EPA Approval Received: May 24, 2001

Eleven Total Maximum Daily Loads for Legacy Pollutants in Streams and Reservoirs in Fort Worth

For Segments 0806, 0806A, 0806B, 0829, and 0829A

Prepared by the:
Field Operations Division, Region 4
Strategic Assessment Division, TMDL Team

TEXAS NATURAL RESOURCE CONSERVATION COMMISSION

Eleven Total Maximum Daily Loads for Legacy Pollutants in Streams and Reservoirs in Fort Worth

Introduction

Section 303(d) of the Clean Water Act requires all states to identify waters that do not meet, or are not expected to meet, applicable water quality standards. For each listed water body that does not meet a standard, states must develop a total maximum daily load (TMDL) for each pollutant that has been identified as contributing to the impairment of water quality in that water body. The Texas Natural Resource Conservation Commission (TNRCC) is responsible for ensuring that TMDLs are developed for impaired surface waters in Texas.

In simple terms, a TMDL is a quantitative plan that determines the amount of a particular pollutant that a water body can receive and still meet its applicable water quality standards. In other words, TMDLs are the best possible estimates of the assimilative capacity of the water body for a pollutant under consideration. A TMDL is commonly expressed as a load, with units of mass per time period, but may also be expressed in other ways. TMDLs must also estimate how much the pollutant load needs to be reduced from current levels in order to achieve water quality standards.

The Total Maximum Daily Load Program, a major component of Texas' statewide watershed management approach, addresses impaired or threatened streams, reservoirs, lakes, bays, and estuaries (water bodies) in or bordering the state of Texas. The primary objective of the TMDL Program is to restore and maintain the beneficial uses (such as drinking water, recreation, support of aquatic life, or fishing) of impaired or threatened water bodies.

The ultimate goal of these TMDLs is the reduction of fish tissue contaminant concentrations to levels that constitute an acceptable risk to consumers, allowing TDH to remove the bans on fish consumption and the beneficial use to be restored to these water bodies.

Section 303(d) of the Clean Water Act and the U.S. Environmental Protection Agency's (EPA) implementing regulations (40 Code of Federal Regulations, Section 130) describe the statutory and regulatory requirements for acceptable TMDLs. The TNRCC guidance document, *Developing Total Maximum Daily Load Projects in Texas* (GI-250, 1999), further refines the process for Texas. This TMDL document has been prepared in accordance with these guidelines, and is composed of the following six elements:

- Problem Definition
- Endpoint Identification
- Source Analysis
- Linkage Between Sources and Receiving Waters

- Margin of Safety
- Pollutant Load Allocation

This TMDL document was prepared by:

- Region 4 of the Field Operations Division of the Office of Compliance and Enforcement of the Texas Natural Resource Conservation Commission, and
- the TMDL Team in the Strategic Assessment Division of the Office of Environmental Policy, Analysis, and Assessment of the Texas Natural Resource Conservation Commission.

It was adopted by the Texas Natural Resource Conservation Commission on November 17, 2000. Upon adoption, the TMDL became part of the state Water Quality Management Plan. The Texas Natural Resource Conservation Commission will use this document in reviewing and making determinations on applications for storm water permits and in its nonpoint source pollution abatement programs.

Background Information

These TMDLs address the contamination of fish tissue by several legacy pollutants in:

- portions of two classified segments of the Trinity River, and
- three unclassified urban lakes in Tarrant County, north-central Texas.

Legacy pollutant is a collective term used to describe substances whose use has been banned or severely restricted by the U.S. Environmental Protection Agency (EPA). Because of their slow rate of decomposition, these substances frequently remain at elevated levels in the environment for many years after their widespread use has ended. No additional loading of legacy pollutants is allowed or expected due to the EPA restrictions. Gradual declines in environmental legacy pollutant concentrations occur as a result of natural attenuation processes.

EPA guidance (*Draft Guidance for Water Quality-based Decisions: The TMDL Process, Second Edition, EPA 841-D-99-001, 1999*) on the development of TMDLs offers flexibility in addressing particular situations and unusual circumstances, allowing States the discretion to adopt different approaches where appropriate. The guidance states that the allowable pollutant load "must be expressed in a manner ... that represents attainment and maintenance of water quality standards." The guidance allows for the use of a surrogate target for situations where "no .... quantifiable pollutant load can be used to .... express the TMDL."

In preparing these TMDLs for legacy pollutants, the TNRCC has refined the typical loading allocation approach of a TMDL, which limits the amount of a pollutant that can be added to an impaired water body. Because these legacy pollutants are already restricted, and no significant additional loading is expected, these TMDLs do not specifically attempt to quantify allowable loads for these contaminants.

Problem Definition

The water bodies covered by this TMDL document were included on the State of Texas 1998, 1999, and 2000 §303(d) lists (see corresponding *State of Texas Clean Water Act Section 303(d) List and Schedule of Development of Total Maximum Daily Loads, SFR-58*) as a result of the issuance of Aquatic Life Orders by the Texas Department of Health (TDH), which prohibit the consumption of fish. Consumption bans were issued following determinations of unacceptable human health risk due to elevated concentrations of one or more legacy pollutants in fish tissue (Table 1). The impacted water bodies lie within Tarrant County, in the upper end of the Trinity River Basin (Figure 1).

Table 1. Tarrant County water bodies on the State of Texas 303(d) list due to concentrations of legacy pollutants in fish tissue that have resulted in the issuance of a fish consumption ban by the Texas Department of Health.

Segment Number	**Segment Name (Portion Covered by TDH Fish Consumption Ban)**	**Fish Tissue Contaminants on the 303(d) List**	**TDH Ban Issued**
0829	Clear Fork Trinity River Below Benbrook Lake (lower one mile of the segment from 7th Street to the confluence with the West Fork Trinity River in Segment 0806 in downtown Fort Worth)	Chlordane	01/1990
0806	West Fork Trinity River Below Lake Worth (lower 22 miles of the segment from the Clear Fork Trinity River confluence in downtown Fort Worth to the end of the segment at the confluence with Village Creek)	Chlordane	01/1990
0829A	Lake Como (entire lake)	Chlordane DDT Dieldrin PCBs	04/1995
0806A	Fosdic Lake (entire lake)	Chlordane DDE Dieldrin PCBs	04/1995
0806B	Echo Lake (entire lake)	PCBs	12/1995

All or part of four classified segments of the Trinity River in Tarrant and Dallas Counties do not support the fish consumption use. The impacted portions of these segments extend from the Clear Fork Trinity River at 7th Street in Fort Worth, downstream to the Trinity River at Interstate 20 in southeast Dallas County. TDH issued Aquatic Life Order 2 on 4 January 1990, declaring the entire length a "prohibited area for the taking of finfish" due to elevated levels of chlordane in fish tissue. Trinity River segments addressed in this document are:

