the Current Situation, May 1992 (SED PC, ABSA 2, 10, 11 and SW ABSA 1,2,5,8, 11)
Surface Water Data for ABSAs 1 and 2 from samples taken at location near border of ABSA 1 and 2
Surface Water Data for ABSAs 1 and 2 estimated from two samples, less than detection limit, using half the detection limit
4) Blasland, Bouck & Lee, Former Impoundment Sediment and Geochronologic Dating Investigation, 1994, includes data analyzed in 1997 (ABSA 11 data from wetland sediments/soils)
Blasland, Bouck & Lee Description of the Current Situation, 1992 (ABSA 10, single sample)
5) Blasland, Bouck & Lee, Results of Phase I TBSA Soil Sampling, February 1994
6) K_d calculated only for ABSAs where reasonably synoptic (1993/1994) SED data were collected

Table 4-5b
Concentration and Distribution of Total PCBs in Bird Eggs
API/PC/KR

Species	PCB Conc (mg/kg)	Location	Year Collected	Collected/Analyzed by	Reference
RW Blackbird	1.64	Trowbridge Dam	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
	1.61	Trowbridge Dam	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
	0.0094	Ottawa Marsh, ASGA	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
	1.77	Otsego Dam	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
	1.05	Otsego Dam	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
Robin	3.77	Plainwell Dam	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
	0.405	Plainwell Dam	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
GH Owl	22.46	Caulkin's Dam, ASGA	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
	90.8	Koopman's Marsh, ASGA	1993	C. Mehne/Animal Health Diag. Lab., Lansing, Michigan	2
	15.94	High Banks Game Refuge, ASGA	1994	C. Mehne/Illinois Dep. of Agriculture, Centralia, Illinois	2
Wood Duck	0.736	Otsego Dam	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
	0.265	Ottawa Marsh, ASGA	1994	C. Mehne/Illinois Dep. of Agriculture, Centralia, Illinois	1
	0.446	Ottawa Marsh, ASGA	1994	C. Mehne/Illinois Dep. of Agriculture, Centralia, Illinois	1
	0.315	Ottawa Marsh, ASGA	1994	C. Mehne/Illinois Dep. of Agriculture, Centralia, Illinois	1
	0.446	Ottawa Marsh, ASGA	1994	C. Mehne/Illinois Dep. of Agriculture, Centralia, Illinois	1
	0.373	Ottawa Marsh, ASGA	1994	C. Mehne/Illinois Dep. of Agriculture, Centralia, Illinois	1
GB Heron	1.48	ASGA, Ottawa Marsh	1993	C. Mehne/Animal Health Diag. Lab., Lansing, Michigan	2
	4.74	ASGA, Ottawa Marsh	1993	C. Mehne/Animal Health Diag. Lab., Lansing, Michigan	2
	7.67	ASGA, Ottawa Marsh	1993	C. Mehne/Animal Health Diag. Lab., Lansing, Michiganl	2
	2.30	ASGA, Ottawa Marsh	1993	C. Mehne/Animal Health Diag. Lab., Lansing, Michigan	2
	2.31	ASGA, Ottawa Marsh	1993	C. Mehne/Animal Health Diag. Lab., Lansing, Michigan	2
	44.38	ASGA, Ottawa Marsh	1993	C. Mehne /Animal Health Diag. Lab., Lansing, Michigan	2
Wood Thrush	1.93	Plainwell Dam	1995	C. Mehne /Animal Health Diag. Lab., Lansing, Michigan	2

Species	PCB Conc (mg/kg)	Location	Year Collected	Collected/Analyzed by	Reference
Yellow Warbler	1.31	Otsego Dam	1995	C. Mehne/A.D. Little Lab., Cambridge, Massachusetts	1
RT Hawk	2.31	High Banks Game Refuge, ASGA	1993	C. Mehne/Animal Health Diag. Lab., Lansing, Michiganl	2
	4.47	Caulkins Dam, ASGA	1994	C. Mehne/Illinois Dep. of Agriculture, Centralia, Illinois	2
	27.12	Ottawa Marsh, ASGA	1994	C. Mehne/Illinois Dep. of Agriculture, Centralia, Illinois	2
Bald Eagle	102.29	Ottawa Marsh, ASGA	1994	J. Marshall and C. Mehne/Mississippi State Chem. Lab, Mississippi State, Mississippi	2
	123.27	Ottawa Marsh, ASGA	1994	J. Marshall and C. Mehne/Mississippi State Chem. Lab, Mississippi State, Mississippi	3
	53.34	Highbanks Game Refuge, ASGA	1996	J. Marshall and C. Mehne/Mississippi State Chem. Lab, Mississippi State, Mississippi	2
	31.68	ASGA	1996	J. Marshall and C. Mehne/Mississippi State Chem. Lab, Mississippi State, Mississippi	2

References

1. Stratus Consulting Inc. 1999a. Laboratory Data Sheets and Chain of Custody Forms, Copies from D. Beltman, Stratus Consulting. Laboratory Data from 1995 Collection of Bird Eggs for PCB Analysis. Submitted to Camp Dresser & McKee Inc. (CDM) in September 1999.
2. C. Mehne 1994 in MDEQ, MDAG, USFWS, NOAA 2000 - Michigan Department of Environmental Quality (MDEQ), Michigan Department of Attorney General (MDAG), U.S. Fish and Wildlife Service (USFWS), and National Oceanic and Atmospheric Administration (NOAA). 2000. Notice of Intent to Perform an Assessment and Preassessment Screen. Kalamazoo River Environment Site, Michigan.
3. Letter from D. Best, USFWS, to S. Cornelius, MDEQ, 1996

Table 4-6
Measured Soil-to-Terrestrial Plant BAFs for PCBs
(garden plot data, ABSA 8, CDM 2000)
API/PC/KR

Soil PCB Conc. (mg/kg)	Plant PCB Conc. (mg/kg)	Plant Species	Soil to Plant BAF
3.33	0.0236	Peppers	0.0071
3.33	0.0415	Carrots	0.0125
3.33	<0.0025	Tomatoes	<0.0008[1]
3.33	0.0093	Rhubarb	0.0028
16.7	0.00318	Potatoes	0.00019
0.66 and 4.04	0.00931	Horseradish	0.008 (mean)
0.66 and 4.04	0.025	Cucumber	0.022 (mean)
0.66 and 4.04	0.0692	Lettuce	0.061 (mean)
Mean	**0.016**		
U95 BAF	**0.037**		

[1] 0.0008 used as BAF for fruits and berries in food chain modeling

Table 4-7
Literature-Based Soil-to-Terrestrial Plant BAFs for PCBs
API/PC/KR

Plant BAF	PCB Soil Application Rate	Receptor	Method	Reference
0	0.05, 0.5, 1 ppm	Carrot, radish	Experimental	Moza, et al. 1976 and Wallnofer, et al. 1975 in Pal, et al. 1980
0	Unknown	Mature tomato plants	Experimental	Wallnofer 1973 - 1974 (unpub) in Pal, et al. 1980
0.0008	-	**Green tomatoes (represents fruit/berries)**	**Measured, Co-Located Soil and Plant**	**CDM 2000**
0.002	100 ppm	Soybean sprouts	Experimental	Suzuki 1977 in Pal, et al. 1980
0.01	0.3 ppm	Sugarbeet leaves	Experimental	Wallnofer, et al. 1975 in Pal, et al. 1980
0.015	–	Aboveground vegetation	Theoretical, log TF=1.588-log (K_{ow})	Travis and Arms 1988
0.016	0 - 1,000 ppm	Soybean	Experimental	Weber, et al. 1979 in Pal, et al. 1980
0.03	0.17 B 0.24 ppm	Sugarbeet leaves	Experimental	Moza, et al. 1978b in Pal, et al. 1980
0.04	–	**8 Crop Species (all tissues)**	**Measured, Co-Located Soil and Plant**	**CDM 2000**
0.07	0.17 B 0.24 ppm	Sugarbeet roots	Experimental	Moza, et al. 1978b in Pal, et al. 1980
0.16	100 ppm	Carrot roots	Experimental	Iwata, et al. 1974 in Pal, et al. 1980
0.16	0.05, 0.5, 5 ppm	Carrot roots	Experimental	Wallnofer, et al. 1975 in Pal, et al. 1980
0.17	0 - 1,000 ppm	Fescue	Experimental	Weber, et al. 1979 in Pal, et al. 1980
0.25	1 ppm	Carrot roots and leaves	Experimental	Moza, et al. 1976 in Pal, et al. 1980
0.5	0.3 ppm	Sugarbeet whole plant	Experimental	Wallnofer, et al. 1975 in Pal, et al. 1980
0.80	0.17 B 0.24 ppm	Weeds	Experimental	Moza, et al. 1978b in Pal, et al. 1980
0.96	1 ppm	Weeds	Experimental	Moza, et al. 1976 in Pal, et al. 1980
1.3	1 - 2 ppm	Fresh plant B barley	Mean of measured conc in plant/mean measured conc in soil	Trapp, et al. 1990

Table 4-8
Calculated Aquatic BCFs[1]/BSAFs[1] and Terrestrial BAFs[1] for Representative Food Web Species (based on primary exposure media)
API/PC/KR

Location	SM Bass BAF (SW)	SM Bass BSAF (SED)	Sucker BAF (SW)	Sucker BSAF (BSAF)	Carp BAF (SW)	Carp BSAF (SED)	Earthworm BAF[2] (SS)	White-footed/Deer Mouse BAF (SS)
ABSA 3	305,000	0.9	47,000	0.1	547,000	1.6		
ABSA 4 TBSA 10	113,000	0.5	156,000	0.7	1,000,000	4.7	0.07	0.03
ABSA 5	NA	0.1	NA	0.2	NA	0.9		
ABSA 6	NA	0.3	NA	0.2	NA	1.0		
ABSA 7	235,000	0.4	88,000	0.2	404,000	0.8		
ABSA 8 TBSA 3, 5	NA	1.2	NA	0.1	NA	1.1	0.113 (TBSA 3) 0.073 (TBSA 5)	0.016 (TBSA 3) 0.013 (TBSA 5)
ABSA 9	342,000	2.6	42,000	0.3	375,000	2.9		
ABSA 10/TBSA 1	NA	NA	NA	NA	NA	NA	0.109	1.52
Average	249,000	0.88	83,000	0.28	583,000	1.9	0.09	0.40
Average FISH BAF = 305,000 **Average FISH BSAF = 1.02**								

[1] BCFs/BAFs based on U95 PCB Conc (biota)/U95 total PCB Conc (exposure media) Data from Table 4-5a. Values are derived only for locations where reasonably synoptic data were collected
Values are rounded to the nearest one thousand. SW: Surface Water SED: Instream Sediment SS: Surface Soil/Floodplain Sediment from TBSAs
[2] Worm BAFs based on depurated carcass (measured).
NA: Not Applicable because 1) media quality and/or biological data not collected or 2) PCBs were not detected in sampled biota.

Table 4-9
Adverse Effects Associated with Bird Egg PCB Concentrations
API/PC/KR

Species	Egg PCB Conc (mg/kg)	Effect	Reference
Chicken	0.36	NOAEC, egg hatchability	Scott 1977 in 2
	0.95	NOAEC, egg hatchability	Britton and Huston 1973 in 2
	1.5	LOAEC, egg hatchability	Britton 1973 in 1
	2.5	LOAEC, egg hatchability	Scott 1977 in 1
	2.8	**Mean NOAEC**	Calculated, N = 4
	3.0	egg hatchability	Brunstrom 1988 in 1
	4.0	LOAEC, deformities and egg hatchability	Tumasonis, et al. 1973 in 2
	4.8	egg hatchability	Lillie 1975 in 1
	<5.0	NOAEC, egg production and female fertility	Platonow and Reinhart 1973 in 2
	5.0	LOAEC, egg production and femaile fertility	Platonow and Reinhart1973 in 1 and 2
	6.2	**Mean LOAEC**	Calculated, N = 6
	5.0	NOAEC, egg hatchability and 2-fold increase in deformities	Summer et al. 1996 a,b
	24	LOAEC, egg hatchability and 2-fold increase in deformities	Summer et al. 1996 a,b
Ring-necked Pheasant	1.0	egg lethality	Brunstrom 1986 in 1
	1.8	egg hatchability	Dahlgren 1972 in 1
	16	egg lethality	Peakall 1972 in 1
Tree Swallow	5.7	LOAEC, reproductive behavior	McCarty and Secord 1999 in 2
Herring Gull	5	Egg hatchability	Ludwig 1993 in 1
Foster's Tern	4.5	NOAEC, hatching success	Kubiak, et al.1989 in 2
	7.0	NOAEC, population size or reproductive success	Bosveld and Van den Berg 1994 in 2
	19.0	LOAEC, population size or reproductive success	Bosveld and Van den Berg 1994 in 2
	22.2	LOAEC, egg lethality	Kubiak, et al.1989 in 1
Caspian Tern	4.2	LOAEC, egg hatchability	Yamashita 1993 in 1
Double-crested Cormorant	3.5	egg hatchability	Tillitt 1993 in 1
Bald Eagle	1.5	NOAEC (est. from mean LOAEC/10) 5	Calculated, LOAEC N = 5
	4.0	LOAEC, egg lethality	Kubiak 1991 in 1
	4.0	LOAEC, population size or reproductive success	Ludwig et al. 1993 in 2
	4.5	LOAEC, 40% decrease in productivity	Wiemeyer 1984
	7.2	NOAEC, "successful" nests	Wiemeyer et al. 1984
	7.7	Mean LOAEC	Calculated, N = 5
	13	LOAEC, "unsuccessful" nests	Wiemeyer et al. 1984
	13	LOAEC, population size or reproductive success	Bosveld and Van den Berg 1994 in 2

1: RCG/Hagler, Bailly, Inc. 1994
2: Stratus Consulting 1999b

Table 4-10
PCB Stressor-Response Profiles
API/PC/KR

Chemical Stressor	Media of Concern	Measurement Endpoint Concentrations	Measurement Endpoint Data Data Type/Species/Effects	References
Total PCBs (µg/L)	SW	0.00012	Wildlife Protection Criterion for Surface Water - Michigan	Act 451 1994, Part 4
		0.0016	Site-specific value to protect mink. Based on mean site-specific BAF for fish (305,000) and dietary no effect concentration for mink (0.5 mg/kg).	See text
		0.00197	Site-specific value to protect mink. Based on mean site-specific BAF for fish (305,000) and dietary low effect concentration for mink (0.6 mg/kg).	See text
		0.014	Chronic Ambient Water Quality Criterion	EPA 1980
		0.14	Lowest chronic value, freshwater aquatic plants	Suter and Tsao 1996
		0.2 – 9	Range of chronic values (mean of ranges) for Aroclors 1242-1260, fathead minnow	EPA 1980
		0.8 – 15	Range of chronic values (mean of ranges) for freshwater invertebrates	EPA 1980
Total PCBs (mg/kg) (Aquatic/Semi-aquatic/Wetland)	SED FP SED	0.0029	Freshwater Screening Level Concentration (SLC)	Long & Morgan 1991
		0.01	No Effect Level, benthic organisms, Ontario	Persaud, et al. 1993
		0.054 – 3.1	Range of apparent effects concentrations (AET), multiple species	Long & Morgan 1991
		0.07	Lowest Effect Level, benthic organisms, Ontario	Persaud, et al. 1993
		0.1	Carp-based values based on GLI default values to protect mink	See Table 5-5 (MDEQ-SWQD)
		0.37	Concentration at which adverse effects are always observed	Long & Morgan 1991
		0.4	Effects Range-Median (ER-M)	EPA 1988b see text, EP approach*
		0.5	Calculated value to allow IW to remain below site-specific no effect SW threshold (0.0016 µg/L)	EP Approach/ Site-specific
		0.6	Calculated value to allow IW to remain below site-specific low effect SW threshold (0.00197 µg/L)	EP Approach/ Site-specific
		3.5	Calculated value to allow IW to remain below chronic AWQC (theoretical Kd)	EP Approach
		4.2	Calculated value to allow IW to remain below chronic AWQC (site-specific Kd: 302,000)	EP Approach

Table 4-10
PCB Stressor-Response Profiles
API/PC/KR

Chemical Stressor	Media of Concern	Measurement Endpoint Concentrations	Measurement Endpoint Data Data Type/Species/Effects	References
Total PCBs (mg/kg) (Terrestrial/upland)	FP SED SS	0.1	"A" concentration (background pollution), Quebec	Siegrist 1989
		1	"B" concentration (threshold), Quebec	Siegrist 1989
		6.5 – 21	Range of no effect PRGs (API/PC/KR-specific) to protect terrestrial / upland receptors (lowest value for robin)	See text
		10	"C" concentration, (contaminated), Quebec	Siegrist 1989
		8.1 – 63	Range of low effect PRGs (API/PC/KR-specific) to protect terrestrial / upland receptors (lowest value for robin)	See text

SW: Surface Water SED: Sediment FP SED: Floodplain Sediment/SS: Surface Soil
Equilibrium Partitioning approach (SED CONC=KD*IW CONC), (Site-specific: mean Kd=302,000, IW CONC = Chronic AWQC (0.000014 mg/l)

(Theoretical): SED CONC (mg/kg) = KD*IW CONC (mg/L)
KD = Koc * Foc
Foc = 0.084 (sitewide mean Foc)
KD = 2,944,422 * 0.082 = 247,331
log Koc = 0.937 log Kow - 0.006 (EPA Foc 1988b) = 6.469 (Koc = 2,944,422)
Mean log Kow (Aroclor 1260) = 6.91 (EPA 1988b)
SED CONC (mg/kg) = KD*IW CONC (mg/L
3.5 mg/kg = 247,331* 0.000014 mg/L

5 Section Five

Section 5
Risk Characterization

Risk characterization integrates exposure data (e.g., PCB concentrations in surface water) and effects data (e.g., concentrations of PCBs in surface water that protect sensitive resident biota) to estimate risk. For this ERA, the integration of exposure and effects data includes but is not limited to the use of hazard quotients. The hazard quotient approach consists of dividing a single exposure point concentration (e.g., U95 PCB concentration) by a single, preferred toxicity reference value (TRV, e.g., chronic AWQC). The result is the hazard quotient or HQ.

$$HQ = \frac{\textit{Exposure Point Concentration}}{\textit{Toxicity Reference Value}}$$

HQs greater than 1.0 are indicative of risk, while those less than 1.0 indicate no significant risk. Numerically high HQs are not necessarily associated with more severe effects, but instead suggest *greater likelihood* of adverse effects actually occurring. Although such quotients are useful, limiting risk estimation to this simplistic approach fails to consider the variability and uncertainty in exposure and effects data. This ERA therefore supplements the hazard quotient method with other information to provide multiple lines of evidence to reduce uncertainty and increase confidence in risk estimation.

Contributing to the multiple lines of evidence approach used in this ERA are the following:

- comparisons of key exposure data (e.g., mean, U95, maximum PCB concentrations in exposure media) to one or more relevant effects concentrations or thresholds
- the results of the food chain model that estimates PCB dose via dietary exposure
- qualitative evaluations of observations and discussions of ecological significance
- HQs using carefully selected exposure and effects data.

Risks for ecological receptors are assessed on a media-specific basis. There is no appropriate method for combining risks from multiple exposure sources because the relative contribution to total risk from each source (e.g., surface water, sediment, soil, and biota) is unknown. For example, the relative contribution to overall risks to muskrats from surface water, sediment, soil, and food cannot be reliably determined. Also, the relative risk contribution from each source and for each species surely varies both spatially and temporally, especially as seasonal migratory and dietary habits change.

5.1 Risks from Chemical Stressors

The primary risks to ecological receptors at this site are from chemical stressors. A large variety of chemical contaminants have been detected in onsite media and in resident biota. However, this ERA is focused on assessing the risks from PCB exposures via direct contact with contaminated surface water, streambed sediment, floodplain (exposed) sediment, and surface soil, as well as ingestion of PCB-contaminated food items. Risks from drinking surface water and, except for food chain modeling for select species, from incidental ingestion of sediment and soil are not evaluated in this ERA because such risks are likely to be much lower than the risks from direct contact with exposure media and ingestion of contaminated prey. As stated previously, this ERA is focused on the most important stressors (PCBs) and exposure pathways for resident ecological receptors.

The following discussions of media-specific risks are based on presentations of ABSA-specific arithmetic mean, U95, and maximum exposure concentrations and relevant effects concentrations from multiple sources. For estimating risks, the most useful comparisons of exposure and effects concentrations are based on U95 exposure concentrations and site-specific effects concentrations or thresholds. These comparisons best represent reasonable upper-bound estimates of risk for site receptors. Although less useful, comparisons of more general effects concentrations to arithmetic mean and maximum exposure concentrations are included in the following discussions so that other levels of site contamination can be evaluated.

5.1.1 Risk from PCBs in Surface Water

Figure 5-1 presents mean, U95, and maximum total PCB concentrations in surface water for all sampled ABSAs and Portage Creek. Non-detect values are included in the mean and U95 values as either half the detection limit or a randomly assigned value between zero and the detection limit, depending on data source. Also included in Figure 5-1 are horizontal lines representing relevant effects concentrations, thresholds, or criteria for aquatic receptors. These concentrations are, from lowest to highest total PCB concentrations, the

- Michigan state water quality standard to protect wildlife (0.00012 µg/L)
- API/PC/KR-specific No Effect threshold to protect sensitive piscivorous consumers such as mink (0.0016 µg/L), based on 100% fish diet
- API/PC/KR-specific Low Effect threshold to protect sensitive piscivorous consumers such as mink (0.00197 µg/L), based on 100% fish diet
- EPA national chronic AWQC for PCBs (0.014 µg/L), to protect general piscivorous wildlife
- Lowest chronic value for aquatic plants (0.14 µg/L)

- Lowest chronic value for freshwater fish (0.2 µg/L).

These thresholds are taken from Table 4-9. The lowest three values listed are based on protection of wildlife rather than direct effects to aquatic biota. The EPA national chronic AWQC is based on protection of general piscivorous wildlife. The last two values are based on direct toxic effects to exposed aquatic biota. A comparison of these values supports the assumption that PCBs pose greater risks to wildlife, specifically piscivorous mammals and birds, and lower risks to aquatic biota.

Figure 5-1 reveals that all measured surface water total PCB concentrations exceed the Michigan water quality standard for the protection of wildlife and both the No Effect and Low Effect values for mink protection via dietary intake. Except for ABSAs 1 and 2, most surface water PCB concentrations exceed or approach the EPA national chronic criterion of 0.014-µg PCB/L surface water.

Only occasionally have measured surface water PCB concentrations exceeded or approached chronic effects thresholds for fish or aquatic plants. Direct toxic effects to invertebrates (lower range of chronic effects = 0.8 µg/L), or aquatic plants are therefore considered unlikely except at specific locations or times when PCB water column concentrations are likely to be highest (e.g., during storm events).

5.1.2 Risks from PCBs in Streambed Sediment

Figure 5-2 presents mean, U95, and maximum total PCB concentrations in streambed sediment for all sampled ABSAs and Portage Creek. Also included in Figure 5-2 are horizontal lines representing relevant thresholds or PRGs for selected representative receptors. These thresholds or PRGs are, from lowest to highest total PCB concentrations, the

- Sediment value (0.036 mg/kg) associated (based on site-specific sediment-water relationships) with the Michigan state surface water standard (0.00012 µg/L) to protect wildlife

- API/PC/KR-specific No Effect PRG derived to protect sensitive piscivorous consumers such as mink (0.5 mg/kg), based on 100% fish diet, site-specific mean BSAF, and calculated EC_{10} (dietary no effect TRV)

- API/PC/KR-specific Low Effect PRG derived to protect sensitive piscivorous consumers such as mink (0.6 mg/kg), based on 100% fish diet, site-specific mean BSAF, and calculated EC_{25} (dietary low effect TRV)

These sediment thresholds or PRGs are taken from Table 4-9.

Figure 5-2 clearly shows that mean, U95, and maximum total PCB concentrations in streambed sediments exceed all three thresholds or PRGs at ABSAs 2-9. At ABSAs 10 and 11, the maximum detected total PCB concentration in sediment exceeds or approximately equals all thresholds or PRGs.

PCB concentrations in API/PC/KR streambed sediments are likely to pose risks to sensitive benthic aquatic biota (e.g., macroinvertebrates) and water-column biota (e.g., invertebrates and fish) through release of PCBs from sediment particles. Also, sensitive piscivorous consumers such as mink are likely to be adversely affected by PCB-contaminated streambed sediments via the SED-IW-SW-fish pathway. The ingestion pathway is discussed in Section 5.1.4.

5.1.3 Risks from PCBs in Floodplain Sediment and Surface Soil

Figure 5-3 presents mean, U95, and maximum total PCB concentrations in floodplain sediment/soil for all sampled areas. Sample areas include floodplain sediments at the Plainwell former impoundment (ABSA 5), Otsego former impoundment (ABSA 7), and the Trowbridge former impoundment (ABSA 8).

Figure 5-4 presents similar values for PCB concentrations in surface soil for all sampled areas. Surface soil is defined here as floodplain sediment/soil taken from the TBSAs, and these samples may in fact represent semi-aquatic sediments that are covered with water for significant portions of the year. Alternative PRGs such as those derived for protection of mink are more appropriate for floodplain sediments that are frequently inundated. This recommended application of PRGs is based on the direct link between these riparian sediments and aquatic and semi-aquatic food webs.

Also included in Figures 5-3 and 5-4 are horizontal lines representing relevant thresholds or PRGs for potential receptors. The threshold or PRG concentrations for both surface soil and floodplain sediment are, from lowest to highest total PCB concentrations, the

- NOAEL-based PRG for great horned owl (2.9 mg/kg)
- NOAEL-based PRG for red fox (5.9 mg/kg)
- NOAEL-based PRG for American robin (6.5 mg/kg)
- LOAEL-based PRG for American robin (8.1 mg/kg)
- LOAEL-based PRG for great horned owl (8.5 mg/kg)
- NOAEL-based PRG for mouse (21 mg/kg)
- LOAEL-based PRG for red fox (29.5 mg/kg)
- LOAEL-based PRG for mouse (63 mg/kg)

Figure 5-3 reveals that maximum total PCB concentrations in floodplain sediments/soils exceed all NOAEL-based PRGs at all sampled locations. Average and U95 total PCB concentrations at all sampled locations exceed all NOAEL-based PRGs

except the mouse NOAEL PRG. Average floodplain sediment total PCB concentrations at all three former impoundments (Plainwell, Otsego, and Trowbridge) exceed or nearly equal the LOAEL-based PRGs for great horned owl and robin.

For surface soils (Figure 5-4), limited sampling from TBSAs 1, 3, 5, 10, and 11 reveals greatest potential for concern at TBSAs 3 and 5. Mean, U95, and maximum total PCB concentrations in surface soils at TBSAs 3 and 5 exceed all PRGs except the LOAEL PRGs for mouse and fox. Mean, U95, and maximum total PCB concentrations in surface soils at TBSA 10 exceed or approximately equal the NOAEL PRG concentrations for fox and robin and the LOAEL PRGs for robin and owl. PCBs in surface soils at TBSAs 11 and 1 appear to present little risk to most terrestrial receptors.

Surface soils and floodplain sediments have potential to pose risks to sensitive terrestrial receptors that consume PCB-contaminated invertebrates. Terrestrial omnivores such as mice and terrestrial carnivores such as red fox might be at risk if they forage predominately in floodplain areas that are highly contaminated with PCBs. Foraging outside the floodplain, where surface soil PCB concentrations are lower and less variable than floodplain sediments, is likely to reduce risks to terrestrial omnivores and carnivores. Certain songbirds (e.g., vermivores) foraging within the floodplain are predicted to be at substantial risk because elevated PCB concentrations have been measured in surface soil, floodplain sediment, and most importantly, in earthworms. Onsite PCB risks to most terrestrial biota are expected to be substantially lower than risks to piscivorous birds and mammals. Finally, because some floodplain sediments (including some termed "surface soils") are frequently inundated and support aquatic and semi-aquatic biota, the application of PRGs based on protection of mink should be considered for these locations.

5.1.4 Risks from PCBs in Food Items (Ingestion)

Risks to consumers of onsite plants and animals are expected to be highly variable. Only limited site-specific PCB values are available for determining PCB concentrations in site plants. PCBs bioaccumulate in plants to a much lower degree than in animals. However, PCB concentrations in site plants can, based on limited site-specific data and literature soil-to-plant uptake values, be of concern. This is because onsite soil PCB concentrations are sufficiently elevated in some areas to cause elevated PCB concentrations in exposed plants, especially riparian or semi-aquatic plants that grow in aquatic environments or wet soils. It is unknown if the estimated or measured PCB concentration in plants is due primarily to uptake from soil, volatilization from soil, or aerial deposition. Although all three processes have potential to contribute to plant PCB burdens, the dominant process is unimportant to consumers of PCB-contaminated vegetation.

Table 5-1 summarizes the dose estimates from the PCB food web model and presents dose-based LOAELs or Low Effect TRVs (ED_{25}) and NOAELs or No Effect TRVs (ED_{10}) for representative receptors. Table 5-2 presents ranges (No Effect to Low

Effect) of PCB PRGs for terrestrial receptors (mouse, robin, great horned owl, and red fox). These PRGs are based on NOAELs and LOAELs taken directly from the literature, on calculated ED_{25} and ED_{10} values based on multiple studies from the literature, and on dietary data and site-specific PCB concentrations in floodplain sediment/surface soil.

Table 5-3 presents hazard quotients (HQs) for terrestrial and aquatic biota. HQs for mink, bald eagle, robin, owl, fox, mouse, and muskrat are based on estimated doses from the results of food chain modeling (Appendix C-2).

HQ = Daily Dose (mg/kg-d) / NOAEL (or ED_{10}) or LOAEL or (ED_{25}) (mg/kg-d)

Based on the calculated NOAEC-based HQs, mink are at most risk, followed by bald eagle,, great horned owl, American robin, and red fox. White-footed or deer mouse and muskrat appear to be at little or no risk (HQs<1).

Estimated risks to great horned owls should be viewed with caution, based on the level of PCB contamination in great horned owl eggs collected downstream of Lake Allegan. The apparent discrepancies between egg data and relatively low estimated risks based on food web modeling are discussed in subsequent sections of the ERA.

The types of consumers most likely to be at serious risk at this site are consumers of aquatic prey, especially piscivores. Aquatic biota within the API/PC/KR area, especially carp, are much more seriously contaminated with PCBs than are terrestrial biota that are likely to serve as prey for mostly piscivorous predators such as mink. Mink are at most risk from PCB contamination through ingestion of prey because they

- Consume large amounts fish (with seasonal variation) that are highly contaminated
- Are likely to obtain most or all prey within or near aquatic environments within site boundaries and
- Are the most sensitive to PCBs of all animals studied to date (Eisler 1986)

The maximum allowable tissue concentration for dietary items of mink ranges from 0.5 to 0.6 mg/kg, based on the No Effect ED_{10} and the Low Effect ED_{25} values from the studies described in Appendix D. Mink should be adequately protected if the average PCB concentrations of all prey items contain less than 0.5 mg PCB/kg prey. Prey PCB concentrations greater than 0.5 mg/kg are associated with some degree of risk. When the average PCB concentration in mink prey approaches 0.6 mg/kg, measurable adverse effects are expected. These are primarily adverse reproductive effects that can affect population status.

The calculated ED_{10} and ED_{25} values for mink fall within the range of the dietary NOAELs and LOAELs for total PCBs derived by Heaton et al. (1995) of 0.015 and 0.72 mg/kg. The Heaton et al. (1995) NOAEL is based on a daily dose of 0.004 mg/kg bw-

d, while the LOAEL is based on a daily dose of 0.134 mg/kg bw-d. The estimated daily doses of PCBs calculated for mink in this study are 0.091 and 0.11 mg/kg-d (Tables 5-1 and 5-3). The ED_{25} dose is nearly the same value as the LOAEL-based dose derived by Heaton et al. (1995), while the calculated ED_{10} dose exceeds the NOAEL-based dose derived by Heaton et al. (1995).

Estimated doses and corresponding HQs for mink based on food chain modeling are directly related to mink dietary assumptions. Mink diet is expected to vary spatially and temporally, and is likely to differ substantially depending on the predominant foraging areas. Mink foraging along the river are expected to consume more fish and aquatic biota than mink foraging in areas more removed from the river. The latter may consume fewer fish and more birds and small mammals, for example. The fraction of fish in mink diet directly affects the PRGs determined for mink. The mink-based PRGs based on surface water-sediment-fish PCB relationships (presented in Section 4 .2.1) assume a 100% fish diet. PRGs for mink protection would be different (higher) if mink diet was not predominately fish-based. In some cases, food chain modeling can be used to estimate dietary PCB doses. However, food chain modeling based on a highly variable and mostly unknown diet would be associated with considerable uncertainties. Also, the gut contents of the small numbers of mink collected onsite are unlikely to provide much useful information regarding the overall annual diet of mink. Frogs, crayfish, and whole body songbirds, all likely prey of mink, have not been collected onsite and analyzed for PCBs. The assumptions that mink diet is comprised primarily of fish and that fish provide the major source of PCBs to mink are not unreasonable, as discussed below.

U95 PCB concentrations in fish collected from ABSAs 3-9 (the primary areas of impact) range from 0.90 (sucker) to 16.1 mg/kg (carp). Carp collected just downstream of the site, below Allegan Dam, contained up to 36 mg/kg PCBs, and even higher values resulting from long-term monitoring have been recently observed. Where and when readily available, fish are expected to comprise the majority of the diet for mink. This assumption is supported by mink diets for Michigan presented in EPA Exposure Factors Handbook (1993), which suggests that 85 percent of mink diet is comprised of fish.

Fish consumption by certain individual mink, or by most mink during certain seasons, is likely to be supplemented by consumption of mammals, birds, amphibians, reptiles, and invertebrates (e.g., crayfish). Site-specific data are unavailable to assess PCB contamination in crayfish, frogs, and birds, and for this reason food chain modeling based on these dietary items is not performed.

PCB contamination of mammals that may be consumed by mink is expected to vary from low to moderate. PCBs were measured in the whole bodies of muskrat and deer/white-footed mouse and in liver of muskrat. These data are used to estimate doses used to calculate HQs for mink and to support food chain modeling for certain other receptors. Muskrat and mice collected from the API/PC/KR site reveal moderate to relatively low (respectively) whole body PCB concentrations compared to

carp. Maximum whole body total PCB concentrations (wet weight) range from 0.28 to 0.45 mg/kg in mice and up to 2.9 mg/kg in muskrat. These potential prey items are, therefore, expected to contribute low (mice) to moderate (muskrat) levels of PCBs to mink diet. Consumption of muskrat by mink could contribute to adverse effects because in some areas whole body PCB concentrations in muskrat exceed the dietary low effect TRV (0.6 mg/kg) derived for mink. However, muskrat are most likely to make up a large portion of mink diet in areas that do not support fish or in winter when fish and crayfish are not as readily available. Consumption of mice by mink is not a major concern because mean whole body PCB concentrations in sampled mice remained well below the dietary thresholds for mink.

Preliminary data on shrews collected onsite suggests that these animals, as expected from their diet, contain substantially greater PCB concentrations than mice or muskrat. Consumers of shrews would therefore be at greater risk than predators eating mice or muskrat. It is not unreasonable to assume some small portion of mink diet is comprised of shrews. Therefore, food chain modeling that bases small mammal consumption on only mice and muskrat probably underestimates PCB dietary exposures.

Fish contamination is also a critical issue for piscivorous birds, such as bald eagle. Avian predators associated with aquatic environments are likely to be exposed to PCBs primarily through ingestion of fish and other aquatic prey. The selected No Effect and Low Effect dose-based TRVs for birds, based on chicken data, are 0.4 and 0.5 mg/kg-d. The calculated dose for bald eagles, based on the food web model and on input parameters presented in Appendices C-1 and C-2, is 2.1606 mg/kg-d. Bald eagles with a diet similar to that presented in Appendices C-1 and C-2 can therefore be adversely affected by PCB contamination. Because this potential risk is based on a diet of 77 percent fish, risks may be reduced where diets include a smaller proportion of fish or where fish are less contaminated than the values used in the food web model. Preliminary site-specific information on the dietary composition of bald eagles suggests that the 77 percent fish value is appropriate for this site.

Table 5-3 also presents HQs for piscivorous wildlife, which are also protective of aquatic biota. One set of HQs for piscivorous wildlife and aquatic biota is based on a comparison of the average of ABSA-specific U95 value for total PCBs in surface water (0.043 ug/L) to the EPA national chronic ambient water quality criterion (AWQC, 0.014 ug/L). The chronic AWQC for PCBs is intended to protect 95% of aquatic species as well as sensitive piscivorous wildlife species. This comparison reveals that PCB concentrations in the Kalamazoo River and Portage Creek surface water have potential to pose risks to piscivorous wildlife (HQ=3.1, Table 5-3). Additional comparisons are made between the same U95 surface water concentration, NOAECs and LOAECs for various fish and invertebrates. This comparison reveals little or no direct risk to fish and invertebrates (HQs<1).

An important goal for the API/PC/KR site is re-establishment of an anadromous salmonid fishery. Toxicity data indicate that salmonids are likely to be among the most sensitive aquatic biota to PCBs (EPA 1980). The re-establishment of a self-

sustaining salmonid fishery must, therefore, consider PCB effects on salmonid eggs, larvae, and young as well as effects on adult salmonids and prey species consumed by salmonids. In general, early life stages of fish are more sensitive to contaminants than adults, and reproductive success depends on providing safe exposures for these life stages. Obviously, suitable spawning and rearing habitats must also be present if a self-reproducing fishery is to become established in the Kalamazoo River.

5.1.5 Reproductive Risks to Birds (Bird Egg Data)

Many bird eggs have been collected within the site boundaries within the past several years. Most of these were collected from 1993 through 1996. These data are summarized on Table 4-5b, and are used to calculate egg-based HQs. Tables 5-4.a and 5-4.b provide comparisons of egg-based NOAECs and LOAECs for total PCBs to PCB data for birds eggs collected onsite from 1993 to 1996. These comparisons are presented as hazard quotients (HQs) where bird egg PCB concentrations are divided by NOAECs or LOAECs for bird eggs.

Egg-based HQs are calculated using two sets of relevant egg-based toxicity data. First, PCB concentrations in eggs collected onsite are compared to egg-based toxicity values from Table 4-10, resulting in the HQs shown on Table 5-4.a.. The toxicity data shown on Table 4-9 are associated with adverse reproductive effects due to PCB contamination of bird eggs. As noted on Table 4-10, for most tested species, total PCB concentrations in bird eggs ranging about 1 to 2 mg/kg are associated with no adverse effects. Unacceptable adverse effects have been observed in most species at egg concentrations ranging from about 3 to 6 mg/kg. Chickens appear to be among the most sensitive species to PCBs, while Forster's tern appears to be among the most resistant.

Second, PCB concentrations measured in bird eggs collected onsite are compared to egg-based NOAECs and LOAECs derived using the EC_{10} and EC_{25} approach detailed in Appendix D. The HQs resulting from these comparisons are presented on Table 5-4.b, and in general exceed the HQs derived using the toxicity data presented on Table 4-10. These exceedences are likely due to due the sensitivity of chickens to PCBs, and this sensitivity underlies the TRVs derived using the EC_x approach detailed in Appendix D.

Although there are differences in the HQs depending on the source of the toxicity data used (Table 4-10 or Appendix D), the general trends remain the same. The data presented on Tables 5-4.a and b. reveal a wide range of risk estimates (HQs) based on PCB contamination of bird eggs collected onsite. The magnitude of HQs appears directly related to diet. Average PCB contamination of eggs of *piscivorous* birds (bald eagle, great blue heron) is the highest (bald eagle) or among the highest (great blue heron). *Carnivorous* raptors such as red tailed hawk and great horned owl are also associated with elevated PCB contamination of eggs. These species are presumed to feed primarily on terrestrial rodents and birds. *Omnivorous* birds such as robins are associated with moderate risks based on degree of PCB contamination of eggs. PCB

contamination of eggs of *insectivorous* birds (e.g., yellow warbler, red winged blackbird, wood thrush) appears low but possibly significant (HQs range from less than 1.0 to 1.9). Finally, *herbivorous* waterfowl, represented by wood duck, appear to be at low risk based on low levels of PCB contamination in eggs. In summary, PCB contamination of bird eggs can be approximated as follows:

Piscivores > Carnivores > Omnivores > Insectivores > Herbivores

Most of the risk estimates presented on Tables 5-4.a. and b. are more or less expected, given the measured or estimated degree of PCB contamination in dietary items such as fish, rodents, and earthworms. However, the high HQs of red tailed hawk and especially those of great horned owl are unexpected.

PCB contamination of expected major prey items of great horned owl, such as white footed or deer mice, is low, based on measured whole body PCB concentrations in these species collected onsite. PCB contamination of songbirds, the other likely prey item of great horned owls based on dietary studies in Michigan (Appendix C-1), are predicted to be quite high for whole body songbirds based on the selected diet-to-carcass BAF (Appendix C-1). It is currently unclear if great horned owls are obtaining much of their total PCBs from songbirds or from some other unidentified source.

Other potential dietary sources of PCBs to great horned owls include prey with stronger associations with aquatic environments. These may include muskrat (which are associated with moderate levels of PCB contamination), shrews (which appear to have higher PCB concentrations than mice or muskrat based on preliminary data), waterfowl, fish carcasses, other small mammals such as young raccoons or mink, crayfish, and frogs. The aquatic-associated prey items are not expected to be major components of great horned owl diet, but local diet along the river corridor may differ from what is generally expected or reported in the literature.

In summary, there does not appear to be a clear link between PCB levels in floodplain sediments or soils near the nests where owl eggs were taken and the elevated levels of PCBs in owl eggs. For example, eggs taken downstream of Lake Allegan contained PCBs in the range of about 16 to over 90 ppm, yet floodplain sediments in this area remain low, generally less than 1 ppm. Since the primary route of exposure of great horned owls to PCBs is poorly understood at this site, protection of great horned owls and other similar birds should not be the basis of PRGs for floodplain sediment or surface soil.

5.1.6 Sitewide Summary of Risks

Table 5-3 presents the results of a simplified HQ approach (e.g., exposure concentration/effects concentration) that presents risk in a very general manner for representative receptors. This table presents the estimated risks for all representative species of concern based on estimated PCB dose (birds and mammals) or on the sitewide average of U95 SW PCB concentration (aquatic receptors). For risks based on

surface water exposure, the risk estimates consider only the direct potential toxicity to exposed receptors. Risks to aquatic biota resulting from bioaccumulation are not included. Risks to birds and mammals are based on estimated PCB dose compared to no effect and low effect doses from the literature or calculated using the ED_x approach discussed previously (and discussed in detail in Appendix D).

The risks presented on Table 5-3 are based on sitewide averages of (1) U95 total PCB concentrations for abiotic media and fish, and (2) maximum total PCB concentrations for sampled terrestrial biota serving as input to food chain modeling (earthworms, mice, muskrat). These exposure concentrations are used to describe reasonable upper bound exposures across the entire site. For most species or individuals, these risks probably over-estimate actual risks in relatively clean areas. Similarly, these risks are probably under-estimated for highly contaminated areas, often described as "hot spots". Sitewide average risks are therefore unlikely to be highly useful for evaluating location-specific contamination.

5.2 Risks from Nonchemical Stressors

The major non-chemical stressors contributing to biological impairment of the Kalamazoo River are disturbed aquatic and terrestrial habitats. Disturbances of aquatic habitat appear to be primarily caused by conditions related to urban environments and sediment inputs from upstream sources and streambank erosion. Impacts from urbanization may include degradation of streambanks, flow alterations, channelization, etc. Deposition of fine-grained sediments often results in the loss or degradation of preferred habitats for most desirable benthic macroinvertebrates. Spawning areas for many fish species would also be similarly affected where deposition of fine-grained sediments predominates. Also, certain fish species would be indirectly affected by conditions that impaired the colonization, survival, growth, and reproduction of prey species, including benthic macroinvertebrates.

Finally, fine-grained sediments commonly contain higher concentrations of chemicals than coarser materials. Fine-grained sediments within the Kalamazoo River channel are expected to be more toxic to aquatic life than large grained sediments because of increased sorption of PCBs on fine-grained materials. Sedimentation in the Kalamazoo River is, therefore, a source of both physical (habitat disturbance) and chemical (PCB toxicity) stress on resident aquatic biota.

Terrestrial/upland habitats are also disturbed in some areas. This disturbance includes long-term impacts related to urbanization and more temporary impacts in some areas related to remedial activities. Also, the physical presence of PCB-contaminated surface soils and deposited sediments, and the toxic conditions associated with these media, preclude the maintenance of a diverse and healthy plant community in some cases. Physical or chemical stressors that impair the establishment and/or maintenance of vegetative growth can adversely affect animals that require sufficient food (herbivorous species) and cover (most all species) for survival and reproduction. Sensitive soil-dwelling animals, along with sensitive plant

species, are not expected to inhabit areas where PCB contaminated media substantially replaces or covers native soils. The expected decrease in abundance and diversity of soil biota, including important microorganisms critical to nutrient recycling, can be due to both physical (displacement or covering of native soil) and chemical (toxicity) causes. As stated previously, PCB-contaminated streambank sediments/surface soils are also likely to contribute to impairment of the Kalamazoo River through erosion and runoff.

5.3 Risk Summary and Ecological Significance

Section 5.3.1 summarizes the risks for this site. The ecological significance of these risks is also included in this summary. The risk summary is followed (Section 5.3.2) by other observations or information that contributes to the multiple lines of evidence presented in the ERA.

5.3.1 Risk Summary

Table 5-3 presents the summary of risks for all representative ecological receptors based on doses (terrestrial receptors) or direct toxicity (aquatic receptors). Figures 5-5 and 5-6 present total PCB concentrations in terrestrial biota and fish, respectively, for sampled locations. Figures 5-7, 5-8, and 5-9 present the mean, U95, and maximum whole body total PCB concentrations measured in smallmouth bass, carp, and suckers, respectively. These values are overlaid with the calculated no effect (EC_{10}) and low effect (EC_{25}) dietary concentrations associated with critical reproductive effects in mink.

The risks from the sitewide representation presented in Table 5-3 are considered in addition to the location-specific distribution and concentration of PCBs described in previous sections (e.g., Table 4-5) and presented in part of Figures 5-5 and 5-6. The data presented in Figures 5-7, 5-8, and 5-9 are also used to describe important risk-related information. Together this information is used to summarize risks in the following discussion.

- Most aquatic biota such as invertebrates and fish are unlikely to be adversely affected by direct contact with and ingestion of surface water because of relatively low PCB toxicity to most aquatic biota. Adverse effects may be exhibited by sensitive aquatic biota such as some species of aquatic plants, but such effects are likely to be spatially and temporally limited.

- PCB contamination of surface water and streambed sediment (and floodplain sediment that is frequently inundated or has potential to erode into the river) is likely to adversely affect sensitive piscivorous predators such as mink through consumption of PCB-contaminated prey, especially fish.

  - Impaired reproduction of mink and ultimately decreases in mink populations are the most likely effects of PCB contamination in aquatic prey. Henry, et al. (1998) demonstrated that concentrations of PCBs in smallmouth bass from a

remote lake in the Upper Peninsula of Michigan were of concern to mink populations, even with the low levels of PCBs in fish tissue from this lake.

- Other piscivorous predators, such as bald eagles, also appear to be at high risk based on the exposure assumptions presented in Appendices C-1 and C-2. The level of PCB contamination in eagle eggs suggests that these assumptions are valid. Furthermore, field investigations of bald eagles by U.S. Fish and Wildlife indicate there has been a loss of reproductive capacity and decrease in the populations of bald eagles within the site boundaries.

- Terrestrial and semi-aquatic biota may be at risk from PCB-contaminated floodplain sediment and surface soil, depending on life history (e.g., foraging behavior, diet, mobility) and sensitivity to PCBs.

  - Omnivorous birds (represented by the robin) that consume substantial numbers of soil invertebrates, such as earthworms, appear to be at moderate but significant risk.

  - Carnivorous terrestrial species (represented by the red fox) are unlikely to be at significant risk unless foraging is concentrated in riparian areas with contaminated floodplain sediment and diet consists of prey that (1) reside in PCB-contaminated areas, and (2) have taken up substantial amounts of PCBs.

  - Omnivorous terrestrial species (represented by mice) are also unlikely to be at significant risk unless they reside in the most contaminated areas. PCB uptake in mice appears to be low.

  - Semi-aquatic herbivorous mammals (represented by muskrat) may be at risk from PCB contamination because estimated dietary doses exceed recommended threshold values for rats. This conclusion is based on the assumption that laboratory rats and muskrats are equally sensitive to PCBs via ingestion. Muskrats contaminated with PCBs may also cause adverse effects to muskrat predators because some muskrats contain PCBs in excess of recommended dietary limits for PCB-sensitive predators such as mink.

5.3.2 Other Supporting Information

This section presents a compilation of qualitative findings, anecdotal information, and observations that support the risk estimates presented in this ERA. This information by itself cannot be used to derive risks or characterize the site in any particular way. However, the following information is considered useful to add to the multiple lines of evidence presented in this ERA. The following is therefore intended to support the conclusions and assumptions presented and discussed in this ERA.

- Yearling smallmouth bass (<8 months old) had whole body PCB concentrations exceeding 3 mg/kg, well above the calculated dietary low effect concentration to protect mink (0.6 mg/kg)

- Mink trapping success was inversely correlated to level of PCB contamination at TBSAs

  - Habitats were similar at all locations, based on both qualitative assessments by local trappers and on preliminary data from quantitative habitat assessments conducted by MSU

  - Equal trapping time was expended at each location

- Bald eagles at the Allegan State Game Area have had very poor reproductive success (Best 1999)

  - Since monitoring began in 1960, two fledged young have been produced in 15 breeding attempts (0.13 fledged young per occupied breeding area - 0.7 is indicative of stable population) (Best 1999)

- Great horned owl eggs from the Allegan State Game Area contained up to 90.8 mg/kg total PCBs

- Redtail hawk eggs from the Allegan State Game Area contained up to 27.1 mg/kg total PCBs

- Eggs of other bird species from the Allegan State Game Area contained low to moderate levels of PCBs

- Previously observed great blue heron colony alongside Kalamazoo River is gone, and heron eggs from the Allegan State Game area contained PCBs at concentrations averaging over 10 mg/kg (max over 40 mg/kg)

- Regional bald eagle sightings reported to MDNR have all been from alongside the Kalamazoo River within the site boundaries

  - This supports the use of 1.0 for a SFF for bald eagles

- Non-normalized average BSAFs for other sites in the Great Lakes region consistently range from a little less than 1 to about 2

  - Average BSAFs for this ERA range from 0.28 to 1.9, with an overall average of 1.02

- Muskrat and mink liver PCB concentrations (mg/kg wet weight) support the conclusion of significant exposure to PCBs.

- Maximum PCB concentrations in muskrat liver range from non-detect (ABSA 1, reference) to 1.2 mg/kg (Trowbridge).

- Maximum PCB concentrations in mink liver range from 1.5 mg/kg (ABSA 1, reference) to 12.5 mg/kg (ABSA 10, Allegan).

Figures 5-10 through 5-13 show the concentrations of total PCBs in muskrat whole body (Figure 5-10), muskrat liver (Figure 5-11), mink whole body (Figure 5-12), and mink liver (Figure 5-13). These concentrations are shown as both wet weight and lipid weight values. LOAELs, NOAELs, or other effects type data are unavailable for comparisons to whole body mink or muskrat PCB concentrations or to muskrat liver concentrations.

