Environmental Contaminants

Chemical contamination of the environment is a pervasive, insidious side effect of human population growth and technological development. Unlike many other environmental stressors, casual observers often become aware of chemical pollutants only after some catastrophic event, such as an oil or chemical spill that kills many animals. Typically, chemical discharges that can be seen or smelled—or barren mine spoils (Fig. 1) and fishless streams—call attention to chemical pollutants. More commonly, sophisticated instruments are needed to detect toxic chemicals.

The effects of chemical contaminants on individual organisms, populations, and ecosystems may also be difficult to detect. The imperceptible accumulation of DDT (Table 1) and its metabolites in bald eagles and other predatory birds led to eggshell thinning, reproductive failure, and, ultimately, greatly reduced populations over much of North America (for example, Carson 1962; Colborn 1991; Wiemeyer et al. 1993). Much of this happened before the problem was understood sufficiently to take the steps necessary to correct it; populations of bald eagles are now recovering (U.S. Fish and Wildlife Service 1994).

Effects of other contaminants on less visible species may be more subtle and localized. In addition to killing plants and animals, contaminants may reduce growth and reproduction or cause animals to avoid contaminated areas or to become more vulnerable to predators. The effects of chemical contaminants may also be masked by the more obvious effects of human population growth and technological development with which chemicals often co-occur. Sedimentation occurs in mined areas (Fig. 1); pesticides accompany sediments, nutrients, salts, and physical habitat changes as a consequence of intensive mechanized agriculture; and industrial chemicals, petroleum, pesticides, sewage, and combustion products accompany manufacturing, commerce, and urban growth.

Contaminants may be transported long distances in air and water, be modified in form and toxicity on release into the environment, and cause ecological injury far from their original sources. The resiliency of biological systems at all levels of organization—from subcellular through organism, population, and ecosystem—may also mask the effects of chemicals.

Environmental contaminants are generally perceived as a twentieth-century problem. Although this is true for synthetic organic chemicals, some of our most ecologically significant contaminant problems derive from the historical use of older technologies. Other contaminant problems existed for decades until technology and knowledge advanced sufficiently to detect and understand them. Some—including those resulting from mining, irrigation, and energy extraction—represent the accumulation of naturally occurring substances to harmful concentrations as a result of human-induced acceleration of natural weathering processes. In this chapter, it is not possible to discuss the thousands of potentially toxic substances released to the environment nor to evaluate their individual and combined effects on U.S. biota. Instead, I will present a historical overview of those human activities from which contaminant releases have been most significant—mineral and energy exploitation, agriculture, and industrial and urban growth. I also summarize the extent to which the ecological effects of the releases are known, and I present some information on future contaminant threats to biological diversity and some suggestions for where information is most needed.

Courtesy M. Mac, USGS

Fig. 1. Mine tailings from historical lead mining near Desloge, Missouri.

Mineral and Energy Exploitation

The extraction and processing of minerals and fossil fuels have had widespread environmental consequences. Mining, drilling, smelting, refining, and other technologies associated with the extraction and exploitation of minerals and fossil fuels produce toxic by-products that can be released into the environment and transported long distances. Mineral and energy extraction activities also physically disrupt habitats and ecosystems.

Nonferrous Metals

Obtaining metals from ores requires separating the sought-after materials from the host rock and other minerals in the ore, a process that generates large quantities of wastes: noncoal-mining wastes in the United States totaled 50 billion metric tons in 1985 and were accumulating at an estimated 1.3 billion metric tons per year (U.S. Environmental Protection Agency 1985). Ores vary, but metal carbonates, oxides, and sulfides constitute the majority of commercially exploitable U.S. mineral deposits. Of these, the mining of sulfide minerals—which constitute important copper, nickel, lead, zinc, and silver deposits—has been the most environmentally significant. In the process of extracting metals, sulfur is oxidized and lost to the atmosphere as SO_2, which reacts with water and other atmospheric constituents to produce sulfuric acid. Therefore, smelters contribute substantially to acid precipitation. Because most ores are mixtures of minerals, potentially toxic elements other than the sought-after metals—including arsenic, cadmium, and mercury—may also be present and released into the environment. Following extraction—which varies in efficiency but is never complete—the natural oxidation of metal sulfides remaining in the solid wastes can produce acidic, metals-rich leachates, which may contaminate surface- and groundwaters. In addition to toxins, the large volumes of solid waste generated by mining and milling may also cause environmental problems (Fig. 1).

The technology for extracting and purifying gold, silver, copper, lead, and other metals from sulfide ores was brought to the Western Hemisphere by Europeans. North American colonization and subsequent westward expansion in the United States were stimulated first by the search for precious metals and later by the needs of industry. As the United States grew westward, new ore deposits were discovered and exploited. Although extraction efficiency also improved, the nineteenth and twentieth centuries brought tremendous increases in scale to the mining industry through the advent of steam, internal combustion, and electric power. The basic methods for extracting metals, though, continued to rely largely on mechanical separation, gravity, water, and heat. Consequently, the increasingly large quantities of toxic metals and by-products released into the environment left their marks on U.S. biota. No information on the cumulative effects of metals mining and refining on biota exists, but 557,650 abandoned mines in the United States are estimated to have contaminated 728 square kilometers of lakes and reservoirs and 19,000 kilometers of streams and rivers (Mining Policy Center 1994). Areas where toxic releases to the environment from mining and smelting have caused large-scale effects on United States biological diversity or have jeopardized particularly rare or valuable living resources are numerous; some examples are listed in Table 2.

In addition to the toxic releases generally associated with extracting metals from sulfide ores, gold and silver mining caused additional problems—placer and hydraulic mining and amalgamation with mercury. A placer is a sediment deposit from which gold and silver are mined; the sediments are dredged and washed in place. In hydraulic mining, water is pumped from a stream to wash the precious metals from surrounding banks and hillsides. Placer and hydraulic mining alter habitats and release metals, other toxins, and suspended solids directly into waterways. Aquatic plant, invertebrate, and fish production is generally reduced in placer-mined streams (Cordone and Kelley 1961; Lloyd et al. 1987). Both historical and ongoing instream mining still affect streams in California and Alaska (for example, Buhl and Hamilton 1990).

Table 1. Chemical names, common names, and acronyms of compounds mentioned in text.

Chemical name, common name, or acronym	Primary use(s)
Alachlor [Lasso® ; 2-chloro-2',6'-diethyl-N-(methoxymethyl)-acetanilide]	Herbicide
Aldrin (1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4:5,8-dimethanonaphthalene)	Organochlorine insecticide
Atrazine (Aatrex®; 2-chloro-4-ethylamino-6-isopropylamino-s-triazine)	Herbicide
Azinphos-methyl {O,O-dimethyl S[4-oxo-1,2,3-benzotriazin 3(4H)-methyl] phosphorodithioate}	Organophosphate insecticide
BHC (Benzene hexachloride; 1,2,3,4,5,6-hexachlorocyclohexane)	Organochlorine insecticide
Butylate (S-ethyl diisobutylthiocarbamate)	Carbamate insecticide
Carbofuran (Furadan®; 2,2-dimethyl-2,3-dihydro-7-benzofuranyl-N-carbamate)	Carbamate insecticide
Carbaryl (Sevin®; 1-naphthyl N-methylcarbamate)	Carbamate insecticide
CFC's (Chlorofluorocarbons, a family of gases comprising halons and freons)	Refrigerants, propellants, and fire supressants
Chlordane (1,2,4,5,6,7,8,8-octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methanoindane, and other similar compounds)	Organochlorine insecticide
Chlorinated dibenzofurans (a family of compounds that are structurally and toxicologically similar to chlorinated dioxins)	Impurities; combustion products
Chlorinated dioxins (a family of structurally similar compounds, of which 2,3,7,8 tetrachloro-*p*-dibenzodioxin [TCDD; dioxin] is the most toxic)	Impurities; combustion products
Cyanazine [Bladex®; 2-chloro-4-(1-cyano-1-methylamino)-6-ethylamino-s-triazine]	Herbicide
2,4-D (2,4-dichlorophenoxyacetic acid)	Herbicide
2,4,5-T (Silvex®; 2,4,5-trichlorophenoxyacetic acid)	Herbicide
DDD {Rhothane®; TDE; [1,1-dichloro-2,2-bis(*p*-chlorophenyl) ethane]}	Organochlorine insecticide; DDT breakdown product
DDE [1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethylene]	DDT breakdown product
DDT [1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane]	Organochlorine insecticide
Dicofol (Kelthane; 1,1-bis(chlorophenyl)-2,2,2-trichloroethanol]	Miticide
Dieldrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4:5,8-dimethanonaphthalene)	Organochlorine insecticide; aldrin breakdown product
Endosulfan (6,7,8,9,10,10-hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,4,3-benzodioxathiepin 3-oxide)	Organochlorine insecticide
Endrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4:5,8-dimethanonaphthalene)	Organochlorine insecticide; aldrin breakdown product
Fonofos (Dyfonate®; O-ethyl-S-phenylethylphosphonodithioate)	Organophosphate insecticide
Glyphosate [Roundup®; N-(phosphonomethyl)glycine, isopropylamine salt]	Herbicide
Heptachlor (1,4,5,6,7,8,8-octachloro-2,3,3a,4,7,7a-heptahydro-4,7-methanoindane)	Organochlorine insecticide; chlordane constituent
Hexachlorophene (2,2'-dihydroxy-3,3'5,5'6,6'-hexachlorodiphenylmethane)	Disinfectant
Kepone® (chlordecone; decachlorooctahydro-1,3,4-metheno-2H-cyclobuta(cd)pentalen-2-one)	Organochlorine insecticide
Methomyl {S-methyl-N-[(methylcarbamoyl)oxy] thioacetamide}	Carbamate insecticide
Methoxychlor [1,1,1-trichloro-2,2-bis(*p*-methoxyphenyl)ethane]	Organochlorine insecticide
Methyl parathion (O,O-dimethyl-O-4-nitrophenyl phosphorothioate)	Organophosphate insecticide
Metribuzin [4-amino-6-(1,1-dimethyl)-3-(methylthio)-1,2,4-triazin-5(4H)-one]	Herbicide
Metolachlor [2-chloro-N-(2-ethyl-6-methylphenyl)-N-(2-methoxy-1-methylethyl)acetamide]	Herbicide
Mirex [dodecachlorooctahydro-1,3,4-metheno-2H-cyclobuta(cd)-pentalene]	Organochlorine insecticide; fire retardant
PAH's (polycyclic aromatic hydrocarbons; a family of compounds—some of which are toxic and carcinogenic—containing primarily carbon and hydrogen)	Combustion products and petroleum constituents
PCB's (polychlorinated biphenyls of varying composition and toxicity)	Dielectric, heat transfer, and hydraulic fluids; lubricants; plasticizers; copy papers; extenders in many products
Pentachlorophenol (PCP)	Wood preservative
TCDD	(see chlorinated dioxins)
Terbufos {Counter®; S-[[(1,1-dimethylethyl)thio]methyl] O,O-diethyl phosphorodithioate}	Organophosphate insecticide
Toxaphene (chlorinated camphene mixture averaging 62% chlorine by weight)	Organochlorine insecticide
Trifluralin [Treflan®; 2,6-dinitro-N,N-dipropyl-4-(trifluoromethyl) benzenamine]	Herbicide
VOC's (volatile organic compounds—trichloroethylene, tetrachloroethylene, carbon tetrachloride, chloroform, chlorobenzenes and related compounds, and methylene chloride)	Solvents, cleaning agents, industrial feedstocks