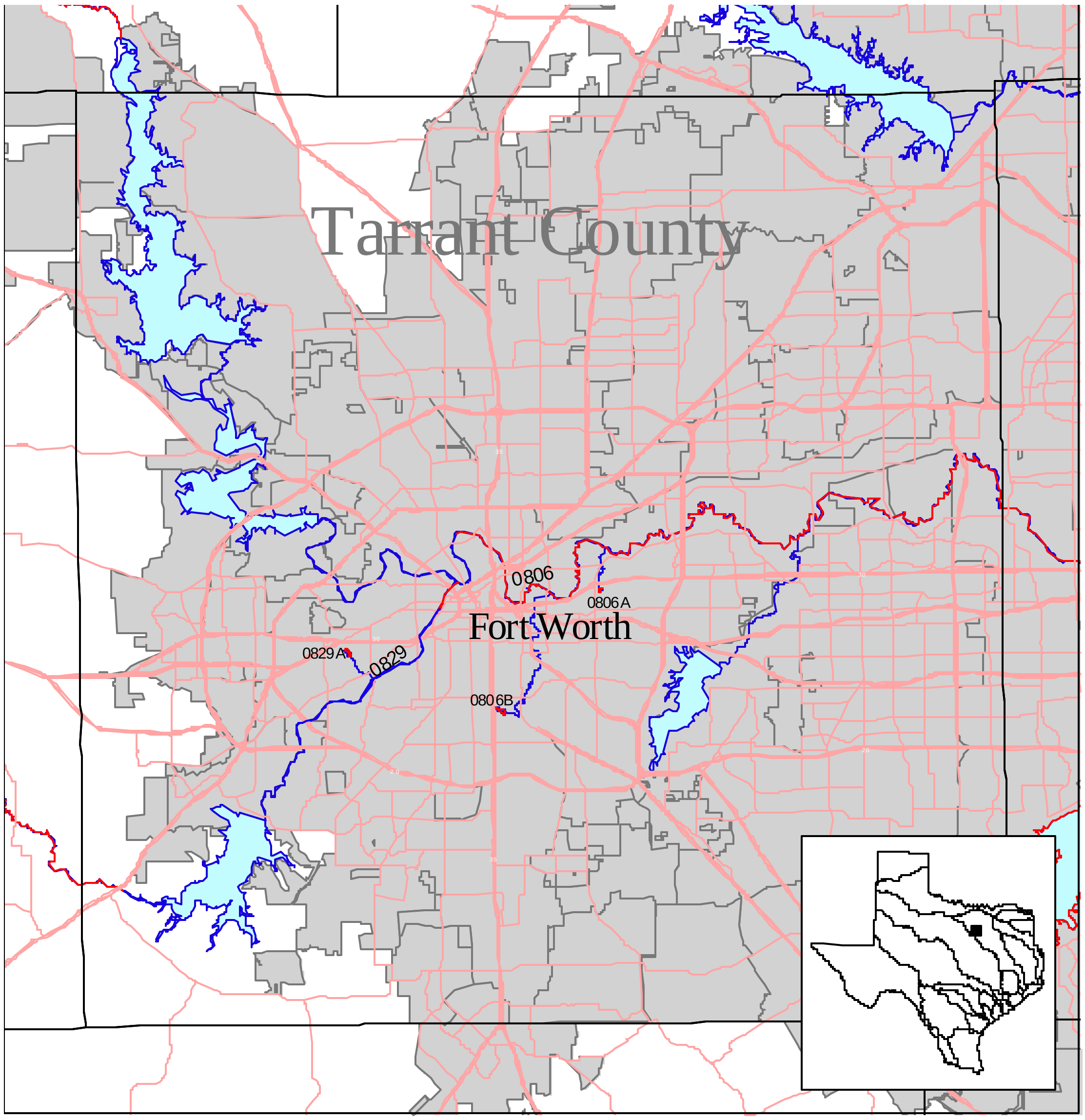

Figure 1. Study area, Tarrant County in upper Trinity River Basin.

- Segment 0829 (Clear Fork Trinity River Below Benbrook Lake) extends from the Benbrook Lake dam in southwest Tarrant County, downstream to the confluence with the West Fork Trinity River (Segment 0806) in downtown Fort Worth. The fish consumption use is not supported through the lower one mile, from the 7th Street bridge at the north end of Forest Park in the City of Fort Worth, downstream to the West Fork Trinity confluence.

- Segment 0806 (West Fork Trinity River Below Lake Worth) extends from the Lake Worth dam in west-central Tarrant County, downstream to a point immediately upstream of the confluence of Village Creek in east-central Tarrant County. The fish consumption use is not supported through the lower 22 miles, from the confluence with the Clear Fork

Trinity River (Segment 0829) in downtown Fort Worth, downstream to the end of the segment.

Segments 0829 and 0806 drain a 188,666-acre watershed downstream from the dams on Benbrook Lake and Lake Worth. The watershed is 62 percent urban and 34 percent agricultural/undeveloped land use. The City of Fort Worth accounts for most of the watershed area and 74 percent of the population (*1999 Annual Water Quality Management Plan, North Central Texas Council of Governments, Arlington, Texas*).

The fish consumption use is not supported in three unclassified urban lakes located in public parks within the City of Fort Worth. All three impound small drainage tributaries that collect storm water runoff from small urban watersheds (watershed information obtained from City of Fort Worth Department of Environmental Management):

- Lake Como (Segment 0829A) is a 10.1-acre impoundment of an unnamed tributary of the Clear Fork Trinity River (Segment 0829), located approximately five blocks south of Interstate 30, in Lake Como Park in west Fort Worth. Lake Como was impounded in 1889, and drains a 743-acre watershed that is 65 percent residential land use.

- Fosdic Lake (Segment 0806A) is a seven acre impoundment of an unnamed tributary of the West Fork Trinity River (Segment 0806), located approximately two blocks south of Interstate 30, in Oakland Lake Park in east Fort Worth. Fosdic Lake was impounded in 1927, and drains a 292-acre watershed that is 76 percent residential land use.

- Echo Lake (Segment 0806B) is a 16.8-acre impoundment of an unnamed tributary of Sycamore Creek, located approximately two blocks east of Interstate 35W, in Echo Lake Park in south-central Fort Worth. Echo Lake drains a 632-acre watershed that is 55 percent residential and 30 percent industrial land use.

TDH issued Aquatic Life Order No. 10 on 4 April 1995, declaring Lake Como and Fosdic Lake “prohibited areas for possession of all fish species” due to elevated levels of several legacy pollutants in fish tissue. Aquatic Life Order No. 11 was issued on 5 December 1995, declaring Echo Lake a “prohibited area for possession of all fish species” due to elevated levels of polychlorinated biphenyls in fish tissue.

The fish consumption use of a water body is not supported when the TDH issues an Aquatic Life Order prohibiting the consumption of aquatic life. The fish consumption bans on the water bodies addressed in this document are the result of contamination by one or more organochlorine insecticides, degradation products of organochlorine insecticides, and polychlorinated biphenyls (PCBs).

Organochlorine insecticides and PCBs were widely used in the U.S. prior to EPA restriction, and are common environmental contaminants (Moore and Ramamoorthy 1984; Schmitt *et al.* 1985, 1990; Smith *et al.* 1988; USGS 2000). These substances are a frequent cause of fish consumption advisories in the U.S. (EPA 1999b,c), and elevated concentrations of some of these contaminants are frequently found in game fish tissue (Kuehl *et al.* 1994). Fish consumption can be a primary route of human exposure to these contaminants (Schwartz *et al.* 1983; Humphrey 1987; Fiore *et al.* 1989), which can cause a variety of adverse health effects (Swain 1988; Longnecker *et al.* 1997). The Aquatic Life Orders for the Fort Worth water bodies were issued on the basis of an unacceptable carcinogenic risk of liver cancer and a noncarcinogenic risk of adverse liver effects due to fish tissue contaminant levels (see TDH 1995a,b,c).

Endpoint Identification

The ultimate goal of these TMDLs is the reduction of fish tissue contaminant concentrations to levels that constitute an acceptable risk to fish consumers, allowing TDH to remove the bans on fish consumption. The allowable load of contaminant is based on fish tissue concentrations.

