CHAPTER II
EXPOSURE AND EFFECTS

Section 112(m) of the CAA requires EPA to assess the environmental and human health effects attributable to atmospheric deposition to the Great Waters and to assess whether atmospheric pollutant loadings to the Great Waters cause or contribute to exceedances of drinking water or water quality standards.

Adverse effects on environmental and human health and exceedances of drinking water or water quality standards that result from the pollutants of concern are pieces of a larger puzzle of what happens to the pollutants of concern after they are deposited to the Great Waters. After being deposited to water, the pollutants can bind to particles, concentrate at the water surface, dissolve in the water, and/or (if sufficiently volatile) escape as gases back into the air. Ecosystems and humans may be exposed to these pollutants through various exposure routes (e.g., food consumption). Exceedances of water quality criteria or standards are one means of assessing the levels of the pollutants in water and biota to which ecosystems and humans may be exposed. Following exposure to the pollutants of concern, ecosystems and humans may experience adverse health effects.

At this time, it is not possible to distinguish between effects caused by airborne pollutants and the same pollutants delivered by waterborne or other routes. In the absence of data to the contrary, EPA takes the position that the contribution of airborne pollutants to adverse effects corresponds to the relative air contribution by various routes to pollutant loads, including releases of historic loadings from sediments.

This chapter is divided into five sections that discuss:

- Exposure routes and extent of contamination in the Great Waters (Section II.A);
- Level of contamination in biota of the Great Waters (Section II.B);
- Potential ecological effects that may result from exposure to the pollutants of concern (Section II.C);
- Potential human health effects that may result from exposure to the pollutants of concern (Section II.D); and
- Other potential effects, such as recreational fishing losses, attributable to the pollutants of concern (Section II.E).

Sections II.A, II.C, and II.D update information that was presented in the First Report to Congress (U.S. EPA 1994a). These sections begin with a brief summary of the information presented in the First Report to Congress to provide a foundation for the subsequent discussion of the recent information available for this report.

II.A Exposure Routes and Extent of Contamination

This section presents information on the exposure routes of concern for humans and ecosystems and the extent of contamination in the Great Waters. The measures used in this report to assess pollutant exposure and the extent of contamination are exceedances of water quality criteria and the issuance of fishing restrictions and fish consumption advisories. Levels of contamination in biota are discussed separately in Section II.B.

As noted earlier, the relationship between exposure and resulting adverse effects of toxic pollutants and atmospheric deposition is not well understood. As described in the First Report, some correlations and linkages have been established between specific pollutants of concern and exposure and effects in the Great Waters. Many pollutants that are a concern due to atmospheric deposition also have a long history of direct surface water discharges to the Great Waters. In addition, current pollutant levels in waterbodies may include the contribution of pollutants that enter through groundwater, that are recycled from sediments, or that are resuspended, following earlier deposition, and redeposited at other locations. There currently is no evidence available to suggest that the pollutants deposited from the air will have effects on biota any different from the effects of these same pollutants carried in water or found in sediment.

Conclusions from the First Report to Congress

As mentioned above, information from the First Report to Congress is presented here to provide a foundation for the subsequent discussion of the recent information available for the Second Report. The research findings and studies presented in the First Report, as well as the background document on exposure and effects (Swain et al. 1992a), led to the following conclusions concerning exposure routes and extent of contamination in the Great Waters:

- For water pollutants that are derived from atmospheric deposition, the major routes of exposure are fairly well understood. The main exposure routes of concern for animals are intake of food, intake of drinking water, and direct contact with water. Exposure routes for plants include water uptake and direct contact. For humans and fish-eating birds and mammals, intake of food (e.g., contaminated fish) is the main exposure route of concern for pollutants that are persistent in the environment and that tend to bioaccumulate.

- The pollutants of concern generally are persistent in the environment and tend to accumulate in fat or muscle tissue and, as a result of food web interactions, reach the highest concentrations in animals at the top of the food web, including humans. These characteristics allow the pollutants to remain in the environment and animal tissue for long periods of time, increasing the opportunity for exposure and resulting in greater exposures to animals at the top of the food web.

- The tendency of the pollutants of concern to bioconcentrate and biomagnify was supported by numerous studies summarized in the First Report. (See sidebar on next page for definition of terms. Note that these terms may be used with somewhat different emphasis by different authors. This summary is based on common usages from many articles reviewed for this report.) Evidence presented by these studies included: (1) documented cases of elevated levels of persistent toxic pollutants in

various fish species compared to levels in water and, in many cases, levels in sediment; (2) data showing that predators (e.g., the herring gull, bald eagle, and turtle) in the Great Lakes region have had some of the highest reported concentrations of persistent toxic chemicals in their tissues; and (3) data indicating that people who regularly consumed fish from Lake Michigan in the 1970s had significantly higher concentrations of PCBs and pesticides, such as DDT, in their tissues compared with those who did not consume fish.

Distinguishing Common Terms Describing Bioaccumulation

Bioaccumulation is the uptake and retention of a chemical by a living organism as a result of intake of food, intake of drinking water, direct contact, or inhalation.

Bioconcentration is the phenomenon by which chemicals become more concentrated in an organism than in its surrounding environment.

Biomagnification is the phenomenon by which chemicals become more concentrated in animals at higher levels in the food web.

- Based on the most current information available for the First Report to Congress, portions of all of the Great Lakes and many associated waterbodies, Chesapeake Bay, and Lake Champlain had some kind of advisory on fish consumption at that time.

- Understanding of the contribution of atmospheric deposition to overall exposure was limited for the First Report to Congress because: (1) overall exposure to toxic water pollutants had not been adequately quantified; (2) sufficient and accurate information on all pollutant inputs and outputs was not available at that time; and (3) the difficulty in distinguishing the origin of a pollutant (e.g., originated from the air) after it is in the water made it difficult to link exposure, and resultant effects, to particular pathways (e.g., atmospheric deposition).

- Although the exposure routes of concern have been identified, the concentrations of pollutants in water to which humans, animals, and plants are exposed (i.e., the extent of contamination) were not easily determined given available data at that time.

- Few violations of existing drinking water standards (i.e., maximum contaminant levels or MCLs) for the pollutants of concern were found in Great Lakes drinking water systems; for the pollutants that exceeded their MCLs, much of the problem was thought to be caused by the distribution system rather than the water source.

- When maximum open water concentrations from Great Lakes sampling data taken between 1980-1986 were compared to water quality criteria, six pollutants of concern (cadmium, dieldrin, DDT/DDE, hexachlorobenzene, mercury, and PCBs) potentially exceeded at least one criterion in at least one of the Great Lakes (see Appendix B of the First Report to Congress). Maximum concentrations of most of the remaining Great Waters pollutants in most of the lakes approached levels of concern. An updated comparison of sampling data to water quality criteria is presented later in this section.

- In Lake Champlain, limited sampling data indicated that lead was the only pollutant of concern that exceeded applicable water quality criteria. In Chesapeake Bay, a limited number of measured concentrations of cadmium and lead in the tidal

tributaries to the Chesapeake Bay exceeded EPA water quality criteria and state water quality standards prior to 1993.

The remainder of this section presents updated information on exposure routes and extent of contamination.

Current Understanding of Exposure Routes and Extent of Contamination

As indicated above and in the First Report to Congress, the exposure routes of concern for humans and ecosystems are fairly well understood. Exposures can occur through intake of drinking water, direct contact with water, and, especially important for humans and fish-eating birds and mammals, intake of food. This section presents updated information on measures for assessing the extent of contamination in the Great Waters.

COMPARISON TO WATER QUALITY CRITERIA

One means of assessing the extent of contamination in the Great Waters caused by the pollutants of concern is to compare available water sampling data to drinking water standards and other water quality criteria. Such comparisons are consistent with the requirement in section 112(m) of the 1990 CAA Amendments for EPA to assess the contribution of atmospheric deposition to exceedances of drinking water standards and other water quality standards and criteria.

For national drinking water standards, few exceedances are known for the Great Waters pollutants of concern based on current information in the Great Lakes. Since the First Report to Congress, there continue to be few violations of existing maximum contaminant levels (MCLs) in Great Lakes drinking water systems. Interpretation of this information is limited because the exceedance of the MCL for a pollutant may be a result of a problem in the distribution system, rather than the water source.

For other water quality criteria and standards, three sets of relevant water quality criteria are compared with available Great Waters sampling data: EPA's national ambient water quality criteria (AWQC); the U.S.-Canadian Great Lakes water quality objectives (GLWQOs); and Great Lakes water quality criteria (GLWQC) developed by EPA and Great Lakes states. The first two sets of criteria are the same as those used in the First Report, while the third set, GLWQC, was released in 1995. Proposed GLWQC (pGLWQC) were used in the First Report, but these criteria have since been finalized (U.S. EPA 1995a); see Chapter IV for more discussion on the development of GLWQC. The three sets of criteria are briefly defined in Table II-1.

Water sampling data are compared with water quality criteria, rather than comparing sediment contamination data or biological contamination data to appropriate standards, for two main reasons: (1) the specific requirement in section 112(m) to report exceedances of water quality standards and benchmarks, and (2) the limited availability of federal or other widely accepted numerical benchmarks for sediments or living organisms for the selected pollutants of concern. Because many of the pollutants of concern bioconcentrate and biomagnify, water concentrations may understate the full potential for fish and wildlife to contain high concentration levels; only the GLWQC account for the potential for biomagnification. Therefore, the absence of water quality criteria exceedances for pollutants

that have a strong tendency to bind to sediments and to bioaccumulate does not necessarily indicate the absence of contamination levels of potential human health or ecological concern. Contamination levels in biota and sediments in the Great Waters are discussed in Section II.B.

Table II-2 compares recent estimates of total water column concentrations in the Great Lakes for seven pollutants of concern for which sampling data exist (i.e., DDT/DDE, dieldrin, hexachlorobenzene (HCB), α-HCH, lindane, PCBs, and POM). The data are taken from studies conducted between 1986 and 1991 by EPA, researchers funded by EPA, and Environment Canada. Sampling data for the other pollutants of concern and for other Great Waters were not readily available.

TABLE II-1
Summary of Water Quality Criteria Used for Comparison in This Report

Criteria Set[a]	Summary
Ambient water quality criteria (AWQC)	Designed to protect humans, and freshwater and saltwater animals and plants from harmful effects resulting from chronic and acute exposures. Reflect current knowledge on health and welfare effects, dispersal of pollutants across media, and effects on animal and plant reproduction and communities. Derived entirely with risk-based data (not cost or technology considerations). National criteria provided by EPA as guidelines to states for developing regulations.
Great Lakes water quality objectives (GLWQOs)	Developed through joint U.S.-Canadian agreement. Set for certain chemicals to protect the most sensitive user of the water among humans, aquatic life, and wildlife. For chemicals with no specific GLWQO, concentrations in water (does not specify whether ambient water) and in aquatic organisms should be lower than detection levels.
Great Lakes water quality criteria (GLWQC)	Developed by EPA and Great Lakes States. Specific to the Great Lakes system. Form basis for new state water quality standards for ambient waters of the Great Lakes system. Provided as guidelines to protect aquatic life (for both acute and chronic exposure), wildlife (for exposure through food webs), and humans (for chronic exposure through consumption of both fish and drinking water and through water-related recreation). Includes consideration of biomagnification.

[a] Sources: U.S. EPA 1986, IJC 1978, and U.S. EPA 1995a, respectively.

As shown in Table II-2, total water column concentrations of dieldrin and PCBs exceed their GLWQC at some locations in all of the Great Lakes, where data are available. Note that DDT/DDE may also exceed its GLWQC (sampling data of <0.00006 μg/L versus GLWQC of 0.000011 μg/L), and that the criterion for POM is for polycyclic aromatic hydrocarbons (PAHs), a subset of POM, while sampling data were available only for one POM compound (i.e., benzo(a)pyrene, or B(a)P). In addition, the concentrations of PCBs at some locations in Lakes Erie, Huron, and Ontario are above the AWQC for human health. For the pollutants with sampling data reported in both the First Report to Congress and this report (i.e., dieldrin, DDT/DDE, HCB, and PCBs), the total water column concentrations presented in Table II-2 are generally lower than the concentrations reported in the First Report to Congress and the levels for DDT/DDE and HCB no longer exceed any of the water quality criteria.

TABLE II-2
Comparison of Water Quality Criteria to Pollutant Concentrations in the Great Lakes (µg/L)

Pollutant	National AWQC: Fresh Water Aquatic Life[a]	National AWQC: Human Health[b]	Great Lakes Water Quality Agreement Objective[c]	Great Lakes Water Quality Criterion[d]	Total Water Column Concentration[e]				
					Lake Superior	Lake Michigan	Lake Huron	Lake Erie	Lake Ontario
DDT/DDE[f]	0.001	0.00024	0.003	0.000011	<0.00006	NA	<0.00006	<0.00006	<0.00006
Dieldrin	0.0019	0.00071	0.001[g]	0.0000065	0.00026	NA	0.00032-0.00035	0.00038	0.00028-0.00032
HCB	—	0.0072	—	0.00045	<0.00004	NA	0.000072	0.000047	0.000036
α-HCH	—	0.092	—	—	0.0011	0.0016	0.0015	0.0011	0.0008-0.0009
Lindane	0.08	0.186	0.01	0.47	0.0004	0.00034	0.00038	0.00049	0.00036
Total PCBs	0.014	0.00079	—	0.0000039	0.00018	0.00020-0.00036	0.0007-0.0009	0.00122	0.0012
POM[h]	—	0.028	—	—	<0.00046	NA	<0.00046	<0.00046	<0.00046

NA=No data available.
Highlighted boxes indicate exceedances of GLWQC; shaded boxes indicate exceedances of AWQC for human health.

[a] Values are for freshwater chronic criteria (U.S. EPA 1986).
[b] Values are for human chronic exposure through both fish consumption and drinking water (U.S. EPA 1986).
[c] Values are for protection of the most sensitive user of the water among humans, aquatic life and wildlife (IJC 1978).
[d] Values are the most stringent (i.e., lowest) among those for protection of human health, aquatic life, or wildlife (U.S. EPA 1995a).
[e] Concentrations are taken from De Vault et al. (1995) and L'Italien (1993). Concentrations of dieldrin and PCBs that are reported as ranges represent two different concentrations reported in two different studies. For α-HCH, the range of concentrations in Lake Ontario represents the range reported in a single study.
[f] Sampling data are for p,p'-DDE.
[g] Value for aldrin and dieldrin combined.
[h] AWQC for human health is for polycyclic aromatic hydrocarbons (PAHs), a subset of POM; sampling data are for benzo(a)pyrene, a PAH.

Meaningful trend data on pollutant concentrations in the Great Lakes are limited mainly because the technology required to measure pollutants at the trace concentrations found in the water column of the Great Lakes has become widely available only in the last few years (De Vault et al. 1995). One recent study, however, has provided insight into water column trends for PCBs in Lake Superior (Jeremiason et al. 1994). As shown in Table II-3, total PCB concentrations in the Lake Superior water column show an overall decline from 1978 to 1992, though there is some variation from year to year. In addition, based on the concentrations of the same 25 PCB congeners measured in surface water samples collected between 1980 and 1992 by the same laboratory (also shown in Table II-3), the concentrations of these PCB congeners has decreased at a rate of approximately -0.00020 μg/L per year. (For details on the collection methods and laboratory techniques used in this study, refer to Jeremiason et al. (1994).) The researchers believe that, due to the remote location of Lake Superior and the absence of significant point source loadings, the decline in PCB concentrations represents a continental decrease in atmospheric loading of PCBs. (Other research suggests that cycling of PCBs in the environment, including volatilization of gaseous PCBs from waterbodies, is an important consideration (see Chapter IV).) According to the data, the concentrations of PCBs in Lake Superior were above the AWQC for human health until approximately 1983 and still remain above the GLWQC (criteria shown in Table II-2).

TABLE II-3
Concentration of Total PCBs in Lake Superior Water Column

Year	Total PCB Concentration (µg/L)	Total Concentration of 25 PCB Congeners (µg/L)
1978	0.00173 ± 0.00065	NA
1979	0.00404 ± 0.00056	NA
1980	0.00113 ± 0.00011	0.00099 ± 0.00010
1983	0.0008 ± 0.00007	0.00073 ± 0.00006
1986	0.00056 ± 0.00016	0.00055 ± 0.00015
1988	0.00033 ± 0.00004	0.00020 ± 0.00001
1990	0.00032 ± 0.00003	0.00021 ± 0.00001
1992	0.00018 ± 0.00002	0.00009 ± 0.00001

NA = Not applicable
Source: Jeremiason et al. 1994.

FISHING RESTRICTIONS AND FISH CONSUMPTION ADVISORIES

Another measure of contamination of the Great Waters caused by selected pollutants of concern is the existence of fishing restrictions or fish consumption advisories. These advisories are established as a means of limiting human exposure when fish taken from a particular body of water are found to contain levels of pollutants that exceed recommended intake levels (see sidebar on next page). Such advisories have immediate significance to the general public by providing concrete examples of health concerns and affecting the public use of waters and aquatic resources. States issue several different types of advisories for

waterbodies in an effort to reduce health risks associated with exposure to pollutants in certain freshwater fish and shellfish species:

- *Informational health advisories*: advisories that fish tissue contains contaminants but not at levels high enough to warrant advising people to limit consumption.