Amalgamation is a process in which pure liquid mercury is used to separate particles of gold and silver from other materials, a practice that has led to mercury contamination in many areas of the West. For example, mining of the Comstock Lode in Nevada, which began before 1900, led to the release of substantial quantities of mercury to the Truckee-Carson River system (Richins and Risser 1975); this system comprises wetlands, lakes, and rivers important to fishes and migratory birds. Mercury from historical gold and silver mining remains evident in biota on both sides of the Sierra Nevada in California (Phillips 1987; Schmitt and Brumbaugh 1990; Hallock and Hallock 1993).

The second half of the twentieth century brought environmental regulation and improved extraction efficiency to the mining and metal refining industry. The incorporation of flotation processes employing organic reagents, for example, enhanced the efficiency with which metals are recovered from tailings, so wastes contain lower concentrations of leachable metals; SO_2 is recovered from smelter stack emissions, which reduces acid formation; and flue dusts containing metals are trapped and

recycled (Baker and Bhappu 1974). Collectively, these improvements result in less toxic and less widespread emissions.

Some reagents and by-products of the newer technologies also affect biota, however. The organic reagents can induce algal growth and alter invertebrate communities in receiving streams (Hardie et al. 1974). Sodium cyanide, used to extract precious metals, is highly toxic (Baker and Bhappu 1974; Eisler 1991). Accidental discharges of cyanide from tailings ponds and other facilities have caused massive and well-documented fish kills (for example, Leduc 1984). Cyanide-leach ponds at gold mines attract waterfowl and other wildlife and have caused numerous wildlife kills—some recently (Eisler 1991). Consequently, mines and smelters remain ecologically significant sources of pollutants despite recent improvements (Nriagu 1984; U.S. Environmental Protection Agency 1993).

Ferrous Metals and Coal

Commercially important iron deposits occur as oxides from which little sulfur is lost to the environment. Consequently, iron mining is not a major source of toxic contaminants. Of much greater environmental significance than the iron itself is the reducing agent necessary for its recovery. Carbon is supplied to reduce iron from the oxidized to the metallic state; either charcoal or coke—hardwood or coal, respectively, from which elements other than carbon have been driven by slow burning under restricted airflow—is typically used. In North America, iron and steel were made with charcoal until about 1840, when the eastern hardwood forests were exhausted. This coincided with the development of steam power and, consequently, railroads, which opened the vast Appalachian coal fields to the iron and steel industry.

Acid Mine Drainage

The most direct and immediately obvious toxicological effect of the switch to coal was acid mine drainage. Acid drainage is caused by the oxidation of pyrite—iron sulfide—a common mineral constituent of rocks and soils that is often present in coal mine wastes and overburden. Streams affected by coal mine drainage are typically acidic (pH 2.5–6.0) and are characterized by high iron and sulfate concentrations. In addition, the iron, as ferric hydroxide, often precipitates as a fine flock that may coat stream bottoms and further affect aquatic life (Fig. 2). Acid mine drainage may also leach toxic concentrations of metals such as copper, aluminum, and zinc from rocks and soils (Barton 1978). The oxidation, or weathering, of pyrite and other metal sulfides is a natural process that may occur spontaneously on exposure of minerals to air, or it may be mediated by bacteria. Weathering of pyritic rock formations has resulted in naturally acidic streams in some localized areas of the Southeast (Huckabee et al. 1975) and Colorado (Bencala et al. 1987).

By the mid-1960's, a century of U.S. surface mining had disturbed about 8,000 square kilometers, including 21,000 kilometers of streams

Table 2. Examples of sites where mining and mineral exploitation have affected U.S. biological diversity or have affected particularly valuable biological resources.

Location	Effect
Copper Basin, Tennessee	SO_2 emissions from copper smelting beginning in 1843 eliminated vegetation over a 130-square-kilometer area (Tyre and Barton 1986) and may have contributed to the endangered status of Ruth's golden aster, a federally listed plant endemic to the Ocoee Valley. Metals and sediment have also contaminated Tennessee Valley Authority reservoirs on the Ocoee River (U.S. Environmental Protection Agency 1977; Tennessee Valley Authority 1991).
Palmerton, Pennsylvania	Zinc smelting emissions from 1898 to 1980 completely denuded an 8-square-kilometer area and affected plants and animals for a much greater distance (Jordan 1975). Stream aquatic communities were not measurably affected (Carline and Jones 1993).
Tri-State Mining District—Missouri, Kansas, Oklahoma	A century of zinc mining and smelting and attendant acidification and toxic metals releases have left bare areas and eliminated animal life from small streams. Among affected species are the Neosho madtom, a federally listed threatened fish (Williams et al. 1989), and the Neosho mucket, a rare mussel that is a candidate for federal listing (Missouri Department of Conservation 1992).
Torch Lake, Michigan	The disposal of tailings and other copper mining wastes from the late 1860's to the 1960's is believed responsible for an outbreak of liver cancer in sauger and walleye (Black and Baumann 1991). The sauger were ultimately eliminated, and the walleye are sustained by stocking (Ellenberger et al. 1994).
Leadville, Colorado	Mining in the headwaters of the Arkansas River system since the 1860's has resulted in acidification and toxic metals pollution that continues to affect aquatic communities for 50 kilometers downstream (Roline 1988; Kiffney and Clements 1993).
Clark Fork River system, Montana	Some 15 million cubic meters of mine tailings containing elevated metal concentrations, generated since mining began in 1880, have visibly contaminated and affected the aquatic biota in more than 230 kilometers of the Clark Fork main-stem (Woodward et al. 1994). Acidic, metals-laden mine drainage has also affected the benthic and fish communities and reduced the productivity of sport fisheries in the Blackfoot River, a tributary (Moore et al. 1991).
Blackbird Mine, Idaho	Mining contaminated 40 kilometers of Panther Creek, a tributary of the Salmon River. Releases of copper, colbalt, and other metals for more than 50 years decimated the resident fishery and the spring-summer run of chinook salmon, a threatened species.
Coeur d'Alene Valley, Idaho	Mining and smelting in and around Kellogg since 1879 have contaminated the South Fork of the Coeur d'Alene River, obliterated area vegetation (U.S. Environmental Protection Agency 1977), and contaminated biota (Henny et al. 1994; Hoiland et al. 1994). Mining wastes were responsible for repeated deaths of tundra swans into the late 1980's (Henny et al. 1991).
Iron Mountain Mine, Redding, California	Fish kills caused by metals released from mines and mine wastes have occurred in the Sacramento River for more than 100 years (Finlayson and Wilson 1979). Threatened are populations of steelhead (the sea-run form of rainbow trout) and chinook salmon, which have been denied access to all but 32 kilometers of Sacramento River spawning habitat since construction of Shasta Dam. Metals from Iron Mountain Mine, together with warm summer discharges from Shasta Dam, may be responsible for the imperiled status of the spring-run chinook salmon population (Finlayson and Verrue 1980; M. K. Saiki, Dixon, California, personal communication).

Courtesy D. Hardesty, USGS

Fig. 2. Iron hydroxide precipitate (orange) in a Missouri stream receiving acid drainage from surface coal mining.