However, the level of PCB contamination of mink liver collected onsite can be compared to NOAELs and LOAELs based on mink liver PCB concentrations. For example, Kannan et al. (2000) derived (from other studies) a lipid-normalized mink liver total PCB NOAEL of 2.03 mg/kg, lipid weight, and a LOAEL of 44.4 mg/kg, lipid weight. Based on lipid-normalized values, total PCB concentrations in liver in the eight mink collected to support this ERA range from 2.25 to 57.51 mg/kg, lipid weight. The range for background locations (n=5) is 2.25 to 5.17 mg/kg lipid weight. For Plainwell the single mink liver collected contained 11.26 mg/kg lipid weight. The single value for Trowbridge equals 17.02 mg/kg lipid weight. Finally, the two mink livers collected at Allegan contained 11.38 to 57.51 mg/kg lipid weights.

Figure 5-13 shows these lipid-normalized mink liver PCB concentrations as well as the same values expressed as wet weight concentrations. This figure reveals that all mink livers (lipid wt.) collected onsite exceed the lipid-normalized mink liver NOAEL presented by Kannan et al. (2000). The LOAEL of 44.4 mg/kg total PCBs, lipid weight, was exceeded by one of the livers collected at Allegan. The mink livers collected at the background locations slightly exceed the NOAEL, while all others from Plainwell, Trowbridge, and Allegan exceed the NOAEL by about five-fold (Plainwell and one Allegan sample), eight-fold (Trowbridge), or 26-fold (second sample at Allegan).

The small sample sizes (n=1 to 5 at any location) precludes using these liver data to make definitive statements regarding risks to mink, but they appear to support the overall conclusions regarding mink exposure and risk from PCBs at this site. This conclusion is based in part on the finding that all mink livers collected from the site contained total PCBs at levels exceeding the liver-based NOAEL and approaching (or in one case exceeding) the liver-based LOAEL.

Finally, the large spread between the lipid-normalized liver NOAEL (2.03 mg/kg) and LOAEL (44.4 mg/kg) adds uncertainty to the actual threshold concentration at which adverse effects would begin to be observed in exposed mink. The values of the NOAEL and LOAEL calculated by Kannan et al. (2000) are a function of the treatment concentrations used in the original studies. Additional studies with treatment

concentrations closer to one another may reveal that the actual LOAEL is lower than the LOAEL of 44.4 mg/kg reported by Kannan et al. (2000).

5.4 Uncertainty Evaluation - Risk Characterization

By definition, uncertainties in risk characterization are influenced by uncertainties in exposure assessment and effects assessment. Uncertainties in exposure assessment are reduced by the adequate sampling and analysis of surface water, streambed sediment, floodplain sediment, surface soil, and biota. Descriptions of the magnitude and distribution of PCBs within the API/PC/KR site are considered to be representative of current conditions because of the environmental persistence of PCBs.

Effects data can also contribute to overall uncertainty in risk characterization. Science and scientific investigations cannot prove any hypothesis beyond doubt. The scientific method is instead based on stating hypotheses, testing these hypotheses, and either accepting or rejecting the hypotheses based on the weight-of-evidence provided by test data. Cause and effect relationships can be inferred, and evidence can support hypotheses, but cause and effect relationships can rarely be proven.

In this ERA, the primary null hypothesis is that the Kalamazoo River and associated aquatic and riparian habitats have not been and are not being adversely affected by PCBs and related physical stressors. These stressors are assumed to have originated primarily from past industrial activities along the Kalamazoo River. This null hypothesis is tested by using multiple lines of evidence, which provide support for either rejection or acceptance of the proposed hypotheses. No data are conclusive. Site-specific biological and chemical data are subject to concerns of representativeness and availability and the sensitivity of sampled species used to derive such data. Toxicity data that are not site specific may not be totally applicable to the site being investigated. There are concerns about laboratory-to-field extrapolation of effects data. Taxa-to-taxa extrapolations are a concern as well. All effects data are, therefore, subject to some degree of uncertainty. Confidence in the ability of selected effects data to assess potential for ecological risks varies for each data value selected.

This ERA presents effects data in the risk characterization phase that be used to assess potential for adverse ecological impacts. While each and every effects data value used in this and every other ERA is associated with some degree of uncertainty, it is the general trend described by the comparisons between exposure concentrations and effects concentrations, and the overall confidence in such comparisons, that are most important.

Another potential source of uncertainty is the lack of extensive biological or ecological surveys conducted over time to support this ecological risk assessment. The types of surveys needed to aid in the determination of cause and effect relationships are highly dependent on data quality and data quantity. For example, historical data on fish and furbearer populations could be used to evaluate population-level effects over time

that might be associated with PCB contamination or other sources of ecological stress. Other useful long-term data such as gut contents of key predators (e.g., mink) could help refine the estimated average dietary composition critical to food chain modeling. In contrast, the gut contents of a few mink taken during one season cannot be used to reliably estimate the average annual diet of mink. For the most part, these types of long-term data are not currently available. Still, observations based on recent fieldwork can be used to provide important qualitative information and in some cases evidence of adverse impacts.

For example, trapping success of mink appears to be associated with PCB contamination in sediment and fish. While equal trapping effort was expended at all locations, trapping success was substantially greater within the reference areas upstream of the API/PC/KR site. Of the 10 mink collected for tissue analyses, 5 (50 percent of total) were taken from the upstream reference area (ABSA 1). Of the remaining 5 mink, 1 was taken from ABSA 6 upstream of Otsego City Dam, 2 from TBSA 5 upstream of Trowbridge Dam, and 2 from ABSA 10 downstream of Allegan Dam. Although data are insufficient for making conclusions relating cause and effect of possible population level effects on mink, it is noted that fish tissue PCB concentrations are correlated with numbers of mink collected. Substantially fewer mink were collected within and downstream of the API/PC/KR where fish tissues contained the highest levels of PCBs. Similarly, fish tissue PCB concentrations were substantially lower in areas where mink trapping was highly successful.

The risk characterization method itself can also contribute to uncertainty. This type of uncertainty is minimized by not relying on a single exposure point concentration (e.g., mean or maximum value) or on a single effects concentration (e.g., AWQC or LC_{50}). The multiple lines of evidence used to conduct this ERA provides a more meaningful approach that minimizes the effects associated with the inherent uncertainty in any particular exposure or effects data value. This can be best demonstrated with the selection of TRVs for mink and non-raptor birds. For these receptors, multiple studies were evaluated and the final TRVs were determined using an approach (EC_x or ED_x) that incorporates data from several studies determined to be most appropriate. This approach is in contrast to the more common method where multiple studies are evaluated and one value is selected from a single study to serve as the TRV of choice.

Uncertainties with risk characterization differ for each receptor or receptor group. For example, risks to great horned owl and red fox are likely to be overestimated because these risks are based in part on the consumption of songbirds, represented by robin. Granivorous bird species and others that do not consume earthworms are likely to have much less exposure to PCBs than robins. Using robins as a representative avian prey item for owls and foxes is therefore likely to result in an overestimation of risks.

This ERA presents overwhelming evidence that, despite uncertainties identified in the ERA, two and possibly three of the four proposed null hypotheses introduced in Section 3.4 and presented below can be rejected with little reservation.

1. *The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the structure or function of the fish populations in the Kalamazoo River and Portage Creek System.*

This hypothesis is *accepted* because there is no direct evidence that fish communities are being affected by PCB contamination. The impaired fish community of Lake Allegan is comprised primarily of stunted and often malformed carp. The cause of these findings cannot be determined from the available data. It is noted, however, that PCBs cause a wasting syndrome in several mammalian species. There is insufficient evidence to determine if similar effects are occurring in fish.

2. *The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the survival, growth, and reproduction of plant and animal aquatic receptors utilizing the Kalamazoo River and Portage Creek system.*

This hypothesis is *conditionally rejected.* This is based on the finding that at some locations the maximum detected surface water PCB concentration exceeds the lowest chronic value for freshwater fish, invertebrates, or aquatic plants.

3. *The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the survival, growth, and reproduction of mammalian receptors utilizing the Kalamazoo River and Portage Creek system.*

This hypothesis is *rejected* because there is sufficient evidence that adverse effects are likely to be experienced by mammalian predators, especially those that consume fish.

4. *The levels of PCBs in water, sediment, and biota are not sufficient to adversely affect the survival, growth, and reproduction of avian receptors utilizing the Kalamazoo River and Portage Creek system.*

This hypothesis is *rejected* because there is sufficient evidence that adverse effects are likely to be experienced by avian predators, especially those that consume fish.

In summary, the ecosystem associated with the API/PC/KR portion of the Kalamazoo River has been and is currently being adversely affected by PCBs originating from past industrial activities. The environmental persistence of PCBs suggests that adverse impacts to ecological resources at this site will continue into the foreseeable future without significant remedial/removal actions.

5.5 Remediation Issues

The Kalamazoo River and nearby riparian areas are currently being adversely affected by nonpoint sources of chemical contamination. It is expected that remediation of the most serious and most ubiquitous contaminants (i.e., PCBs) would result in remediation of other less serious contaminants that are not as uniformly distributed

or are present at lower concentrations. For this reason, this preliminary discussion of remediation issues is focused on remediation of PCBs in aquatic and terrestrial media.

Instream and floodplain sediments, surface water, surface soil, and biota within the API/PC/KR site are contaminated with PCBs. Contaminated groundwater may discharge to the Kalamazoo River and Portage Creek as well, but groundwater inputs have not been quantitatively evaluated. It is expected that the most critical current nonpoint source of PCBs to the Kalamazoo River and Portage Creek are erosion and runoff of contaminated streambank sediments/soils and release of PCBs from streambed sediments to surface water. Surface water within the API/PC/KR area is probably also affected by upstream, offsite inputs of both contaminated surface water and contaminated sediments, but such inputs appear to be small compared to onsite sources (e.g., areas of former impoundments). Again, contaminated groundwater may contribute to elevations in surface water PCB concentrations during certain times of the year and in certain locations, depending on groundwater/surface water relationships. Fine-grained instream sediments probably move downstream at a rate dependent on flow. During and immediately following storm events, fine grained sediments are likely to move downstream rapidly, eventually entering depositional areas within the API/PC/KR site or Lake Michigan. Lake Michigan probably acts as a sediment trap for sediments that reach far downstream. Several areas of the API/PC/KR site are likely to trap substantial amounts of fine-grained sediment, and removal of fine-grained sediment from these depositional areas is likely to decrease biological impairment by removing a primary source of toxicity and instream siltation.

Stabilizing streambank materials is also expected to decrease the potential chemical and physical effects of erosion. Surface water concentrations of PCBs are unlikely to return to safe levels without consideration of both streambank and streambed sediments. Siltation must be controlled if a diverse and healthy aquatic community is to be established in affected areas of the API/PC/KR site. Removal and/or capping of streambank sediments contaminated with PCBs is necessary to prevent erosion and runoff which ultimately contaminates and physically degrades the river.

Finally, the use of a single sitewide cleanup value for sediments is supported by the dynamic nature of the sediment environment. A single protective value derived for the entire site assumes that conditions can and do change both seasonally and from year to year, while multiple values assumes stable conditions at each location where a separate cleanup value may be derived. Since sediments are unstable and are continuously moving into the aquatic environment and downstream, the use of multiple ABSA-specific or other location-specific cleanup values is unwarranted.

Table 5-5 presents a compilation of total PCB limits, criteria, and site-specific PRGs proposed to be considered in the selection of a single media-specific cleanup value for the API/PC/KR site. For each media type, the selection of indicator chemicals is appropriate. That is, remediation of the most critical chemical component within each

media type (e.g., PCBs) is likely to result in remediation of the less critical chemical stressors as well. Total PCBs can, therefore, serve as indicator chemicals for remediation purposes.

For surface water, control of streambank erosion and runoff and elimination or decrease in streambed sediment volumes and/or PCB concentrations is most critical. For streambed and streambank sediment, substantial decreases in total PCBs are warranted because these media will continue to provide a toxicant source to the Kalamazoo River and resident aquatic and terrestrial biota. For surface soil, concentrations of PCBs need to be substantially reduced where such soils have potential to erode into aquatic environments.

The selection of the most appropriate methods for achieving remediation goals is not a risk assessment issue but is a risk management issue to be addressed in the feasibility study (FS) for the API/PC/KR site. The application of specific PRGs is also considered a risk management decision. This risk assessment derives and recommends a range of receptor- and media-specific PRGs. It is most appropriate for risk managers rather than risk assessors to decide how to best apply these PRG ranges to meet remedial goals and objectives.

5.5.1 Summary of Recommended Cleanup Values

Table 5-5 summarizes the proposed cleanup levels for various media for the Kalamazoo River Superfund Site. This summary is based on the Low Effect PCB concentrations calculated for site media, and as such are analogous to "not to exceed" concentrations.

- *Surface water* total PCB concentrations should not exceed 0.00197 µg/L to protect mink, the most sensitive of all animals tested to date. This is based on the low effect dietary concentration (EC_{25}) determined from long-term studies in which mink were fed PCB-contaminated fish and on site-specific BAFs for fish. The corresponding No Effect PCB concentration is 0.0016 ug/L.

- *Streambed sediment* total PCB concentrations should not exceed 0.6 mg/kg to protect mink, the most sensitive of all animals tested to date. This is also based on the low effect dietary concentration (EC_{25}) determined from long-term studies in which mink were fed PCB-contaminated fish, site-specific BAFs for fish, and sediment/water relationships. The corresponding no effect dietary concentration (EC_{10}) to protect mink is 0.5 mg/kg.

- *Surface soil* and in some cases *floodplain sediment* PCB concentrations should not exceed 8.1 mg/kg (low effect PRG based on ED_{25}) to protect omnivorous birds such as American robin. The corresponding no effect PRG (based on ED_{10}) for robin is 6.5 mg/kg.

Figure 5-1
Total PCB Concentrations - Thresholds/Criteria
API/PC/KR Surface Water

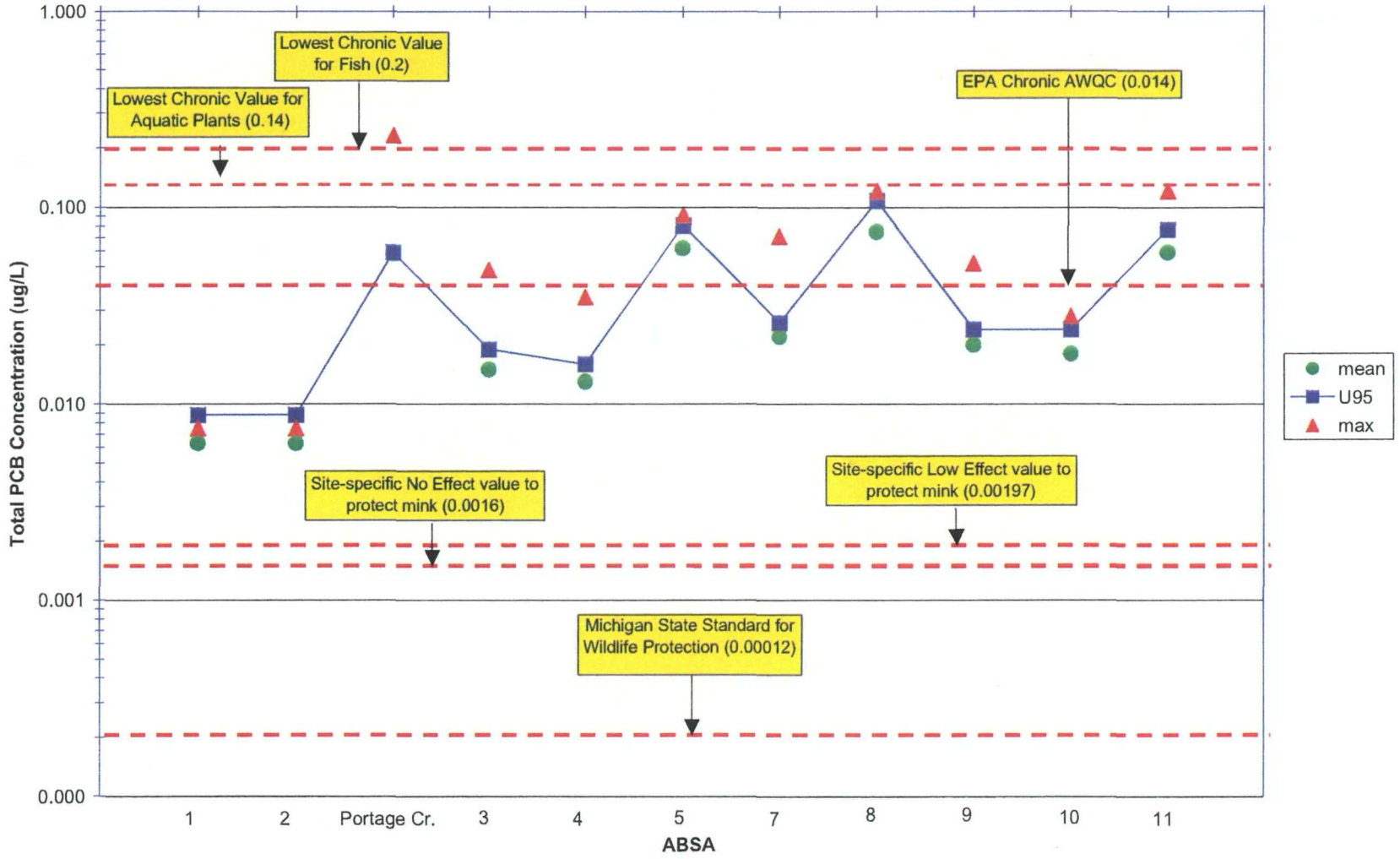


FIG5-1_Rev42303.XLSfig5-1sw

Figure 5-2
Total PCB Concentrations - Thresholds - PRGs
API/PC/KR Instream Sediment

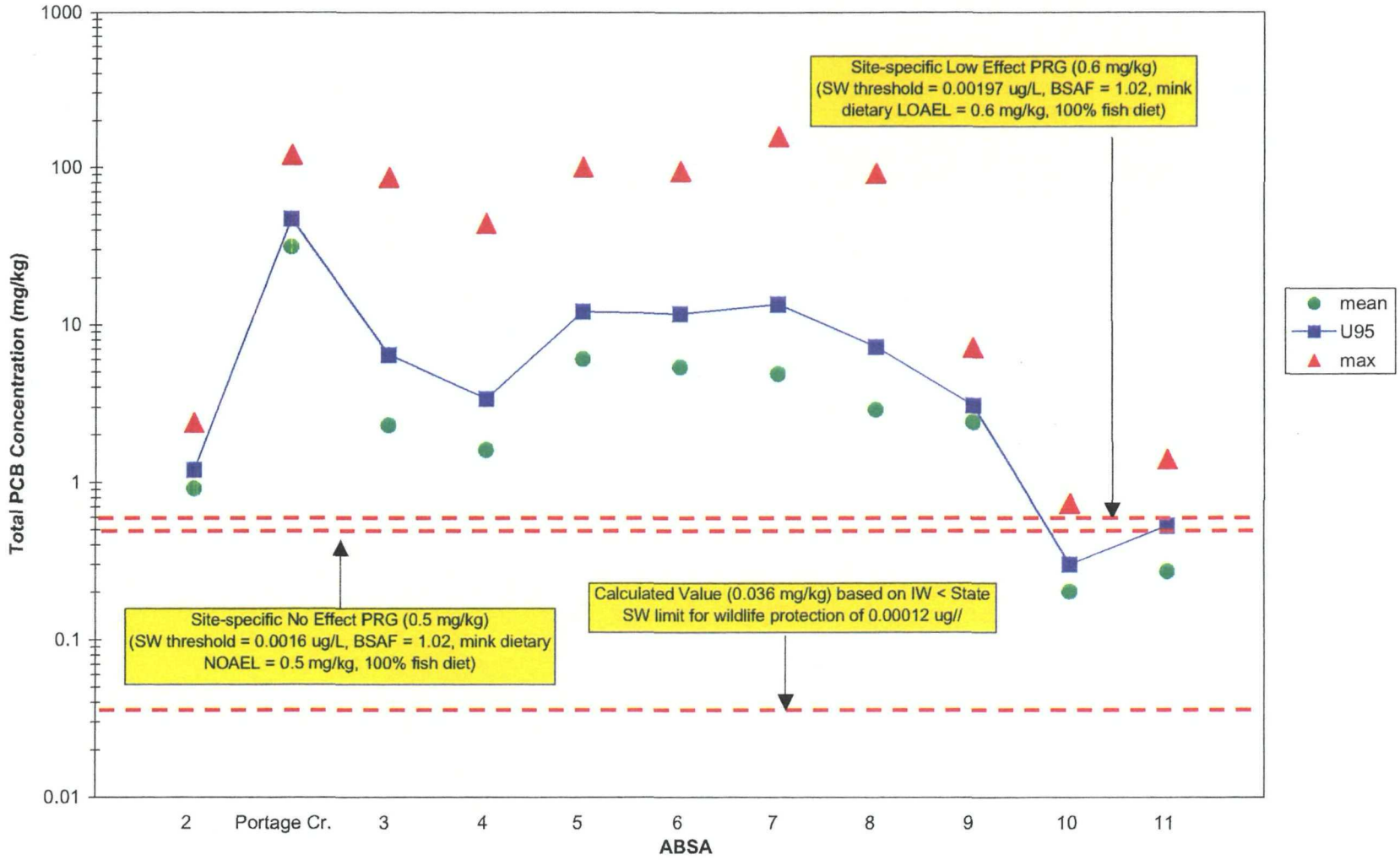


FIG5-2-4_Rev042303.xlsfig5-2sed

Figure 5-3
Total PCB Concentrations - PRGs
API/PC/KR FP SED/SS (Terrestrial/Floodplain Exposures)

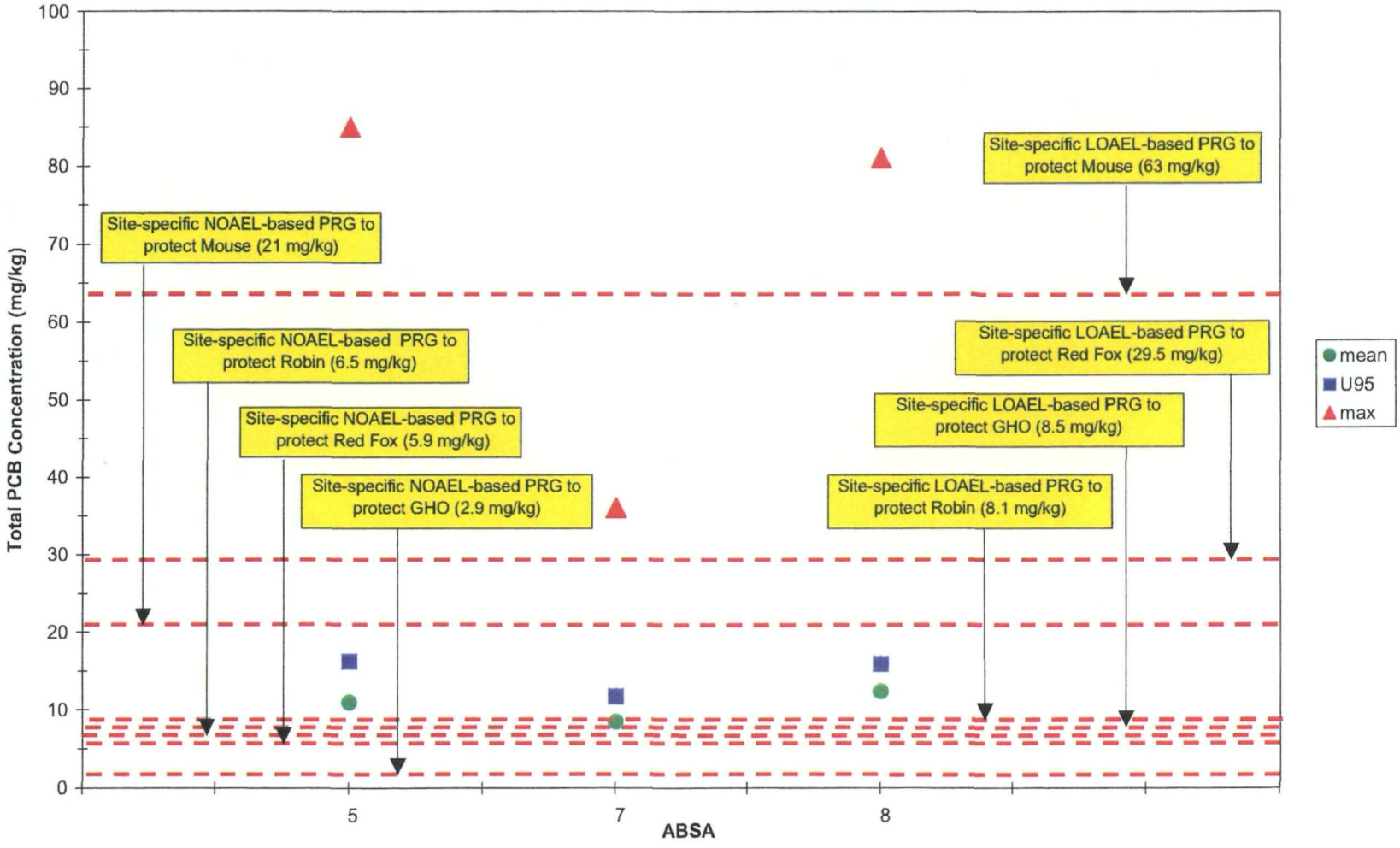


FIG5-2-4_Rev042303.xlsfig5-3fpsed

Figure 5-4
Total PCB Concentrations - PRGs
API/PC/KR FP TBSA Surface Soil (Terrestrial Exposures)