- *Advisories to limit fish consumption*: advisories to either the general population or subopulations potentially at greater risk (e.g., pregnant or nursing women, those who fish for subsistence reasons) to restrict the size and frequency of meals of fish and shellfish;

- *Advisories against fish consumption*: advisories to either the general population or subpopulations potentially at greater risk (e.g., pregnant or nursing women) against consuming fish and shellfish;

- *No-kill zones*: notification that it is illegal to take, kill, or possess any fish from the specified waters; and

- *Commercial fishing bans*: bans on the commercial harvest and sale of fish and shellfish from the specified waterbody.

Interpreting Fish Advisory Data

Individual states are responsible for issuing fish advisories. Generally, an advisory is issued for a particular waterbody (or portion of waterbody), pollutant, fish species, and advisory type. In many advisories, the size of the fish affects the type of advisory issued (e.g., for walleye < 22", restricting meals in the general population may be advised, while for walleye > 22" not consuming the fish may be advised). For several reasons, comparing advisories quantitatively (e.g., counting the number of advisories per waterbody) is difficult and therefore, this Report does not do so. For example, a waterbody may appear to have more fish advisories than another waterbody, but it may be that: (1) more states are involved (e.g., advisories in Lake Ontario are issued only by New York, while four states issue advisories for Lake Michigan; (2) states have different methods or use different standards for identifying fish species affected by advisories, some of which may be more comprehensive than others; or (3) a state may issue an advisory for "all fish" making it difficult to count this advisory with advisories for particular fish species.

State advisory data are collected by EPA in a national data base. For each advisory, the data base contains information such as waterbody name, pollutant name, fish species, population targeted by advisory (called advisory type in this report), advisory status (e.g., active), and a contact name and telephone number. The data base does not, however, contain information on the levels of pollutants in fish or the benchmark levels set by a particular state for each advisory type. The information in the data base available for use in this report was current through 1995 (U.S. EPA 1996b). For this report, EPA reviewed the data base for any of the above advisories related to the Great Waters pollutants of concern in the Great Lakes, Lake Champlain, Chesapeake Bay, and several coastal waters.[7]

According to the fish advisory data base, no informational health advisories were in effect in any of the Great Waters. A portion of the Hudson River, which empties into the New York/New Jersey Harbor, had an active no-kill zone for all fish related to PCBs. Table II-4

[7] A more detailed description of the criteria EPA used to obtain information related to the Great Waters from the fish advisory data base is provided in Appendix B.

lists the commercial fishing bans that were in effect in the Great Waters. As the table shows, the commercial fishing bans in the Great Waters are all due to PCBs or dioxins.

TABLE II-4
Commercial Fishing Bans in the Great Waters

Waterbody (State Issuing Advisory)	Great Waters Pollutant of Concern	Fish Species
Lake Champlain (NY)	PCBs	Yellow perch
Lake Ontario (NY)	Dioxins, PCBs	Eel
Long Island Sound (NY)	PCBs	Striped bass
New York/New Jersey Harbor (various waters; NJ, NY)	PCBs	American eel, blue crab, carp, goldfish, striped bass
	Dioxins	Blue crab, crustaceans, fish, shellfish, striped bass

Source: U.S. EPA 1996b.

There are many active advisories to limit or avoid fish consumption in the Great Waters. Table II-5 (the Great Lakes and Lake Champlain) and Table II-6 (selected coastal waters, including Chesapeake Bay) indicate the type of advisories in effect for the Great Waters pollutants of concern, in increasing order of severity of the advisory (i.e., from advisories to at-risk subpopulations to restrict fish consumption up to advisories to the general population to not eat certain fish). For the Great Lakes and Lake Champlain, only lakewide advisories are included in Table II-5. Advisories for particular "hot spots" in these lakes (e.g., Green Bay), as well as the lakewide advisories, are presented in Appendix B. In Table II-6, the advisories for coastal waters represent the advisories as they are identified in the data base. Therefore, advisories for some coastal waters represent the entire waterbody (e.g., Tampa Bay), while others represent smaller estuaries or portions of the waterbody (e.g., Baltimore Harbor). Appendix B provides details on the waterbodies that represent the coastal water advisories. The fish consumption advisories shown in the tables have been issued for at least one fish species and, in many cases, have been issued for several fish species. For more detail on the fish species affected by the advisories and the states that issued the advisories, refer to Appendix B.

As shown in Table II-5, fish consumption advisories in the lakes of the Great Waters are most commonly associated with PCBs, followed by mercury, dioxins, and chlordane. For one lake, Lake Superior, an advisory related to toxaphene also has been issued. In the selected coastal waters, as shown in Table II-6, fish consumption advisories are most commonly associated with PCBs, followed by dioxins. Several advisories related to mercury, chlordane, and DDT also have been issued. Current fish advisories generally are associated with the same pollutants of concern as in the First Report. Furthermore, in this report, fish advisory data are available in more detail than were reported in the First Report (e.g., advisories by each state), especially because of the recent availability of the fish advisory data base. In addition, some states may have become more aggressive in their fish advisory programs. Therefore, the increase in number of advisories presented in Appendix B does not necessarily reflect a higher level of contamination in the Great Waters.

TABLE II-5
Fish Consumption Advisories in the Great Lakes and Lake Champlain

Pollutants of Concern	Great Lakes						Lake Champlain
	Lake Superior	Lake Michigan	Lake Huron	Lake Erie	Lake Ontario	Connecting Channels	
Chlordane	■	● □ ■	● □ ■	■			
Dioxins			● □ ■		● □ ■	● □ ■	
Mercury	○ ● □ ■	○ ●				○ ● ■	● □
PCBs	● □ ■	● □ ■	● □ ■	● ■	● □ ■	● □ ■	● □
Toxaphene	■						

TABLE II-6
Fish Consumption Advisories in Selected Coastal Waters

Pollutants of Concern	Chesapeake Bay	Other Coastal Waters					
		Long Island Sound	New York/New Jersey Harbor	Delaware Bay	Tampa Bay	Galveston Bay	San Francisco Bay
Chlordane	●	●		■	●		
Dioxins			○ ● □ ■			● □	○ ● □ ■
DDT							○ ● □ ■
Mercury							○ ● □ ■
PCBs	●	● □ ■	● □ ■	○ ● ■	●		○ ● □ ■

KEY: ○ Advisories to subpopulations potentially at greater risk (e.g., pregnant women) to restrict the size and frequency of meals of fish and shellfish
● Advisories to the general population to restrict the size and frequency of meals of fish and shellfish
□ Advisories to subpopulations potentially at greater risk (e.g., pregnant or nursing women) against consuming fish and shellfish
■ Advisories to the general population against consuming fish and shellfish

Source: U.S. EPA 1996b.

II.B Contamination of Biota

Measurements of pollutant levels in biota provide information about the extent of contamination in the waterbody, as well as potential bioaccumulation in the food web. Section 112(m)(1)(E) of the CAA requires EPA to sample biota, including fish and wildlife, in the Great Waters for hazardous air pollutants and to identify the sources of these pollutants. Because studies are already being performed under national programs such as the National Status and Trends Program, Great Lakes National Program Office, Chesapeake Bay Program, as well as state programs, EPA relies largely on these studies to support implementation of this CAA requirement. These programs provide information on the extent of contamination in the waterbodies, as reflected in tissues of living organisms. Contamination of biota also suggests potential contribution from various exposure routes such as air and water, as well as bioaccumulation in the food web. This section presents a brief overview of biota sampling approaches and how the sampling data provide useful information for assessing the extent of pollutant contamination in a waterbody. This overview includes a summary of two large-scale studies that are designed to assess national pollutant levels in aquatic biota, and how the results from these studies can apply to the Great Waters. This discussion is followed by a summary of research efforts addressing biota contamination specific to the Great Waters.

Sampling Biota for Contamination

Different sampling approaches are used to determine pollutant levels in biota because each monitoring study has its own objectives, such as to identify "hot spots" or characterize a waterbody's general condition. Therefore, caution must be taken in the interpretation of data, as well as statistical analyses applied to these data. To estimate spatial or temporal patterns of contamination, sophisticated sampling designs are often used. Figure II-1 illustrates the importance of sampling various components of an aquatic ecosystem when attempting to characterize the condition of the system.

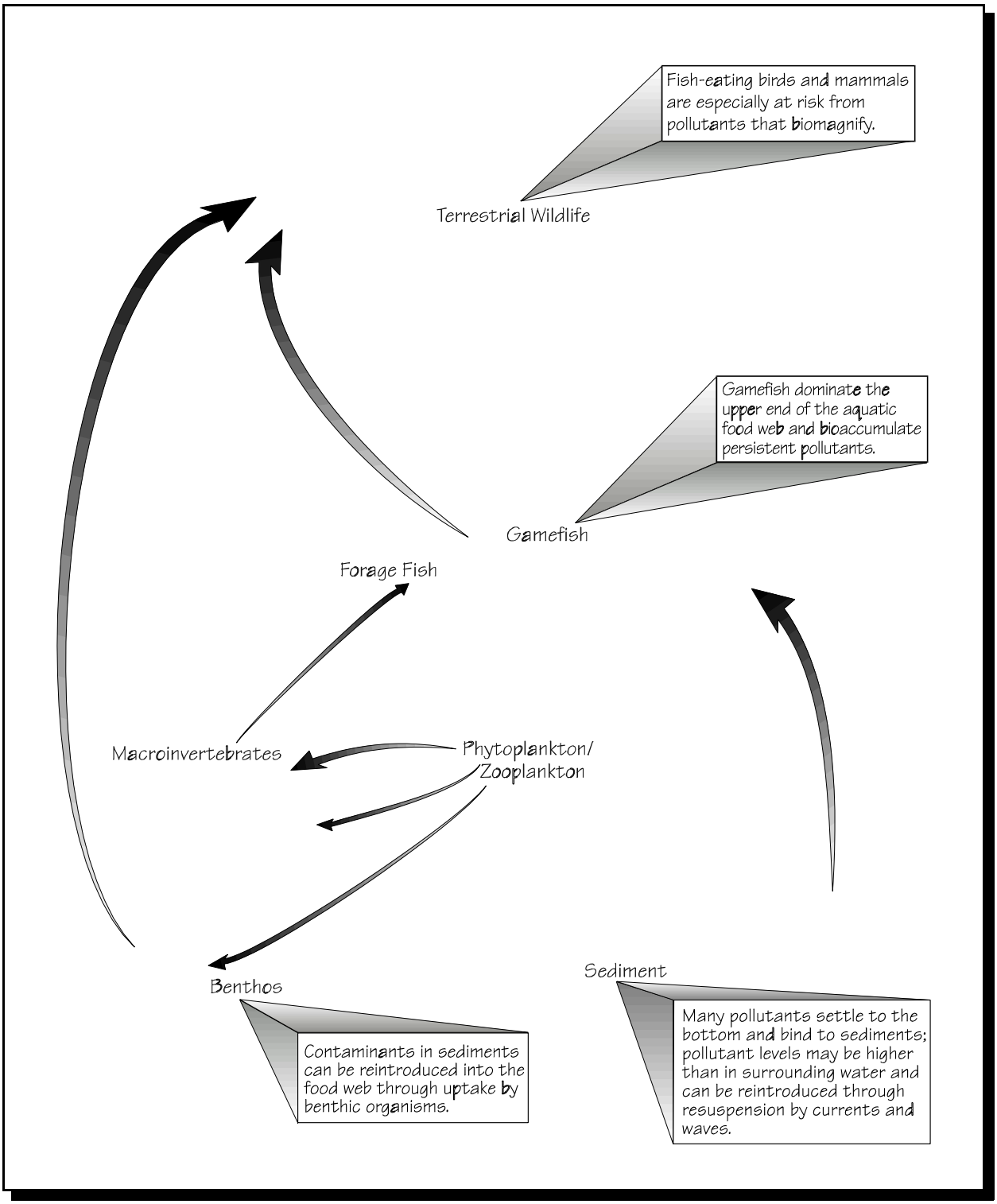
An Example of Bioaccumulation in the Food Web

Of the pathways by which ecosystems and components of ecosystems may be exposed to atmospheric mercury, exposure of high trophic level predatory wildlife to mercury in food is particularly important. Mercury biomagnifies in aquatic food chains, with the result that mercury concentrations in tissue increase as trophic levels increase. Therefore, the trophic level and feeding habits of an animal influence the degree to which that species is exposed to mercury. Predatory animals primarily associated with aquatic food chains accumulate more mercury than those associated with terrestrial food chains. Thus, fish eaters and their predators generally have the highest exposure to mercury. Species with high tissue levels of mercury include otter and mink, which are top mammalian predators of aquatic food chains. Top avian predators of aquatic-based food chains include raptors such as the osprey and bald eagle.

Sources: Bloom 1992; Eisler 1987a; Roelke et al. 1997; Wobeser and Swift 1976; Wren 1985.

In assessing contamination in a waterbody, aquatic and terrestrial species are often collected and analyzed. Game fish are useful bioindicators because they have long life spans, dominate the upper end of the aquatic food web, and can bioaccumulate many of the persistent pollutants (see sidebar). Their population levels may be affected by continued exposure to environmental stresses, such as eutrophication and pollutant contamination, and people become aware of changes in abundance of game fish. Reduced dissolved oxygen concentrations in the water may affect growth, survival, or structural development of fish,

FIGURE II-1
Assessing Contamination in a Waterbody



while pollutant contamination may result in decreased growth, reproduction, or survival of fish (see Section II.C for information on ecological effects). Contamination in fish can enter the diet of humans and other animals (see sidebar). Therefore, terrestrial wildlife, such as fish-eating birds or mammals, are often monitored. While all animals in the aquatic environment have the potential to be affected by pollutants, fish-eating birds and mammals are especially at risk from pollutants that biomagnify because they are frequently exposed to high levels of these pollutants.

Contaminated sediments pose a potential hazard to human health due to biomagnification up the food web. Many pollutants in the water settle to the bottom and bind to sediments or remain in solution in the water between the sediment particles. Although some pollutants can be degraded by bacteria, many persist in sediments for years, even after the original source of contamination has been removed (Howdeshell and Hites 1996). Therefore, pollutant levels in the sediment may be higher than the concentrations in the surrounding water and/or biota, and should be interpreted cautiously when extrapolating to potential levels in biota. Pollutants in the contaminated sediment can also be reintroduced into the water column and can enter the food web through benthic (bottom-dwelling) organisms (e.g., clams, crustaceans, and worms), which are prey to larger fish. Benthic community structure, as well as pollutants in tissues of benthic organisms, may serve as useful indicators of sediment contamination.

EPA Guidance For Issuing Fish and Wildlife Consumption Advisories

In response to a 1990 request from the states, EPA established The Fish and Wildlife Contamination Program to assist states and tribes assess and reduce health risks associated with exposure to chemical contaminants in noncommercially harvested (e.g., subsistence) fish and wildlife. In partnership with states and tribes, EPA has developed a series of guidance documents which provide a scientifically sound, cost-effective method for developing, issuing, managing, and communicating fish consumption advisories. Though many states use the EPA guidance for developing fish and wildlife consumption advisories, some states use parts of the guidance selectively. A few states still apply outdates methods for characterizing contaminants. EPA continues to work with the states and tribes to establish a national consistency in the methods used for characterizing the risks posed by contaminants in noncommercial fish and wildlife.

The Fish and Wildlife Contamination Program has also worked with the states and tribes in organizing training workshops and national conferences. In addition, EPA manages national data bases such as the *Listing of Fish and Wildlife Consumption Advisories* (discussed in Section II.A). The listing, which is updated annually, includes an inventory of all fish and wildlife consumption advisories issued by the states and tribes. Because of additional sampling due to the increased attention given to fish contamination, the number of advisories issued by the states and tribes has increased 72 percent, from 1,278 advisories in 1993 to 2,193 advisories issued in 1996. Most of these advisories have been issued due to mercury contamination.

Wide-scale monitoring studies have been conducted on pollutant contamination of biota to assess large-scale regional and national impacts in U.S. waters. These programs provide valuable information about major pollutants (e.g., where they are found and where they bioaccumulate) and the extent of contamination in the waterbodies, although the source of contamination (or contribution of atmospheric deposition) is generally not evaluated.

Two large-scale programs that assess the contamination of biota in major U.S. waterbodies or coastal areas are the NOAA National Status and Trends (NS&T) Program and the EPA Environmental Monitoring and Assessment Program (EMAP). The NS&T Program monitors trends of more than 70 chemical contaminants (organic compounds and trace

metals) in bottom-feeding fish, shellfish, and sediments at almost 300 coastal and estuarine locations throughout the United States. A well-known project of the NS&T is the Mussel Watch. EMAP has carried out regional-scale studies, on both terrestrial and aquatic species. The data collected for EMAP-Estuaries, to quantify conditions in coastal regions of the United States, are useful to the Great Waters. Sampling and subsequent analyses in EMAP studies are generally not directed at a specific waterbody, but encompass a larger geographic region, which may include waterbodies of the Great Waters. These two projects provide information on assessing the extent of pollutant contamination in U.S. waterbodies; however, the application of their findings to individual waterbodies is limited since these projects were not designed to address concerns specific to the Great Waters.