(totaling 550 square kilometers), 281 natural lakes (419 square kilometers), and 168 reservoirs (168 square kilometers; Spaulding and Ogden 1968). Coal mining accounted for 41% of the total disturbed lands, the bulk of the injury coming from acid mine drainage occurring in the East and the Midwest (Spaulding and Ogden 1968). Acid drainage from coal mining affected 9,480 kilometers of streams in the Appalachians (Kinney 1964), with surface mining accounting for about 25% and underground mines about 75% (Amhad 1974). The Appalachian Regional Commission (1969) estimated that about 12,000 kilometers of streams and 120 square kilometers of impoundments in the eastern coal-mining regions were seriously affected by acid mine drainage. Acidified streams typically result in reduced diatom, benthic invertebrate, and fish communities (for example, Matter et al. 1978; Vaughan et al. 1978; Winger 1978).

Injury from acid mine drainage has been extensive and well-documented in the Cumberland River system of Kentucky and Tennessee. Streams of the Cumberland Plateau historically supported unique fish and invertebrate faunas that have been severely affected by surface mining; 23% (22 species) of the historical mussel fauna is either extinct or federally listed as endangered, and several other species have either been eliminated from significant portions of their historical ranges or reduced to small, nonreproducing populations (Layzer et al. 1993). Rare fishes and other invertebrates have also been affected (Layzer and Anderson 1992).

Injury to aquatic biota has also been extensive elsewhere in the East, but documentation on a species-by-species basis has not been undertaken. In the arid regions of the West, acid drainage is not typically associated with coal mining because deposits are generally overlain by alkaline soils and rocks (Moore and Mills 1977). In addition, the intensive mining of western coals is a relatively recent phenomenon; mining in the West has been more closely regulated and mined land reclamation has been more rigorously practiced than in historically mined areas farther east.

Current U.S. surface mining regulations mandate the restoration of disturbed lands. Moreover, restoration of abandoned coal mines has been under way since 1977; programs to seal mine openings and revegetate surface-mined lands have improved water quality in many areas and, through the incorporation of new technology, have the potential to improve more. Despite these gains, the National Stream Survey estimated that in 1986, 4,590 kilometers of streams—about 10% of the total in Pennsylvania, West Virginia, and Maryland—remained acidic because of acid mine drainage and that an additional 5,780 kilometers were strongly affected but not acidic (Herlihy et al. 1990). Other estimates range as high as 20,000 kilometers (for example, Girts and Kleinmann 1986). Moreover, the long-term success of remediation efforts on restoring biological diversity remains to be evaluated.

Acid Precipitation and Other Inorganic Contaminant Releases Into the Atmosphere

Coal is primarily carbon, but because it consists of decomposed plants and animals, it also contains variable concentrations of other elements, including sulfur, arsenic, selenium, aluminum, mercury, and cadmium. Eastern and midwestern coals are especially high in sulfur content. The combustion of these coals releases large quantities of SO_2, the major component of acid precipitation, to the atmosphere. Most of the high-sulfur coal consumed in the United States during this century has been used to make steel and to generate electricity in the East and Midwest. From there, atmospheric pollutants responsible for acid precipitation are transported northward and eastward by prevailing winds and storms (Haines 1981). These trends are reflected in the geographic distribution of rainfall pH (Fig. 3). Emissions from coal-fired electric generating plants (Fig. 4) presently constitute the largest source of atmospheric SO_2 (Placet 1991). Other constituents of acid precipitation,

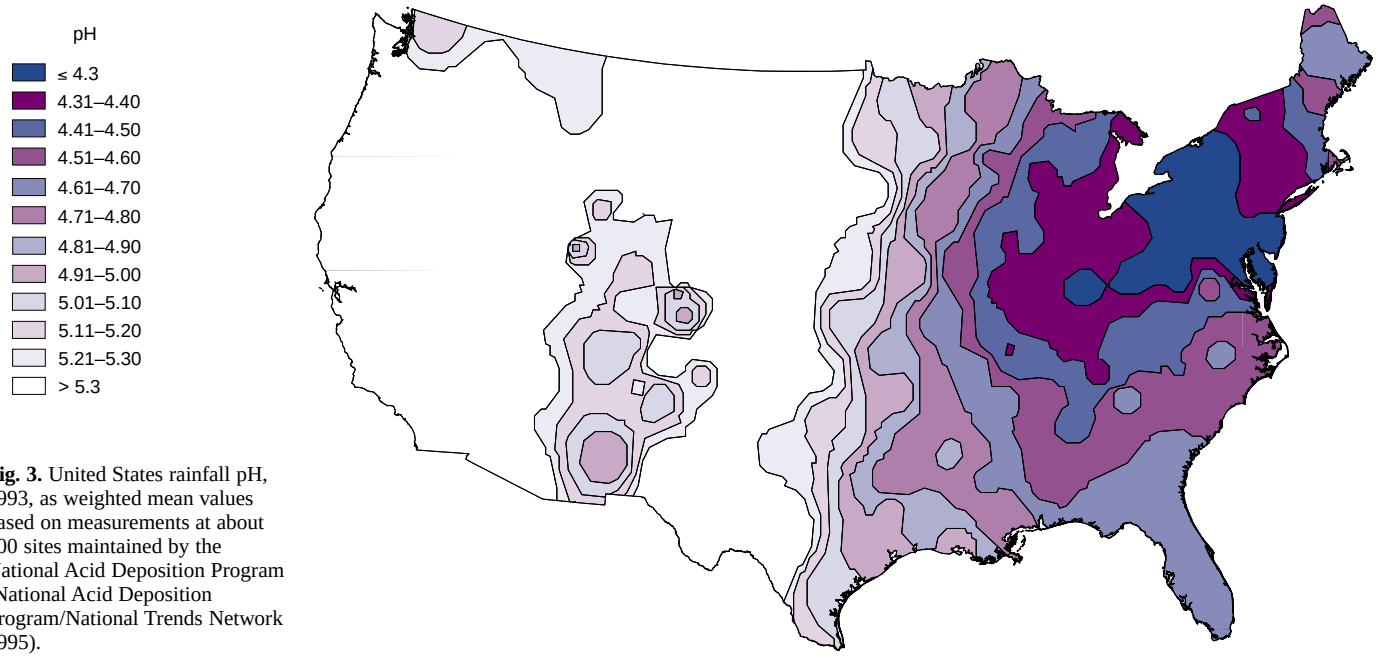


Fig. 3. United States rainfall pH, 1993, as weighted mean values based on measurements at about 200 sites maintained by the National Acid Deposition Program (National Acid Deposition Program/National Trends Network 1995).

including those from automotive exhausts, are distributed similarly (Sisterson 1991).

The chemistry of acid precipitation is complex and involves atmospheric and terrestrial components. Hydrogen ions in precipitation and their deleterious effects may be buffered by carbonates, organic matter, and other rock and soil constituents. Consequently, areas underlain by crystalline rock, shale, and sandstone are more susceptible to acidification than those underlain by limestones and other carbonate-rich rock. Potentially sensitive areas are widely distributed in North America and include much of the Appalachian Mountains, where rainfall is most acidic (Fig. 3); the Canadian Shield region of the upper Midwest (that is, the northern parts of Michigan and Wisconsin, as well as eastern Minnesota and parts of eastern and central Canada); the higher elevations of the Sierra Nevada, Rocky Mountains, and Cascade Range; and parts of the Ozark and Ouachita uplands, mid-Atlantic Coastal Plain, and Florida. Buffering by ions in groundwater and constituents leached from watersheds makes large lakes and rivers at lower elevations less susceptible to acidification than smaller, higher-elevation lakes and streams.

Fig. 4. A coal-fired electric generating plant near Concord, New Hampshire.

The interactions of ions in precipitation (that is, H^{+}, SO_4^{+2}, NO_3^{-2}) with organic and inorganic constituents of soil and water affect toxicity. Particularly important is the leaching of potentially toxic elements, especially aluminum, from rocks and soils by acidic precipitation. Aluminum toxicity occurs in acidic waters with low organic content; organic matter binds aluminum, making it less toxic (Wright and Snevik 1978; Baker and Schofield 1980; Driscoll et al. 1980). Sensitivity to pH and aluminum varies with taxon and life stage, but acid-sensitive species occur among most groups of aquatic organisms, including mollusks, mayflies, zooplankton, and fishes (Haines 1981; Baker 1991a). Acidified waters typically contain zooplankton faunas dominated by a few resistant species, reduced benthic macroinvertebrate diversity and productivity, altered fish communities, and no mollusks (Haines 1981). Among fishes, certain minnows (Cyprinidae) and the early life stages of some salmonids, including Atlantic salmon and rainbow trout, are more sensitive than those of other taxa, such as brook trout (Haines 1981).

Toxicity attributable to pH and aluminum is often episodic, occurring during high surface-water discharge; pH during spring snowmelt is characteristically low and aluminum is correspondingly high in those areas that develop significant snowpack (Wigington 1991). Spring is also the time when salmonid alevins emerge from stream gravel, making them susceptible to episodic acidification and aluminum toxicity (Fiss and Carline 1993). Significant episodes can also occur during large rainfall events in other seasons (Wigington 1991).

