
6. Injuries to Fish and Aquatic Invertebrates

This chapter presents the Stage I Injury Assessment for the fish and aquatic invertebrate resources of the KRE. The Kalamazoo River fish community upstream of the Lake Allegan Dam includes primarily carp, white sucker, smallmouth bass, walleye, northern pike, channel catfish, and black crappie (J. Wesley, MDNR Fisheries Division, personal communication, 2004). When dissolved oxygen in the river was low prior to the 1980s, carp and white suckers dominated the fishery (Knight and Lauff, 1969; MWRC, 1972a; Towns, 1984). Downstream of the Lake Allegan Dam, the fish community includes carp, smallmouth bass, largemouth bass, northern pike, channel catfish, flathead catfish (*Pylodictis olivaris*), black crappie, yellow perch, white sucker, freshwater drum, and some white bass/hybrid striped bass (Knight and Lauff, 1969; MWRC, 1972a; Towns, 1984; J. Wesley, MDNR Fisheries Division, personal communication, 2004). Additional species migrate upstream from Lake Michigan. Chinook salmon, coho salmon, rainbow trout, brown trout, walleye, and lake sturgeon run the lower river to spawn, and Lake Allegan Dam prevents passage of these fish to upstream areas. Stocking of salmonid species in this reach began in the early 1970s. Currently, chinook salmon, rainbow trout, brown trout, and walleye are stocked in the lower river (J. Wesley, MDNR Fisheries Division, personal communication, 2004).

Benthic invertebrates are important components of the aquatic food chain that live in close contact with river sediment. Many such invertebrates are the larval stages of insects, and mussels and other bivalves are also important components. Benthic invertebrates also play an important role in nutrient and energy cycling within the aquatic food chain. Their close contact with PCB contaminated sediment can make them relatively highly exposed to PCBs in the KRE, which coupled with their importance in the aquatic food chain, makes benthic invertebrates relevant to the Stage I assessment.

Ecosystem services provided by fish include prey for carnivorous and omnivorous wildlife, and nutrient and energy cycling. Human use services include fishing for recreation and as a food source. Benthic invertebrates provide important ecological services as well, as prey for fish, birds, and mammals, in nutrient cycling, and in energy transfer.

6.1 Injury Definitions

Biological resources are defined in the DOI regulations as “those natural resources referred to in section 101(16) of CERCLA as fish and wildlife and other biota. Fish and wildlife include marine and freshwater aquatic and terrestrial species; game, nongame, and commercial species; and threatened, endangered, and State sensitive species. Other biota encompass shellfish, terrestrial and aquatic plants, and other living organisms not listed in this definition” [43 C.F.R. § 11.14(f)]. This chapter addresses injuries to aquatic biota, specifically fish and aquatic invertebrates. Injuries to terrestrial wildlife are addressed in Chapter 7 of this document.

In this chapter, injuries to aquatic biota are determined using the following injury definition. According to DOI regulations, “an injury to a biological resource has resulted from the . . . release of a hazardous substance if concentration of the substance is sufficient to cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction), or physical deformations” [43 C.F.R. § 11.62(f)(1)(i)].

An injury to biological resources can be demonstrated, per the DOI regulations, “if the biological response under consideration can satisfy all of the following acceptance criteria” [43 C.F.R. § 11.62 (f)(2): (i-iv)]:

- ▶ The biological response is often the result of exposure to . . . [the] hazardous substances [43 C.F.R. § 11.62 (f)(2)(i)].
- ▶ Exposure to . . . [the] hazardous substances is known to cause this biological response in free-ranging organisms [43 C.F.R. § 11.62 (f)(2)(ii)].
- ▶ Exposure to . . . [the] hazardous substances is known to cause this biological response in controlled experiments [43 C.F.R. § 11.62 (f)(2)(iii)].
- ▶ The biological response measurement is practical to perform and produces scientifically valid results [43 C.F.R. § 11.62 (f)(2)(iv)].

Injuries to biological resources may include death (e.g., fish kills), cancer (e.g., neoplasm), disease (e.g., fin erosion), physical deformation (e.g., deformities or lesions), behavioral abnormalities (e.g., avoidance), and physiological malfunctions (e.g., reduced reproduction) [43 C.F.R. § 11.62 (f)(4)].

6.2 Stage I Injury Assessment Approach

Exposure of fish to PCBs is known to cause a wide range of adverse effects (Table 6.1). Adverse effects in fish range from death (fry mortality and reduced egg survival rates) to sublethal and reproductive effects. PCBs have been found to promote the growth of cancerous tumors, and to reduce the effectiveness of the immune system in disease resistance. Physical deformities such as skeletal deformities and organ hemorrhaging have also been found in fish exposed to PCBs, particularly in fry. Physiological impairments caused by exposure to PCBs include endocrine system malfunction, decreases in fertility and other reproductive impairment, and biochemical changes. Chronic exposure of invertebrates to PCBs is known to cause reductions in growth and survival (Dillon et al., 1990; Eisler and Belisle, 1996; Ingersoll et al., 2000), even though invertebrates lack the aryl hydrocarbon receptor through which the coplanar PCB congeners act (West et al., 1997).

Table 6.1. Overview of adverse effects in fish caused by exposure to PCBs

Category	Response measure	Documented response	Example studies
Death	Mortality	Fry mortality and reduced egg hatchability	Nebeker et al., 1974; Eisler, 1986; Walker et al., 1994; Mac, 1988; Mac and Schwartz, 1992; Mac et al., 1993; Monosson et al., 1994
Cancer	Tumorigenesis	Tumor formation, especially in the liver	Hendricks et al., 1981, 1990; Baumann, 1992a, 1992b; Teh et al., 1997; Barron et al., 2000
		Enhanced tumorigenesis, via potentiation of estrogen responsiveness	Teh and Hinton, 1998
Disease	Immune system impairment	Reduced antibody levels	Thuvander and Carlstein, 1991
		Reduced immune cell activity	Jones et al., 1979; Arkoosh et al., 1994; Rice and Schlenk, 1995; Rice et al., 1996
		Reduced resistance to introduced bacteria	Jones et al., 1979
		Increased susceptibility to disease, parasitism, and cancer	Kahn and Thulin, 1991; Zelikoff, 1994; Anderson and Zeeman, 1995

Table 6.1. Overview of adverse effects in fish caused by exposure to PCBs (cont.)