NS&T MUSSEL WATCH PROJECT

Initiated in 1986, this continuing project is directed at tracking temporal trends in concentrations of pollutants (e.g., PAHs, PCBs, pesticides, metals) found in whole soft-parts of mollusks at about 255 coastal and estuarine sites on the Atlantic, Pacific, and Gulf coasts (including Great Waters such as Chesapeake Bay and Galveston Bay).

Systematic Sampling Approach

The Mussel Watch project is designed to provide long-term and large-scale monitoring of pollutant distribution, looking for temporal (not spatial) trends. The sampling design is set up to be *representative of large areas* rather than small-scale patches of contamination. The objective is to get a representative picture of the general or "average" conditions of the U.S. coastal waters. Therefore, a systematic sampling approach is used. Also, it is useful for estimating statistically the average concentration of pollutants when general trends or patterns in concentration are known from other sources of information.

Since no single species of mollusk is common to all waters, a number of species have been sampled for this project, including several common to the Great Waters — blue mussels (*Mytilus edulis*) from the Northeast and West Coasts, American oysters (*Crassostrea virginica*) from the Mid-Atlantic and Gulf coasts, and zebra mussels (*Dreissena polymorpha*) from the Great Lakes. Mussels and oysters are useful for monitoring changes in the pollutant levels because they remain at fixed sites, and concentrations in their tissues reflect, in general, changes in the concentrations in the surrounding water. There are species differences, so data need careful interpretation. Sampling for this project is performed during the same season each year to reduce the influence of seasonal cycles on natural factors (e.g., salinity, reproductive state). Sites that were selected support adequate populations of these mollusks such that sufficient samples are available annually over many years (O'Connor 1992; O'Connor and Beliaeff 1995).

Table II-7 is a general representation of the pollutant trends in mollusks during the 1986-1993 period at Great Waters sites based on analysis of data collected for the Mussel Watch Project (O'Connor and Beliaeff 1995). All of the trends shown in Table II-7 are statistically significant (confidence level of 90 percent and above); however, quantitative information regarding the changes in pollutant levels was not provided by O'Connor and Beliaeff (1995). Consequently, the magnitudes of the increases or decreases cannot be compared between sites (i.e., one cannot determine which sites showed the most improvement). However, useful information regarding general pollutant trends is presented.

As shown in Table II-7, most of the Great Waters sites did not exhibit a statistically significant trend or change in pollutant levels in mollusk tissues during the eight-year period.

Trends, when present, were mainly downward since 1986 (i.e., for cadmium, PCBs, DDT, PAHs, chlordane, and dieldrin). According to the investigators, decreases in levels of these chemicals are probably the result of bans on the use of chlorinated hydrocarbons and the reduced use of certain pollutants (O'Connor and Beliaeff 1995).

The level of mercury in mollusks showed an increasing trend over the eight-year period only in one Galveston Bay site (O'Connor and Beliaeff 1995). During the four-year period from 1986 to 1990, mercury concentrations increased at several other sites (O'Connor 1992); however, mercury levels stabilized at these sites by 1993 so that no statistically significant trend was apparent over the eight-year period (O'Connor and Beliaeff 1995). Lead levels in mollusks also exhibited an increasing trend at one site in Galveston Bay and one site in Tampa Bay during the eight-year period (O'Connor and Beliaeff 1995); however, no change in lead levels was evident at these sites between 1986 and 1990 (O'Connor 1992). The investigators concluded that because the high concentrations of mercury and lead were found, for the most part, in the vicinity of population centers, they may be attributable to human activities (O'Connor and Beliaeff 1995).

The information presented in Table II-7 and discussed above focuses only on trends in contaminant levels in mollusks based on the Mussel Watch Project data. For the pollutants that show no significant trends over the eight-year period, it does not necessarily mean that no changes are occurring with respect to contamination of biota at Great Waters sites. Other analyses of the Mussel Watch Project data may indicate different patterns of pollutant concentrations in mollusks. In addition, gamefish and fish-eating birds and mammals are much higher in the food web than mussels and oysters, and therefore, may exhibit different patterns of pollutant concentrations in their tissues.

TABLE II-7
Eight-Year Trends of Pollutant Concentrations in Mussel Watch Project (1986-1993)

Waterbodies (number of sites examined)	Contaminant Trend (number of sites affected)[a]							
	Mercury	Lead	Cadmium	PCB	DDT	PAH	Chlordane	Dieldrin
Chesapeake Bay (5 sites)	↔(5)	↔(5)	↓ (1) ↔(4)	↓ (3) ↔(2)	↓ (3) ↔(2)	↔(5)	↓ (4) ↔(1)	↓ (3) ↔(2)
Delaware Bay (4 sites)	↔(4)	↔(4)	↓ (1) ↔(3)	↓ (2) ↔(2)	↔(4)	↔(4)	↓ (1) ↔(3)	↔(4)
Long Island Sound (9 sites)	↔(9)	↓ (1) ↔(8)	↓ (4) ↔(5)	↓ (5) ↔(4)	↓ (2) ↔(7)	↓ (1) ↔(8)	↓ (3) ↔(6)	↓ (1) ↔(8)
Narragansett Bay (2 sites)	↔(2)	↓ (1) ↔(1)	↔(2)	↔(2)	↔(2)	↔(2)	↔(2)	↔(2)
Tampa Bay (3 sites)	↔(3)	↑ (1) ↔(2)	↔(3)	↓ (1) ↔(2)	↓ (1) ↔(2)	↔(3)	↓ (2) ↔(1)	↔(3)
Galveston Bay (6 sites)	↑ (1) ↔(5)	↑ (1) ↔(5)	↔(6)	↓ (2) ↔(4)	↓ (1) ↔(5)	↔(6)	↓ (4) ↔(2)	↓ (3) ↔(3)

[a] Represents trends in annually measured concentrations of contaminants in mollusks over 8-year period; trend indicated by arrow (i.e., ↓ = decreasing trend; ↑ = increasing trend; ↔ = no trend). Number of sites showing trend within each waterbody is indicated in parentheses. Sites were sampled in at least six of the eight years. All trends shown are statistically significant.

Adapted from O'Connor and Beliaeff (1995).

EMAP-ESTUARIES

> **EMAP** is a national program initiated in 1989 in response to the EPA Science Advisory Board's recommendation to monitor the status and trends of the U.S. ecological resources -- terrestrial, freshwater, and marine. The program is directed by EPA's Office of Research and Development, with participation by other federal agencies (e.g., NOAA, U.S. Forest Service, U.S. Fish and Wildlife Service).

One goal of EMAP is to quantitatively evaluate the condition of coastal estuaries, by investigating several environmental conditions: hypoxia (low oxygen levels), sediment contamination, coastal eutrophication, and habitat loss. A probability-based approach is used, which allows estimates to be made of the uncertainty associated with assessments and improves the ability to identify ecological responses to pollution. Of interest to the Great Waters are the EMAP-Estuaries results obtained for the Virginian Province (Cape Cod to the Chesapeake Bay) and Louisianian Province (Texas to west coast of Florida). Statistical data collected provide primarily quantitative information on a regional scale.

Results are available for the Virginian Province for 1992 (trend data for 1990-1993 are currently being analyzed), which include sampling results for two relevant Great Waters sites, the Chesapeake Bay (as well as connecting tributaries and small water systems) (53 sampling stations) and Long Island Sound (14 stations). Together, the two waterbodies represent approximately 63 percent of the surface area of the entire province. One of the environmental indicators assessed in the study was sediment contamination. Results showed that metal concentrations in the sediment for the Chesapeake Bay were similar to the concentrations for the overall Province (≈24 μg/g for lead; ≈0.054 μg/g for mercury; ≈0.206 μg/g for cadmium). In Long Island Sound, the concentrations of some metals in the sediment (44.2 μg/g lead; 0.088 μg/g for mercury) were higher than those reported for the Chesapeake Bay and the overall Virginian Province. These results suggest that Long Island Sound exhibits slightly higher contamination of some metals in sediment compared to most other waterbodies in the east coast of the United States. Comparing results among specific waterbodies within the regional area is limited because the density of sampling points was not designed to thoroughly characterize each waterbody separately. Also, uncertainty exists in the analysis of these waterbodies due to the short data collection period (one year) (Strobel et al. 1994).

Biota Contamination by Major Waterbody

This subsection presents information from some monitoring studies and investigations of the pollutant levels in tissues of biota living in the Great Waters. These studies, most of which measure concentrations of metals and organochlorines in fish, provide evidence that toxic contamination is occurring in biota from these waters. In most instances, the researchers focused on pollutant levels in tissues of the biota, and did not look for toxic lesions or other adverse effects. Nevertheless, some of the long-term studies provide information on potential contamination trends in the species examined. In addition, studies that monitor toxic contamination in sediment are presented because contaminated sediment may be a long-term source of pollutant exposure to the aquatic biota and food web. These studies suggest continuing contamination in the waterbody.

GREAT LAKES

Contaminant concentrations in gamefish from the open waters of the Great Lakes have been monitored for over 20 years and provide one of the most extensive data bases on trends of environmental contaminants in organisms at the upper end of the food web. Three monitoring efforts that data have come from include: (1) lake trout monitored by Fisheries and Oceans Canada; (2) lake trout and walleye cooperatively monitored in U.S. waters by EPA's Great Lakes National Program Office, U.S. National Biological Service, and the Great Lakes States; and (3) coho salmon fillets cooperatively monitored in U.S. waters by the Great Lakes States, FDA, and EPA's Great Lakes National Program Office.

PCB Contamination in Great Lakes Biota

Lake Trout/Walleye: During the period 1977-1992, PCB concentrations in lake trout, as well as walleye in Lake Erie, declined significantly, but in recent years, concentrations have generally remained stable or increased slightly in Lakes Michigan, Huron, Superior, and Erie.

Coho Salmon: PCB concentrations in coho salmon collected from Lake Michigan declined from 1.9 μg/g (1980) to 0.38 μg/g (1983), but then increased to 1.09 μg/g (1992). A similar pattern was observed in Lake Erie and the upper reaches of the Saint Lawrence River.

Herring Gull Eggs: Monitored since 1974, the greatest decline in PCB contamination in herring gull eggs occurred between 1974 and 1981. Since then, the rate of decrease has leveled off, and by 1991, slight increases were reported in the levels of some PCBs.

Sources: De Vault et al. 1995, 1996.

These monitoring efforts have demonstrated that, while significant declines in PCB and DDT concentrations in lake trout, walleye, and coho salmon have been observed over the past two decades, the amount of residues of PCBs and DDT in these fish have leveled off or even increased slightly in the last ten years (De Vault et al. 1995, 1996) (see sidebar). This change in trend has occurred despite declining ambient water concentrations of PCBs. A similar trend has been noted in the levels of PCBs and DDT in herring gull eggs, which have been monitored by the Canadian Wildlife Service since 1974.

Lake condition changes can complicate pollutant contamination issues. The strong correlation between trends in DDT and PCBs suggests that changes in the composition of the food web (or trophic structure) may be partly responsible for increases in contaminant concentrations at the upper end of the food web (i.e., gamefish) (De Vault et al. 1995, 1996). For example, research by Haffner (1994) and Stow et al. (1995) on the exposure dynamics of organic pollutants in Lakes Erie and Michigan suggests that changes in the food web could be the cause of the observed increase in PCB contamination in biota. Large, regional pools of PCBs can be reintroduced from sediments by benthic organisms. Changes in the species composition at the mid-trophic levels of the food web may biomagnify greater amounts of PCBs to higher trophic levels. Increased PCB levels in certain predator fish also may be due to reductions in their growth rate.

Evidence of changes in the exposure dynamics of organic contaminants has been observed in the western basin of Lake Erie with the invasion of zebra and quagga mussels (Haffner 1994). These mussels increase biomagnification of pollutants in the benthic food web by consuming significant amounts of phytoplankton that are contaminated with pollutants. The major predator of the zebra mussel is the drum (a low-trophic level fish),

which in turn is a preferred prey of the herring gull. By concentrating contaminants contained in phytoplankton and other suspended organic particles, zebra and quagga mussels can cause PCB levels in certain fish and in herring gulls to increase even though ambient water concentrations of contaminants are decreasing.

The three monitoring programs discussed above also provide information on the levels of two pesticides, dieldrin and toxaphene, in upper trophic-level fish from the Great Lakes. Dieldrin concentrations have exhibited a general pattern of decline in the Great Lakes since the 1970s (De Vault et al. 1995, 1996). Peak levels were noted in 1979 in Lakes Michigan, Superior, Huron, and Ontario and again in 1984 in Lakes Superior, Huron, Ontario, and Erie. Toxaphene concentrations are highest in lake trout from Lakes Michigan and Superior and lowest in lake trout from Lakes Erie and Ontario (De Vault et al. 1996). Lake trout from Lake Michigan sampled between 1982 and 1992 suggest declining levels of toxaphene during this period; however, there was no significant change in concentrations of Lake Superior lake trout (Glassmeyer et al. 1997). A similar trend was observed in rainbow smelt. The drop in toxaphene concentrations coincides with the U.S. ban on toxaphene in 1982. The investigators speculate that the lack of decline in Lake Superior is due to either a lake-specific source that continues to load toxaphene into Lake Superior or a slower removal rate in Lake Superior compared to the other Great Lakes (more discussion in Section IV.A on the Great Lakes).

Sediment Core Data in the Great Lakes

Atmospheric pollutant loadings into the Great Lakes region are estimated from mass balance studies and modeling data, although indirect measures of contaminant loadings, such as sediment core data, are also desirable. Recent measurements of sediment core data have shown declining concentrations of PAHs in Lake Michigan, PCBs and DDT in Lakes Michigan and Ontario, and lead and mercury in Lakes Superior, Michigan, and Ontario. Comparison of sediment data between the Great Lakes provides additional information on sources of loadings. For example, toxaphene has long been thought to result from long-range atmospheric transport from the southeastern U.S.; however, sediment cores from Lake Superior and upper Lake Michigan suggest little decline in toxaphene (contrary to declines observed for DDT, mercury, and PCBs in these lakes). Efforts are underway to examine this issue.

Sources: De Vault et al. 1995; Simcik et al. 1996.

In contrast to the monitoring studies of gamefish, monitoring of forage fish provides an indication of contamination at lower levels of the food web. Rainbow smelt have been routinely monitored in Lakes Superior, Huron, Erie, and Ontario by Fisheries and Oceans Canada since 1977. During this time, concentrations of PCBs, mercury, and total DDT have declined significantly in smelt from these lakes (De Vault et al. 1995). Smelt from Lake Ontario consistently have the highest tissue concentrations of PCBs (~0.5–2.25 μg/g) and total DDT (~0.15–0.6 μg/g), while those from Lake Superior have the highest mercury levels (~0.02–0.1 μg/g).

Contaminant concentrations in young-of-the-year spottail shiners are useful indicators of local, recent pollutant inputs into aquatic ecosystems because they do not travel extensively during their first year of life. Surveillance of these fish by the Ontario Ministry of Environment and Energy (primarily in Canadian waters) has shown a general decline in tissue PCB and DDT concentrations (De Vault et al. 1995). Contaminant levels also have been assessed in young-of-the-year fish from the New York waters of the Great Lakes (Skinner et al. 1994). Elevated concentrations of PCBs were found mainly in fish from the St. Lawrence River drainage area

below the Moses-Saunders Dam in Massena; these levels were attributed to industrial activities in the area. Levels of mercury in these fish were low (<100 ng/g).

Zebra mussels have been considered to be potential system-wide biomonitors of organic contamination trends in the Great Lakes (Comba et al. 1996). Researchers collected and analyzed specimens from 24 sites in Lake Erie, Lake Ontario, and the St. Lawrence River between 1990 and 1992 for residues of PCBs and organochlorine pesticides. Mean concentrations of 154 ng/g total PCBs, 8.4 ng/g total DDT, and 3.5 ng/g total chlordane (whole mussel dry weight basis) were reported. Concentrations varied greatly between sites (e.g., 22–497 ng/g total PCBs), which the researchers attributed to the sensitivity of these mussels to different levels of contamination (Comba et al. 1996). The investigators also indicated that the observations of spatial contaminant trends in the study were similar to findings from other biomonitoring programs.

LAKE CHAMPLAIN

Current efforts to monitor toxic pollution in Lake Champlain have focused on fish and sediment contamination by metals and organic compounds. The Vermont Department of Environmental Conservation implemented a study to analyze soft tissue from mussels (*Elliptio complanata*) as a bioindicator for the lake. Mussels were collected at mouths of several Lake Champlain tributaries; chlordane and PAHs were detected in the mussels (LCBP 1994). In 1987-1988, the States of Vermont and New York analyzed fish tissue collected from Lake Champlain for 17 contaminants. Elevated levels of PCBs were found in large lake trout and in American eel and brown bullhead. The findings of this study led, in part, to health advisories being issued against eating these fish species in Lake Champlain (LCBP 1994).

Because of elevated levels of pollutants in Lake Champlain, the Lake Champlain Sediment Toxics Assessment Program was initiated (McIntosh 1994). Pollutants that were measured included trace metals (cadmium, mercury, lead) and organic compounds (PCBs, PAHs, dioxins/furans). Pollutant levels were measured at nine sites during 1991 and 1992. Findings after the end of the first phase (May 1993) provided little evidence of widespread high-level contamination (although high levels of PCBs and PAHs were measured in sediment near two dock sites). The study did find widely varying patterns of contamination. It appears that, at some sites, some pollutants had higher concentrations in the deeper layers of the sediments, with the upper layers of sediment showing less contamination, while other pollutants exhibited a reversal of this pattern (McIntosh 1994); however, the investigator did not provide an explanation for this deposition pattern. The local and/or regional source of the contamination is not known.