EPA (1997a) provides guidance for assessing contaminant data for risk assessment. This guidance and TDH assumptions were used to develop target values for tissue contaminant levels that result in an acceptable risk level. EPA (1997a) presents equations for calculating the maximum allowable fish consumption rate given consumer body weight, contaminant concentration, an acceptable cancer risk level, and the contaminant risk values for carcinogenic and noncarcinogenic risk. A cancer potency value (q_1*) is the risk value for carcinogens. The oral reference dose (RfD) is the risk value used to protect against chronic exposure by noncarcinogens.

The consumption rate and consumer body weight were set at the TDH constants of 30 grams of fish per day (0.03 kg/d) for a 70-kg adult, and at 15 grams of fish per day (0.015 kg/d) for a 15- and 35-kg child, both over a 30-year time period. TDH uses an acceptable cancer risk level of 1×10^{-4}, adjusted to 2.33×10^{-4} to account for the use of the 30-year time period. Cancer potency and chronic RfD values (Table 2) were obtained from EPA (1997a) and the EPA IRIS database.

Equations in EPA (1997a) were solved to calculate the maximum allowable concentration of contaminant in tissue (1) at a given cancer risk level, and (2) based on the noncarcinogenic health effects of a contaminant:

(1) $$C_m = (ARL)(BW) / (C_{lim})(q_1^*)$$

(2) $$C_m = (RfD)(BW) / (C_{lim})$$

where:

C_m = maximum allowable concentration of contaminant in tissue (mg/kg)
ARL = acceptable cancer risk level = 2.33×10^{-4}
BW = consumer body weight (kg)
C_{lim} = allowable fish consumption rate (kg/d)

q_1* = cancer slope factor for given contaminant (see Table 2)
RfD = oral reference dose for given contaminant (see Table 2).

Substituting adult and child consumption rates and body weights used by TDH, the maximum tissue contaminant concentrations that can be consumed within an acceptable level of risk, were calculated (see Table 2).

Table 2. Maximum fish tissue concentrations (mg/kg) for individual contaminants that can be ingested by consumers of given body weights, within the acceptable cancer risk level (ARL) used by TDH, and without causing adverse noncarcinogenic health effects. Carcinogenic (q_1*) and noncarcinogenic (RfD) risk values were obtained from EPA 1997a and the EPA IRIS database.

	CARCINOGENIC RISK ARL = 2.33×10^{-4}			NONCARCINOGENIC RISK		
	MAXIMUM FISH TISSUE CONCENTRATION (mg/kg)					
CONTAMINANT		CONSUMER BODY WT.			CONSUMER BODY WT.	
	q_1*	15-kg	35-kg/70-kg	RfD	15-kg	35-kg/70-kg
Total Chlordane	0.35	0.67	1.55	5×10^{-4}	0.5	1.17
Total DDT[1]	0.34	0.68	1.6	5×10^{-4}	0.5	1.17
DDD	0.24	0.97	2.3	na	na	na
DDE	0.34	0.68	0.68	na	na	na
Dieldrin	16	0.015	0.034	5×10^{-5}	0.05	0.12
Total PCBs	2.0	0.12	0.27	2×10^{-5}	0.02	0.05

[1]Sum of 4,4'- and 2,4'- isomers of DDT, DDE, and DDD (EPA 1997a).
na = Separate RfDs not available for DDE and DDD.

The calculated contaminant concentrations are valid targets only for each contaminant individually. The chlordane and total PCB targets can be used for the Trinity River segments and Echo Lake, respectively, because TDH risk assessments identified each compound as the risk factor. In both cases, the target concentration that will achieve an acceptable noncarcinogenic risk is less than that needed for an acceptable carcinogenic risk. Both target concentrations for a 15-kg child are less than those of a 35-kg child and 70-kg adult (see Table

2). The noncarcinogenic values for a 15-kg child are therefore most protective, and these values become the endpoint targets for chlordane in the Trinity River segments and total PCBs in Echo Lake (Table 3).

The situation is different where multiple contaminants were determined to be contributing to overall risk, as is the case in Lake Como and Fosdic Lake. TDH assumes that risk is additive when more than one contaminant is present at sufficient levels. In such cases, the additive risk of all contaminants cannot exceed either the cancer risk level or a noncarcinogenic hazard index. When multiple contaminants are present, the concentration of one or more must be reduced so that the additive carcinogenic risk does not exceed 2.33×10^{-4}. The endpoint target for carcinogenic risk in this case is an additive risk that is no greater than the acceptable cancer risk level (Table 3).

The noncarcinogenic hazard index is the sum of the hazard ratios of each individual contaminant, and must be no greater than one for noncarcinogenic risk to be acceptable. The hazard ratio of a contaminant is the ratio of the actual noncarcinogenic exposure level to the oral reference dose (RfD). When multiple contaminants are present, the concentration of one or more must be reduced so that the additive hazard index does not exceed one. The endpoint target for noncarcinogenic risk is a hazard index that is no greater than one (Table 3).

Table 3. Most protective endpoint targets for fish tissue contamination in each §303(d) listed water body that will allow removal of the TDH fish consumption ban.

Segment	**Primary Endpoint Target**
Clear Fork Trinity River (0829)	≤ 1.17 mg/kg chlordane in fish tissue for adults ≤ 0.50 mg/kg chlordane in fish tissue for children
West Fork Trinity River (0806)	≤ 1.17 mg/kg chlordane in fish tissue for adults ≤ 0.50 mg/kg chlordane in fish tissue for children
Lake Como (0829A)	additive cancer risk $\leq 2.33 \times 10^{-4}$ cumulative noncarcinogenic hazard index ≤ 1
Fosdic Lake (0806A)	additive cancer risk $\leq 2.33 \times 10^{-4}$ cumulative noncarcinogenic hazard index ≤ 1
Echo Lake (0806B)	≤ 0.05 mg/kg total PCBs in fish tissue for adults ≤ 0.02 mg/kg total PCBs in fish tissue for children
All Water bodies	Removal of fish consumption bans

The calculated target values are valid only under the assumed conditions. TDH has the authority and jurisdiction for the decision to issue or remove fish consumption bans. Subsequent risk assessments by TDH may result in no change to a ban, removal of the ban, or a shift to an advisory for certain groups at greater risk. The ultimate endpoint goal for the affected water bodies is the protection of all groups and complete removal of the fish consumption bans.

Source Analysis

Production and use of legacy pollutants has been banned or severely restricted by the EPA. Because of their past heavy and widespread use, strong affinities for sorption to sediment organic matter and tissue, and slow rates of decomposition, these substances and/or their degradation products frequently remain at elevated levels in the environment for many years after widespread use has ended (Moore and Ramamoorthy 1984; Smith *et al.* 1988; Jones and de Voogt 1999; USGS 2000).

- Chlordane was introduced in 1948, and was used extensively as a broad spectrum insecticide to control soil insects on agricultural crops, as a home lawn and garden insecticide, as a fumigating agent, and for termite control (EPA 1980b; Dick 1982; Dearth and Hites 1991). EPA suspended use of chlordane on food crops in 1978, and phased out other above-ground uses over the following five years (EPA 1997b; Mattina *et al.* 1999). All uses except underground application for termite control were banned in 1983 (Mattina *et al.* 1999). Manufacture and domestic sales were halted in 1987, and use of existing stores was allowed until April 1988 when sale and use were terminated (Dearth and Hites 1991; EPA 1997b; Mattina *et al.* 1999).

- DDT was initially used in World War II for control of disease-carrying insects, and was used extensively as a broad spectrum insecticide for the control of almost all agricultural and disease-carrying insects (EPA 1980c; NPTN 1999). It was used extensively in the 1950s and 1960s for mosquito control in urban areas. DDD is a metabolite of DDT, and was itself manufactured as a pesticide for several years. Most uses of DDT, and all uses of DDD, were banned by EPA in December 1972 (EPA 1980c). DDE is the major degradation product of DDT and DDD, and is among the most widely occurring pesticide residues (Schmitt *et al.* 1990; Kuehl *et al.* 1994).