Category	Response measure	Documented response	Example studies
Physical deformation	Deformities, especially in developing fry	Yolk sac edema	Walker et al., 1991
		Hemorrhaging in various organs	Spitsbergen et al., 1991; Walker and Peterson, 1992
		Skeletal deformation including domed skulls and craniofacial deformities	Walker et al., 1994
		Overt external malformations such as opercular defects	Helder, 1980, 1981
Physiological malfunction	Endocrine system and reproductive impairment	Promotion, inhibition, and mimicking of estrogens; competition with or alteration of thyroid hormone levels in the blood	Hansen, 1994; Gillesby and Zacharewski, 1998
		Altered sex determination and sex ratios	Matta et al., 1998
		Delayed maturity	Munkittrick et al., 1997
		Decreased fertility and egg production	Arcand-Hoy and Benson, 1998
		Gonadal abnormalities	Matta et al., 1998
		Reduced gonadal growth	Jobling et al., 1996
Physiological malfunction	Biochemical changes	Induced CYP1A activity, which is linked to tumorigenesis via metabolic activation	Van Der Oost et al., 1991; Wirgin et al., 1992; Stegeman and Hahn, 1994; Sleiderink et al., 1995; Eggens et al., 1996; Schrank et al., 1997; Courtenay et al., 1999

The approaches used in this chapter to assess injury to aquatic biota include comparisons of measured tissue PCB concentrations to toxicological benchmarks and in situ fish health studies (Table 6.2). The PCB concentrations measured in the last two decades in Kalamazoo River fish tissue are well below concentrations shown to cause direct mortality to adult fish (> 100 mg/kg ww) or adverse impacts to fish growth (> 50 mg/kg ww) (Niimi, 1996). Therefore, these endpoints are not assessed further. Instead, the Stage I assessment of injuries to fish focuses on embryomortality caused by PCBs deposited in fish eggs, and on histopathological or biochemical changes associated with PCB accumulation in fish livers. Fish are relatively sensitive to these

Table 6.2. Approaches to evaluate injury to aquatic biota

Injury definition	Stage I injury assessment approach	Chapter section
Cause the biological resource or its offspring to have undergone adverse changes in viability [43 C.F.R. § 11.62(f)(1)(i)].	Compare measured concentrations of PCBs in smallmouth bass and walleye eggs to toxicological benchmarks.	6.3
	Compare measured PCB concentrations in smallmouth bass livers to concentrations associated with adverse effects.	6.4
	Compare PCB concentrations and histopathological, immunological, and endocrine function parameters between fish in assessment locations and fish from reference locations.	6.5
	Compare surface sediment concentrations to consensus-based sediment effect concentrations for benthic invertebrates.	6.6

endpoints, and PCBs have been shown to cause these effects in laboratory studies and in field studies (Niimi, 1996). Embryomortality is assessed using a comparison of measured fish egg PCB concentrations with toxicity reference values from the literature. Histopathological and biochemical effects are assessed using a comparison of measured fish liver PCB concentrations with toxicity reference values from the literature and an evaluation of data collected on immunological, histopathological, and endocrine system function in KRE smallmouth bass. The data for these three evaluations come from the same study, a 1995 fish health study conducted by Stratus Consulting staff (Anderson et al., 2003). The Trustees are not aware of any other relevant studies of Kalamazoo River fish health or viability, nor of any other data on PCB concentrations in Kalamazoo River fish eggs or livers.

However, it should be noted that the data collected in the 1995 study are limited in species addressed, spatial coverage, sample size, and time period. A much larger amount of data is available for PCB concentrations (measured as Aroclors) in KRE fish fillets or whole bodies. However, using these data to assess potential injuries to fish necessarily involves uncertainties. No clear and generalizable relationship between PCB concentrations in fish fillet or whole bodies and sublethal adverse effects has been demonstrated across fish species and PCB composition (Monosson, 1999; Barron et al., 2002). Furthermore, the embryotoxicity of PCBs in fish eggs is believed to be caused by specific coplanar PCB congeners (see Section 6.3), and data from the 1995 study are the only data available on the concentrations of these congeners in KRE fish tissue. Furthermore, available data are not sufficient to reliably estimate across all of the sampled fish species and sample locations the concentrations of the coplanar PCB congeners in fish tissue from the available fish fillet and whole body Aroclor PCB data. Therefore, the Stage I Assessment of adverse effects injuries to fish is based on the site-specific data on PCB concentrations in fish eggs and livers from the 1995 study, and the larger dataset of PCB concentrations in fish fillets and whole bodies is not used.

This chapter also evaluates adverse effects to benthic invertebrates caused by releases of PCBs into the KRE (Table 6.2).

6.3 TCDD-Equivalents in Fish Eggs

In this section, measured concentrations of PCB congeners in fish eggs from the Kalamazoo River are compared to toxic thresholds for egg and fry mortality as a means of determining reproductive injuries to KRE fish. Fish eggs are the focus because developing embryos and fry are the most sensitive life stages to PCB toxicity, and because toxicity thresholds for PCBs in eggs are available from the literature (Cook et al., 1993; Peterson et al., 1993; Walker and Peterson, 1994; Eisler and Belisle, 1996). Many of the toxic effects of PCBs on developing fish are produced by specific coplanar PCB congeners that have a structure similar to that of 2,3,7,8-tetrachloro-*p*-dibenzodioxin (TCDD) and cause toxicity through a similar mechanism, and fish embryos and fry tend to be more sensitive to the TCDD-like toxicity of coplanar PCB congeners than they are to toxicity caused by other PCB congeners (Giesy and Kannan, 1998). TCDD has been studied in laboratory toxicity tests as the model compound for causing embryotoxicity to fish since it is the most potent compound at causing these effects. Therefore, to assess the reproductive toxicity of PCBs in fish eggs the Trustees use the dioxin equivalents approach, in which the concentrations of coplanar PCB congeners in fish eggs are converted to an equivalent concentration of TCDD. The dioxin equivalents approach to assessing the developmental toxicity of PCBs to fish is widely used and accepted (Van den Berg et al., 1998).

6.3.1 Data sources

The following data source was used in this evaluation:

- ▶ Study of fish health conducted by Stratus Consulting in 1995 (Anderson et al., 2003; analytical data in: A.D. Little, 1996; Midwest Research Institute, 1996).

As part of a larger study on smallmouth bass and walleye in the Kalamazoo River, Stratus Consulting collected eggs from 14 smallmouth bass and 7 walleye in the spring of 1995. Smallmouth bass samples were collected from below Morrow Dam, Verburg Park, Trowbridge Dam, and Lake Allegan. Walleye samples were collected at Lake Allegan. Eggs were taken from ripe females that were collected for tissue sampling. These egg samples were analyzed for a suite of 45 PCB congeners, including the coplanar PCB congeners PCB 77, 81, 126, and 169 (A.D. Little, 1996). However, the coplanar congener concentrations were below the detection limits of the analytical method used. Split samples of seven of the smallmouth bass and three of the walleye egg samples were analyzed separately for coplanar PCB congeners and polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) using a high-resolution, low-detection analytical technique designed specifically for these congeners (Midwest Research Institute, 1996). Since the high-resolution method has lower detection limits and fewer potential interferences than the method used by A.D. Little, only the Midwest Research Institute data were used for these congeners.