As part of the Lake Champlain Sediment Toxics Assessment Program, a biological assessment of the contaminated sediments was also performed (McIntosh 1994). Most of the year, lake trout do not inhabit bottom waters near the sediment-water interface. However, concerns exist for the mechanisms that may link lake trout to PCB-contaminated sediment. This issue was evaluated by looking at one possible link, the freshwater shrimp *Mysis relicta* (or mysids). Mysids are believed to be a major component in the Lake Champlain food web, and the high lipid content of these organisms make them potential accumulators of PCBs. Laboratory experiments demonstrated that exposure to PCB-contaminated sediment results in high levels of PCBs in the mysids (McIntosh 1994). However, there was no attempt to predict the potential of mysids to redistribute PCBs within the sediments in Lake Champlain.

CHESAPEAKE BAY

Adverse effects that could be related to pollutants accumulating in the tissues of organisms, such as reduced growth, reproduction effects, and tumor development, have been reported in aquatic organisms in a variety of habitats in the Bay from the 1980s to the early 1990s. The Chesapeake Bay Program has sponsored forums to assess contaminant levels in biota and to reach a consensus regarding the trends in the pollutants found in biota and in sediment. While significant declines in metal contamination of fish tissue have been observed over the past two decades, elevated metal concentrations have been measured in fish in specific, more industrialized areas of Chesapeake Bay (CBP 1994b).

Studies conducted from 1970 to 1992 show that chemical contamination has caused various effects to wildlife in the Chesapeake Bay during the 1970s and 1980s (CBP 1994b). In 1991, the *Status and Assessment of Chesapeake Bay Wildlife Contamination Forum* was held to critically review data on the effects of exposure and uptake of pollutants on Chesapeake Bay basin birds, mammals, reptiles, and amphibians. The committee concluded that there was little evidence to suggest pollutants were posing a serious *direct* hazard to birds in the early 1990s. Instead, it is more likely that indirect effects on wildlife habitats and food sources (e.g., excessive nutrients, suspended sediments, herbicides) have greater impacts on bird populations. The forum found that there were insufficient information available at the time to fully assess potential adverse impacts of chemical contamination on mammalian, reptile, and amphibian populations in the Chesapeake Bay basin (CBP 1994b). Due to more recent scientific information on some pollutants' mechanisms of action such as endocrine disruption (see Sections II.C and II.D), the conclusions of the 1991 forum may need to be reevaluated with more current data on pollutant contamination in Chesapeake Bay biota.

In 1993, the *Chesapeake Bay Finfish and Shellfish Tissue Contamination Critical Issues Forum* was held to address the following issues: (1) magnitude and extent of fish and shellfish contamination in the Chesapeake Bay and its basin; (2) impact (i.e., bioaccumulation, toxicity) of the contamination at basinwide, baywide, regional, or local scales; and (3) comparison of the contamination to that of other waterbodies (e.g., Puget Sound, Great Lakes) (CBP 1994b). The data compiled by the forum indicate that finfish and shellfish tissue contaminant concentrations declined significantly after the 1970s for several metals, pesticides, and organic chemicals. For fish species combined, concentrations of PCBs and DDT in fish liver tissue are in the low range relative to national data. However, at the species-specific level, Atlantic croaker and spot collected from 1984 to 1987 had levels of chlordane, PCBs, dieldrin, and total DDT in the liver above the national average and national median for these species. Lead and mercury concentrations in croaker livers were generally above the national average and national median, while lead concentrations in spot livers were sporadically high. The concentrations of PCBs, chlordane, dieldrin, DDT, cadmium, and mercury in oysters in the Chesapeake Bay have declined between 1986 and 1991. Levels of mercury, chlordane, toxaphene, and PCBs in finfish from the Chesapeake Bay basin "hot spots" (e.g., Baltimore Harbor, Back River) are generally well below those found at other areas considered contaminated (e.g., New York/New Jersey Harbor, Lake Michigan, Boston Harbor).

OTHER COASTAL AREAS

Several monitoring studies of biota contamination also have been performed in other coastal waterbodies of the Great Waters. Some relevant findings are highlighted below;

however, additional research is needed to provide a more complete picture of biota contamination in some of these coastal areas.

- *Galveston Bay*. There is little information about historical trends and concentrations of pollutants in aquatic organisms from Galveston Bay. For this reason, the Galveston Bay National Estuary Program initiated a study to characterize pollutant contamination in edible fish and shellfish in the bay. Between 1991 and 1993, 14 fish species, two shellfish species, and three bird species were sampled for numerous pollutants, including several Great Waters pollutants of concern. No "hot spots" of biota contamination were detected and the fish tissue concentrations rarely exceeded FDA criteria for these contaminants (Brooks et al. 1992). The study did not evaluate dioxins, but fish consumption advisories to protect the general population currently exist for dioxins in Galveston Bay.

- *Tampa Bay*. Tissue concentrations of organic compounds and trace metals in Tampa Bay oysters (*C. Virginica*) (sampled between 1986 and 1989) were compared to reported levels for adverse effects to mussels at other sites (Long et al. 1991). The average concentration of total PCBs in the Tampa Bay oysters collected over four years was 0.15 ppm dry weight; total PCB concentration of 0.18 ppm or greater occurred in 11 of 55 samples. Investigators determined that, for total PCBs, adverse effects (e.g., reproductive effects, cellular damage, and biochemical changes) in mussels were associated with tissue concentrations of 0.18 to 1.43 ppm dry weight. The average concentration of mercury in the Tampa Bay oysters collected over four years was 0.27 ppm dry weight; mercury concentration of 0.4 ppm or greater occurred in 13 of 55 samples. Adverse effects (e.g., pathological and enzymatic responses) in mussels were associated with mercury tissue concentration of 0.4 ppm dry weight. Based on these data, Long et al. (1991) concluded that the PCB and mercury concentrations in some oysters in Tampa Bay may be sufficient to cause potential adverse biological effects.

- *New York-New Jersey Harbor and New York Bight*. In 1993, 23 species of fish, six species of bivalves, two species of crustaceans, and one species of cephalopod were collected from six areas of the New York-New Jersey Harbor, including the New York Bight Apex (Skinner et al. 1996). The samples were analyzed for contaminants, including PCBs, organochlorine pesticides, and mercury. Of these compounds, PCBs were the primary chemical contaminants of concern. Average total PCB concentrations for American eel, striped bass, white perch, bluefish, rainbow smelt, and the hepatopancreas of blue crab and American lobster in one or more areas of the harbor estuary exceeded the FDA tolerance level for PCBs (2,000 ng/g). In blue crab and American lobster, PCB and organochlorine pesticide residue concentrations were particularly elevated in tissues with high lipid content (e.g., hepatopancreas). The researchers noted that although relatively low levels of contamination were found in muscle tissue, increased contaminant levels in the hepatopancreas may present a substantially increased risk to those people who choose to eat this tissue (Skinner et al. 1996). PCB concentrations were highest in the Hudson River and Upper Bay, the East River, and the Arthur Kill-Kill Van Kull-Newark complex; concentrations were lowest in Jamaica Bay and the New York Bight Apex. The principal components of the PCB concentrations observed were the higher chlorinated congeners.

Residues of certain other contaminants (e.g., DDT, chlordane, dieldrin, and mercury) also exceeded regulatory criteria in some fish tissue samples (Skinner et al. 1996). Analyses were performed for hexachlorobenzene and toxaphene, but they were seldom or never detected.

II.C Ecological Effects

This section is intended to provide a brief overview of the current literature on the potential ecological effects (generally, adverse effects to aquatic organisms, birds, and mammals) from exposure to the Great Waters pollutants of concern. In general, the information presented in this section represents data published since the background document from the First Report to Congress on exposure and effects (Swain et al. 1992a) was completed through 1995. Because the information presented in this report covers only recent studies, it cannot be used alone to determine whether these effects are widespread in the environment. As in the First Report to Congress, the contribution of atmospheric deposition to toxic contamination and potential ecological effects associated with exposure to the pollutants cannot be quantified at this time.

This section presents information on effects observed in both laboratory and field studies; it does not, however attempt to establish a relationship between the two types of studies. This section also does not provide information on the exposure levels of the pollutants responsible for the observed effects. In addition, this section does not distinguish effects that may occur in wildlife with long-term exposure to the pollutants of concern from effects caused by acute, high-level exposures (e.g., accidental spills). The potential effects of a pollutant may vary with duration of exposure, possibly due to a breakdown of the chemical in the body to another chemical that is more toxic or affects other target organs than the exposed chemical. Furthermore, adverse effects on ecological health caused by exposure to toxic contaminants are not often easy to distinguish from other stresses. For example, fish populations in the Great Lakes suffer from habitat loss, overfishing, and the introduction of non-native species, in addition to the effects from toxic contaminant exposure (U.S. EPA 1995a). Finally, studies on ecological effects generally do not determine the exposure pathway of the pollutants (e.g., atmospheric deposition). In this report, under future directions for research, EPA recommends coordinated analyses of persistent pollutants that relate field measurements of concentrations to estimated exposure and associated effects observed in biota.

Conclusions from the First Report to Congress

Information from the First Report to Congress is presented here to provide a foundation for the subsequent discussion of the recent information available for this report. The First Report to Congress, as well as the background document on exposure and effects (Swain et al. 1992a), identified many adverse ecological effects, at both the individual species level and the ecosystem level, associated with the pollutants of concern. In addition, the First Report to Congress discussed eutrophication of estuarine waters, which is the main ecological effect relevant to this report associated with nitrogen loading. Research findings and studies presented in the First Report to Congress led to the following general conclusions concerning potential ecological effects:

- The selected pollutants of concern have been linked to a broad range of effects at the individual species level in aquatic organisms and other wildlife, including effects on the reproductive, nervous, immune, and endocrine systems, and changes in enzyme functioning.

- Reproductive effects of certain pollutants of concern include reduced fertility, increased embryo toxicity, reduced hatchability, reduced survival of offspring, abnormalities in

offspring, parental behavior change, and changes in mating behavior (e.g., impaired hormone activity, changed adult sexual behavior). For example, eggshell thinning in a number of bird species and associated reproductive loss were linked to exposure to DDT (and its metabolite DDE) in the 1960s and 1970s. Recent decreases in environmental concentrations of reproductive pollutants of concern, such as DDT and PCBs, are correlated with population recoveries in many bird and other wildlife species; however, some populations in certain regions of the Great Lakes still exhibit higher rates of reproductive failure than in other areas.

Pollutants of Concern for Reproduction

Pollutants of concern that have been linked with reproductive impairment in aquatic and terrestrial wildlife include cadmium, DDT/DDE, dieldrin, lead, lindane, mercury, PCBs, and 2,3,7,8-TCDD.

- Effects on the nervous and endocrine systems may occur at very low exposure levels. For example, wild populations of Great Lakes herring gulls, Forster's terns, and ring-billed gulls exposed to various pollutants of concern have exhibited behavioral changes such as female-female pairings, which result in abnormal incubation activities and nesting behavior, including nest abandonment.

- Several of the pollutants of concern cause changes in enzyme functioning. Studies reported that the activity of enzymes responsible for the breakdown of foreign compounds is greatly increased by most of the pollutants of concern. In fish, the increased activity of these enzymes has been shown to result from exposure to PCBs and PAHs. In birds, "wasting" syndrome (i.e., the condition in which an animal slowly loses body weight until it can no longer sustain itself) has been related to altered enzyme activity resulting from exposure to pollutants such as TCDD.

- Exposure of communities of bottom-dwelling aquatic species in the Great Lakes to toxic chemicals has resulted in significant changes in species diversity and populations. In addition, fish-eating birds such as bald eagles, herring gulls, and Forster's terns in the Great Lakes region have undergone significant population declines since the 1960s. Only in recent years, as water concentrations of pollutants in the Great Lakes have declined, have some species begun to recover. Certain current population recoveries of fish-eating birds are still dependent on migration to Great Lakes breeding colonies from other areas.

- Eutrophication[8] is one of the most serious pollution problems facing estuarine waters of the United States. Atmospheric deposition of various nitrogen compounds (mostly nitrates and ammonium) can contribute significantly to eutrophication in coastal waters where productivity is usually limited by nitrogen availability. Accelerated eutrophication results in severe ecological effects such as nuisance algal blooms, dieback of underwater plants (due to reduced light penetration), reduced oxygen levels in the water, and reduced populations of fish and shellfish. The reduction in oxygen levels

[8] Eutrophication is an overabundance of nutrients (e.g., nitrogen) in a water body. It is a natural process that typically takes place over hundreds of years, but can be greatly accelerated by additions of nutrients from human activities.

may reduce or eliminate bottom-feeder populations, create conditions that favor different species, or cause dramatic fish kills, resulting in an altered food web.

The remainder of this section presents updated information on ecological effects associated with the Great Waters pollutants of concern.

Current Understanding of Ecological Effects

Since the First Report to Congress, updated information on the pollutants of concern and their effects on aquatic and terrestrial wildlife has become available. This section first discusses some notable research efforts on ecological effects relevant to some Great Waters pollutants of concern and then provides a brief overview of recent data on the potential ecological effects specific to each pollutant of concern.

As introduced in the First Report to Congress, the role of endocrine disruptors in causing adverse effects wildlife and humans is an emerging and controversial issue. Endocrine disruptors were termed "environmental estrogens" in the First Report to Congress; however, because the interference with hormone action was found not to be limited to estrogen, these pollutants are now more generally referred to as "endocrine disruptors." For example, p,p'-DDE (a breakdown product of DDT) has been shown to inhibit the binding of androgen, a male hormone, to receptors, among other androgen actions (Kelce et al. 1995). Several pollutants have been identified as possible endocrine disruptors, including 11 of the 15 pollutants of concern for atmospheric deposition to the Great Waters: chlordane, dieldrin, DDT/DDE, hexachlorobenzene, lead, lindane, mercury (in the form dimethylmercury), PCBs, TCDDs, TCDFs, and toxaphene (e.g., Cassidy et al. 1994; Chowdhury et al. 1993; Colborn et al. 1993; McKinney 1994; Soto et al. 1994; U.S. EPA 1994c). Since the First Report to Congress, scientific research on endocrine disruptors has continued to provide evidence of their adverse effects and has investigated their mechanisms of action (i.e., how they disrupt the endocrine system within the body).

The existence and effects of the hormone-like action of environmental pollutants were first hypothesized in the 1950s and 1960s. In the late 1980s, scientists concerned with noncancer effects of toxic pollutants brought this issue into focus. In July 1991, many scientists whose diverse research interests touched on some aspect of endocrine system disruption convened at the Wingspread Conference. The conference helped identify future research needs for improving the understanding of endocrine disruptors, their mechanisms of action, and their effects (NWF 1994). Many of the adverse effects in wildlife and humans (e.g., reproductive, developmental, and immunological effects) associated with the pollutants of concern are now theorized to be associated with the endocrine-disrupting action of the pollutants. Recent articles published in the mass media have brought this issue widespread attention (Begley and Glick 1994; Suplee 1996; Weiss 1994; Weiss and Lee 1996).

Endocrine disruptors are believed to interfere with the operation of the endocrine system in many ways, such as by mimicking natural hormones or by blocking natural hormones. This interference can potentially disrupt the reproductive and immune systems and adversely affect metabolism, growth, and behavior.

Some of the recent articles demonstrating endocrine disruption by a few of the Great Waters pollutants of concern in aquatic and terrestrial wildlife are briefly summarized below.

- Scientists in central Florida have been observing alligators from a contaminated lake and a control lake. The researchers have found that alligator eggs and newborns from the contaminated lake differ significantly from those in the control lake, showing reduced hatchability, reduced viability of offspring, endocrine "demasculation" of males, and "superfeminization" of females (Guillette et al. 1994). Juvenile alligators exhibit significantly smaller penis size (24 percent decrease) and lower plasma concentrations of testosterone (70 percent lower) when compared to alligators from the control lake (Guillette et al. 1996). The alligators from the contaminated lake have elevated levels of p,p'-DDE, a known endocrine disruptor, in their tissues, which researchers believe are associated with a large spill of a pesticide containing DDT. Studies into the mechanism of action of these effects are ongoing (Guillette et al. 1995, 1996).

- Researchers have been studying the mechanism of action of endocrine disruption in a common turtle species (the red-eared slider) and the African clawed frog (Palmer and Palmer 1995). Their work has focused on a potential biomarker for exposure to endocrine disruptors called vitellogenin (egg-yolk protein in the blood of egg-laying vertebrates). When stimulated by estrogen, the liver produces this protein and releases the protein into the bloodstream, where it then circulates to the ovaries and is deposited into an egg. Usually only females possess a sufficient amount of estrogen to produce vitellogenin; however, in a laboratory study, DDT induced vitellogenin production in male turtles and frogs. In another study, PCBs and lindane were found to induce estrogen receptor and vitellogenin accumulation in rainbow trout liver (Flouroit et al. 1995).