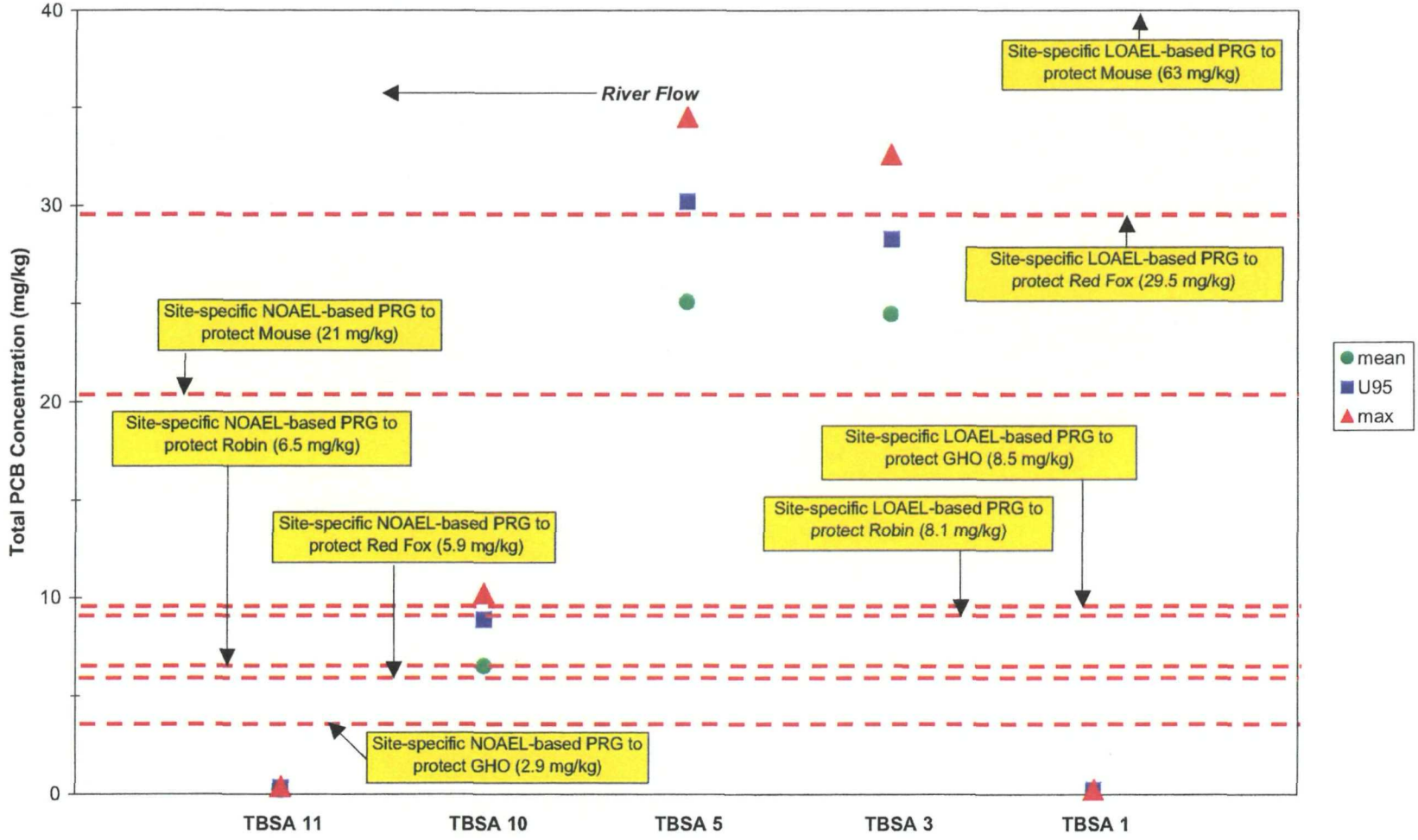


FIG5-2-4_Rev042303.xlsfig5-4ss

Figure 5-5
Maximum Total PCB
Concentrations in Terrestrial Biota

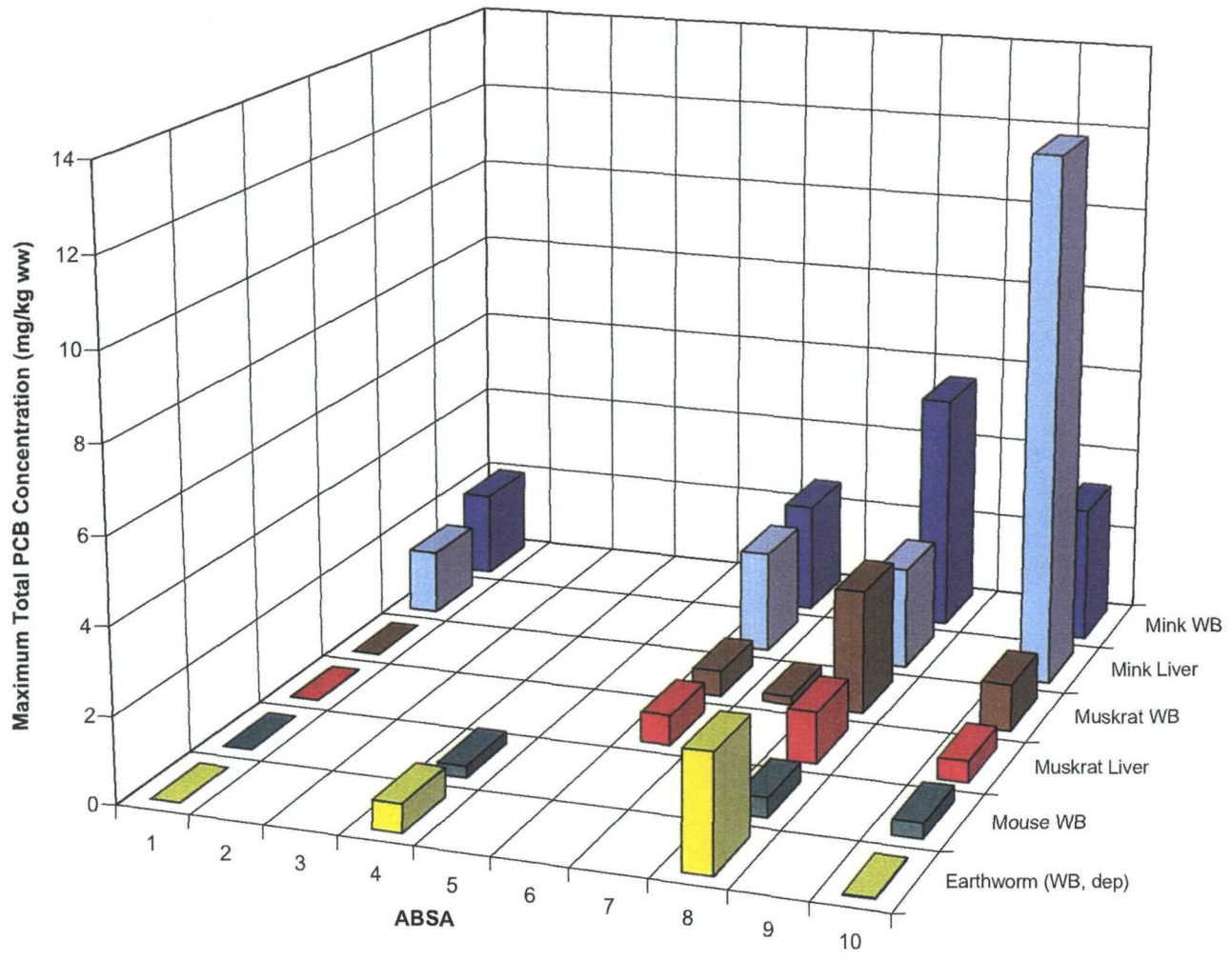


FIG5-5-6_Rev12-04-02.XLSfig5-5terr

Figure 5-6
U95 Whole Body Total PCB
Concentrations in Fish

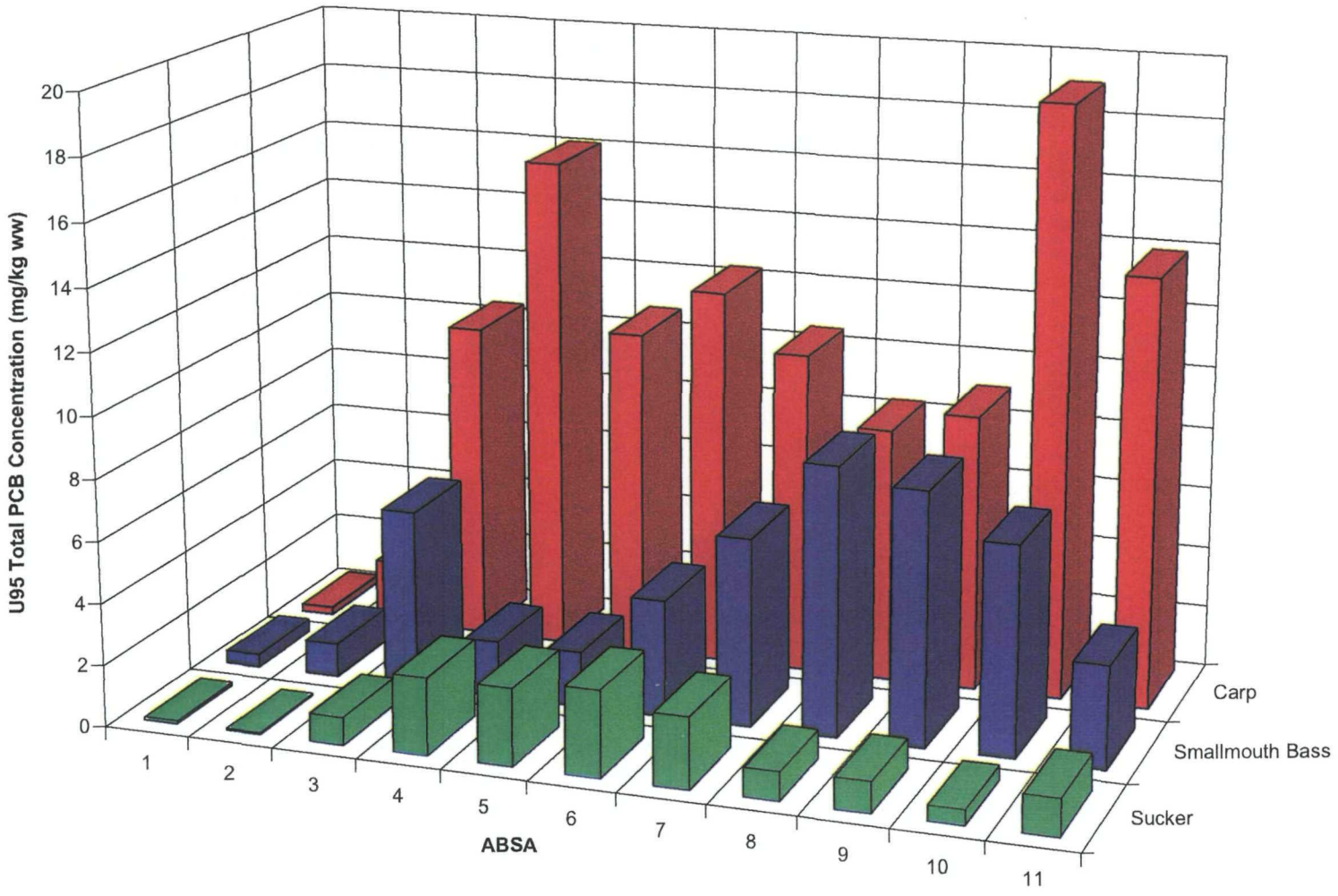


FIG5-5-6_Rev12-04-02.XLSfig5-6fish

Figure 5-7
Smallmouth Bass Whole Body
Total PCB Concentrations

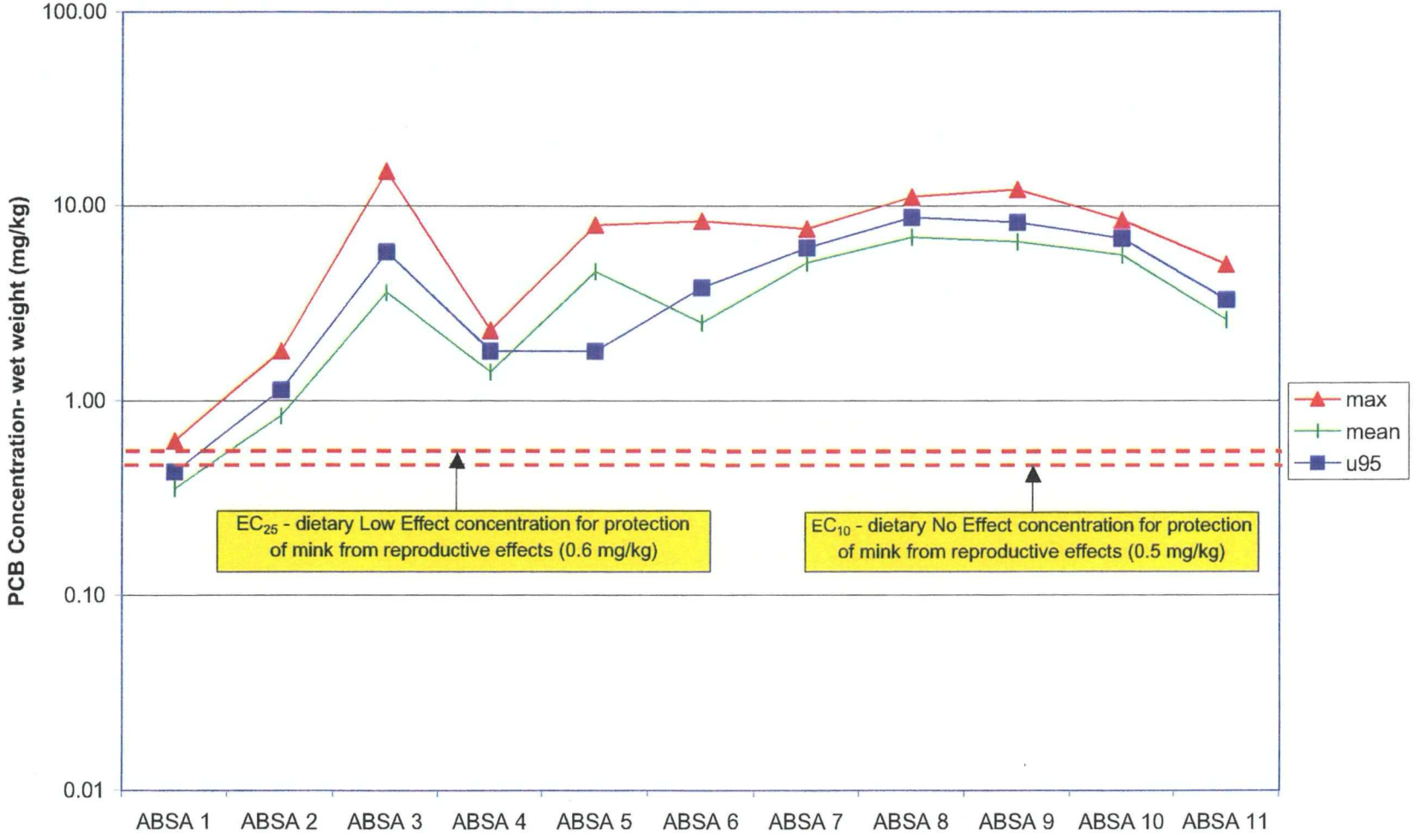


FIG5-7-9_Rev042303.xlsfig5-7

Figure 5-8
Common Carp Whole Body
Total PCB Concentrations

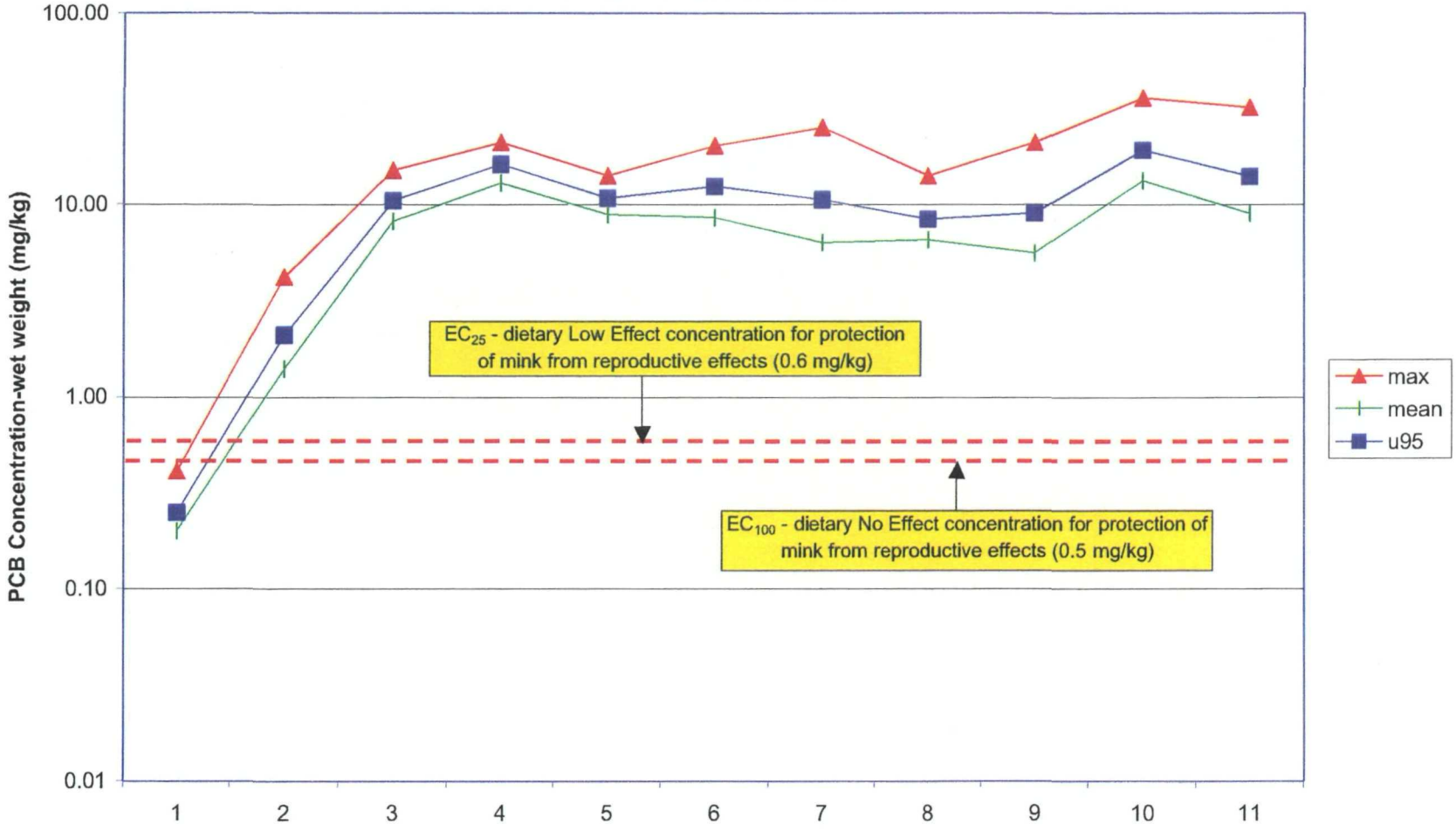


FIG5-7-9_Rev042303.xlsFig5-8

Figure 5-9
Sucker Whole Body
Total PCB Concentrations

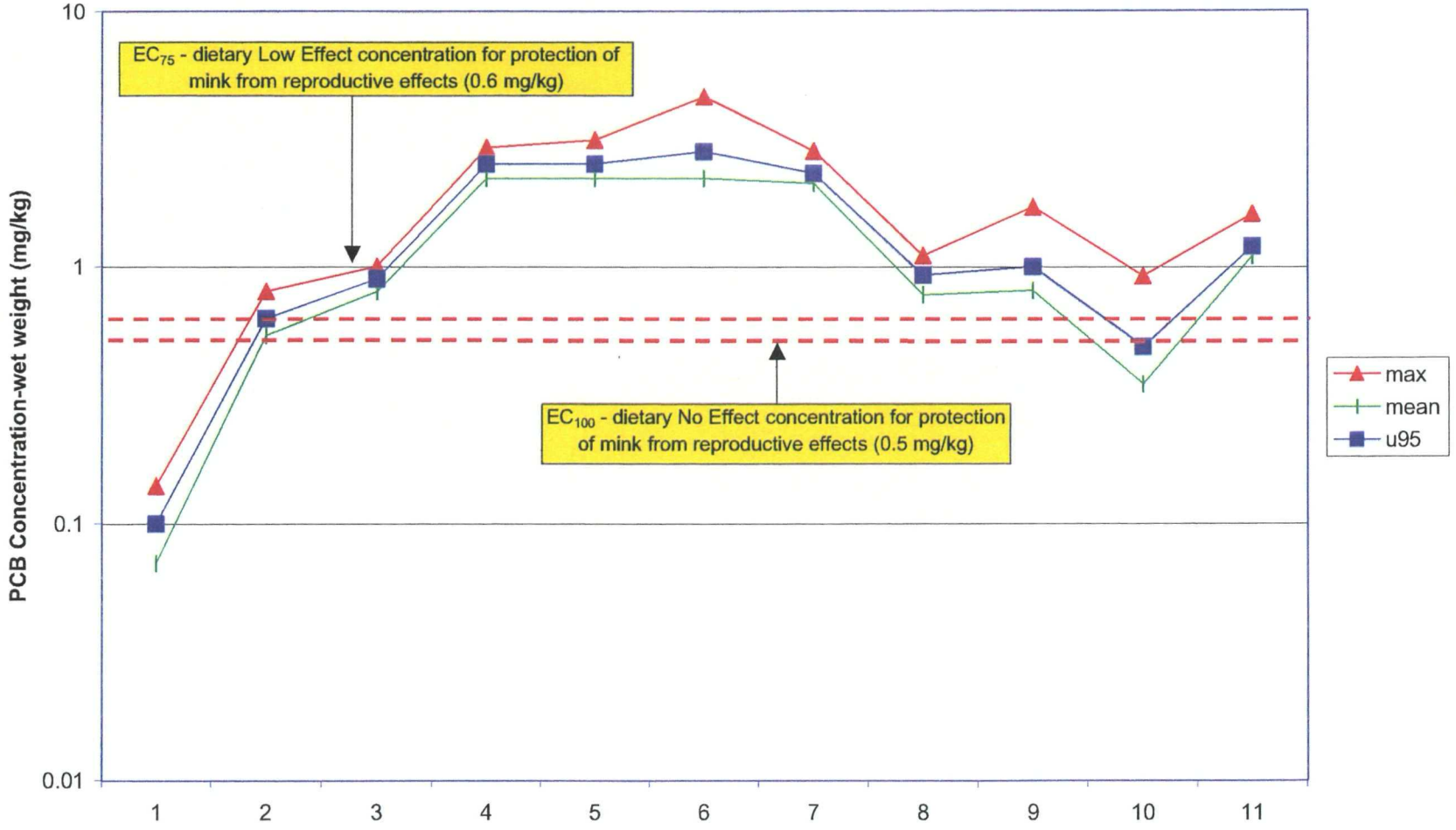


FIG5-7-9_Rev042303.xlsFig5-9

Figure 5-10
Total PCB Concentrations
Muskrat Carcass

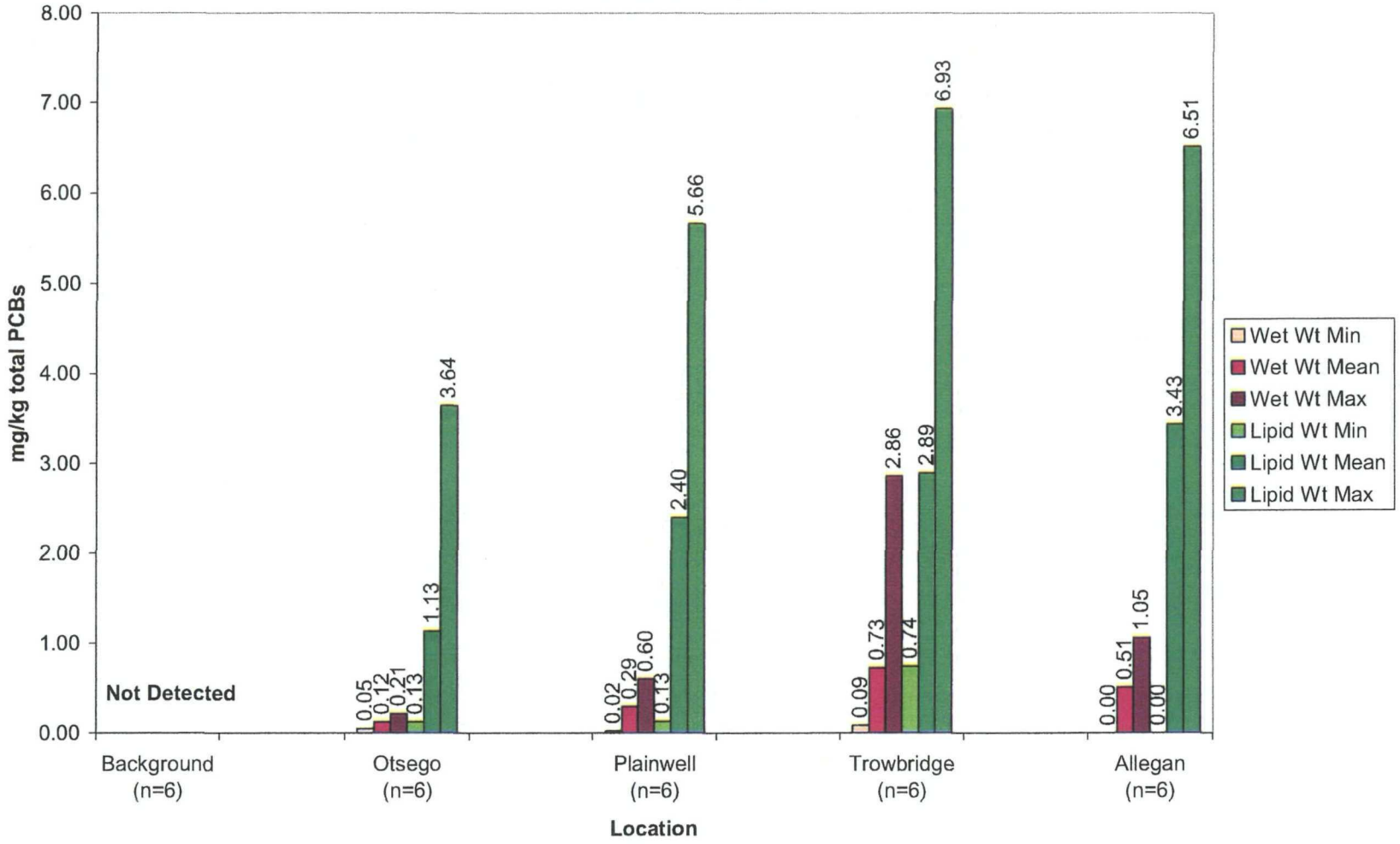


Figure 5-11
Total PCB Concentrations
Muskrat Liver

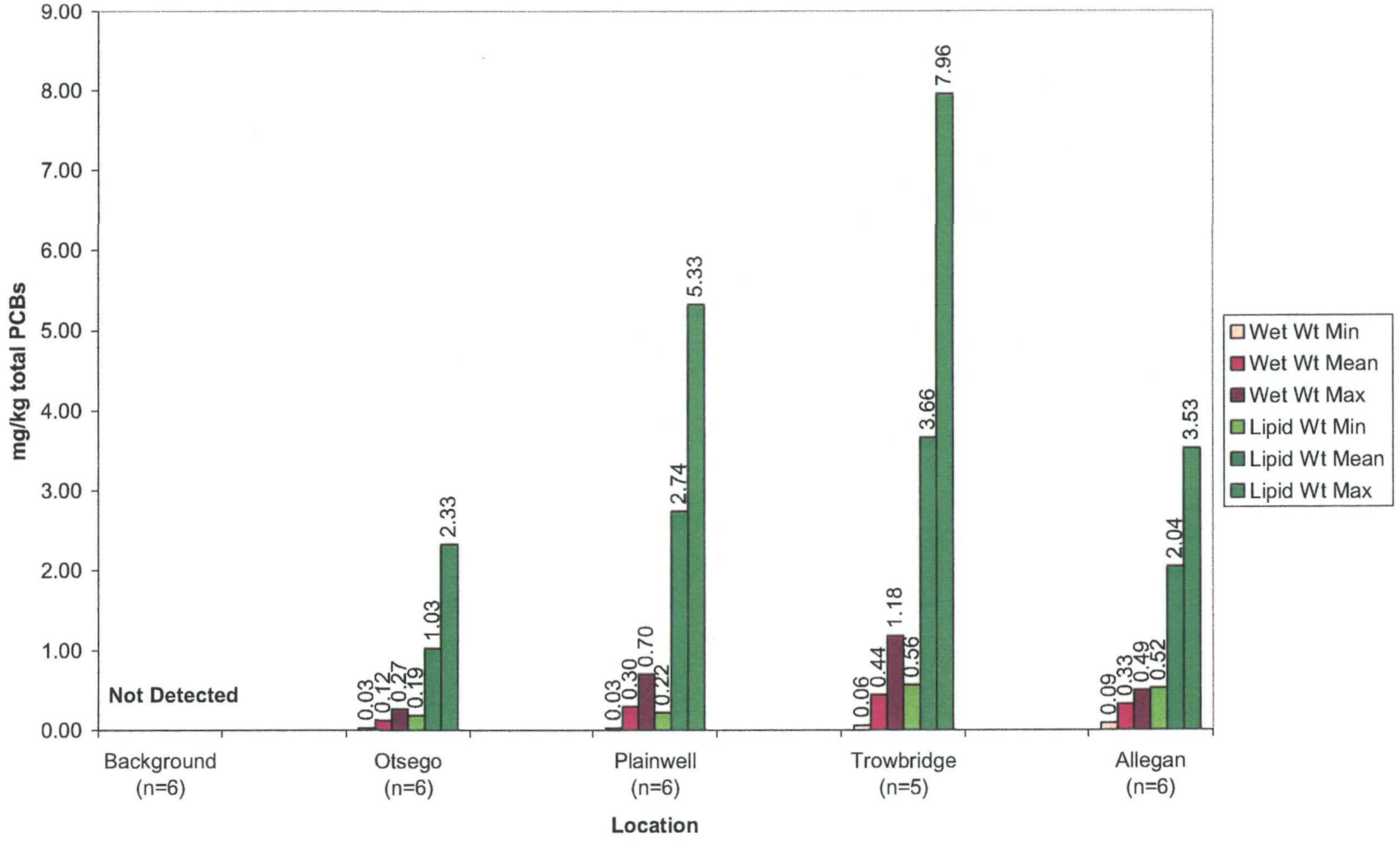


Figure 5-12
Total PCB Concentrations
Mink Carcass

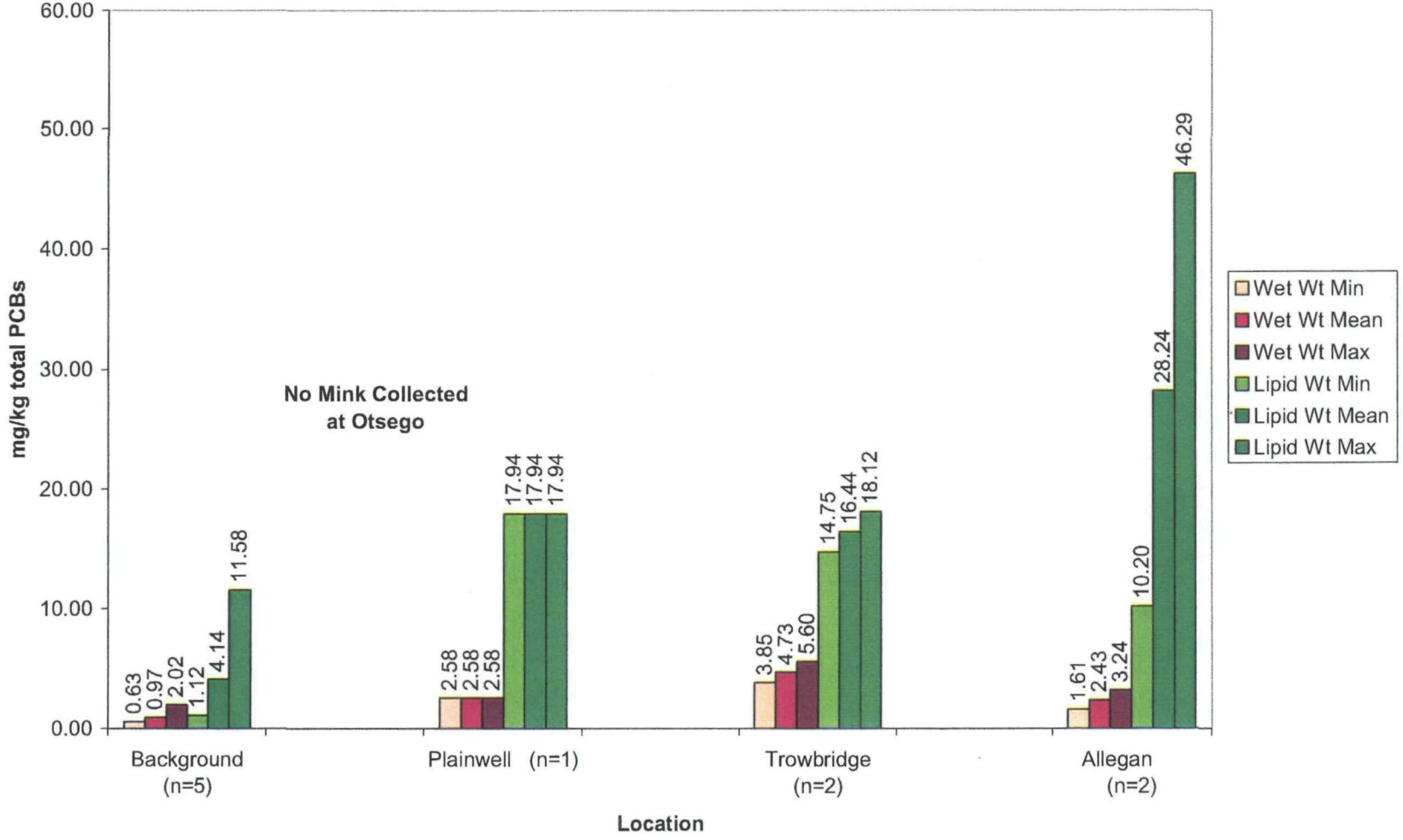


Figure 5-13
Total PCB Concentrations
Mink Liver

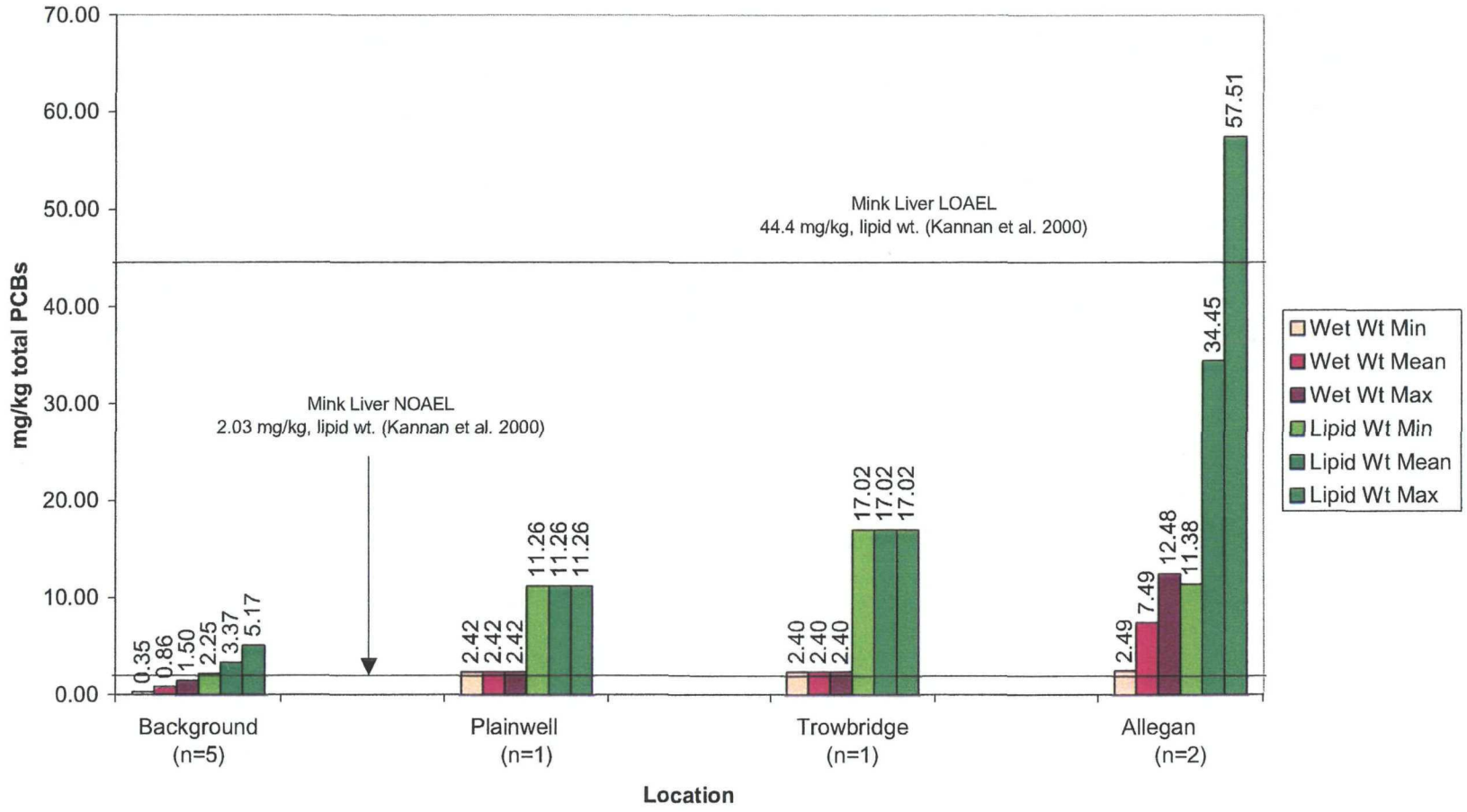


Table 5-1
Summary of the PCB Food Web Model, Terrestrial/Wetland Species
API/PC/KR

Receptor	Estimated Average Potential Daily Dose (mg/kg/d)	Low Effect Concentration LOAEL or ED_{25} (mg/kg/d) (target species)	No Effect Concentration NOAEL or ED_{10} (mg/kg/d) (target species)	Reference
American Robin	0.9044[1]	0.5 (chicken)	0.4 (chicken)	See Appendix C-1, C-2
Mink	1.6988[2]	0.11 (mink)	0.091 (mink)	See Appendix C-1, C-2
White-footed/ Deer Mouse	0.3109	1.35 (mouse)	0.45 (estimated from mouse LOAEL/3)	See Appendix C-1, C-2
Bald Eagle	2.1606	0.5 (chicken)	0.4 (chicken)	See Appendix C-1, C-2
Muskrat	0.4167	5 (rat)	1.7 (estimated from rat LOAEL/3)	See Appendix C-1, C-2
Red Fox	2.4764	5 (dog)	1 (dog)	See Appendix C-1, C-2
Great Horned Owl	2.0551	1.2 (estimated from NOAEL*3)	0.41 (screech owl)	See Appendix C-1, C-2

[1] Terrestrial plant component of diet based on soil-to-fruit BAF (tomato, CDM 2000)
[2] Diet from Alexander 1977 (river, year-round) in EPA 1993
unadjusted values = 85% fish
adjusted values:
birds/mammals = 6%, adjusted for birds = 5%, mammals = 10% (5% mouse, 5% muskrat)
vegetation = 1%, adjusted to 0%
unidentified = 1%, adjusted to 0%
crustaceans = 4%, adjusted to 0%
amphibians = 3%, adjusted to 0%
adjustments made to include only prey items for which site-specific PCB data are available

Table 5-2
PRGs for PCBs in FP Sediment/Surface Soil for Representative Terrestrial Food Web Species
API/PC/KR

Receptor	Low Effect DOSE (mg PCB/kg-d)	No Effect DOSE (mg PCB/kg-d)	Daily Dose (mg/kg-d)	FPSED/SS Total PCB Concentration[1] (mg/kg)	PRG RANGE (No Effect to Low Effect[2]) (mg PCB/kg FPSED)
Robin	0.5	0.4	0.9044	14.6	6.5 –8.1
Great Horned Owl	1.2	0.41	2.0551		2.9 – 8.5
Red Fox	5	1	2.4764		5.9 – 29.5
White-footed/ Deer Mouse	1.35	0.45	0.3109		21.1 – 63.4

[1] FP SED/SS total PCB concentration based on mean of U95 PCB concentration for ABSAs 5, 7, and 8 (Plainwell, Otsego, and Trowbridge areas)
[2] NOAEL to LOAEL or ED_{10} to ED_{25}, see Appendix C-2-A for detailed calculations and text for discussion

Table 5-3
Summary of Total PCB Risks to Ecological Receptors
API/PC/KR

Ecological Receptor Group or Target Species	Exposure Concentration (dose or exposure media)	No Effect Dose	No Effect Dose-Based HQ	Low Effect Dose	Low Effect Dose-Based HQ
Mink	1.6988 mg/kg/d	0.091 mg/kg/d	**19**	0.11 mg/kg/d	**15**
Bald Eagle	2.1606 mg/kg/d	0.4 mg/kg/d	**5.4**	0.5 mg/kg/d	**4.3**
Great Horned Owl	2.0551 mg/kg/d	0.41 mg/kg/d	**5.0**	1.2 mg/kg/d	**1.7**
American Robin	0.9044 mg/kg/d	0.4 mg/kg/d	**2.3**	0.5 mg/kg/d	**1.8**
Red Fox	2.4764 mg/kg/d	1.0 mg/kg/d	**2.5**	5.0 mg/kg/d	0.5
White-footed/Deer Mouse	0.3109 mg/kg/d	0.45 mg/kg/d	0.7	1.35 mg/kg/d	0.2
Muskrat	0.4167 mg/kg/d	1.7 mg/kg/d	0.3	5.0 mg/kg/d	0.08
	(mean U95 SW conc)	**Chronic AWQC[1]**	**AWQC-based Hazard Quotient**		
Generic Piscivorous Wildlife	0.043 µg/L surface water	0.014 µg/L surface water	3.1		
		NOAEC[2]	**NOAEC-based HQ**	**LOAEC[3]**	**LOAEC-based HQ**
Carp	0.043 µg/L surface water	0.02 µg/L surface water	**2.2**	0.2 µg/L	0.22
Sucker	0.043 µg/L surface water	0.02 µg/L surface water	**2.2**	0.2 µg/L	0.22
Smallmouth Bass	0.043 µg/L surface water	0.04 µg/L surface water	**1.1**	0.4 µg/L	0.11
Aquatic Invertebrates	0.043 µg/L surface water	0.08 µg/L surface water	0.54	0.8 µg/L	0.05
Salmonid Fish	0.043 µg/L surface water	0.1 µg/L surface water	0.43	1.0 µg/L	0.04

[1] Chronic AWQC (Final Residue Value) for PCBs is based on protection of piscivorous wildlife. Data specifically from studies of mink and ingestion of salmonid fish. In most cases, chronic AWQC are intended to protect 95 percent of the aquatic species. EPA modifies this approach for certain chemicals that readily bioaccumulate and move easily through food chains to upper trophic level predators. In these cases, AWQC are further lowered to protect sensitive wildlife that may consume contaminated prey. For PCBs, the chronic AWQC (0.014 ug/L) is specifically based on (1) the lowest maximum permissible tissue concentration for dietary items consumed by mink and (2) the geometric mean whole body BCF values for salmonid species. The derivation of the chronic AWQC follows:

$$\text{Freshwater chronic AWQC} = \frac{\text{maximum permissible tissue concentration}}{\text{geometric mean BCF for salmonid fish}}$$

$$0.014\text{ug/L} = \frac{0.64\text{ mg/kg}}{45{,}000}$$

All values used in the derivation of the national chronic AWQC are presented in EPA 1980. Because the national chronic AWQC for PCBs is based on wildlife protection, it is more accurately referred to as the Freshwater Final Residue Value.

[2] Estimated from LOAEC/10

[3] From Appendix C-1, except for salmonid value (brook trout chronic value, Mauck, et al. 1978 in EPA 1980)

Table 5-4.a
Hazard Quotients for Birds Eggs – Egg TRVs from Table 4-9

Bird Species	Mean Egg PCB Conc (n)	Egg NOAEC[1]	Egg LOAEC[1]	NOAEC HQ	LOAEC HQ
Bald eagle	77.6 (4)	1.5 (bald eagle)	7.7 (bald eagle)	**52**	**10**
Great horned owl	43.1 (3)	1.3 (bald eagle)	6.4 (bald eagle)	**33**	**6.7**
Red tailed hawk	11.3 (3)	1.3 (bald eagle)	6.4 (bald eagle)	**8.7**	**1.8**
Great blue heron	10.5 (6)	5.8 (Foster's tern)	20.6 (Foster's tern)	**1.8**	0.5
Wood thrush	1.93 (1)	1.1[2] (tree swallow)	5.7 (tree swallow)	**1.8**	0.3
Yellow warbler	1.31 (1)	1.1[2] (tree swallow)	5.7 (tree swallow)	**1.2**	0.2
Red winged blackbird	1.2 (5)	1.1[2] (tree swallow)	5.7 (tree swallow)	**1.1**	0.2
American robin	2.1 (2)	2.8 (chicken)	6.2 (chicken)	0.8	0.3
Wood duck	0.43 (6)	2.8 (chicken)[3]	6.2 (chicken)[3]	0.2	0.07

All data in mg/kg total PCBs
[1] Mean NOAEC or LOAEC for most closely related species or species with similar diet (Table 4-9)
[2] Estimated from LOAEC/5, based on similar data for other species
3 NOAEC and LOAEC based on mean value for egg hatchability

Table 5-4.b
Hazard Quotients for Birds Eggs – Egg TRV from Appendix D (chicken studies)

Bird Species	Mean Egg PCB Conc (n)	Egg NOAEC[1]	Egg LOAEC[1]	NOAEC HQ	LOAEC HQ
Bald eagle	77.6 (4)	1.0	1.5	**78**	**52**
Great horned owl	43.1 (3)	1.0	1.5	**43**	**29**
Red tailed hawk	11.3 (3)	1.0	1.5	**11**	**7.3**
Great blue heron	10.5 (6)	1.0	1.5	**11**	**7.0**
American robin	2.1 (2)	1.0	1.5	**2.1**	**1.4**
Wood thrush	1.93 (1)	1.0	1.5	**1.9**	**1.3**
Yellow warbler	1.31 (1)	1.0	1.5	**1.3**	0.9
Red winged blackbird	1.2 (5)	1.0	1.5	**1.2**	0.8
Wood duck	0.43 (6)	1.0	1.5	0.4	0.3

All data in mg/kg total PCBs
[1] NOAEC or LOAEC from Appendix D

Table 5-5
Media-Specific and Species-Specific Levels of Protection
API/PC/KR

Media	Total PCB Concentration	Receptor	Description	Equation
Surface Water	0.00012 µg/L	Avian and Mammalian Wildlife	MDEQ Surface Water Quality Division value for protection of avian and mammalian wildlife.	NA
	0.0016 µg/L	Mink	No Effect value for fish tissue threshold (0.5 mg/kg) to protect mink. Mean fish BAF = 305,000.	0.5 mg/kg / 305,000 * 1,000
	0.00197 µg/L	Mink	Low Effect value for fish tissue threshold (0.6 mg/kg) to protect mink (mean fish BAF = 305,000) .	0.6 mg/kg / 305,000 * 1,000
Instream Sediment **Floodplain Sediment/ Soil**[1]	0.036 mg/kg	Avian and Mammalian Wildlife	Calculated from MDEQ Surface Water Quality Division SW value for protection of avian and mammalian wildlife (0.00012 µg/L) and mean site-specific K_d (302,000).	0.00012 µg/L * 302,000 / 1,000
	0.1 mg/kg	Avian and Mammalian Wildlife	NOAEC-base value based on MDEQ-SWQD default variables (from GLI) for water value protective of mink (0.000132 ug/L), NOAEC for mink (0.5 mg/kg, BAF for trophic level 3 fish (1,139,000), fish lipid (6.46%), and site-specific values for sediment Foc (0.082) and carp BSAF (1.9).	[(0.000132 ug/L)(1,139,000 L/kg) / 6.46%] (8.2%) / 1.9
	0.5 - 0.6 mg/kg	Mink	No Effect (EC_{10}) and Low Effect (EC_{25}) values to allow pore water PCB concentration to remain below SW thresholds of 0.0016 and 0.00197 µg/L, respectively. Mean site-specific SED/SW partition factor (K_d) = 302,000. No and Low Effect fish tissue thresholds = 0.5 and 0.6 mg/kg, mean site-specific Biota/SED partition factor = 1.02.	No Effect = 0.0016 µg/L * 302,000 / 1,000 or 0.5 mg/kg * 1.02 Low Effect = 0.00197 µg/L * 302,000 / 1,000 or 0.6 mg/kg * 1.02
	1.4 - 1.7 mg/kg	Bald Eagle	No Effect (ED_{10}) and Low Effect (ED_{25}) values resulting from food chain modeling, assuming fish-based diet (77%), dietary No Effect Dose = 0.4 mg/kg-d, dietary Low Effect Dose = 0.5 mg/kg-d, average daily dose = 2.1606 mg/kg-d, and U95 PCB Conc SED = 7.3 mg/kg.	No Effect = 0.4 mg/kg-d / 2.1606 mg/kg-d * 7.3 mg/kg Low Effect = 0.5 mg/kg-d / 2.1606 mg/kg-d * 7.3 mg/kg

Surface Soil **Floodplain Sediment/ Soil**[2]	6.5 – 8.1 mg/kg	Robin	No Effect (ED_{10}) and Low Effect (ED_{25}) values to protect omnivorous songbirds, represented by American robin. Dietary No Effect Dose = 0.4 mg/kg-d, dietary Low Effect Dose = 0.5 mg/kg-d, average daily dose = 0.9044 mg/kg-d, mean site-wide U95 PCB Conc FP SED = 14.6 mg/kg.	No Effect = 0.4 mg/kg-d / 0.9044 mg/kg-d * 14.6 mg/kg Low Effect = 0.5 mg/kg-d / 0.9044 mg/kg-d * 14.6 mg/kg
	2.9 - 8.5 mg/kg	Great Horned Owl (GHO)	NOAEL- and LOAEL-based value to protect non-piscivorous raptors, represented by GHO. Dietary NOAEL = 0.41 mg/kg-d, LOAEL = 1.2 mg/kg-d, average daily dose = 2.0551 mg/kg-d, mean site-wide U95 PCB Conc FP SED = 14.6 mg/kg	NOAEL = 0.41 mg/kg-d / 2.0551 mg/kg-d * 14.6 mg/kg LOAEL = 1.2 mg/kg-d / 2.0551 mg/kg-d * 14.6 mg/kg
	5.9 - 29.5 mg/kg	Red Fox	NOAEL- and LOAEL-based value to protect top mammalian predators, represented by red fox. Dietary NOAEL = 1 mg/kg-d, LOAEL = 5 mg/kg-d, average daily dose = 2.4764 mg/kg-d, mean site-wide U95 PCB Conc FP SED = 14.6 mg/kg	NOAEL = 1 mg/kg-d / 2.4764 mg/kg-d * 14.6 mg/kg LOAEL = 5 mg/kg-d / 2.4764 mg/kg-d * 14.6 mg/kg
	21 - 63 mg/kg	White-footed/ Deer Mouse	NOAEL- and LOAEL-based value to protect omnivorous rodents, represented by white-footed/deer mouse. Dietary NOAEL = 0.45 mg/kg-d, LOAEL = 1.35 mg/kg-d, average daily dose = 0.31094 mg/kg-d, mean site-wide U95 PCB Conc FP SED = 14.6 mg/kg	NOAEL = 0.45 mg/kg-d / 0.31094 mg/kg-d * 14.6 mg/kg LOAEL = 1.35 mg/kg-d / 0.31094 mg/kg-d * 14.6 mg/kg

[1] Assumes aquatic environment, exposures to instream sediment , site-wide (ABSAs 3-9) U95 total PCB concentration = 7.3 mg/kg

[2] Assumes terrestrial environment, exposure to floodplain sediments/soils, site-wide (ABSAs 3-9) U95 total PCB concentration = 14.6 mg/kg

6 Section Six

Section 6
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Appendices

Appendix A
Lists of Plant and Animal Species Present or Potentially Present at the Kalamazoo River Superfund Site

Appendix A
Lists of Plant and Animal Species Present or Potentially Present at the Kalamazoo River Superfund Site

Endangered Species

Table A-1 presents plant and animal species of special concern that may potentially occur in or near the API/PC/KR area.

Table A-1 Plant and Animal Species of Special Concern Potentially Occurring In or Near the API/PC/KR Area

Scientific Name	Common Name	County
Endangered Vertebrates		
Acipenser fulvescens	Lake Sturgeon	Allegan
Acris crepitans lanchardi	Blanchard's Cricket Frog	Kalamazoo, Allegan
Alasmidonta marginata	Elk Toe Mussel	Kalamazoo, Allegan
Ambystoma opacum	Marbled Salamander	Allegan
Ardea herodias	Great Blue Heron	Kalamazoo, Allegan
Clemmys guttata	Spotted Turtle	Kalamazoo, Allegan
Clemmys insculpta	Wood Turtle	Allegan
Clonophis kirtlandii	Kirtland's Snake	Kalamazoo
Cryptotis parva	Least Shrew	Kalamazoo
Erimyzon oblongus	Creek Chubsucker	Kalamazoo
Gavia immer	Common Loon	Allegan
Haliaeetus leucocephalus	Bald Eagle	Allegan
Ictiobus niger	Black Buffalo	Allegan
Lanius ludovicianus migrans	Loggerhead Shrike	Allegan
Lepisosteus oculatus	Spotted Gar	Kalamazoo
Microtus ochrogaster	Prairie Vole	Kalamazoo
Microtus pinetorum	Woodland Vole	Kalamazoo, Allegan
Notropis anogenus	Pugnose Shiner	Kalamazoo
Notropis texanus	Weed Shiner	Kalamazoo, Allegan
Rallus elegans	King Rail	Allegan
Sistrurus catenatus catenatus	Massasauga	Kalamazoo, Allegan
Terrapene carolina carolina	Eastern Box Turtle	Kalamazoo, Allegan
Endangered Invertebrates		
Calephelis mutica	Swamp Metalmark	Kalamazoo
Cyclonaias tuberculata	Purple Wartyback	Allegan
Hesperia ottoe	Ottoe Skipper	Allegan
Incisalia irus	Frosted Elfin	Allegan
Lycaeides melissa samuelis	Karner Blue	Allegan
Neonympha mitchellii mitchellii	Mitchell's Satyr	Kalamazoo
Nicrophorus americanus	American Burying Beetle	Kalamazoo
Pygaruei Spraguei	Sprague's Pygarctia	Kalamazoo, Allegan
Speyeria idalia	Regal Fritillary	Kalamazoo
Stylurus laurae	Laurea Snaketail	Kalamazoo
Endangered Vascular Plant Communities		
Agalinis gattingeri	Gattinger's Gerardia	Kalamazoo
Amorpha canescens	Leadplant	Kalamazoo
Angelica venenosa	Hairy Angelica	Kalamazoo
Arabis missouriensis var deamii	Missouri Rock-Cress	Kalamazoo, Allegan
Aristida dichotoma	Shinner's Three-Awned-Grass	Kalamazoo
Aster sericeus	Western Silvery Aster	Kalamazoo
Astragalus canadensis	Canadian Milk-Vetch	Kalamazoo

Table A-1 Plant and Animal Species of Special Concern Potentially Occurring In or Near the API/PC/KR Area

Scientific Name	Common Name	County
Astragalus neglectus	Cooper's Milk-Vetch	Kalamazoo
Baptisia lactea	White False Indigo	Kalamazoo, Allegan
Baptisia leucophaea	Cream Wild Indigo	Kalamazoo
Berula erecta	Cut-Leaved Water-Parsnip	Kalamazoo, Allegan
Besseya bullii	Kitten-Tails	Kalamazoo
Cacalia plantaginea	Prairie Indian-Plantain	Kalamazoo
Calamagrostis stricta	Narrow-Leaved Reedgrass	Kalamazoo
Carex albolutescens	Greenish-White Sedge	Kalamazoo, Allegan
Carex festucacea	Fescue Sedge	Kalamazoo, Allegan
Carex frankii	Frank's Sedge	Kalamazoo
Carex oligocarpa	Eastern Few-Fruited Sedge	Kalamazoo
Carex seorsa	Sedge	Kalamazoo
Carex straminea	Straw Sedge	Kalamazoo
Carya laciniosa	Shellbark Hickory	Kalamazoo
Cirsium hillii	Hill's Thistle	Kalamazoo
Cirsium pitcheri	Pitcher's Thistle	Allegan
Coreopsis palmata	Prairie Coreopsis	Kalamazoo
Corydalis flavula	Yellow Fumewort	Kalamazoo
Cuscuta campestris	Field Dodder	Kalamazoo
Cuscuta pentagona	Dodder	Kalamazoo
Cuscuta polygonorum	Knotweed Dodder	Kalamazoo
Cyperus flavescens	Yellow Nut-Grass	Kalamazoo, Allegan
Cypripedium candidum	White Lady-Slipper	Kalamazoo
Diarrhena americana	Beak Grass	Kalamazoo
Draba reptans	Creeping Whitlow-Grass	Kalamazoo
Dryopteris Celsa	Log Fern	Kalamazoo
Echinodorus tenellus	Dwarf Burhead	Allegan
Eleocharis compressa	Flattened Spike-Rush	Kalamazoo
Eleocharis engelmannii	Engelmann's Spike-Rush	Kalamazoo, Allegan
Eleocharis melanocarpa	Black-Fruited Spike-Rush	Kalamazoo, Allegan
Eleocharis microcarpa	Small-Fruited Spike-Rush	Allegan
Eleocharis tricostata	Three-Ribbed Spike-Rush	Allegan
Eryngium yuccifolium	Rattlesnake-Master	Kalamazoo
Euphorbia commutata	Tinted Spurge	Allegan
Eupatorium sessilifolium	Upland Boneset	Kalamazoo
Filipendula rubra	Queen-of-the-Prairie	Kalamazoo
Fuirena squarrosa	Umbrella Grass	Kalamazoo, Allegan
Gentiana flavida	White Gentian	Kalamazoo
Gentiana puberulenta	Downy Gentian	Allegan
Geum triflorum	Prairie-Smoke	Allegan
Gillenia trifoliata	Bowman's Root	Kalamazoo
Glyceria acutiflora	Manna Grass	Kalamazoo
Gymnocladus dioicus	Kentucky Coffee Tree	Kalamazoo
Helianthus hirsutus	Whiskered Sunflower	Kalamazoo
Hemicarpha micrantha	Dwarf-Bulrush	Kalamazoo, Allegan
Hibiscus moscheutos	Swamp Rose Mallow	Allegan
Hybanthus concolor	Green Violet	Kalamazoo
Hydrastis canadensis	Goldenseal	Kalamazoo, Allegan
Hypericum gentianoides	St. John's Wort	Kalamazoo
Isoetes engelmannii	Appalachian Quillwort	Allegan
Isotria verticillata	Whorled Pogonia	Kalamazoo
Juncus biflorus	Two-Flowered Rush	Kalamazoo, Allegan
Juncus brachycarpus	Short-Fruited Rush	Allegan

Table A-1 Plant and Animal Species of Special Concern Potentially Occurring In or Near the API/PC/KR Area

Scientific Name	Common Name	County
Juncus scirpoides	Scirpus-Flowered Rush	Kalamazoo, Allegan
Juncus vaseyi	Vasey's Rush	Allegan
Kuhnia eupatorioides	False Boneset	Kalamazoo
Lechea minor	Least Pinweed	Kalamazoo
Lechea pulchella		Kalamazoo
Lechea stricta	Erect Pinweed	Kalamazoo
Lemna valdiviana	Pale Duckweed	Kalamazoo
Liatris punctata	Dotted Blazing Star	Kalamazoo
Lindernia anagallidea	False Pimpernel	Kalamazoo
Linum sulcatum	Furrowed Flax	Kalamazoo
Linum virginianum	Virginia Flax	Kalamazoo
Ludwigia alternifolia	Seedbox	Kalamazoo, Allegan
Lycopodium appressum	Fern	Kalamazoo, Allegan
Lygodium palmatum	Climbing Fern	Kalamazoo
Morus rubra	Red Mulberry	Kalamazoo
Muhlenbergia richardsonis	Mat Muhly	Kalamazoo
Nelumbo lutea	American Lotus	Kalamazoo
Panax quinquefolius	Ginseng	Kalamazoo, Allegan
Panicum leibergii	Leiberg's Panic-Grass	Kalamazoo
Panicum longifolium	Long-Leaved Panic Grass	Allegan
Platanthera ciliaris	Orange-Finged Orchid	Kalamazoo, Allegan
Poa Paludigena	Bog Bluegrass	Kalamazoo
Polygala cruciata	Cross-Leaved Milkwort	Kalamazoo, Allegan
Polygonum careyi	Carey's Smartweed	Allegan
Populus heterophylla	Swamp Cottonwood	Kalamazoo
Potamogeton bicupulatus	Waterthread Pondweed	Allegan
Pycnanthemum verticillatum	Whorled Mountain-Mint	Allegan
Querus alba	White Oak	Allegan
Rhexia mariana var mariana	Maryland Meadow-Beauty	Allegan
Rhexia virginica	Meadow-Beauty	Kalamazoo, Allegan
Rhynchospora macrostachya	Tall Beak-Bush	Kalamazoo, Allegan
Rosa setigera	Prairie Rose	Kalamazoo
Rotala ramosior	Tooth-Cup	Kalamazoo, Allegan
Rudbeckia sullivantii	Showy Coneflower	Kalamazoo, Allegan
Sabatia angularis	Rose-Pink	Kalamazoo
Scirpus hallii	Hall's Bulrush	Allegan
Scirpus torreyi	Torrey's Bulrush	Allegan
Scleria reticularis	Netted Nut-Rush	Allegan
Scleria triglomerata	Tall Nut-Rush	Kalamazoo, Allegan
Scutellaria elliptica	Hairy Skullcap	Kalamazoo
Silene stellata	Starry Campion	Kalamazoo
Silphium intergrifolium	Rosinweed	Kalamazoo
Silphium laciniatum	Compass-Plant	Kalamazoo
Silphium perfoliatum	Cup-Plant	Kalamazoo
Sisyrinchium atlanticum	Atlantic Blue-Eyed Grass	Allegan
Smilax herbacea	Smooth Carrion-Flower	Kalamazoo
Spiranthes ovalis	Lesser Ladies'-Tresses	Kalamazoo
Sporobolus heterolepis	Prairie Dropseed	Kalamazoo, Allegan
Stellaria crassifolia	Fleshy Stitchwort	Kalamazoo
Trichostema dichotomum	Bastard Pennyroyal	Kalamazoo, Allegan
Trillium sessile	Toadshade	Kalamazoo
Triphora trianthrophora	Three-Birds Orchid	Kalamazoo
Utricularia subulata	Zigzag Bladderwort	Allegan

Table A-1 Plant and Animal Species of Special Concern Potentially Occurring In or Near the API/PC/KR Area

Scientific Name	Common Name	County
Valeriana ciliata	Edible Valerian	Kalamazoo
Valerianella chenopodiifolia	Goosefoot Corn-Salad	Kalamazoo
Viola pedatifida	Prairie Birdfoot Violet	Kalamazoo
Zizania aquatica var aquatica	Wild Rice	Kalamazoo

Table A-2 Plant Species Potentially Occurring In or Near the API/RI/KR Area

Family	Species	Common Name
Trees and Woody Plants		
Pinaceae	*Larix laricina*	Tamarack
	Pinus strobus	Eastern White Pine
	Pinus banksiana	Jack Pine
	Pinus resinosa	Red Pine
Annonaceae	*Asimina triloba*	Pawpaw
Magnoliaceae	*Liriodendron tulipifera*	Tuliptree
	Tilia americana	American Basswood
Salicaceae	*Populus deltoides*	Eastern Cottonwood
	Salix amygdaloides	Peachleaf Willow
	Salix nigrum	Black Willow
	Salix exigna	Sandbar Willow
	Salix discolor	Pussy Willow
Rosaceae	*Malus coronaria*	Wild Crab Apple
	Malus pumila	Common Apple
	Amelanchier arborea	Downy Serviceberry
	Prunus nigra	Canada Plum
	Prunus pensylvanica	Pin Cherry
	Prunus serotina	Black Cherry
	Prunus virginiana	Chokecherry
Fabaceae	*Gymnocladus dioicus*	Kentucky Coffeetree
	Gleditsia triacanthos	Honeylocust
	Cercis canadensis	Red Bud
Cornaceae	*Cornus alternifolia*	Alternate Leaf Dogwood
	Cornus florida	Flowering Dogwood
	Cornus stolonifera	Red Osier Dogwood
Hippocastanaceae	*Aesculus glabra*	Ohio Buckeye
Aceraceae	*Acer nigrum*	Black Maple
	Acer saccharum	Sugar Maple
	Acer rubrum	Red Maple
	Acer saccharinum	Silver Maple
	Acer negundo	Boxelder
Juglandaceae	*Juglans cinerea*	Butternut
	Juglans nigra	Black Walnut
	Carya cordiformis	Bitternut Hickory
	Carya glabra	Pignut Hickory
	Carya laciniosa	Shellbark Hickory
	Carya ovata	Shagbark Hickory
Hamamelidaceae	*Hamamelis virginiana*	Witch-Hazel
Betulaceae	*Betula alleghaniensis*	Yellow Birch
	Betula papyrifera	White Birch
	Alnus rugosa	Speckled Alder
	Carpinus caroliniana	Blue Beech
	Ostrya virginiana	Hop-Hornbeam
Ulmaceae	*Celtis occidentalis*	Northern Hackberry

Table A-2 Plant Species Potentially Occurring In or Near the API/RI/KR Area

Family	Species	Common Name
	Celtis tenuifolia	Dwarf Hackberry
	Ulmus americana	American Elm
	Ulmus thomasii	Rock Elm
	Ulmus rubra	Slippery Elm
Moraceae	*Morus rubra*	Red Mulberry
Fagaceae	*Castanea dentata*	American Chestnut
	Fagus grandifolia	Beech
	Quercus alba	White Oak
	Quercus bicolor	Swamp White Oak
	Quercus muehlenbergii	Chipkapin Oak
	Quercus prinoides	Dwarf Chinkapin Oak
	Quercus rubra	Red Oak
	Quercus velutina	Black Oak
	Quercus coccinea	Scarlet Oak
	Quercus ellipsoidalis	Northern Pin Oak
	Quercus palustris	Pin Oak
	Quercus imbricaria	Shingle Oak
Platanaceae	*Platanus occidentalis*	Sycamore
Caprifoliaceae	*Viburnum lentago*	Nannyberry
Oleaceae	*Fraxinus americana*	White Ash
	Fraxinus nigra	Black Ash
	Fraxinus pennsylvanica	Red Ash
	Fraxinus quadrangulata	Blue Ash
Lauraceae	*Lindera benzoin*	Spicebush
Aquifoliaceae	*Ilex verticillata*	Winterberry
Anacardiaceae	*Toxicodendron vernix*	Poison Sumac
Grasses, Wildflowers, and Shrubs		
	Salix discolor	Pussy Willow
	Typha latifolia	Cattail
	Saururus cernuus	Lizard's Tail
	Rosa palustris	Swamp Rose
	Lythrum salicaria	Purple Loosestrife
	Iris versicolor	Blue Flag
	Pinguicula vulgaris	Common Butterwort
	Peltandra virginica	Arrow arum
	Lemna	Duckweed
	Polygonum amphibium	Smartweed
	Nymphaea odorata	Fragrant Water Lily
	Sambucus canadensis	Elderberry
	Nyssa sylvatica	Black Tupelo
	Salix discolor	Pussy Willow
	Salix bebbiana	Bebb Willow

References: Barnes and Wagner 1981; Vines 1984; Nierung 1985; MDNR 1971 and 1994

Table A-3 Insect Species Potentially Occurring In or Near the API/PC/KR Area

Family	Species	Common Name
Arthropods (Phylum Arthropoda) (aquatic and terrestrial)		
Insects	***Class Insecta***	
	Order Hymenoptera	Ants, Bees, Wasps
	Order Diptera - (Two species of aquatic Diptera)	Flies, Midges, Mosquitoes
	Order Odonata - Two species of Odonata	Dragonflies and Damselflies
	Order Ephemeroptera - Six species of Ephemeroptera	Mayflies
	Order Tricoptera - Five species of Trichoptera	Caddisflies
	Order Plecoptera	Stoneflies
	Order Orthoptera	Grasshoppers and Crickets
	Order Coleoptera - Two species of aquatic Coleoptera	Beetles
	Order Hemiptera	True Bugs
	Order Lepidoptera - One species of aquatic Lepidoptera	Butterflies and moths
	Class Arachnida	Spiders, Scorpions, Mites, Ticks
	Class Isopoda	Isopods
	Class Branchiopoda - One species of Daphnia	Cladocerans
	Class Amphipoda	Amphipods
	Class Chilopoda	Centipedes
	Class Diplopoda	Millipedes
Flatworms Phylum Platyhelminthes		
	Class Turbellaria - two species	Turbellarians
Segmented Worms and Leeches		
Phylum Annelida	*Class Oligochaeta*	Earthworms and related worms
	Class Hirudinea	Leeches
Molluscs		
Phylum Mollusca	*Class Gastropoda - Two species of Gastropoda*	Snails and Slugs
	Class Bivalvia	Freshwater Clams
Bryozoans		
Phylum Ectoprocta	*- two species of Bryozoa*	

References: MDNR 1987; Niering 1985; Milne and Milne 1980.

Table A-4 Fish Species Potentially Occurring In or Near the API/PC/KR Area

Family	Species	Common Name
Amiidae	*Amia calva*	Bowfin
Clupeidae	*Alosa pseudoharengus*	Alewife
	Dorsoma cepedianum	Gizzard shad
Umbridae	*Umbra limi*	Central mudminnow
	Esox americanus	Mud pickerel
	Esox lucius	Northern pike
Characidae	*Cyprinus carpio*	Common Carp
	Notemigonus crysoleucas	Golden shiner
	Semotilus atromaculatus	Creek chub
	Nocomis biguttatus	Hornyhead chub
	Rhinichthys atratulus	Blacknose dace
	Luxilus chrysocephalus	Striped shiner
	Luxilus cornutus	Common shiner
	Cyprinella spilopterus	Spotfin shiner
	Pimephales notatus	Bluntnose minnow
	Notropis atherinoides	Emerald shiner
	Notropis ludibundus	Sand shiner
	Notropis volucellus	Mimic shiner
	Notropis hudsonius	Spottail shiner
Catostomidae	*Carpiodes cyprinus*	Quillback
	Catostomus commersoni	White sucker
	Minytrema melanops	Spotted sucker
	Erimyzon oblongus	Creek chubsucker
	Hypentelium nigricans	Northern hog sucker
	Moxostoma breviceps	Shorthead redhorse
	Moxostoma duquesnei	Black redhorse
	Moxostoma erythrurum	Golden redhorse
	Moxostoma anisurum	Silver redhorse
	Moxostoma macrolepidotum	Northern redhorse
Ictaluridae	*Ameiurus natalis*	Yellow bullhead
	Ameiurus melas	Black bullhead
	Ameiurus nebulosus	Brown bullhead
	Ictalurus punctatus	Channel catfish
	Pylodictis olivaris	Flathead catfish
	Noturus flavus	Stonecat
	Noturus gyrinus	Tadpole madtom
Aphredoderidae	*Aphredoderus sayanus*	Pirate perch
Gadidae	*Lota lota*	Burbot
Atherinidae	*Labidesthes sicculus*	Brook silverside
Centrarachidae	*Pomoxis nigromaculatus*	Black crappie
	Ambloplites rupestris	Rock bass
	Micropterus salmoides	Largemouth bass
	Micropterus dolomieu	Smallmouth bass
	Lepomis cyanellus	Green sunfish
	Lepomis macrochirus	Bluegill
	Lepomis gibbosus	Pumpkinseed
	Lepomis megalotis	Longear sunfish
Percidae	*Stizostedion vitreum*	Walleye
	Perca flavescens	Yellow perch
	Percina maculata	Blackside darter
	Percina caprodes	Logperch
	Etheostoma nigrum	Johnny darter
	Etheostoma exile	Iowa darter
Sciaenidae	*Aplodinotus grunniens*	Freshwater drum

Amphibians

Table A-5 identifies all amphibian species and subspecies that occur within the general site area. Occurrence onsite is expected to be limited by specific habitat requirements. Species recently observed onsite are identified with an asterisk (*).

Table A-5 Amphibians Potentially Occurring In or Near the API/PC/KR Area

Family	Species	Common Name
Proteidae	*Necturus masculosus*	Mudpuppy
Sirenidae	*Siren intermedia nettingi*	Western Lesser Siren
Ambystomatidae	*Ambystoma laterale*	Blue Spotted Salamander
	Ambystoma maculatum	Spotted Salamander
	Ambystoma opacum	Marbled Salamander
	Ambystoma tigrinum tigrinum	Tiger Salamander
Salamandridae	*Notophthalmus viridescens louisianensis*	Central Newt
Plethodontidae	*Plethodon cinereus*	Red-Backed Salamander
	Hemidactylium scutatum	Four-Toed Salamander
Bufonidae	**Bufo americanus americanus*	Eastern American Toad
	Bufo woodhousii fowleri	Fowler's Toad
Hylidae	*Acris crepitans blanchardi*	Blanchard's Cricket Frog
	Pseudacris triseriata triseriata	Western Chorus Frog
	Pseudacris triseriata maculata	Boreal Chorus Frog
	Pseudacris crucifer crucifer	Northern Spring Peeper
	Hyla versicolor	Eastern Gray Treefrog
	Hyla chrysoscelis	Cope's Gray Treefrog
Ranidae	*Rana clamitans melanota*	Green Frog
	**Rana catesbeiana*	Bull Frog
	**Rana pipiens*	Northern Leopard Frog
	Rana palustris	Pickerel Frog
	Rana sylvatica	Wood Frog

References: Conant 1975; Behler and King 1979; Harding 1992

Reptiles

Table A-6 identifies all reptile species and subspecies that occur within the general site area. Occurrence onsite is expected to be limited by specific habitat requirements. Species recently observed onsite are identified with an asterisk (*).