6.3.2 Concentrations in eggs causing toxicity

Poor correlations have been observed between reproductive effects in fish and total PCB concentrations, especially concentrations reported in eggs (Williams and Giesy, 1992). This may be because of the range in toxicity of different PCB congeners and the variable proportion of congeners in PCB mixtures. To account for the variations in toxicity of different congeners, a PCB congener mixture can be converted to a toxicologically equivalent concentration of TCDD using toxicity equivalency factors (TEFs). TEFs are specific to each congener, and they represent the toxicity of the congener relative to TCDD. TCDD is used to calculate TEFs because it is the most toxic of the planar halogenated aromatic compounds, a group which includes PCDDs, PCDFs, and the coplanar PCB congeners. The TEF of a congener is determined by dividing the concentration of TCDD causing a given adverse response (e.g., concentration at which 50% mortality occurs) by the concentration of a congener causing the same level of response:

$$\text{TEF} = \frac{\text{TCDD concentration}}{\text{PCB congener concentration}}.$$

An international group of toxicology experts (Van den Berg et al., 1998) developed TEFs for several PCB and PCDD, and PCDF congeners based on their relative toxicity to fish (Table 6.3). These values were derived from multiple studies and are not specific to any single fish species.¹ Using these TEFs, contaminant concentrations in fish eggs can be converted to TCDD-equivalents (TCDD-eq). TCDD-eq is the concentration of TCDD that would have the same potency as the congener mix in a sample.

A TCDD-eq for a sample is calculated by summing the product of each congener's measured concentration and TEF in the sample (Giesy et al., 1994):

$$\text{TCDD - eq} = \sum ([\text{congener}]_i \times \text{TEF}_i).$$

Calculated TCDD-eq concentrations can then be compared to TCDD concentrations that cause toxicity. Table 6.4 summarizes the toxic effects concentrations of TCDD in eggs, including no observed effect level (NOEL) and lowest observed effect level (LOEL). The concentrations at which effects are seen vary dramatically by species. The Giesy et al. (2002) chronic study on rainbow trout eggs produced the lowest LOEL in any published study to date of 0.1 pg/g ww in eggs. Other LOEL concentrations range from 40 pg/g for lake trout embryomortality to 3,330 pg/g for white perch embryomortality.

1. TEFs specific to embryomortality in rainbow trout are available from several studies (Walker and Peterson, 1991; Zabel et al., 1995). However, the appropriateness of the TEFs from the rainbow trout studies to smallmouth bass and walleye is difficult to assess. Therefore, the more general TEFs from Van den Berg et al. (1998) are used here.

Table 6.3. Toxic equivalency factors of PCB congeners, PCDDs, and PCDFs relative to TCDD in fish eggs

Congener	TEF
PCB congener 77	0.0001
PCB congener 81	0.0005
PCB congener 126	0.005
PCB congener 169	0.00005
2,3,7,8 TCDD	1
2,3,7,8 TCDF	0.05
1,2,3,7,8 PeCDD	1
1,2,3,7,8 PeCDF	0.05
2,3,4,7,8 PeCDF	0.5
1,2,3,4,7,8 HxCDD	0.5
1,2,3,6,7,8 HxCDD	0.01
1,2,3,7,8,9 HxCDD	0.01
1,2,3,4,7,8 HxCDF	0.1
1,2,3,6,7,8 HxCDF	0.1
1,2,3,7,8,9 HxCDF	0.1
2,3,4,6,7,8 HxCDF	0.1
1,2,3,4,6,7,8 HpCDD	0.001
1,2,3,4,6,7,8 HpCDF	0.01
1,2,3,4,7,8,9 HpCDF	0.01
OCDD	0
OCDF	0

Source: Van den Berg et al., 1998.

Toxicity data can also be expressed as an LC50, which is the concentration at which a 50% mortality rate occurs. LC50s for lake trout eggs range from 58 to 80 pg/g TCDD, depending on exposure route (Walker et al., 1994). An LC50 concentration for brook trout eggs has been reported at 200 pg/g (Walker and Peterson, 1994) and LC50s for rainbow trout eggs range from 230 to 488 pg/g (Walker and Peterson, 1991). It should be noted that no LC50 value was derived in the Giesy et al. (2002) study because no consistent dose-response relationship existed. Eggs of other species that have been tested experience 50% mortality at TCDD concentrations of 250 pg/g or greater (Monosson et al., 1994; Elonen et al., 1998; Toomey et al., 2001).

Because of the range of the values shown in Table 6.4 and uncertainties with regard to species sensitivity, a single toxicity value is not derived from the values shown in the table. Rather, the calculated TCDD-eq concentrations in KRE fish eggs are compared to the entire range of reported effects concentrations.

Table 6.4. Effect endpoints based on TCDD concentrations in fish eggs (pg/g ww)

Species	Exposure route	NOEL ^a	LOEL	LC _{egg-50} ^b	Source
Rainbow trout	Maternal, chronic exposure	—	0.1	—	Giesy et al., 2002
Lake trout	Maternal	23	50	58 (36-90)	Walker et al., 1994
Lake trout	Waterborne	34	40	69 (64-75)	Walker et al., 1994
Lake trout	Injection	44	55	80 (68-91)	Walker et al., 1994
Brook trout	Maternal	—	—	127 (106-145)	Johnson et al., 1998
Brook trout	Waterborne	135	185	200 (179-215)	Walker and Peterson, 1994
Rainbow trout, McConaughy strain	Injection	—	—	230 (208-249)	Walker and Peterson, 1991
Rainbow trout, Erwin strain	Injection	—	—	240 (209-264)	Walker and Peterson, 1991
Mummichog	Injection	—	—	250	Toomey et al., 2001
Rainbow trout, Arlee strain	Injection	—	—	374 (280-412)	Walker and Peterson, 1991
Rainbow trout, Eagle Lake strain	Injection	—	—	488 (338-580)	Walker and Peterson, 1991
Fathead minnow	Waterborne	235	435	539 (476-611)	Elonen et al., 1998
Channel catfish	Waterborne	385	855 ^c	644 (576-721)	Elonen et al., 1998
Lake herring	Waterborne	175	270	902 (783-1,040)	Elonen et al., 1998
Medaka	Waterborne	455	949	1,110 (932-1,320)	Elonen et al., 1998
White sucker	Waterborne	848	1,220	1,890 (1,760-2,030)	Elonen et al., 1998
Northern pike	Waterborne	1,190	1,800	2,460 (2,100-2,880)	Elonen et al., 1998
Zebrafish	Waterborne	424	2,000	2,610 (2,310-2,950)	Elonen et al., 1998
White perch ^d	Injection	—	3,330	—	Monosson et al., 1994

a. For white sucker, effect endpoint was decreased growth, for all other species effect endpoint was decreased survival.

b. Concentrations in eggs causing 50% mortality to fish at test termination. Values in parentheses represent the 95% confidence interval, where available.

c. LOEL represents lowest experimental concentration in eggs where a significant decrease in mortality was observed. The next lowest exposure group had a concentration in eggs of 385 pg/g. The LC_{egg-50} of 644 pg/g was derived from a best fit concentration-response curve, and falls between these two values.

d. Concentration based on exposure to PCB-77 and converted to TCDD-eq using a TEF of 0.0001.

6.3.3 Results

The concentrations of TCDD-eqs from PCB, PCDD, and PCDF congeners in smallmouth bass and walleye egg samples are presented in Table 6.5. The highest concentrations are seen in smallmouth bass eggs collected from Verburg Park in the city of Kalamazoo and from Lake Allegan. Walleye eggs were collected only from Lake Allegan, and have lower concentrations of TCDD-eqs than smallmouth bass eggs from the same location.