- In many egg-laying reptiles, the temperature of the incubating egg determines the sex of the offspring. PCBs applied to the shells of turtle eggs during the period of sexual differentiation counteracted male-producing temperatures and induced ovarian development (Bergeron et al. 1994). Further study of the mechanism of action and synergistic effects of different PCB congeners are ongoing (Crews et al. 1995).

- Researchers have recently been testing the hypothesis that endocrine disruptors, such as DDE, mercury, and PCBs, are playing a role in the decline of the endangered Florida panther population (Facemire et al. 1995). (Many have considered inbreeding the main factor up to this point.) A large percentage of males have exhibited abnormal reproductive organs, sterility, and production of abnormal or deformed sperm. Both males and females exhibit abnormal hormone ratios, with little difference in estradiol levels between males and females (i.e., evidence that males have been demasculated and feminized).

- The effluent from sewage treatment plants has been shown to induce vitellogenin synthesis is male fish (Folmar et al. 1996; Sumpter and Jobling 1995). The investigators attribute this effect to the estrogenic properties of environmental contaminants in the effluent, though specific chemicals were not identified.

In recent years, several organizations and governmental agencies, including EPA, have begun to support research efforts to further study endocrine disruptors. For example:

- The National Science and Technology Council, which advises the President and his Cabinet on directions for federal research and development efforts, has given EPA

the task of developing a national research strategy on endocrine disruptors by 1998. EPA recently conducted two workshops to plan research in this area (see sidebar).

Research Planning Workshops on Endocrine Disruptors

Purpose: To develop a national research strategy on endocrine disruptors, in response to growing public concern over their adverse effects.

Findings: The hypothesis that endocrine disruptors cause a variety of adverse effects in wildlife and humans is of sufficient concern to warrant a concerted research effort. Research priorities include: identifying and characterizing effects on developing reproductive systems; and refining exposure assessments and research on toxicology of mixtures.

Outcomes: EPA has published workshop findings and recommendations in scientific literature, begun implementing some of the identified research initiatives, and has formed an Endocrine Disruptor Research Coordination Committee.

Sources: Ankley et al. 1997; CENR 1996; Kavlok et al. 1996; U.S. EPA 1995b.

- The Chemical Industry Institute of Toxicology (CIIT) has developed a comprehensive research program to evaluate the potential for selected chemicals to affect the human endocrine system. One of the critical goals of this program is to understand the relationship between dose levels that produce an effect in laboratory cell cultures and the dose level needed for effects to be seen in laboratory animals.

- EPA's Risk Assessment Forum, which promotes scientific consensus on risk assessment issues, assembled a technical panel to study environmental endocrine disruptors. The panel has released a draft report that is intended to serve as an interim assessment and analysis of the environmental endocrine disruption hypothesis until a more extensive exploration of the issue can be completed by the National Academy of Science (U.S. EPA 1996d).

In addition to the recent focus on endocrine disruptors and their potential impact on ecological health, other research efforts relevant to potential ecological effects of the pollutants of concern include the following:

- EPA submitted a draft Mercury Study Report to the Science Advisory Board (SAB) for review in June 1996. The report was reviewed by SAB in February 1997. EPA expects to receive the opinion of SAB in the summer of 1997. The final Mercury Study Report will fulfill the requirements of CAA section 112(n)(1)(B), including a requirement to assess the environmental effects of mercury emissions.

- Dioxin has been classified as the most potent known animal carcinogen, and as a probable human carcinogen, since 1985. Increased concerns that dioxins in aquatic environments may be a major contributor to overall human dioxin exposure through fish and shellfish consumption, as well as increased evidence of its hazard to fish and wildlife, prompted EPA to reassess dioxin's effects on aquatic ecosystems. Work on characterizing ecological risks is in progress at EPA's Mid-Continent Ecology Division of the National Health and Ecological Effects Research Laboratory (NHEERL) in Duluth, Minnesota. EPA published the *Interim Report on Data and Methods for Assessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin Risks to Aquatic Life and Associated Wildlife* (U.S. EPA 1993a) on this research. The goal of the report is to review and evaluate relevant published and unpublished data and models currently available for

analyzing dioxin exposure to and effects on aquatic life and wildlife. Information on related compounds, such as TCDF and PCBs, is not discussed in detail in the interim report; however, it is expected that the final report will assess the contribution of these related compounds to the risk for aquatic life and wildlife. The interim report findings are presented under the discussion of TCDD in this section.

Based on information from the above research on ecological effects of pollutants of concern, as well as findings from recently published data, a summary of the potential ecological effects of the pollutants of concern is presented in Table II-8. For each effect attributable to a pollutant of concern, the table identifies the organism type(s) in which the effect has been observed (i.e., plants, invertebrates, fish, amphibians/reptiles, birds, and mammals). Table II-8 presents data on the following types of effects: death; cancer; reproductive/developmental, immunological, metabolic/enzyme, and neurological/ behavioral effects; damage to the kidney, liver, heart, lungs or gills, or gastrointestinal tract; exterior changes; and decreased growth or biomass. The information in this table is based on both field and laboratory studies. This report does not attempt to assess how the observed effects on individual animals contributes to changes in species populations within an ecosystem or how the laboratory studies relate to studies of effects observed in the environment. In addition, information on the exposure levels related to these effects is not provided.

Pollutants of Concern and Health Effects in St. Lawrence Estuary Beluga Whales

A 9-year epidemiological study in an isolated population of beluga whales documented higher levels of many pollutants of concern, including dieldrin, DDT, HCB, lead, mercury, PAHs, and PCBs, in these whales compared to Arctic belugas (Beland et al. 1993). The St. Lawrence whales exhibited high prevalence of tumors; high incidence of lesions to the digestive system, mammary glands, and other glandular structures; some evidence of immune system suppression; and frequent tooth loss and gum disease. No such lesions were observed in Arctic belugas or in other St. Lawrence aquatic mammals (e.g., seals). Researchers propose this case as a model for the potential long-term consequences of pollutants in the environment on human health (De Guise et al. 1995).

Following the table, recent information on the ecological effects of the pollutants of concern is discussed by pollutant group. The ecological effects of nitrogen (i.e., eutrophication and its consequences) also are discussed in this section.

MERCURY AND COMPOUNDS

The data presented in Table II-8 summarize the potential effects of mercury exposure on plants, invertebrates, fish, birds, and mammals. A brief summary of some recent research findings is presented below:

- The effects of mercury on plants may include death, growth inhibition, leaf or root damage, chlorophyll decline, and reduced photosynthesis (Godbold 1991; Lindquist 1991; Schlegel et al. 1987).

- In fish and aquatic invertebrates, mercury exposure can cause death, reduced reproductive success, impaired growth and development, and behavioral abnormalities (U.S. EPA 1985).

TABLE II-8
Potential Effects of the Pollutants of Concern on Aquatic Life and Wildlife

Pollutant of Concern	Potential Ecological Effects[a]					
	Death	Cancer	Reproductive/ Developmental[b]	Immunological	Metabolic/ Enzyme	Neurological/ Behavioral
Cadmium and compounds	I F		I F B		F B M	I B
Chlordane	I F A B M	M	I F A B M	M	I M	F B M
DDT/DDE	P I		I B A M	M		F B
Dieldrin	B		A			B
HCB	P I F B		I F			I B
α-HCH						
Lead and compounds	I F A B M		I F A B M	F A	I F B M	F A B M
Lindane	I		I	F		I
Mercury and compounds	I F A B M	M	I F A B M	B	P F B	I F B M
PCBs	P I F B M	M	I F B M	F M	F B M	F B M
POM (PAHs)	I F M	F A M	I F B M	F B M	F	I F
TCDD (dioxins)	F B M		F B M	M		B
TCDF (furans)						
Toxaphene	I F A B M	M	I F B M			A F B

[a] Blank areas indicate that no data were found correlating the effect with the pollutant of concern (as opposed to data found indicating no correlation between the effect and the pollutant of concern).
[b] Includes endocrine-disrupting effects.

Key: P = Plants I = Invertebrates F = Fish A = Amphibians/Reptiles B = Birds M = Mammals

Sources: Arkoosh et al. 1994; Baturo et al. 1995; Constable and Orr 1994; Di Pinto et al. 1993; Doust et al. 1994; Dunier and Siwicki 1994; Eisler 1985, 1986a, 1986b, 1987a, 1987b, 1988, 1990; Eisler and Jacknow 1985; Ferrando et al. 1995; Fitchko 1986; Geyer et al. 1993; Government of Canada 1994; Hermsen et al. 1994; Hill and Nelson 1992; Hugget et al. 1992; Johnson et al. 1993; Lahvis 1995; Malbouisson et al. 1994; Schulz and Liess 1995; Tidou et al. 1992; Trust et al. 1994; and U.S. EPA 1993a, 1993b, 1993c.

TABLE II-8
Potential Effects of the Pollutants of Concern on Aquatic Life and Wildlife
(continued)

Pollutant of Concern	Potential Ecological Effects[a]						
	Kidney Damage	Liver Damage	Heart Damage	Lung/Gill Damage	Gastrointestinal Damage	External Damage	Decreased Growth/Biomass
Cadmium and compounds	B		B				I F B
Chlordane	M	M	M	M	M		I M
DDT/DDE							P
Dieldrin						B	
HCB		F B					P I B
α-HCH							
Lead and compounds[b]	F B	F B		F	A M	F B	I M
Lindane							I
Mercury and compounds[b]	B M	B		M			I F B
PCBs	F M	B M			B M	F B M	P I F B
POM (PAHs)[b]	M	F M			M		F B
TCDDs	M	F M		M	M	F M	F
TCDFs							
Toxaphene	F B				F	F B	I F B

[a] Blank areas indicate that no data were found correlating the effect with the pollutant of concern (as opposed to data found indicating no correlation between the effect and the pollutant of concern).
[b] Also causes other noncancer effects in plants, such as leaf and root damage, chlorophyll decline, and reduced photosynthesis.

Key: P = Plants I = Invertebrates F = Fish A = Amphibians/Reptiles B = Birds M = Mammals

Sources: Arkoosh et al. 1994; Baturo et al. 1995; Constable and Orr 1994; Di Pinto et al. 1993; Doust et al. 1994; Dunier and Siwicki 1994; Eisler 1985, 1986a, 1986b, 1987a, 1987b, 1988, 1990; Eisler and Jacknow 1985; Ferrando et al. 1995; Fitchko 1986; Geyer et al. 1993; Government of Canada 1994; Hermsen et al. 1994; Hill and Nelson 1992; Hugget et al. 1992; Johnson et al. 1993; Lahvis 1995; Malbouisson et al. 1994; Schulz and Liess 1995; Tidou et al. 1992; Trust et al. 1994; and U.S. EPA 1993a, 1993b, 1993c.

- Effects of mercury on birds may include death, liver and kidney damage, neurobehavioral effects, impaired growth and development, and reproductive effects (Eisler 1987a; MDNR 1993; Scheuhammer 1987, 1991). Reproductive effects are the primary concern for avian mercury poisoning and may occur at dietary concentrations well below those that cause overt toxicity.

- Extensive research on the toxicity of mercury to mammals indicates that effects vary depending on the form of mercury ingested, with methylmercury being the most toxic form. Methylmercury ingestion by mammals may cause death, neurological and behavioral effects, and damage to the heart, lung, liver, kidney, and stomach (ATSDR 1992e).

Population-level Effects of Mercury

Studies conducted on various communities have shown mercury to: (1) reduce species diversity of freshwater, brackish-water, and soil microbial communities; (2) reduce carbon fixation in phytoplankton communities; and (3) change the species composition of phytoplankton in an aquatic community. Although clear causal links between mercury contamination and population declines in various wildlife species have not been established, mercury may be a contributing factor to population declines of the endangered Florida panther and the common loon. Other researchers have concluded, however, that mercury levels in most areas are not sufficient to adversely affect bird populations.

Sources: Barr 1986; Ensor et al. 1992; FPIC 1989; Roelke et al. 1991.

OTHER METALS

Cadmium. As shown in Table II-8, adverse effects from exposure to cadmium have been demonstrated in invertebrates, fish, and birds. Recent studies have focused on the toxicity of cadmium to aquatic species (Kraak et al. 1992; Weinstein et al. 1992) and the identification of potential indicator species for cadmium exposure (Naimo et al. 1992). For example, researchers concluded that accumulation of some metals contributes to lesions to the shell of blue crabs in the Albemarle-Pamlico Estuarine System; however, the researchers did not observe this result for cadmium and lead (Weinstein et al. 1992).

Lead. Many noncancer ecological effects are associated with lead exposure, as shown in Table II-8. However, the availability of recent studies on the ecological effects of lead is limited. Rather, there is a strong interest currently focused on the neurological and behavioral effects in infants and children (see Section II.D). One recent study of fish demonstrated that exposure to waterborne lead may result in multiple effects on reproductive behavior and overall reproductive success in fathead minnows (Weber 1993). A recent review of wildlife contamination in Chesapeake Bay cited older articles that found that lead adversely affected embryo development and neurological behavior in the green and bull frogs (Heinz et al. 1992).

COMBUSTION EMISSIONS

As discussed in Chapter I, this pollutant group is comprised of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD, dioxins), 2,3,7,8-tetrachlorodibenzofuran (TCDF, furans), and polycyclic organic matter (POM). Polychlorinated biphenyls (PCBs) are also discussed under this pollutant group; they were historically produced for use in electrical equipment and hydraulic fluids but are often now released into the environment in combustion gas emissions when PCB-containing materials

are incompletely burned. The potential ecological effects associated with these pollutants are discussed below.

TCDD. As shown in Table II-8, dioxins are associated with effects in fish, birds, and mammals, including reproductive effects and other noncancer effects. EPA's assessment of ecological effects in aquatic life and associated wildlife discusses the effects of TCDD in detail (U.S. EPA 1993a). A brief summary of some of the findings is presented below; please refer to this document for the primary references for these studies.

- Available laboratory toxicity information indicates that aquatic invertebrates, plants, and amphibians may be substantially less sensitive to TCDD than fish. The report emphasized that the data are limited.

- The data indicate that young fish may be more sensitive to TCDD than older fish. Fish fry were most sensitive to effects from TCDD following exposure of eggs before or shortly after fertilization.

- In mammals and birds, the primary effect of concern is reproductive impairment. In addition, because of the bioaccumulation of TCDD in aquatic food webs, the species most exposed to TCDD are those species whose diet consists mostly of fish.

Ecological Effects of TCDD in Lake Trout Population in Lake Ontario

Commercial over-fishing and sea lamprey predation are thought to be the primary causes of lake trout decline in Lake Ontario. However, several findings suggest that TCDD toxicity could have contributed to the reproductive failure of stocked lake trout in Lake Ontario in the post-1950s: (1) the strong association between the occurrence of blue-sac disease (a stress syndrome) in sac-fry from TCDD-exposed fish in laboratory studies and from eggs collected from Lake Ontario; (2) the historical record of lake trout exposure to TCDD and related chemicals in Lake Ontario; and (3) the consistency of predicted toxicity with the field sampling record of no natural reproduction until 1986. Attainment of a self-sustaining population of lake trout in Lake Ontario requires exposures to TCDD and related chemicals below the level that causes significant reproductive impairment, in addition to sea lamprey control, maintenance of adequate spawning beds and water quality, and introduction of lake trout strains appropriate for Lake Ontario's conditions and food web.

Source: U.S. EPA 1993a.

One of the main issues of current interest is the mechanism of action of dioxins (i.e., how they bring about the effects). The unusual potency of dioxins in eliciting toxic effects suggested to researchers that a dioxin receptor existed. Based on a substantial amount of biological and genetic evidence, an intracellular protein called the aryl hydrocarbon receptor (Ah receptor) is believed to mediate biological responses to TCDD and related compounds. Figure II-2 presents a simplified diagram of how biological responses to dioxin-like compounds are mediated by the Ah receptor. Although our understanding of the Ah receptor is limited, mammals (including humans), birds, and fish all have exhibited detectable concentrations of the Ah receptor in a number of different tissues. In laboratory mammal studies, binding of dioxins to the Ah receptor has been associated with weight loss, edema, liver damage, promotion of tumors, and adverse effects on the immune and reproductive systems (Fox 1993). Researchers believe that responses to TCDD are likely to vary within and between species, as well as between tissues in individual species, based on differential responses to Ah-receptor binding. Several proteins are believed to contribute to TCDD's gene regulatory effects; the response to TCDD probably involves a complex relationship between multiple genetic and environmental factors.

Recent studies of dioxins and furans that were found through the literature search conducted for this Report to Congress demonstrated reproductive effects (e.g., embryo anomalies, decreased hatching success) in nesting wood ducks in an Arkansas bayou (White and Hoffman 1995) and potential neurotoxic effects in great blue heron hatchlings, as indicated by asymmetrical brain development (Henschel et al. 1995).

FIGURE II-2
Role of Ah Receptor in Biological Responses to Dioxin Exposure

Dioxin Exposure
↓
Free Dioxin in Tissues
↓
Dioxin Binding to the Ah Receptor in Cells
↓
Ah Receptor-Dioxin Complex Binding with DNA
↓
Gene Regulation
↓
m-RNA Regulation
↓
Protein Synthesis
↓
Biochemical Alterations
↓ Interactions of
↓ Multiple Target
↓ Genes
Early Cellular Responses (cell growth stimulation)
↓
Late (irreversible) Tissue Response (cancer, deformity)

Source: Adapted from EPA 1994c.