Table A-6 Reptiles Potentially Occurring In or Near the API/PC/KR Area

Family	Species	Common Name
Chelydridae	**Chelydra serpentina*	Common Snapping Turtle
Kinosternidae	*Sternotherus odoratus*	Musk Turtle (Stinkpot)
Emydidae	*Clemmys guttata*	Spotted Turtle
	Clemmys insculpta	Wood Turtle
	**Terrapene carolina carolina*	Eastern Box Turtle
	Emydoidea blandingii	Blanding's Turtle
	**Graptemys geographica*	Map Turtle
	**Chrysemys picta marginata*	Midland Painted Turtle
Trionychidae	*Trionyx spinifera spinifera*	Eastern Spiny Softshell
Scincidae	*Eumeces fasciatus*	Five Lined Skink
Colubridae	*Clonophis kirtlandii*	Kirtland's Water Snake
	Nerodia erythrogaster neglecta	Northern Copperbelly Snake
	Nerodia sipedon sipedon	Northern Water Snake
	Regina septemvittata	Queen Snake
	Storeria dekayi	Brown Snake
	Storeria occipitomaculata occipitomaculata	Northern Redbellied Snake
	**Thamnophis sirtalis sirtalis*	Eastern Garter Snake
	Thamnophis sauritus septentrionalis	Northern Ribbon Snake
	Diadophis punctatus edwardsi	Northern Ringneck Snake
	Heterodon platyrhinos	Eastern Hognose Snake
	Coluber constrictor foxi	Blue Racer
	Elaphe obsoleta obsoleta	Black Rat Snake
	Lampropeltis triangulum triangulum	Eastern Milk Snake
	Opheodrys vernalis vernalis	Eastern Smooth Green Snake
Viperidae Crotalinae	*Sistrurus catenatus catenatus*	Eastern Massasauga Rattlesnake

References: Conant 1975; Behler and King 1979; Harding 1990; Holman 1989
* Species recently observed

Table A-7 Avian Species Potentially Occurring In or Near the API/PC/KR Area

Family	Species	Common Name	Status	Abundance
Gaviidae	*Gavia immer*	Common Loon	Transient	Accidental
Ardeidae	*Ardea herodias*	Great blue heron	Summer	Irregular
Gruidae	*Grus canadensis*	Sandhill crane	Transient	Accidental
Anatidae	*Cygnus columbianus*	Whistling swan	Transient	Accidental
	Cygnus buccinator	Trumpeter swan	Transient	Accidental
	Chen caerulescens	Snow Goose	Transient	Accidental
	Anser c. caerulescens	Blue goose	Transient	Accidental
	Branta canadensis	Canada goose	Transient	Irregular
	Anas platyrhynchos	Mallard duck	Permanent	Common
	Anas rubripes	Black duck	Permanent	Irregular
	Anas strepera	Gadwall	NA	NA
	Anas crecca	Green winged teal	Summer	Irregular
	Anas acuta	Northern pintail	NA	NA
	Anas discors	Blue winged teal	Summer	Irregular
	Aix sponsa	Wood duck	Summer	Uncommon
	Aythya valisineria	Canvasback duck	NA	NA
	Aythya americana	Redhead duck	NA	NA
	Aythya affinis	Lesser scaup	Transient	Accidental
	Bucephala clangula	Common goldeneye	Winter	Common
	Bucephala albeola	Bufflehead	NA	NA
	Mergus merganser	American merganser	Winter	Accidental
Rallidae	*Porphyrula martinica*	American gallinule	NA	NA
	Fulica americana	American coot	Transient	Accidental
Charadriidae	*Charadrius vociferus*	Killdeer	Summer	Common
Scolopacidae	*Tringa solitaria*	Solitary sandpiper	Transient	Irregular
	Actitis macularia	Spotted sandpiper	Transient	Rare
	Gallinago gallinago	Wilson's snipe	Transient	Irregular
	Scolopax minor	American woodcock	Transient	Rare
	Calidris melantos	Pectoral sandpiper	Transient	Accidental
	Bartramia longicauda	Upland sandpiper	NA	NA
Laridae	*Larus philadelphia*	Bonaparte's gull	Transient	Accidental
	Larus delawarensis	Ring-billed gull	Transient	Rare
	Larus argentatus	Herring gull	Transient	Rare
	Chlidonias niger	Black tern	Transient	Accidental
Cathartidae	*Cathartes aura*	Turkey vulture	Summer	Uncommon
Accipitridae	*Aquila chrysaetos*	Golden eagle	NA	NA
	Haliaeetus leucocephalus	Bald eagle	Transient	Accidental
	Accipiter striatus	Sharp-shinned hawk	Transient	Uncommon
	Accipiter cooperii	Cooper's hawk	Permanent	Rare
	Buteo lineatus	Red-shouldered hawk	Transient	Rare
	Buteo platypterus	Broad-winged hawk	Transient	Irregular
	Buteo jamaicensis	Red-tailed hawk	Permanent	Uncommon
	Buteo lagopus	Rough-legged hawk	Winter	Irregular
	Pandion haliaetus	Osprey	Transient	Irregular
Phasianidae	*Bonasa umbellus*	Ruffed grouse	Permanent	Uncommon
	Colinus virginianus	Bobwhite quail	Permanent	Uncommon
	Phasianus colchicus	Ring-necked pheasant	Permanent	Common
	Meleagris gallopavo	Wild turkey	Permanent	Common
Columbidae	*Columba livia*	Rock dove	Permanent	Common
	Zenaida macroura	Mourning dove	Permanent	Common
Cuculidae	*Coccyzus americanus*	Yellow-billed cuckoo	Summer	Uncommon
	Coccyzus erythropthalmus	Black-billed cuckoo	Summer	Uncommon

Table A-7 Avian Species Potentially Occurring In or Near the API/PC/KR Area

Family	Species	Common Name	Status	Abundance
Tytonidae & Strigidae	*Tyto alba*	Barn owl	NA	NA
	Asio flammeus	Short-eared owl	Winter	Accidental
	Asio otus	Long-eared owl	Winter	Accidental
	Bubo virginianus	Great horned owl	Permanent	Uncommon
	Strix varia	Barred owl	Summer	Accidental
	Otus asio	Screech owl	Permanent	Uncommon
	Aegolius acadicus	Northern saw-whet	Transient	Accidental
Caprimulgidae	*Caprimulgus vociferus*	Whip-poor-will	Transient	Accidental
	Chordeiles minor	Common nighthawk	Transient	Uncommon
Trochilidae	*Archilochus colubris*	Ruby throated-hummingbird	Summer	Rare
Alcedinidae	*Ceryle alcyon*	Belted kingfisher	Permanent	Uncommon
Picidae	*Melanerpes carolinus*	Red bellied-woodpecker	Permanent	Uncommon
	Melanerpes erythrocephalus	Red headed-woodpecker	Summer	Common
	Picoides pubescens	Downy woodpecker	Permanent	Common
	Picoides villosus	Hairy woodpecker	Permanent	Uncommon
	Dryocopus pileatus	Pileated woodpecker	Transient	Accidental
Tyrannidae	*Tyrannus tyrannus*	Eastern Kingbird	Summer	Common
	Myiarchus crinitus	Great crested-flycatcher	Summer	Common
	Sayornis phoebe	Eastern phoebe	Summer	Uncommon
	Empidonax virescens	Acadian flycatcher	Summer	Irregular
	Empidonax traillii	Willow flycatcher	Summer	Common
	Empidonax flaviventris	Yellow bellied-flycatcher	Transient	Irregular
Alaudidae	*Eremophila alpestris*	Horned lark	Permanent	Common
Hirundinidae	*Tachycineta bicolor*	Tree swallow	Summer	Common
	Progne subis	Purple martin	Summer	Uncommon
	Riparia riparia	Bank Swallow	Transient	Irregular
	Stelgidopteryx serripennis	Rough-winged swallow	Transient	Irregular
	Hirundo pyrrhonota	Cliff Swallow	Transient	Accidental
	Hirundo rustica	Barn Swallow	Summer	Common
Corvidae	*Cyanocitta cristata*	Blue jay	Permanent	Common
	Corvus brachyrhynchos	Common Crow	Permanent	Common
Paridae	*Parus bicolor*	Tufted titmouse	Permanent	Common
	Parus atricapillus	Black capped-chickadee	Permanent	Common
Certhiidea	*Certhia americana*	Brown creeper	Winter	Uncommon
Sittidae	*Sitta carolinensis*	White breasted-nuthatch	Permanent	Common
	Sitta canadensis	Red breasted-nuthatch	Transient	Rare
Troglodytidae	*Troglodytes aedon*	House wren	Summer	Common
	Troglodytes troglodytes	Winter wren	Transient	Uncommon
	Cistothorus palustris	Marsh wren	Transient	Accidental
Muscicapidae	*Regulus satrapa*	Golden crowned-kinglet	Transient	Common
	Regulus calendula	Ruby crowned-kinglet	Transient	Common
	Polioptilla caerulea	Blue-gray gnatcatcher	Summer	Irregular
	Sialia sialis	Eastern bluebird	Summer	Common
	Hylocichla mustelina	Wood thrush	Summer	Uncommon
	Catharus fuscescens	Veery	Transient	Uncommon
	Catharus ustulatus	Swainson's thrush	Transient	Common
	Catharus minimus	Gray-cheeked thrush	Transient	Common
	Catharus guttatus	Hermit thrush	Transient	Common
	Turdus migratorius	American robin	Summer	Common
Laniidae	*Lanius ludovicianus*	Loggerhead shrike	Transient	Accidental
	Lanius excubitor	Northern shrike	Winter	Accidental
Mimidae	*Dumetella carolinensis*	Gray catbird	Summer	Common
	Mimus polyglottos	Mockingbird	Summer	Accidental
	Toxostoma rufum	Brown thrasher	Summer	Common

Table A-7 Avian Species Potentially Occurring In or Near the API/PC/KR Area

Family	Species	Common Name	Status	Abundance
Cinclidae	*Bombycilla cedrorum*	Cedar waxwing	Permanent	Common
Sturnidae	*Sturnus vulgaris*	European Starling	Permanent	Common
Vireonidae	*Vireo flavifrons*	Yellow-throated vireo	Summer	Rare
	Vireo solitarius	Solitary vireo	Transient	Rare
	Vireo olivaceus	Red-eyed vireo	Summer	Common
	Vireo philadelphicus	Philadelphia vireo	Transient	Rare
Emberizidae	*Vermivora pinus*	Blue-winged warbler	Summer	Common
	Vermivora chrysoptera	Golden winged-warbler	Transient	Uncommon
	Vermivora peregrina	Tennessee warbler	Transient	Common
	Vermivora celata	Orange crowned-warbler	Transient	Rare
	Vermivora ruficapilla	Nashville warbler	Transient	Common
	Dendroica caerulescens	Black-throated blue-warbler	Transient	Uncommon
	Dendroica cerulea	Cerulean warbler	Transient	Rare
	Dendroica fusca	Blackburnian warbler	Transient	Uncommon
	Dendroica pennsylvanica	Chesnut-sided-warbler	Transient	Common
	Dendroica coronata	Yellow rumped-warbler	Transient	Common
	Dendroica virens	Black-throated green-warbler	Transient	Common
	Dendroica pinus	Pine warbler	Transient	Irregular
	Dendroica palmarum	Palm warbler	Transient	Uncommon
	Dendroica petechia	Yellow warbler	Summer	Common
	Oporornis philadelphia	Mourning Warbler	Transient	Irregular
	Oporornis agilis	Connecticut Warbler	Transient	Accidental
	Wilsonia canadensis	Canada warbler	Transient	Uncommon
	Wilsonia pusilla	Wilson's Warbler	Transient	Uncommon
	Wilsonia citrina	Hooded warbler	Transient	Accidental
	Seiurus aurocapillus	Ovenbird	Summer	Common
	Seiurus motacilla	Louisiana water-thrush	Summer	Irregular
	Seiurus noveboracensis	Northern water-thrush	Transient	Rare
	Geothlypis trichas	Common yellow-throat	Summer	Common
	Setophaga ruticilla	American Redstart	Transient	Common
	Cardinalis cardinalis	Northern Cardinal	Permanent	Common
	Passerina cyanea	Indigo bunting	Summer	Common
	Pipilo erythropthalmus	Rufous-sided towhee	Summer	Common
	Ammodramus savannarum	Grasshopper sparrow	Summer	Uncommon
	Ammodramus henslowii	Henslow's sparrow	Summer	Irregular
	Pooecetes gramineus	Vesper sparrow	Summer	Uncommon
	Melospiza melodia	Song sparrow	Permanent	Common
	Spizella arborea	Tree sparrow	Winter	Common
	Spizella pusilla	Field sparrow	Summer	Common
	Spizella pallida	Clay-colored sparrow	Transient	Accidental
	Junco hyemalis	Dark-eyed junco	Winter	Common
	Zonotrichia albicollis	White throated-sparrow	Transient	Common
	Zonotrichia leucophrys	White crowned-sparrow	Transient	Common
	Passerella iliaca	Fox sparrow	Transient	Uncommon
	Melospiza lincolnii	Lincoln's sparrow	Transient	Rare
	Melospiza georgiana	Swamp sparrow	Transient	Uncommon
	Dolichonyx oryzivorus	Bobolink	Summer	Uncommon
	Sturnella magna	Eastern Meadowlark	Summer	Common
	Sturnella neglecta	Western Meadowlark	Summer	Accidental
	Agelaius phoeniceus	Red-winged blackbird	Summer	Common
	Molothrus ater	Brown headed-cowbird	Summer	Common
	Quiscalus quiscalus	Common grackle	Summer	Common
	Icterus spurius	Orchard oriole	Summer	Accidental
	Icterus galbula	Northern oriole	Summer	Common

Table A-7 Avian Species Potentially Occurring In or Near the API/PC/KR Area

Family	Species	Common Name	Status	Abundance
	Piranga olivacea	Scarlet tanager	Summer	Uncommon
Passeridae	*Passer domesticus*	House sparrow	Permanent	Common
Fringillidae	*Carduelis tristis*	American goldfinch	Permanent	Common
	Carpodacus purpureus	Purple finch	Winter	Common
	Coccothraustes vespertinus	Evening grosbeak	Winter	Irregular

Definitions:

Permanent resident	Species which remain year round and breed in the area during Spring and/or Summer.
Summer resident	Species which nest in the area, but migrate to the south for the winter.
Winter resident	Species which arrive in the Fall and leave for more northern breeding grounds in the Spring.
Transient resident	Species which pass through in the Spring and/or Fall and normally do not remain in Summer or Winter.
Common	Regularly recorded in large numbers.
Uncommon	Regularly recorded in small numbers.
Rare	Seldom recorded more than two or three times per year/season.
Irregular	Not recorded every year, but may be somewhat common in certain areas.
Accidental	Recorded on less than five occasions.
NA	Data not available.

References: Adams 1974; McPeek and Adams 1994; National Geographic Society (2nd ed.)

Mammals

Table A-8 identifies mammals whose range encompasses the general site area. Species examples are the most common or wide-ranging species within the group. Rare mammals, those known to occur only within certain limited areas, or those that do not occur in areas impacted by human use are not included.

Table A-8 Mammals Potentially Occurring In or Near the API/PC/KR Area

Family	Species	Common Name
Didelphidae	*Didelphis virginiana*	Opossum
Soricidae	*Sorex cinereus*	Masked shrew
	Blarina brevicauda	Short-tailed shrew
	Cryptotis parva	Least shrew
Talpidae	*Scalopus aquaticus*	Eastern mole
	Condylura cristata	Star-nosed mole
Vespertilionidae	*Myotis lucifugus*	Little brown bat
	Lasionycteris noctivagans	Silver-haired bat
	Eptesicus fuscus	Big brown bat
	Lasiurus borealis	Red bat
	Lasiurus cinereus	Hoary bat
	Nycticeius humeralis	Evening bat
Leporidae	*Sylvilagus floridanus*	Eastern cottontail
Sciuridae	*Tamias striatus*	Eastern chipmunk
	Marmota monax	Woodchuck
	Spermophilus franklinii	Franklin's ground squirrel
	Spermophilus tridecemlineatus	Thirteen-lined ground squirrel
	Sciurus carolinensis	Gray squirrel
	Sciurus niger	Fox squirrel
	Tamiasciurus hudsonicus	Red squirrel
	Glaucomys sabrinus	Northern flying squirrel
	Glaucomys volans	Southern flying squirrel
Castoridae	*Castor canadensis*	Beaver
Cricetidae	*Peromyscus leucopus*	White-footed mouse
	Peromyscus maniculatus	Deer mouse
	Microtus pennsylvanicus	Meadow vole
	Microtus pinetorum	Woodland vole
	Ondatra zibethicus	Muskrat
	Synaptomys cooperi	Southern bog lemming
Muridae	*Mus musculus*	House mouse
Zapodidae	*Zapas hudsonius*	Meadow jumping mouse
Canidae	*Canis latrans*	Coyote
	Vulpes vulpes	Red fox
	Urocyon cinereoargenteus	Gray fox
Procyonidae	*Procyon lotor*	Raccoon
Mustelidae	*Mustela erminea*	Ermine
	Mustela frenata	Long-tailed weasel
	Mustela nivalis	Least weasel
	Mustela vison	Mink
	Taxidea taxus	Badger
	Mephitis mephitis	Stiped Skunk
	Lutra canadensis	River otter
Cervidae	*Odocoileus virginianus*	White-tailed deer

References: Baker 1983; Davis 1978

Appendix B
Exposure-Related Data for Representative Receptors

Appendix B
Exposure-Related Data for Representative Receptors

Red Fox (*Vulpes vulpes*)

Red fox are native to most of North America, but are most abundant in Canada and the northern United States. Red fox are most often found in rural areas; however, they may also inhabit small areas within urban communities where suitable habitat is available. In Michigan, red fox are found in every county and on most of the major islands of the Great Lakes.

Habitat. Red fox prefer habitats that provide both adequate cover and prey. The most suitable habitats for red fox are fallow fields, cultivated fields, meadows, bushy fence lines, woody streams, and low shrub cover adjacent to woodlands or waterbodies (Baker 1983). Red fox construct burrows, which are used as refuges and for rearing young. The burrows are usually located in a well-drained area; however, red fox may sometimes construct dens on river islands (Arnold 1956). These burrows may extend 10 to 30 feet below the ground surface (Baker 1983).

Density and Movement. Red fox are highly mobile and forage extensively when food is limited. The home range is dependent on topography, vegetation, and prey availability (Baker 1983). Typically, a home range area will be comprised of an adult pair, their offspring, and occasionally a stray adult. The home range of red fox varies seasonally. During autumn, juvenile foxes are dispersing from the burrows in search for their own home range. Males will disperse an average of 18.4 miles during late September to early October. However, females will only disperse an average of 6.2 miles and do not leave the burrow until a month after the males (Phillips, et al. 1972). In the winter months the daily average home range is 900 acres, and nightly travels average 5 miles (Arnold and Schofield 1956). In the spring, there is commonly one fox family, averaging 7.4 individuals, sharing a home range of 2,471 acres (Shick 1952). In Michigan, the typical home range for a pair of red fox is 1,200 acres (Murie 1936).

Behavior. Red fox are nocturnal, and are active 8 to 10 hours per 24-hour day. Eighty percent of this time is spent traveling. Red fox are also capable of swimming, which allows utilization of streams and rivers for food sources. In addition, red fox are burrowing animals and therefore spend much of their time digging.

Reproductive Activities. Red fox are capable of producing one litter of pups per year. The breeding season begins in December and continues through March. The gestation period is 51 to 54 days. The average litter is five pups (average range is four to six pups), depending on location. In the Upper Peninsula of Michigan, the average litter is four pups, while six pups are average in the Lower Peninsula (Schofield 1958). The pups are weaned at 60 days, and after 120 days the pups are able to hunt. The average life expectancy of a red fox is 3 years (Baker 1983). Hunting and trapping account for

80 percent of fox mortalities (Baker 1983). There is also evidence that red fox populations fluctuate in 10-year cycles (Baker 1983).

Food Habits. Red fox are omnivores, but about 90 percent of the diet are of animal origin. Red fox consume on average 10 percent plants, 20 percent invertebrates, 15 percent reptiles and amphibians (herps), 15 percent birds, and 40 percent mammals (EPA 1994). The diet includes several species identified in the Kalamazoo River Food Web, including deer mice, muskrat, mink, snapping turtles, and great horned owls.

Economic Importance. Red fox are hunted and trapped. Their furs are valued at $5 to $150 each, depending on the annual supply and demand (Baker 1983).

Deer Mouse (*Peromyscus maniculatus bairdii*)

Deer mice are small ground-dwelling rodents that live in a wide variety of habitats throughout North America. The genus *Peromyscus* is widespread throughout North America. The subspecies *bairdii* is most common in the southwestern portions of Michigan. Deer mice are distinguished by large black beady eyes, pointed nose, and long whiskers. On average adult deer mice are 4.8 to 6.2 inches in length and weigh from 0.4 to 0.8 ounces (Baker 1983).

Habitat. Deer mice are found in a wide variety of habitats and are capable of adapting to many environments, including sandy beaches or lake shores, the edges of marshes, open woodlands, agricultural areas, and grassy fields and prairies (Baker 1983).

Density and Movement. The density of deer mice in any given area is a function of food supplies, habitat quality, and spatial needs of individual animals (Baker 1983). Deer mice populations also fluctuate seasonally. All wild deer mice populations experience an annual low in the early spring due to winter die-off and predation. This annual low is followed by a population explosion in the late spring (Howard 1949).

Deer mice are typically sedentary, and have home ranges from 0.5 to 2.5 acres (Baker 1983). Male deer mice have larger home ranges than females. Male home ranges encompass the home ranges of many females (Cranford 1984). The female's home range encompasses their foraging and nesting areas (Cranford 1984). Woodland deer mice, on average, have larger home ranges than prairie deer mice (Blair 1942).

Behavior. The behaviors of deer mice are categorized into three classes: (1) Motor Patterns, (2) Sensory Capacities, and (3) Learning Ability (King 1968). Motor patterns refer to the ability to swim, climb, gather food, and move around within its home range, while sensory capacities refer to the ability to detect light, odor, taste, temperature, gravity, and sound. Learning ability, which is generally unknown in wild populations, is measured by using mazes and rewards. In the winter months deer mice tend to congregate in one nest to conserve heat (Howard 1951). Within this group are three basic social units: (1) a mature male, (2) a mature female, and (3) juveniles.

Reproductive Activities. Deer mice reach sexual maturity 35 days after birth (EPA 1993). The breeding season extends from March through November. As the temperature increases in the spring, the reproduction rate of deer mice also increases. Each mouse is capable of producing two or three litters per breeding season (Johnson, et al. 1970). An average litter size includes four to six mice. Deer mice are also able to have consecutive litters without an estrus cycle (Baker 1983). Over a 1-year period the mortality rate of deer mice is 95 percent (Hansen, et al. 1974).

Food Habits. The average diet of deer mice is comprised of 60 percent terrestrial plants and 40 percent terrestrial invertebrates (CDM 1994). Food items may include insects, other invertebrates, seeds, fruits, flowers, and plants (Baker 1983). During periods of food shortages, deer mice will consume fecal pellets to sustain themselves (Baker 1983).

Predators. Deer mice serve as prey for many different animals including owls, hawks, snakes, coyotes, foxes, mink, and domestic cats.

Economic Importance. Deer mice serves a useful purpose in the environment as a principal food item for a wide variety of carnivores, including valuable fur-bearing animals such as mink (Baker 1983).

American Robin (*Turdus migratorius*)

The American robin is a medium-sized migratory bird found throughout the United States, Canada, Mexico, and Central America, and is distinguished by its black or dark grey/brown plumage with a dark orange breast.

Habitat. The American robin is found in a large variety of habitats. The preferred habitats are moist forests, swamps, open woodlands, orchards, parks, and suburban lawns. These types of habitat provide the robin with adequate cover, foraging areas, and water supplies (EPA 1993). The American robin utilizes trees or hedges for nesting sites.

Density and Movement. The density of the American robin is dependent on the type of cover available and the abundance of food supplies. Areas with very dense cover and adequate foraging areas yield very high densities of nesting robins, while areas with sparse cover do not support high densities of birds (EPA 1993). American robins are migratory, and spend the winter months in the southern United States, Mexico, and Central America. In the early spring they migrate to the northern United States and Canada. Male robins will return to the summer breeding ground just before the female robins arrive. This allows the males to establish breeding territories. It is very common for the same birds to return to the same breeding grounds year after year (EPA 1993). During the summer months, at the peak of the breeding season, the home range of the American robin is approximately 0.33 acres (CDM 1994). In the winter months when the robin is migrating southward the home range can be very large.

Reproductive Activity. The breeding season of the American robin begins in April and extends through July. As the males return from their wintering grounds they establish dominant breeding territories. Then as the females return, the males defend their territory from other males. Once a pair of robins mate, they remain united for the entire breeding season (Young 1951). The female prepares the nest from dried vegetation and mud. Only the female incubates the eggs, and incubation lasts for 10 to 14 days (EPA 1993). A female's first clutch usually produces three or four eggs. Later clutches produce fewer eggs. Once the eggs hatch, both the male and female participate in feeding the nestlings (Young 1955). After the nestlings are able to fly, the family forms a foraging flock and feeds together in areas of high food availability (EPA 1993). The longevity of the American robin is from 1.3 to 1.4 years (Farner 1949). Half of the adult birds survive from year to year.

Food Habits. The American Robin consumes a combination of fruits and invertebrates. During the breeding season, the diet may be composed of 90 percent invertebrates and 10 percent vegetation. However, the rest of the year the robins diet is usually comprised of 80 to 99 percent fruit and 1 to 20 percent invertebrates (Martin, et al. 1951). The robin's food choices for fruits include plums, dogwood, summac, hackberries, blackberries, cherries, greenbriers, and raspberries. The robin's food choices for invertebrates include beetles, caterpillars, moths, grasshoppers, spiders, millipedes, and earthworms. The American robin's daily intake of food must exceed their body weight to meet their metabolic needs (Karasov and Levey 1990). Robins have a digestive efficiency of 55 percent for fruits and 70 percent for invertebrates (Karasov and Levey 1990).

Predators. Predation is the leading cause of mortality for the American robin (EPA 1993).

Economic Importance. The American robin is not economically important, but is the state bird of Michigan. In addition, all songbirds are protected by Federal law.

Great Horned Owl (*Bubo virginianus*)

Great horned owls, found throughout the United States and Canada, are the largest and most powerful owl. They are recognized by brown spotted plumage, white throat feathers, and the distinguishing characteristic of "ears" that point upward, making these owls look as if they have horns growing from their heads.

Habitat. Great horned owls may be found in a wide variety of habitats ranging from wooded wilderness to urban parks. The most suitable habitats for great horned owls are woods, marshes, dunes, open deserts, and mountainous regions, which provide abundant hunting areas (Terres 1980).

Density and Movement. The home range of great horned owls is approximately 180 acres (CDM 1994).

Behavior. Great horned owls do not construct a nest but instead utilize old hawk, eagle, or crow nests. They prefer to use nests that are situated in the hollow of a tree or on the edge of a cliff (Terres 1980).

Reproductive Activity. Winter is the breeding season for great horned owls, and eggs are usually laid in January or February. Each female is capable of laying from one to six eggs. The incubation period ranges from 26 to 30 days, and only the female incubates eggs (Granlund, et al. 1994). After hatching, it takes 63 to 70 days before nestlings start to fly (Terres 1980). Great horned owls may live up to 29 years (Terres 1980).

Food Habits. Great horned owls are primarily nocturnal, and use old abandoned nests to roost and consume prey. Prey includes rabbits, squirrels, chipmunks, mink, weasels, skunks, woodchucks, opossum, snakes, cats, bats, and birds (Terres 1980). Of these, rabbits are the most preferred. Average dietary composition consists of approximately 20 percent invertebrates, 20 percent herps, 20 percent birds, and 40 percent mammals (CDM 1994).

Muskrat (*Ondatra zibethicus*)

Muskrats are semi-aquatic mammals found throughout North America. They are one of the largest rodents found in Michigan, and are recognized by robust size, long-flattened tail, and dense fur, which provides insulation and buoyancy.

Habitat. Muskrats are found in a large variety of aquatic environments, especially marshes with constant water levels and no flowing water (Johnson 1925). Less favorable habitats for muskrats are ponds, lakes, streams, canals, reservoirs, and swamps (Johnson 1925). The high productivity of marshes make them the most suitable environment for muskrats providing that the water level does not drop below 4 to 6 feet. Low water levels during the winter months can result in freeze out and high mortality among local muskrat communities (Baker 1983). Marshes are also most suitable for muskrats due to the diversity of the vegetation, which provides food resources and materials for den construction.

Density and Movement. The density of muskrat populations is affected by severe winters, flooding, drought, disease, and over-trapping (Errington 1939). On average, there are one to three muskrats per acre in habitats of low suitability. Under optimum conditions there may be as many as 35 muskrats per acre (Banfield 1974). Muskrats experience annual and semi-annual fluctuations in their populations due to periods of high mortality and high reproduction (Baker 1983). Muskrats typically have a very small home range averaging about 0.05 acres (CDM 1994). During the summer, muskrats rarely stray more than about 600 feet from their dens, and during winter muskrats forage within about 36 feet of their dens (Baker 1983). Muskrats are capable of moving up to 20 miles during their lifetime (Errington 1939). The primary reasons why muskrats may travel such distances are: (1) overcrowding; (2) dispersal of young;

(3) reproductive activity; (4) severe cold (winter freeze-out); (5) drought; and/or (6) food shortages (Baker 1983).

Behavior. Muskrats typically live in groups that consist of related individuals (Baker 1983). Muskrats are also territorial and use their scent glands to mark and maintain their territories. They usually have two different houses, one of which is a feeding house while the other is a dwelling and rearing den. These dens are typically constructed of vegetation and have multiple entrances and tunnels. Muskrats also dig burrows in the banks of rivers, streams, or lakes (Baker 1983). Muskrats may be active 24 hours a day. However, they usually forage in the late evening hours.

Reproductive Activities. The breeding season is from March to August. Females are capable of producing up to three litters per year, and each litter may have from 1 to 11 newborns. The average litter size is six. The normal gestation period is 25 to 35 days. Ten days after birth the young are capable of moving about the nest. At 14 to 16 days the newborns are able to swim. The young begin to consume green vegetation at 30 days. After about 200 days the young reach full independence (Baker 1983). The life expectancy for muskrats is 3 to 4 years. The mortality rate during the first year of life is 87 percent and increases to 98 percent during the second year (Baker 1983).

Food Habits. Muskrats are primarily herbivorous. They consume one third of their body weight in vegetation each day. During the summer months muskrats primarily consume emergent vegetation. However, in the winter months when emergent vegetation is scarce, muskrats will consume primarily submergent vegetation. The foods of choice for the muskrat include cattails, bulrush, arrowhead, water lily, corn, reed, and duckweed. When vegetation is limited, muskrats will consume crayfish, frogs, turtles, mollusks, and fish (Baker 1983).

Predators. Muskrats serve as prey to many different predators, including snapping turtles, bass, northern pike, pickerel, herons, bald eagles, owls, hawks, red fox, and mink (Errington 1939). Mink are the primary predators of muskrat (Errington 1943). Muskrats are also trapped for furs and meat.

Economic Importance. Muskrats are valued for their furs. They are the most important fur-bearing animal in Michigan (Ruhl and Baumgartner 1942). In 1981, muskrat pelts were selling for $7.30 per pelt (Baker 1983). Muskrats are also valued for their meat, and muskrat meat can be found in markets for up to $0.70 per pound (Dufresne 1982).

Mink (*Mustela vison*)

Mink are long slender mammals with short legs, thick soft under fur, and long glossy oily guard hairs. Most mink are black and have a characteristic white blotch under their chin. Mink are one of the most abundant and widespread carnivores in North America, found across North America except in extremely arid regions of the

southwest United States and Mexico and extreme northern regions of Canada (Baker 1983).

Habitat. Mink are semi-aquatic mammals, and may be found along streams, rivers, lakes, ponds, and marshes. They prefer habitat with irregular shorelines (Allen 1986). When away from water, mink prefer mixed shrubs, weeds, and grasses. The only type of habitat that mink will not use on a regular basis is heavily wooded uplands (Baker 1983).

Density and Movement. The density of mink populations depends on food and habitat availability. Mink populations are highest in large marshes that contain cattails and numerous muskrat dens (Errington 1943). Mink populations are also a function of hunting and trapping seasons. Prior to the trapping season, mink density ranges from 8 to 22 animals per square mile. After trapping season mink density ranges from three to four animals per square mile (Baker 1983). The movements of mink are influenced in part by intraspecific living space interaction (Baker 1983). The home range encompasses foraging areas, surrounding waterways, and dens (EPA 1993). A mink's home range depends on food availability, sex, and season (EPA 1993). The average home range for mink is about 20 acres (CDM 1994). However, along rivers or streams, male mink may travel up to 1.6 miles from their dens, while females travel up to 1.1 miles from their home site (Gerell 1970).

Behavior. Mink are generally nocturnal. They are also solitary except during the breeding season. Mink of the same sex usually avoid interactions with one another. Females are solely responsible for raising the young (Baker 1983). Mink usually establish their dens near water, and have a tendency to invade old beaver or muskrat dens (Baker 1983). Mink excavate ground burrows under root masses, beneath fallen logs, under brush piles, or in stream banks. Most tunnels are frequently inundated with water. Mink are also excellent swimmers, capable of diving to depths of 18 feet and swimming under water for distances up to 100 feet (Baker 1983).

Reproductive Activity. The breeding season begins in February and ends in April. Mink are only capable of producing one litter per year. The average litter size is four (EPA 1993). The mink's reproductive cycle is unique. After the egg is fertilized, the embryo goes dormant (Hannson 1947). The length of this dormancy depends on the amount of daylight during a 24-hour period (Holcomb 1963). Therefore, the total gestation period varies from 39 to 76 days. Only 30 to 32 days are needed for full development of the fetus (Enders 1952). The young are usually born in late April or May, and they are able to catch their own prey 42 to 56 days after birth. In August the young disperse because they no longer need maternal care (Baker 1983). The life expectancy of mink is 3 to 4 years (Baker 1983).

Food Habits. Mink are primarily carnivorous. However, they may consume some plant material from time to time (Baker 1983). The typical diet of the mink consists of approximately 30 percent fish, 20 percent herps, 20 percent birds, and 30 percent

mammals (CDM 1994). Mink are opportunistic in food selection (Iverson 1972). Primary terrestrial food items include shrews, moles, squirrels, mice, rats, bats, rabbits, voles, and muskrats. In the winter, the primary food choice of the mink is either muskrat or rabbit (Baker 1983).

Predators. Humans are the main predator of mink. Hunters and trappers account for the majority of mink mortality. Other natural predators include great horned owls, red fox, and domestic animals (Baker 1983).

Economic Importance. Mink are economically important because of the value of their furs. Mink are commercially raised for their pelts. This has helped alleviate hunting and trapping pressures on wild mink (Baker 1983). However, mink pelts are still highly valued. In 1969, mink pelts sold for $12 each. By 1980 they were selling for $30 each (Baker 1983). With such trends, it is expected that mink furs will continue to be valued. The fur market is subject to highs and lows that are influenced by fashion trends, excise taxes, imports, and synthetic furs (Baker 1983).

Appendix C
PCB Food Web Model
Kalamazoo River Superfund Site

Table C-1
Input Parameters for PCB Food Web Model
API/PC/KR

A	B	C	D	E	F	G	H	I	J	K	L	M	N
Receptor	**Total PCB Conc[1] (ABSAs 3-10) (ppm)**	**Method**	**Primary Exposure Media**	**Mean BCF/BAF**	**Home Range (hectares)**	**Site Foraging Frequency (SFF)[2]**	**Dietary Fraction (DF)**	**PCB Conc Diet (ww food, dw SED/FPSED, mg/kg)**	**Ingestion Rate IRww (kg/d) NIRww (kg/kg-d) IRdw (kg/kg-d)**	**Body Weight BW (kg)**	**DOSE (Sum (NIRww * PCB Conc food item * DF food item) + (NIRdw * U95 PCB Conc SED or FPSED * DF SED or FPSED) mg/kg-d)**	**LOAEC (conc) or LOAEL (dose) (exposure duration) Species –Effect – Reference**	**Criteria, Threshold, or NOAEC (conc) or NOAEL (dose)**
SW (range of U95) (mean of U95)	0.000016 - 0.000108 0.000043	Measured	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0.000014 mg PCB/L - EPA 1980 (protection of piscivorous wildlife, dietary exposure)
SED (range of U95) (mean of U95)	0.30-13.6 7.3	Measured	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	19.5 mg PCB/kg carbon - EPA 1988b (protection of piscivorous wildlife, dietary exposure)
FP SED/SS (range of U95) (mean of U95)	11.7 - 16.2 14.6	Measured (ABSAs 5, 7, 8)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	see SED see SS
SS (TBSA) (range of U95) (mean of U95)	0.23-30.2 16.9	Measured (TBSAs 3, 5, 10, 11)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	Species-specific LOAEC/(SUM (BAF*DF)SFF) - Boucher 1990
Algae (range of U95) (mean of U95)	0.016-0.108 0.043	Estimated (U95 PCB Conc SW *BCF)	SW	1,000 (Diatom, Keil, et al. 1971 in EPA 1980)	NA	NA	NA	NA	NA	NA	NA	0.0001 mg/L Algae (diatoms) -Delayed and Reduced Growth - Fisher and Wurster 1973 in EPA 1980	None
Aquatic Macrophyte (range of U95) (mean of U95)	0.05 – 2.5 1.3	Estimated (U95 PCB Conc SED*BCF)	FP SED/SS	0.182 (*Hydrilla*, mean of n=2 (Hopple and Foster 1996) assume 87% water content (EPA 1993),	NA	NA	NA	NA	NA	NA	NA	No Available Data	None
Terrestrial Macrophyte (range) (mean)	<0.04325 – 0.0692 0.023	Measured (n = 8)	FP SED/SS	0.037 (U95 of onsite garden plot data, 0.0008 (mean of tomato BAF (rep. fruit) (CDM 2000)	NA	NA	NA	NA	NA	NA	NA	No Available Data	None
Aquatic Invertebrate (Water Column) (range of U95) (mean of U95)	0.058-0.39 0.16	Estimated (U95 PCB Conc SW *BCF)	SW SED	3,650 - geo mean of mosquito and cladoceran (Mayer, et al. 1977 in EPA 1980)	NA	NA	NA	NA	NA	NA	NA	0.0008 mg/L Midge Larva - Nebeker and Puglisi 1974 in EPA 1980	None

Table C-1
Input Parameters for PCB Food Web Model
API/PC/KR

A	B	C	D	E	F	G	H	I	J	K	L	M	N
Receptor	**Total PCB Conc [1] (ABSAs 3-10) (ppm)**	**Method**	**Primary Exposure Media**	**Mean BCF/BAF**	**Home Range (hectares)**	**Site Foraging Frequency (SFF) [2]**	**Dietary Fraction (DF)**	**PCB Conc Diet (ww food, dw SED/FPSED, mg/kg)**	**Ingestion Rate IRww (kg/d) NIRww (kg/kg-d) IRdw (kg/kg-d)**	**Body Weight BW (kg)**	**DOSE (Sum (NIRww * PCB Conc food item * DF food item) + (NIRdw * U95 PCB Conc SED or FPSED * DF SED or FPSED) mg/kg-d)**	**LOAEC (conc) or LOAEL (dose) (exposure duration) Species –Effect – Reference**	**Criteria, Threshold, or NOAEC (conc) or NOAEL (dose)**
Aquatic Invertebrate (Benthic) (range of U95) (mean of U95) (crayfish)	 0.46-3.1 1.2 0.0323	Estimated (U95 PCB Conc SW *BCF)	SW SED	28,900 - geo mean of *Gammarus*, n = 3 (Nebeker and Puglisi 1974 in EPA 1980) 750 – crayfish (Meyer et al. 1977)	NA	NA	NA	NA	NA	NA	NA	0.0008 mg/L Nebeker and Puglisi 1974 in EPA 1980	None
Earthworm (range of max) (mean of max)	 0.025-3.2 1.5	Measured (Max PCB Conc, depurated carcass)	SS FP SED/SS	0.09 (depurated worms)	NA	NA	NA	NA	NA	NA	NA	No Available Data	None
Sucker (range of U95) (mean of U95)	 0.49-2.8 1.7	Measured (U95 PCB Conc, WB)	SW SED	83,000 (calculated SW)	NA	NA	NA	NA	NA	NA	NA	0.0002 mg/L Fathead Minnow - Defoe, et al. 1978 in EPA 1980	None
Carp (range of U95) (mean of U95)	 9.0-19.1 12.1	Measured (U95 PCB Conc, WB)	SW SED	583,000 (calculated SW)	NA	NA	NA	NA	NA	NA	NA	0.0002 mg/L Fathead Minnow - Defoe, et al. 1978 in EPA 1980	None
SM Bass (range of U95) (mean of U95)	 1.8-8.7 5.4	Measured (U95 PCB Conc, WB)	SW Prey	249,000 (calculated SW)	NA	NA	NA	NA	NA	NA	NA	0.0004 mg/L Largemouth Bass - Acute LC_{50} (2.3 µg/L)/geo mean ACR for FW Fish (6.4) - Birge, et al. 1979 in EPA 1980	None
Fish (mean of mean of U95, 3 species)	6.4	Measured (U95 PCB Conc, WB)	SW Prey	305,000 (mean, 3 species)									
Muskrat (range of max) (mean of max)	 0.21-2.9 1.2	Measured (Max PCB Conc ww, WB)	SED FP SED/SS Vegetation	0.08 Whole body (mean of max PCB conc / mean of U95 FPSED)	0.13 EPA 1993	1.0	Semi-Aquatic Plants 1.0 (based on mean *Hydrilla* BAF, 0.182) SED 0.094 (raccoon) EPA 1993	1.3 7.3	0.42 0.3 0.037 EPA 1993	1.4 EPA 1993	0.417	100 mg/kg FW diet (240 days) - 5 mg/kg-d per Burse, et al. 1974 in EPA 1980) - Rat – Substantial Weight Loss - Kimbroughj, et al. 1972 in EPA 1980	33 mg/kg FW diet - 1.7 mg/kg-d (based on LOAEC/3 = NOAEC)

Table C-1
Input Parameters for PCB Food Web Model
API/PC/KR

A	B	C	D	E	F	G	H	I	J	K	L	M	N
Receptor	**Total PCB Conc [1] (ABSAs 3-10) (ppm)**	**Method**	**Primary Exposure Media**	**Mean BCF/BAF**	**Home Range (hectares)**	**Site Foraging Frequency (SFF) [2]**	**Dietary Fraction (DF)**	**PCB Conc Diet (ww food, dw SED/FPSED, mg/kg)**	**Ingestion Rate IRww (kg/d) NIRww (kg/kg-d) IRdw (kg/kg-d)**	**Body Weight BW (kg)**	**DOSE (Sum (NIRww * PCB Conc food item * DF food item) + (NIRdw * U95 PCB Conc SED or FPSED * DF SED or FPSED) mg/kg-d)**	**LOAEC (conc) or LOAEL (dose) (exposure duration) Species –Effect – Reference**	**Criteria, Threshold, or NOAEC (conc) or NOAEL (dose)**
Mink (range of max) (mean of max)	2.6-5.6 3.8	Measured (Max PCB Conc ww, WB)	Prey	Not Applicable because of unknown contribution from multiple exposure pathways	14.1 EPA 1993	1.0	Fish 0.850 Mammal 0.10 Bird 0.05 SED 0.0 (est. from raccoon) Based diet modified from Michigan river (year-round) of Alexander 1977 in EPA 1993). Mammal = 5% mouse and 5% muskrat	6.4 0.79 76.64	0.218 0.18 Heaton, et al. 1995 (LOAEL treatment) 0.08 EPA 1993	1.2 Calculated from Heaton, et al. 1995 (LOAEL treatment)	1.70	0.60 mg/kg FW diet (low effect, EC_{25}, see text) - (0.11 mg/kg-d) Mink - Kit body wt. and no. of live kits/mated female, multi-generational, Aroclor 1254, multiple studies – see Appendix D	0.5 mg/kg FW diet (EC_{10}, see text) - (0.091 mg/kg-d) - Mink – estimated from low effect (EC_{25}), see Appendix D
Mouse (range of max) (mean of max)	0.28-0.45 0.37	Measured (Max PCB Conc, WB)	Vegetation and Prey	0.025 Whole body (mean of max PCB conc / mean of U95 FPSED)	0.06 EPA 1993	1.0	Terr. plants 0.44 Terr. inverts 0.56 FPSED 0.02 EPA 1993	0.54 (all tissues) 1.314 14.6	0.0055 0.262 0.0035 EPA 1993	0.021 EPA 1993	0.311	10 mg/kg FW diet (540 days) - 1.35 mg/kg-d, NIR = 0.135 kg/kg-d - White-footed Mouse - Reduced no. of young - Linzey 1987 and 1988)	3.3 mg/kg FW diet - 0.45 mg/kg-d (based on LOAEC/3 = NOAEC)
Robin (mean)	76.64	Estimated for whole body carcass (see Appendix C-2-A for equations)	Vegetation and Invert Prey	93 - mean diet to carcass BAF for alewife to gull (Braune and Norstrom 1989)	0.48 EPA 1993	1.0	Terr. Plants 0.49 Terr. Iinverts 0.51 FPSED 0.1 EPA 1993	0.012 (fruit only) 1.314 14.6	0.069 0.896 0.016 EPA 1993	0.077 EPA 1993	0.904	0.5 mg/kg-d, ED_{25} for egg hatchability in chicken, Aroclor 1248 (Scott 1977 and Lillie 1975, see Appendix D)	0.4 mg/kg-d, ED_{10} for egg hatchability in chicken, Aroclor 1248 (Lillie et al. 1974 and Cecil et al. 1974, see Appendix D)
GH Owl	Not Determined	NA	Prey	Not Determined	329 Peterson 1979 in Johnsgard 1988	1.0	Birds 0.47 Mammals 0.53 FPSED 0.02 Craighead and Craighead 1956 (4% aq.inverts dist. equally to birds and mammals) Mammals based on average of mouse and muskrat	76.64 0.79 14.6	0.084 0.056 Craighead and Craighead 1956 0.073 EPA 1993	1.505 Mean of male and female geo mean BW, Craighead and Craighead 1956	2.06	estimated 9.0 mg/kg FW diet, 1.2 mg/kg-d (estimated by NOAEL*3)	3.0 mg/kg FW diet (0.41 mg/kg-d, NIR = 0.138 kg/kg-d, from Sample, et al. 1996) - Screech Owl – McLane and Hughes 1980

Table C-1
Input Parameters for PCB Food Web Model
API/PC/KR

A	B	C	D	E	F	G	H	I	J	K	L	M	N
Receptor	**Total PCB Conc [1] (ABSAs 3-10) (ppm)**	**Method**	**Primary Exposure Media**	**Mean BCF/BAF**	**Home Range (hectares)**	**Site Foraging Frequency (SFF) [2]**	**Dietary Fraction (DF)**	**PCB Conc Diet (ww food, dw SED/FPSED, mg/kg)**	**Ingestion Rate IRww (kg/d) NIRww (kg/kg-d) IRdw (kg/kg-d)**	**Body Weight BW (kg)**	**DOSE (Sum (NIRww * PCB Conc food item * DF food item) + (NIRdw * U95 PCB Conc SED or FPSED * DF SED or FPSED) mg/kg-d)**	**LOAEC (conc) or LOAEL (dose) (exposure duration) Species –Effect – Reference**	**Criteria, Threshold, or NOAEC (conc) or NOAEL (dose)**
Red Fox	Not Determined	NA	Prey	Not Determined	708 EPA 1993	1.0	Terr. Plants 0.11 Terr. Inverts 0.04 Herps 0.08 Birds 0.19 Mammals 0.58 FPSED 0.028 EPA 1993 Mammals = mean of mouse and muskrat Herps (see footnote)	0.54 (all tissues) 1.314 3.36 76.64 0.79 14.6	0.752 0.16 0.245 EPA 1993	4.7 EPA 1993	2.48	5 mg/kg-d - dog - (est. 31 mg/kg diet) - reproductive effects - Earl, et al. 1974	1 mg/kg-d - dog - (est. diet = 6 mg/kg) - no reproductive effects - Earl, et al. 1974
Bald Eagle	Not Determined	NA	Prey	Not Determined	2,500 EPA1993	1.0	Fish 0.77 Birds 0.17 Mammals 0.06 SED 0.0 EPA 1993 Mammals = mean of muskrat and mouse	6.4 76.64 0.79	0.45 0.12 0.145 EPA 1993	3.75 EPA 1993	2.16	0.5 mg/kg-d, ED_{25} for egg hatchability in chicken, Aroclor 1248 (Scott 1977 and Lillie 1975, see Appendix D)	0.4 mg/kg-d, ED_{10} for egg hatchability in chicken, Aroclor 1248 (Lillie et al. 1974 and Cecil et al. 1974, see Appendix D)

Acronyms, Footnotes, and Assumptions

ACRONYMS

WB	Whole Body
BCF/BAF	Whole Body Concentration Biota / Concentration Exposure Medium
LOAEC(L)	Lowest observed adverse effect concentration (level)
NOAEC(L)	No observed adverse effect concentration (level)
SW	Surface Water
SED	Streambed Sediment
FP SED/SS	Floodplain Sediment/Surface Soil
SS	Surface Soil from TBSAs
FW	Fresh Weight
ACR	Acute to Chronic Ratio
*	Value based on half the analytical detection limit (< detection limit value)
NIR	Normalized ingestion rate (IR/BW) – from EPA 1993 unless indicated otherwise
ED_{25} or EC_{25}	Effective dose (mg/kg-d) or concentration (mg/kg), low effect is 0.75 of control response a toxicological endpoint (EC_{25}, which represents a 25% decrease in response)
ED_{10} or EC_{10}	Effective dose (mg/kg-d) or concentration (mg/kg), no effect is equal to 0.90 of the control response (EC_{10}, which represents a 10% decrease in response)

FOOTNOTES

1. Estimated PCB concentration for Biota = (Conc SW * BCF) or (Conc SED or SS * BAF).
2. SFF = Site Area 518,000 hectares / Home Range

ASSUMPTIONS (see footnotes in Appendix C-2 for additional assumptions)

- Earthworms are conservative and appropriate representatives for terrestrial invertebrate prey (depurated worm data used with soil intake to account for whole body burden)
- Consumers of fish ingest equal amounts of forage, rough, and game fish (represented by sucker, carp, smallmouth bass)
- Whole body PCB concentrations for HERPS (reptiles and amphibians) consumed as prey based on mean SED-to-Frog BSAF (0.23) from Unnamed Tributary, NY (CDM 2001)
- Birds most representative of species consumed by predators are omnivorous passerine birds, represented by American robin
- Bird PCB Conc (whole body) based on diet-to-carcass BAF of 93, from Braune and Norstrom 1989 (alewife to gull BAF)

Table C-2-A. Terrestrial Risk Estimates and PRG Derivation

Floodplain Sediment/Soil-based Exposures

Receptor	BW	IRww	NIRww	IRdw	NIRdw	Soil PCB	DFsoil	Worm BAF	Worm PCB
	kg	kg/d	kg/kgbw-d	kg/d	kg/kgbw-d	mg/kg	fraction	ww/dw	mg/kg
Robin	0.077	0.069	0.89	0.0160	0.2074	14.6	0.1	0.09	1.314
Mouse	0.021	0.0055	0.27	0.0035	0.1647	14.6	0.02	0.09	1.314
Red Fox	4.7	0.752	0.16	0.2452	0.0522	14.6	0.028	0.09	1.314
GH Owl	1.505	0.084	0.056	0.0733	0.0487	14.6	0.02	-	-

DFworm	Veg BAF	Veg PCB	DFveg	DFHerp	HerpBAF	HerpPCB	DFBird	BirdBAF	BirdPCB
fraction	ww/dw	mg/kg	fraction	fraction	ww/dw	mg/kg	fraction	ww/dw	mg/kg
0.51	0.0008	0.01168	0.49	0	-	-	0	-	-
0.56	0.037	0.5402	0.44	0	-	-	0	-	-
0.04	0.0008	0.01168	0.11	0.08	0.23	3.358	0.19	5.25	76.7
0	-	-	0	0	-	-	0.47	5.25	76.7

DFMammal	MammalBAF	MammalPCB	Dose	LOAEL	**LOAEL HQ**	NOAEL	**NOAEL HQ**	**LOAEL PRG**	**NOAEL PRG**
fraction	ww/dw	mg/kg	mg/kg-d	mg/kg-d	**ratio**	mg/kg-d	**ratio**	**mg/kg**	**mg/kg**
0	-	-	0.9044	0.5	**1.81**	0.4	**2.26**	**8.07**	**6.46**
0	-	-	0.3109	1.35	**0.23**	0.45	**0.69**	**63.39**	**21.13**
0.58	0.0530	0.79	2.4764	5	**0.50**	1	**2.48**	**29.50**	**5.90**
0.53	0.0530	0.79	2.0551	1.2	**1.71**	0.41	**5.01**	**8.53**	**2.91**

Soil PCB = mean of U95 values for FPSED measured in ABSAs 5,7, and 8

DFsoil = from EPA 1993 or estimated

VegBAF = U95 BAF, all plant species and tissues (0.037) or fruit BAF, from soil-to-tomato BAF (0.0008) (CDM 2000)

Robin NIRww - Skorupa and Hothem 1985 in USEPA 1993, 11.5 % animal prey, remainder fruit and vegetation

Mouse NIRww - mean of 6 adult values in USEPA 1993

IRdw (passerine) = (((BW * 1000)^0.85) * 0.398)/1000 (USEPA 1993 equation 3-4)

IRdw (rodents) = (((BW * 1000)^0.564) * 0.621)/1000 (USEPA 1993 equation 3-8)

IRdw (fox-mammal) = (((BW * 1000)^0.822) * 0.235)/1000 (USEPA 1993 equation 3-7)

IRdw (non-passerine) = (((BW * 1000)^0.751) * 0.301)/1000 (USEPA 1993 equation 3-5)

(Receptor) PCB = Soil PCB * (Receptor) BAF

HerpBAF = mean of green frog and leopard frog sediment-to-whole body PCB conc, Un-named Tributary, New York (CDM 2000)

MammalBAF = mean of muskrat and mouse BAF, where BAF = mean of max whole body PCB conc / mean of U95 FPSED/soil PCB conc

BirdBAF = est.bird whole body (WB) PCB conc (carcass) / soil PCB conc

Robin Dietary PCB Conc (mg/kg) = dose (mg/kg_{bw}-d) / (NIR_{ww} (mg $food_{ww}$ /kg_{bw}-d) + NIR_{dw} (mg $soil_{dw}$/kg_{bw}-d))

Robin Dietary PCB Conc (0.824 mg/kg) = 0.9044 mg/kgbw-d / (0.89 mg foodww /kgbw-d + 0.2074 mg soildw/kgbw-d)

BirdPCB = Robin dietary PCB Conc (mg/kg) * diet to carcass BAF for birds (93, alewife to gull, Braune and Norstrom 1989)

BirdPCB (76.7 mg PCB/kg bird) = Robin dietary PCB Conc (0.824 mg/kg) * 93

Soil-to-bird BAF = BirdPCB / Soil PCB Conc = 76.7 mg PCB/kg bird / 14.6 mg PCB/kg soil

Dose = SUM (NIRww * PCBPrey1...x * DFPrey1...x) + (NIRdw * PCBSoil * DFSoil)

Dose (Example = Robin) = (NIRww * Worm PCB * DFworm) + (NIRww * Veg PCB * DFveg) + (NIRdw * Soil PCB * DFsoil)

HQ = Dose / LOAEL or NOAEL

PRG = LOAEL or NOAEL / SUM ((NIRww * BAFPrey1...x * DFPrey1...x) + (NIRdw * DFSED)) = LOAEL or NOAEL / DOSE * SED PCB CONC

PRG (Example = Robin) = LOAEL or NOAEL / ((NIRww * Worm BAF * DFworm) + (NIRww * Veg BAF * DFveg) + (NIRdw * DFsoil))

Table C-2-. .mi-Aquatic Risk Estimates and PRG Derivation

Instream Sediment-based Exposures

Receptor	BW	IRww	NIRww	IRdw	NIRdw	SED PCB	DFSED	FishBAF	Fish PCB	DFFish
	kg	kg/d	kg/kgbw-d	kg/d	kg/kgbw-d	mg/kg	fraction	ww/dw	mg/kg	fraction
Muskrat	1.4	0.42	0.300	0.037	0.026	7.3	0.094	-	-	0
Bald Eagle	*3.75*	*0.45*	*0.120*	*0.145*	*0.039*	*7.3*	*0*	*0.877*	*6.4*	*0.77*
Mink	1.2	0.218	0.182	0.080	0.067	7.3	0	0.877	6.4	0.85

Veg BAF	Veg PCB	DFveg	DFBird	BirdBAF	BirdPCB	DFMammal	MammalBAF	MammalPCB	Dose	LOAEL
ww/dw	mg/kg	fraction	fraction	ww/dw	mg/kg	fraction	ww/dw	mg/kg	mg/kg-d	mg/kg-d
0.182	1.3286	1	0	-	-	0	-	-	0.4167	5
-	-	-	0.17	10.5	76.64	0.06	0.108	0.79	2.1606	0.5
-	-	0	0.05	10.5	76.64	0.10	0.108	0.79	1.6988	0.11

LOAEL HQ	NOAEL	NOAEL HQ	LOAEL PRG	NOAEL PRG
ratio	mg/kg-d	**ratio**	**mg/kg**	**mg/kg**
0.08	1.7	**0.25**	**87.60**	**29.78**
4.32	0.4	**5.40**	**1.69**	**1.35**
15.44	0.055	**30.89**	**-**	**-**

SED PCB (instream) = mean of U95 values for instream SED (7.3) measured in ABSAs 3-10

SW PCB (instream) = mean of U95 values (0.000043 mg/L) measured in ABSAs 3-10

DFSED = from EPA 1993 or estimated

VegBAF (muskrat) = mean BAF, *Hydrilla*, (from dw/dw BAF of 1.5, fraction moisture = 0.87, Hopple and Foster 1996)

Mouse NIRww - mean of 6 adult values in USEPA 1993

IRdw (muskrat/mouse - rodents) = (((BW * 1000)^0.564) * 0.621)/1000 (USEPA 1993 equation 3-8)

IRdw (mink-mammal) = (((BW * 1000)^0.822) * 0.235)/1000 (USEPA 1993 equation 3-7)

IRdw (bald eagle - non-passerine) = (((BW * 1000)^0.751) * 0.301)/1000 (USEPA 1993 equation 3-5)

(Receptor) PCB = Soil PCB * (Receptor) BAF

MammalBAF = mean of mean MuskratBAFand MouseBAF, where BAF = max whole body PCB conc / mean of U95 SED PCB conc

BirdBAF = est.bird whole body (WB) PCB conc (carcass) / soil PCB conc

BirdPCB = Robin dietary PCB Conc (mg/kg) * diet to carcass BAF for birds (93, alewife to gull, Braune and Norstrom 1989)

Robin Dietary PCB Conc (mg/kg) = dose (mg/kg_{bw}-d) / (NIR_{ww} (mg $food_{ww}$ /kg_{bw}-d) + NIR_{dw} (mg $soil_{dw}$/kg_{bw}-d))

Robin Dietary PCB Conc (0.824 mg/kg) = 0.9044 mg/kgbw-d / (0.89 mg foodww /kgbw-d + 0.2074 mg soildw/kgbw-d)

Dose = SUM (NIRww * PCBPrey1...x * DFPrey1...x) + (NIRdw * PCBSED * DFSED)

HQ = Dose / LOAEL or NOAEL

PRG = LOAEL or NOAEL / SUM ((NIRww * BAFPrey1...x * DFPrey1...x) + (NIRdw * DFSED)) = LOAEL or NOAEL / DOSE * SED PCB CONC

Appendix D
EPA Region 5 Recommended Avian and Mink PCB Toxicity Reference Values

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 5

DATE: March 6, 2003

SUBJECT: Toxicity Reference Values (TRVs) for Mammals and Birds Based on Selected Aroclors

FROM: James Chapman, Ph.D., Ecologist

TO: Shari Kolak, RPM

1 Summary

Toxicity reference values (TRVs) are developed for polychlorinated biphenyl (PCB) mixtures based on studies of controlled exposures to commercial Aroclor products for sensitive mammal (mink) and bird (chicken) species. The TRVs are interpolated from dose-response plots of Aroclor exposure and reproductive or growth endpoints, with data collated from multiple studies. The interpolated low-effect level is the dose that results in a 25 % decrease in an endpoint response compared to that of the control group, and the interpolated no-effect level a 10 % decrease.