Acid rain is defined as rainfall with a pH lower than about 5.0; the pH of distilled water in equilibrium with atmospheric CO_2 is 5.6, but other atmospheric constituents tend to make rainfall more acidic even in areas unaffected by air pollution. In eastern North America, the 1970–1971 mean pH based on weekly measurements was about 4.0 at monitoring stations in upstate New York and New Hampshire, with rainfall pH of individual storms regularly less than 4.0. Historical records indicated pH values as low as 2.1 in 1964 (Likens and Bormann 1974). The National Surface Water Survey (NSWS) estimated that in addition to waters acidified from coal mining, 1,181 lakes (4.2% of those surveyed) and 5,506 kilometers of streams (2.7%) were acidic and that potentially toxic aluminum concentrations were present in 3% of the lakes and 13% of the streams surveyed (Baker 1991b). Almost all the affected waters were in six susceptible areas—the southwestern part of the Adirondack Mountains, New England, forested mid-Atlantic highlands, Atlantic Coastal Plain, northern Florida highlands, and the eastern part of the upper Midwest (Baker 1991b).

The biological consequences of acid precipitation in North America were first publicized with the discovery of fishless lakes—110 lakes above 610 meters elevation in the Adirondack Mountains (Schofield 1976). Significant changes in fish community composition, including the elimination of smallmouth bass, also occurred in many lakes that contained fish (Schofield 1976). Further study revealed more widespread ecological injury. The NSWS estimated that nearly 60% of headwater stream reaches in the mid-Atlantic Coastal Plain, 23% of Adirondack lakes, and 18% of mid-Appalachian streams are too acidic to support brook trout, an acid-tolerant species (Baker 1991a). Among Adirondack lakes, 24% have no fish; the 16% for which historical data are available have lost one or more fish species; and brook trout have been eliminated from 11% of those lakes that historically supported the species (Baker 1991a). Trends in Vermont and New Hampshire are similar to those reported for the Adirondacks, and acid-sensitive species have been eliminated from headwater streams in Massachusetts (Baker 1991a). In Maine, acidified lakes are unsuitable for natural reproduction of Sunapee char, a rare landlocked form of arctic char (Jagoe et al. 1984). In Pennsylvania, an estimated 3,000 kilometers of trout streams are affected by acid precipitation (Carline et al. 1992; Fiss and Carline 1993), as are 10% of trout streams in West Virginia (Cosby et al. 1991). Some precipitation-acidified headwater streams in Pennsylvania are toxic to salmonids, including brook trout, during spring runoff (Carline et al. 1992). In the Northeast as a whole, about 5% of lakes otherwise suitable for brook trout are too acidic, and 9% will not support other acid-tolerant species (Baker 1991a). In the mid-Appalachian region, about 30% of stream reaches surveyed by the NSWS will not support acid-sensitive species such as blacknose dace (Baker 1991a). Moreover, the NSWS did not survey small lakes (less than 4 hectares) and headwater streams and may have consequently underestimated the full extent of injury (Baker 1991a).

Acidification has also affected migratory fishes, amphibians, and terrestrial organisms in the Northeast. Atlantic salmon have been eliminated from coastal streams in northern Europe (Rosseland et al. 1986) and in maritime Canada (Elson et al. 1973; Watt 1986; Haines 1992). Although New England coastal streams receive acidic deposition (Fig. 3), they are not chronically acidic, and effects on Atlantic salmon have not been as severe. Nevertheless, parr production in tributaries of Maine coastal streams may occasionally be reduced by episodic acidification (Haines 1992). In tributaries of Chesapeake Bay, episodic acidification and elevated aluminum concentrations associated with spring rains may reduce the survival of larval striped bass (Hall et al. 1985, 1991). In addition, about 65% of the small coastal streams surrounding the upper Chesapeake Bay in Maryland are too acidic for blueback herring, an acid-sensitive species (Baker 1991a). Acidification of breeding sites in the Northeast was suspected of reducing the survival of larval frogs, toads, and salamanders (Haines 1981), but amphibians avoid the most acidified habitats (Freda 1986). Many northeastern amphibians breed in temporary pools fed by acidified spring meltwaters and rain; these small habitats may also have been overlooked by regional water-quality surveys (Blancher 1991). Elsewhere, studies on the effects of acid precipitation on amphibians have proven equivocal (for example, Bradford et al. 1994).

Acidification-induced changes in the aquatic fauna of small lakes in the Northeast have affected the distribution of fish-eating birds, including ospreys, common loons, belted

kingfishers, and common mergansers, as well as other ducks (Diamond 1989; Longcore et al. 1993; Desgrangies and Gagnon 1994). The reduced benthic macroinvertebrate productivity typical of acidified waters may indirectly depress production of American black ducks (Haramis and Chu 1987), a species that has been in decline in the Northeast since the 1950's (Steiner 1984). Acidification also reduces the availability of soil calcium to forest insects, which may ultimately lead to calcium deficiency and eggshell thinning in insectivorous birds (Drent and Woldendorp 1989; Blancher 1991). Direct injury by acid precipitation to forest vegetation is believed limited to red spruce at high elevations in the northern Appalachians (Barnard and Lucier 1991); however, indirect effects on wildlife caused by subtle vegetation shifts have been documented. The distributions of tree swallows and eastern kingbirds have been altered by acidification-induced forest changes—including effects on invertebrate food organisms (Diamond 1989; Longcore et al. 1993)—as was the production of birds in acidified areas of Europe (Graveland et al. 1994). The long-term effects of acidification on wildlife populations and on the productivity of terrestrial ecosystems have not been thoroughly assessed (Barnard and Lucier 1991; Blancher 1991; Schreiber 1995).

In addition to sulfur, the combustion of coal releases other potentially toxic elements, including arsenic, cadmium, lead, mercury, and selenium into the environment (Dvorak and Lewis 1978). Arsenic, cadmium, and selenium are concentrated in coal ash, from which they may be leached into surface waters and accumulated to toxic concentrations by aquatic organisms (Hall and Burton 1982). In North Carolina (Lemly 1985; Baumann and Gillespie 1986) and Texas (Garrett and Inman 1984), selenium from power plant ash caused mortality and reproductive failure in fishes. Mercury, some selenium, and other elements in coal are released into the atmosphere in stack emissions (Fig. 4) and may be transported long distances. Mercury, like selenium, tends to accumulate in birds, mammals, and predatory fishes; it is also highly toxic to most organisms. Mercury was formerly a pollutant associated with gold mining and point sources such as caustic soda (that is, sodium hydroxide) plants and paper mills (May and McKinney 1977). The accumulation of mercury in the biota of remote lakes in the Northeast (for example, Driscoll et al. 1994), however, has raised concerns about atmospheric transport and natural chemical processes that tend to keep mercury available for accumulation by organisms. Mercury of presumed atmospheric origin has accumulated in the biota of remote inland lakes in Maine, where it is believed to be the cause of reduced reproductive success in bald eagles relative to coastal populations (Welch 1994). Coal-fired electric generating plants are the greatest sources of atmospheric mercury; other important sources include municipal and hospital waste incinerators (U.S. Environmental Protection Agency 1993).

Courtesy P. C. Baumann, USGS

Fig. 5. External neoplasms (tumors) and deformed chin barbels on a brown bullhead from the lower Black River, a Lake Erie tributary, in Lorain, Ohio. Polycyclic aromatic hydrocarbons (PAH's) from coke ovens, long discharged to Great Lakes harbors and estuaries in steel-making cities, are believed responsible for outbreaks of these and other types of cancer in fishes. Fish populations in which the frequency of such lesions is high typically contain few old individuals, suggesting that the tumors are ultimately lethal (Baumann et al. 1990).

Polycyclic Aromatic Hydrocarbons

Coal combustion also releases organic chemicals, especially polycyclic aromatic hydrocarbons (PAH's; Table 1), into the atmosphere. Few of these are directly toxic; however, some are potent human and animal genotoxins (that is, mutagens, carcinogens, and teratogens). These compounds can also lower the disease resistance of many animals (Hodgins et al. 1977). The most toxic PAH's, including the genotoxins, are rapidly metabolized by vertebrates and hence do not tend to accumulate in food chains (Eadie 1984). Nevertheless, benthic invertebrates inhabiting contaminated sediments are a source of PAH's to fishes, shorebirds, and mammals. The full extent and significance of releases of PAH's from coal combustion are unknown (Heit 1985).

The production of coke from coal also generates PAH's, and the indiscriminate disposal of coking wastes has led to the contamination of many industrialized waterways. Contamination by PAH's is especially evident in the harbors of steel-making cities of the Great Lakes basin (Great Lakes Water Quality Board 1985; Baumann and Whittle 1988). The benthic invertebrate and fish faunas of these waters are characteristically reduced in diversity (Great Lakes Water Quality Board 1985; Smith et al. 1994), and bottom-dwelling fishes in some waters are afflicted with liver cancer and other tumors (Baumann 1989; Black and Baumann 1991; Fig. 5) that reduce life expectancy in affected populations (Baumann et al. 1990). Significant quantities of PAH's derived from coal are also released from other metallurgical sources, including foundries and electric arc furnaces that produce aluminum and steel (Quilliam et al. 1985; Thrane 1987).

The use and disposal of other coal-derived by-products, especially creosote, were also significant sources of PAH's to the environment. Creosote, a condensate distilled from coal tar, was used for decades as a wood preservative. Contamination from creosote wood treatment facilities is common, and such facilities have affected biota by inducing tumors in fish, reducing benthic diversity, or both, in many locations (Malins et al. 1984; Bender et al. 1988; Swartz et al. 1989; Pastorok et al. 1994). Creosote was also used to separate metals from ore and is among the possible causes of liver cancer in walleye and sauger of Torch Lake, Michigan

(Baumann 1989; Black and Baumann 1991). Creosote is no longer registered for use as a wood preservative, but nevertheless it continues to enter the environment from historical sites of use and formulation as well as from treated wood products.