- Dieldrin was used as a pesticide, and is a degradation product of the pesticide aldrin. Although aldrin was used in greater quantity, it is rapidly converted to the more persistent dieldrin in the environment (EPA 1980a; Dick 1982). Both pesticides were used primarily for the control of corn rootworm and cutworm, with some use in the citrus industry and for mosquito larvae and termite control (EPA 1980a; Dick 1982). All food crop uses of both compounds were canceled in May 1975, and only subsurface injection for termite control was allowed after that time. All remaining uses were canceled in 1987.

- Polychlorinated biphenyls (PCBs) are a group of synthetic organic chemicals containing 209 possible individual compounds, which vary in chemical and physical properties, toxicity, environmental persistence, and degree of bioaccumulation (EPA 1980d, 1999b). PCBs were manufactured as mixtures of different congeners, and generally sold under the trade name Aroclor. PCBs were most widely used as coolants and lubricants in transformers, capacitors, and other electrical equipment. In 1976 the Toxic Substances Control Act (TSC A) banned, with limited exceptions, the manufacture, processing, distribution in commerce, and use of PCBs (EPA 1994). TSCA also required the EPA to promulgate regulations for proper use, cleanup, and disposal. TSCA and subsequent EPA rules did not require PCB-containing materials to be removed from service, and many are still in use (EPA 1999b). A substantial portion of the PCBs manufactured before 1977 remain in service, although these are being phased out as equipment is replaced or decontaminated.

Organochlorine insecticides have entered aquatic systems as a result of direct application to a water body, drift from aerial spraying, urban and agricultural runoff, spills, industrial and municipal wastewater discharges, and erosion of contaminated soils (Dick 1982; Smith *et al.* 1988; Van Metre *et al.* 1998). PCBs can enter the environment via spills and leaks from sites where they are used, improper disposal methods, and leaching from landfills (Tanabe 1988).

Studies examining the relationship between land use and contamination by legacy pollutants suggest that problems can originate from both urban and agricultural land uses, although determination of a specific source can be very difficult (Tate and Heiny 1996; Munn and Gruber 1997; Webster *et al.* 1998). Numerous studies have associated organochlorine pesticide and PCB residues in tissue and sediment with urban land uses (Stamer *et al.* 1985; Arruda *et al*. 1987; Smith *et al.* 1988; Pereira *et al.* 1996; Mattina *et al.* 1999; Black *et al.* 2000).

Legacy pollutant contamination in the Trinity River and urban lakes appears to have originated from urban areas, as the watersheds of these water bodies have been highly urbanized for many years. Erosion as a result of extensive urban development over the past 10 to 15 years may have contributed contaminants attached to source soils. Ulery and Brown (1995) evaluated a number of available data sets from the Trinity River Basin, and found a significant correlation between sediment chlordane presence and urban land use. Irwin (1988) found concentrations of total chlordane in mosquitofish to be strongly associated with residential runoff from the area. Kleinsasser and Linam (1989) found elevated chlordane levels in fish collected within the area covered by the TDH consumption ban on the Trinity River, and suggested urban or suburban runoff as the source.

Recent household hazardous wastes received at the City of Fort Worth Environmental Collection Center have included chlordane, suggesting some recent or possible continued use in the area . Urban residents may have continued using existing stocks of chlordane since it was in use longer than some of the other legacy insecticides. Van Metre and Callender (1997) found the chlordane peak in sediment cores from White Rock Lake in Dallas to have occurred around 1990, reflecting its relatively recent urban use.

Linkage Between Sources and Receiving Waters

The time required for the reduction of legacy pollutant tissue concentrations to endpoint levels is a function of their persistence and fate in the environment. Organochlorine insecticides and PCBs are extremely hydrophobic, and their affinity for sorption to soil and sediment, along with their tendency to partition into the lipid tissues of aquatic organisms, determine their transport, fate, and distribution (Smith *et al.* 1988).

Numerous studies have documented the long-term persistence of organochlorine pesticides and their degradation products in soil. Pesticide residue concentrations in soils can span several orders of magnitude, and are a reflection of application history and loss rates (Lichtenstein *et al.* 1971; Harner *et al.* 1999). Heavily used pesticides will be present in higher concentrations years later. Degradation rates of organochlorine residues are highly variable, and soil half-lives of as much as 20 to 35 years have been reported (Nash and Woolson 1967; Dimond and Owen 1996; Mattina *et al.* 1999).

The primary method of transport of legacy pollutants into aquatic systems is by erosion of soil and attached contaminants (Munn and Gruber 1997). Aquatic sediments act as a reservoir for hydrophobic pesticides and PCBs (Moore and Ramamoorthy 1984). Contaminants may be present in sediment at concentrations that are orders of magnitude higher than in the water column, where they are typically very low or undetectable (see Smith *et al.* 1988). These contaminants degrade slowly, and may be present for long periods of time (Oliver *et al.* 1989; EPA 1999b). Van Metre *et al*. (1998) analyzed sediment core samples from 11 reservoirs, including White Rock Lake in Dallas, and determined mean sediment half-lives of 7.7 to 17 years for chlordane, 13 $\pm$ 5.8 years for total DDT, and 9.5 $\pm$ 2.2 years for PCBs. Field and laboratory studies of contaminated sediments have found that the greatest amount of PCB dechlorination occurs during a relatively short and rapid initial phase after contaminant input, but then slows or effectively ceases (Rhee *et al.* 1993; Sokol *et al.* 1998).

Sediments may act as long-term sources of contamination through desorption of contaminants, and as a result of the resuspension of sediment particles by disturbances (Oliver *et al.* 1989; Baker *et al.* 1991; Zaranko *et al.* 1997; Maher *et al.* 1999). Sediment-associated contaminants can be a long-term source of chronic toxicity to organisms that live or feed in contact with the sediments, and provide a source for the introduction of contaminants into the food web (Reynoldson 1987; Farrington 1991; Larsson 1986).

Organochlorine insecticides and PCBs are highly lipophilic and rapidly accumulate in the tissue of aquatic organisms. Contaminant concentrations are found in fish tissue at levels considerably higher than that of the water column and sediments (Smith *et al.* 1988; Rinella *et al.* 1993; EPA 1997a, 1999b). Fish tissue contaminant concentrations can vary within the same water body (Stow *et al*. 1995; Lamon and Stow 1999), and among different fish species, size classes within a fish species, and various tissues within a fish (Swackhamer and Hites 1988; EPA 1997a).

A large number of factors have been found to influence contaminant uptake, accumulation, and elimination in fish and other aquatic organisms. Characteristics of fish species and their environments are very important to uptake and elimination processes (Swackhamer and Hites 1988). Fish characteristics include lipid content, age, length, weight, diet and feeding habits, reproductive status, contaminant transfer from females to young, growth dilution, metabolism, and other species-specific physiological factors. Environmental factors include contaminant levels in food items, trophic position and length of the food chain, habitat use and movement, seasonal variation in contaminant availability, water column contaminant concentration, and sediment contaminant concentration and bioavailability. The relative importance of these factors is much debated, and research has found the effects of many of them to be interrelated (Smith *et al.* 1988; Farrington 1991; Pritchard 1993; Jones and de Voogt 1999; Sijm *et al.* 2000).