Table 6.5. TCDD-equivalents of PCBs and PCDDs/PCDFs in fish eggs

Fish ID	Species	Location	TCDD-eq from PCBs (pg/g)^a	TCDD-eq from PCDDs/PCDFs (pg/g)^b	Total TCDD-eq (pg/g)
SMMP01	Smallmouth bass	Downstream of Morrow Lake	11.24	15.02	26.26
SMMP02	Smallmouth bass	Downstream of Morrow Lake	5.66	NA	NA
SMMP03	Smallmouth bass	Downstream of Morrow Lake	5.50	8.44	13.94
SMMP04	Smallmouth bass	Downstream of Morrow Lake	5.27	NA	NA
SMVP01	Smallmouth bass	Verburg Park	11.10	14.32	25.42
SMVP02	Smallmouth bass	Verburg Park	15.87	NA	NA
SMVP03	Smallmouth bass	Verburg Park	0.91	NA	NA
SMVP04	Smallmouth bass	Verburg Park	15.83	31.59	47.42
SMBT01	Smallmouth bass	Trowbridge Dam	3.65	NA	NA
SMLA01	Smallmouth bass	Lake Allegan	14.93	12.37	27.30
SMLD01	Smallmouth bass	Downstream of Lake Allegan	11.55	NA	NA
SMLD02	Smallmouth bass	Downstream of Lake Allegan	14.45	20.42	34.87
SMLD03	Smallmouth bass	Downstream of Lake Allegan	6.65	NA	NA
SMLD04	Smallmouth bass	Downstream of Lake Allegan	6.73	24.49	31.23
KZAD01	Walleye	Downstream of Lake Allegan	3.92	4.46	8.37
KZAD02	Walleye	Downstream of Lake Allegan	0.26	NA	NA
KZAD03	Walleye	Downstream of Lake Allegan	0.20	NA	NA
KZAD04	Walleye	Downstream of Lake Allegan	2.31	NA	NA
KZAD05	Walleye	Downstream of Lake Allegan	2.88	3.93	6.80
KZAD06	Walleye	Downstream of Lake Allegan	2.72	3.55	6.27
KZAD07	Walleye	Downstream of Lake Allegan	1.42	NA	NA

NA = sample not analyzed for these contaminants.

a. Calculated using the sum of TCDD-equivalents of detected PCB congeners.

b. Calculated using the sum of TCDD-equivalents of detected PCDDs and PCDFs.

Source: Midwest Research Institute, 1996.

When the concentrations in Table 6.5 are compared to the toxic effects endpoint concentrations in Table 6.4, TCDD-eq concentrations from PCBs are all lower than toxic effects thresholds, with the exception of the rainbow trout LOEL from the chronic exposure study conducted by Giesy et al. (2002). All of the fish egg samples contained TCDD-eq from PCBs at concentrations that exceed the 0.1 pg/g LOEL from this study. When the TCDD-eq concentration from PCBs is added to the TCDD-eq concentration from PCDDs and PCDFs, the total TCDD-eq concentrations are still less than the LOELs reported in Table 6.4, with the exception of the 0.1 pg/g LOEL from the Giesy et al. (2002) study.

It is difficult to draw conclusions regarding embryomortality injuries to fish from the available fish egg TCDD-eq data. Relevant data are available only for smallmouth bass and walleye eggs, and these data show that these two species are exposed to PCBs at concentrations lower than most of the available thresholds for embryomortality. However, the sensitivity of these two species (and most other KRE species) to embryomortality caused by PCBs or TCDD is not known. Fish species display widely variable sensitivity to TCDD toxicity. Lake trout are generally believed to be the most sensitive, although research by Cook (2000) suggests that bull trout may be more sensitive. In addition, the 0.1 pg/g long-term exposure LOEL for TCDD in rainbow trout eggs from the Giesy et al. (2002) study suggests that all of the sampled fish eggs contain PCBs at concentrations well above a LOEL. Finally, egg PCB data are available for KRE walleye and smallmouth bass only from 1995, and PCB exposures of these species (and others) in the past was higher than it was in 1995.

Therefore, the available data may indicate embryomortality injuries to KRE fish from PCB egg exposure. Additional information on the sensitivity of KRE species to PCB embryotoxicity and on the applicability of the Giesy et al. (2002) study results to KRE fish would help reduce the uncertainty in the injury determination.

6.4 Total PCBs in Smallmouth Bass Livers

In this section, PCB concentrations measured in livers of Kalamazoo River smallmouth bass are compared to concentrations associated with adverse sublethal effects from published literature, and to concentrations found to be associated with histopathological effects in walleye exposed to PCBs in Green Bay, Wisconsin. The amount of data available on PCB concentrations in KRE fish livers is small relative to the amount of whole body or fillet PCB concentration data. However, the liver is a site both of PCB accumulation and toxic action, and the relationship between adverse effects and PCB concentrations measured in livers is expected to be more robust than that for PCB concentrations in whole bodies or fillets (Monosson, 1999). Furthermore, although PCB concentrations measured in whole bodies or fillets could be converted to an estimated liver concentration on a lipid normalized basis (Russell et al., 1999), measurements of liver lipid content, which would be necessary to convert lipid normalized PCB concentrations in livers to equivalent wet weight concentrations for comparison to toxicological benchmarks, are not available for the fish sampled for whole body or fillet PCB concentrations. The liver lipid fractions measured in the Trustees' 1995 study are highly variable, making it unreliable to simply assume a single lipid fraction in all KRE fish livers. Therefore, the Trustees rely exclusively on the available liver PCB data for KRE fish from the Trustees' 1995 study.

6.4.1 Data sources

The following data source is used in this section:

- ▶ Study of fish health conducted by Stratus Consulting in 1995 (Anderson et al., 2003; analytical data in A.D. Little, 1996).

Twenty-nine smallmouth bass from assessment areas of the Kalamazoo River were collected and analyzed for PCBs (A.D. Little, 1996; State of Michigan Community Public Health Agency, 1996; Anderson et al., 2003). Livers from nine fish collected at D Avenue in Kalamazoo were collected and analyzed for 18 PCB congeners (8, 18, 28, 44, 52, 66, 101, 105, 118, 128, 138, 153, 170, 180, 187, 195, 206, 209; A.D. Little, 1996). Livers were also analyzed for coplanar PCB congeners (PCB 77, 81, 126, 169; Midwest Research Institute, 1996); however, there was insufficient tissue mass for obtaining detectable concentrations of these congeners at detection limits ranging from 0.0004 to 0.007 mg/kg ww (Anderson et al., 2003). The sum of the 18 PCB congeners was calculated using a value of zero where individual congeners were not detected.² An estimate of total PCB concentration in each liver sample was made from the sum of 18 measured congeners using a linear regression model.³

2. Non-detects were infrequent and occurred at detection limits less than 0.005 mg/kg, except for congener number 209, decachlorobiphenyl. This congener was not detected in any sample at detection limits up to 0.12 mg/kg, but this congener occurs at non-measurable amounts in Aroclor mixtures (Frame et al., 1996). Therefore, the use of zero for non-detects introduces only negligible bias to the analysis.