PCBs. As shown in Table II-8, PCBs are associated with adverse effects in plants, invertebrates, fish, birds, and mammals. These effects include reproductive/ developmental, metabolic/enzyme, and neurological/behavioral effects. The specific effects and the levels at which these effects are seen may vary among the different PCB congeners and mixtures. Recent articles on the ecological effects of PCB exposure are summarized below. These studies focused on reproductive effects in invertebrates, fish, birds, and mammals, immune effects in fish and aquatic mammals, and other effects in plants and fish.

♦ *Reproductive and Developmental Effects*. A group of related studies investigated the reproductive effects of commercial PCB and combinations of PCB congeners in mink. Two of these studies found that commercial PCBs and combinations of PCB congeners adversely affected reproduction, but that exposure to single PCB congeners did not produce these same effects (Bäcklin and Bergman 1992; Kihlström et al. 1992). One of these studies showed a difference in adverse reproductive effects (e.g., late fetal death versus early fetal death) between two different commercial PCBs, Clophen A50 and Aroclor 1254 (Bäcklin and Bergman 1992). In another study, PCBs were found to reduce egg production and total reproductive capacity in a small crustacean species under laboratory conditions (DiPinto et al. 1993). A study that investigated the developmental toxicity of PCB congeners in minnows determined that several congeners caused severe teratogenic effects (e.g., inhibition of yolk absorption) and early hatching (Silberhorn et al. 1992). Recent studies of Great Lakes bird populations (e.g., common terns) provided further evidence of the developmental problems linked to PCBs during the 1980s (Becker et al. 1993; Hoffman et al. 1993; Yamashita et al. 1993). A literature review of wildlife contamination in Chesapeake Bay cited historical evidence of elevated post-hatch mortality in the leopard frog and in unspecified toad species related to PCB exposure (Heinz et al. 1992).

- *Immune System Effects*. In a recent study, production of antibodies was suppressed in juvenile chinook salmon exposed to the commercial PCB mixture, Aroclor 1254 (Arkoosh et al. 1994). In another study, delayed immune system responses in harbor seals fed herring from the Baltic Sea were associated with PCB exposure (Ross et al. 1995). In a study of dolphins, decreased immune system response was associated with elevated levels of DDT and PCBs in their blood (Lahvis et al. 1995).

- *Other Noncancer Effects*. Researchers have correlated detrimental effects on the quality and quantity of growth, as well as increased mortality, in aquatic plants with concentrations of PCBs (Doust et al. 1994). One study of winter flounder did not find a strong association between exposure to PCBs and the development of particular liver lesions (Johnson et al. 1993). Some recent studies have shown that effects from PCB exposure may be amplified in the presence of other chemicals. For example, a study in quail found that high levels of cadmium can amplify both the quantitative and qualitative retention of PCB congeners in muscle tissue, especially some of the congeners that are most toxic and resistant to metabolic degradation (Leonzio et al. 1992).

POM. The data in Table II-8 indicate that POM is associated with a range of adverse effects to aquatic and terrestrial wildlife. Studies on the adverse ecological effects from exposure to PAHs (a subset of POM) that have been completed in the last few years are summarized below. These studies examined immune effects in fish and birds as well as various other effects in algae, aquatic invertebrates, fish, and birds.

- *Immune System Effects*. In a recent study, production of antibodies was suppressed in juvenile chinook salmon exposed to PAHs (Arkoosh et al. 1994). Another study found that PAH exposure suppressed the immune system in both adult and young starlings, although only young starling showed overt signs of general toxicity (e.g., decreased body weight and blood hemoglobin concentration) (Trust et al. 1994). A study of effects on fish from PAH exposure found that macrophage activity, an important component of the cellular immune system (i.e., protects the host by eliminating foreign material), was markedly reduced (Huggett et al. 1992). The study results suggested that these effects may be reversible.

- *Other Noncancer Effects*. Recent research has investigated the increased toxicity of PAHs following exposure of the pollutants to solar radiation. One study that investigated the photo-induced toxicity of anthracene (a PAH compound) to a species of green algae showed that the combination of ultraviolet (UV-A) radiation and anthracene produced significant toxic effects. The study also concluded that algae may be slightly more resistant to photo-induced toxicity than fishes and invertebrates (Gala and Giesy 1992). Two recent studies on fish suggest a strong association between exposure to PAHs and the development of internal lesions, commonly found in the liver and kidney, as well as external lesions, such as lens cataracts (Huggett et al. 1992; Johnson et al. 1993). One of these studies found a strong correlation between sediment PAH concentration and stimulation of enzyme activity, which suggests that the PAHs are a main component of the adverse effects observed in the fish (Huggett et al. 1992). In another study, dramatic declines in PAH concentrations in sediment following the closure of a steel facility were followed by a decreased incidence of liver cancer and liver lesions in brown bullhead catfish (Baumann and Harshbarger 1995). A study of aquatic insect larvae from a

contaminated area of the Niagara River found that higher body burdens of PAHs were associated with menta ("teeth") deformities. The researchers determined that the deformities were not passed on to future generations (i.e., that PAHs caused malformations but did not permanently alter genes or chromosomes) (Dickman et al. 1992).

PESTICIDES

Information on the adverse effects of the pesticides of concern in the Great Waters (chlordane, dieldrin, α-HCH, DDT/DDE, HCB, lindane, and toxaphene) is summarized in Table II-8. These data suggest that the pesticides of concern may produce a wide range of adverse effects in terrestrial and aquatic wildlife of the Great Waters. Recent literature on the ecological effects of pesticides was available mainly for DDT/DDE, HCB, and lindane; this information, except for information on the potential for endocrine disrupting effects (which was discussed earlier in this section), is summarized below.

DDT. Much of the recent literature on the ecological effects of DDT (and DDE) focuses on reproductive impairment in birds. Other studies demonstrate effects to the immune systems of mammals and impaired growth and survival of aquatic plants following DDT exposure.

- *Reproductive and Developmental Effects*. Recent studies on natural bird colonies in the Great Lakes indicate a decreased role of DDT/DDE in the reproductive success of these populations (Weseloh et al. 1994; Weseloh and Ewins 1994; Bowerman et al. 1995). One of these studies determined that for herring gull colonies in Lake Superior, eggshells were only eight percent thinner than before the introduction of DDT (Weseloh et al. 1994). In this study, reproductive failure was attributed to causes other than toxic contamination (e.g., predation and shortage of food supply). Similarly, another study attributed increasing populations of double-crested cormorants in Lake Ontario to reduced levels of contaminants (especially DDT/DDE) and increased availability of forage fish (Weseloh and Ewins 1994). A study investigating the combined effects of DDE and decreased food intake on reproduction in ringed turtle doves found that the lower the percentage of normal food intake, the greater the effects of DDE exposure on breeding success. DDE restricted breeding success apparently by limiting levels of hormones necessary to develop and maintain active gonads, adequate courtship and brooding behavior, and functional crop glands (Keith and Mitchell 1993). A study of freshwater pond snails, which are an important component of invertebrate fauna in most eutrophic and mesotrophic lakes, found that DDT exposure reduced their reproductive output (Woin and Brönmark 1992).

- *Immune System Effects*. In a study of dolphins, decreased immune system response was associated with elevated levels of DDT and PCBs in their blood (Lahvis et al. 1995).

- *Other Noncancer Effects*. In one study in aquatic plants, exposure to DDE caused mortality and had detrimental effects on the quality and quantity of growth (Doust et al. 1994).

Hexachlorobenzene. Recent studies on ecological effects from HCB exposure demonstrate reproductive and neurological effects in aquatic plants and animals. Impaired growth and survival of aquatic species and liver damage in fish and birds have also been shown.

- *Reproductive and Developmental Effects*. Reproductive effects from HCB exposure documented in recent literature include reduced reproduction in protozoa and fathead minnows and reduced fertility in waterflea crustaceans (Constable and Orr 1994; Government of Canada 1994).

- *Neurological/Behavioral Effects*. In freshwater snails, exposure to HCB inhibited body growth, altered metabolic activity, and stimulated egg production, which researchers attributed to neurotoxic effects (Baturo et al. 1995). Kestrels, a small falcon species, have shown ruffling of feathers and tremors following exposure to HCB (Government of Canada 1994).

- *Other Noncancer Effects*. In aquatic species, HCB was lethal to some marine invertebrates and significantly reduced the survival rates of freshwater caddisfly larvae and fathead minnows (Constable and Orr 1994; Government of Canada 1994; Schulz and Liess 1995). In another study, exposure to HCB caused mortality and had detrimental effects on the quality and quantity of growth in aquatic plants (Doust et al. 1994). One report documented the following other effects in aquatic species related to HCB exposure: reduced production of chlorophyll, dry matter, carbohydrate and nitrogen in some algae; decreased growth of algae and protozoa; digestive gland damage in crayfish; and liver necrosis in largemouth bass (Government of Canada 1994). In birds, HCB exposure induced porphyria (a group of disorders related to altered metabolism of breakdown products of hemoglobin known as porphyrins), increased liver weight, and slightly damaged livers in Japanese quail and produced significant weight loss, increased liver weight, and decreased heart rate in kestrels (Government of Canada 1994).

Lindane. Recent studies show effects in aquatic species following exposure to lindane, including reproductive effects in aquatic invertebrates, immune effects in fish, and behavioral effects in aquatic invertebrates.

- *Reproductive and Developmental Effects*. One study documented reduced reproduction in waterflea crustaceans from lindane exposure (Ferrando et al. 1995).

- *Immune System Effects*. Laboratory experiments on rainbow trout found that antibody production was significantly suppressed from lindane exposure at doses comparable to those found in fish in polluted fresh waters, and that as the dose increased the more the immune system was suppressed (Dunier and Siwicki 1994; Dunier et al. 1994).

- *Neurological/Behavioral Effects*. As a result of brief exposures to high concentrations of lindane, the mating behavior of freshwater crustaceans was disrupted (Malbouisson et al. 1994). In another study, the common mussel, *Mytilus edulis*, showed lower rates of feeding-type behavior after exposure to lindane-contaminated sediments (Hermsen et al. 1994). Also, an annelid worm exposed to lindane in seawater exhibited delays in settlement and metamorphosis; this species is otherwise known to be a colonizer of disturbed or polluted areas (Hill and Nelson 1992).

- *Other Noncancer Effects*. In two different studies, survival of a waterflea crustacean species was inhibited by lindane exposure (Ferrando et al. 1995; Tidou et al. 1992); in one of these studies, lindane also significantly repressed growth in the waterflea crustaceans (Ferrando et al. 1995). In studies comparing data for several species of fish, acute toxicity of lindane decreased with increasing total body fat in fish (Geyer et al. 1993, 1994).

NITROGEN COMPOUNDS

The availability of biologically-usable nitrogen normally limits biological productivity in coastal waters, but an over-abundance of nitrogen is a concern in areas where nutrient enrichment problems, known as eutrophication, have developed. In addition to increasing productivity, nutrient enrichment generally alters the normal ratios of nitrogen to phosphorus and to other elements, such as silicon. This alteration may induce changes to phytoplankton community structure. Species that normally occur in low abundance may be favored, and in some cases, toxic and/or noxious algal blooms may result. On the New England coast, for example, the number of red and brown tides and shellfish problems from nuisance and toxic plankton blooms have increased over the past two decades. In coastal areas with poor or stratified circulation patterns (e.g., Chesapeake Bay, Long Island Sound), the "overproduced" algae tends to sink to the bottom and decay, using all (anoxia) or most (hypoxia) of the available oxygen in the process, killing or driving away organisms that require oxygen. In addition, the increase in suspended matter due to overproduction of algae decreases light penetration, causing a loss of underwater seagrass and coral communities.

Atmospheric deposition of nitrogen compounds is recognized by all U.S. estuarine programs on the east coast as either a significant contributor to estuarine eutrophication or a mechanism of possible concern (ECARA 1996). An important consideration for controlling atmospheric deposition of nitrogen is that the region from which the atmospheric nitrogen pollution arises (i.e., the "airshed") is larger than the watershed that drains into the waterbody and much larger than the water surface that is potentially affected. The extent of "airsheds" is now starting to be recognized (e.g., see the discussion of the Chesapeake Bay airshed in Section C of Chapter IV).

II.D Human Health Effects

This section is intended to provide a brief overview of the current literature on the potential human health effects from exposure to the Great Waters pollutants of concern. In general, the information presented in this section represents data published since the background document from the First Report to Congress on exposure and effects (Swain et al. 1992a) was completed through 1995. Because the information presented in this report covers only recent studies, it cannot be used alone to determine whether these effects are widespread in the environment. As in the First Report to Congress, the contribution of atmospheric deposition to toxic contamination and potential human health effects associated with exposure to the pollutants cannot be quantified at this time.

Much of the information presented in this section on human health effects is based on effects observed in animals, mainly in laboratory animal studies, that are suggestive of potential human health effects. Unlike the effects of certain pollutants of concern on ecological health in the Great Waters (discussed in Section II.C), data on adverse health effects observed in humans from non-occupational exposure to the pollutants of concern are limited. This section focuses on the effects of the pollutants of concern and does not attempt to summarize the exposure levels responsible for the observed effects. In addition, this section does not attempt to distinguish effects that may occur with long-term exposure to the pollutants of concern from those effects caused by acute, high-level exposures (e.g., accidental spills). The potential effects of a pollutant may vary with duration of exposure, possibly due to a breakdown of the chemical in the body to another chemical that is more toxic or affects other target organs than the exposed chemical.

Conclusions from the First Report to Congress

Information from the First Report to Congress is presented here to provide a foundation for the subsequent discussion of the recent information available for this report. The research findings and studies presented in the First Report to Congress, as well as the background document on exposure and effects (Swain et al. 1992a), led to the following conclusions regarding the potential human health effects from exposure to the pollutants of concern:

- Numerous studies indicated potential human health effects associated with the pollutants of concern (see sidebar).

- Though many of the pollutants of concern are probable carcinogens, the noncancer effects of these pollutants are also a significant concern and may be as detrimental as cancer, or more so, to individuals and populations.

- It is possible for low-level exposure to several of the pollutants to have little or no measurable effect on an adult, yet alter the formation and function of critical physiological systems and organs in

Potential Human Health Effects Caused by Pollutants of Concern

- Cancer
- Reproductive effects
- Developmental effects, including effects on embryos, fetuses, and children
- Neurological (i.e., brain and nervous system) effects
- Immune system effects
- Endocrine system effects, including effects on hormone synthesis and function
- Other noncancer effects, including liver and kidney damage

children of the exposed adult, especially when the child is exposed at critical developmental stages in the mother's womb.

- Some human health effects caused by the pollutants of concern are subtle, result from long-term exposures to low levels of pollutants, and may be delayed in onset and occur across several generations. For example, long-term exposure to low levels of mercury may result in kidney or nervous system damage only after gradual exposure and bioaccumulation in the body.

The remainder of this section presents updated information on human health effects associated with the Great Waters pollutants of concern.

Current Understanding of Human Health Effects

Since the First Report to Congress, updated information on the potential human health effects from exposure to the pollutants of concern has become available. This section summarizes the information first by discussing some notable research efforts relevant to the potential human health effects of some Great Waters pollutants of concern, followed by a brief overview of recent data on the potential human health effects for each pollutant of concern.

As discussed in Section II.C, the role of endocrine disruptors in causing adverse effects in wildlife and humans is an emerging and controversial issue. Effects of endocrine disruptors have been seen mostly in wildlife and laboratory experiments; however, there are a few known cases of accidental exposure of humans to high concentrations of endocrine disruptors (see sidebar for an example). Humans are especially susceptible to adverse effects when exposure to endocrine disruptors occurs during periods that are tightly controlled by hormonal activity, such as during embryo and fetal development (Bern 1992). As an example, the chemical hormone diethylstilbestrol (DES) was given to millions of women to prevent miscarriages between the 1940s and 1970s. Though the women were largely unaffected by their exposure to DES, their children exhibited adverse reproductive effects such as decreased fertility in both sexes, testicular cancer in males, and abnormal pregnancies in females (Colborn et al. 1993; Hileman 1994). Scientists use the DES incident as a model of how endocrine disruptors may affect humans.

Human Exposure to Endocrine Disruptors

Accidental exposure to cooking oil contaminated with PCBs occurred in Japan in 1968 and Taiwan in 1979, affecting 3,000 to 5,000 people overall. Children born to women who consumed the oil when pregnant exhibited birth defects and developmental and behavioral deficiencies that scientists believe may be related to hormonal changes caused by exposure (Lai et al. 1993; Rogan and Gladen 1992). Upon reexamination of the Taiwanese children in the early 1990s, researchers found that the children scored lower than controls on IQ tests, girls were shorter than average, and boys' penises were significantly smaller than normal (Chen and Hsu 1994; Guo et al. 1993).

Some researchers have cautioned that because of the complex interactions involved, proving a cause and effect link between adverse effects in humans and endocrine disruptors will be difficult. Some raise doubts about the severity of the effects of endocrine disruptors shown in some of the scientific literature (Safe 1995). The investigators note that these chemicals are only weak hormones; natural estrogens have been shown to be over a thousand times stronger than

estrogenic pesticides (Birnbaum 1994). In addition, some researchers theorize that the net effect of natural and chemical endocrine disruptors may be zero, especially given that some environmental estrogens are found in plants and have been shown to have beneficial effects, such as inhibiting tumor formation (Safe 1995). The significance of endocrine-disrupting effects in humans remains unclear.