Characteristics of the contaminants also affect their tissue concentrations. These factors include differences in isomer and residue bioavailability, equilibrium time, and susceptibility to uptake, biotransformation, and elimination. Schmitt *et al.* (1985) found that changes in tissue concentrations over time vary with differences in chlordane and PCB isomers. Significant differences have been found in the accumulation rates of different PCB congeners, and in the degree of accumulation within different fish body tissues (Gruger *et al.* 1975; van der Oost *et al.* 1988; Zhou and Wong 2000).

The time necessary for a contaminant to reach equilibrium in tissue is variable, hard to determine, and generally very long. Stable organic compounds with low aqueous solubilities, such as many legacy pollutants, generally exhibit the longest equilibrium times. Time to equilibrium is also a function of fish size, with larger fish accumulating contaminants at a slower rate (Smith *et al.* 1988).

Once equilibrium is reached, the time necessary for a contaminant to be eliminated from tissue is also long, often on the order of years, and variable, generally increasing with the hydrophobicity and lipophilicity of the compound (Larsson 1986). Contaminant elimination may occur through respiration, metabolism, egestion, growth dilution, and transfer to eggs or young (Sharpe and Mackay 2000). Elimination rates can also be affected by the form of the contaminant (Niimi and Oliver 1983; Sijm *et al.* 1992; de Boer *et al.* 1994), especially in the case of PCBs. Schnoor (1981) calculated a dieldrin decrease of 15 percent per year in reservoir fish tissue. Half-lives for DDT, DDE, and PCBs in lake trout have been estimated at 9 to 10 years (see Borgmann and Whittle 1992; Van Metre *et al.* 1998). Delorme *et al.* (1999) suggest that hydrophobic contaminants may not remobilize from fish tissue unless severe nutritional stress occurs.

In addition to generally excluding the effects of contaminated sediment and food, most studies of contaminant uptake and elimination are relatively short-term laboratory experiments (de Boer *et al.* 1994; Sijm *et al.* 2000). Long-term field studies have generally found that elimination rates are considerably longer than in those measured in the shorter laboratory studies (de Boer *et al.* 1994; Delorme *et al.* 1999). The interval between bioconcentration and elimination may be too short in laboratory studies to allow equilibrium within all tissues, allowing elimination to proceed much faster than in a field situation. Published uptake and elimination rates derived from laboratory studies may not reflect field conditions, limiting their use for the prediction of contaminant behavior (Swackhamer and Hites 1988; de Boer *et al.* 1994).

Available sediment and fish tissue data from the water bodies discussed by this document indicates that legacy pollutant concentrations are decreasing as a result of natural attenuation processes. Davis and Bastian (1990) evaluated mean chlordane levels in Trinity River sediment samples collected between 1974 and 1989, and found some evidence that levels were decreasing. Subsequent data collected in 1992-1993 by Moring (1997) appear to support this trend. The data suggest that chlordane levels are decreasing in the sediments of the affected area, thus removing a significant source of potentially bioavailable contamination.

Samples of recent sediment deposits were collected from three locations within each of the three urban lakes in May 1999 by the Trinity River Authority and the City of Fort Worth. Analyses were conducted for a number of pesticides and metabolites (including DDT, DDD, DDE, and dieldrin), and for seven PCB congeners. Concentrations were less than the detection limit (0.0007 mg/kg for pesticides and 0.02 mg/kg for PCBs) at all locations in all three lakes. The data suggest that there have been no recent contaminant inputs to the lakes, and that any remaining contaminated sediments are being buried, thus reducing their bioavailability.

Fish tissue data are available for two or three sample dates in each of the impacted water bodies (Table 4). Samples frequently consisted of a relatively small number of fish, and

Table 4. Fish tissue contaminant means and ranges time in Segments 0829 and 0806 in Fort Worth, and in the three urban lakes. N = number of fish. na = not applicable. nd = not detected.

SAMPLE MONTH/YEAR===>			**01-04/1988**	**10/1990**	**07/1996**	**11/1998**
Water Body	**Pollutant**	**Statistic**	**Fish Tissue (Fillets) Concentration (mg/kg)**			
0829	Chlordane	N Mean Range	1 0.780 na	3 0.03 0.02-0.05	4 0.14 0.03-0.36	---
0806	Chlordane	N Mean Range	---	5 0.13 nd-0.28	3 0.03 nd-0.06	10 0.13 nd-0.37
SAMPLE MONTH/YEAR===>			**08/1994**	**04/1995**	**09/1997**	**05/1999**
Como		N	4	---	5	7
	Chlordane	Mean Range	1.78 1.00-2.90	---	0.665 0.371-0.849	0.02 0.01-0.04
	Dieldrin	Mean Range	0.074 0.034-0.160	---	0.05 0.017-0.129	0.03 0.01-0.06
	DDE	Mean Range	0.107 0.085-0.130	---	0.002 <0.00005-0.011	0.06 0.01-0.1

	PCBs	Mean Range	0.220 0.150-0.340	---	---	0.23 <0.20-0.30
Fosdic		N	1	---	6	18
	Chlordane	Mean Range	0.350 na	---	0.165 0.032-0.437	0.004 <0.0004-0.01
	Dieldrin	Mean Range	0.013 na	---	0.008 <0.0002-0.037	0.002 <0.001-0.006
	DDE	Mean Range	0.054 na	---	0.008 <0.00005-0.031	0.005 <0.0004-0.01
	PCBs	Mean Range	0.190 na	---	---	<0.04 <0.04
Echo		N	---	8	---	12
	PCBs	Mean Range	---	0.252 0.05-1.20	---	0.053 <0.04-0.21

SOURCES OF DATA: Kleinsasser and Linam (1989)
Texas Department of Health (*Fish Tissue Sampling Data 1970-1997* and unpublished 11/1998 data)
Texas Parks & Wildlife Department (unpublished 12/1990 and 07/1996data)
City of Fort Worth (unpublished urban lakes data)
Trinity River Authority and City of Fort Worth (unpublished urban lakes data)

most often included largemouth bass and/or one or more of the bottom-feeding common carp, smallmouth buffalo, and blue catfish. Mean contaminant levels for each sampling event and water body were calculated using available fish fillet data, to investigate changes in tissue concentrations.

There is some evidence of a decline in chlordane fish tissue concentrations in Segment 0829, and fish tissue concentrations in Segment 806 are relatively low; however, the amount of data for these segments is very limited. Fish sample sizes from the Fort Worth urban lakes are generally larger than those from the Trinity River, particularly for samples collected in May 1999, providing more confidence in the apparent trends. Decreases in tissue concentrations through time are evident for all except PCBs in Lake Como.

Margin of Safety

The margin of safety is required in a TMDL in order to account for any uncertainty about the pollutant load and its association with water quality. The margin of safety may be an explicit component that leaves a portion of the assimilative capacity of a water body unallocated, or an implicit component established through the use of conservative analytical assumptions (EPA 1999a).

These TMDLs use an implicit margin of safety. EPA (1997a) guidance on the assessment of contaminant data for use in fish advisories contains an extensive discussion of the assumptions and uncertainties present in the calculation of fish consumption limits. Conservative assumptions and calculations are used throughout the guidance to provide a margin of safety for the various uncertainties. Strict criteria is used concerning the types of studies and the data required to support assumptions and calculations. Numeric adjustments are made for the extrapolation of study results from animals or humans to the general population, and to provide a conservative upper bound on cancer risk values and a conservative RfD for noncarcinogens. Adjustments are designed to provide a safe margin between observed toxicity and potential toxicity in a sensitive human.