Petroleum

Petroleum, like coal, is a complex mixture containing thousands of compounds derived from the decay of prehistoric plants and animals. The most abundant of these compounds are aliphatic (straight-chained) and aromatic (ring-structured) hydrocarbons (Clark and Brown 1977). Petroleum hydrocarbons are composed of mostly carbon and hydrogen, but some also contain oxygen, nitrogen, sulfur, and other elements and vary greatly in molecular weight, volatility, solubility, persistence, and toxicity (Clark and Brown 1977; Neff 1979; Robotham and Gill 1989; Shales et al. 1989). Crude oils and refined products, as well as wastes from petroleum production and processing facilities, are also highly variable in composition and toxicity. In general, the more volatile, water-soluble, and lower molecular weight aromatic components are the most directly toxic to aquatic organisms. These are also proportionally more abundant in refined products than in crudes (Shales et al. 1989; Mielke 1990).

In addition to direct toxicity by aromatic hydrocarbons, other petroleum components may harm biota through different mechanisms. Oil slicks may especially affect organisms concentrated at the air–water interface—including marine birds and mammals and the eggs and larvae of recreationally and commercially important fishes and invertebrates. Oil may coat shorelines, beaches, and tidal flats (Fig. 6), suffocating intertidal organisms. Several mechanisms may cause the incorporation of oil into marine particulates—including small planktonic organisms—which may result in the incorporation of petroleum hydrocarbons into pelagic food chains, or the particles may sink to the bottom and affect benthic organisms. The decomposition of petroleum hydrocarbons by bacteria depletes oxygen, which may cause oxygen stress, and the loss of insulating capacity caused by oil on feathers and fur increases the vulnerability of birds and mammals to cold (Geraci and Smith 1977; Holmes and Cronshaw 1977; Kooyman et al. 1977). Microliter quantities of oil transferred to eggs from the feathers of oiled birds can be toxic to developing embryos (Albers 1977; Hoffman 1979, 1990; King and Lefever 1979), and ingestion of a single dose by female birds may alter the yolk structure and reduce the hatchability of eggs (Grau et al. 1977).

Courtesy D. Chapman, USGS

Fig. 6. Oiled salt marsh in Corpus Christi Bay, a Texas estuary.

Large quantities of petroleum enter the environment, mostly from land-based sources. The National Academy of Sciences estimated that annual inputs of petroleum to the oceans during the 1970's were about 4.5 million metric tons annually, derived from the following major sources: land-based discharges other than refineries (including sewage, urban runoff, and waste oils), 53%; natural seeps, 13%; atmospheric transport and deposition, 13%; marine operations (bilge and ballast discharges, and so forth), 11%; offshore production, 4%; marine accidents (oil spills and well blowouts), 3%; and refineries, 0.4% (Clark and MacLeod 1977). Land-based, nonindustrial sources similarly predominate even in Raritan Bay, New Jersey, a heavily industrialized estuary ringed by petroleum storage, transport, and processing facilities. About 92,000 kilograms of petroleum hydrocarbons enter Raritan Bay daily. Of this total, sewage treatment effluents and urban runoff represent 76%; other industrial effluents, 19%; and spills about 2% (Connell 1982).

On release into the environment, the composition and potential toxicity of petroleum mixtures change rapidly and continuously as individual compounds are volatilized, solubilized, dispersed, and degraded at differing rates by physical, chemical, and biological processes. The rates of these weathering processes vary depending on temperature, currents, wind, concentrations of suspended and dissolved components of the receiving water, and biological activity. The timing of petroleum releases relative to the distribution and life cycles of organisms determines the potential exposure and, correspondingly, the biological effects of exposure. Ecosystems also vary in their susceptibility to oil (Gundlach and Hayes 1978; Getter et al. 1984). Nearshore areas, especially sheltered marshes and estuaries, are generally more vulnerable than those of the open ocean; coastal habitats are more diverse and productive, and the restricted circulation characteristic of some causes longer residence time at higher concentrations of the oil (Fig. 6). Arctic and subarctic

ecosystems may be more vulnerable than those in warmer climates because of the slower rates of dispersion and decomposition of petroleum components especially under ice, where oil decomposes very slowly. Animals dependent on openings in the ice may be at particular risk. The comparatively low productivity and regenerative capacity of cold-climate organisms also increase their vulnerability; arctic vegetation is also very sensitive and slow to recover from oil exposure (Clark and Finley 1977). Conversely, the toxicity of oil increases with temperature (Mayer and Ellersieck 1986). Collectively, these factors make the environmental fate and effects of oil highly variable and dynamic.

Oil Spills

Much of what is known about oil in the environment is derived from the study of transportation and production accidents, despite their relatively small contribution to the global flux of petroleum. Spills of varying proportions involving both crudes and refined products have occurred often during this century, and recent events have been widely publicized. Studies of major North American spills and well blowouts—and of many other smaller spills, and of those that occurred outside North America—have provided dramatic evidence of short-term ecological injury (Phillips and Lincoln 1930; Clark and Finley 1977; Mielke 1990). Massive direct mortalities of plants, shellfish, mammals, and birds are common, with regularly reported kills of tens and hundreds of thousands of birds as a result of individual spills (Holmes and Cronshaw 1977). A 1952 spill off Chatham, Massachusetts, for example, reduced the overwintering population of eiders from 500,000 to 150,000, and a 1969 well blowout in Santa Barbara, California, killed 64% of the loons and grebes in the area, which represented 7%–10% of the regional populations (Perry 1980). Direct deaths of adult fishes occur infrequently, but the early life stages of many fish species are vulnerable because the fishes are then highly concentrated in inshore areas and near the surface; losses, however, are difficult to observe and quantify. Inland spills also occur regularly, but they have received much less study than marine spills. Reported findings are nevertheless similar: short-term effects include kills of plants, invertebrates, fishes, amphibians, reptiles, water birds, and mammals; one 1988 Ohio River diesel spill killed an estimated 5,000 waterfowl and innumerable fish and invertebrates (Cronk et al. 1990). Unlike marine spills, however, long-term effects of inland spills have received little study (Shales et al. 1989).

The effects of the 1989 *Exxon Valdez* spill in Alaska are still being investigated, but findings to date indicate that about 40% of the estimated 6,500 endangered sea otters inhabiting Prince William Sound were killed (Garrott et al. 1993), and as many as 300,000 seabirds (Piatt et al. 1990), including 120,000 adult common murres, 12,000 marbled murrelets, and 150 bald eagles (*Exxon Valdez* Trustee Council 1994). Moreover, mortality figures based on counts of dead animals recovered or observed shortly after oil spills usually underestimate the total kill; in addition to dead animals being difficult to find, delayed deaths and indirect effects are also underestimated (Holmes and Cronshaw 1977). In Prince William Sound, for example, the number of dead birds recovered was estimated to be only 10%–15% of the total killed (*Exxon Valdez* Trustee Council 1994). By 1994, bald eagles in Prince William Sound had nearly recovered, and common murres and marbled murrelets were recovering, but numbers of northwestern crows, cormorants, arctic terns, and tufted puffins remained depressed in oiled areas (*Exxon Valdez* Trustee Council 1994). Survival of river otter pups was poor in years following the spill (*Exxon Valdez* Trustee Council 1994), and sublethal effects in adult otters remained evident in oiled areas (Duffy et al. 1993). Survival of larval Pacific herring was not affected despite the presence of large quantities of oil on spawning beaches (McGurk et al. 1990, 1992), but biochemical evidence of exposure remained evident in intertidal fishes 14 months after the spill (Woodin and Stegeman 1993). Lower than normal survival of eggs and larvae in oiled areas may have reduced the adult pink salmon population of Prince William Sound by as much as 10%, with further reductions possible from secondary effects (*Exxon Valdez* Trustee Council 1994).

Longer-term effects of spills on populations and communities are less obvious, and follow-up studies of individual events, when they have been conducted, have provided equivocal evidence of significance and persistence. In addition, it is not clear whether sufficient studies can be conducted to discriminate the effects of single events, especially in areas of natural seepages and repeated spills. Events such as spills are always difficult to evaluate because they are not repeatable and because prespill data are lacking for most locations (Norcross 1992). Generally, 10 years is the estimated recovery time for local effects from individual oil spills, but each spill is a separate event governed by different physical and biological constraints (Mann and Clark 1978). Coral reefs and mangroves are highly vulnerable to spills and may take longer to recover (Gundlach and Hayes 1978; Fucik et al. 1984; Getter et al. 1984). In addition, the long-term effects of spills in cold climates have not yet been fully evaluated and

await the *Exxon Valdez* findings; the seabird colonies of Prince William Sound may take 20 years or more to recover (*Exxon Valdez* Trustee Council 1994).

Chronic Oil Pollution

Although chronic pollution has been studied much less intensively, chronic pollution by oil and other contaminants associated with petroleum production, transport, and refining is generally regarded as more widespread and serious than individual spills and other accidents (Holmes and Cronshaw 1977). In addition, at some point along a continuum, repeated spills become chronic pollution; such spills have been implicated as a factor responsible for the long-term decline of seabird colonies in parts of northern Europe and maritime Canada (Holmes and Cronshaw 1977). Annual bird losses from chronic oil pollution in the North Sea and North Atlantic Ocean were estimated as 150,000–450,000—murres, scoters, grebes, and gulls were the most commonly recovered groups (Perry 1980).