Recent work on mixtures of endocrine disruptors raises possible answers to some of these arguments. Researchers found that, in a simple yeast estrogen system, combinations of two weak endocrine disruptors, such as dieldrin and toxaphene, were 10 to 1,600 times more potent than individual compounds in eliciting binding and activation of the human estrogen receptor (Arnold et al. 1996). This research is preliminary, but has important implications, especially because pollutants in the environment are typically found in mixtures.

In addition to the recent focus on endocrine disruptors, other research efforts relevant to the potential human health effects of the pollutants of concern include the following:

- EPA submitted a draft Mercury Study Report to the Science Advisory Board (SAB) for review in June 1996. The report was reviewed by SAB in February 1997. EPA expects to receive the opinion of SAB in the summer of 1997. The final Mercury Study Report will fulfill the requirements of CAA section 112(n)(1)(B), including a requirement to assess the public health impacts of mercury emissions.

- In response to growing scientific controversy regarding the potential effects from exposure to dioxins and related compounds, EPA began a scientific reassessment of the health risks from exposure to TCDD and chemically-related compounds (known collectively as dioxins and including TCDFs) in April 1991. Activities under the dioxin reassessment included updating and revising the health assessment and exposure assessment documents and performing research to characterize ecological risks in aquatic ecosystems. The dioxin reassessment document was published in draft form in June 1994 and is found in two reports (each 3 volumes): the health assessment document (U.S. EPA 1994c) and the exposure document (U.S. EPA 1994d). More detailed information on the human health effects related to TCDDs and TCDFs are discussed later in this section.

Based on the above research areas of interest plus human health effects data from the First Report to Congress and from recently published studies, Table II-9 presents the potential human health effects associated with the Great Waters pollutants of concern, except nitrogen. Nitrogen compounds are not included in this table because nitrogen compounds that are atmospherically deposited generally are not a direct hazard to human health. (Nitrates in drinking water from wells are of concern in many areas, but are not linked to atmospheric deposition.) For balance in understanding the importance of nitrogen oxides and other nitrogen emissions to the atmosphere, effects on human health due to ozone formation resulting from nitrogen compounds are briefly discussed later in this section. The data in Table II-9 generally are based on a compilation of results from laboratory studies on whole animals, toxicity/cellular studies in "test tubes" (i.e., in vitro studies), and human epidemiological studies describing occupational or accidental exposure to high concentrations of chemicals. Although these studies identify serious potential effects for humans, it is difficult to determine the adverse effects that would actually occur with chronic and low-level exposure to these pollutants in the environment. Since the First Report to Congress, Table II-9 has been updated to recognize the

TABLE II-9
Potential Human Health Effects Associated With Pollutants of Concern [a]

Pollutant	Potential Effects on Human Health [b]					
	Cancer [c]	Reproductive/ Developmental	Neurological/ Behavioral	Immunological	Endocrine	Other Noncancer [d]
Cadmium and compounds	Probable	●	●	●		Respiratory and kidney toxicity
Chlordane	Probable	●	●	●	●	Liver toxicity
DDT/DDE	Probable	●	●	●	●	Liver toxicity
Dieldrin	Probable	●	●	●	●	Liver toxicity
Hexachloro-benzene	Probable	●	●	●	●	Liver toxicity
α-HCH	Probable					Kidney and liver toxicity
Lead and compounds	Probable	●	●	●	●	Kidney toxicity
Lindane	Probable [f]	●	●	●	●	Kidney and liver toxicity
Mercury and compounds	Possible [g]	●	●	●	●	Kidney toxicity
PCBs	Probable	●	●	●	●	Liver toxicity
Polycyclic organic matter [e]	Probable	●		●	●	Blood cell toxicity
TCDF (furans)	Not classifiable	●		●	●	Liver toxicity
TCDD (dioxins)	Probable [f]	●	●	●	●	Chloracne
Toxaphene	Probable	●	●	●	●	Cardiovascular effects; liver toxicity

[a] Sources: Barnea and Shurtz-Swirski 1992; Cassidy et al. 1994; Chowdhury et al. 1993; Clayton and Clayton 1994; Colborn et al. 1993; Howard 1991; Linder et al. 1992; Soto et al. 1993; ATSDR Toxicological Profiles (see list in References chapter); and U.S. EPA 1987a, 1987b, 1988, 1989, 1990, 1991a, 1993d, and 1994c.
[b] For purposes of this table, a pollutant was considered to induce an effect if human or laboratory mammal data indicating a positive result were available. Blanks mean that no data indicating a positive result were found in the references cited (not necessarily that the chemical does not cause the effect).
[c] Cancer classifications: (1) "probable human carcinogen" when there is limited or no evidence of human carcinogenicity from epidemiological studies but sufficient evidence of carcinogenicity in animals (corresponds to EPA weight-of-evidence category B); (2) "possible human carcinogen" when there is limited evidence of carcinogenicity in animals and inadequate or lack of human data (corresponds to EPA weight-of-evidence category C); and (3) "not classifiable as to human carcinogenicity" when there is inadequate human and animal evidence of carcinogenicity or when no data are available (corresponds to EPA weight-of-evidence category D). Data on cancer classifications are obtained from EPA's Integrated Risk Information System (IRIS), unless otherwise noted.
[d] This column reports only a sample of other noncancer effects that may occur as a result of chronic exposure to the pollutant. Additional adverse human health effects may be associated with each chemical.
[e] POM represents a class of numerous compounds; not every compound is responsible for the potential effects on human health.
[f] Data from Health Effects Assessment Summary Tables (HEAST), which classify these chemicals as probable human carcinogens; however, these carcinogenic evaluations are currently under review by EPA.
[g] Inorganic (mercuric chloride) and organic (methylmercury) forms are classified as "possible," whereas elemental is "not classifiable."

potential endocrine-disrupting effects of DDT/DDE and lindane, and the "possible human carcinogen" classification for mercury (organic and inorganic forms).

The current information on the human health effects associated with each of the pollutant groups is highlighted in the remainder of the section.

MERCURY AND COMPOUNDS

Mercury, a metal, is discussed separately from other metals because of the significant emphasis placed on mercury in section 112 of the CAA and the difference in its behavior and effects compared to other metals. It has long been known that organic mercury (methylmercury) bioaccumulates in fish, and can biomagnify in the food web. In the air, mercury exists primarily as elemental (Hg^0) and inorganic (Hg[II]) mercury. Most of the mercury in water, soil, or sediments occur in the form of elemental mercury and inorganic mercury salts.

Humans are most likely exposed to mercury indirectly as methylmercury through a diet containing contaminated fish, instead of directly from inhalation of mercury in air. Ingestion of mercury-contaminated fish can result in various health effects, particularly toxicity to the nervous system in adults and in children exposed as fetuses. As shown in Table II-9, mercury may also affect the reproductive and immune systems. Since the First Report to Congress, EPA has recommended the current rating of the scientific weight-of-evidence regarding the human carcinogenicity of mercury (U.S. EPA 1993d):

- Elemental mercury: not classifiable as to human carcinogenicity;
- Inorganic (mercuric chloride) and organic (methylmercury) mercury: possible human carcinogens.

In recent years, researchers, including EPA, have focused on several issues related to the health effects of methylmercury: (1) improving the quantitative estimate of the relationship between mercury levels in air and exposure levels; (2) using refined statistical approaches and the application of physiologically-based pharmacokinetic models to evaluate the critical dose levels at which health effects occur from mercury; and (3) effects on nervous system development in populations that consume mercury-contaminated fish. New data are available from a recently-published study investigating neurological effects in children belonging to a fish-consuming population in the Seychelles Islands. Data from evaluation of these children (up to six years of age) have been collected and are being analyzed. Data from a similar study in the Faroe Islands have been published in abstract form; however, further investigation is being conducted to determine if the study subjects also may have been exposed to PCBs. Smaller scale studies evaluating effects in populations around the Great Lakes also are in progress. The above data and methodologies have not yet been incorporated into an EPA risk assessment of methylmercury because a majority of the new data are either not yet published or have not yet been subjected to rigorous review.

OTHER METALS

Cadmium. As indicated in Table II-9, cadmium has been linked to numerous adverse human health effects, including respiratory and kidney toxicity, probable carcinogenicity,

reproductive and developmental effects, and immunological effects. Recent literature discussing the adverse human health effects of exposure to cadmium is limited and generally is focused on how cadmium alters the function of the kidney, which is known to be the critical organ for cadmium exposures (WHO 1992). Cadmium exposure has been found to cause deficient vitamin D metabolism in the kidney, which subsequently affects the calcium balance and bone density and may result in osteoporosis or osteomalacia (both bone diseases characterized by a change in the mineral and matrix phases of bone tissue). Exposure to cadmium has also been linked to developmental effects (e.g., particularly low birth weight).

Lead. Recent literature on human health effects from lead exposure generally supports the findings presented in II-9: lead is considered a probable carcinogen and may affect reproduction and development, alter the immune, nervous, and endocrine systems, and damage the kidney. While the correlation between high levels of lead in blood and adverse human health effects is relatively well known, current research has focused on the adverse effects associated with low levels of lead in blood (<30 micrograms of lead per deciliter of blood, or μg/dL) (see sidebar). Low levels of lead may be found in blood of a significant portion of the general public (U.S. EPA 1991b). Effects from low level exposure range from subtle cellular changes, such as effects on red blood cell metabolism, to pronounced effects on physical and mental development (Hovinga et al. 1993; Huseman et al. 1992; Kim et al. 1995). Blood lead levels as low as 10 μg/dL (and possibly lower) may result in adverse human health effects (Mushak et al. 1989; U.S. EPA 1991b). EPA has also noted that infants and young children may be most susceptible to adverse effects resulting from lead exposures (U.S. EPA 1991b).

Low-Level Lead Exposure

Because of the adverse human health effects associated with low-level lead exposure coupled with the numerous exposure pathways for lead, EPA has abandoned its standard approach for evaluating chemical toxicity in favor of a site-specific modeling approach. In 1994, EPA released a revised version of the *Integrated Exposure Uptake Biokinetic Model for Lead in Children* as its recommended methodology for assessing lead exposure. The model considers the principal lead exposure pathways (e.g., drinking water, diet, inhalation) to estimate lead levels in blood of children. Overall lead intake is then integrated in the model to estimate blood lead concentration. This concentration can then be used to predict: (1) the percentage of the exposed population that will have blood lead levels greater than 10 µg/dL; or (2) the probability that a child exposed to this level will have a blood lead level greater than 10 µg/dL. EPA defines 10 µg/dL blood lead as the lower bound of the range that is known to cause adverse effects on behavior in young children.

Source: U.S. EPA 1994b.

COMBUSTION EMISSIONS

TCDD and TCDF. As shown in Table II-9, the potential human health effects from exposure to TCDD include probable carcinogenicity, and reproductive and developmental, neurological and behavioral, immunological, and endocrine system effects. For TCDF, the potential human health effects are reproductive and developmental, immunological, and endocrine system effects, and liver toxicity. The dioxin reassessment document (U.S. EPA 1994c) discusses the effects of TCDD and related (compounds collectively referred to as dioxins) in detail. A summary of some of the research presented in the reassessment is discussed below; please refer to the reassessment document for the primary literature references for these studies.

- *Carcinogenicity*. A number of new studies provide further evidence that dioxins are probable human carcinogens. Because available human studies alone cannot confirm whether there is a causal relationship between dioxin exposure and increased cancer incidence, assessors have extrapolated from available animal data to potential human cancer. Recent animal studies have demonstrated dioxins to be carcinogenic in hamsters and small fish. All of these data have contributed to the weight-of-evidence that dioxins and related compounds may be carcinogenic, under certain circumstances, in humans.

- *Reproductive and Developmental Effects*. The potential for dioxins and related compounds to cause adverse reproductive and developmental effects in animals has been recognized for many years. Recent laboratory studies have suggested that altered development may be among the most sensitive TCDD endpoint in laboratory animals, although the likelihood and level of response in humans is much less clear. One study of monkeys found that chronic exposure to TCDD increased the risk of endometriosis, a female reproductive system disorder that can be painful and produce infertility. Human epidemiologic studies on the relationship between dioxin exposure and endometriosis are planned as part of follow-up research to a 1976 dioxin release in Seveso, Italy.

- *Immune System Effects*. Recent animal studies confirm that dioxins may cause immunological effects and suggest that some effects may occur after low-level exposure. There is conflicting evidence, however, concerning the effects of these compounds on humans. A developing human immune system is believed to be particularly sensitive to the effects of exposure to dioxins compared to adults, but additional research is necessary to confirm this hypothesis.

- *Endocrine System Effects*. Two human epidemiologic studies linked exposure to TCDD with changes in male reproductive hormone levels. Two of three studies found decreased testosterone levels and one of two studies observed an increase in a female ovulation hormone in males. Animal studies have produced similar results.

- *Other Noncancer Effects*. Chloracne is a severe acne-like disorder that develops within humans after a few months of exposure to dioxin. It may disappear in some individuals after exposure is discontinued, or persist in others for many years. Limited data exist to determine the doses at which chloracne is likely to occur, but long lasting, high-intensity exposures that begin at an early age are believed to increase an individual's chances of contracting this skin disorder. Another potential effect is enzyme induction, which has occurred in animals exposed to TCDD compounds. The biochemical alteration may either benefit the animal or result in adverse effects to the animal (i.e., alter metabolism of certain chemicals by increasing or decreasing toxicity). Studies linking this effect directly to humans are not available.

Most dioxins are thought to affect animals and humans by binding with the Ah receptor. The Ah receptor, which has been detected in humans, and its mechanism of action are discussed in Section II.C.

PCBs. This section summarizes the potential human health effects that are generally common to all PCB compounds, although the levels at which these effects may occur vary between compounds. In addition, specific compounds may produce their own range of effects.

As shown in Table II-9, PCBs are classified as probable carcinogens, and potentially affect reproductive, neurological, immunological, and endocrine processes in the human body. Recent research suggests that PCBs may be able to act as endocrine disruptors in ways similar to many pesticides and dioxins (McKinney and Waller 1994).

PCBs remain a significant concern because, although they have not been produced in this country for over a decade, they are often released into the environment in combustion gas emissions when PCB-containing materials are incompletely burned. In addition, appreciable concentrations are still found in water, and animal and human tissue, milk, and blood. As reported in Section II.A, the majority of fish advisories in the Great Waters are due to the presence of PCBs.

One recent study comparing serum PCB levels in people who ate Great Lakes fish (i.e., consumers) to a control population found that serum PCB levels did not change substantially over a seven-year period (Hovinga et al. 1992), as shown in Table II-10. The study proposes a number of reasons for the static PCB levels in the fish-consuming population: (1) restrictions on PCB production alone may not ensure decreasing levels of PCB exposure in human populations; (2) other sources of PCB contamination may be major sources of exposure; or (3) seven years may not be a long enough time to see a decrease in body burdens of PCBs, due to the persistent nature of these compounds, in human populations.

Changes in Serum PCB Levels in Great Lakes Populations

Hovinga et al. (1992) compared data from a 1982 study that examined 115 individuals who consumed Great Lakes fish and 95 who did not eat fish (i.e., controls) to a similar study in 1989 that reevaluated these individuals. Comparison of the data from the two studies (Table II-10) shows a significant decrease in mean serum DDT levels in both fish-eaters (almost 40 percent) and controls (approximately 30 percent). In contrast, serum PCB levels decreased only slightly in fish-eaters and remained relatively constant in controls. These results support other studies that have found stable PCB levels over time when DDT levels have been decreasing and suggest that, despite their ban, restrictions on PCBs have not been as effective in reducing levels of contamination in humans as those placed on DDT.

TABLE II-10
Mean Serum PCB and DDT Levels in Fish Eaters and Controls (1982 vs. 1989) [a]

	Fish Eaters		Controls	
	1982	1989	1982	1989
Serum PCB (ppb)	20.5	19.0	6.6	6.8
Serum DDT (ppb)	25.8	15.6	9.6	6.8

[a] Values represent 111 fish eaters and 90 controls (out of original 115 and 95, respectively).

Potential sources of exposure to PCBs other than fish consumption are disposal of previously manufactured products containing PCBs; atmospheric transport from other countries where PCBs are still produced and used; and cycling of PCBs in the environment. The

widespread use of PCBs in electrical equipment is of concern, but there has been an increased awareness of the combustion of waste containing PCBs. The continued detection of PCB concentrations in the environment suggests that there is a reservoir of PCBs from "old" pollution that cycles between sediments, water, and air.

In view of this evidence of continuing persistence of PCBs in the environment and in the human population, effects on humans related to this group of chemicals remain a concern and many studies assessing their effects are currently underway. A 1992 workshop sponsored by EPA documented several human health effects from exposure to PCBs (see sidebar). Other recent findings include:

Workshop on Developmental Neurotoxic Effects Associated With PCB Exposure

In September 1992, EPA sponsored a workshop on the developmental nervous system effects of PCBs. At this meeting, experts addressed whether currently available health effects data are sufficient to support developmental neurotoxicity data in risk assessment. Some current findings regarding human health effects associated with PCBs were discussed and are summarized below.