3. The relationship between the sum of 18 measured congeners and total PCBs was developed from whole body fish tissue data from the Kalamazoo River, described in Section 7.4.1 of this report (Michigan State University Aquatic Toxicology Laboratory, 2002j). In that study seventy-six PCB congeners were measured in fish tissue samples, including the 18 congeners measured in smallmouth bass livers in the Trustees' study. The data from the Michigan State University study can thus be used to develop a relationship for Kalamazoo River fish between the sum of the 18 congeners and the sum of the 76 congeners. The relationship is characterized by the equation: $\text{sum}_{76 \text{ congeners}}(\text{mg/kg}) = 2.18 \times \text{sum}_{18 \text{ congeners}}(\text{mg/kg})$ (multiple R-squared = 0.9923, residual standard error = 0.458 mg/kg). The Trustees assumed that these 76 congeners represented total PCBs, and used this equation (forced through an intercept of zero) to estimate total PCBs in the Kalamazoo River fish sampled by the Trustees. The Trustees evaluated the assumption that the sum of the 76 congeners can be used to represent total PCBs by reviewing PCB concentration data for 112 congeners in biota collected in the Lower Fox River and Green Bay, Wisconsin. Although the Wisconsin biota dataset contained some different coelutions of PCB congeners, 72 of the 76 congeners reported in the Michigan State University Kalamazoo River data also were measured in the Wisconsin study. On average, the sum of the 72 congeners was approximately 91% of the sum of the 112 congeners measured in the Wisconsin study. Additionally, Laurenstein et al. (1993) report that total PCB concentrations as the sum of homologues are approximately two times the sum of the 18 PCB congeners which were measured in the Kalamazoo River smallmouth bass livers.

6.4.2 Toxicological benchmarks

Table 6.6 lists available studies from the literature that exposed fish to PCBs in a laboratory setting and observed adverse sublethal effects that are associated with a PCB concentration accumulated in the liver. There are few toxicological benchmark values available for PCBs in fish livers. Although numerous toxicological studies are available from which fish liver PCB concentrations associated with toxicity could be developed, most of the studies are on individual PCB congeners, from field studies where other contaminants are also present, or rely on rough approximations to estimate liver PCB concentrations. Table 6.6 lists the results from available laboratory studies where fish were dosed with a PCB mixture and LOEL values for measured PCB concentrations in fish livers can be obtained. Other studies are available with TCDD (e.g., Walter et al., 2000), but KRE fish contaminant data are insufficient to estimate TCDD-eq in fish livers. The effects concentrations for PCBs in fish liver shown in Table 6.6 range from approximately 24.2 mg/kg for reduced fecundity, egg hatching rate, and alterations in liver microstructure in barbel to 45-100 mg/kg for alterations in hormone levels and testicular growth in Atlantic cod. Based on the studies listed in Table 6.6, the Trustees use the lowest reported LOEL concentration of 24.2 mg/kg PCBs in fish liver as a laboratory study-based injury threshold for fish. However, it should be noted that few directly relevant studies have been reported in the literature.

Table 6.6. PCB concentrations in livers of adult fish associated with adverse effects in laboratory studies^a

Species	PCB mixture	Liver PCB concentration (mg/kg ww)	Adverse effect	Study
Barbel	Aroclor 1260	24.2 ^b	Reduced fecundity; reducing egg hatching rate; liver ultrastructure alterations	Hugla and Thome, 1999
Atlantic croaker	Aroclor 1254	25	Decreased serotonin (by 38%), dopamine (by 35%), testicular growth (by 79%), testosterone (by 60%), 11-ketotestosterone (by 73%); inhibition of gonadotropin secretion	Khan and Thomas, 1996, 1997
Atlantic cod	Aroclor 1254	45-100	Abnormal testes; altered steroid hormone metabolism; decreased spermatogenic elements	Sangalang et al., 1981; Freeman et al., 1982
Chinook salmon	Aroclor 1254	54	Immune system suppression	Arkoosh et al., 1994

a. Only studies where PCBs were directly measured in the liver are shown. Data from Black et al. (1998), in which killifish were injected with PCBs, are not included because the injection was done with a unique mixture of 8 PCB congeners and it is difficult to compare the results with the measurements of PCBs in KRE fish. A qualitative evaluation of the data indicates that the fish in the Black et al. (1998) study were exposed to higher levels of PCBs than measured in KRE bass.

b. Value is calculated from a mean dry weight concentration of 6.3 mg/kg dw from the paper using an assumed moisture content of 74% (Connolly et al., 1992).

Most of the studies listed in Table 6.6 used Aroclor 1254 as the PCB dosing mixture. Although the specific mixture of PCB congeners in the KRE is not the same as in Aroclor 1254, the general pattern of the congeners present in fish tends to match the general pattern of Aroclor 1254 (Blasland, Bouck & Lee, 2000c). This occurs even though the bulk of the PCBs released from the paper company facilities was Aroclor 1242 because of changes in the congener pattern that can occur once the PCBs were released (Erickson, 1997; Cacula et al., 2002). Therefore, although the PCB mixture used in the laboratory does not match the mixture that was originally released to the KRE, the general congener pattern of the mixtures used in the laboratory is similar to that found in fish from the KRE.

Additional benchmarks are drawn from a field study on adverse effects in walleye exposed to PCBs in the Fox River and Green Bay, Wisconsin (Barron et al., 2000). The Fox River/Green Bay environment is similar to the KRE in that it has been contaminated by releases of Aroclor 1242 from paper companies involved in the production or deinking of carbonless copy paper containing PCBs. This study found a significantly greater prevalence of cancerous tumors and precancerous lesions in the livers of walleye from assessment areas than in the livers of walleye from reference areas in Lake Winnebago and Patten Lake ($p = 0.004$). After removal of sections for histopathological analysis and disease screening, the remaining liver samples were analyzed for PCB concentrations as Aroclors. Mean concentrations of total PCBs in the livers of Fox River and Green Bay fish ranged from 3.6 to 6.4 mg/kg ww, and were significantly greater than concentrations in reference areas ($p < 0.01$) (Table 6.7). This range of PCB concentrations in fish livers associated with adverse effects is similar to the results of Mikaelian et al. (2002), who observed liver tumors and pre-neoplastic lesions in lake whitefish from the St. Lawrence River with mean liver PCB concentrations of 1.75 mg/kg (quantified as Aroclor 1254).

6.4.3 Results

Estimated total PCB concentrations in the 9 smallmouth bass livers collected from Kalamazoo River fish range from 1.68 to 12.80 mg/kg ww with an arithmetic mean concentration of 4.77 mg/kg ww and a geometric mean concentration of 3.83 mg/kg ww (Table 6.8). These concentrations are less than liver Aroclor 1254 concentrations reported in laboratory studies as being associated with adverse sublethal effects (Table 6.6). However, few laboratory studies are available that have assessed the sublethal impacts of PCBs to fish and simultaneously measured liver PCB concentrations, and no studies are available for species of interest in the KRE. Thus, a comparison of measured PCB concentrations in KRE fish livers with laboratory toxicity data suggests that concentrations in KRE fish are less than effects levels, but the comparison is limited in scope.