The TRVs are recommended for mink or conservative application to bird species that lack species-specific PCB toxicity data. Since the TRVs are derived from studies of sensitive species to PCBs, use of uncertainty factors for extrapolation to other species is not recommended. The TRVs are given as bodyweight normalized doses (mg PCB per kilogram bodyweight per day) for ingestion by birds to facilitate application to bird species of different sizes. Dietary TRVs (mg PCB per kg food) on a wet weight (ww) basis are given for mink since interspecific extrapolation is not necessary to assess risk to wild mink. The TRVs for bird eggs are given as the concentration in whole eggs on a wet weight basis (mg PCB per kilogram egg).

The TRVs are summarized in Table 1. See the text for details.

Table 1. Interpolated PCB Toxicity Reference Values (TRVs) Based on Controlled Exposures of Mink and Chicken to Commercial PCB Products.

Commercial PCB Product (Aroclor)	Mink Diet [a]		Bird Dose		Bird Egg	
	mg/kg ww		mg/kg_{BW}-d		mg/kg whole egg ww	
	no effect	low effect	no effect	low effect	no effect	low effect
1242	1.3	1.4	0.1 - 0.5 [b]	0.4 - 0.8 [b]	1.0	1.5
1248	see 1254 [c]	see 1254 [c]	0.4	0.5	0.7	1.3
1254	0.5	0.6	0.6	1.2	9	12

Notes for Table 1:

a) Mink TRVs are adjusted for continuous exposure over multiple years or generations at the same site (see text).

b) Two response patterns are exhibited in the published studies, which are separately assessed (see text).

c) A1248 has not been tested in mink. The mink A1254 TRVs are applied because A1248 is as potent as A1254 in an *in vitro* mammalian bioassay (Tillitt, et al. 1992).

The TRVs for mink are adjusted for continuous exposure through two breeding seasons or generations because mink feeding studies with one of the European commercial PCB formulations (Clophen A50) and, independently, with field-contaminated fish have shown pronounced increases in toxicity compared to exposure over a single breeding season. The A1254 TRV is based on the number of live kits per mated female and kit bodyweight at birth. Although kit survival following birth might be a more sensitive endpoint compared to live kit production or kit bodyweight at birth (see Clophen A50 below), the data are insufficient for determining kit survival TRVs for A1254, other than to state that the low-effect dietary concentration is less than 1 mg/kg for a single season of exposure. Surprisingly, no mink feeding studies were located for A1248. However, A1248 is as potent as A1254 in an *in vitro* [1] mammalian bioassay (Tillitt, et al. 1992), so the A1254-based TRVs are applied to A1248. The TRVs for A1242 are based on live kit production. Data are insufficient for other endpoints for A1242.

For comparison, the mink dietary TRVs for Clophen A50, one of the European commercial PCB products, over 2 seasons exposure are 1.1 to 1.3 mg/kg for live kit production (no effect to low effect), 2.3 mg/kg for kit bodyweight (low effect), and less than 0.8 mg/kg for kit survival (low effect). Data are insufficient to determine no effect TRVs for the latter two endpoints, other than to state that the no effect TRVs are greater than the control dietary concentration of 0.01 mg/kg.

All of the TRVs from chicken studies are based on hatchability, the most frequently reported endpoint of PCB studies with chicken. Chick bodyweight is a less sensitive endpoint in the few cases for which comparisons can be made with hatchability. Chick survival appears to be a more sensitive endpoint than hatchability in the sole available comparison (low effect TRV of 0.3 mg/kg_{BW}-d for A1248), but is less reliable compared to the A1248 hatchability TRV because the survival TRV is based on sparser data requiring interpolation over a much wider dose gradient.

A1242 exhibits two dose-response patterns in chicken studies--one with TRVs somewhat lower than A1248, and another approaching the A1254 TRVs. The two A1242 patterns may be due to differences in A1242 batches, chickens, feed, or experimental designs. Instead of choosing between the two patterns, both sets of A1242 TRVs are shown.

TRVs calculated from exposure to commercial PCB products may underestimate the toxicity of PCBs in the field because of environmental weathering and selective retention in biota that alter the proportions of dioxin-like congeners compared to the source product. Concurrent exposures to other chemicals in the field that contribute to dioxin-like toxicity reduces the margin of exposure to PCBs that can be tolerated without exhibiting adverse effects. Use of the lower of the TRVs given above is recommended to account for increased toxicity due to these effects (A1254 TRVs for mink and A1248 TRVs for birds). The TRVs are probably not applicable to sites

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[1] The literal meaning of *in vitro* is "in glass", which refers to experiments performed outside of a living body, for example, in test tubes , petri dishes., or other laboratory apparatus. In this case, the bioassay measures the response of cultured cells to PCBs and other chemicals with dioxin-like toxicity.

with source PCBs different from the Aroclors assessed in this effort, for example, A1260, which is less toxic than A1242, A1248, or A1254 in an *in vitro* mammalian bioassay (Tillitt, et al. 1992).

The methodology used for deriving the TRVs was internally peer-reviewed by USEPA scientists. The peer review charge included review of the data normalization procedure for combining the results of different studies, effect size selection, linear interpolation method (including the following modifications–restriction of interpolation to the linear portion of the data plots, use of log-linear interpolation, no adjustment for violations of monotonicity for hormetic responses, and lack of confidence interval estimation), and adjustment of mink TRVs for increased toxicity associated with continuous exposure over 2 breeding seasons or 2 generations. The peer reviewers also made additional comments regarding meta-analysis, uncertainty associated with Aroclor approaches, TEQ as an alternative approach, and editorial comments. The peer review comments and responses are summarized in Responses to Peer Review Comments, Wildlife PCB Toxicity Reference Values. March 6, 2003. USEPA Region 5 Superfund Division, Chicago. The present version of this work product has been revised in accordance with these comments and responses.

2 Acronyms

A1242, A1248, A1254, A1260 - different Aroclors (commercial PCB products produced in America)
A50 - one of the Clophen commercial PCB products produced in Europe
AhR - aryl hydrocarbon receptor (cellular protein that binds with dioxin-like chemicals in the initial step of a cascade of interactions leading to expression of toxic effects)
AWQC - federal ambient water quality criteria
BMF - biomagnification factor (= concentration in animal / concentration in food or environmental media)
BW - bodyweight
Ca^{2+} - calcium ion
d - day
EC_x - effective concentration resulting in a treatment response x % less than the control response
ED_x - effective dose resulting in a treatment response x % less than the control response
fw - fresh weight (weight including moisture content at the time of measuring)
g - gram
GLI - Great Lakes Initiative
H4IIE - designates a particular cultured rat cell line used in an *in vitro* bioassay for dioxin-like activity
I-TEF - international toxic equivalency factors
kg - kilogram (1000 g)
LD_{50} - lethal dose to 50 % of the exposed population
LOAEL - lowest observed adverse effect level (lowest tested dose that caused a statistically discernible response compared to the control group)
LOEC - lowest observed effect concentration (lowest tested concentration that caused a statistically discernible response compared to the control group)
lw - lipid weight (concentration on a lipid (fat) basis, e.g., mg PCB per kg fat)
mg - milligram (0.001 g)
pg - picogram (one trillionth gram)

NOAEL - no observed adverse effect level (highest tested dose that did not cause a statistically discernible response compared to the control group)
NOEC - no observed effect concentration (highest tested concentration that did not cause a statistically discernible response compared to the control group)
OECD - Organization for Economic Co-operation and Development (Europe)
PCB - polychlorinated biphenyl
ppb - parts per billion (equal to 0.001 ppm)
ppm - parts per million (equal to mg/kg)
ppt - parts per trillion (equal to 0.000001 ppm or pg/g)
PRG - preliminary remedial goal
REP - relative potency (the fractional response of a dioxin-like chemical compared to 2,3,7,8-TCDD in a particular test or approach)
RR - relative response (normalized treatment response = treatment response / control response of the same study)
TCDD - tetrachlorodibenzo-*p*-dioxin
TEF - toxic equivalency factor (the consensus fractional response of a dioxin-like chemical compared to 2,3,7,8-TCDD based on variety of research approaches and results)
TEQ - toxic equivalent concentration (the concentration of 2,3,7,8-TCDD that is expected to equal the potency of a mixture of dioxin-like chemicals, calculated by multiplying the concentrations of each dioxin-like chemical by their respective TEFs, or measured directly by an *in vitro* bioassay)
TRV - toxicity reference value (the concentration or dose of a chemical used to assess risk–no effect TRVs are not expected to cause adverse effects, and low effect TRVs are the levels at which adverse effects first become apparent)
USEPA - United States Environmental Protection Agency
WHO - World Health Organization
wk - week
ww - wet weight (weight including the normal moisture content)

3 Background

One of the issues raised concerning the Baseline Ecological Risk Assessment for the Allied Paper, Inc./Portage Creek/Kalamazoo River Superfund site concerns the appropriate PCB TRVs for wildlife. Inclusion of studies performed with field-contaminated prey from Saginaw Bay, MI, in the derivation of PCB TRVs for mink and birds was criticized because the observed effects may have been confounded by contaminants other than PCBs.[2] One of the alternatives suggested in written and oral comments was to use the TRVs developed for the Great Lakes Initiative (GLI) water quality criteria (WQC) for wildlife (USEPA 1995a). This was looked into, but a difficulty occurred in attempting to apply the TRVs used by the GLI to Superfund purposes.

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[2] Whether PCBs appear to be major or minor contributors to the observed toxicity in the Saginaw Bay studies depends on which set of toxic equivalency factors (TEFs) are used to convert the measured contaminant data to dioxin toxic equivalents (TEQs). PCBs are the major contributor according to the International TEF (I-TEF) scheme, but are minor contributors according to the TEFs [better termed relative potencies (REPs) because they are based on a single experimental approach] reported for the H4IIE bioassay (an *in vitro* assay performed with a rat hepatoma cell line) (Tillitt, et al. 1996; Geisey, et al. 1997). The I-TEF scheme has been replaced by World Health Organization TEFs (WHO-TEFs) (Van den Berg, et al. 1998), but the new scheme does not significantly alter the outcome.

The GLI WQC are based solely on the no observed adverse effect level (NOAEL), but the guidance for Superfund ecological risk assessments recommends evaluation of risks and calculation of site-specific preliminary remedial goals (PRGs) for both the NOAEL and lowest observed adverse effect level (LOAEL) (USEPA 1997). At first this did not appear to be problematic since the GLI reported both the available NOAELs and LOAELs of the studies reviewed for calculating the WQC. The issue in applying these TRVs for Superfund use is that the GLI did not evaluate the appropriateness of the LOAEL data for regulating LOAEL-based risks. The mink assessment represents an extreme example. The LOAEL chosen by the GLI for mink reproduction resulted in complete kit mortality--only 2 of 7 exposed females whelped (gave birth), producing only 1 live but underweight kit that died before reaching 4 weeks age (Aulerich and Ringer 1977). Since a NOAEL was not identified in this study, the LOAEL was converted to a NOAEL by dividing by an uncertainty factor of 10 (USEPA1995a). The calculated NOAEL was equivalent to the NOAEL of a mink feeding study performed with field-contaminated fish, which indicated that the conversion provided an adequate margin of safety for ensuring no adverse effects (USEPA 1995a), and therefore satisfied the objectives of the GLI WQC. However, at the LOAEL, zero successful reproduction is not an adequate representation of a lowest adverse effect level, instead it represents the maximum possible adverse effect on reproduction, and therefore does not satisfy the Superfund objectives of characterizing the risk range between no effects and the level at which adverse effects become detectable.

The problem in applying the LOAEL identified by the GLI is inherent in the methodology of the NOAEL/LOAEL approach, which has been criticized in numerous publications (for examples see Crump 1984; Suter 1996; OECD 1998; Crane and Newman 2000). The main limitations of the NOAEL/LOAEL approach are that the values are significantly affected by factors other than toxicity, and the available dose-response information is not utilized. NOAELs and LOAELs are statistically defined–a LOAEL is the lowest tested dose that exhibited a statistically discernible response compared to the control response, and a NOAEL is the highest tested dose that did not show a statistically discernible response from that of the control. An obvious issue is that, by this approach, NOAELs and LOAELs are restricted to the particular doses tested. This is the source of the problem with the GLI selected LOAEL for mink–the lowest treatment dose tested resulted in 0 % successful reproduction, so by default, it was identified as the "lowest" adverse effect level, even though it is obvious that lower doses, if tested, would also show adverse reproductive effects. Also, determination of statistical significance depends not only on toxicity, but also on the study design (the particular dose levels tested and number of replicates per dose) and the particular statistical procedure chosen to compare the treatment and control responses, all of which affects the statistical power of the comparison. An unfortunate result is that "poor" studies with low statistical power are rewarded from the perspective of potentially liable parties because they result in higher (less protective) NOAELs and LOAELs compared with more rigorous and expensive studies with higher statistical power. Similar considerations pertain to the number of dose levels tested–fewer doses are less expensive, but may "miss" appropriate effect levels by wide margins. Another way of considering these issues is that, because of the widely ranging statistical power associated with toxicity tests, and differences in the doses selected for study, the level of adversity associated with statistically determined TRVs varies uncontrollably. For example, in a ring test of aquatic toxicity laboratories, the mean decrease in response associated with the statistically identified no observed effect concentration (NOEC) was about 10 % across laboratories, but ranged as high as 37 % in individual cases (cited in Crane and Newman 2000). In another evaluation, statistically determined no effect concentrations could be associated with as much as 50 % decreases in responses compared to controls depending on the data and the choice of statistical method, leading

the investigators to conclude that "the NOEC is rarely if ever an indicator of no effect" (Crane and Newman 2000). The same issues apply to LOAEL determinations. Another limitation of the NOAEL/LOAEL approach is that it does not make use of the available dose-response information. See Crump (1984) for an example showing how statistically determined effect levels can give misleading results for chemicals with markedly different dose-response patterns.

An alternative is to use the data from toxicological studies to develop dose-response relationships, and to use the relationships to determine the no-effect and low-effect doses that correspond to selected effect levels. This frees the analysis from the specific doses used in a study (a TRV can now be interpolated between the tested doses), and from the non-conservative bias of tests with inadequate statistical power. In this approach, the effect size is selected first (effect size is the percentage decrease in performance compared to control), for example, that the low effect level should be a 20 % decrease in treatment response compared to the control response. Then the dose corresponding to the selected effect size is determined from the dose-response relationship. This approach is referred to as "ED_x" or "EC_x", where ED is effective dose, EC is the effective concentration, defined as the dose or "concentration that produces a specified size of effect relative to an untreated control" [3] (Chapman 1998), and x represents the effect size–the selected change in response compared to the control response (for example, the dose resulting in a decrement in response of 25 % is designated as ED_{25}). A particular ED_x (the dose that would result in a decrease in performance by the percentage chosen as the effect size) may be determined from dose-response data through several procedures including graphical techniques, calculation from a fitted equation, or interpolation between the measured responses that bracket the selected effect size. A modification is to calculate the TRV for the lower confidence limit of the data, which is termed a "benchmark dose" (USEPA 1995b).

Some of the advantages of the ED_x approach for determining TRVs are that the size of the effect is known (because it is selected beforehand), the TRVs are not constrained to the particular doses tested (because they are determined from the dose-response relationship revealed by the test data), the TRVs do not depend on the particular statistical test chosen, and confidence intervals can be calculated. One of the main limitations is in choosing the appropriate regression model for curve-fitting approaches. Confidence limits may be quite large for threshold [4] and hormesis [5] models (Chapman 1998). Also, determination of TRVs for very low effect levels (less than ED_{10}) becomes strongly model dependent (Moore and Caux 1997; Scholze, et al. 2001). Fortunately, determination of TRVs for effect levels greater than 10 % has low model dependence, that is, the choice of

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[3] Dose is the rate of exposure of an animal or plant to a chemical, usually expressed as the amount of chemical per unit bodyweight per day. Instead of dose, the concentration of the chemical under investigation may be given for contaminated media (water, soil, air), food, or in a tissue or the whole body of the exposed animal or plant.

[4] For threshold models, treatment responses are flat (not different from the control response) at low doses until a critical level of dose is reached above which the treatment responses decrease as the dose increases.

[5] Hormesis refers to enhanced responses (treatment responses greater than control responses) at low doses of a chemical that has adverse effects at higher doses. For hormesis, treatment responses are flat (same as control) as the dose initially increases above the control dose, but, before reaching the critical threshold for adverse effects, the treatment responses become greater than the control response. As the critical threshold is approached, the treatment response decreases to the control level, and, as the doses increase above the critical threshold, the treatment responses decrease below the control response (adverse effects occur).

regression model has relatively minor effects on TRVs when calculated for ED_{10} or higher (Moore and Caux 1997).

An ED_x approach therefore is applied to the PCB toxicity data for mink and chicken to develop TRVs appropriate for assessing the risk range between no effect and low effect levels.

Although congener-specific analyses are recommended for assessing risks to PCBs, Aroclor-based toxicity reference values (TRVs) are still useful for several reasons. 1) The PCB database at many sites is predominantly or solely Aroclor data. This is especially true of historic data. 2) At contentious sites, the lengthy process for resolving disagreements has resulted in a need to finalize Aroclor-based risk assessments initiated prior to the current emphasis on congener-based approaches. In these situations, abandonment of the an Aroclor approach could entail substantial delay and cost for resampling media and biota to provide synoptic congener data. 3) There is a large database available on the ecotoxicological effects of PCBs on an Aroclor basis. 4) The utility of the available TEQ-based ecotoxicological studies is compromised by the use of inconsistent toxic equivalency factors (TEF). Conversion to a common TEQ basis is feasible only if the original congener data is reported so that the TEF scheme of choice can be applied (Dyke and Stratford 2002), but the underlying congener data are rarely reported in journal articles, which reduces the pool of comparable TEQ studies. Results of *in vitro* bioassay TEQs cannot be directly compared to calculated TEQs because bioassay results and congener relative potencies (REPs) may vary with changes in test protocols, for example, the solvent for dosing the cells (Tillitt, et al. 1991), exposure time (Clemons, et al. 1997), or the species from which the cell line is derived (Aarts, et al. 1995); and bioassays may show responses to chemicals not having significant effects in animals because of toxicokinetic processes not present *in vitro*. 5) The currently available TEQ approach assesses only toxicity related to aryl hydrocarbon receptor (AhR)-mediated processes (dioxin-like effects). Although AhR-mediated effects are frequently reported to be more sensitive endpoints compared to non-AhR effects, it is not clear how generally this relationship applies across taxa and endpoints. In the absence of a non-AhR TEF scheme, an Aroclor-based assessment can provide an indication whether significant non-AhR effects may have been missed in a TEQ-based assessment.

4 Methods

4.1 Linear Interpolation

The effluent toxicity testing guidance in the water program (e.g., Klemm, et al. 1994; Chapman, et al. 1995) is modified for deriving PCB TRVs from multiple mink or chicken studies. The guidance recommends linear interpolation between the treatments showing effects that bracket the chosen effect level. The linear interpolation method avoids the complications associated with selection of the appropriate regression model by focusing on the mean dose-response trend in the region surrounding the chosen effect level. Confidence intervals are then calculated through a bootstrap method. The method assumes monotonicity, that is, that the mean response decreases as the test concentration increases, and data are smoothed (adjusted) if this pattern is violated.

The linear interpolation method was developed for deriving TRVs from the results of individual toxicity studies. However, for the present effort, the results of multiple studies are combined to better reveal the shape of the

dose-response relationship for PCBs. This is necessary because most of the individual PCB toxicity studies tested a limited number of doses. Interpolation is strictly implemented for this effort–no extrapolations beyond the empirical data range are performed.

The first modification is to normalize the data so multiple studies can be compared on a common basis. The reason for combining research results is to better define the shape of the dose-response relationship compared to that shown by the relatively low number of doses tested in any single experiment (Section 4.7). Normalization is accomplished by dividing each mean treatment response by the respective mean control response (Equation 1). Two examples of this normalization procedure for combining multiple studies are Leonards, et al. (1995) and Tananka and Nakanishi (2001) (the latter normalized both response and exposure concentration, but only response is normalized for the present effort). The normalized responses are termed "relative response" (RR).

RR = treatment response / control response of the same study [1]

The relative responses are plotted on semi-log graphs (base 10 logarithm dose or concentration vs. relative response). The plots showing interpretable dose-response relationships (Section 6.1.1) are used to derive the no- and low-effect TRVs by a linear interpolation between the treatments that bracket the effect level of concern. The plots showing obviously inconsistent dose-response relationships, either because there is no relationship or because the combined studies are incompatible for some reason, are excluded for TRV derivation.

The second modification is interpolation is only performed when the selected effect size falls within the steep linear portion of the dose-response plot. There are two purposes: 1) the linear interpolation method is applicable to linear responses, but will over- or underestimate for nonlinear portions of the dose-response relationship; and 2) this avoids interpolation over excessively large exposure gradients for which the shape of the dose-response relationship is poorly known. The practical result is that most of the interpolations are performed between relatively small gradients in exposure values. The majority of the TRV interpolations for mink occur between treatments that differ in dietary concentrations by 2-fold or less, with the largest difference for the interpolations for Clophen A50 and live kits (3-fold for exposure over 2 breeding seasons, and 5-fold over 1 breeding season). Interpolation is not performed for the TRV for A1254 and kit survival, for example, because there is a 100-fold difference between the dietary concentrations of the treatments that bracket the target low-effect response. Many of the bird TRVs are interpolated between small gradients (2- or 3-fold for A1242 or A1248 dose and hatchability, less than 4-fold for A1254 dose and hatchability, and 2-fold or less for A1242 or A1254 egg residue and hatchability). A few bird TRVs are interpolated over larger gradients (6- fold for A1242 egg residue and chick bodyweight, 7-fold for A1248 egg residue and hatchability, and 10-fold for A1242 or A1248 dose and chick bodyweight, and A1248 dose and survival). Interpolations are not performed for greater than 10-fold differences in treatment doses.

A third modification is log-linear interpolation (Equation 2) is used since it gives a better fit within the linear portion of the data plots compared to the linear interpolation in the guidance.

$$\text{Log}_{10}\,\text{TRV} = \text{Log}_{10}\,C_j + (((M_i * P) - M_j) * ((\text{Log}_{10}\,C_{j+1} - \text{Log}_{10}\,C_j) / (M_{j+1} - M_j))) \quad [2]$$

$$\text{TRV} = 10^{\text{Log10 TRV}}$$

Where TRV is the interpolated toxicity reference value, P is the chosen effect size (Section 4.2), M_1 is the control relative response (1.0 by definition because the response data is normalized to controls), C_j is the test concentration of the treatment that produced a relative response (M_j) greater than P, and C_{j+1} is the test concentration of the treatment that produced a relative response (M_{j+1}) less than P. The symbols used in Equation 2 are the same as the ones in the guidance for effluent toxicity testing. Equation 2 is used for interpolating TRVs on the basis of PCB concentration in mink diet or chicken eggs. A similar equation is used for interpolating TRVs on the basis of bodyweight-normalized dose to chicken, where C is replaced by D for dose.

A fourth modification is data are not smoothed when treatment responses exceed control responses (relative responses > 1) to allow for hormesis (enhanced response at very low doses). One of the response patterns used for bird TRV derivation, chick bodyweight vs. A1242 egg residues (Figure 27), was attributed to hormesis by the investigators (Gould, et al. 97). The same investigators also reported a hormetic effect of A1254 on chick bodyweight (Figure 26). Gould, et al.'s conclusion is accepted because hormesis is evident at two dose levels for two different endpoints. All three of the commercial PCB products tested in mink feeding studies show possible hormetic effects on the number of live kits per mated female (Aroclors 1242 and 1254, and Clophen A50) (Figures 2, 3, 7). Hormesis is evident in the Clophen A50 experiment for exposure durations of both 1 and 2 breeding seasons (Figure 7). This effect is also shown by some of the feeding trials performed with field-contaminated prey for the same endpoint (Figures 8 and 13). Therefore, acceptance of hormetic responses is justified for the effects of egg residues on chick bodyweight (as attributed by the researchers), and the effect of dietary exposure on the number of live kits per mated female mink (exhibited in multiple studies). This indicates that adjustment of deviations in monotonicity is unwarranted for a treatment response exceeding the control response. The same modification to the linear interpolation method to allow for potential hormesis was made in a recent comparison of techniques for calculating effective doses (Isnard, et al. 2001). Data smoothing for monotonicity is performed in a few cases when the treatment responses are less than the control response, that is, when hormesis can not explain the deviations (documented in the notes to Tables 2 and 3).

A fifth modification is the procedure for deriving confidence intervals is not implemented since the only available data from the published mink and chicken studies are the treatment means (the underlying data for the individual replicates were not presented for any of the studies). The bootstrapping method for generating confidence intervals for the linear interpolation method requires the full replicate data.

An additional modification was made for the mink TRVs only. Two mink feeding studies, one performed with Clophen A50-supplemented feed and one with field-contaminated prey, reported the reproductive effects of PCBs associated with exposures over both one and two breeding seasons, and the latter study also reported the reproductive effects in two generations of exposed females. Both studies showed increased adverse effects in the second year or generation of continuous exposure. Since only single-season exposures have been reported for commercial Aroclor feeding studies, TRVs protective for long-term occupancy of a site by female mink are calculated by multiplying the single-season Aroclor TRVs by the mean ratio of the Clophen A50 and field-contamination TRVs for exposure over two breeding seasons or generations divided by the corresponding TRVs for single-season exposure in the same studies (the ratios are given in Table 2).

4.2 Effect Size

Effect size is the amount of decrease in response of animals or plants exposed to a chemical compared to unexposed controls that is selected as the level of concern for assessing risk (the x of ED_x, Section 3). The selected effect sizes for this effort are not based on receptor-specific life history/population models. The bird TRVs, derived from chicken data, are intended to provide conservative TRVs for application to species of unknown sensitivity to PCBs, for which no single population model would be applicable. The mink TRVs may also be applied to mammalian receptors of unknown sensitivity to PCBs (this requires bodyweight normalization of the mink dietary TRVs), in addition to mink for which it is derived. The effect sizes used in this effort are chosen for pragmatic reasons–to minimize model dependence, approximate the power of well-designed toxicity studies, and maintain general consistency in approach with other regulatory uses of toxicity test data. In short, to select a low effect size that is expected to be detectable in a well-designed study, and is reasonably consistent with prior Agency practice. The very steep PCB dose-response plots make the question of the appropriate low effect level somewhat moot, since there is a small range between no-effect and total-effects levels.

A pragmatic consideration is to avoid choosing an effect size for which interpolation may be strongly model dependent. In an examination of aquatic toxicity data sets, Moore and Caux (1997) concluded that interpolation becomes strongly model-dependent for less than 10 % decreases in response compared to that of controls (see also Scholze, et al. 2001). The various models gave reasonably consistent results for response differences of at least 10 % compared to controls. A related consideration is the effect size commonly associated with statistically-determined lowest observed effect concentrations (LOECs) in well-designed toxicity studies. The LOECs of the toxicity studies for the ambient water quality criteria (AWQC) and pesticide programs generally correspond to 20 to 25 % effect sizes (Suter, et al. 2000), and interpolation of the 25 % effect size is recommended for effluent toxicity testing (e.g., Klemm, et al. 1994; Chapman, et al. 1995). Another pragmatic consideration is consistency with the basis for regulatory decision-making in other programs that utilize toxicity testing results. A *de minimis* effect size of 20 % was identified in one such review (summarized in Suter, et al. 2000) [note: this is not a standard written in the regulations, but the minimum effect size associated with regulatory actions in practice].

This indicates that a reasonably detectable effect size consistent with Agency practices in other programs would fall between 20 and 25 %. The higher of these values is chosen for this effort to ensure that the low effect size represents a non-trivial departure from the control response (equivalent to 75 % relative response). In other words, the interpolated low effect TRV is the ED_{25} or EC_{25}.

The no effect size is set a t 10 % (relative response of 90 %), so the interpolated no effect TRV is the ED_{10} or EC_{10}. Similar to the rationale for the choice of low effect size, 10 % is chosen for no effect size because it is unlikely to be identified as a LOAEL in a reasonably well-designed toxicity study, is lower than the *de minimis* effect-level identified in a review of regulatory decision-making, but is at the minimum size so that the calculated ED_{10} is not strongly model-dependent (various regression techniques will likely give similar values).

The effect sizes could be further refined by linking them to species-specific population models to derive effect levels from projected population dynamics–the models probably need to be both region- and habitat-specific, but even so, there may be significant uncertainty (Section 6.1.6). However, because of the nature of the dose-response relationships for PCBs and reproductive endpoints in mammals and birds, such refinement would have relatively minor impact on the final TRV values.

The question of the appropriate value for the low effect size is made somewhat moot by the very steep dose-response plots for PCBs. For example, the A1248 oral dose to hens associated with complete hatch failure (~1 mg/kg-d) is less than 3 times greater than the dose showing no effect (~0.4 mg/kg-d) (Figure 19). The same is true for mink endpoints. Live kit production is completely suppressed at a dietary concentration of 5 mg/kg A1242, but no effect is reported at 2 mg/kg (exposure over a single breeding season) (Figure 2). The range in A1254 dietary concentrations for the same endpoints are 2 and approximately 1 mg/kg, respectively (exposure over a single breeding season) (Figure 3). Refinements of the effect level will therefore produce only relatively small changes in the TRVs.

4.3 Study Selection

Study results are selected according to the following criteria: 1) studies published in journals (gray literature [6] excluded), 2) primary sources (secondary sources [7] excluded), 3) matched control and treatment responses, 4) continuous PCB exposure up to or through the initiation of breeding (responses following cessation of exposure are excluded if sufficient time elapsed to allow depuration [8] to occur prior to breeding), and 5) treatment responses individually reported by dose and Aroclor (aggregated responses based on combinations of exposure levels or combinations of Aroclors are excluded). The individual Aroclor constraint is not applied to studies with field-contaminated prey. Statistical significance is not a criterion for selection of treatments within a study since the objective is to develop dose-response relationships over the full gradient tested (treatments that do not differ from the control response are as important for delineating the dose-response relationship as the treatments that do differ). When response data are reported for more than one exposure time, data for later exposure periods take precedence over earlier exposure periods or data averaged over the entire exposure period. Data are taken from text, tables, or figures so long as the selection criteria are met.

Only studies in which the test animals were exposed to commercial PCB products are used for calculating TRVs. Studies performed with field-contaminated prey are not directly used for calculating TRVs (to avoid possible confounding effects of contaminants not occurring in PCB products), but are included to contribute to the weight-of-evidence for response trends (e.g., evidence of hormesis), to contribute to the estimation of the proportional change in mink responses when the exposure duration increases from one breeding season to two breeding seasons or generations, and for overall comparison with Aroclor studies. Aroclor and field contamination studies are plotted separately for mink, but since only one chicken study is included with field-contaminated feed, it is plotted on the same graphs with chicken Aroclor studies to conserve space (the field-contaminated study is shown as "PCB" in Figures 17, 21, 25, 26, and 29-31).

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[6] Gray literature refers to studies not published in journals or books, or abstracts of results that provide insufficient information on methods and data. Examples of gray literature include meeting abstracts , government reports, master's or doctoral theses, unpublished research notes, and prepublication drafts.

[7] Primary sources are to the original publications reporting research results. Secondary sources are review articles, compilations, or other summaries of previously published work.

[8] Depuration is the elimination of chemicals from an animal after the cessation of exposure, through metabolic conversion and/or excretion.

Of the studies used for TRV derivation, only one did not continue exposure throughout breeding. Käkelä, et al. (2002) exposed mink to A1242-supplemented food for 21 weeks, but then switched to the control diet at the onset of breeding. This treatment is included because there was no delay between the cessation of A1242 exposure and initiation of breeding, therefore depuration did not occur prior to breeding. The sole TRV calculation involving this treatment is for live kits per mated female for A1242, in which the Käkelä, et al. datum is consistent with the trend of the other studies (Figure 2).

One of the "field-exposed diet" studies (mink fed meat from A1254-exposed cows) reported the control response for only one of the endpoints in the study (live kits per mated female) (Platanow and Karstad 1973). Other responses are included only when the treatment response was zero (e.g., 0 % kit survival in the 0.64 ppm treatment), because the relative response in this case is not affected by the specific value of the control response. This study is not included in the A1254 TRV derivation because A1254 was not fed directly to mink. The bioaccumulation process in cows increased the toxicity of the PCBs to the next higher trophic level (animals feeding on cows) as does bioaccumulation in wild animals (PCB toxicity to predators is usually greater than to their prey), so this study is included as one of the field-exposure studies.

It is not feasible to exactly match the exposure durations between studies. Exposure durations range from 6 to 14 wk for chicken feeding studies, with most between 6 and 9 wk (Table 7) (an individual 39-wk treatment by Platanow and Reinhart (1973) is not used for TRV derivation), and from 3 to 10 months for mink studies performed over a single breeding season (Table 6) (the results of the 2-month exposure duration by Jensen (1977) is not used for TRV derivation because the type of PCB in this study was not identified). For mink, the studies are segregated by the number of breeding seasons exposure was maintained (the results of exposure over 2 breeding seasons or 2 generations are analyzed separately from 1-season results). The data show no obvious effects due to the range in exposure durations (other than the 1-season vs. 2-season or 2-generation results for mink which are therefore disaggregated) (see Sections 6.1.2 and 6.1.5 for further discussion).

The exposure route for all of the mink studies is the same–contaminated diet. For oral dose to chicken, the exposure route is contaminated diet with one exception–contaminated water in the study by Tumasonis, et al. (1973). The data do not show an effect related to this difference in exposure media. The relative effect due to exposure to contaminated water is consistent with the effect trends of exposure to contaminated diet (Figures 20 and 24). As it turns out, the Tumasonis, et al. results had no direct influence any of the TRV interpolations. For egg concentration, the exposure route was through maternal dietary exposure except for Gould, et al. (1997) in which PCBs were injected into egg yolks on day 0 of incubation. The Gould, et al. study influenced one TRV (chick bodyweight vs. A1242 egg residue). Again, the response trend is consistent between exposure routes (Figure 27) (see Section 6.1.3 for further discussion).

4.4 Toxicity Endpoints

Data for the following reproductive and growth endpoints were collected from a review of mink PCB studies: whelping frequency (number of female mink giving birth / number mated), total kits (live and stillborn at birth) per whelped female, live kits per whelped female (at birth), live kits per mated female (at birth), kit bodyweight, and kit survival (Table 4). Since the effects of the first three endpoints are integrated in the number of live kits per mated female, TRVs are not separately calculated for whelping frequency or for total or live kits per whelped

female. Kit bodyweight and survival are reported for various times following birth as given in the original studies. TRVs are calculated for kit bodyweight at birth, but not for later times, because the database for later times is smaller than for bodyweight at birth. Kit survival was reported for 4 to 6 weeks following birth in the studies used for TRV derivation.

For chicken PCB studies, the toxicity endpoints include egg productivity, egg fertility, hatchability, chick bodyweight, chick survival, and chick deformity. To maintain comparability among the dose-response plots (reduced response at higher doses for endpoints exhibiting a relationship with PCB exposure), chick deformity is converted to chick normality, that is, the relative proportion of chicks *without* deformities is plotted. Chick normality is calculated as 1.0 - the proportion of deformed chicks. As with other endpoints, treatment normality is divided by the corresponding control normality to calculate the relative response, in this case, relative normality (or normalized normality!).

4.5 Data Conversions

Normalization of response data is discussed in Section 4.1. The data sources, relative response calculations, and other data conversions are documented in Tables 6 and 7.

The mink dietary PCB concentrations are as given in the original studies when available. Two studies expressed the exposure in terms of daily ingestion (mg PCB/mink/d), instead of dietary concentration (Brunström, et al. 2001; Kihiström, et al. 1992). The dietary concentration is calculated by dividing the daily PCB ingestion by the daily food ingestion reported in each study (see notes to Table 6). For some of the study results, the reported data are converted to make them consistent with the toxicity endpoints assessed in this effort. For example, if the number of live kits per mated female is not given in the original study, it is calculated by multiplying the number of live kits per whelped female by the fraction of females whelped of those mated. The conversions are documented in the notes to Table 6.

The chicken dietary PCB concentrations are converted to bodyweight-normalized doses by multiplying by the food ingestion rate reported in the study, or by a default leghorn hen food ingestion rate of 0.067 kg feed/kg_{BW}-d (Medway and Kare 1959). For the single study with PCB exposure through water (Tumasonis, et al. 1973), the bodyweight-normalized dose is calculated by multiplying the PCB concentration in water by the reported daily water consumption per hen divided by the reported hen bodyweight (see note to Table 7). When egg PCB concentrations were reported for egg yolks, the data are converted to whole-egg concentrations by multiplying by 0.364, the proportion of yolk in chicken eggs on a wet weight basis (Sotherland and Rahn 1987).

The relative "chick" normality (see Section 4.4) for Lillie, et al. (1975) is based on abnormal embryos, not on deformities in hatched chicks. However, data are insufficient for deriving deformity-based Aroclor TRVs. The relative "chick" bodyweight for Gould, et al. (1997) is based on 17-d embryos, not on hatched chicks. This data set plays an important role in the A1242 egg TRVs for chick bodyweight.

4.6 Presentation

The source data, data conversions, and relative response calculations are documented in Tables 6 and 7. The relative responses are summarized in Tables 4 and 5, and plotted in Figures 1-32 in semi-log graphs (dose or concentration on a base 10 logarithmic scale). To aid interpretation, the data points of commercial PCB feeding studies that exhibit interpretable dose-response relationships are linearly connected in the figures showing the effects of a single commercial product (an exception is made for Figures 25 and 28 because of the small number of data points). Data points are also linearly connected in the figures illustrating the Restum, et al. (1998) study performed with field-contaminated diets because the results are used in part to estimate the effect of increasing exposure duration from 1 breeding season to 2 breeding seasons or generations. Data are presented as scatterplots (unconnected) in the figures simultaneously showing the effects of multiple Aroclors or multiple field-contaminated diet studies on an individual toxicity endpoint, and in the figures of endpoints that do not exhibit an interpretable dose-response relationship.

The TRV interpolations are presented in Tables 2 and 3. Although the TRVs are derived through calculation, and not through a graphical approach, their derivation can be visually understood by examining the figures. The low effect size is shown in the figures for endpoints used for TRV derivation by a horizontal line indicating 0.75 relative response (effect size of 25 %). The low effect TRV (ED_{25} or EC_{25}) is represented by the dose or concentration corresponding to the intersection of the 0.75 relative response line and the line connecting the scatterplot data. The two data points nearest to the intersection are the data used for interpolation (see Tables 2 and 3 for the sources and values of the interpolation data). Similarly, a no effect TRV (ED_{10}) is the intersection of the 0.90 relative response line (not shown) and the line connecting the scatterplot data.

4.7 Example

A comparison between the results of individual studies and combined studies is illustrated in Figure 16 for the effect of A1248 dose to hen on hatchability. The 9 mean data points in this plot come from 3 studies–one contributing 4 means, one 3 means, and another 2 means (the exposure durations of these 3 studies are similar, 8 to 9 wk). There is an internally consistent dose-response relationship based on the combined data that exhibits a threshold for significant adverse effects above 0.3 mg/kg_{BW}-d, with a steep decrease in hatchability to nearly complete suppression above 1.0 mg/kg_{BW}-d. Based on the combined data, the interpolated no effect TRV (ED_{10}) is 0.38 mg/kg_{BW}-d, and the low effect TRV (ED_{25}) 0.48 mg/kg_{BW}-d (Table 3). Taken individually, the interpolated ED_{25} for the separate studies are approximately 0.2, 0.25, and 0.45 mg/kg-d. Two of the studies provide inaccurate estimates of the ED_{25} because the doses chosen for those studies do not adequately reveal the steep portion of the dose-response relationship. In both cases, the doses used for interpolation differ by an order of magnitude, that is, interpolation is performed over a 10-fold dose gradient The one study (Lillie, et al. 1975) that adequately reveals the steep portion of the dose-response relationship was performed with closely spaced doses (2-fold gradients) specifically selected between the doses showing no and severe effects in an earlier investigation by the same research group.

Statistical analyses were presented in two studies [9] for the effect of A1248 dose on hatchability. The NOAEL was 0.12 mg/kg_{BW}-d (2 ppm treatment), and LOAEL 1.2 mg/kg_{BW}-d (20 ppm treatment) for Lillie, et al. (1974). Compared to the dose-response relationship in Figure 16, the NOAEL is much lower and LOAEL much higher than the actual threshold for effects. In the study by Scott (1997), the NOAEL was 0.07 mg/kg_{BW}-d (1.0 ppm treatment) and LOAEL 0.67 mg/kg_{BW}-d (10 ppm treatment). In this case, the LOAEL is closer to the ED_{25} of the combined data, but the NOAEL is much lower than the ED_{10}, in other words, one treatment dose was fortuitously chosen that fell within the narrow transition between no and severe effects, but the 10-fold gradient to the next lower dose tested was too large to adequately represent the threshold for adverse effects.

5 Results

5.1 Mink Studies

The results of mink studies are shown in Figures 1-15. Exposure-response relationships are evident for number of live kits per mated female (Figures 1-3, 7, 8, and 13), kit bodyweight (Figures 5, 9, 10, and 14), and kit survival (Figures 11, 12, and 15). Data were also normalized for whelping frequency, total kits per whelped female, and live kits per whelped female, but these effects are integrated in the live kits per mated female endpoint, so are not separately analyzed.

The interpolated TRVs are given in Table 2. The dietary TRVs (mg/kg ww) for exposure in a single breeding season are as follows: A1242–2.5 (no effect) to 2.7 (low effect) for live kits per mated female; A1254–1.0 (no effect) to1.1 (low effect) for live kits per mated female and 1.1 (low effect) for kit bodyweight; and Clophen A50–2.4 (no effect) to 3.1 (low effect) for live kits per mated female. The A1254 TRVs for kit survival cannot be interpolated because of data complications (described below) and, for the no effect TRV, excessively large dose gradients, but are greater than 0.02 and less than 1.0 mg/kg ww diet.

The A1254 relative response for kit survival appears to show a no effect level of 1.0 mg/kg ww (Wren, et al. 1987) and complete mortality at 2.0 mg/kg ww (Aulerich and Ringer 1977) (Figure 6). Although Wren, et al. (1987) show the same kit survival for controls and the 1 mg/kg treatment, they reported a dramatic shift in the cause of the mortality in the two groups–mainly trauma and infection in the control kits (9 of 12 kits that died after birth), but predominantly starvation in the treatment kits (13 of 14 treatment kits that died after birth). In contrast, they reported that none of the control kit mortality was due to starvation. These observations raise the possibility that the treatment mortality might have been related to wasting syndrome, a "starvation-like" syndrome of chemicals with dioxin-like effects (Seefeld, et al. 1984; Lu, et al. 1986). Although the Wren, et al. study does not prove that wasting syndrome occurred, the major shift in the causes of mortality between the treatment and control groups indicates that there is substantial uncertainty in concluding that the 1 mg/kg treatment is, in fact, the no effect dietary concentration for kit survival in the Wren, et al. study. This means that the no effect dietary A1254 TRV for kit survival may be less than 1 mg/kg ww, and greater than 0.02 mg/kg ww (control), but more precise determinations cannot be made with the existing data.

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[9] Unfortunately, the statistical analyses in Lillie, et al. (1975) were only performed to compare the effects of different Aroclors (with the results of the multiple doses combined for any single Aroclor), or different doses (with the results of multiple Aroclors combined for any single dose). Statistical comparisons were not made to compare the effects of different doses of any single Aroclor.

Two studies, one performed with a commercial PCB product (Brunström, et al. 2001), and one with field-contaminated prey (Restum, et al. 1998), reported the reproductive effects of PCBs associated with exposures over both one and two breeding seasons. Restum, et al., also reported the reproductive effects in two generations of exposed females. Both studies showed increased adverse effects in the second year or generation of continuous exposure compared to the first (Figures 7-10, and 12). Brunström, et al. (2001) wrote:

> "In the second season, the effects on reproduction were more pronounced and clearly dose dependent... In our study, the concentration in the feed was the same during the two reproduction seasons, resulting in a reduced frequency of whelping females in the second season only. This finding suggests that the PCB concentration in the animals increased from the first to the second reproduction season, showing the relevance of long-term exposure for estimation of a LOAEL."