A significant component of chronic pollution from petroleum exploitation is produced water—the water that often accompanies oil and natural gas in geologic formations. Water may also be injected into wells to force oil to the surface. Produced waters may contain toxic concentrations of petroleum hydrocarbons, salts and other ions (including metals and radionuclides), and reagents used to enhance the separation of oil from water (Andreasen and Spears 1983; Woodward and Riley 1983; Roach et al. 1993). Some 36 billion liters of produced water are discharged annually to coastal waters in the United States (U.S. Environmental Protection Agency 1995). Historically, produced waters were discharged untreated; however, this practice has been regulated by most states, and the elimination of discharges to all coastal waters except Cook Inlet, Alaska, has been proposed (U.S. Environmental Protection Agency 1995).

Disposal of produced waters often represents a significant source of injury to biota in oil production areas (Leppäkoski and Lindström 1978; Armstrong et al. 1979), especially in estuarine marshes with restricted freshwater inflow and limited tidal exchange (for example, Nance 1991). In Texas, chronic pollution from produced water discharges has affected the biota of Nueces, Trinity, and Galveston bays (Armstrong et al. 1979; Henley and Rauschuber 1981; Carr 1993; Roach et al. 1993; D'Unger et al. 1996; Fig. 7); about 11 million liters of produced waters are discharged daily into Galveston Bay (Andreasen and Spears 1983). In freshwater streams that drain oil fields and refineries, the diatom, benthic macroinvertebrate, and fish communities are often affected by produced water discharges (Wilhm and Dorris 1968; Woodward and Riley 1983; Olive et al. 1992). At some inland sites, produced waters are stored in reserve pits or lagoons for evaporation, or they may be used to create wetlands. These may represent sources of continuing mortality for waterfowl and wading birds (Ramirez 1993). In arctic regions where evaporation rates are low and reserve pits must be discharged, benthic invertebrate diversity is reduced in receiving waters (West and Snyder-Conn 1987).

Courtesy D. Chapman, USGS

Fig. 7. Produced waters—the water accompanying petroleum in geologic formations—being discharged to Nueces Bay, Texas.

Because many point sources of water pollution associated with petroleum production and refining have been regulated, the contribution of nonpoint sources has become more apparent. As noted earlier, waste oils, discharges from marine operations, and urban runoff are significant, pervasive sources of petroleum hydrocarbons in the environment. Petroleum-derived aromatic hydrocarbons are present in the sediments of all urban waterways, and concentrations tend to be elevated in areas of petroleum storage and transport. Especially high concentrations occur in the sediments of Casco Bay, Maine; Boston and Salem harbors, Massachusetts; western Long Island Sound; Raritan Bay, New Jersey; Baltimore Harbor, Maryland; San Diego Harbor and parts of San Francisco Bay, California; Elliot Bay, Washington (Calder and Means 1987; Helz and Huggett 1987; Turgeon and Robertson 1995); and in many Great Lakes harbors and bays (Meyers 1984; Fabacher et al. 1991). Petroleum-contaminated sediments typically contain genotoxic PAH's, and tumors have been reported in bottom-dwelling fishes from many of these locations (Baumann 1989; Turgeon and Robertson 1995; Fig. 5). Fin rot, which may indicate reduced resistance to disease, is also

common in fish from areas chronically contaminated by petroleum (Minchew and Yarbrough 1977; Turgeon and Robertson 1995). The specific linkages between these disease outbreaks and petroleum pollution have been challenged (Mix 1986). It is also difficult to attribute such effects, which may have multiple causes, to one chemical or group of chemicals among the complex mixtures usually present in urban, industrial environments.

Air Pollution

Petroleum constituents and combustion products contribute significantly to air pollution. Refineries and fuel-handling and transport facilities are major sources of benzene and other organic components of smog, and the combustion of petroleum releases PAH's to the atmosphere. Although coal combustion, primarily by electric utilities (Fig. 4), is the greatest source of atmospheric SO_2, vehicular exhausts also contribute significantly. In addition, refineries emit large quantities of sulfur into the atmosphere. Vehicular emissions are the largest source of nitrogen oxides (NO_x), which contribute to smog and acid precipitation, as well as carbon monoxide (CO) and carbon dioxide (CO_2; Placet 1991). Both NO_x and CO are precursors of ozone, the air pollutant most responsible for injury to vegetation (Barnard and Lucier 1991). Automobile exhausts also contain ethylene, which is toxic to vegetation and also contributes to ozone formation (Heck and Anderson 1980). Methane, CO, and CO_2 are among the so-called greenhouse gases responsible for global warming; their concentrations are increasing in response to increasing global consumption of fossil fuels (Boden et al. 1994). The combustion of leaded gasoline, which was introduced in 1923 (Nriagu 1990), remains the greatest source of lead to the global atmosphere (Settle and Patterson 1980). Leaded gasoline also contributes to urban lead pollution; however, environmental lead concentrations in the United States have generally declined over the last decade (Fig. 8), ostensibly as a result of the removal of lead from gasoline and the control of emissions from mining and point sources (Smith et al. 1987).

Agriculture and Forestry

In the twentieth century, the advent of machinery run with fossil fuels greatly aided the conversion of vast expanses of native forests, prairies, wetlands, and deserts into croplands across North America. In addition to physical changes, the irrigation of formerly arid lands leaches minerals from soils at accelerated rates, resulting in accumulations of some elements to

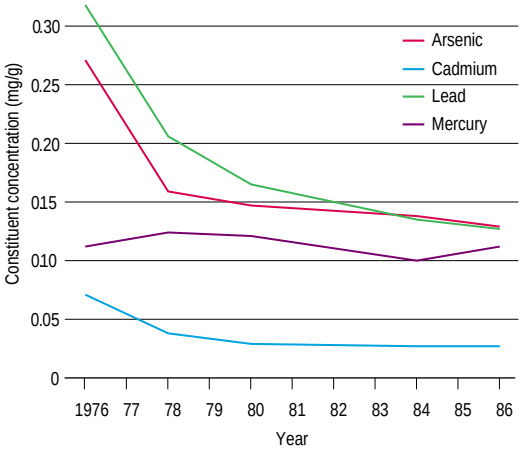


Fig. 8. Concentrations of arsenic, cadmium, lead, and mercury in United States freshwater fish, 1976–1986 (U.S. Fish and Wildlife Service, National Contaminant Biomonitoring Program, unpublished data).

toxic concentrations. Agricultural conversion also simplifies complex natural ecosystems, which facilitates pest and disease infestations and creates a market for pesticides. Agricultural runoff may contain high concentrations of sediments and plant nutrients, which have profoundly affected stream biota in the Midwest and elsewhere (for example, Trautmann 1957). Nutrients (that is, nitrogen and phosphorus) from agricultural runoff, with increased temperature and light penetration, can stimulate the growth of algae and nuisance rooted aquatic vegetation. The decomposition of excess vegetation may cause oxygen deficiency in affected waters. Bacteria can also convert nitrogen from fertilizers and animal wastes to ammonia, which is highly toxic to aquatic organisms.

Toxic Minerals From Irrigation

Wetlands in the arid and semiarid regions of the West support migratory birds and rare endemic fishes, wildlife, and plants (Preston 1981; Thompson and Merritt 1988). That the evaporation of water from these "arid wetlands" (Lemly et al. 1993) could cause toxic accumulations of minerals was known to wildlife biologists since at least 1891, when an estimated 35,000 eared grebes and northern shovelers were found dead at Owens Lake, California (Phillips and Lincoln 1930). Subsequent extensive kills of ducks and other birds occurred on the Bear River and Great Salt Lake marshes of Utah during droughts early in the twentieth century. Similar incidents occurred widely during the 1920's, from the Dakotas and Canadian prairies to Oregon and California, and in Kansas, Oklahoma, Texas, and northern Mexico; particularly serious outbreaks occurred

in the Klamath River basin in Oregon (Phillips and Lincoln 1930). These episodes were termed alkali poisoning and were believed caused by the ingestion of plant and animal material containing toxic levels of salts—primarily of sodium, magnesium, and calcium—concentrated by evaporation (Phillips and Lincoln 1930). Subsequent studies revealed the involvement of selenium in these episodes (Twomey and Twomey 1936). It was known in the 1920's that evaporation and the concentration of salts to toxic levels were worsened by water withdrawals for irrigation (Phillips and Lincoln 1930).

Selenium, a natural constituent of the earth's crust, is essential to all plants and animals. At high concentrations, however, it can be toxic. Elevated concentrations commonly occur west of the Mississippi River in areas of arid, alkaline soils derived from marine shales (Lemly and Smith 1987). Irrigation of arid soils leaches selenium salts and those of other elements into surface waters. Once in solution, the salts may be concentrated by evaporation and are available for accumulation by aquatic organisms and animals that feed on them. Among the high-selenium areas of the West is the extensively drained, intensively irrigated Central Valley of California. Beginning in the 1970's, irrigation return water from selenium-rich Central Valley farmland was used to create and maintain wetland habitat at Kesterson National Wildlife Refuge; drain water constituted nearly all of Kesterson's water supply by 1981 (Lemly et al. 1993). Primarily selenium, but also boron, accumulated in the refuge, causing widely publicized incidents involving wildlife kills, poor hatching success, and embryo deformities in ducks and other birds (Fig. 9) in the early and middle 1980's (Ohlendorf et al. 1986; Lemly et al. 1993). Aquatic organisms were also affected: a massive fish kill and subsequent reproductive failure eliminated all fishes except mosquitofish from irrigation drains (Saiki 1986) and refuge ponds, and a high incidence of embryo deaths occurred even in this resistant species (Saiki et al. 1991). At Kesterson, selenium also accumulated to high concentrations in snakes, frogs, raccoons, and small mammals, but no deleterious effects were detected (Clark 1987; Ohlendorf et al. 1988; Clark et al. 1989).