Table 6.7. Mean and SD of concentrations of total PCBs in walleye livers collected in the Lower Fox River and Green Bay, Wisconsin, in 1996 and 1997, and associated prevalence of foci of cellular alteration and hepatic tumors

Sample location	Number of samples	Total PCB mg/kg ww mean (SD)	Total PCB mg/kg (lipid) mean (SD)	Prevalence of foci of cellular alteration	Prevalence of hepatic tumors
<i>Reference areas</i>					
Lake Winnebago	12	0.94 (0.55)	27.0 (18.0)	10%	0%
Patten Lake	13	0.02 (0.07)	0.47 (1.7)	0%	0%
<i>Assessment areas: Fox River/Green Bay</i>					
Lower Fox River	20 ^a	6.4 (1.7)	61.8 (13.7)	8%	4%
Lower Green Bay	16	4.3 (3.4)	44.3 (37.6)	25%	6%
Eastern Green Bay	21	3.6 (2.7)	36.4 (17.9)	24%	5%
Western Green Bay	18	4.0 (2.0)	33.3 (10.2)	33%	11%
Upper Green Bay	4	4.4 (1.4)	32.4 (9.5)	0%	50%

a. Number of samples indicates the number of separate sample analyses. Analyses were of 19 individual fish livers and 1 composite sample of livers from four fish.

Source: Barron et al., 2000.

Table 6.8. Total PCB concentrations in smallmouth bass livers from D Avenue, Kalamazoo

Fish ID	Sum of 18 PCB congeners in liver (mg/kg ww)	Estimated total PCBs in liver (mg/kg ww)
SBDA01	1.90	4.15
SBDA02	1.03	2.25
SBDA05	5.87	12.80
SBDA06	3.14	6.85
SBDA09	0.80	1.68
SBDA10	1.87	4.07
SBDA11	0.93	2.02
SBDA12	3.16	6.88
SBDA15	1.04	2.26
Arithmetic mean	2.19	4.77
Geometric mean	1.76	3.83

Source: A.D. Little, 1996.

The mean estimated concentration of total PCBs in Kalamazoo River smallmouth bass livers, 4.70 mg/kg ww, is within the range of the lowest mean concentrations observed in the livers of Green Bay walleye with increased prevalence of foci of cellular alteration and hepatic tumors (3.6 mg/kg ww in livers of fish from eastern Green Bay to 6.4 mg/kg ww in livers of fish from the lower Fox River).

These data indicate that the PCB concentrations in KRE smallmouth bass livers are similar to those observed in Green Bay walleye (Figure 6.1). Therefore, depending on the PCB sensitivity of smallmouth bass compared to walleye, KRE smallmouth bass may be exposed to PCBs at concentrations associated with liver tumors and pre-tumors. In the next section, the results of an investigation of the health of KRE smallmouth bass are presented and discussed.

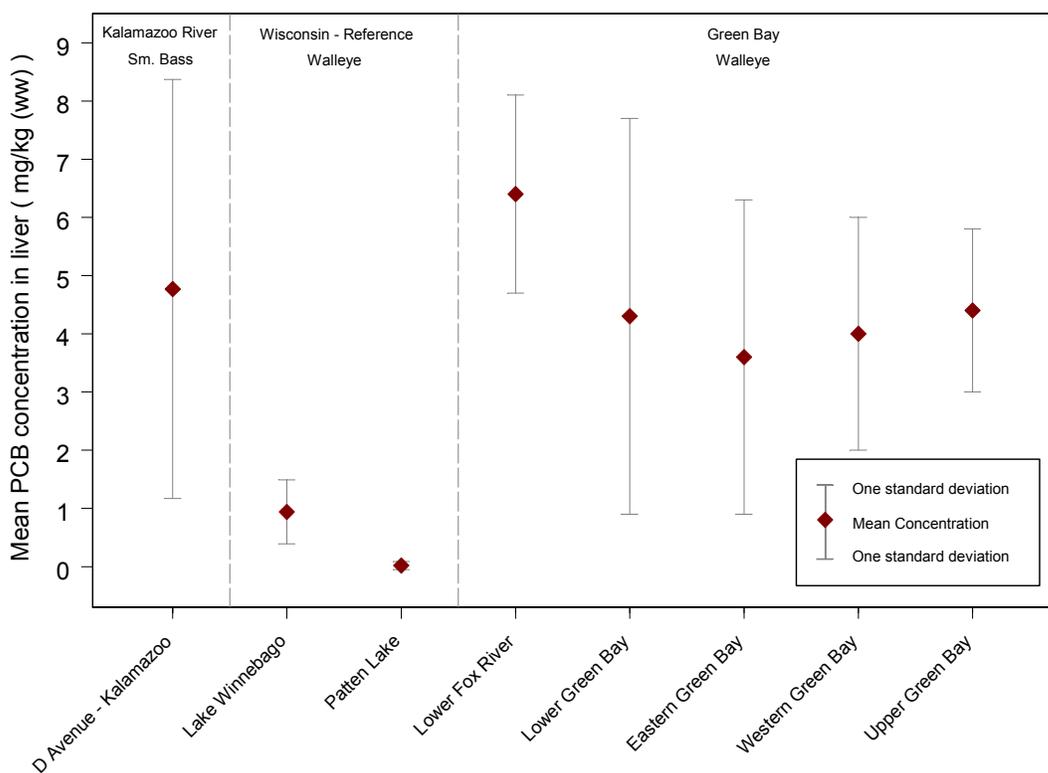


Figure 6.1. Mean and SD of concentrations of estimated total PCBs ww in smallmouth bass livers collected in the Kalamazoo River in 1995 and total PCBs in walleye livers collected in the Lower Fox River and Green Bay, Wisconsin, in 1996 and 1997.

Sources: A.D. Little, 1996; Barron et al., 2000.

6.5 Smallmouth Bass Condition, Biochemical, and Histopathological Status

6.5.1 Data sources

The following data source is described and discussed in this section:

- ▶ Study of smallmouth bass health conducted by the Trustees in 1995 (Anderson et al., 2003).

Thirty smallmouth bass, aged 3 to 5 years, were collected from the Kalamazoo River in 1995 using electroshocking (Anderson et al., 2003). Half of the bass were sampled from an upstream area near Ceresco, and half were sampled from an area near D Avenue in Kalamazoo. The upstream area served as a reference area because sediments of the upstream area were known to be relatively uncontaminated with PCBs (Blasland, Bouck & Lee, 2000b). The presence of Morrow Dam (Figure 1.1) prevents fish from migrating from the assessment area to the upstream area, but downstream migration is not prevented.