Brunström, et al. (2001) fed mink diets spiked with Clophen A50, one of the European commercial PCB products, and reported results for exposure over both 1 breeding season (6 months) and 2 breeding seasons (16 months). This study showed a dramatic decrease in the whelping frequency from 90 % of mated females for the first breeding season to 39 % for the second season in their "A50 high" treatment (2.3 mg/kg ww diet). The control whelping frequency was 93 % in both years. Live litter size per whelping female decreased nearly by half between the two exposure periods for the same treatment (from 3.8 live kits/whelped female the first year to 2.0 the second year) (control values 4.0 and 4.4, respectively). Mean kit bodyweight also decreased for this treatment (from 7.9 g to 6.7 g) (control values 9.6 and 8.9, respectively). Only kit bodyweight was statistically discernible from the control in the first breeding season, but, in addition to kit bodyweight, both whelping frequency and live litter size per whelped female were also statistically discernible from control values in the second breeding season. Sufficient data are available to calculate TRVs for both exposure periods for the number of live kits per mated female [10] (Table 2 and Figure 7). The low effect TRV for exposure over 2 breeding seasons (1.3 mg/kg) is 0.42 of the corresponding TRV for 1 season exposure (3.1 mg/kg), and the 2-season no effect TRV (1.1 mg/kg) is 0.47 of the 1-season value (2.4 mg/kg).

Restum, et al. (1998) fed mink various proportions of field-contaminated carp from Saginaw Bay, Michigan, and reported results for exposures over 1 breeding seasons (6 months), 2 breeding seasons (16 months), or 2 generations (exposure *in utero* [11] followed by 12 months exposure) (Figures 8, 10, and 12). Six comparisons are shown in Table 1 between 1-season and 2-season or 2-generation TRVs for live kits per mated female, kit bodyweight, and kit survival. Note that for live kits per mated female, the ratios of 2-season or 2-generation responses divided by the 1-season response result in maximum ratios. This is because the 1-season live kit per mated female TRV cannot be interpolated (it is at a higher dietary concentration than the highest tested). Instead of making an uncertain extrapolation, the relative response at the highest dietary concentration tested is used for the 1-season low effect TRV (0.9 relative response at 1.0 mg/kg). Since the 1-season EC_{25} is at a dietary concentration greater than 1 mg/kg, the actual product of dividing the 2-season or 2-generation TRVs by the 1-

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[10] The data for live kit production for single-season exposure is supplemented with the results of a single Clophen A50 treatment (12 mg/kg) reported by Kihlström, et al. (1992).

[11] Maternal exposure for 6 months including pregnancy. *In utero* means "in the womb", in other words, before birth.

season TRV would be smaller than the ratios shown in Table 1 for live kit per mated female (0.39 and 0.28, respectively). There are no such issues for the other endpoints. Overall, the ratio of 2-season or 2-generation TRVs divided by 1-season TRVs ranges from <0.28 to 0.87 for the various endpoints in the Restum, et al., study (Table 1).

For the purposes of adjusting the single-season Aroclor TRVs so they will be protective for sustainable occupancy by mink for multiple years or generations at a given location, the 1-season TRVs are multiplied by the mean ratio of the 2-season or 2-generation low effect TRVs divided by the 1-season TRVs based on the studies by Brunström, et al. (2001) and Restum, et al. (1998). The mean ratio of the seven comparisons is 0.52, that is, on average, the low effect TRV for 2-seasons or 2-generations exposure is 52 % of the low effect TRV for 1-season exposure to PCBs. Accordingly, the single-season TRVs for A1242 and A1254 are multiplied by 0.52 to derive TRVs for long-term sustainability. By this approach, the A1254 low effect TRV is 0.6 mg PCB/kg ww diet for live kit production and kit bodyweight, the A1254 no effect TRV is 0.5 mg PCB/kg ww diet for live kit production, and the A1242 TRVs are 1.3 (no effect) to 1.4 mg/kg ww (low effect) for live kit production.

The more conservative TRVs of the ones calculated for mink in this effort–no effect of 0.5 and low effect of 0.6 mg/kg ww diet based on A1254–are recommended for risk assessment purposes to account for the increased toxicity of PCBs that occurs with bioaccumulation and trophic transfer (foodchain transfer from prey to predators), or additive effects of concurrent exposure to co-contaminants that act through the same toxicological mechanisms as PCBs (Section 6.2.1.1).

5.2 Chicken Studies

The results of chicken studies are shown in Figures 17-32. Dose-response relationships are evident for hatchability (Figures 17-24) and chick bodyweight (Figures 25-27). Two dose-response patterns are evident for the effect of A1242 on hatchability (Figure 18)–one based on 3 studies by two research groups [12] (Briggs and Harris 1972; Cecil, et al. 1974; Lillie, et al. 1974, 1975), the other on 1 study by a third research group (Britton and Huston 1973). Each of these response patterns is separately analyzed instead of attempting to choose between the research results. An effect on chick survival is apparent for A1248, but not other Aroclors at the doses tested (Figure 28). There are no consistent dose-response relationships for egg productivity (Figure 29) or egg fertility (Figure 30). Although trends are apparent for chick deformities, studies were not performed at doses sufficiently high to allow interpolation of ED_{25}, except for the field study using field-contaminated feed (Figure 31) (studies based on field contamination are not used for TRV derivation). Only single data points are available for egg concentration and chick survival for each of the Aroclors considered in this effort (Figure 32), so concentration-response relationships cannot be evaluated precluding TRV derivation.

The interpolated TRVs are given in Table 3. The bodyweight-normalized dose TRVs (mg/kg_{BW}-d) are as follows: A1242–0.1-0.5 (no effect) to 0.4-0.8 (low effect) for hatchability, and 0.2 (no effect) to 0.9 (low effect) for chick bodyweight; A1248–0.4 (no effect) to 0.5 (low effect) for hatchability, 0.2 (no effect) to 0.6 (low effect)

[12] Two papers report data from the same experiment (Cecil, et al. 1974 and Lillie, et al. 1974).

for chick bodyweight, and 0.2 (no effect) to 0.3 (low effect) for chick survival; and A1254–0.6 (no effect) to 1.2 (low effect) for hatchability.

The interpolated egg TRVs (mg/kg whole egg, ww) are as follows: A1242–1.0 (no effect) to 1.5 (low effect) for hatchability, and 3 (no effect) to 10 (low effect) for chick bodyweight; A1248–0.7 (no effect) to 1.3 (low effect) for hatchability; and A1254–9 (no effect) to 12 (low effect) for hatchability.[13]

Although the lowest TRVs for hen dose are for A1248 and chick survival, little confidence can be placed in the calculated ED_{10} or ED_{25} because the interpolations are performed over a 10-fold dose gradient (Figure 28). Based on the shapes of the better defined dose-response plots for other endpoints, the interpolated values are probably underestimated. A similar concern applies to the no effect TRVs for A1242 or A1248 doses and chick bodyweight (Figure 25). Since two dose-response patterns are evident for A1242 and hatchability (Figure 18), the recommended bird TRVs are based on A1248 and hatchability–0.4 mg/kg_{BW}-d (no effect) and 0.5 mg/kg_{BW}-d (low effect) (bracketed by the two A1242 values).

For egg TRVs, the best defined concentration-response plots are for A1242 and hatchability (Figure 22) and A1254 and hatchability (Figure 24), in which interpolations are performed within gradients of 2-fold or less. Although the egg TRVs for A1242 chick bodyweight are interpolated over a 7-fold concentration gradient (Figure 27), and combines disparate exposure routes (egg injection and contaminant transfer from exposed hens), the low effect TRV is very close to the treatment mean based on dosed hens and not significantly influenced by the egg injection study (the converse is true for the no effect TRV). The egg TRVs for A1248 and hatchability are interpolated over a 7-fold concentration gradient (Figure 23), and therefore are have greater uncertainty than the A1242 or A1254 TRVs for the same endpoint. The recommended egg TRVs are based on the more sensitive of the Aroclors with well-defined concentration-response plots, that is, A1242 and hatchability–1.0 (no effect) to 1.5 mg/kg ww whole egg (low effect).

6 Uncertainty

Uncertainty is discussed for the method for deriving the TRVs and the application of the TRVs for risk assessment.

6.1 TRV Uncertainty

6.1.1 Confounding Factors

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[13] Adverse effects have been reported at whole-egg concentrations greater than 4 mg/kg based on the A1254 study by Tumasonis, et al. (1973) in reviews by Barron, et al. (1995) and Hoffman, et al. (1996), which is lower than the egg A1254 TRVs presented here also based in part on Tumasonis, et al. (1973). The difference is that the treatment response used in the present effort is based on the effects occurring during exposure to PCBs (maximal suppression of hatchability at 100 mg/kg in yolk). Tumasonis, et al. (1973) also reported deformities in chicks at yolk concentrations at or above 10-15 mg/kg in the weeks following cessation of exposure to PCBs, which is the basis for the effect levels reported in the reviews. These data were not used in the present effort because the effects occurred after cessation of exposure, and quantitative data on deformity rates were not provided.

An important potential source of uncertainty is associated with combining the results of separate studies together into aggregated dose-response plots because the studies were not performed under standardized protocols. Differences in results between studies may have occurred that are not linked to treatment doses for several reasons including differences in rearing conditions, feed, animal strains, health or nutritional status, age, exposure routes, or exposure durations. Other possible confounding factors include unsuspected alternate sources of contamination in the feed, water, or experimental facility (either to the same chemical being tested or to another unmeasured chemical), or differences in the composition of the Aroclor batches tested (different lots of the same Aroclor may differ in toxicity due to fluctuations in the composition of toxic PCB congeners or co-contaminants formed during manufacture).

The significance of these potentially confounding factors is assessed by examination of the dose-response plots of the combined studies. Marked deviations from interpretable dose-response patterns indicate that study results are incompatible for some reason. An interpretable dose-response pattern is one that is consistent with known patterns and toxicological theory. The basic pattern is a sigmoid curve in which low doses have minor effects, higher doses exhibit increasingly adverse effects, and the effects at the highest doses asymptotically approach maximum adversity. Two modifications are threshold models, in which increases in dose at low dose levels cause no significant changes in response until a threshold dose is reached, above which the sigmoid pattern applies, and hormetic models, in which doses lower than a threshold for adverse effects show an enhanced (positive) response. Of the endpoints considered in this effort, only two exhibit uninterpretable dose-response patterns–A1254 and egg productivity (Figure 29) or fertility (Figure 30). Either A1254 has no effect on egg productivity or fertility (at the doses tested), or the studies combined into these plots are incompatible for one or more of the factors described above. Regardless of the reason, these endpoints are excluded from the TRV process. Chick survival is also excluded because there are insufficient data to reveal dose-response patterns for any Aroclor (Figure 32). The rest of the endpoints of studies performed with commercial PCB products exhibited interpretable dose-response patterns consistent with one of the models described above, which indicates that the results of the combined studies were not significantly affected by confounding factors (with the possible exception of A1242 and hatchability discussed below).

6.1.2 Exposure Duration

In addition to the overall screening of interpretable dose-response patterns, it is also possible to specifically assess the possible effects of combining studies with different exposure durations or exposure routes. It is not feasible to exactly match the exposure durations between the studies combined into single plots. Exposure duration ranged from 6 to 14 wk for chicken feeding studies (most between 6 and 9 weeks), and from 3 to 10 months for mink studies performed over a single breeding season. The data are consistent within the range of exposure durations of the combined studies as discussed below.

The studies combined for A1248 and hatchability have similar exposures durations–8 (Lillie, et al. 1975) and 9 wk (Lillie, et al. 1974; Cecil, et al. 1974; Scott 1977)–and exhibit a consistent dose-response pattern (Figure 19). Three studies were combined to evaluate the effect of A1254 on hatchability with exposure durations of 6 (Tumasonis, et al. 1973), 9 (Lillie, et al. 1974 and Cecil, et al. 1974), and 14 wk (Platanow and Reinhart 1973); however, the relative response plots show internally consistent responses (no obvious duration effects) on the basis of either hen dose (Figure 20) or egg concentration (Figure 24). This is partly because the shortest duration

treatment (6 wk) was at a high dose that completely suppressed hatchability, but mainly because the results of the 9- and 14-wk studies are remarkably consistent. At first impression, the divergent A1242 and hatchability patterns appear to be related to exposure duration (Figure 18). The pattern showing greater toxicity is largely based on 8- to 9-wk durations (Lillie, et al. 1974, 1975; Cecil, et al. 1974), and the one showing lesser toxicity on 6-wk duration (Britton and Huston 1973), except that the data by Briggs and Harris (1972) with 6-wk exposure is consistent with the pattern exhibited by the 8- to 9-wk exposure studies, and inconsistent with the Britton and Huston study. The divergent A1242 patterns are inexplicable with the available information and therefore are separately assessed. This uncertainty is reflected in the TRV ranges presented for A1242 dose and hatchability.

All of the mink Aroclor feeding studies were performed over single breeding seasons. Three studies are combined for A1242 and live kit production (Figure 2) with rounded exposure durations of 5 (Käkelä, et al. 2001), 8 (Bleavins, et al. 1980) and 10 months (Aulerich and Ringer 1977). No and low effects are bracketed by the hormetic response at 2 mg/kg ww dietary concentration (Aulerich and Ringer 1977) and complete reproductive suppression at 5 mg/kg (Bleavins, et al. 1980) with roughly comparable exposure durations. The treatment at an intermediate dietary concentration (3 mg/kg) has the shortest exposure duration of the combined studies (5 months), which was terminated at the onset of breeding (Käkelä, et al. 2001) in contrast to the other studies, but exhibits a response consistent with the longer duration studies (in fact, plots close to a direct log-linear line between the other studies). Again, there is no evidence that the difference in exposure durations among studies has distorted the concentration-response relationship. Three studies are combined for A1254 and live kit production (Figure 3) with four rounded exposure durations of 3 (Kihlström, et al. 1992), 4 (Aulerich and Ringer 1977), 6 (Wren, et al. 1987), and 10 months (Aulerich and Ringer 1977). Live kit production is almost completely suppressed at all the tested dietary concentrations of 2 mg/kg or greater (3-, 4-, and 10-month exposure durations). An apparent inconsistency occurs at 1 mg/kg, with a 6-month exposure study exhibiting hormesis (Wren, et al. 1987) and a 4-month exposure study showing adverse effects (Aurlerich and Ringer 1977), which are the opposite trends expected based solely on the respective exposure durations (the data are smoothed at this dietary concentration by averaging the two responses). However, since reproduction is unsuccessful at 2 mg/kg (the sole live kit in that treatment soon died), there is no margin for increasing the A1254 low effect TRV, that is, it must be less than 2 mg/kg ww diet (for a single breeding season). The A1254 TRVs might be overestimated (too high) because they are bracketed at the no-effect side by the results of shorter exposure durations (4 to 6 months), that is, greater adverse effects may occur if mink were exposed to 1 mg/kg for 10 months instead of 4-6 months. The same consideration applies to the low effect TRVs for A1254 and kit bodyweight (Figure 5), which is bracketed by a 10-month exposure study for severe effects and a 6-month exposure study for lesser effects. However, a similar disparity in exposure durations of A1242 studies did not result in an obvious inconsistency in responses.

Two studies are combined for one of the Clophen A50 endpoints (live kits per mated female), with exposure durations of 3 (Kihlström, et al. 1992) and 6 months (Brunström, et al. 2001) (Figure 7). The responses are consistent because the single 3-month exposure treatment was performed at a sufficiently high dose to completely suppress reproduction. Once maximum adversity occurs, there is no scope for further change in response with increased exposure duration.

In contrast to the generally consistent results of combining single breeding season studies of varying exposure durations, exposure duration effects are apparent in both of the studies that included continuous exposures over

both 1 breeding season and 2 breeding seasons or 2 generations (Figures 7-10 and 12). The exposure duration was 6 months for the single breeding season treatments in both studies, and was 16 (Restum, et al. 1998) and 18 months (Brunström, et al. 2001) for females continuously exposed over 2 breeding seasons. The second generation females were exposed in the womb (6-month maternal exposure) followed by 12 months postnatal exposure (Restum, et al. 1998). The effect may be more pronounced for live kit production and possibly kit survival compared to kit bodyweight (compare Figures 7 with 9, and 8 or 12 with 10), and appears to be more pronounced for exposure over 2 generations compared to the same adult female continuously exposed over 2 breeding seasons (Figures 8, 10, 12). Since the concentration-response patterns differ for exposures over single versus double breeding seasons or generations, the data are not aggregated.

To summarize, there is no evidence that the range of exposure durations of the studies combined for assessing effects during single breeding seasons resulted in significant inconsistencies in the dose-response patterns for either chicken or mink. The A1254 TRVs for mink might be overestimated (too high) because the effect sizes for live kit production and kit bodyweight are bracketed by shorter exposure duration studies on the no effect side (4 to 6 months) as compared to the severe effect side (10 months), however, a similar disparity for A1242 showed no inconsistencies (a 5-month exposure duration treatment is intermediate in both dietary concentration and response to 8- to 10-month treatments). However, two studies show that the responses to 6-month exposures during a single breeding season differ from the responses to continuous 16- to 18-month exposures over two breeding seasons, and therefore should not be combined into aggregated dose-response plots. Similarly, a study shows that the responses to exposure over a single breeding season should not be aggregated with the responses of females exposed *in utero* followed by 12 months postnatal exposure.

6.1.3 Exposure Route

The same approach can be used to assess the effect of different exposure routes. The exposure route for all of the mink studies was the same, that is, through contaminated diet. For oral dose to chicken, the exposure route was contaminated diet with one exception–contaminated water in the study by Tumasonis, et al. (1973). The data do not show an effect related to this difference in exposure media. The response due to exposure to contaminated water is consistent with the effect trends of exposure to contaminated diet (Figures 20 and 24). For egg concentration, the exposure route was through hen dietary exposure except for Gould, et al. (1997) in which PCBs were injected into egg yolks. The Gould, et al. study influenced one TRV (A1242 egg residue and chick bodyweight), for which the egg injection data are combined with a single treatment from a hen feeding study (Lillie, et al. 1974, Cecil, et al. 1974) (Figure 27). In addition to the difference in exposure route, the relative "chick" bodyweight for Gould, et al. (1997) is based on 17-d embryos, not on hatched chicks. However, the response trend is reasonably consistent between exposure routes, or, better put, there is no obvious inconsistency between the response of the two studies. In any case, because of the spacing of the treatments, the low effect egg A1242 TRV for chick bodyweight is predominantly influenced by the hen feeding treatment, and the no effect TRV by the egg injection study. This means that the no effect egg TRV for A1242 and chick bodyweight may be less certain in comparison with the low effect TRV.

6.1.4 Linear Interpolation

The appropriate regression technique is a source of uncertainty for the ED_x procedure because the results depend on how well the dose-response relationship is modeled (Section 3). Model uncertainty in the present effort is minimized in three ways. 1) Uncertainties related to characterization of complex dose-response relationships, such as threshold or hormesis models, are avoided by linear interpolation of TRVs between the treatments that bracket the selected effect sizes for no and low effects. It is not necessary to mathematically represent the entire dose-response curve to calculate the ED_{10} or ED_{25}, so long as the overall shape of the dose-response relationship conforms with one of the known patterns. Related to this, extrapolation beyond the empirical data is strictly excluded. 2) The effect sizes (10 % decrease from control for no effect, and 25 % decrease for low effect) are selected to minimize model dependence (Section 3). 3) The results of linear interpolations are only accepted when performed within the steep linear portion of the dose-response plots, and, related to this restriction, confidence in the TRVs interpolated between narrow dose gradients is greater (less uncertainty) than for TRVs interpolated between wider dose gradients. The Aroclor TRVs for mink are interpolated within 2-fold or less gradients in dietary concentration (A1242 or A1254 and live kit production, and the low effect A1254 TRV for kit bodyweight). Most of the bird TRVs are interpolated within 2-fold gradients in dose (A1242 or A1248 and hatchability) or egg concentration (A1242 or A1254 and hatchability), and one of the no effect TRVs for A1242 dose and hatchability is interpolated over a 3-fold gradient. This indicates that uncertainty related to appropriate characterization of the dose-response relationship is low.

Although the TRVs for A1254 dose and hatchability are interpolated over a 4-fold gradient, there is low model uncertainty for the low effect TRV because it coincides with one of the treatment means (Figure 20). However, there is greater model uncertainty for the no effect TRV for A1254 and hatchability because the shape of the dose-response relationship is uncertain over the 4-fold gradient. Similarly, the TRVs for A1242 or A1248 and chick bodyweight (Figure 25), or A1248 and survival (Figure 28) have high model uncertainty because they are interpolated over 10-fold dose gradients (although modeling uncertainty is appreciably less for the low effect TRV for A1242 and hatchability because the treatment mean plots close to the low effect size). Despite the apparent greater sensitivity of chick survival for A1248 (or the no effect TRV for chick bodyweight) compared to hatchability, the A1248 TRVs are based on hatchability because the modeling uncertainty is high for the other endpoints.

To summarize, modeling uncertainty is low for the final TRVs because they are interpolated over narrow dose gradients within well-defined dose-response relationships.

6.1.5 Adjustment of Mink TRVs for Exposure Over 2 Breeding Seasons or 2 Generations

Another source of uncertainty for the mink TRVs concerns the empirical observations that continuous exposure over 2 breeding seasons or 2 generations increases the severity of the reproductive effects of PCBs compared to exposure over a single season, "showing the relevance of long-term exposure for estimation of a LOAEL" (Brunström, et al. 2001). Since the effect has been observed in mink feeding studies both with controlled dosing with one of the European commercial PCB products and with field-contaminated fish from a site in the United States, it is unlikely that it is caused by some unique attribute of the European product or some non-PCB-related contaminant in the field-contaminated fish (also, the field-contaminated fish of the latter study were collected at one time, homogenized, and stored for use throughout the study, so co-contaminant levels did not vary between breeding seasons). This indicates the increased toxicity of PCBs to mink with continuous exposures over

multiple breeding seasons or generations may be a general characteristic of PCBs, with implications for long-term occupancy of contaminated sites.

The potential for increased PCB toxicity with extended exposure is relevant for assessing the long-term suitability of habitats for mink because the estimated longevity in the wild is 3 to 6 years, with maximum longevity of 8 to 12 years during which mink are fecund for 7 or more years (Chapman and Feldhamer 1982; Merritt 1987). Unfortunately, mink Aroclor studies have only been performed for single breeding seasons and single generations, so there is uncertainty in either accounting for or ignoring the increase in toxicity associated with exposures over 2 breeding seasons or 2 generations in other studies. If excluded, a habitat remediated on the basis of single-breeding season TRVs may allow for unimpaired mink reproduction during the initial year of occupancy, but not in succeeding years or generations of continued occupancy. The net effect would be that only transient mink would have unimpaired reproduction, but not resident mink that remain in the same locality through multiple years or generations. In other words, the habitat might remain a population sink in which the presence of mink would depend on regular immigration from other areas. If the increase in toxicity related to exposure over multiple years or generations is accounted for by adjusting the single-season TRVs, reproductive impairment by PCBs would not be expected in mink regardless of residence time or number of generations at the site. The uncertainty in this scenario is in determining the appropriate adjustment to Aroclor TRVs when the empirical data are limited to Clophen A50 and field-contaminated fish.

The uncertainty in not making this adjustment would be low if the difference between the effects of exposures to 1 versus 2 breeding seasons or generations was relatively small. However, the study with Clophen A50 showed large decreases in the proportion of females giving birth (57 % decrease in whelping frequency) and the number of live kits per whelped female (47 % decrease) compared to exposures over 1 breeding season (Brunström, et al. 2001), so that only one-fourth of the number of live kits were produced per mated female in the second breeding season compared to the first (Figure 7). The Restum, et al. (1998) study with field-contaminated fish showed similarly large effects for live kit production (Figure 8) and kit survival (Figure 12), as well as a pronounced effect on the bodyweight of kits whelped by 2nd generation females (themselves exposed *in utero* and postnatally) much greater than the effect on kit bodyweight due to exposure to adult female mink over either 1 or 2 breeding seasons (Figure 10).

The weight of evidence indicates that the uncertainty associated with excluding an exposure duration or generational effect may be high, that is, potentially severe adverse effects may be overlooked. However, there is a large range in the ratio of 2-season or 2-generation exposure-based TRVs divided by 1-season exposure TRVs for the various endpoints reported in the two studies, from less than 0.3 to 0.9 (Table 2), which means that selection of an adjustment factor for Aroclor TRVs is correspondingly uncertain. Although the ratios are lowest for live kit production (<0.3-0.4) and kit bodyweight of 2nd generation-exposed females (0.4), the two endpoints used for the mink Aroclor TRVs, the approach taken in this effort is to use the mean ratio of all the endpoints for which low effect TRVs could be calculated (mean of 0.52, n = 7). The mean ratio should have lower uncertainty compared to ratios selected from either end of the range, and is therefore used to adjust the mink Aroclor TRVs in the absence of Aroclor-specific data.

For comparison, the mink TRV for the GLI water quality criteria is based on an A1254 dietary LOEC of 2 mg/kg (Aulerich and Ringer 1977), which was converted to a NOEC of 0.2 mg/kg by dividing by an uncertainty

factor of 10 (USEPA 1995a). These values bracket the mink A1254 TRVs derived in this effort. The low effect dietary TRV of 0.6 mg/kg is significantly lower than 2 mg/kg, but, as discussed in Section 3, the LOEC used by the GLI resulted in complete reproductive suppression, therefore the actual lowest dietary concentration associated with the onset of adverse effects is expected to be lower than 2 mg/kg. Since the LOEC resulted in severe effects, the NOEC for the GLI (the sole basis for decision-making in the GLI effort) was conservatively estimated by using a large uncertainty factor, which resulted in a value somewhat lower than the no effect dietary TRV of 0.5 mg/kg based on long-term sustainability. This comparison indicates that an appropriate level of conservatism was used in the GLI effort in estimating a no effect level from less than ideal toxicity data, and that the TRVs derived in this effort are reasonably consistent with the GLI even though the values are adjusted to account for the observed increase in toxicity with continuous exposure over multiple years or generations.

6.1.6. Endpoints and Effect Size

Consistent with the guidance for ecological risk assessment in the Superfund program (USEPA 1997), the toxicological endpoints included in this effort are one that could impact populations–live kit production, kit survival, and kit bodyweight for mink; and hatchability, deformities, chick survival, and chick bodyweight for birds (bodyweight is an indicator of the potential for long-term survival). The main uncertainties with the toxicological endpoints relied on for the TRVs are that data are insufficient for fully evaluating all of the considered endpoints, for example, kit or chick survival might be a more sensitive endpoint than live kit production or hatchability; and data are sparse for other endpoints that could impact populations, such as immune system effects, or neurological or other somatic effects that could impair performance of essential activities such as mating, rearing, hunting, evading predation, migrating, or competing with other species. A possible field example involves Caspian tern exposure to PCBs at Saginaw Bay, MI. Although productivity did not appear to be affected by exposures, elevated plasma PCB level was associated with decreased return of adults to the colonies, suggesting a possible effect on survival (see discussion and references in Hoffman, et al. 1998). The possibility that other endpoints might be more sensitive or result in greater overall impact in the field compared to the endpoints used for TRV derivation in this effort (live kit production, kit bodyweight, and hatchability) is an underlying uncertainty.

The effect sizes used in this effort are chosen for pragmatic reasons–to minimize model dependence, approximate the power of well-designed toxicity studies, and maintain general consistency in approach with other regulatory uses of toxicity test data (Section 4.2). The main uncertainty with the effect size selection is that they are not linked to population models, that is, the effects of 10 or 25 % decrements in hatchability, live kit production, or kit bodyweight on local populations are not explicitly modeled. There is uncertainty in both directions–a 10 % decrease may result in larger impacts than appropriate for a no effect level, or a 25 % decrease may not result in discernible impacts. As discussed in Section 4.2, this uncertainty is low because of the very steep slope of the dose-response relationship between no effects and severe effects–mostly separated by less than 3-fold gradients in dose or dietary concentration. Since population modeling is irrelevant for either zero impacts or 100 % adverse impacts (the local population will not be impacted by exposures that do not affect individuals, but is clearly not sustainable when reproduction is completely suppressed), modeling could only influence the TRVs within the 2- or 3-fold gradient between the extremes in response.

Such modeling for mink or bird populations would itself have large uncertainty associated with it. There are multiple sources of uncertainty in modeling or measuring population responses to stresses (Lester, et al. 1996; Power, 1997; NRC 1998; Rose 2000; Forbes, et al. 2001; Shea and Mangel 2001; Tyre, et al. 2001). A significant uncertainty in choosing effect sizes based on population models is that "simple, general, *a priori* predictions are not feasible" even with knowledge of life history dynamics and how life history traits are affected by toxicant exposure, because of the large number of factors influencing the outcome (Forbes, et al. 2001). Uncertainty is further increased because exposure to new stressors can change which population traits most influence population growth rates (referred to as "vital rates"). This means that identification of sensitive population traits with prospective demographic studies (prior to exposure to stressors) does not reliably predict which population trait is most important for population impacts following exposure (Cooch, et al. 2001 and references).

> "[T]he vital rate which contributes most to the observed variability in life histories is not necessarily the one to which life histories are most sensitive (which is revealed by the prospective analysis), nor the one that will necessarily make the biggest contribution to variability in another environment. This is especially true in wild populations, where natural selection is likely to minimize variation in those parameters to which population growth (i.e., fitness) is potentially the most sensitive, such that observed variation in growth over time might be reasonably expected to reflect changes in one or more of the parameters to which growth is less sensitive." [citations omitted] (Cooch, et al. 2001).

Exposure to toxic chemicals not only "switches the sensitivity of [population growth rate] to changes in vital rates", but also "increases the sensitivity of organisms to stressors that affect vital rates other than the ones that have been affected by the toxicant" (Kammenga, et al. 2001). An additional uncertainty in identifying sensitive population traits is that the results depend on both the spatial and temporal scales of the assessment (Power 1997; Rose 2000). These considerations mean that there is large uncertainty in applying general population models, and significant uncertainty may be associated even with species- and site-specific models because contaminant exposure may change the interactions between the various population traits and population growth, that is, the pre-exposure demographic model may not apply to post-exposure conditions.

Since the PCB dose-response relationships show a narrow range between the onset of adverse effects and maximum severity, the uncertainty associated with population modeling to refine the choice of effect size for determining TRVs is considered excessive relative to the constrained range over which the TRVs can vary.

6.2 Application Uncertainty

There are several sources of uncertainty associated with the application of the TRVs to field situations. In addition to the usual uncertainties of extrapolating from laboratory studies to field conditions, and, in the case of the bird TRVs, extrapolating between species, there are additional uncertainties associated with measuring PCBs as Aroclors in environmental samples, or measuring or estimating TEQ, and their use in risk assessments.

6.2.1 PCBs and Risk Assessment

Polychlorinated biphenyls (PCBs) are not a single chemical, but are mixtures of large numbers of different chemicals based on a common structure--a biphenyl "frame" with variable numbers of chlorine atoms attached to it. Each different arrangement of the number of chlorine atoms and their spatial position on the biphenyl is a separate PCB chemical, referred to as a "congener". There are 209 possible PCB congeners, each with slightly to very different chemical, physical, and toxicological properties. The complex mix of congeners with differing properties presents several challenges for assessing the risks of PCB exposures.

First, the toxicity of PCBs is caused by a subset of the congeners. The best understood subset is the dioxin-like congeners that act wholly or in part through the same mechanism as dioxin (Van den Berg, et al. 1998). The dioxin-like congeners, often referred to as "planar" or "coplanar" congeners, are capable of binding with the same cellular protein--aryl hydrocarbon receptor (AhR)--that binds with dioxin in the initial step of a cascade of interactions leading to expression of toxic effects. However, some of the non-coplanar, non-dioxin-like PCB congeners or their metabolites also have toxic effects through separate toxic mechanisms that are not as well understood (Fisher, et al. 1998). Some of the coplanar congeners may act through multiple pathways, that is, they may contribute to both dioxin-like and non-dioxin-like toxicity. The combined toxicity of the dioxin-like congeners can be estimated through a toxic equivalent (TEQ) approach (described below), but, at present, there is no comparable approach for estimating the combined effect of non-dioxin-like congeners.

Second, each of the different commercial PCB products are comprised of different proportions of congeners, which means that the toxicity varies for the different Aroclors, for example, A1242 is more toxic than A1260 because A1242 has a higher proportion of dioxin-like congeners. The uncertainty related to differences in congener composition between Aroclors is addressed in this effort by separately assessing the toxicity of each Aroclor. The toxicity of a European product (Clophen) is assessed separately from American products (Aroclors) for the same reason.

Third, once released into the environment, the differences in the chemical and physical properties of the congeners result in differences in their fate and transport, that is, in their persistence, how they move through the environment, and in which components they are likely to accumulate in greater concentrations. For example, the lower chlorinated congeners (ones with few chlorine atoms) volatilize (evaporate), solubilize (partition to water), and degrade more readily so they tend to decrease over time, while the heavier, more chlorinated congeners are less volatile, less soluble, often less readily degraded, and therefore are more persistent in the environment. Conversely, under anaerobic conditions (without free oxygen), some of the higher chlorinated congeners may be more readily degraded than lower chlorinated ones. Therefore, congener composition of PCBs in the environment can change over time, a process described as "weathering". The congener composition may also be altered as PCBs are passed through foodchains, that is, the congener pattern retained in animals may differ from the pattern in their food. The changes in congener proportions mean that the toxicity of PCBs in the environment differs from the toxicity of the source Aroclors depending on the type and degree of weathering and bioaccumulation.

6.2.1.1 Aroclor-based Risk Assessment

The original toxicity testing of PCBs was performed with commercial Aroclors, with the results presented in terms of Aroclor dose or concentration. An advantage of the Aroclor approach is that studies show the

combined effects of all the toxicological modes of actions of the various congeners (both dioxin-like and non-dioxin-like) and manufacturing impurities, and their net interactions (additive, synergistic, and antagonistic). This means that, for exposures to tested commercial PCB products that have not been significantly weathered, there is little uncertainty related to multiple toxic mechanisms or interactions among congeners or other co-contaminants formed in the PCB manufacturing process. Also, there is a large ecotoxicological database for Aroclor effects.

The main uncertainties of Aroclor-based risk assessment are related to the changes in congener composition following release to the environment (weathering and bioaccumulation), which can affect measurements of PCB levels and estimations of risk. Various methods have been used to determine the amount of PCBs in a sample as a concentration of an Aroclor or a mix of Aroclors (summarized in Eisler and Belisle 1996). Uncertainty is introduced because the congener composition of environmental samples may differ from that of any particular Aroclor or combinations of Aroclors, which results in larger variability in analytical results between laboratories than is usual for other chemical analyses. In formal terms, measurement error is larger for Aroclor analyses compared to congener-specific analyses.

Changes in congener patterns also can affect toxicity. Loss of lower chlorinated congeners to volatilization or degradation can increase the proportional dioxin-like toxicity of the remaining PCBs because many of the dioxin-like congeners are persistent. Anaerobic degradation may reduce toxicity due to higher chlorinated dioxin-like congeners, although the products may also be toxic (e.g., Ganey, et al. 2000). Foodchain transfers may increase the toxicity of the PCBs retained in organisms (see references in Lugwig, et al. 1996). For example, the biomagnification factors (BMF) for dioxin-like congeners are twice as high as the BMFs for total PCBs in zooplankton or *Mysis* (a freshwater invertebrate) feeding on phytoplankton, or *Diporeia* (another invertebrate) feeding on *Mysis* (Trowbridge and Swackhamer 2002). This preferential biomagnification increases the toxicity of the PCBs in the organism relative to the source PCBs because of the increased proportion of dioxin-like congeners accumulated in their tissues. Since the organisms in this example are representative of the base of an aquatic foodchain, the altered pattern with increased toxicity will be passed to animals feeding on zooplankton or aquatic invertebrates. This is evident in one study of animals that feed on plankton, the sediment-to-biota BMF for bioassayed TEQ was 10 times greater than the BMF for PCBs (Jones, et al. 1993). There is inconsistent evidence for preferential biomagnification of dioxin-like congeners by piscivorous (fish-eating) fish (Jones, et al. 1993; Metcalfe and Metcalfe 1997), but marked preferential biomagnification of dioxin-like congeners has been reported in some studies of piscivorous birds (gulls and cormorants) and mammals (otters) (Koslowski, et al. 1994; Guruge and Tanabe 1997; Leonards, et al. 1997). In general, risk assessments based on the original source Aroclor are likely to underestimate the risk of bioaccumulated PCBs (Ludwig, et al. 1996; Giesy and Kannan 1998).

Another potential source of uncertainty in Aroclor-based assessments is that total risk in the field may be underestimated because the approach does not readily allow for combined assessment of the effects of PCBs and additional contaminants with the same toxicological mode of action. For example, contributions to dioxin-like toxicity may be made by dioxins, polychlorinated dibenzofurans, and other chemicals in addition to PCBs. The source of the additional chemicals may be from the same facility that released PCBs or from separate sources (either local or distant through atmospheric transport). Regardless of the sources, the presence of additional chemicals with dioxin-like activity in the field reduces the amount of PCB exposure that can be tolerated by

wildlife in comparison to controlled exposures to commercial PCB products in captive animals not simultaneously exposed to additional dioxin-like chemicals.

6.2.1.2 Dioxin Toxic Equivalent-based Risk Assessment

Another approach for assessing the risks of PCBs is based on the total dioxin-like effects (TEQ), either calculated from congener-specific analytical data or measured by *in vitro* bioassays. Some advantages of these approaches are that they are not subject to the analytical uncertainties related to the potential mismatches between Aroclor standards and weathered PCBs, they facilitate assessment of the combined toxicity of dioxin-like PCB congeners and other dioxin-like contaminants, and TRVs can be based on studies of any chemical with dioxin-like toxicity when the results are given as TEQ (in contrast to Aroclor-specific results, which can not be generalized to other dioxin-like chemicals).

The main uncertainties associated with the currently available TEQ approaches for risk assessments are related to the methods used to determine the TEQ, and the potential significance of non-dioxin-like effects.

One TEQ approach is based on congener-specific analytical data in which the concentration of each dioxin-like congener is multiplied by its toxic equivalency factor (TEF), the fractional toxicity of that congener compared to 2,3,7,8-TCDD, which are summed for all dioxin-like congeners to give the toxic equivalent concentration (TEQ). By this approach, TEQ represents the concentration of the most toxic dioxin congener that is expected to equal the potency of the mix of PCB congeners in the sample. The approach permits inclusion of additional chemicals with dioxin-like potency such as polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans.

An obvious source of uncertainty are the TEF values. The current consensus TEFs are "order of magnitude estimates of the toxicity of a compound relative to TCDD" based on a tiered evaluation of the relative potencies (REPs) reported in a variety of studies (Van den Berg 1998). The order of magnitude estimate is an "illustration of the overall uncertainty in TEF values based on the differences in outcomes of the different end points and the variation in available data for the different congeners" (van Leeuwen 1999). Another indication of TEF uncertainty is the difference in TEF schemes by different groups and at different times, which also limits the usability and comparability of TEQ studies unless the full congener data were reported so that results can be converted to a common basis (Dyke and Stratford 2002). Another source of uncertainty is the additivity assumption in the TEQ calculation. Although dose additivity is supported by many studies (Van den Berg 1998), non-additive interactions also are reported. These uncertainties are believed to be less than the level of uncertainty associated with Aroclor-based assessments, supported by examples of good correlations in practice between TEQs and toxic effects (Van den Berg 1998; van Leeuwen 1999; Birnbaum 1999; Tillitt 1999), however, caution has also been expressed for the use of the TEF approach for PCBs based on "nonadditive interactions, coupled with the unusually broad range of TEF values observed for some PCB congeners" (Safe 1998). An uncertainty related to analytical issues is that most of the dioxin-like PCB congeners occur in very low concentrations, which means that measurement errors of congeners with high TEF values will be magnified in TEQ calculations. An extreme example in a recent study is unuseable analytical data for congener 126 due to interference (Trowbridge and Swackhamer 2002). Since congener 126 is often one of the greatest contributors to the TEQ of PCBs, the calculated TEQs of this study are underestimated and inappropriate for risk assessment

purposes.[14] Since the TEFs for different dioxin-like congeners vary by several orders of magnitude, small measurement errors for highly potent congeners can result in large errors in TEQ calculations. Another uncertainty is that TEFs are not presently available for all chemicals with potential dioxin-like activity, although TEFs are available for the ones shown to account for the majority of the dioxin-like toxicity in intact animals.

Another approach for determining TEQs is by *in vitro* bioassays, in which the response of cultured cell lines exposed to dioxin-like chemicals is measured. An advantage of the bioassay approach is that it provides an integrated measure of the effects of all the chemicals in a mixture that affect dioxin-like responses with all of their interactions (additive, synergistic, and antagonistic). Interactions can occur between dioxin-like chemicals or with non-dioxin-like chemicals that modulate dioxin-like responses. The main uncertainties are related to interspecific differences in cell responses, and issues involved in extrapolation of effects in isolated cells to intact animals. Cells of different species show differences in interactive effects between PCB congeners. For example, at high doses, PCB congener 52, one of the di-*ortho*-substituted congeners [15], inhibits cellular responses to dioxin or dioxin-like PCB congeners in bioassays performed with mouse and rat cell lines, but not with guinea pig or human cell lines (Aarts, et al. 1995). This means that the presence of di-*ortho*-substituted congeners in Aroclors may reduce the TEQ measured in bioassays performed with cultured mouse or rat cell lines (reportedly by as much as 2 orders of magnitude in comparison with a calculated TEQ that assumes additivity, see references in Aarts, et al. 1995), but not in bioassays performed with cultured guinea pig or human cell lines. In addition to measurement uncertainties related to interspecific differences in cellular responses, there are uncertainties related to extrapolation of *in vitro* responses of isolated cell cultures to *in vivo* [16] responses of intact animals. One of the advantages of bioassays–an integrated response to direct administration of complex environmental mixtures to cells–also introduces uncertainty because the dosing does not reflect the pharmacokinetics [17] in intact animals. Although many chemicals are capable of binding with the Ah receptor, their ability to cause dioxin-like toxicity also depends on their pharmacokinetic behavior, for example, how rapidly they are metabolized (degraded) (Birnbaum 1999) or distribution patterns within an animal (for examples of species differences in PCB distribution among organs see Bachour, et al. 1998). *In vitro* bioassays may therefore show responses to chemicals that have little or no effect in intact animals.

> "In summary, a single *in vitro* assay based on a single surrogate species may not accurately predict the toxicity of a chemical or complex mixture following exposure to other species.

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[14] The purpose of this particular study was to investigate the transfer of PCB congeners through selected trophic levels in an aquatic ecosystem, for which the loss of data for a single dioxin-like congener is not crucial. However, a similar data gap would be unacceptable for a risk assessment.

[15] Di-*ortho*-substituted congeners have 2 chlorine atoms attached in the positions closest to the bond that holds the biphenyl "frame" together, with variable numbers of chlorines attached at other positions. The 2 *ortho* chlorines prevent these congeners from taking on the planar configuration necessary for activating the Ah receptor, and therefore they do not exhibit dioxin-like toxicity, but, at high concentrations, inhibit the Ah receptor (with varying efficiency in different species) so that it becomes less responsive to dioxin-like congeners.

[16] *In vivo* means "in the living", and refers to experiments performed with intact living organisms.

[17] Pharmacokinetics refer to the rates of various processes that affect the movement and form of chemicals in living organisms including uptake, distribution, binding, biotransformation, and elimination.

> Nevertheless, the use of *in vitro* assays provides a general tool as a prescreening method of TEQs in environmental samples. However, it does not replace *in vivo* experiments when determining TEFs for dioxinlike compounds." (Van den Berg, et al. 1998).

Another source of uncertainty for TEQ-based risk assessments is that the current approach does not include non-dioxin-like toxicity (by definition). Non-dioxin-like toxicity, that is, toxic effects not mediated by the Ah receptor, may be induced by non-coplanar PCB congeners (Fisher, et al. 1998), or biotransformed PCB products such as hydroxylated metabolites (Schuur, et al. 1998) or methylsulfonyl metabolites (Johansson, et al. 1998). The uncertainty would be low if the thresholds for non-dioxin-like effects are lower than for dioxin-like effects, in which case assessments based on dioxin-like effects would be protective for all adverse effects. A comparison of the available data on non-AhR-mediated neurotoxicity [18] and dioxin-like effects in wildlife indicated that the dioxin-like effects are more sensitive endpoints (Giesy and Kannan 1998). Although encouraging, the comparison is provisional because the neurotoxic effects are not as well studied as dioxin-like effects, non-dioxin-like effects include endpoints other than neural effects, and some endpoints may be affected through both AhR-mediated and non-dioxin-like pathways. For example, thyroid function may be affected by both pathways. In one study, the relative potency of different extracts in depressing serum levels of thyroxine (the main thyroid hormone) in rats was not well predicted by TEQ. An air extract proportionally enriched in lower chlorinated congeners and depleted in higher chlorinated congeners, dioxins, and dibenzofurans, exhibited more severe effects on thyroxine levels at the same TEQ concentrations as soil or dust extracts with the converse congener compositions (Figure 2A in Li and Hansen 1996). Although in most situations, TEQ-based assessments show good correlations with toxic effects and appear to provide an adequate margin of safety for non-dioxin-like effects as well, the potential for non-dioxin-like processes remains an uncertainty until our understanding of non-AhR-mediated processes improves.

> "The spectrum of activity produced by [non-coplanar] congeners has not been fully explored, and the mechanisms by which their known actions are produced are emerging but remain to be fully elucidated. The toxicodynamic interactions between non-coplanar PCBs and the actions produced by coplanar PCBs which bind to the Ah-receptor remain to be investigated. Similarly, the actions and interactions of hydroxylated and other metabolites of PCBs remain to be studied in sufficient depth. At the present time, it is clear that non-coplanar PCBs alter signal transduction pathways and interrupt intracellular Ca^{2+} homeostasis. A common site of action responsible for all of the actions of non-coplanar PCBs, analogous to the Ah-receptor utilized by coplanar PCBs, has not been found ..." (Fisher, et al. 1998).

In summary, the two major approaches for PCB risk assessment have converse strengths and uncertainties. For Aroclor-based approaches, uncertainties are low for interactions between congeners and multiple toxic mechanisms, but uncertainties increase as the congener composition of environmental samples is altered from the original Aroclor composition by weathering or bioaccumulation. The Aroclor approach does not readily allow for assessment of combined risk of PCBs and other chemicals with dioxin-like toxicity. For the currently available TEQ-based approaches, results are not affected by weathering, but uncertainties are associated with TEF values and additivity assumptions for calculated TEQs, interspecific differences in cellular responses and *in*

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[18] The situation is complicated by possible neurotoxicity caused by dioxin-like congeners as well as non-dioxin-like congeners.

vitro to *in vivo* extrapolations for bioassay TEQs, and an inability to account for non-dioxin-like effects. The TEQ approaches facilitate assessment of combined risk of PCBs and other chemicals with dioxin-like toxicity, although uncertainty remains for calculated TEQs by the limited number of consensus TEFs (risks may be underestimated due to dioxin-like chemicals without TEFs), and for bioassay TEQs by toxicokinetic considerations (risks may be overestimated by cellular responses to chemicals that would not cause toxicity in intact animals).

6.2.2 Interspecific Extrapolation and Laboratory-to-Field Extrapolation

Extrapolation of toxicity data from tested species to wildlife is another source of uncertainty in TRVs that includes two categories–extrapolations between different species, and extrapolations from laboratory conditions (captivity) to field conditions.[19] There is no interspecific extrapolation for mink because the TRVs are based on studies of captive mink, but the difference between conditions in captivity and in the wild is a source of uncertainty. Both categories of uncertainty pertain to the bird TRVs, which are based on studies of captive chicken.

Captive animals are well fed, do not have to compete for resources, are less active, usually protected from weather extremes, and in general are subject to less stress compared to wild animals.[20] The toxicity of a tested chemical is often greater in stressed animals, for example, in a review of fish toxicity, nutritional status altered the relative toxicity between laboratory and field situations by as much as 10-fold, and temperature stress by as much as 100-fold (Heugens, et al. 2001). Stressor interactions are often nonlinear, complicating their assessment (Power 1997), and may involve complex interactions. The adverse effects of PCBs on stress responses were increased by poor nutritional status (Quabius, et al. 2000), which implies that a synergistic interaction of PCB exposure and nutritional stress could decrease the capability to respond to additional stressors. Kammenga, et al. (2001) discuss examples in which exposure to toxic substances increases sensitivity to other environmental variables such that the exposed population becomes more vulnerable to changes in these other variables than to the direct toxicant effects. Another difference between captive and wild animals is that wild animals are exposed to a wider variety of toxic chemicals. In addition to interactions between stresses due to chemicals with different toxicological actions, wild animals may be exposed to chemicals that act though the same toxicological mechanisms as the chemical of concern, thereby increasing the toxicity of a given level of exposure compared to captive animals with controlled exposures. Other endpoints might be more sensitive or result in greater overall impact in the field compared to the endpoints studied under controlled conditions (Section 6.1.6). Related to this, laboratory studies are usually not performed over an entire life cycle, and effects in the field may differ from those in laboratory studies because of cumulative effects, greater sensitivity at other developmental or life stages than the ones investigated, or interactions between generations (for example, impaired parental care).

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[19] Another source of uncertainty for risk assessment involves the exposure assumptions. This is not addressed here because it does not affect the TRV values. For example, risk in the field may differ from modeled risk because the wildlife are feeding on a different mix of food items or in other locations than assumed in the model that results in differences between field and modeled exposures. However, exposure uncertainty concerns whether the TRVs have been or are likely to be exceeded, not the particular values of the TRVs.

[20] This may not hold for species that can not tolerate captivity, that is, the stress of being confined may outweigh the reduced stress of being cared for, but species intolerant of captivity can not be used for toxicity testing.

An example of greater adverse effects in a field study than expected from laboratory studies on related species is the high sensitivity of wood ducks to egg TEQ concentrations in the field–significant reductions in hatchability and live duckling production occurred at egg TEQs of 20-50 ppt (White and Seginak 1994; White and Hoffman 1995), which are comparable to the sensitivity of chicken–onset of embryonic mortality and deformities at 10-20 ppt dioxin egg concentration (Verrett 1976 as cited in Hoffman, et al. 1996), and LD_{50} (lethal dose to 50 % of embryos) of 122-297 ppt (Henshel, et al. 1997). This outcome would not be expected on the basis of laboratory studies with other ducks, which show much less sensitivity to PCBs compared to chicken–LD_{50} of 3-40 ppb congener 77 (one of the dioxin-like congeners) in chicken eggs, but no effects in mallard or goldeneye duck eggs at 5000 ppb congener 77 (various studies, see Table 3 in Hoffman, et al. 1996); and reduced hatchability at less than 1 ppm A1242 in chicken eggs, but no effects on hatchability at 105 ppm A1242 in mallard eggs (various studies, see Table 2 in Hoffman, et al. 1996). Based on these laboratory comparisons, ducks are at least 100 times less sensitive than chicken to PCBs and dioxin-like effects. The unexpected sensitivity of wood ducks in the field may have occurred because of differences among duck species (wood duck may be orders of magnitude more sensitive than mallard or goldeneye), unmeasured co-contaminant exposure contributing to toxicity in the field, stressor interactions not present in captivity, or exposure duration effects. Another example involves adverse effects on terns in the Great Lakes (see discussion in Hoffman, et al. 1998).