EPA assumes no safe threshold for exposure to carcinogens. Any exposure is assumed to pose some cancer risk. Noncarcinogenic effects occur with chronic exposure over a significant period of time. The oral reference dose (RfD) is defined in EPA (1997a) as "an estimate (with uncertainty perhaps spanning an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime." Calculated RfDs reflect the assumption that, for noncarcinogens, a threshold exists below which exposure does not cause adverse health effects. RfD calculations use modifying and uncertainty factors to account for variables such as the variability of responses in human populations, differences in responses between animal study species and humans, and gaps in available data. The RfD is calculated so there is little probability of an adverse health effect due to chronic exposure to concentrations below the RfD (EPA 1997a).

The flexibility provided by having numerous combinations of contaminants and concentration reductions that can produce acceptable carcinogenic and noncarcinogenic risk when multiple contaminants are present provides an inherent margin of safety that the goal of acceptable risk will be met. Use of the most protective target concentration for single contaminants provides additional assurance that protection from both carcinogenic and noncarcinogenic effects will be achieved. Because the goal of this TMDL is removal of fish consumption bans through reduction of the consumption risk, the margin of safety inherent in the EPA guidance, combined with the conservative use of endpoint targets, will provide an adequate margin of safety for the protection of human health. The decline of tissue contaminant concentrations to within an acceptable level of risk will allow TDH to remove the fish consumption bans, which effectively restores the fish consumption use to these water bodies.

Pollutant Load Allocation

Restrictions on the use of legacy pollutants generally have resulted in a slow but steady decline in environmental residues (Smith *et al.* 1988). Contaminant levels in lake sediment cores have shown good agreement with production and usage histories of the parent compounds, with peak concentrations appearing at the times of peak use (Ricci *et al.* 1983; Oliver *et al.* 1989; Van Meter and Callender 1997; Van Metre *et al.* 1998). Higher concentrations generally appeared deeper in the cores, indicating that input and accumulation were decreasing with time. Although residues continue to persist in deeper parts of the cores, burial by more recently deposited sediments may result in effective removal of the contaminants from bioavailability to aquatic life (Ricci *et al.* 1983).

Decreases in fish (and human) tissue concentrations of organochlorine insecticides and PCBs have been observed where no major additional inputs are occurring (see Moore and Ramamoorthy 1984; Brown *et al.* 1985; Hovinga *et al.* 1992; Bremle and Larsson 1998). Reviews of tissue data collected from a variety of water bodies in northern Europe between 1967 and 1995 have found a significant decrease in organochlorine concentrations over time (Skåre *et al.* 1985; Bignert *et al.* 1998). Fish tissue concentrations of total DDT, chlordane, and dieldrin have declined across the U.S. since use of these substances was discontinued (Schmitt *et al.* 1990; USGS 2000). The DDE component of total DDT has increased as a result of continued degradation. Declining tissue DDT and PCB concentrations have been reported in various locations and fish species in the Great Lakes (Scheider *et al.* 1998). Less consistent trends in tissue PCB levels may be a reflection of the congener-specific nature of PCB metabolism and degradation. In addition, strong oscillations in PCB levels influenced by food web interactions can be superimposed on a gradual decline (see Borgmann and Whittle 1992).

Continuing decreases in environmental legacy pollutant levels are expected, although the necessary time frame is subject to debate. Within the context of these TMDLs, legacy pollutants are considered background sources that reflect the site-specific application history and loss rates of the subject area. All continuing sources of pollutant loadings occur from nonpoint source runoff , leaching, or erosion of the various sinks that may exist within the watersheds. No authorized point source discharges of these pollutants are allowed by law. Therefore, any contribution from point source discharges would be the result of illegal disposal of these contaminants by customers of the treatment systems.

Available evidence suggests that legacy pollutants are generally declining in both the surface sediments and the fish tissue of the affected Fort Worth water bodies. Continuing natural attenuation of these pollutants is expected via degradation and metabolism of the contaminants, burial of contaminated sediment through natural sedimentation in the urban lakes, and scouring and redistribution of sediments in the river.

Natural attenuation is generally a preferred option for the elimination of legacy pollutants. More drastic alternatives, such as sediment removal by dredging, can result in considerable habitat disturbance and destruction, and sediments resuspended during dredging further expose aquatic life to contaminants and the potential for additional uptake, cause abrasive damage to gills and sensory organs of fish and invertebrates, and interfere with fish prey selection (O'Brien 1990; Newcombe and MacDonald 1991; Waters 1995). More drastic alternatives such as dredging or eradication of contaminated fish communities and restocking (O'Meara *et al.* 2000) are generally better justified at heavily contaminated sites impacted by point source discharges and major spills.

References

Arruda, J.A., M.S. Cringan, D. Gilliland, S.G. Haslouer, J.E. Fry, R. Broxterman, and K.L. Brunson. 1987. Correspondence between urban areas and the concentrations of chlordane in fish from the Kansas River. Bulletin of Environmental Contamination and Toxicology 39:563-570.

Baker, J.E., S.J. Eisenreich, and B.J. Eadie. 1991. Sediment trap fluxes and benthic recycling of organic carbon, polycyclic aromatic hydrocarbons, and polychlorobiphenyl congeners in Lake Superior. Environmental Science and Technology 25:500-509.

Bignert, A., M. Olsson, W. Persson, S. Jensen, S. Zakrisson, K. Litzén, U. Eriksson, L. Häagberg, and T. Alsberg. 1998. Temporal trends of organochlorines in northern Europe, 1967-1995. Relation to global fractionation, leakage from sediments and international measures. Environmental Pollution 99:177-198.

Black, R.W., A.L. Haggland, and F.D. Voss. 2000. Predicting the probability of detecting organochlorine pesticides and polychlorinated biphenyls in stream systems on the basis of land use in the Pacific northwest, USA. Environmental Toxicology and Chemistry 19:1044-1054.

Borgmann, U., and D.M. Whittle. 1992. Bioenergetics and PCB, DDE, and mercury dynamics in Lake Ontario lake trout (*Salvelinus namaycush*): a model based on surveillance data. Canadian Journal of Fisheries and Aquatic Sciences 49:1086-1096.

Bremle, G., and P. Larsson. 1998. PCB concentration in fish in a river system after remediation of contaminated sediment. Environmental Science and Technology 32:3491-3495.

Brown, M.P., M.B. Werner, R.J. Sloan, and K.W. Simpson. 1985. Polychlorinated biphenyls in the Hudson River. Environmental Science and Technology 19:656-661.

Davis, J.R., and M.V. Bastian. 1990. Analysis of Fish Kills and Associated Water Quality Conditions in the Trinity River, Texas. III. Final Toxicological Considerations. Report No. LP 90-03, Texas Water Commission, Austin, Texas.

Dearth, M.A., and R.A. Hites. 1991. Complete analysis of technical chlordane using negative ionization mass spectrometry. Environmental Science and Technology 25:245-254.

de Boer, J., F. van der Valk, M.A.T. Kerkhoff, P. Hagel, and U.A.Th. Brinkman. 1994. 8-year study on the elimination of PCBs and other organochlorine compounds from eel (*Anguilla anguilla*) under natural conditions. Environmental Science and Technology 28:2242-2248.

Delorme, P.D., W.L. Lockhart, K.H. Mills, and D.C.G. Muir. 1999. Long-term effects of toxaphene and depuration in lake trout and white sucker in a natural ecosystem. Environmental Toxicology and Chemistry 18:1992-2000.

Dick, M. 1982. Pesticide and PCB Concentrations in Texas - Water, Sediment, and Fish Tissue. Report 264, Texas Department of Water Resources, Austin, Texas.

Dimond, J.B., and R.B. Owen. 1996. Long-term residues of DDT compounds in forest soils in Maine. Environmental Pollution 92:227-230.