Selenium also accumulated in reservoirs constructed in seleniferous areas of western Colorado; those impoundments became unable to support fish in the 1950's (Baumann and May 1984). Following the discoveries at Kesterson, further studies of irrigation drain water by the U.S. Department of the Interior revealed either overt symptoms of selenium poisoning, selenium concentrations sufficiently high to induce such symptoms, or both at nine other refuges in the West and in the Gunnison River basin, Colorado, and the middle Arkansas River basin, Kansas (Stephens et al. 1992; Lemly et al. 1993; Presser et al. 1994). Elevated selenium concentrations also occur in water (Dvorak and Lewis 1978; Stephens et al. 1992) and fishes (Schmitt and Brumbaugh 1990; Fig. 10) throughout much of the basins of the Colorado and Missouri Rivers. In the Colorado and Green River systems, irrigation and other water-development activities have further increased naturally elevated concentrations to the extent that selenium may be at least partly responsible for the currently imperiled status of the razorback sucker, humpback chub, bonytail chub, and Colorado squawfish—all federally listed threatened or endangered fishes (Williams et al. 1989; Stephens et al. 1992; Hamilton and Waddell 1994). Elevated selenium concentrations in the Rio Grande (International Boundary and Water Commission 1994) may also be caused by water withdrawals for irrigation.

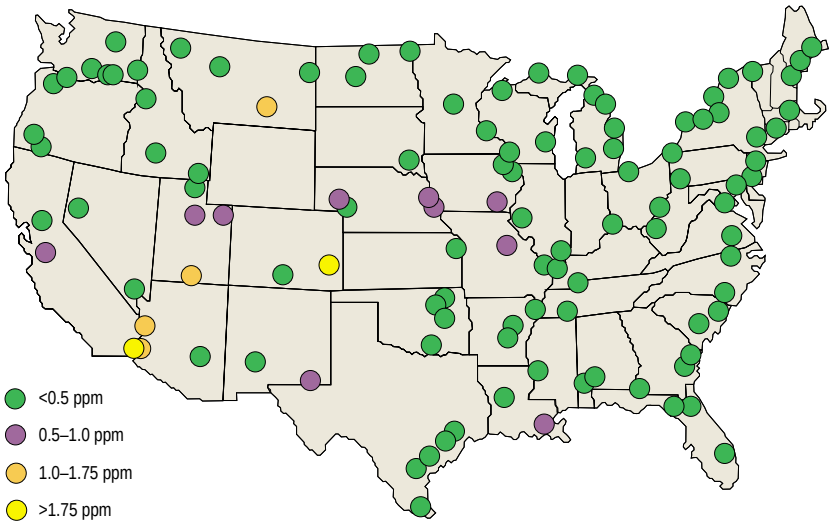


Fig. 10. Geographic distribution of selenium in freshwater fish collected from the indicated sites in the conterminous United States in 1986.

Courtesy H. M. Ohlendorf, U.S. Fish and Wildlife Service

Fig. 9. An American coot embryo from Kesterson National Wildlife Refuge, California, with selenium-induced developmental abnormalities including a deformed lower bill and no eyes.

Pesticides

Widespread use of pesticides did not occur until the twentieth century; however, chemicals were used to control insects, fungi, and weeds much earlier. Inorganic pesticides, including copper sulfate, mercuric chloride, lead and sodium arsenates, sodium dichromate, hydrogen cyanide, and sodium chlorate became widely available in the nineteenth and early twentieth centuries (Clarkson 1991; Ghering et al. 1991; Stevens and Sumner 1991). These were followed by organo-metallic compounds (that is, organic chemicals containing arsenic, mercury, tin, and others), most of which remained in use until after World War II; some organo-arsenicals are still used (Clarkson 1991). Plant-derived organic pesticides, including pyrethrum and strychnine, were available commercially by the end of the nineteenth century (Ray 1991). Organic compounds obtained from coal and oil, including kerosene, naphthalene, and carbon disulfide, were in use as pesticides by 1900 (Ghering et al. 1991).

Organochlorine Pesticides

Synthetic organic chemicals containing chlorine, including carbon tetrachloride and trichloroethylene, were available commercially by 1925 (Ghering et al. 1991). Benzene hexachloride (Table 1) was first synthesized in 1825, and DDT in 1874; however, the insecticidal properties of these compounds were not recognized until around 1940 (Smith 1991). The military developed and produced DDT during World War II to control mosquitoes and, thereby, the spread of malaria and other diseases. Released into civilian markets in 1945, DDT was used heavily over the next two decades to control agricultural and forest insects as well as disease vectors; by 1961, 1,200 formulations were available for use on 334 crops (U.S. Environmental Protection Agency 1992). DDT was also used to control fishes, bats, and other wildlife. After World War II, additional organochlorine pesticides—including methoxychlor, aldrin, dieldrin, and chlordane—became available. These were followed in the 1950's–1960's by endosulfan, endrin, mirex, kepone, toxaphene, and others (Smith 1991; Table 1). In addition to being highly toxic, organochlorine pesticides are relatively insoluble in water, adhere strongly to soil particles, and are resistant to physical, chemical, and biological degradation. These properties were viewed as desirable, and negative consequences from bioaccumulation and toxicity to nontarget organisms were not foreseen.

The use of DDT in the United States peaked in the late 1950's; maximum production, 80–90 million kilograms per year, occurred in the early 1960's (Hayes 1991; U.S. Environmental Protection Agency 1992). Cotton farming in the Southeast, the Delta states, and the southern plains accounted for 74% of the 15 million kilograms of DDT used in 1964 on farms in the United States (Eichers et al. 1971). Other heavily treated crops included tobacco, soybeans, and fruits and vegetables. Cotton pests developed resistance to DDT, and it was replaced in the 1960's by newer compounds, especially toxaphene. Consequently, DDT represented only about one-third of the 34 million kilograms of insecticides applied to cotton in 1964, with toxaphene and nonorganochlorine chemicals accounting for the rest in about equal proportions (Eichers et al. 1971).

As the use of DDT declined, total organochlorine chemical use increased; in 1966, 18 million kilograms of organochlorine insecticides were used in agriculture, including 6 million kilograms of DDT and DDD (a DDT derivative; Table 1), 7 million kilograms of toxaphene, and 3 million kilograms of aldrin (Fox et al. 1968; Eichers et al. 1971; Andrilenas 1974). After the 1969 DDT ban, toxaphene became the most heavily used insecticide, averaging about 13 million kilograms annually through the early 1970's; most was used for insect control in cotton (Eichers et al. 1978). Cotton insects eventually developed resistance to toxaphene, and usage declined through the late 1970's, well before its registration was canceled in 1983.

Aldrin, heptachlor, chlordane, and similar compounds were used heavily to control insects in corn during the 1970's, and organochlorine chemical use in the Midwest increased accordingly (Eichers et al. 1978). As insect resistance and environmental concerns grew, use of these and other organochlorine compounds declined from 46% of the total used in 1971 to 29% in 1976 (Andrilenas 1974; Eichers et al. 1978). At present, only endosulfan and methoxychlor are registered for agricultural use, although other organochlorine insecticides are still manufactured for export.

The ecological consequences of organochlorine pesticides were extensive, and some remain evident. Because of their insolubility in water and resistance to complete metabolic degradation, many organochlorine compounds bioaccumulate. Upon accumulation by vertebrates, DDT is metabolized to DDE (Table 1), which is stable and toxic; it impairs calcium metabolism in the shell gland of adult female birds. At sufficiently high concentrations, eggshell thickness can be reduced to the extent that eggs cannot support the weight of the incubating parents. Shell breakage and death of the developing embryo can result. Susceptibility varies, but predatory birds are most vulnerable, both physiologically

A History of Aquatic Toxicology

Most disciplines of biology have evolved along a path of hypothesis testing, but the field of aquatic toxicology is an exception. Society's demand for information in an atmosphere of increasing litigation initially drove, then hastened, the development of the field of aquatic toxicology. Before 1962, pollution concerns were largely focused on sewage treatment facility operations and eutrophication of lakes, streams, and estuaries. With the publication of *Silent Spring* by Rachel Carson in 1962, however, the warning alarm was sounded, proclaiming that organic contaminants, particularly pesticides such as DDT, posed a threat to the health of natural resources and humans.

One of the most salient features of the development of environmental toxicology as a scientific discipline has been the expressed need for chemical detection and identification simultaneously with the need to determine the biological effects associated with chemicals. Lags in methodological progress, both chemical and biological, limited observable progress in the early years of environmental toxicology and chemistry. Thin-layer chromatography and gas chromatography were first used to detect and identify pesticides and organic chemicals; atomic adsorption spectrophotometry was used to detect metals and other inorganic materials. At the same time, acute lethality (in which the subject organism dies in 96 hours or less) toxicity tests were initially developed and standardized to bracket extreme biological effects.