Bass were euthanized and samples were collected for a suite of analyses designed to evaluate health parameters that can be adversely affected by exposure to PCBs. Blood was collected for analysis of plasma vitellogenin, a protein produced by females that is used in the formation of eggs and is an indicator of estrogenic or antiestrogenic effects in fish. Liver tissue samples were analyzed for ethoxyresorufin-O-deethylase (EROD) activity, an enzyme that is involved in detoxifying pollutants, and for the enzyme cytochrome P4501A (CYP1A), a protein involved in the metabolism of planar aromatic hydrocarbons, including PCBs. Liver and spleen tissue samples were analyzed for superoxide dismutase activity (SOD), a critical antioxidant enzyme. Finally, various tissues (gill, liver, spleen, head kidney, trunk kidney, thyroid, and gonad) were histopathologically examined. Fillet samples were collected for analysis of PCB as Aroclors. Additionally, the livers of 9 bass from the assessment area and 10 bass from the reference area were analyzed for the concentrations of 18 PCB congeners. Total PCBs in livers was estimated from the sum of the 18 congeners using the method described in Section 6.4.1. PCB concentrations in both livers and fillets were normalized by lipid content and both wet weight and lipid normalized concentrations were used in further analyses.

6.5.2 Summary of results

Means of sum of PCB congeners in livers and total PCBs in fillets are significantly ($p < 0.001$) elevated in the assessment area compared to the reference area (Table 6.9). When PCB concentrations are lipid-normalized, liver sum of PCB congeners are positively correlated with fillet total PCBs across all fish ($p < 0.001$, $\rho = 0.85$).⁴

Table 6.9. PCB concentrations in liver and fillet of smallmouth bass from the Kalamazoo River, 1995

Location	N ^a	Liver				N ^a	Fillet	
		Sum of 18 PCB congeners (mg/kg ww) ^b		Estimated total PCB concentration (mg/kg ww) ^c			Total PCB concentration in fillet (mg/kg ww) ^d	
		Mean	SD	Mean	SD		Mean	SD
Ceresco (reference)	10	0.26	0.10	0.60	0.21	15	0.09	0.04
D Avenue (assessment)	9	2.19 ^e	1.65	4.77	3.60	15	1.01 ^e	0.43

a. N = number of samples. Fewer liver samples were obtained for PCB analysis than for fillet analysis because liver mass was sometimes insufficient.

b. As reported in Anderson et al., 2003.

c. Total PCB concentration estimated from results of 18 congeners (see Section 6.4.1 for details).

d. Skin-on fillet total PCBs estimated by Aroclor method.

e. Significantly different than reference ($p < 0.001$).

Source: Anderson et al., 2003.

Bass in the assessment area have statistically significant differences in body condition and biochemical status compared to reference area bass (Table 6.10). Overall indices of fish health such as liver organosomatic index (liver weight as a percentage of total body weight) and condition factor (body weight divided by length cubed) are significantly lower in assessment area bass compared to reference area bass. In pooled samples from both sites, these indices were negatively correlated with liver and fillet PCB concentrations.

4. ρ = Spearman's rank correlation.

Table 6.10. Summary of body condition and biochemical results for KRE smallmouth bass and correlations with tissue PCB concentrations

Parameter	Statistical significance ^{a,b}	Significant correlations with sum of PCB congeners on wet weight and lipid bases in liver		Significant correlations with total PCBs on wet weight and lipid bases in fillet	
		ww	Lipid	ww	Lipid
General indices					
Length	Not sig.	— ^c	—	—	—
Weight	Not sig.	—	—	—	—
Gender	Not sig.	—	—	—	—
Age	Not sig.	—	—	—	—
Spleen organosomatic index ^d	Not sig.	—	—	—	—
Testis organosomatic index ^d	Not sig.	—	—	—	—
Ovary organosomatic index ^d	Not sig.	—	—	—	—
Liver organosomatic index ^d	a < r (p < 0.05)	Negative (p = 0.05, rho = -0.45)	Not sig.	Not sig.	Negative (p < 0.01, rho = -0.50)
Condition factor	a < r (p < 0.001)	Negative (p = 0.001, rho = -0.77)	Negative (p < 0.01, rho = -0.64)	Negative (p < 0.02, rho = -0.46)	Negative (p < 0.001, rho = -0.70)
Biochemical indices					
Liver EROD (males)	a < r (p = 0.003)	Negative (p = 0.022, rho = -0.76)	Negative (p = 0.035, rho = -0.70)	Negative (p = 0.032, rho = -0.57)	Negative (p = 0.043, rho = -0.54)
Liver EROD (females)	Not sig.	Not sig.	Not sig.	Not sig.	Not sig.
Liver CYP1A concentration (males)	Not sig.	Not sig.	Not sig.	Not sig.	Not sig.
Liver CYP1A concentration (females)	Not sig.	Not sig.	Not sig.	Not sig.	Not sig.

Table 6.10. Summary of body condition and biochemical results for KRE smallmouth bass and correlations with tissue PCB concentrations (cont.)

Parameter	Statistical significance ^{a,b}	Significant correlations with sum of PCB congeners on wet weight and lipid bases in liver		Significant correlations with total PCBs on wet weight and lipid bases in fillet	
		ww	Lipid	ww	Lipid
SOD activity in spleen	a < r (p = 0.001)	Negative (p = 0.016, rho = -0.56)	Negative (p = 0.029, rho = -0.51)	Not sig.	Negative (p = 0.03, rho = -0.40)
SOD activity in liver	a < r (p = 0.015)	Negative (p = 0.026, rho = -0.52)	Negative (p = 0.005, rho = -0.66)	Negative (p = 0.002, rho = -0.58)	Negative (p = 0.001, rho = -0.60)
Plasma vitellogenin (females)	a < r (p < 0.05)	Not sig.	Negative (p = 0.035, rho = -0.79) ^e	—	—

a. a = assessment, r = reference.

b. Not sig. = not significant, p > 0.05.

c. — = not reported.

d. Organosomatic index = (organ weight/body weight) × 100.

e. Correlation using only females with mature oocytes (three fish from reference area were eliminated from this analysis because they contained predominately immature oocytes).

Source: Anderson et al., 2003.

Biochemical indices previously shown to be associated with or modulated by PCBs include liver EROD activity, liver CYP1A relative protein concentrations, liver and spleen SOD activity, and plasma vitellogenin concentrations (Anderson et al., 2003). Liver EROD activity in male assessment area bass is significantly lower than in male reference area bass, but there is no significant difference in female bass (Table 6.10). Liver EROD activity in males is significantly lower in the assessment area and is negatively correlated with liver and fillet PCB concentrations. CYP1A protein expression is not significantly different between the sampling locations. Fish exposed to PCBs generally exhibit EROD induction (elevated EROD activity) (Barron et al., 2000) and CYP1A protein expression (Anderson et al., 2003). However, extended exposure to PCBs has also been associated with low or depressed EROD activity (Besselink et al., 1998). The depressed concentrations of these detoxification enzymes may be associated with negative consequences in fish such as accelerated accumulation of PCBs in tissue and the lessened ability to detoxify other contaminant stressors (Anderson et al., 2003).

SOD is a critical antioxidant enzyme involved in the conversion of superoxide anion to hydrogen peroxide and water, and can be elevated in fish exposed to oxidant stress. Liver and spleen SOD are significantly lower in assessment area bass of both genders relative to reference area bass. Liver and spleen SOD activity are negatively correlated with liver and fillet PCB concentrations, suggesting that PCBs may impair detoxification (Anderson et al., 2003).