EPA (U.S. Environmental Protection Agency). 1980a. Ambient Water Quality Criteria for Aldrin/Dieldrin. EPA 440/5-80-019, Office of Water, U.S. EPA, Washington, D.C.

EPA (U.S. Environmental Protection Agency). 1980b. Ambient Water Quality Criteria for Chlordane. EPA 440/5-80-027, Office of Water, U.S. EPA, Washington, D.C.

EPA (U.S. Environmental Protection Agency). 1980c. Ambient Water Quality Criteria for DDT. EPA 440/5-80-038, Office of Water, U.S. EPA, Washington, D.C.

EPA (U.S. Environmental Protection Agency). 1980d. Ambient Water Quality Criteria for Polychlorinated Biphenyls. EPA 440/5-80-068, Office of Water, U.S. EPA, Washington, D.C.

EPA (U.S. Environmental Protection Agency). 1994. PCB Q&A Manual. EPA TSCA Assistance Document, Operations Branch, Chemical Management Division, Office of Pollution Prevention and Toxics, U.S. EPA, Washington, D.C.

EPA (U.S. Environmental Protection Agency). 1997a. Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories. Volume 2: Risk Assessment and Fish Consumption Limits, Second edition. EPA 823-B-97-009, Office of Water, U.S. EPA, Washington, D.C.

EPA (U.S. Environmental Protection Agency). 1997b. Toxicological Review of Chlordane (Technical): In Support of Summary Information on the Integrated Risk Management System (IRIS), U.S. EPA, Washington, D.C.

EPA (U.S. Environmental Protection Agency). 1999a. Draft Guidance for Water Quality-based Decisions: The TMDL Process (Second Edition). EPA 841-D-99-001, Office of Water, U.S. EPA, Washington, D.C.

EPA (U.S. Environmental Protection Agency). 1999b. Polychlorinated Biphenyls (PCBs) Update: Impact on Fish Advisories. EPA-823-F-99-019, Fact Sheet, Office of Water, U.S. EPA, Washington, D.C.

EPA (U.S. Environmental Protection Agency). 1999c. Update: National Listing of Fish and Wildlife Advisories. EPA-823-F-99-005, Fact Sheet, Office of Water, U.S. EPA, Washington, D.C.

Farrington, J.W. 1991. Biogeochemical processes governing exposure and uptake of organic pollutant compounds in aquatic organisms. Environmental Health Perspectives 90:75-84.

Fiore, B.J., H.A. Anderson, L.P. Hanrahan, L.J. Olson, and W.C. Sonzogni. 1989. Sport fish consumption and body burden levels of chlorinated hydrocarbons: A study of Wisconsin anglers. Archives of Environmental Health 44:82-88.

Gruger, E.H., Jr., N.L. Karrick, A.I. Davidson, and T. Hruby. 1975. Accumulation of 3,4,3',4'-tetrachlorobiphenyl and 2,4,5,2',4',5'- and 2,4,6,2',4',6'-hexachlorobiphenyl in juvenile coho salmon. Environmental Science and Technology 9:121-127.

Harner, T., J.L. Wideman, L.M.M. Bidleman, and W.J. Parkhurst. 1999. Residues of organochlorine pesticides in Alabama soils. Environmental Pollution 106:323-332.

Hovinga, M.E., M. Sowers, and H.E.B. Humphrey. 1992. Historical changes in serum PCB and DDT levels in an environmentally-exposed cohort. Archives of Environmental Contamination and Toxicology 22:362-366.

Humphrey, H.E.B. 1987. The human population - an ultimate receptor for aquatic contaminants. Hydrobiologia 149:75-80.

Irwin, R.J. 1988. Impacts of Toxic Chemicals on Trinity River Fish and Wildlife. U.S. Fish & Wildlife Service Ecological Services Contaminants Report, Fort Worth, Texas.

Jones, K.C., and P. de Voogt. 1999. Persistent organic pollutants (POPs): state of the science. Environmental Pollution 100:209-221.

Kleinsasser, R., and G. Linam. 1989. Water quality and fish assemblages in the Trinity River, Texas, between Fort Worth and Livingston. Appendix B in J.R. Davis, 1989. Use Attainability Analysis of Trinity River Segment 0805. Texas Water Commission, Austin, Texas.

Kuehl, D.W., B. Butterworth, and P.J. Marquis. 1994. A national study of chemical residues in fish. III: Study results. Chemosphere 29:523-535.

Lamon, E.C., III, and C.A. Stow. 1999. Sources of variability in microcontaminant data for Lake Michigan salmonids: statistical models and implications for trend detection. Canadian Journal of Fisheries and Aquatic Sciences 56(Supplement):71-85.

Larsson, P. 1986. Zooplankton and fish accumulate chlorinated hydrocarbons from contaminated sediments. Canadian Journal of Fisheries and Aquatic Sciences 43:1463-1466.

Lichtenstein, E.P., T.W. Fuhremann, and K.R. Schulz. 1971. Persistence and vertical distribution of DDT, lindane, and aldrin residues, 10 and 15 years after a single soil application. Journal of Agricultural and Food Chemistry 19:718-721.

Longnecker, M.P., W.J. Rogan, and G. Lucier. 1997. The human health effects of DDT (dichlorodiphenyltrichloroethane) and PCBs (polychlorinated biphenyls) and an overview of organochlorines in public health. Annual Review of Public Health 18:211-244.

Maher, W., G.E. Batley, and I. Lawrence. 1999. Assessing the health of sediment ecosystems: use of chemical measurements. Freshwater Biology 41:361-372.

Mattina, M.J.I., W. Iannucci-Berger, L. Dykas, and J. Pardus. 1999. Impact of long-term weathering, mobility, and land use on chlordane residues in soil. Environmental Science and Technology 33:2425-2431.

Moore, J.W., and S. Ramamoorthy. 1984. Organic Chemicals in Natural Waters - Applied Monitoring and Impact Assessment. Springer-Verlag, New York, 289p.

Moring, J.B. 1997. Occurrence and Distribution of Organochlorine Compounds in Biological Tissue and Bed Sediment from Streams in the Trinity River Basin, Texas, 1992-93. U.S. Geological Survey Water-Resources Investigations Report 97-4057.

Munn, M.D., and S.J. Gruber. 1997. The relationship between land use and organo chlorine compounds in streambed sediment and fish in the Central Columbia Plateau, Washington and Idaho, USA. Environmental Toxicology and Chemistry 16:1877-1887.

Nash, R.G., and E.A. Woolson. 1967. Persistence of chlorinated hydrocarbon insecticides in soils. Science 157:924-927.

NPTN (National Pesticide Telecommunication Network). 1999. DDT. NPTN Technical Fact Sheet, National Pesticide Telecommunication Network, Oregon State University, Corvallis, Oregon. Online publication available at http://ace.orst.edu/info/nptn/

Niimi, A.J., and B.G. Oliver. 1983. Biological half-lives of polychlorinated biphenyl (PCB) congeners in whole fish and muscle of rainbow trout (*Salmo gairdneri*). Canadian Journal of Fisheries and Aquatic Sciences 40:1388-1394.

O'Brien, W.J. 1990. Perspectives on fish in reservoir limnology. Pages 209-225 in K.W. Thornton, B.L. Kimmel, and F.E. Payne (editors). Reservoir Limnology: Ecological Perspectives. John Wiley and Sons, Inc., New York

Oliver, B.G., M.N. Charlton, and R.W. Durham. 1989. Distribution, redistribution, and geochronology of polychlorinated biphenyl congeners and other chlorinated hydrocarbons in Lake Ontario sediments. Environmental Science and Technology 23:200-208.