The sensitivity of different bird species to PCBs spans several orders of magnitude, and chicken are the most sensitive of the species tested to date (Bosveld and Van den Berg 1994; Barron, et al. 1995; Eisler and Belisle 1996; Hoffman, et al. 1996 and 1998). Use of chicken-based TRVs is inappropriate when species-specific toxicity data are available, and is generally considered inappropriate when data are available for closely related species (although the available toxicity data for ducks poorly predicted field effects for wood duck). The chicken-based PCB TRVs are recommended as a conservative estimator of risk for birds of unknown sensitivity to PCBs. Since chicken are more sensitive than other bird species tested so far, the likelihood of chicken TRVs under predicting risk for other species of unknown sensitivity is probably low, therefore use of uncertainty factors for interspecific extrapolation is not recommended. Although the same rationale indicates that chicken data for PCB toxicity is likely to overestimate risks to PCBs for other bird species, the wood duck example shows that this is not certain–the margin between laboratory effect levels in chicken and field effect levels in other species may be unexpectedly small. Also, PCB or dioxin toxicity has been studied in a relatively small number of bird species under controlled conditions. While the extremes of sensitivity are known to widely diverge, the overall distribution of species sensitivities within this range is poorly known.

The degree of conservatism of applying unmodified chicken-based PCB TRVs to species of unknown sensitivity can be evaluated by comparison to the bird PCB TRV used in the Great Lakes Initiative (GLI) for deriving water quality criteria for the protection of wildlife (USEPA 1995a). The GLI PCB TRV for birds is based on a LOAEL of 1.8 mg/kg_{BW}-d in pheasant (Dahlgren, et al. 1972), which was divided by an interspecific extrapolation uncertainty factor of 3 and a LOAEL to NOAEL uncertainty factor of 3. Therefore the calculated LOAEL for species of unknown sensitivity was 0.6 mg/kg_{BW}-d and the NOAEL 0.2 mg/kg_{BW}-d (only the NOAEL was used for deriving the water quality criteria). These values bracket the recommended TRVs of 0.4 to 0.5 mg/kg_{BW}-d based on chicken PCB TRVs without uncertainty factors. This comparison demonstrates that the conservatism of chicken-based PCB TRVs is consistent with that of previous agency practice for determining environmental PCB limits for protection of wildlife.

In summary, the bird TRVs proposed in this effort provide an appropriate level of conservatism for estimating risk to species of unknown sensitivity to PCBs. The TRVs are unlikely to underestimate risk. By design, they are more likely to overestimate risk, which is a necessary bias for accounting for the uncertainty regarding the sensitivity of untested species. Although interspecific differences in PCB sensitivity span several orders of magnitude, indicating potentially large uncertainty in assessing risk to untested species, the degree of conservatism associated with the TRVs in the present effort is consistent with prior agency practice.

There is no interspecific extrapolation for the mink TRVs, but uncertainty is associated with laboratory to field extrapolation. The uncertainty of laboratory to field extrapolations is that potential effects are more likely to be underestimated, rather than overestimated, for the various reasons discussed above. For Aroclor-based risk estimates in particular, a common observation is that toxicity is underestimated. This may be due to preferential biomagnification of toxic congeners that increase toxicity compared to the source Aroclor; exposure to other contaminants that either act through the same toxicological mechanisms as PCBs, thereby decreasing the amount of PCB exposure that can be tolerated without adverse effects, or acting as separate but additional stressors; or other non-chemical stressor interactions. These sources of uncertainty are addressed by the recommendation to use the lower of the derived TRVs.

As discussed in Section 6.1.5, the recommended mink TRVs are reasonably consistent with the value used by the GLI for calculating water quality criteria for protection of wildlife.

7. Conclusions

This effort demonstrates that toxicity reference values (TRVs) can be successfully derived through evaluation of dose-response plots in which data are aggregated from multiple studies by normalizing the treatment responses by the respective control responses of each study. The combined data sets better define the shape of dose-response relationship by increasing the number of doses plotted, thereby providing more information for decision-making compared to statistically-defined no or lowest observed adverse effect levels (NOAELs or LOAELs), which are influenced by multiple factors unrelated to toxicity and do not provide dose-response information. Although uncertainties may be introduced by differences in the experimental protocols of the various studies that are combined, such as differences in exposure duration or route, significant effects are readily apparent as inconsistencies in the dose-response plots.

The results of this exercise show that dose-response plots are not highly sensitive to moderate differences in exposure duration. The few differences in exposure route among the aggregated studies also did not result in obvious distortions of dose-response relationships (contaminated food vs. contaminated water, or egg injection vs. maternal transfer to eggs). In the cases in which dose-response inconsistencies are apparent between study results, the data can be stratified (considered separately) for analysis if multiple patterns are evident, or that endpoint can be dropped from further consideration if the data exhibit no interpretable pattern. In other words, the dose-response plots provide their own safeguard against utilization of incompatible data by exhibiting divergent patterns or uninterpretable relationships inconsistent with known toxicological models.

The dose-response plots exhibit very steep transitions between PCB exposures causing no adverse effects and those resulting in severe adversity–mostly less than 2- or 3-fold gradients in dose or dietary concentration

between the response extremes. This has two implications: 1) small exceedances of PCB TRVs are likely to result in severe effects on reproductive success, and 2) the calculated PCB TRVs are relatively insensitive to the choice of effect size (the percent decrease in response that is of concern for risk management) because the range of values over which the TRVs can vary is narrow.

Two significant observations can be made from the dose-response plots for mink (actually dietary concentration-response plots). 1) PCBs exhibit a hormetic effect (enhanced reproductive performance) at doses lower than the threshold for adverse effects for the number of live kits produced per mated female in feeding trials performed with either commercial PCB products or field-contaminated prey. 2) In both commercial PCB product (Clophen A50) and field-contaminated prey studies with mink, the exposure-response relationships differ between studies performed over a single breeding season versus those in which exposures are continued over 2 breeding seasons or 2 generations of female mink. Continuous PCB exposure over 2 breeding seasons or 2 generations of female mink results in more severe adverse effects on live kit production, kit survival, and, to a lesser extent, kit bodyweight, in comparison to the effects of exposure over a single breeding season. The mean difference in low effect TRVs for the various endpoints in the two studies is a 50 % decrease associated with 2-breeding season or generation exposures as compared to single-breeding season exposure. This has obvious implications for long-term sustainability of mink at contaminated sites. Since 2-breeding season or generation studies have not been performed with Aroclors, the mink Aroclor TRVs are adjusted by the mean response decrement observed in the Clophen and field-contaminated studies to ensure long-term sustainability.

TRVs based on controlled exposures to Aroclors are given in Table 1 (Section 1). The lower of the TRVs are recommended to account for increases in toxicity PCBs in the field compared to that of Aroclors under controlled conditions, which may be related to changes in source congener composition by weathering and bioaccumulation, concurrent exposure to other contaminants acting through the same toxicological mechanisms as PCBs (thereby reducing the tolerable exposure to PCBs), or interactions with other stressors (chemical, physical, or biological) not present in captivity. Uncertainty factors are not recommended for interspecific extrapolation because the TRVs are based on data for sensitive species.

Although the TRVs are conservatively derived (chicken are sensitive to PCBs, and mink values are adjusted for long-term exposures), the recommended values and level of conservatism are consistent with prior agency practice. Both the bird and mink TRVs are bracketed by the NOAEL and LOAEL values used in the development of PCB water quality criteria for the protection of wildlife by the Great Lakes Initiative. As such, the recommended TRVs represent a refinement of the toxicity information used for the GLI, and share a similar degree of conservatism in their application.

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Figure 1. Live Kits per Mated Female Mink Exposed to Commercial PCB Product for 1 Breeding Season

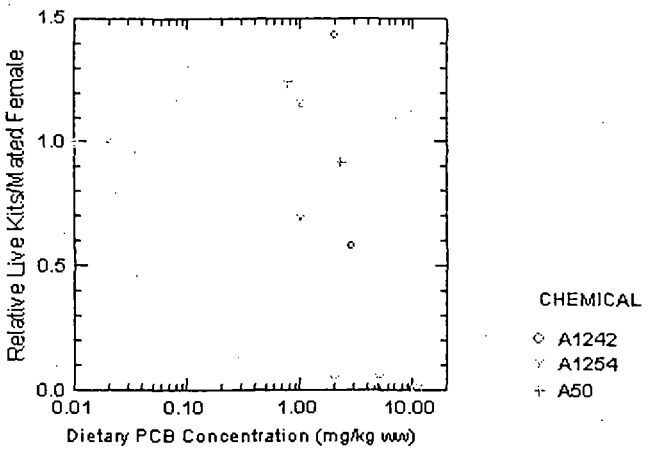
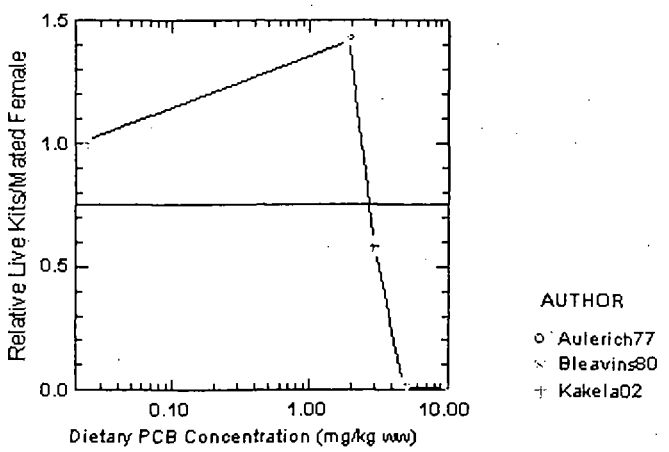


Figure 2. Live Kits per Mated Female Mink Exposed to Commercial Aroclor 1242 for 1 Breeding Season



Author is lead author and date. See notes to Table 3 for citations

Figure 3. Live Kits per Mated Female Mink Exposed to Commercial Aroclor 1254 for 1 Breeding Season

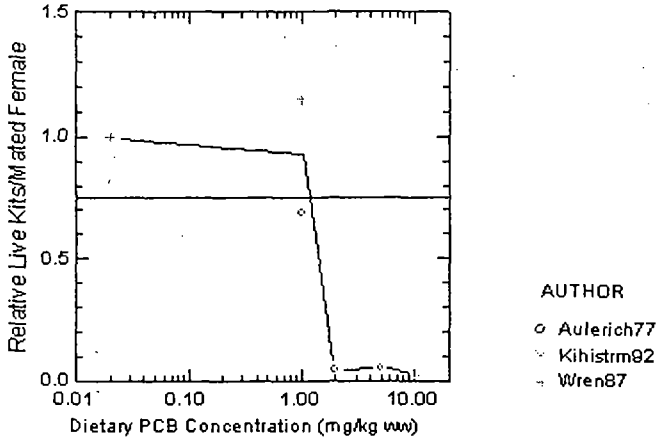


Figure 4. Mink Kit Bodyweight, Matemal Exposure to Commercial PCB Product for 1 Breeding Season

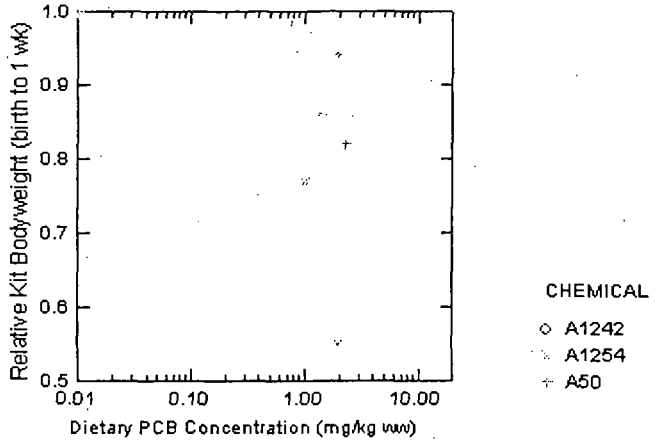


Figure 5. Mink Kit Bodyweight at Birth, Maternal Exposure to Commercial Aroclor 1254 for 1 Breeding Season

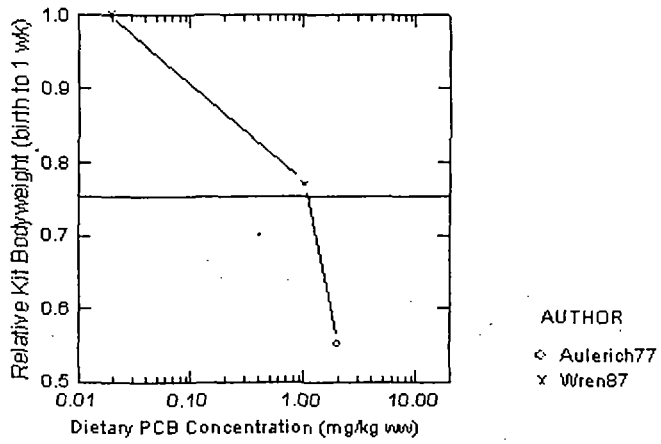


Figure 6. Mink Kit Survival, Maternal Exposure to Commercial Aroclor 1254 for 1 Breeding Season

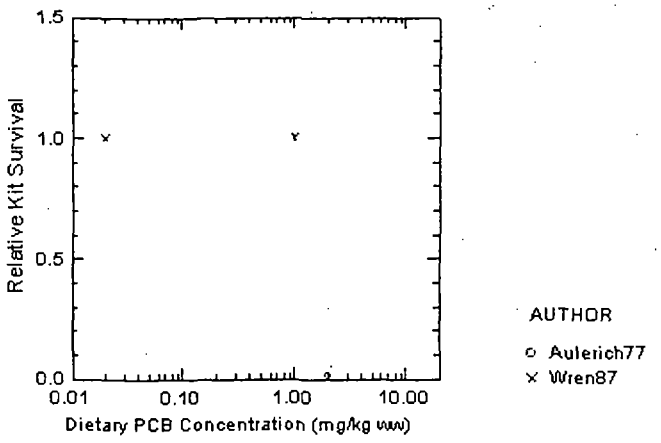


Figure 7. Live Kits per Mated Female Mink Exposed to Commercial Clophen A50 for Multiple Breeding Seasons (Brunström, et al. 2001; Kihlström, et al. 1992)

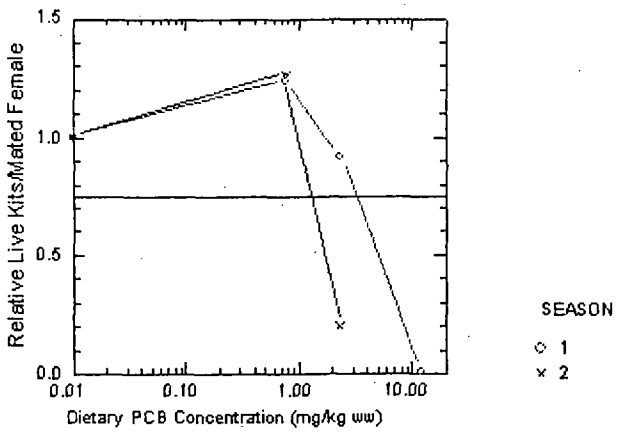


Figure 8. Live Kits per Mated Female Mink Exposed to Field-contaminated Fish for Multiple Breeding Seasons or Generations (Restum, et al. 1998)

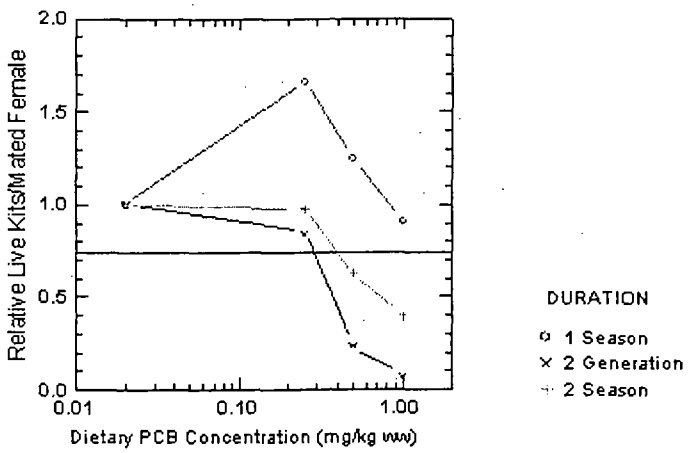


Figure 9. Mink Kit Bodyweight at Birth, Maternal Exposure to Commercial Clophen A50 for Multiple Breeding Seasons (Brunström, et al. 2001)

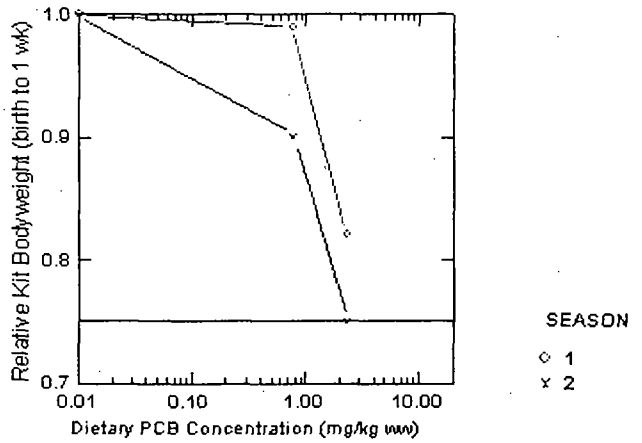


Figure 10. Mink Kit Bodyweight at Birth, Maternal Exposure to Field-contaminated Fish for Multiple Breeding Seasons or Generations (Restum, et al. 1998)

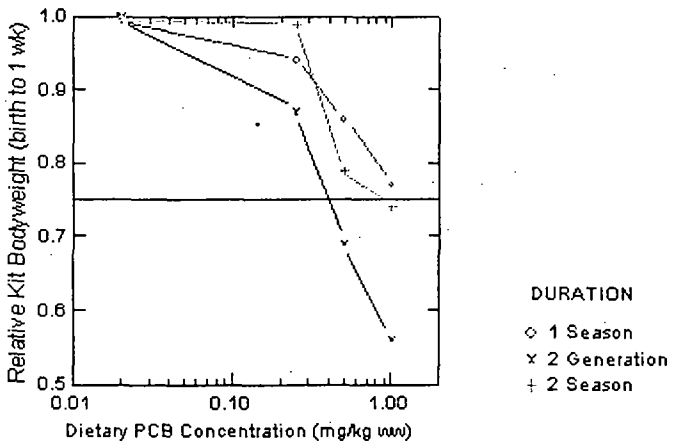


Figure 11. Mink Kit Survival, Maternal Exposure to Commercial Clophen A50 for 2 Breeding Seasons (Brunström, et al. 2001)

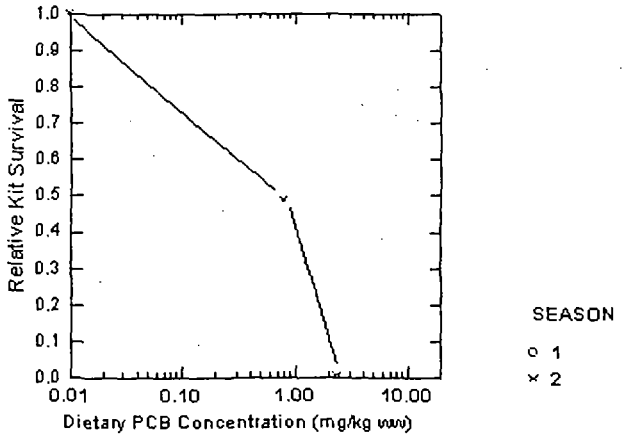


Figure 12. Mink Kit Survival, Maternal Exposure to Field-contaminated Fish for Multiple Breeding Seasons or Generations (Restum, et al. 1998)

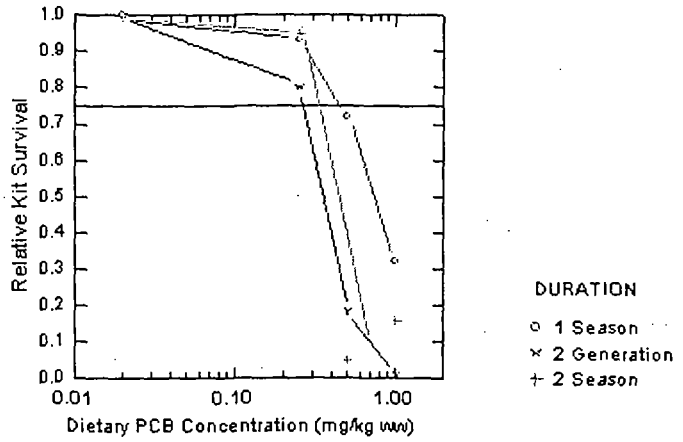


Figure 13. Live Kits per Mated Female Mink Exposed to Field-contaminated Prey for 1 Breeding Season

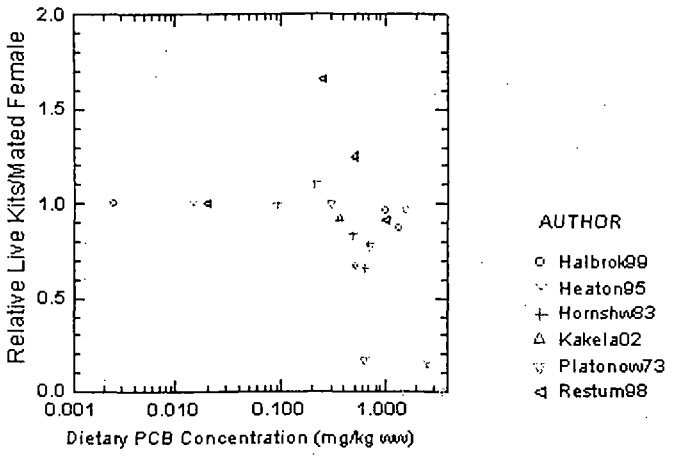


Figure 14. Mink Kit Bodyweight, Maternal Exposure to Field-contaminated Fish for 1 Breeding Season

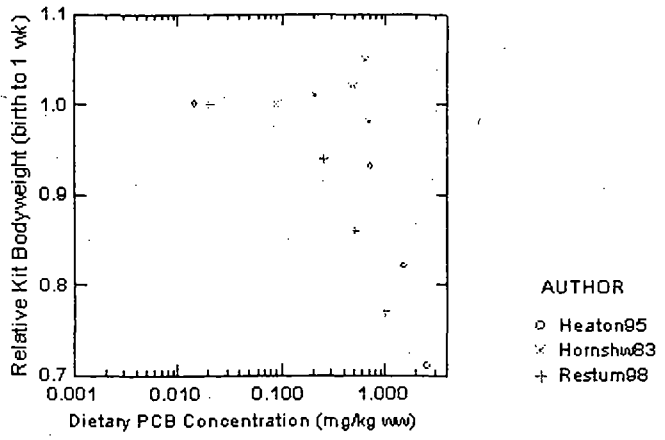


Figure 15. Mink Kit Survival, Maternal Exposure to Field-contaminated Prey for 1 Breeding Season

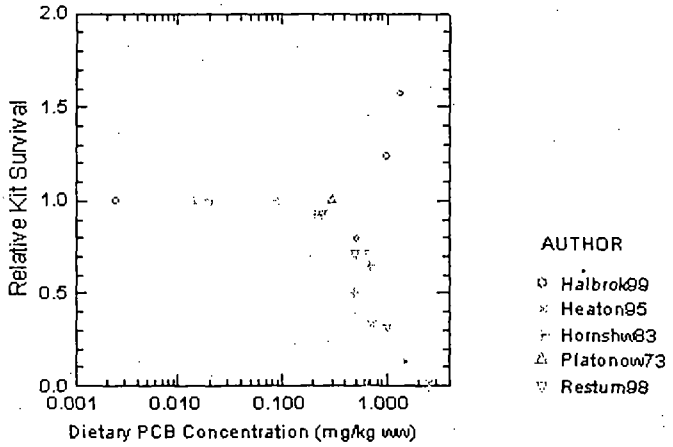


Figure 16. Comparison of Dose-response Relationships for Individual and Aggregated Studies of Hatchability vs. A1248 Dose to Hens

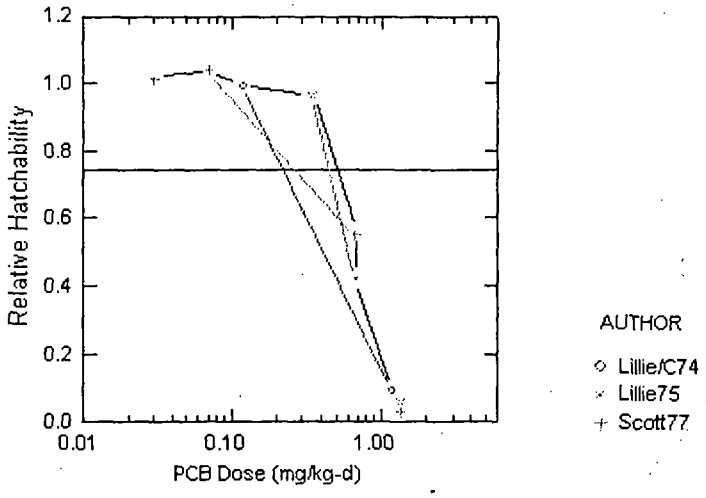


Figure 17. Hatchability, PCB Dose to Chicken Hens

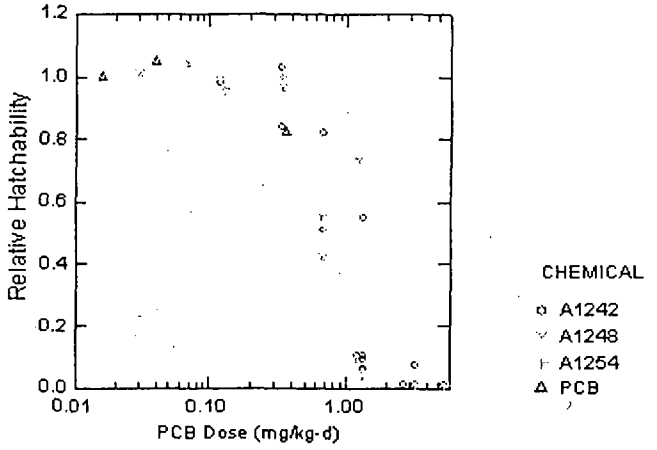
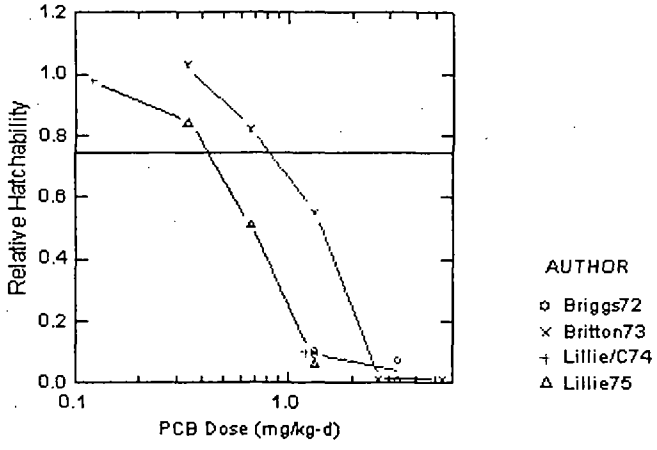


Figure 18. Hatchability, Aroclor1242 Dose to Chicken Hens



Author is lead author and date. See notes to Table 3 for citations.

Figure 19. Hatchability, Aroclor 1248 Dose to Chicken Hens

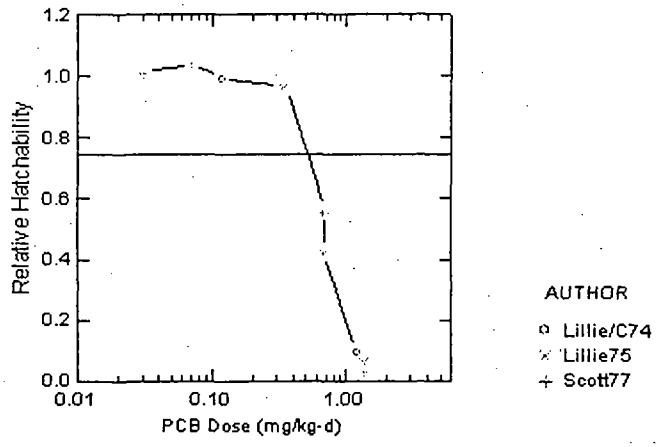


Figure 20. Hatchability, Aroclor 1254 Dose to Chicken Hens

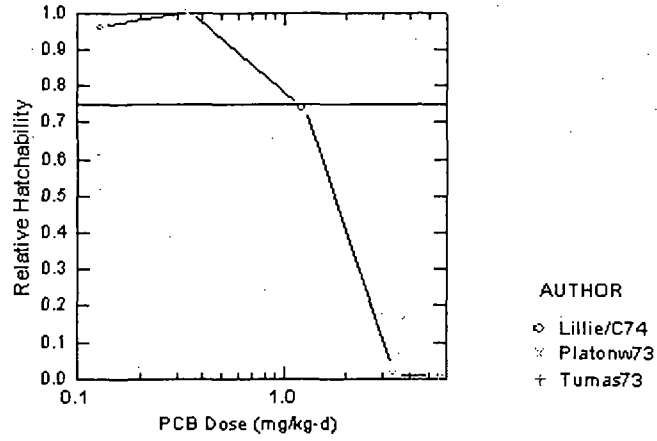


Figure 21. Hatchability, PCB Residues in Chicken Eggs

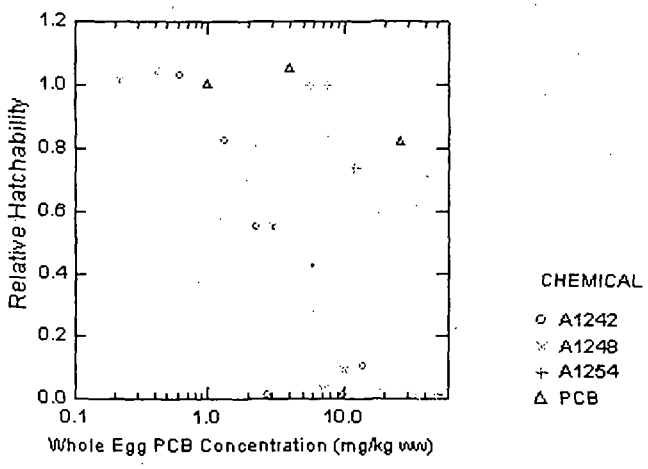


Figure 22. Hatchability, Aroclor 1242 Residues in Chicken Eggs

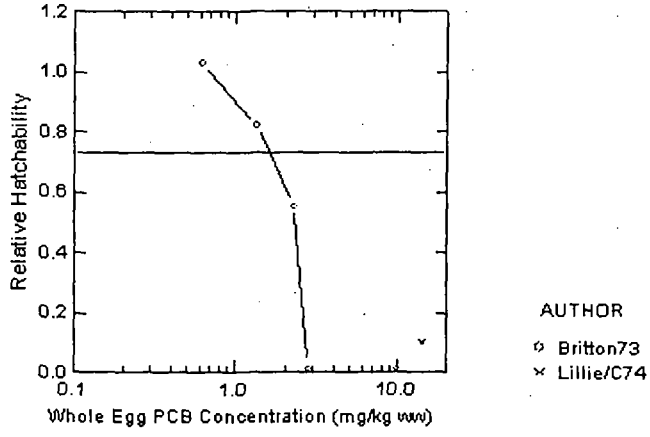


Figure 23. Hatchability, Aroclor 1248 Residues in Chicken Eggs

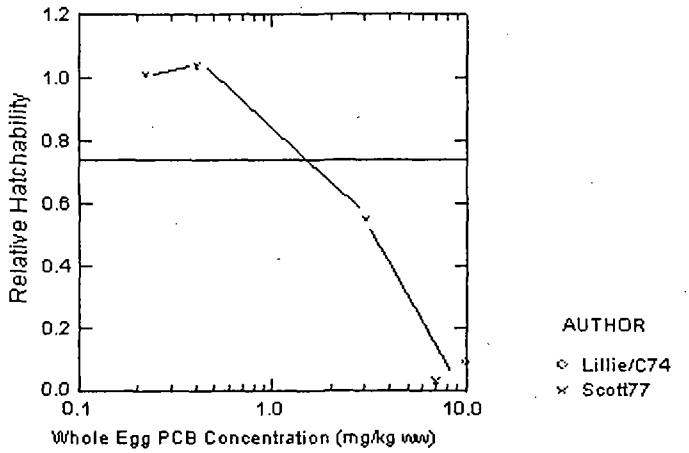


Figure 24. Hatchability, Aroclor 1254 Residues in Chicken Eggs

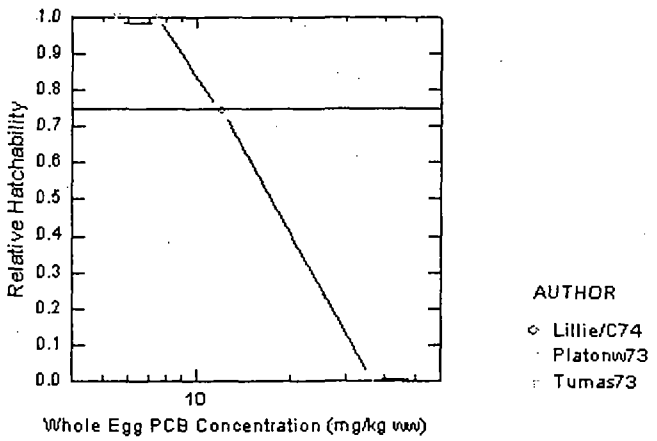


Figure 25. Chick Bodyweight, PCB Dose to Chicken Hens

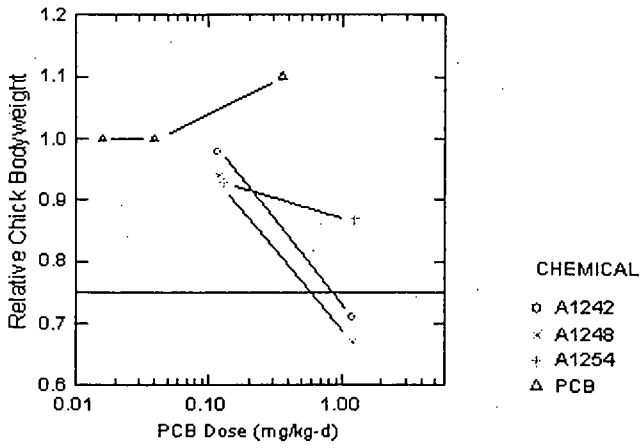


Figure 26. Chick Bodyweight, PCB Residues in Chicken Eggs

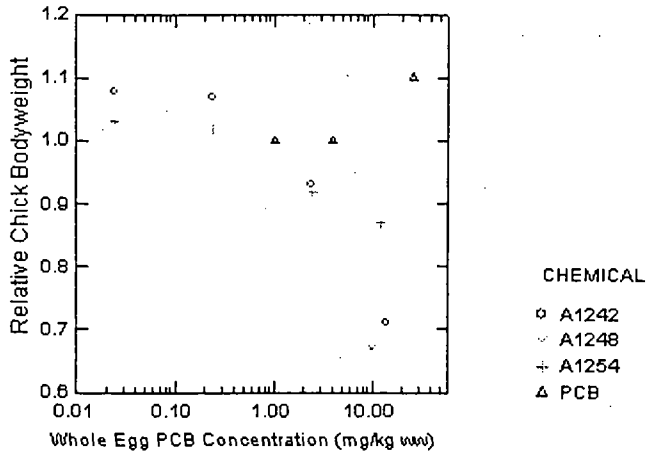


Figure 27. Chick Bodyweight, Aroclor 1242 Residues in Eggs

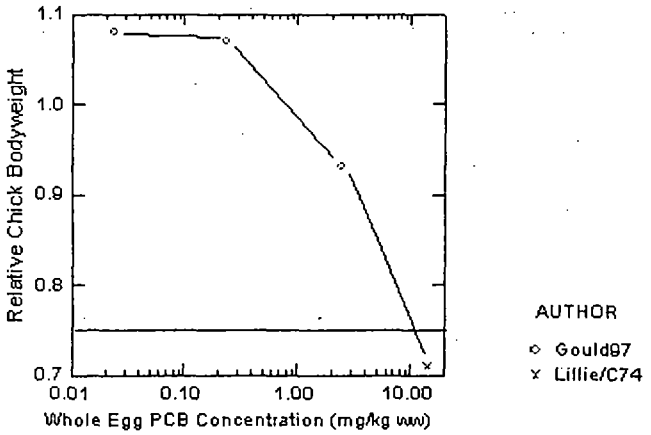


Figure 28. Chick Survival, PCB Dose to Chicken Hens

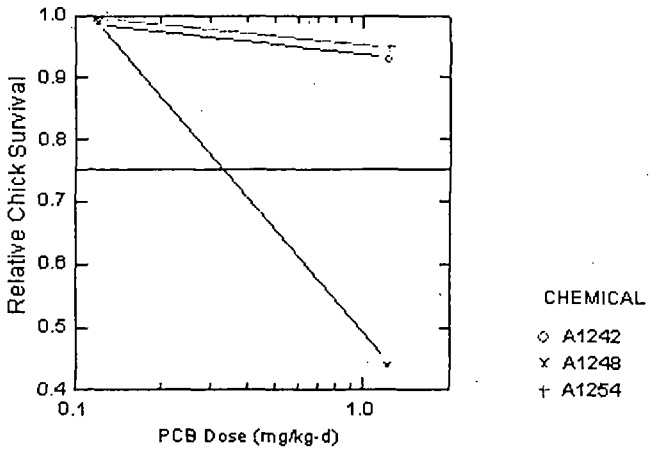


Figure 29. Egg Productivity, PCB Dose to Chicken Hens

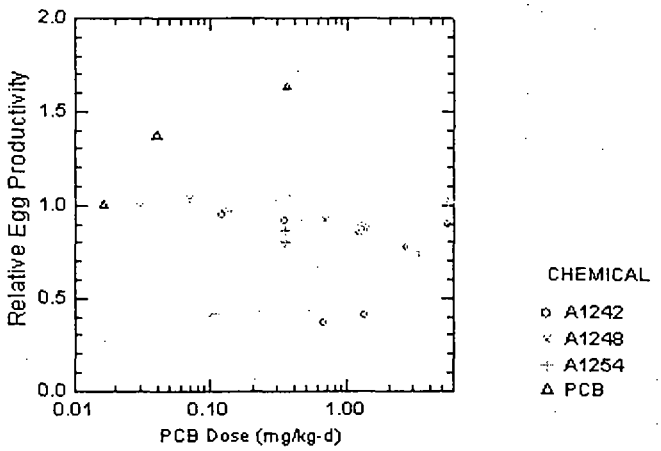


Figure 30. Egg Fertility, PCB Dose to Chicken Hens

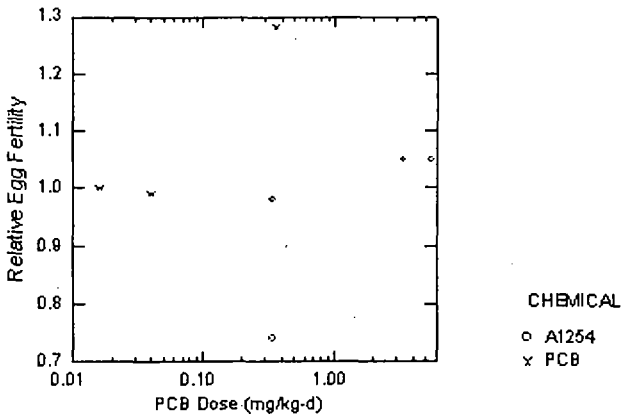


Figure 31. Chick Deformity, PCB Dose to Chicken Hens

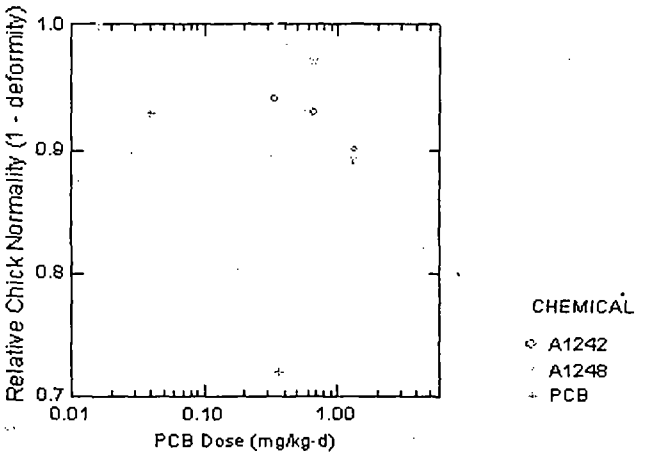


Figure 32. Chick Survival, PCB Residues in Chicken Eggs

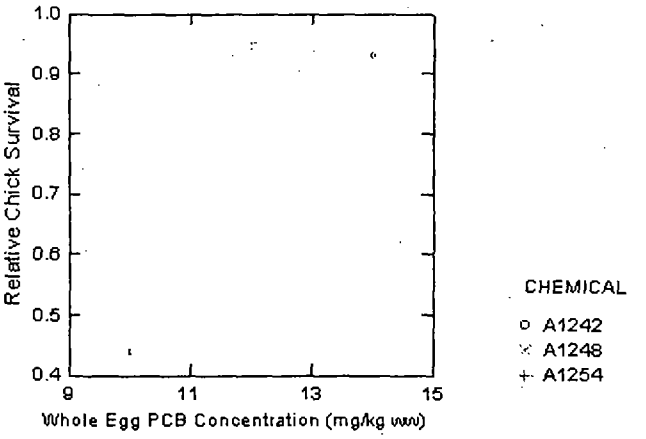


Table 2. Log-Linear Interpolation of PCB Toxicity Reference Values (TRV) for Mink

Chemical or Field study author	Response	Exposure Duration	Control RR	Treatment conc < TRV conc	Treatment conc < TRV RR	Treatment conc > TRV conc	Treatment conc > TRV RR	Target RR	P	TRV	Effect level	Study
			M_1	C_j	M_j	C_{j-1}	M_{j-1}					
Aroclor feeding studies												
A1242	live kit/ mated ♀	1 season	1	2	1.43	2.88	0.58	0.75		2.68	low effect	Aulerich77, Kakela02
			1	2	1.43	2.88	0.58	0.9		2.51	no effect	Aulerich77, Kakela02
A1254	live kit/ mated ♀	1 season	1	1	0.92	2	0.04	0.75		1.14	low effect	Wren87, Aulerich77
			1	1	0.92	2	0.04	0.9		1.02	no effect	Wren87, Aulerich77
A1254	kit bodywt	1 season	1	1	0.77	2	0.55	0.75		1.07	low effect	Wren87, Aulerich77
			1	0.02	1			0.9		>0.02	no effect	Wren87
A1254	kit survival	1 season	1	0.02	1	2	0	0.75		<1.00	low effect	Wren87, Aulerich77
			1	0.02	1			0.9		>0.02	no effect	Wren87
Comparison of 1 breeding season exposure vs 2 breeding seasons or generations continuous exposure												
A50	live kit/ mated ♀	1 season	1	2.31	0.92	12	0	0.75		3.13	low effect	Brunstm01, Kihistm92
		1 season	1	2.31	0.92	12	0	0.9		2.39	no effect	Brunstm01, Kihistm92
		2 season	1	0.77	1.27	2.31	0.2	0.75		1.31	low effect	Brunstm01
		2 season	1	0.77	1.27	2.31	0.2	0.9		1.13	no effect	Brunstm01
Ratio 2 season / 1 season			*0.47 no effect*									
Ratio 2 season / 1 season			*0.42 low effect*									
Restum	live kit/ mated ♀	1 season	1	1	0.91			0.75		>1.00	low effect	Restum98
		2 season	1	0.25	0.98	0.5	0.63	0.75		0.39	low effect	Restum98
		2 generation	1	0.25	0.84	0.5	0.23	0.75		0.28	low effect	Restum98
Ratio 2 season / 1 season			*<0.39 low effect*									
Ratio 2 generation / 1 season			*<0.28 low effect*									
Restum	kit bodywt	1 season	1	1	0.77			0.75		1.00	low effect	Restum98
		2 season	1	0.5	0.79	1	0.74	0.75		0.87	low effect	Restum98
		2 generation	1	0.25	0.87	0.5	0.69	0.75		0.40	low effect	Restum98
Ratio 2 season / 1 season			*0.87 low effect*									
Ratio 2 generation / 1 season			*0.40 low effect*									
Restum	kit survival	1 season	1	0.25	0.93	0.5	0.72	0.75		0.45	low effect	Restum98
		2 season	1	0.25	0.95	0.75	0.11	0.75		0.32	low effect	Restum98
		2 generation	1	0.25	0.8	0.5	0.18	0.75		0.26	low effect	Restum98
Ratio 2 season / 1 season			*0.72 low effect*									
Ratio 2 generation / 1 season			*0.58 low effect*									
Mean ratio 2 season or gen./ 1 season			*0.52 low effect*			(all studies)						

Notes for Table 2.

bodywt - bodyweight
conc - dietary concentration of PCBs (mg/kg wet weight (ww))
RR - relative response = treatment response / control response
Kit bodyweight is for birth to 1 week age.
TRV - toxicity reference value for dietary PCBs (mg/kg wet weight (ww))

$$\text{Log}_{10}\,TRV = \text{Log}_{10}\,C_j + (((M_1 * P) - M_j) * ((\text{Log}_{10}\,C_{j-1} - \text{Log}_{10}\,C_j) / (M_{j-1} - M_j)))$$

$$TRV = 10^{\text{Log}10\,TRV}$$

Study - lead author, date; see notes for Table 4 for citations

A1254 live kit/mated 1 season M_j of 0.92 is the mean of 1.15 (Wren87) and 0.69 (Aulerich77) both at 1 mg/kg dietary concentration.
Restum kit survival 2 season M_j of 0.11 at C_j of 0.75 are the means of 0.05 and 0.16 (M_j) at 0.5 and 1.0 (C_j), respectively.