Levels of plasma vitellogenin, an egg-yolk precursor protein, are significantly lower in females of equivalent reproductive status in the assessment area than in females in the reference area, and concentrations in pooled females from both sites are negatively correlated with lipid normalized PCBs in the liver (Table 6.10). This result suggests that PCBs may be having an antiestrogenic influence on female bass in the Kalamazoo River.

Analysis of histopathological lesions and parameters also shows statistically significant differences between reference and assessment area bass (Table 6.11). Liver glycogen depletion, which is a common lesion observed in fish under a variety of stressful conditions, is more severe in assessment area fish compared to reference area fish. Macrophage aggregates in the liver, head kidneys, and female ovaries are also more severe in assessment area bass than in reference area bass. These lesions are similarly considered nonspecific indicators of contaminant exposure or other stressors.

Table 6.11. Summary of selected histopathology results of KRE smallmouth bass study, 1995

Parameter	Difference between assessment and reference^a
Severity of liver glycogen depletion	a > r (p = 0.019)
Severity of macrophage aggregates in liver	a > r (p = 0.017)
Severity of macrophage aggregates in head kidney	a > r (p = 0.006)
Severity of macrophage aggregates in posterior ovary (females)	a > r (p = 0.028)
Frequency of liver neoplasms	a = 1, r = 0 (not sig., p > 0.5)
Frequency of liver foci of cellular alteration	a = 3, r = 0 (not sig., p = 0.224)
Frequency of stomach foci of cellular alteration	a = 1, r = 0 (not sig., p > 0.5)
Severity of foreign body granuloma in liver	a < r (p = 0.025)
Severity of foreign body granuloma in head kidney	a < r (p = 0.002)
Severity of foreign body granuloma in trunk kidney	a < r (p = 0.01)
Severity of eosinophilic granular leukocytes in thyroid	a < r (p = 0.005)

a. a = assessment, r = reference.
Source: Anderson et al., 2003.

Preneoplastic (foci of cellular alteration) and neoplastic lesions are found only in bass from the assessment area, although the incidences are not statistically significant between the assessment and reference areas. Of the 15 fish collected from the assessment area, three bass (two females and one male) had foci of cellular alteration in the liver and one male possessed a large neoplastic hepatocellular adenoma in the liver and multiple foci of cellular alteration in the stomach. In the Green Bay study described in the previous section, the incidence of these endpoints (preneoplastic and neoplastic lesions) was also higher in PCB-exposed fish than in reference area fish.

Some histopathological parameters are more severe in reference area bass than in assessment area bass (Table 6.11). Foreign body granulomas in the liver, head kidney, and trunk kidney are more prevalent and severe in the reference area than in the assessment area. The prevalence and severity of eosinophilic granular leukocytes in the thyroid is also significantly higher in reference area bass than in assessment area bass. It is notable that many of the foreign body granulomas observed in reference bass were collocated with parasites.

The types of lesions observed in Kalamazoo River assessment area bass are consistent with those seen in other sites where freshwater fish have been exposed to PCBs (Dey et al., 1993; Schrank et al., 1997; Teh et al., 1997; Barron et al., 2000), and there is laboratory evidence that PCBs act as tumor promoters in fish (Bailey et al., 1987; Hendricks et al., 1990; Fabacher et al., 1991).

This study shows that a suite of organosomatic, biochemical and histopathological indices are spatially and statistically associated with PCB exposure in KRE smallmouth bass. Although cause and effect were not established by this study, the results of this study suggest that smallmouth bass within the assessment area of the Kalamazoo River may be adversely affected by exposure to PCBs. Furthermore, the results of the previous section show that PCB concentrations in KRE smallmouth bass livers are within the range of those in Green Bay walleye that also had adverse histopathological changes. Although this study sampled smallmouth bass from only one assessment location, sediment contamination extends throughout the Portage Creek and Kalamazoo River downstream of PRP facilities. Thus, to the extent that the adverse effects observed in the smallmouth bass collected near D Avenue in Kalamazoo are related to PCB exposure, bass in other locations of the river are most likely similarly affected, and the adverse effects would have been occurring for many years prior to the 1995 study.

6.6 Effects on Benthic Invertebrates

Section 4.3 presents a comparison of PCB concentrations in surficial sediment of the Kalamazoo River and Portage Creek with sediment thresholds developed to predict adverse toxicological effects to benthic invertebrates. The comparison presented in that section shows that PCB concentrations in sediments of the KRE exceed concentrations predicted to cause adverse

impacts to benthic invertebrates, leading to the conclusion that PCB sediment concentrations are sufficient to cause injury to benthic invertebrates.

There are no data available to directly assess the effects of PCBs in the KRE on benthic invertebrates. There are no co-located PCB concentrations and benthic invertebrate community data available, nor are any sediment toxicity tests available. Nevertheless, because surficial sediment PCB concentrations exceed concentrations predicted to cause adverse effects to benthic invertebrates, the Trustees conclude that benthic invertebrates are injured in the Kalamazoo River and Portage Creek.

6.7 Conclusions

Available data on TCDD-eq concentrations from PCBs in KRE smallmouth bass and walleye eggs are inconclusive regarding whether the PCBs are sufficient to cause embryomortality in these fish. The measured TCDD-eq concentrations in the KRE bass and walleye egg samples are less than most effects thresholds. However, the concentrations are greater than the threshold concentration from one study in which rainbow trout were chronically exposed to PCBs prior to egg laying. The sensitivity of walleye and smallmouth bass eggs to embryotoxicity from PCB exposure is not known. Therefore, the available data are inconclusive.

PCB concentrations in smallmouth bass livers suggest that the bass may be injured [per the definition in 43 C.F.R. § 11.62(f)(1)(i)]. Concentrations were generally below literature effects levels, but few studies are available for direct comparison. However, many samples had PCB concentrations in the range of concentrations associated with cancerous tumors and precancerous lesions from a field study in Green Bay, Wisconsin (Barron et al., 2000).

Smallmouth bass collected from the Kalamazoo River assessment area had significant alterations of body condition, endocrine function, and histopathological status compared to those collected in upstream reference areas. The types of biochemical responses and histopathological observations were consistent with those seen in other sites where freshwater fish have been exposed to PCBs. These observations, in combination with the comparison of PCB concentrations in fish livers from the KRE with those from Green Bay, suggest that smallmouth bass (and potentially other species, as well) are exposed to PCBs at concentrations sufficient to cause adverse health effects. To the extent that the alterations observed in the smallmouth bass are associated with PCB exposure, it is likely that smallmouth bass throughout the Kalamazoo River downstream of PRP facilities have been experiencing similar effects from the time of the initial releases. However, the available data do not allow for a determination of injury to be made with a reasonable degree of certainty, and additional studies may be necessary to evaluate and define the scope of injuries. The Trustees conclude that smallmouth bass may be and may have been injured as a result of PCB releases.

Additionally, PCB concentrations in surface sediments are high enough to cause injury to benthic invertebrates. PCB concentrations exceed toxic threshold concentrations for benthic invertebrates by up to two orders of magnitude. Thus, the Trustees conclude that benthic invertebrates are injured throughout the Kalamazoo River and Portage Creek downstream of PRP facilities.