O'Meara, J., J. Murray, and J. Ridgway. 2000. $11.8 million project removes PCBs, restores life to Newburgh Lake. Water Environment Federation Watershed and Wet Weather Technical Bulletin 5(3):12-15.

Pereira, W.E., J.L. Domagalski, F.D. Hostettler, L.R. Brown, and J.B. Rapp. 1996. Occurrence and accumulation of pesticides and organic contaminants in river sediment, water and clam tissues from the San Joaquin River and tributaries, California. Environmental Toxicology and Chemistry 15:172-180.

Pritchard, J.B. 1993. Aquatic toxicology: Past, present, and prospects. Environmental Health Perspectives 100:249-257.

Reynoldson, T.B. 1987. Interactions between sediment contaminants and benthic organisms. Hydrobiologia 149:53-66.

Rhee, G.-Y., R.C. Sokol, B. Bush, and C.M. Bethoney. 1993. Long-term study of the anaerobic dechlorination of Aroclor 1254 with and without biphenyl enrichment. Environmental Science and Technology 27:714-719.

Ricci, E.D., W.A. Hubert, and J.J. Richard. 1983. Organochlorine residues in sediment cores of a midwestern reservoir. Journal of Environmental Quality 12:418-421.

Rinella, J.F., S.W. McKenzie, J.K. Crawford, W.T. Foreman, G.J. Fuhrer, and J.L. Morace. 1993. Surface-Water-Quality Assessment of the Yakima River Basin, Washington. Distribution of Pesticides and Other Organic Compounds in Water, Sediment, and Aquatic Biota, 1987-91. U.S. Geological Survey Water-Supply Paper 2354-B.

Scheider, W.A., C. Cox, A. Hayton, G. Hitchin, and A. Vaillancourt. 1998. Current status and temporal trends in concentrations of persistent toxic substances in sport fish and juvenile forage fish in the Canadian waters of the Great Lakes. Environmental Monitoring and Assessment 53:57-76.

Schmitt, C.J., J.L. Zajicek, and M.A. Ribick. 1985. National Pesticide Monitoring Program: Residues of organochlorine chemicals in freshwater fish, 1980-81. Archives of Environmental Contamination and Toxicology 14:225-260.

Schmitt, C.J., J.L. Zajicek, and P.H. Peterman. 1990. National Contaminant Biomonitoring Program: Residues of organochlorine chemicals in U.S. freshwater fish, 1976-1984. Archives of Environmental Contamination and Toxicology 19:748-781.

Schnoor, J.L. 1981. Fate and transport of dieldrin in Coralville Reservoir: Residues in fish and water following a pesticide ban. Science 211:840-842.

Schwartz, P.M., S.W. Jacobson, G. Fein, J.L. Jacobson, and H.A. Price. 1983. Lake Michigan fish consumption as a source of polychlorinated biphenyls in human cord serum, maternal serum, and milk. American Journal of Public Health 73:293-296.

Sharpe, S., and D. Mackay. 2000. A framework for evaluating bioaccumulation in food webs. Environmental Science and Technology 34:2373-2379.

Sijm, D., R. Kraaij, and A. Belfroid. 2000. Bioavailability in soil or sediment: Exposure of different organisms and approaches to study it. Environmental Pollution 108:113-119.

Sijm, D.T.H.M., W. Seinen, and A. Opperhulzen. 1992. Life-cycle biomagnification study in fish. Environmental Science and Technology 26:2162-2174.

Skåre, J.U., J. Stenersen, N. Kveseth, and A. Polder. 1985. Time trends of organochlorine chemical residues in seven sedentary marine fish species from a Norwegian fjord during the period 1972–1982. Archives of Environmental Contamination and Toxicology 22:33-41.

Smith, J.A., P.J. Witkowski, and T.V. Fusillo. 1988. Manmade Organic Compounds in the Surface Waters of the United States - A Review of Current Understanding. U.S. Geological Survey Circular 1007.

Sokol, R.C., C.M. Bethoney, and G.-Y. Rhee. 1998. Reductive dechlorination of preexist ing sediment polychlorinated biphenyls with long-term laboratory incubation. Environmental Toxicology and Chemistry 17:982-987.

Stamer, J.K., T.H. Yorke, and G.L. Pederson. 1985. Distribution and Transport of Trace Substances in the Schuylkill River Basin from Berne to Philadelphia, Pennsylvania. U.S. Geological Survey Water-Supply Paper 2256-A.

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

Swackhamer, D.L., and R.A. Hites. 1988. Occurrence and bioaccumulation of organochlorine compounds in fishes from Siskiwit Lake, Isle Royale, Lake Superior. Environmental Science and Technology 22:543-548.

Tanabe, S. 1988. PCB problems in the future: foresight from current knowledge. Environmental Pollution 50:5-28.

Tate, C.M., and J.S. Heiny. 1996. Organochlorine compounds in bed sediment and fish tissue in the South Platte River Basin, USA, 1992-1993. Archives of Environmental Contamination and Toxicology 30:62-78.

TDH (Texas Department of Health). 1995a. Health Risk Consultation: Fish Tissue Data - Echo Lake, French Lake and Cement Creek Reservoir. Seafood Safety Division, Texas Department of Health, Austin, Texas.

TDH (Texas Department of Health). 1995b. Results and Risk Assessment for Fish Tissue Collected from Fosdic Lake. Seafood Safety Division, Texas Department of Health, Austin, Texas.

TDH (Texas Department of Health). 1995c. Results and Risk Assessment for Fish Tissue Collected from Lake Como. Seafood Safety Division, Texas Department of Health, Austin, Texas.

Ulery, R.L., and M.F. Brown. 1995. Water-Quality Assessment of the Trinity River Basin, Texas – Review and Analysis of Available Pesticide Information, 1968-91. U.S. Geological Survey Water-Resources Investigations Report 94-4218, 88p.

USGS (U.S. Geological Survey). 2000. Pesticides in Stream sediment and Biota. USGS Fact Sheet 092-00.

van der Oost, R., H. Heida, and A. Opperhuizen. 1988. Polychlorinated biphenyl congeners in sediments, plankton, molluscs, crustaceans, and eel in a freshwater lake: Implications of using reference chemicals and indicator organisms in bioaccumulation studies. Archives of Environmental Contamination and Toxicology 17:721-729.

Van Metre, P.C., and E. Callender. 1997. Water-quality trends in White Rock Creek basin from 1912-1994 identified using sediment cores from White Rock Lake reservoir, Dallas, Texas. Journal of Paleolimnology 17:239-249.

Van Metre, P.C., J.T. Wilson, E. Callender, and C.C. Fuller. 1998. Similar rates of decrease of persistent, hydrophobic and particle-reactive contaminants in riverine systems. Environmental Science and Technology 32:3312-3317.

Waters, T.F. 1995. Sediment in Streams: Sources, Biological Effects and Control. American Fisheries Society Monograph 7, Bethesda, Maryland, 251p.

Webster, C.F., T.A. Buchanan, J. Kirkpatrick, and R. Miranda. 1998. Polychlorinated Biphenyls in Donna Reservoir and Contiguous Waters - Results of Intensive Sediment, Water and Fish Sampling and Human Health Risk Assessment. Special Study Report No. AS-161, Field Operations Division, Texas Natural Resource Conservation Commission, Austin.

Zaranko, D.T., R.W. Griffiths, and N.K. Kaushik. 1997. Biomagnification of polychlorinated biphenyls through a riverine food web. Environmental Toxicology and Chemistry 16:1463-1471.

Zhou, H.Y., and M.H. Wong. 2000. Accumulation of sediment-sorbed PCBs in tilapia. Water Research 34:2905-2914.