Table 3. Log-Linear Interpolation of PCB Toxicity Reference Values (TRV) for Chicken

Chemical	Response	Control	Treatment dose < TRV		Treatment dose > TRV		Target	TRV	Effect level	Study
		RR	dose	RR	dose	RR	RR			
		M_1	D_j	M_j	D_{j-1}	M_{j-1}	P			
Hen Dose (mg/kg$_{BW}$-d)										
A1242	hatchability	1	0.67	0.82	1.34	0.55	0.75	0.80	low effect	Britton73
A1242	hatchability	1	0.34	1.03	0.67	0.82	0.9	0.52	no effect	Britton73
A1242	hatchability	1	0.34	0.84	0.67	0.51	0.75	0.41	low effect	Lillie75
A1242	hatchability	1	0.12	0.98	0.34	0.84	0.9	0.13	no effect	Lillie/Cecil74
A1242	chick bw	1	0.12	0.98	1.21	0.71	0.75	0.86	low effect	Lillie/Cecil74
A1242	chick bw	1	0.12	0.98	1.21	0.71	0.9	0.24	no effect	Lillie/Cecil74
A1248	hatchability	1	0.34	0.96	0.67	0.55	0.75	0.48	low effect	Lillie75; Scott77
A1248	hatchability	1	0.34	0.96	0.67	0.55	0.9	0.38	no effect	Lillie75; Scott77
A1248	chick bw	1	0.12	0.94	1.21	0.67	0.75	0.61	low effect	Lillie/Cecil74
A1248	chick bw	1	0.12	0.94	1.21	0.67	0.9	0.17	no effect	Lillie/Cecil74
A1248	survival	1	0.12	0.99	1.21	0.44	0.75	0.33	low effect	Lillie/Cecil74
A1248	survival	1	0.12	0.99	1.21	0.44	0.9	0.18	no effect	Lillie/Cecil74
A1254	hatchability	1	0.34	1	1.22	0.74	0.75	1.16	low effect	Platonw73; Lillie/Cecil74
A1254	hatchability	1	0.34	1	1.22	0.74	0.9	0.56	no effect	Platonw73; Lillie/Cecil74
Egg Concentration (mg/kg, ww)			conc		conc					
		M_1	C_j	M_j	C_{j+1}	M_{j+1}	P	TRV	Effect level	Study
A1242	hatchability	1	1.35	0.82	2.26	0.55	0.75	1.54	low effect	Britton73
A1242	hatchability	1	0.62	1.03	1.35	0.82	0.9	1.00	no effect	Britton73
A1242	chick bw	1	2.44	0.93	14	0.71	0.75	10.19	low effect	Gould97; Lillie/Cecil74
A1242	chick bw	1	2.44	0.93	14	0.71	0.9	3.10	no effect	Gould97; Lillie/Cecil74
A1248	hatchability	1	0.41	1.04	3	0.55	0.75	1.33	low effect	Scott77
A1248	hatchability	1	0.41	1.04	3	0.55	0.9	0.72	no effect	Scott77
A1254	hatchability	1	7.5	1	12	0.74	0.75	11.79	low effect	Platonw73; Lillie/Cecil74
A1254	hatchability	1	7.5	1	12	0.74	0.9	8.99	no effect	Platonw73; Lillie/Cecil74

Notes for Table 3.

bw - bodyweight
conc - whole egg PCB concentration, mg/kg, ww
dose - bodyweight-normalized ingestion, mg PCB/kg$_{BW}$-d
RR - relative response = treatment response / control response
Study - lead author, date; see notes for Table 5 for citations
TRV - toxicity reference value for PCB dose (D) (mg/kg$_{BW}$-d) or whole egg concentration (C) (mg/kg wet weight (ww))

$$\text{Log}_{10}\,TRV = \text{Log}_{10}\,D_j + (((M_1 * P) - M_j) * ((\text{Log}_{10}\,D_{j-1} - \text{Log}_{10}\,D_j) / (M_{j-1} - M_j)))$$

$$\text{Log}_{10}\,TRV = \text{Log}_{10}\,C_j + (((M_1 * P) - M_j) * ((\text{Log}_{10}\,C_{j-1} - \text{Log}_{10}\,C_j) / (M_{j-1} - M_j)))$$

$$TRV = 10^{\text{Log10 TRV}}$$

Table 4. Mink PCB Toxicity Studies

Ref	Exposure				Relative Response Compared to Control					
	Chemical & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
1	reported as A1254, from cow	5.2 month	0.64 mg/kg (control 0.3 mg/kg)	1.23 mg/kg liver (control 0.39 mg/kg); 0.97 mg/kg muscle (control 0.23 mg/kg)				0.17		0, 1 d
		3.4 month	3.6 mg/kg	11.99 mg/kg liver; 3.31 mg/kg muscle	0	0	0	0		
2	A1242 product	9.7 month	2 mg/kg (control NA)		1	1.37	1.43	1.43	0.94 birth	1.42 4 wk
	A1254 product	4.2 month	1 mg/kg (control NA)		0.8	0.90	0.86	0.69		
		9.7 month	2 mg/kg (control NA)		0.29	0.24	0.14	0.04	0.55 birth	0 4 wk
		4.2 month	5 mg/kg (control NA)		0.25	0.50	0.20	0.05		
3	NA (PCB type not identified)	2.2 month	3.3 mg/kg + 3.3 mg/kg DDT (control 0.05 mg/kg)	86 mg/kg fat (control 14 mg/kg)	0.79	0.57	0.20	0.17	0.72 birth	0.21 5 d

Ref	Exposure				Relative Response Compared to Control					
	Chemical & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
			11 mg/kg	280 mg/kg fat	0	0	0	0		
4	A1242 product	8.1 month	5 mg/kg (control NA)		0	0	0	0		
			10 mg/kg		0	0	0	0		
5	reported as A1254, Green Bay alewife	7 month	0.21 mg/kg (control 0.09 mg/kg)	8.1 mg/kg adipose (control 2.9 mg/kg)	0.92	1.15	1.26	1.11	1.01 birth 1.02 4 wk	0.93 4 wk
	L Michigan Whitefish	7 month	0.48 mg/kg	13 mg/kg adipose	0.89	0.91	0.95	0.84	1.02 birth 0.88 4 wk	0.51 4 wk
	Saginaw Bay sucker	7 month	0.63 mg/kg	10 mg/kg adipose	1.00	0.80	0.67	0.66	1.05 birth 0.91 4 wk	0.73 4 wk
	L Erie perch	7 month	0.69 mg/kg	13 mg/kg adipose	0.91	0.93	0.88	0.79	0.98 birth 0.80 4 wk	0.65 4 wk
	Saginaw Bay carp	7 month	1.5 mg/kg	37 mg/kg adipose	0.30	0.56	0	0		
	Erie perch & Saginaw wht sucker	7 month	0.66 mg/kg (control 0.04 mg/kg)		0.58	0.37	0.19	0.11	0.86 birth	0 4 wk

Ref	Exposure				Relative Response Compared to Control					
	Chemical & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
6	A1254 product	6.1 month	1 mg/kg (control 0.02 mg/kg)	2.8 mg/kg liver (control 0.09 mg/kg)	0.99	1.09	1.16	1.15	0.77 1 wk 0.75 3 wk, 0.71 5 wk	1.00 5 wk nearly all starvation (control 75 % trauma or infection, but no starvation)
7	Clophen A50	3 month	12 mg/kg	181 mg/kg fat 4.0 mg/kg muscle	0.11	0.12	0	0		
	A1254	3 month	10 mg/kg	74 mg/kg fat 1.3 mg/kg muscle	0.34	0.66	0	0		
8	PCB - sum of 1242, 1248, 1254, and 1260; TEQ - H4IIE bioassay; Saginaw carp	6 month	PCB 0.72 mg/kg (control 0.015 mg/kg); TEQ 19.4 pg/g (control 1 pg/g)	PCB 2.2 mg/kg liver (control 0.1 mg/kg) TEQ 495 pg/g (control <10 pg/g)	1.00	0.93	0.76	0.76	0.93 birth; 0.67 3 wk; 0.79 6 wk	0.33 6 wk
			PCB 1.53 mg/kg TEQ 40 pg/g	PCB 3.1 mg/kg liver TEQ 439 pg/g	1.00	1.02	0.96	0.96	0.82 birth; 0.67 3 wk 0.41 6 wk	0.13 6 wk

Ref	Exposure				Relative Response Compared to Control					
	Chemical & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
			PCB 2.56 mg/kg TEQ 80.8 pg/g	PCB 6.3 mg/kg liver TEQ 656 pg/g	1.00	0.58	0.14	0.14	0.71 birth	0 3 wk
9	PCB - sum of 1242, 1248, 1254, and 1260; TEQ - H4IIE bioassay; Saginaw carp	6 month (P_1 1992)	PCB 0.25 mg/kg (control 0.02 mg/kg) TEQ 7.1 pg/g (control 1 pg/g)		1.36	1.16	1.19	1.66	0.93-0.94 birth 0.75-0.89 3 wk 0.75-0.85 6 wk	 1.06 3 wk 0.93 6 wk
			PCB 0.5 mg/kg TEQ 13.6 pg/g		1.35	1.02	0.91	1.25	0.84-0.87 birth 0.67-0.75 3 wk 0.65-0.68 6 wk	 0.81 3 wk 0.72 6 wk
			PCB 1.0 mg/kg TEQ 26.4 pg/g		1.16	1.02	0.77	0.91	0.75-0.79 birth 0.51-0.59 3 wk 0.35-0.49 6 wk	 0.32 3 wk 0.32 6 wk
		16 month (P_1 1993)	PCB 0.25 mg/kg TEQ 7.1 pg/g	PCB 0.98 mg/kg liver (control 0.07 mg/kg)	1.02	0.95	0.96	0.98	0.88-1.09 birth 0.87-0.91 3 wk 0.92 6 wk	 0.99 3 wk 0.95 6 wk

Ref	Exposure				Relative Response Compared to Control					
	Chemical & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
			PCB 0.5 mg/kg TEQ 13.6 pg/g	PCB 0.89 mg/kg liver	0.78	0.92	0.80	0.63	0.77-0.81 birth 0.65-0.67 3 wk 0.93 6wk	 0.62 3 wk 0.05 6 wk
			PCB 1.0 mg/kg TEQ 26.4 pg/g	PCB 1.57 mg/kg liver	0.66	0.63	0.59	0.40	0.73-0.74 birth 0.50-0.59 3 wk 0.60-0.66 6 wk	 0.15 3 wk 0.16 6 wk
		12 month F_1 of 6-month exposed parents (F_1-1 1993)	PCB 0.25 mg/kg TEQ 7.1 pg/g	PCB 0.63 mg/kg liver (control 0.02 mg/kg)	0.85	1.05	0.96	0.84	0.87 birth 1.03-1.10 3 wk 0.89-0.95 6 wk	 0.76 3 wk 0.80 6 wk
			PCB 0.5 mg/kg TEQ 13.6 pg/g	PCB 0.96 mg/kg liver	0.76	0.88	0.31	0.23	0.64-0.73 birth 0.42 3 wk 0.54 6 wk	 0.16 3 wk 0.18 6 wk
			PCB 1.0 mg/kg TEQ 26.4 pg/g	1.47	0.63	0.53	0.09	0.07	0.51-0.60 birth	0 3 wk

Ref	Exposure				Relative Response Compared to Control					
	Chemical & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
10	reported as A1260 Poplar Creek & Clinch River fish	7 month	0.52 mg/kg (control <0.005 mg/kg)	<0.005 mg/kg liver (control <0.005); NA fat (control 3.2 mg/kg fat)	0.58	1.20	1.15	0.67	1.02 6 wk	0.79 6 wk
			1.01 mg/kg	<0.005 mg/kg liver; 105.86 mg/kg fat	0.87	0.92	1.10	0.96	0.94 6 wk	1.24
			1.36 mg/kg	7.25 mg/kg liver; 128.63 mg/kg fat	1.16	0.66	0.75	0.87	0.90 6 wk	1.57
11	Clophen A50 product; TEQ calculated by WHO TEFs	6 month	PCB 0.77 mg/kg (control 0.01 mg/kg) TEQ 22 pg/g		0.96	1.20	1.30	1.24	0.99 birth	
			PCB 2.31 mg/kg TEQ 65 pg/g		0.97	1.04	0.95	0.92	0.82 birth	
		18 month	PCB 0.77 mg/kg TEQ 22 pg/g (NOAEC TEQ 3 pg/g)	11 mg/kg lipid muscle (control <1 mg/kg)	0.95	1.22	1.34	1.27	0.90 birth 0.69 2 wk 0.67 5 wk	0.49 2 wk

Ref	Exposure				Relative Response Compared to Control					
	Chemical & Source	Exposure Duration	Dietary Conc	Tissue Conc	whelped ♀ / mated ♀	total kits / whelped ♀	live kits / whelped ♀	live kits / mated ♀	kit BW, time	kit survival, time
			PCB 2.31 mg/kg TEQ 65 pg/g	54 mg/kg	0.42	0.80	0.45	0.20	0.75 birth	0 2 wk
12	reported as PCB (Aroclor not specified); Baltic herring	5.3 month before mating + exposure during mating; TEQ not specified ("internat-ional" TEFs)	PCB 0.36 mg/kg (control 0.024 mg/kg) TEQ 26 pg/g (control 2 pg/g)		1.00	0.92	0.92	0.92	0.87–0.90 10 d 0.87–0.89 50 d	
	A1242 product added to freshwater smelt	5.3 month before mating, control exposure during mating	PCB 2.88 mg/kg TEQ 157 pg/g		0.80	0.76	0.73	0.58	0.78-0.81 10 d 0.95–1.01 50 d	

Notes for Table 4.

Ref - references [abbreviated reference used in the figures and Table 2 in brackets]:

1) Platonow and Karstad. 1973. [Platonow73]
2) Aulerich and Ringer. 1977. [Aulerich77]
3) Jensen 1977. [Jensen77]
4) Bleavins, et al. 1980 [Bleavins80]

5) Hornshaw, et al. 1983. [Hornshw83]
6) Wren, et al. 1987 [Wren87]
7) Kihiström, et al. 1992. [Kihistrm92]
8) Heaton, et al. 1995a, 1995b, and Tillitt, et al. 1996. [Heaton95]
9) Restum, et al. 1998, Shipp, et al. 1998, and Tillitt, et al. 1996. [Restum98]
10) Halbrook, et al. 1999. [Halbrok99]
11) Brunström, et al. 2001. [Brunstm01]
12) Käkelä, et al. 2002. [Kakela02]

Relative Response Compared to Control = treatment response / control response

Source: product is commercial product mixed with food; field is field-contaminated biota prepared as food

TEQ for Restum, et al. (1998) is based on the following regression of total PCB (mg/kg) and H4IIE-bioassay TEQ (pg/g) (data from Tillitt, et al. 1996):

$$TEQ = (25.735 * PCB) + 0.703 \quad r^2 = 1.0, p = 0.005, \text{for PCB range } 0.015–1.53 \text{ mg/kg}$$

Table 5. Chicken PCB Toxicity Studies

Ref	Exposure					Relative Response Compared to Control					
	Chemical, Source	Species	Exposure Duration	Dose to Hen (mg/kg-d)	Egg Conc (whole ww)	Egg Productivity	Egg Fertility	Hatchability	Chick BW	Chick Survival	Chick Normality
1	A1242 product	chicken (white leghom)	6 wk	1.34				0.10, 6 wk			
				3.35				0, 6 wk			
		chicken (broiler)		1.34				0.09, 6 wk			
				3.35				0.07, 6 wk			
2	A1242 product	chicken (white leghom)	6 wk	0.34 (control NA)	0.62 mg/kg 6 wk	0.92, 6 wk		1.03, 6 wk			
				0.67	1.35 mg/kg 6 wk	0.36 6 wk		0.82 6 wk			
				1.34	2.26 mg/kg 6 wk	0.41 6 wk		0.55 6 wk			
				2.68	2.8 mg/kg 6 wk	0.77 6 wk		0 6 wk			
				5.36	10.01 mg/kg 6 wk	0.90 6 wk		0 6 wk			
3	A1254 product	chicken (white leghom)	14 wk	0.34 (control NA)	5.5 mg/kg (max.) 2-14 wk	0.87 1-14 wk	0.98 1-14 wk	1 1-14 wk			
			39 wk	0.34	7.5 mg/kg (max.) 26-35 wk	0.80 26-39 wk	0.74 34-39 wk	1 1-39 wk			

Ref	Exposure					Relative Response Compared to Control					
	Chemical, Source	Species	Exposure Duration	Dose to Hen (mg/kg-d)	Egg Conc (whole ww)	Egg Productivity	Egg Fertility	Hatchability	Chick BW	Chick Survival	Chick Normality
			14 wk	3.35	50 mg/kg (max.) 2-14 wk	0.75 1-14 wk	1.05 1-14 wk	0 3-6 wk			
4	A1254 product	chicken (white leghorn)	6 wk	5.5 (control NA)	10 mg/kg 1 wk; 24 mg/kg 2 wk; 36.4 mg/kg 3 wk; (control NA)	1.02 1-6 wk	1.05 1-6 wk	0.41 2 wk; 0 3-6 wk			
5	A1221 product	chicken (white leghorn)	9 wk	1.30 (control NA)	<1 mg/kg 9 wk	1 0-9 wk		0.99 0-9 wk	0.98 6-9 wk	1	
	A1232 product			1.34	2.5 mg/kg 9 wk	0.91 0-9 wk		0.60 0-9 wk 0.43 8 wk	0.85 6-9 wk	0.93	
	A1242 product			0.12		0.95 0-9 wk		0.98 0-9 wk	0.98 6-9 wk	0.99	
				1.21	14 mg/kg 9 wk	0.85 0-9 wk		0.20 0-9 wk 0.10 8 wk	0.71 6-9 wk	0.93	
	A1248 product			0.12		0.97 0-9 wk		0.99 0-9 wk	0.94 6-9 wk	0.99	

Ref	Exposure					Relative Response Compared to Control					
	Chemical, Source	Species	Exposure Duration	Dose to Hen (mg/kg-d)	Egg Conc (whole ww)	Egg Productivity	Egg Fertility	Hatchability	Chick BW	Chick Survival	Chick Normality
				1.21	10 mg/kg 9 wk	0.85 0-9 wk		0.13 0-9 wk 0.09 8 wk	0.67 6-9 wk	0.44	
	A1254 product			0.13		0.97 0-9 wk		0.96 0-9 wk	0.93 6-9 wk	1	
				1.22	12 mg/kg	0.90 0-9 wk		0.86 0-9 wk 0.74 8 wk	0.87 6-9 wk	0.95	
	A1268 product			1.28	23 mg/kg	0.94 0-9 wk		0.98 0-9 wk	0.96 6-9 wk	1	
6	A1232 product	chicken (white leghorn)	8 wk	0.67 (control NA)				0.86 8 wk			
				1.34				0.57, 8 wk			
	A1242 product			0.34				0.84, 0-8 wk			0.94
				0.67				0.74, 0-8 wk 0.51, 8 wk			0.93
				1.34				0.31, 0-8 wk 0.06, 8 wk			0.90
	A1248 product			0.34				0.96, 0-8 wk			1
				0.67				0.75, 0-8 wk 0.42, 8 wk			0.97

Ref	Exposure					Relative Response Compared to Control					
	Chemical, Source	Species	Exposure Duration	Dose to Hen (mg/kg-d)	Egg Conc (whole ww)	Egg Productivity	Egg Fertility	Hatchability	Chick BW	Chick Survival	Chick Normality
				1.34				0.24, 0-8 wk 0.06, 8 wk			0.89
7	A1248 product	chicken (white leghom)	8 wk	0.03 (control NA)	0.16 mg/kg 4 wk; 0.22 mg/kg 8 wk	0.99 8 wk		1.01 4 wk 1.01 8 wk			
				0.07	0.33 mg/kg 4 wk; 0.41 mg/kg 8 wk	1.03 8 wk		0.98 4 wk 1.04 8 wk			
				0.67	2.2 mg/kg 4 wk; 3 mg/kg 8 wk	0.92 8 wk		0.73 4 wk 0.55 8 wk			
				1.34	4.5 mg/kg 4 wk; 7 mg/kg 8 wk	0.87 8 wk		0.03 4 wk 0.03 8 wk			
8	reported as A1242, 1248, 1254 and 1260; H4IIE bioassay TEQ; Saginaw Bay carp	chicken (white leghom)	8 wk	PCB 0.04 (control 0.016); TEQ 1.4 ng/kg-d (control 0.2)	4 mg/kg 4-8 wk (control 1 mg/kg)	1.37 4-8 wk	0.99 4-8 wk	1.05 4-8 wk	1.0 hatch		0.93 -1 to 8 wk

Ref	Exposure					Relative Response Compared to Control					
	Chemical, Source	Species	Exposure Duration	Dose to Hen (mg/kg-d)	Egg Conc (whole ww)	Egg Productivity	Egg Fertility	Hatchability	Chick BW	Chick Survival	Chick Normality
				PCB 0.36; TEQ 3.2	26 mg/kg 4-8 wk	1.63 4-8 wk	1.28 4-8 wk	0.82 4-8 wk	1.1 hatch		0.72 -1 to 8 wk
9	A1242 product	chicken eggs (white leghorn)	injected in yolk		0.02 mg/kg (control NA)				1.08 embryo		
					0.24 mg/kg				1.07 embryo		
					2.44 mg/kg				0.93 embryo		
	A1254 product				0.02 mg/kg				1.03 embryo		
					0.24 mg/kg				1.02 embryo		
					2.44 mg/kg				0.92 embryo		

Notes for Table 5.

Ref - references [abbreviated reference used in the figures and Table 2 in brackets]:

1) Briggs and Harris. 1972. [Briggs72]
2) Britton and Huston. 1973. [Britton73]
3) Platonow and Reinhart. 1973. [Platonw73]
4) Tumasonis, et al. 1973. [Tumas73]
5) Lillie, et al. 1974 and Cecil, et al. 1974. [Lillie/Cecil74 or Lillie/C74]
6) Lillie, et al. 1975. [Lillie75]

7) Scott 1977. [Scott77]
8) Summer, et al. 1996a., 1996b. [Summer96]
9) Gould, et al. 1997. [Gould97]

Exposures occur through contaminated feed except for Tumasonis, et al. (1973) through contaminated water, and Gould, et al. (1997) through yolk injection.

Relative Response Compared to Control = treatment response / control response

Source: product is commercial product mixed with feed or in water; field is field-contaminated biota prepared as feed

Dose: Calculated from experimental data when available. Generic calculation based on a white leghorn hen food ingestion rate of 0.067 kg feed/kg_{BW}-d (Medway and Kare 1959 cited in USEPA 1995a).

Egg Concentration: Yolk concentration is converted to whole-egg concentration by multiplying by 0.364 (Southerland and Rahn 1987 as cited in Hoffman, et al. 1996).

Chick normality is the proportion of chicks without deformities (= 1 - deformity rate)

Table 6. Summary of Mink PCB Studies and Relative Responses

Lead author Date	Chemical	Dietary PCB conc. mg/kg ww	Treatment name	Chemical source	Dietary TEQ conc. pg/g ww	TEQ source	Exposure duration month	Breeding seasons exposed	Generations exposed	Tissue	Tissue residue PCB conc. mg/kg ww	Lipid cont. % ww	PCB conc. mg/kg lw	TEQ conc. ww	Whelp frequency Control %	Treatment %	RR ratio	Whelp freq. source
Platonow73	A1254	0.64		field			5.2	1	1	liver, muscle	1.23, 0.97							
Platonow73	A1254	3.57		field			3.4	1	1	liver, muscle	11.99, 3.31				NA	0	0.00	text p 393
Aulerich77	A1242	2		product			9.7	1	1						100	100	1.00	table 10
Aulerich77	A1254	1		product			4.2	1	1						100	80	0.80	table 9
Aulerich77	A1254	2		product			9.7	1	1						100	29	0.29	table 10
Aulerich77	A1254	5		product			4.2	1	1						100	25	0.25	table 9
Jensen77	NA	3.3	Group B	NA			2.2	1	1	adipose			86		92	73	0.79	table 1
Jensen77	NA	11	Goup C	NA			2.2	1	1	adipose			280		92	0	0.00	table 1
Bleavins80	A1242	5		product			8.1	1	1						76.2	0	0.00	table 2
Bleavins80	A1242	10		product			8.1	1	1						76.2	0	0.00	table 2
Hornshw83	A1254	0.21	alewife	field			7	1	1	adipose			8.1		90	83	0.92	table 3
Hornshw83	A1254	0.48	whitefish	field			7	1	1	adipose			13		90	80	0.89	table 3
Hornshw83	A1254	0.63	sucker	field			7	1	1	adipose			10		90	90	1.00	table 3
Hornshw83	A1254	0.69	perch	field			7	1	1	adipose			13		90	82	0.91	table 3
Hornshw83	A1254	1.5	carp	field			7	1	1	adipose			37		90	27	0.30	table 3
Hornshw83	A1254	0.66	perch/sucker	field			7	1	1						86	50	0.58	table 3
Wren87	A1254	1	PCB	product			6.1	1	1	liver	2.8				93	92	0.99	87b table 2
Kihistrm92	A50	12	Group 2	product			3	1	1	muscle	3.98	2.2	181.00		90	10	0.11	table 2
Kihistrm92	A1254	10	Group 9	product			3	1	1	muscle	1.33	1.8	74.00		89	30	0.34	table 2
Heaton95	PCB	0.72	10 % carp	field	19.4	H4IIE	6	1	1	liver	2.2			495	50	50	1.00	p 335, table 2
Heaton95	PCB	1.53	20 % carp	field	40	H4IIE	6	1	1	liver	3.1			439	50	50	1.00	p 335, table 2
Heaton95	PCB	2.56	30 % carp	field	80.8	H4IIE	6	1	1	liver	6.3			656	50	50	1.00	p 335, table 2
Restum98	PCB	0.25	P1 0.25 to F1-1	field	7.1	H4IIE	6	1	1						69	94	1.36	table 6
Restum98	PCB	0.5	P1 0.5 to F1-1	field	13.6	H4IIE	6	1	1						69	93	1.35	table 6
Restum98	PCB	1	P1 1.0 to F1-1	field	26.4	H4IIE	6	1	1						69	80	1.16	table 6
Restum98	PCB	0.25	P1 0.25-0.25 to F1-2	field	7.1	H4IIE	16	2	1	liver	0.98				86	88	1.02	table 6
Restum98	PCB	0.5	P1 0.5-0.5 to F1-2	field	13.6	H4IIE	16	2	1	liver	0.89				86	67	0.78	table 6
Restum98	PCB	1	P1 1.0-1.0 to F1-2	field	26.4	H4IIE	16	2	1	liver	1.57				86	57	0.66	table 6
Restum98	PCB	0.25	F1-1 0.25-0.25 to F2	field	7.1	H4IIE	12	2	2	liver	0.63				79	67	0.85	table 6
Restum98	PCB	0.5	F1-1 0.5-0.5 to F2	field	13.6	H4IIE	12	2	2	liver	0.96				79	60	0.76	table 6
Restum98	PCB	1	F1-1 1.0-1.0 to F2	field	26.4	H4IIE	12	2	2	liver	1.47				79	50	0.63	table 6
Halbrok99	A1260	0.52	Diet C	field			7	1	1	liver	<0.005				86	50	0.58	text p 652, table 2
Halbrok99	A1260	1.01	Diet D	field			7	1	1	liver, fat	<0.005		105.86		86	75	0.87	text p 652, table 2
Halbrok99	A1260	1.36	Diet E	field			7	1	1	liver, fat	7.25		128.63		86	100	1.16	text p 652, table 2
Brunstm01	A50	0.77	A50 low	product	22	WHO	6	1	1						93	89	0.96	table 3
Brunstm01	A50	2.31	A50 high	product	65	WHO	6	1	1						93	90	0.97	table 3
Brunstm01	A50	0.77	A50 low	product	22	WHO	18	2	1	muscle	0.26	2.4	11		93	88	0.95	table 5
Brunstm01	A50	2.31	A50 high	product	65	WHO	18	2	1	muscle	1.30	2.4	54		93	39	0.42	table 5
Kakela02	PCB	0.36	Baltic herring	field	26	NA	5.3	1	1						100	100	1.00	table 3
Kakela02	A1242	2.88	Smelt PCB	product	157	NA	5.3	1	1						100	80	0.80	table 3

Notes:
Treatment data only, control data excluded (control RR = 1.0 by definition)
TEQ source - H4IIE - rat hepatoma cell bioassay; WHO - Van den Berg, et al. (1998)
Exposure duration - month = days / 30.5 or weeks / 4; PCB - sum of multiple Aroclors; NA - not available
RR - relative response = treatment response / control response
Default Live kits/mated female = Live kits/whelped female * fraction of females whelped
Plantonow73 - Treatment 0.64 Live kits/mated female = 3 kits / 10 females surviving (2 deaths out of 12 during breeding)
Jensen77 - PCB type or source not identified; Live kits/whelped female = No. of whelps born/pregnant female - number of stillbirths/bitch
Hornshaw83 - Tissue residue for February 1980, mean values
Kihistrm92 - Dietary PCB conc. = 2 mg A50/d or 1.64 mg A1254/d / 0.17 kg food/d (p. 564); Table 2 Stillborn should be 1 (not 100) for Group 2 (fig 4)
Heaton95 - Liver conc. from Tillitt, et al. 96 (Table 4)
Restum98 - Treatment name is parental designation to offspring designation; TEQ interpolated from Tillitt, et al. 96 (Tables 1 and 2)
Restum98 - Live kits/whelped female = Survivability at birth * Litter size
Restum98 - Kit bodyweight in order of male, female kit; – no survivors; RR is the unweighted mean of male and female RRs, or single sex RR if only one sex survived
Halbrook99 - Diet A is used for control; Kit survival = (Alive at 6 weeks / Born alive) * 100
Brunstm01 - Dietary PCB conc. = 0.1 or 0.3 mg A50/d / 0.13 kg/d food ration (p. 2319)
Kakela02 - Smelt PCB treatment was exposed for 21 wk before breeding, then switched to control diet during breeding
Kakela02 - Dietary PCB conc. = Sum PCB per day / Average food consumption; Kit bodyweight in order of male kit, female kit; RR is unweighted mean
Kakela02 - Live kits/whelped female = ((Kits/mother * surviving females) - Dead kits) / surviving females; TEQ - "international" TEFs but no date is given

Table 6. Summary of Mink PCB Studies and Relative Responses

Lead author Date	Chemical	Dietary PCB conc mg/kg ww	Treatment name	Total kits / whelped female Control number	Treatment number	RR ratio	Total kits / whelped source	Live kits / whelped female Control number	Treatment number	RR ratio	Live kits / whelped source	Live kits / mated female Control number	Treatment number	RR ratio	Live kits / mated source	Kit bodyweight 0-1 wk Control g	Treatment g	RR ratio
Platonow73	A1254	0.64										1.8	0.3	0.17	text p 393, 398			
Platonow73	A1254	3.57		NA	0	0.00	text p 393	NA	0	0.00	text p 393	1.8	0	0.00	text p 393, 398			
Aulerich77	A1242	2		4.1	5.6	1.37	table 10	3.5	5	1.43	table 10	3.5	5	1.43	table 10	9.9	9.3	0.94
Aulerich77	A1254	1		6	5.4	0.90	table 9	5.1	4.4	0.86	table 9	5.1	3.5	0.69	table 9			
Aulerich77	A1254	2		4.1	1	0.24	table 10	3.5	0.5	0.14	table 10	3.5	0.14	0.04	table 10	9.9	5.4	0.55
Aulerich77	A1254	5		6	3	0.50	table 9	5.1	1	0.20	table 9	5.1	0.25	0.05	table 9			
Jensen77	NA	3.3	Group B	5.1	2.9	0.57	table 1	4.6	0.9	0.20	text, table 1	4.2	0.7	0.17	text, table 1	9.4	6.8	0.72
Jensen77	NA	11	Goup C	5.1	0	0.00	table 1	4.6	0	0.00	text, table 1	4.2	0	0.00	text, table 1			
Bleavins80	A1242	5		5.8	0	0.00	table 2	4.9	0	0.00	table 2	3.8	0	0.00	table 2			
Bleavins80	A1242	10		5.8	0	0.00	table 2	4.9	0	0.00	table 2	3.8	0	0.00	table 2			
Hornshw83	A1254	0.21	alewife	5.4	6.2	1.15	table 3	4.2	5.3	1.26	table 3	3.8	4.2	1.11	table 3	8.3	8.4	1.01
Hornshw83	A1254	0.48	whitefish	5.4	4.9	0.91	table 3	4.2	4	0.95	table 3	3.8	3.2	0.84	table 3	8.3	8.5	1.02
Hornshw83	A1254	0.63	sucker	5.4	4.3	0.80	table 3	4.2	2.8	0.67	table 3	3.8	2.5	0.66	table 3	8.3	8.7	1.05
Hornshw83	A1254	0.69	perch	5.4	5	0.93	table 3	4.2	3.7	0.88	table 3	3.8	3	0.79	table 3	8.3	8.1	0.98
Hornshw83	A1254	1.5	carp	5.4	3	0.56	table 3	4.2	0	0.00	table 3	3.8	0	0.00	table 3			
Hornshw83	A1254	0.66	perch/sucker	5.4	2	0.37	table 3	5.2	1	0.19	table 3	4.4	0.5	0.11	table 3	9	7.7	0.86
Wren87	A1254	1	PCB	6.9	7.5	1.09	87b table 2	5.8	6.7	1.16	87b table 2	5.4	6.2	1.15	87b table 2	28.1	21.6	0.77
Kihistrm92	A50	12	Group 2	8.1	1	0.12	table 2	5.3	0	0.00	table 2	4.8	0	0.00	table 2			
Kihistrm92	A1254	10	Group 9	5	3.3	0.66	table 2	4.3	0	0.00	table 2	3.7	0	0.00	table 2			
Heaton95	PCB	0.72	10 % carp	5.7	5.3	0.93	table 2	5	3.8	0.76	table 2	2.5	1.9	0.76	p 335, table 2	10.5	9.76	0.93
Heaton95	PCB	1.53	20 % carp	5.7	5.8	1.02	table 2	5	4.8	0.96	table 2	2.5	2.4	0.96	p 335, table 2	10.5	8.66	0.82
Heaton95	PCB	2.56	30 % carp	5.7	3.3	0.58	table 2	5	0.7	0.14	table 2	2.5	0.35	0.14	p 335, table 2	10.5	7.49	0.71
Restum98	PCB	0.25	P1 0.25 to F1-1	5	5.8	1.16	table 6	4.7	5.6	1.19	tables 6, 7	3.2	5.3	1.66	table 6	10, 9.2	9.3, 8.7	0.94
Restum98	PCB	0.5	P1 0.5 to F1-1	5	5.1	1.02	table 6	4.7	4.3	0.91	tables 6, 7	3.2	4	1.25	table 6	10, 9.2	8.7, 7.7	0.86
Restum98	PCB	1	P1 1.0 to F1-1	5	5.1	1.02	table 6	4.7	3.6	0.77	tables 6, 7	3.2	2.9	0.91	table 6	10, 9.2	7.5, 7.3	0.77
Restum98	PCB	0.25	P1 0.25-0.25 to F1-2	6.3	6	0.95	table 6	5.6	5.4	0.96	tables 6, 7	4.8	4.7	0.98	table 6	11.1, 9.9	9.8, 10.8	0.99
Restum98	PCB	0.5	P1 0.5-0.5 to F1-2	6.3	5.8	0.92	table 6	5.6	4.5	0.80	tables 6, 7	4.8	3	0.63	table 6	11.1, 9.9	8.6, 8.0	0.79
Restum98	PCB	1	P1 1.0-1.0 to F1-2	6.3	4	0.63	table 6	5.6	3.3	0.59	tables 6, 7	4.8	1.9	0.40	table 6	11.1, 9.9	8.1, 7.3	0.74
Restum98	PCB	0.25	F1-1 0.25-0.25 to F2	5.7	6	1.05	table 6	5.5	5.3	0.96	tables 6, 7	4.3	3.6	0.84	table 6	9.8, 9.2	8.5, 8.0	0.87
Restum98	PCB	0.5	F1-1 0.5-0.5 to F2	5.7	5	0.88	table 6	5.5	1.7	0.31	tables 6, 7	4.3	1	0.23	table 6	9.8, 9.2	7.2, 5.9	0.69
Restum98	PCB	1	F1-1 1.0-1.0 to F2	5.7	3	0.53	table 6	5.5	0.5	0.09	tables 6, 7	4.3	0.3	0.07	table 6	9.8, 9.2	5.0, 5.5	0.56
Halbrok99	A1260	0.52	Diet C	6.5	7.8	1.20	table 2	5.2	6	1.15	table 2	4.5	3	0.67	text p 652, table 2			
Halbrok99	A1260	1.01	Diet D	6.5	6	0.92	table 2	5.2	5.7	1.10	table 2	4.5	4.3	0.96	text p 652, table 2			
Halbrok99	A1260	1.36	Diet E	6.5	4.3	0.66	table 2	5.2	3.9	0.75	table 2	4.5	3.9	0.87	text p 652, table 2			
Brunstm01	A50	0.77	A50 low	4.9	5.9	1.20	table 3	4	5.2	1.30	table 3	3.7	4.6	1.24	table 3	9.6	9.5	0.99
Brunstm01	A50	2.31	A50 high	4.9	5.1	1.04	table 3	4	3.8	0.95	table 3	3.7	3.4	0.92	table 3	9.6	7.9	0.82
Brunstm01	A50	0.77	A50 low	5.1	6.2	1.22	table 5	4.4	5.9	1.34	table 5	4.1	5.2	1.27	table 5	8.9	8	0.90
Brunstm01	A50	2.31	A50 high	5.1	4.1	0.80	table 5	4.4	2	0.45	table 5	4.1	0.8	0.20	table 5	8.9	6.7	0.75
Kakela02	PCB	0.36	Baltic herring	6.6	6.1	0.92	table 3	6.6	6.1	0.92	table 3	6.6	6.1	0.92	table 3			
Kakela02	A1242	2.88	Smelt PCB	6.6	5	0.76	table 3	6.6	4.8	0.73	table 3	6.6	3.8	0.58	table 3			

Table 6. Summary of Mink PCB Studies and Relative Responses

Lead author Date	Chemical	Dietary PCB conc. mg/kg ww	Treatment name	Kit bodyweight 2-3 wk Control g	Kit bodyweight 2-3 wk Treatment g	Kit bodyweight 2-3 wk RR ratio	Kit bodyweight 4-6 wk Control g	Kit bodyweight 4-6 wk Treatment g	Kit bodyweight 4-6 wk RR ratio	Kit bodyweight source	Kit survival Control %	Kit survival Treatment %	Kit survival RR ratio	Kit survival source
Platonow73	A1254	0.64									NA	0	0.00	text p 393
Platonow73	A1254	3.57												
Aulerich77	A1242	2								table 10	64	91	1.42	table 10
Aulerich77	A1254	1												
Aulerich77	A1254	2								table 10	64	0	0.00	table 10
Aulerich77	A1254	5												
Jensen77	NA	3.3	Group B							text	82	17	0.21	text
Jensen77	NA	11	Goup C											
Bleavins80	A1242	5												
Bleavins80	A1242	10												
Hornshw83	A1254	0.21	alewife				122	124	1.02	table 4	55	51	0.93	table 3
Hornshw83	A1254	0.48	whitefish				122	107	0.88	table 4	55	28	0.51	table 3
Hornshw83	A1254	0.63	sucker				122	111	0.91	table 4	55	40	0.73	table 3
Hornshw83	A1254	0.69	perch				122	98	0.80	table 4	55	36	0.65	table 3
Hornshw83	A1254	1.5	carp											
Hornshw83	A1254	0.66	perch/sucker							table 4	65	0	0.00	table 3
Wren87	A1254	1	PCB	107.3	80.2	0.75	227.8	161.2	0.71	87b table 4	72	72.2	1.00	87b table 2
Kihistrm92	A50	12	Group 2											
Kihistrm92	A1254	10	Group 9											
Heaton95	PCB	0.72	10 % carp	98.7	66.1	0.67	248	197	0.79	table 3	85	28	0.33	table3
Heaton95	PCB	1.53	20 % carp	98.7	65.8	0.67	248	101	0.41	table 3	85	11	0.13	table3
Heaton95	PCB	2.56	30 % carp							table 3	85	0	0.00	table3
Restum98	PCB	0.25	P1 0.25 to F1-1	113, 99	89, 88	0.84	293, 253	220, 214	0.80	table 8	72.7	67.8	0.93	table 7 wk 6
Restum98	PCB	0.5	P1 0.5 to F1-1	113, 99	76, 74	0.71	293, 253	200, 165	0.67	table 8	72.7	52.5	0.72	table 7 wk 6
Restum98	PCB	1	P1 1.0 to F1-1	113, 99	58, 58	0.55	293, 253	102, 125	0.42	table 8	72.7	23	0.32	table 7 wk 6
Restum98	PCB	0.25	P1 0.25-0.25 to F1-2	116, 110	106, 96	0.89	340, 304	312, 280	0.92	table 9	80.3	76.2	0.95	table 7 wk 6
Restum98	PCB	0.5	P1 0.5-0.5 to F1-2	116, 110	78, 72	0.66	340, 304	317, --	0.93	table 9	80.3	4.4	0.05	table 7 wk 6
Restum98	PCB	1	P1 1.0-1.0 to F1-2	116, 110	69, 55	0.55	340, 304	223, 182	0.63	table 9	80.3	12.5	0.16	table 7 wk 6
Restum98	PCB	0.25	F1-1 0.25-0.25 to F2	116, 106	128, 109	1.07	380, 326	361, 291	0.92	table 10	73	58.3	0.80	table 7 wk 6
Restum98	PCB	0.5	F1-1 0.5-0.5 to F2	116, 106	--, 45	0.42	380, 326	--, 177	0.54	table 10	73	13.3	0.18	table 7 wk 6
Restum98	PCB	1	F1-1 1.0-1.0 to F2							table 10	73	0	0.00	table 7 wk 6
Halbrok99	A1260	0.52	Diet C				328	333	1.02	table 2	63.5	50	0.79	table 2
Halbrok99	A1260	1.01	Diet D				328	307	0.94	table 2	63.5	78.9	1.24	table 2
Halbrok99	A1260	1.36	Diet E				328	295	0.90	table 2	63.5	100	1.57	table 2
Brunstm01	A50	0.77	A50 low							table 3				
Brunstm01	A50	2.31	A50 high							table 3				
Brunstm01	A50	0.77	A50 low	70	48	0.69	258	173	0.67	table 5, fig 2	73	36	0.49	text p 2322
Brunstm01	A50	2.31	A50 high							table 5	73	0	0.00	text p 2322
Kakela02	PCB	0.36	Baltic herring	63, 58	55, 52	0.89	566, 505	501, 439	0.88	table 3				
Kakela02	A1242	2.88	Smelt PCB	63, 58	49, 47	0.80	566, 505	573, 481	0.98	table 3				

Table 7. Summary of Chicken PCB Studies and Relative Responses

Lead author Date	Chemical	Dietary conc. mg/kg fw	Food ingestion kg/kgbw fw	Dose mg/kg-d	Exposure duration wk	Yolk conc. mg/kg fw	Whole egg conc. mg/kg fw	Egg conc. source	Control # or %	Productivity Treatment # or %	RR ratio	Productivity source	Control %	Fertility Treatment %	RR ratio	Fertility source
Briggs72	A1242	20	0.067	1.34	6											
Briggs72	A1242	50	0.067	3.35	6											
Briggs72	A1242	20	0.067	1.34	6											
Briggs72	A1242	50	0.067	3.35	6											
Britton73	A1242	5	0.067	0.34	6	1.7	0.62	table 3 wk 6	61	56	0.92	table 1 wk 6				
Britton73	A1242	10	0.067	0.67	6	3.7	1.35	table 3 wk 6	61	22	0.36	table 1 wk 6				
Britton73	A1242	20	0.067	1.34	6	6.2	2.26	table 3 wk 6	61	25	0.41	table 1 wk 6				
Britton73	A1242	40	0.067	2.68	6	7.7	2.80	table 3 wk 6	61	47	0.77	table 1 wk 6				
Britton73	A1242	80	0.067	5.36	6	27.5	10.01	table 3 wk 6	61	55	0.90	table 1 wk 6				
Platonw73	A1254	5	0.067	0.34	14		5.5	fig 4 max. wk 12	82.7	72	0.87	text p 343 wk 1-14	85.5	83.6	0.98	text p 344 wk 1-14
Platonw73	A1254	5	0.067	0.34	39		7.5	fig 4 max. wk 26	72	57.5	0.80	text p 343 wk 26-39	85	63.3	0.74	fig 2 wk 34-39
Platonw73	A1254	50	0.067	3.35	14		50	fig 4 max. wk 12	82.7	62.2	0.75	text p 343 wk 1-14	85.5	89.9	1.05	text p 344 wk 1-14
Tumas73	A1254	50	0.11	5.50	6	100	36.40	fig 2 wk 3	8.6	8.77	1.02	table 1 wk 1-6	92.3	97.2	1.05	table 1 wk 1-6
Lillie/Cecil74	A1221	20	0.0649	1.30	9		<1	Cecil fig 4 wk 9	79.4	79.3	1.00	Lillie table 1 wk 0-9				
Lillie/Cecil74	A1232	20	0.067	1.34	9		2.5	Cecil fig 4 wk 9	79.4	71.9	0.91	Lillie table 1 wk 0-9				
Lillie/Cecil74	A1242	2	0.0615	0.12	9				79.4	75.5	0.95	Lillie table 1 wk 0-9				
Lillie/Cecil74	A1242	20	0.0605	1.21	9		14	Cecil fig 4 wk 9	79.4	67.5	0.85	Lillie table 1 wk 0-9				
Lillie/Cecil74	A1248	2	0.0623	0.12	9				79.4	76.9	0.97	Lillie table 1 wk 0-9				
Lillie/Cecil74	A1248	20	0.0607	1.21	9		10	Cecil fig 4 wk 9	79.4	67.5	0.85	Lillie table 1 wk 0-9				
Lillie/Cecil74	A1254	2	0.0636	0.13	9				79.4	77.1	0.97	Lillie table 1 wk 0-9				
Lillie/Cecil74	A1254	20	0.061	1.22	9		12	Cecil fig 4 wk 9	79.4	71.3	0.90	Lillie table 1 wk 0-9				
Lillie/Cecil74	A1268	20	0.0641	1.28	9		23	Cecil fig 4 wk 9	79.4	74.4	0.94	Lillie table 1 wk 0-9				
Lillie75	A1232	10	0.067	0.67	8											
Lillie75	A1232	20	0.067	1.34	8											
Lillie75	A1242	5	0.067	0.34	8											
Lillie75	A1242	10	0.067	0.67	8											
Lillie75	A1242	20	0.067	1.34	8											
Lillie75	A1248	5	0.067	0.34	8											
Lillie75	A1248	10	0.067	0.67	8											
Lillie75	A1248	20	0.067	1.34	8											
Scott77	A1248	0.5	0.067	0.03	8		0.22	table 1 wk 8	74.5	74	0.99	table 3 wk 8				
Scott77	A1248	1	0.067	0.07	8		0.41	table 1 wk 8	74.5	76.6	1.03	table 3 wk 8				
Scott77	A1248	10	0.067	0.67	8		3	table 1 wk 8	74.5	68.7	0.92	table 3 wk 8				
Scott77	A1248	20	0.067	1.34	8		7	table 1 wk 8	74.5	64.8	0.87	table 3 wk 8				
Summer96	PCB	0.8	0.0553	0.04	8		4	96b table 1 wk 6-10	54	74	1.37	96a table 5 wk 6-10	67	66.6	0.99	96a table 6 wk 6-10
Summer96	PCB	6.6	0.0548	0.36	8		26	96b table 1 wk 6-10	54	88	1.63	96a table 5 wk 6-10	67	85.7	1.28	96a table 6 wk 6-10
Gould97	A1242	yolk inject				0.067	0.02	table 1								
Gould97	A1242	yolk inject				0.67	0.24	table 1								
Gould97	A1242	yolk inject				6.7	2.44	table 1								
Gould97	A1254	yolk inject				0.067	0.02	table 1								
Gould97	A1254	yolk inject				0.67	0.24	table 1								
Gould97	A1254	yolk inject				6.7	2.44	table 1								

Notes:
Default Food ingestion rate - 0.067 kg feed/kgbw-d white leghorn hen (Medway and Kare 1959)
Whole egg conc. = 0.364 yolk conc. (Sotherland and Rahn 1987)
RR - relative response = treatment response / control response; Normality = 1 - deformity
Tumas73 - Dietary conc. is mg/l water conc; Food ingestion rate is l/kgbw-d water ingestion = 0.177 l/hen/d / 1.61 kgbw/hen (p. 314, 315)
Lillie/Cecil74 - Food consumption = treatment food/hen-d (Lillie table 2 wk 0-9) / 1.953 kg mean initial hen bodyweight (Lillie p 727)
Lillie75 - Normality = 100 - % abnormal embryos of fertile eggs
Summer96 - Food ingestion rate - mean for wk 3-10 (96a table 4); Chick deformity recalculated from 96b table 5 (replace rounded percentages)
Gould97 - Yolk injection on day 0 of incubation. Treatment "chick" bodyweight is % difference in 17-d embryo bodyweight compared to control

Table 7. Summary of Chicken PCB Studies and Relative Responses

Lead author Date	Chemical	Dietary conc. mg/kg fw	Hatchability Control %	Hatchability Treatment %	RR ratio	Hatchability source	Chick Bodyweight Control g	Chick Bodyweight Treatment g	RR ratio	Bodyweight source	Chick Survival Control %	Chick Survival Treatment %	RR ratio	Survival source	Chick Normality (1 - defomity) Control %	Chick Normality (1 - defomity) Treatment %	RR ratio
Briggs72	A1242	20	68.9	7.2	0.10	table 1 wk 6 leghorn											
Briggs72	A1242	50	68.9	0	0.00	table 1 wk 6 leghorn											
Briggs72	A1242	20	65.5	6.2	0.09	table 1 wk 6 broiler											
Briggs72	A1242	50	65.5	4.5	0.07	table 1 wk 6 broiler											
Britton73	A1242	5	91	94	1.03	table 3 wk 6											
Britton73	A1242	10	91	75	0.82	table 3 wk 6											
Britton73	A1242	20	91	50	0.55	table 3 wk 6											
Britton73	A1242	40	91	0	0.00	table 3 wk 6											
Britton73	A1242	80	91	0	0.00	table 3 wk 6											
Platonw73	A1254	5	90	90	1.00	text p 344 wk 1-14											
Platonw73	A1254	5	90	90	1.00	text p 344, wk 1-39											
Platonw73	A1254	50	90	0	0.00	text p 344 wk 2-14											
Tumas73	A1254	50	84.7	0	0.00	table 1 wk 3-6											
Lillie/Cecil74	A1221	20	93.7	93.2	0.99	Lillie table 3 wk 0-9	163	159	0.98	Lillie table 4 wk 6-	98.4	98.3	1.00	Lillie table 4 wk 6-9			
Lillie/Cecil74	A1232	20	92.4	40	0.43	Cecil fig 1 wk 8	163	139	0.85	Lillie table 4 wk 6-	98.4	91.9	0.93	Lillie table 4 wk 6-9			
Lillie/Cecil74	A1242	2	93.7	92.2	0.98	Lillie table 3 wk 0-9	163	160	0.98	Lillie table 4 wk 6-	98.4	97.1	0.99	Lillie table 4 wk 6-9			
Lillie/Cecil74	A1242	20	92.4	9	0.10	Cecil fig 1 wk 8	163	115	0.71	Lillie table 4 wk 6-	98.4	91.7	0.93	Lillie table 4 wk 6-9			
Lillie/Cecil74	A1248	2	93.7	92.3	0.99	Lillie table 3 wk 0-9	163	153	0.94	Lillie table 4 wk 6-	98.4	97.5	0.99	Lillie table 4 wk 6-9			
Lillie/Cecil74	A1248	20	92.4	8	0.09	Cecil fig 1 wk 8	163	109	0.67	Lillie table 4 wk 6-	98.4	43.7	0.44	Lillie table 4 wk 6-9			
Lillie/Cecil74	A1254	2	93.7	89.7	0.96	Lillie table 3 wk 0-9	163	151	0.93	Lillie table 4 wk 6-	98.4	98.7	1.00	Lillie table 4 wk 6-9			
Lillie/Cecil74	A1254	20	92.4	68	0.74	Cecil fig 1 wk 8	163	141	0.87	Lillie table 4 wk 6-	98.4	93.7	0.95	Lillie table 4 wk 6-9			
Lillie/Cecil74	A1268	20	93.7	92.2	0.98	Lillie table 3 wk 0-9	163	156	0.96	Lillie table 4 wk 6-	98.4	98.7	1.00	Lillie table 4 wk 6-9			
Lillie75	A1232	10	90	77	0.86	text p 1554 wk 8											
Lillie75	A1232	20	90	51	0.57	text p 1554 wk 8											
Lillie75	A1242	5	91	76	0.84	table 3 wk 4-8									98	92	0.94
Lillie75	A1242	10	90	46	0.51	text p 1554 wk 8									98	91	0.93
Lillie75	A1242	20	90	5	0.06	text p 1554 wk 8									98	88	0.90
Lillie75	A1248	5	91	87	0.96	table 3 wk 4-8									98	98	1.00
Lillie75	A1248	10	90	38	0.42	text p 1554 wk 8									98	95	0.97
Lillie75	A1248	20	90	5	0.06	text p 1554 wk 8									98	87	0.89
Scott77	A1248	0.5	90.5	91.6	1.01	table 4 wk 8											
Scott77	A1248	1	90.5	93.7	1.04	table 4 wk 8											
Scott77	A1248	10	90.5	50	0.55	table 4 wk 8											
Scott77	A1248	20	90.5	2.4	0.03	table 4 wk 8											
Summer96	PCB	0.8	85.8	90	1.05	96b table 2 wk 6-1(	34.49	34.49	1.00	96b table 4 wk 6-10					82.7	76.5	0.93
Summer96	PCB	6.6	85.8	70.2	0.82	96b table 2 wk 6-1(	34.49	37.81	1.10	96b table 4 wk 6-10					82.7	59.9	0.72
Gould97	A1242	yolk inject						+8.4 %	1.08	fig 2 (17-d embryo)							
Gould97	A1242	yolk inject						+6.7 %	1.07	fig 2 (17-d embryo)							
Gould97	A1242	yolk inject						-7.0 %	0.93	fig 2 (17-d embryo)							
Gould97	A1254	yolk inject						+2.8 %	1.03	fig 2 (17-d embryo)							
Gould97	A1254	yolk inject						+2.1 %	1.02	fig 2 (17-d embryo)							
Gould97	A1254	yolk inject						-7.7 %	0.92	fig 2 (17-d embryo)							

Table 7. Summary of Chicken PCB Studies and Relative Responses

Lead author Date	Chemical	Dietary conc. mg/kg fw	Normality source
Briggs72	A1242	20	
Briggs72	A1242	50	
Briggs72	A1242	20	
Briggs72	A1242	50	
Britton73	A1242	5	
Britton73	A1242	10	
Britton73	A1242	20	
Britton73	A1242	40	
Britton73	A1242	80	
Platonw73	A1254	5	
Platonw73	A1254	5	
Platonw73	A1254	50	
Tumas73	A1254	50	
Lillie/Cecil74	A1221	20	
Lillie/Cecil74	A1232	20	
Lillie/Cecil74	A1242	2	
Lillie/Cecil74	A1242	20	
Lillie/Cecil74	A1248	2	
Lillie/Cecil74	A1248	20	
Lillie/Cecil74	A1254	2	
Lillie/Cecil74	A1254	20	
Lillie/Cecil74	A1268	20	
Lillie75	A1232	10	
Lillie75	A1232	20	
Lillie75	A1242	5	Table 3
Lillie75	A1242	10	Table 3
Lillie75	A1242	20	Table 3
Lillie75	A1248	5	Table 3
Lillie75	A1248	10	Table 3
Lillie75	A1248	20	Table 3
Scott77	A1248	0.5	
Scott77	A1248	1	
Scott77	A1248	10	
Scott77	A1248	20	
Summer96	PCB	0.8	96b table 5 wk 1-10
Summer96	PCB	6.6	96b table 5 wk 1-10
Gould97	A1242	yolk inject	
Gould97	A1242	yolk inject	
Gould97	A1242	yolk inject	
Gould97	A1254	yolk inject	
Gould97	A1254	yolk inject	
Gould97	A1254	yolk inject	