- Women exposed to PCBs through contaminated cooking oil experienced irregularities in menstrual cycles, which may suggest alteration in ovarian function.
- Potential nondevelopmental nervous system effects include headaches, numbness, altered peripheral nerve function, and decreased neurobehavioral function measured through visual memory and problem solving ability. In animals, PCB exposure has been shown to affect the actions of dopamine (a brain neurotransmitter).
- PCB exposure has been associated with atrophy of the thymus and immunosuppression in animals (i.e., inhibition of immune cells required for tumor resistance).
- Chloracne and liver dysfunction are associated with occupational exposure to PCBs in humans. In animal studies, increased mortality, skin ailments, hepatotoxicity, and weight loss have been demonstrated.

Source: U.S. EPA 1993b.

- *Carcinogenicity*. In response to evidence that the development of cancer may involve miscommunication between cells, a recent study investigated the effect of PCB exposure on intercellular communication in human breast cells and found that cell communication was inhibited with increasing doses (Kang et al. 1996).

- *Reproductive and Developmental Effects*. In female rat pups given doses of PCBs, one study observed delayed puberty, fertility impairment, and irregular estrus cycle patterns (Sager and Girard 1994). The investigators suggested that these reproductive effects may be related to impairment of endocrine function. One study found that consumption of PCB-contaminated sport fish from Lake Ontario did not increase the risk for spontaneous fetal death in humans, which has been observed in various mammalian species after PCB exposure (Mendola et al. 1995).

- *Neurological/Behavioral Effects*. The effects of prenatal exposure to PCBs on neurological function in children were investigated in a follow-up study of children from the contaminated rice oil incident in Taiwan (introduced in the discussion of endocrine disruptors earlier in this section). Researchers concluded that exposure of the fetus to PCBs may impair the psychological functions of the brain in the child and that PCBs may persist in the brain for a long period (Chen and Hsu 1994).

- *Other Noncancer Effects*. Prenatal exposure to PCBs was associated with lowered intellectual function in school-age children (Jacobsen and Jacobsen 1996). The most highly exposed children were three times as likely to have low average IQ scores and twice as likely to be at least two years behind in reading comprehension. Effects were associated only with prenatal exposure, even though larger quantities of PCBs are transferred by breast-feeding than across the placenta.

As discussed in the recently published health effects reassessment for dioxin, some PCBs have been found to produce similar effects as dioxins.

POM. As shown in Table II-9, adverse human health effects associated with POM include reproductive and immunological effects, as well as probable carcinogenicity. Health effects data on POM are available primarily for polycyclic aromatic hydrocarbons (PAHs), one group of POM chemicals. Recent literature on health effects of PAHs corroborates previous findings and investigates the mechanism of action for PAH effects.

- *Carcinogenicity*. There is some speculation that ultraviolet light acts as a cofactor in the development of PAH-associated skin cancer, although this hypothesis requires further evaluation (Saboori and Newcombe 1992).

- *Reproductive and Developmental Effects*. It has been demonstrated in human placenta samples that short-term exposure to two PAHs (benzo[a]pyrene and 3-methylcholanthrene) increases the secretion of human chorionic gonadotrophin (HCG) (an important hormone in the human placenta) in the first trimester. This effect was maintained after application of the PAHs was discontinued. The secretion of this hormone may be involved in adverse reproductive effects observed with these pollutants (Barnea and Shurtz-Swirski 1992).

- *Immune System Effects*. Exposure of experimental animals in the womb to PAHs was found to alter development of the immune system and cause severe and sustained postnatal immunosuppression (i.e., inhibition of immune cells necessary for tumor resistance) (Holladay and Luster 1994).

PESTICIDES

The pesticides of concern for the Great Waters are chlordane, DDT/DDE, dieldrin, hexachlorobenzene, α-HCH, lindane, and toxaphene. As shown in Table II-9, these pesticides are probable human carcinogens and potentially result in toxic effects to reproductive, immune, and endocrine systems, as well as other noncancer effects.

Much of the recent literature concerning adverse health effects from exposure to the pesticides of concern in the Great Waters discusses their estrogenic and other hormonal effects (see earlier discussion of endocrine disruptors), as well as mechanisms of action of these effects (Chowdhury et al. 1993; Foster et al. 1992a, 1992b; Johnson et al. 1992; Juberg and Loch-Caruso 1992; McNutt and Harris 1993). Current research that has further explored the mechanisms of action of the pesticides in producing their known effects, other than endocrine disruption, are summarized below.

- *Carcinogenicity*. In response to evidence that the development of cancer may involve miscommunication between cells, a few recent studies have investigated the effects and

mechanism of action of cancer-promoting chemicals, including dieldrin, DDT, lindane, and toxaphene, on intercellular communication (Kang et al. 1996; Leibold and Schwarz 1993; Rivedal et al. 1994; Tateno et al. 1994). They have found that cell communication is inhibited with increasing doses of these chemicals.

- *Reproductive and Developmental Effects*. In addition to studies of reproductive effects associated with endocrine disruption, recent studies discuss the effects and accumulation of the pesticides in reproductive organs (e.g., ovary) (Bourque et al. 1994; Lindenau et al. 1994; Singh et al. 1992). One recent study concluded that DDT is not toxic to sperm (Linder et al. 1992).

- *Neurological/Behavioral Effects*. Studies on the adverse effects from exposure to neurotoxic pesticides provided further support of their adverse neurological effects, investigated the mechanisms of action producing these effects, the specific regions of the central nervous system affected (e.g., the motor primary cortex and hippocampus in the brain), and whether there is a critical exposure period for these effects (Barrón et al. 1993; Eriksson et al. 1992, 1993; Gilbert and Mack 1995; Goldey and Taylor 1992; Gopal et al. 1992; Johannson et al. 1993; Kilburn and Thorton 1995; Nagata et al. 1994; Rivera et al. 1992). Many of these studies evaluated nervous system effects during development in young animals, and determined that progression of the effects, as well as severity of these effects, is influenced by the time of exposure (e.g., formation of the nervous system is a sensitive stage of development). Data also suggest that some effects of early exposure to pesticides on certain regions of the central nervous system may be reversible.

- *Immune System Effects*. Studies investigating the mechanisms of action for producing immunological effects and the effects on specific organs in the immune system (e.g., spleen) have confirmed the potential adverse immunological effects related to these pesticides. Immune system effects (e.g., suppression, autoimmunity) were observed in humans with elevated levels of chlordane (McConnachie and Zahalsky 1992). Animal data have demonstrated that immune system stimulation (i.e., increased antibodies) and/or suppression (e.g., decreased production of certain antibody-forming cells) may result with exposure to DDT, dieldrin, HCB, and lindane (Flipo et al. 1992; Meera et al. 1992; Rehana and Rao 1992; Saboori and Newcombe 1992; Saha and Banerjee 1993; Schielen et al. 1993).

- *Other Noncancer Effects*. A review of literature on human health effects from long-term exposure to pesticides concluded that (1) chlordane may cause disorders in bone marrow; (2) DDT can cause chloracne, chromosome aberrations, tremors, muscular weakness, and high levels of cholesterol and triglyceride; and (3) hexachlorobenzene may cause metabolic abnormalities and liver cancer (Maroni and Fait 1993). Another study demonstrated that lindane may impair development of blood cells in the bone marrow (Parent-Massin and Thouvenot 1993).

NITROGEN COMPOUNDS

Nitrogen in the molecular form N_2 is the most abundant gas in the earth's atmosphere, and is essentially chemically inert under ambient environmental conditions. Other compounds of nitrogen, particularly oxides of nitrogen, are common in the air due to their formation mainly through coal and oil fossil-fueled electric power generation and automobile fossil fuel

combustion, and produce a variety of effects on human health and the environment. EPA has several programs, in addition to the Great Waters program, that are evaluating and attempting to reduce the threat to human health from atmospheric nitrogen dioxide, ozone, and acidic precipitation or particulates (U.S. EPA 1995c, 1995d, and 1995e). These other programs also examine general environmental effects of the nitrogen compounds, while the Great Waters program concentrates on the effects from nutrient enrichment, or eutrophication, in waterbodies from atmospheric deposition of nitrogen compounds (see Section II.C on ecological effects).

Oxides of nitrogen are produced abundantly by many modern combustion processes. Of the oxides of nitrogen, nitrogen dioxide (NO_2) is the most abundant in emission plumes or vehicle exhaust. At times, NO_2 itself can reach ambient concentrations associated with a variety of acute and chronic health effects. A more common problem is that, in the presence of sunlight, nitrogen oxides in the atmosphere react with volatile organic compounds (VOCs) to form ozone. Although ozone in the stratosphere is essential for protecting the earth from harsh ultraviolet rays, lower level ozone (or photochemical smog) contributes to a variety of health problems. Additional problems are posed by fine particles in the atmosphere. Under certain conditions in the air, oxides of nitrogen can undergo additional reactions resulting in fine particles or contribute to acids in aerosol droplets. EPA has set health-based National Ambient Air Quality Standards (NAAQS) for NO_2, for ozone, and for fine particulates (which have many sources in addition to nitrogen oxides). Acidic precipitation is also affected by nitrogen oxide emissions, and is the focus of another EPA program. Recent publications from those programs and NAAQS reviews should be consulted for details on the human health effects of nitrogen. The Great Waters program evaluates the reductions in nitrogen compounds that these programs have achieved and have proposed, and considers the net effects on waterbodies, but does not analyze direct human health effects from inhalation.

II.E Other Effects

In addition to adverse effects on human and ecological health, atmospheric deposition of the pollutants of concern may contribute to other adverse effects including environmental justice concerns (e.g., effects on subsistence fishermen), commercial and recreational fishing losses, and other recreational losses. This section provides a very brief overview of these other potential effects. This section does not attempt to establish a link between these effects and atmospheric deposition of the pollutants of concern to the Great Waters.

Environmental Justice Concerns

As introduced in the First Report to Congress, an important environmental justice issue in the Great Waters is whether certain groups of people may have higher exposures to the pollutants of concern than the general population, and therefore potentially greater risks for adverse health effects. One potential effect, cancer, is discussed in the box below (note that noncancer effects may be as or more important).

Subpopulations with Higher Lifetime Cancer Risks

In developing the Great Lakes Water Quality Guidance (GLWQG), EPA estimated baseline human health (cancer and noncancer) risks for certain subpopulations in each of the Great Lakes. Baseline health risks were based on fish tissue concentrations of chlordane, DDT, dieldrin, hexachlorobenzene, mercury, PCBs, 2,3,7,8-TCDD, and toxaphene. Table II-11 presents the low and high estimated lifetime cancer risks, as well as EPA's "acceptable" range of lifetime cancer risk for human health. As shown in the table, the risks to the subpopulations are well above the accepted risk range.

TABLE II-11
Lifetime Cancer Risks in Various Great Lakes Subpopulations Versus EPA's Appropriate Range of Risk to Human Health [a]

Subpopulations	Low	High
Native Americans (subsistence anglers)	18 in 10,000	370 in 10,000
Low income minority sport anglers	25 in 10,000	120 in 10,000
Other sport anglers	9.7 in 10,000	450 in 10,000
EPA's Acceptable Range of Lifetime Cancer Risk for General Population[b]	0.01 in 10,000 to 1 in 10,000	

[a] Cancer risks were driven by fish tissue PCB concentrations which were lowest in Lake Superior and highest in Lake Michigan. Therefore, low end of range represents Lake Superior and high end represents Lake Michigan.
[b] EPA's acceptable range of lifetime cancer risk is based on the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) [40 CFR Part 300].

Sources: U.S. EPA 1995a, 1995h.

Some populations are at higher risk because they may be more vulnerable to effects (e.g., children). Other populations may be more highly exposed because they consume larger quantities of Great Waters fish than the general population (e.g., for subsistence reasons); these populations include Native Americans, the urban poor, and sport anglers. For example, researchers found a relationship between fish consumption levels and socioeconomic characteristics in a study of Michigan licensed sport anglers. A combination of minority status and relatively low annual income (less than $25,000) was correlated with higher levels of fish consumption (West et al. 1993). Higher fish consumption rates have been correlated with higher levels of contaminants in the blood:

- High levels of dioxins and furans were found in frequent consumers of fish living near the Baltic Sea in Sweden. Individuals with the highest percentage of fish in their diet, specifically fishermen and fishing industry workers, had blood levels that were approximately three times that of non-fish consumers (Svensson et al. 1991).

- On the north shore of the Gulf of the St. Lawrence River, fishermen who consumed large amounts of seafood had elevated levels of PCBs in the blood. The blood levels among the highly exposed fisherman were 20 times higher than controls (Dewailly et al. 1994).

As indicated in Section II.A, fish consumption advisories are designed to take into account the fact that some populations are potentially at a greater risk for exposure and potential effects than the general population. Recent studies indicate that some high-risk populations are changing their fish consumption and preparation habits in response to fish consumption advisories. A survey of 8,000 sport anglers in the Great Lakes states found that 36 percent of the respondents had changed their fish consumption behaviors, including modifying fish cleaning and preparation methods and eating less Great Lakes fish (Connelly and Knuth 1993). Another study found that pregnant women of the Mohawk nation had substantially reduced their fish consumption (Fitzgerald et al. 1993).

While fish advisories are designed to the reduce the harmful health effects from eating contaminated fish, they may have negative cultural, societal, and economic impacts. A few of these factors are described briefly below (adapted from U.S. EPA 1996a):

- *Nutritional Value*. Fish are known to be high in important nutrients such as protein and are generally low in fat. By heeding advisories to limit or avoid fish consumption, people may reduce their intake of an important food source without supplementing their diet with other nutritional foods or vitamin supplements. This is especially true for poor subsistence fishers; after fish consumption advisories are issued, these people may not be able to afford other means for eating a well-balanced diet.

- *Health Benefits of Fish Consumption*. A diet rich in fish may have some important health benefits, as demonstrated by studies that have compared diets high in fish with traditional western diets (summarized in U.S. EPA 1996a). Examples of possible health benefits from diets with a high proportion of fish are reduced risk for cardiovascular disease and certain cancers. Therefore, the health risks from not eating fish may outweigh the potential health risks from eating contaminated fish.

- *Traditional Activities*. Eating and catching fish are important cultural activities for many subgroups. For example, for centuries some Native American tribes have built cultural traditions around spearing fish and sharing the catch (U.S. EPA 1992). Tribes that live near large waterbodies often use certain fish species to symbolize characteristics or ideas. For sport anglers, fishing and eating their catch are important social activities. Issuing fish advisories can impose on these important cultural activities, greatly affecting a subgroups' traditional values.

- *Dietary Patterns*. Subgroups such as Native American and Asian American communities have long-standing traditions of eating fish and place importance on celebratory meals highlighted by fish. People may not want to substitute different ingredients or cook in different ways. In addition, the potential substitutes for fish may be more unhealthy and expensive.

- *Fishing and Tourism Industries*. As discussed below, fishing advisories may negatively affect the fishing and tourism industries.

An on-going Canadian study established as a First Nations/Health Canada partnership is assessing the extent of exposure of Native people living in the Great Lakes basin to bioaccumulative pollutants and the associated risk to their health and well-being. The project, Effects on Aboriginals from the Great Lakes Environment (EAGLE), began in September 1990 and is expected to be completed in 1997. EAGLE is a community-based epidemiological project involving the approximately 100,000 First Nations people living in 63 aboriginal communities in the Great Lakes Basin. The project builds on earlier studies and is examining exposure in both adults and children, socio-economic effects, and the impacts on traditional ways of life, culture, and values (Manno et al. 1995).

Commercial and Recreational Fishing Losses

Bans and advisories on commercial fishing due to pollution can cause economic and social losses to owners and employees of commercial fishing enterprises. In addition, bans and advisories on recreational fishing have potentially far-reaching effects on the U.S. economy; expenditures on recreational fishing stimulate the economy, provide jobs in the industry, and generate state and federal taxes (Fedler and Nickum 1992). In 1991, more than 35.6 million Americans over 16 years of age spent a total of $24 billion on sport fishing, averaging 13 trips each and 14 days a year (U.S. FWS 1993). As shown in the sidebar, losses due to fish consumption advisories can be significant.

> The Arkansas Game and Fish Commission has estimated a loss of fishing expenditures due to mercury fish consumption advisories of over $5 million dollars from 1991 to 1992.
>
> Source: Armstrong 1994.

Other Recreational Losses

In addition to recreational fishing, recreational hunting (e.g., for food or fur) may be negatively impacted by exposure of wildlife to the pollutants of concern. These impacts include decreases in the populations of hunted species (e.g., stemming from adverse reproductive effects) and increased health risks to the hunters who consume their kill. Poor water quality (and/or public perception of poor water quality) may negatively affect recreational uses other

than fishing and hunting, such as boating, swimming, and visiting parks. Decreased recreational use also may affect the related tourism economies; the annual outdoor recreation/wildlife industry in Great Lakes states generates approximately $12 billion (NWF 1993). An example of efforts to study the impact of recreational losses in one of the Great Waters is presented in the sidebar.

EPA is currently funding a project that will assess the value of certain recreational services (e.g., bird watching, windsurfing, fishing) to the Corpus Christi Bay National Estuary Program. The objective of this project is to assist in prioritizing management actions for this estuary. Findings from the project will be used, in part, to provide a framework for assessing both economic and natural resource value losses due to negative impacts associated with excess nitrogen loadings to Corpus Christi Bay and other estuaries.