In 1977, a group of scientists working in various areas within the field gathered to discuss the ideal attributes of a toxicity test. This group, made up of representatives from government, academia, and industry, determined that the following attributes were the most important in a toxicity test and ranked them in order of their significance:

- produces ecologically significant results;
- generates scientifically and legally defensible data;
- is based on methods that are routinely available for widespread application;
- is predictive;
- methods are widely applicable across a range of chemicals; and
- test is simple and cost-effective.

The group evaluated available aquatic toxicity tests and scored them on the basis of the previously listed criteria. A score of 100% (out of a possible 100%) was awarded to acute lethality toxicity tests because of their ecologically significant results (death … unarguably important), applicability across chemical classes, simplicity and cost-effectiveness, and their scientifically and legally defensible results.

If evaluated today, acute lethality toxicity tests would still fare well for some of the same reasons but also for some different ones. Acute lethality tests allow for the rapid building of comparative data bases in which species can be compared in terms of their sensitivity to the same chemical or by which chemicals can be compared to one another using the same species. Additionally, water quality can be varied to evaluate potential interactions with toxicity (for example, as pH increases, the toxicity of some metals increases). When dealing with new chemicals, new formulations of existing chemicals, mixtures, changing environmental conditions, and so forth, a rapid screening toxicity test is often invaluable. Consequently, acute lethality tests remain a comparative framework for evaluation.

This approach, however, does not address the important and more likely situation encountered in natural systems, namely, longer-term, sublethal exposure and its ecological consequences. Scientists grew concerned about the inadequacy of so-called "kill 'em and count 'em" tests and developed methods to evaluate changes induced by contaminants that affected reproduction, behavior, physiological processes, biochemical function, and survival of young and other sensitive life stages. Although disruptions in these areas might render an organism "ecologically dead," it may not technically induce direct mortality. For example, courtship behavior may take place normally in the presence of sublethal concentrations of toxicants, but the larvae produced might be malformed or unable to make developmental progress. Because researchers needed chronic, sublethal tests to approximate effects more likely to occur in nature, they developed full life cycle (from birth until the organism reproduces) fish tests. Although the results are ecologically meaningful (such as contaminant effects on the number of eggs produced, percent successful hatch, survival to swim-up, and so forth), such tests are difficult and expensive to run without problems. For example, of the native warmwater, freshwater fishes adaptable to laboratory conditions for full life cycle testing, fathead minnows complete the life cycle in the shortest time; even so, a test on them can last 3 to 6 months depending on broodstock and test conditions. Consequently, to decrease the testing time required, partial life cycle testing began to replace full life cycle tests. Such partial life cycle tests decrease the probability of some test condition complications, can bracket sensitive life stages (such as reproduction), and can increase the potential for testing various species of interest even if their full life cycles could not actually or practically be completed in a reasonable time.

In the late 1970's and early 1980's, invertebrate tests became increasingly important because they took less time than full life cycle testing and because disruptions in food-chain dynamics at lower trophic levels can translate into severe ecological consequences for top predators and species of monetary and ecological concern. In addition, invertebrate testing requires less space and specialized equipment than fish testing.

Behavioral toxicity tests, although ecologically relevant if the endpoints measured are interpretable, have met with limited success because of their intrinsic variability when replicated. The very thing that contributes to their sensitive detection capabilities can backfire if the animals are not acclimated properly or standardized test approaches are not appropriately conducted. The expenditure of time and labor required, however, can be offset by the ecologically interpretable results of such tests.

Biochemical and physiological approaches have become important in aquatic toxicology over the last 5 to 8 years. Not only do such approaches demonstrate the relation between exposure and effects, but they can also sometimes explain the toxic action of the contaminant. As researchers have refined biomarker techniques, such techniques have become more specific and sensitive in detecting contaminants. Baseline information on the normal physiological and biochemical states of aquatic organisms has also grown, making perturbations due to contaminants more discernible.

Currently, scientists are emphasizing portable, field-oriented, sublethal yet acute detectors of dose–response. Microtox (trade name for a bioluminescing bacteria assay) and rotifer tests are receiving much attention. Field approaches, originally survey-oriented and aimed at detecting the presence of a problem, have moved more and more toward hypothesis testing and experimental manipulation. This is a great addition to field data because experimental information

can be expanded with more confidence past the geographic perimeters of the field site, and cause–effect relations can be more realistically investigated.

The realization that no single test approach meets all needs or answers all questions has become even more evident over the last decade. The fact is that many "tools" are needed and each should be selected and combined with others in diverse configurations depending on the contaminants of interest and the questions being addressed. Continued effort is required to further develop meaningful, cost-effective, and field-friendly methodologies to detect contaminants and their effects on aquatic biota.

See end of chapter for reference

Author

Mary G. Henry
U.S. Fish and Wildlife Service
1849 C Street N.W.
Washington, D.C. 20240

and because of their position at the apex of aquatic food chains (Cooke 1973). In North America, population declines attributable to DDE-induced eggshell thinning and reduced recruitment were documented in bald eagles, ospreys, prairie falcons, merlins, double-crested cormorants, and brown pelicans (Cooke 1973). Other contaminants and factors may also have contributed to the declines in these species, especially in the Great Lakes (Colborn 1991). Shell thinning was also documented in other species, including peregrine falcons and herring gulls, but many factors may also have been involved in their declines (Cooke 1973).

The use of DDT and other organochlorine insecticides in agriculture and forestry also affected aquatic organisms. In waters draining cotton-farming areas, the diversity of benthic invertebrate and fish communities was characteristically low, and the development of insecticide-resistant forage fishes led to elimination of predatory species in the 1960's (Ferguson et al. 1964). In addition to directly affecting bald eagle reproduction (Colborn 1991), DDT spraying of United States and Canadian forests in the 1940's and 1950's to control eastern spruce budworms (Fig. 11) and western spruce budworms reduced benthic invertebrate diversity and fish populations in streams over wide areas of the Northeast (Ide 1957; Warner and Fenderson 1962) and the West (Adams et al. 1949; Graham 1960; Cope 1961).

Courtesy U.S. Fish and Wildlife Service

Fig. 11. Forest insecticide spraying.

Agricultural and forest insect control accounted for more than half of organochlorine insecticide usage in the United States during the 1960's and 1970's (Andrilenas 1974; Eichers et al. 1978; Aspelin et al. 1992). Nevertheless, other pesticide uses, mechanisms of toxicity to birds other than eggshell thinning, and effects on other organisms were also significant. Application of chemicals to water for the control of mosquitoes, gnats, and black flies was common. Heavy applications of DDD for gnat control decimated western grebes at Clear Lake, California (Herman et al. 1969). Mosquito control using DDT led to reproductive failure of lake trout in Lake George, New York (Burdick et al. 1964), and of landlocked Atlantic salmon in Sebago Lake, Maine (Anderson and Everhart 1966), and was suspected of reducing the survival of juvenile winter flounder in a Massachusetts estuary (Smith and Cole 1970). During the 1950's, widespread use of DDT to control the bark beetles that transmit Dutch elm disease reduced American robin populations in many northeastern and midwestern communities (Cooper 1991); some 80 bird species were affected by the Dutch elm disease program in Michigan alone (Wallace et al. 1961). In southern Ontario, residual DDT and dieldrin from mosquito control have been suggested as a cause of local amphibian extinctions (Russell et al. 1995).

Accumulations of newer organochlorine insecticides—such as chlordane, aldrin, dieldrin, heptachlor, and endrin—caused many wildlife kills, including endangered gray bats in Missouri (Clark 1981; Clawson and Clark 1989). The use of endrin to control sugarcane borers and of heptachlor (Table 1) to control imported red fire ants in Louisiana during the 1950's–1960's led to repeated massive fish kills in the Atchafalaya and Lower Mississippi rivers and along the gulf coast (Biglane and Lafleur 1967). The consumption by waterfowl of seeds treated with heptachlor and other organochlorine pesticides caused frequent kills in agricultural areas. Dieldrin is suspected of contributing to the decline of the Great Lakes population of bald eagles (Colborn 1991). As presently known, however, the overall extent of

ecological injury attributable to these newer organochlorine insecticides has been less than that of DDT.

In response to declining organochlorine pesticide use in North America, residue concentrations of DDT and other persistent compounds in fish and wildlife declined steadily during the last decade (Mineau et al. 1984; Prouty and Bunck 1986; Bunck et al. 1987; Baumann and Whittle 1988; Schmitt et al. 1990; Wiemeyer et al. 1993; Weseloh et al. 1994; Mora 1995; Figs. 12 and 13). Elevated levels of DDT and other organochlorine insecticides persist in the Great Lakes, some cotton-growing areas of the South and Southwest (White and Krynitsky 1986; Schmitt et al. 1990; U.S. Environmental Protection Agency 1992; Schmitt and Bunck 1995; Fig. 14), and in the Mississippi River

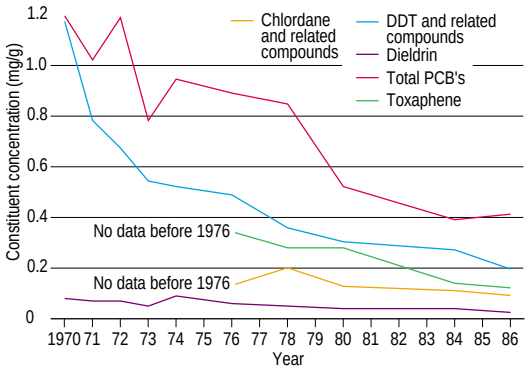
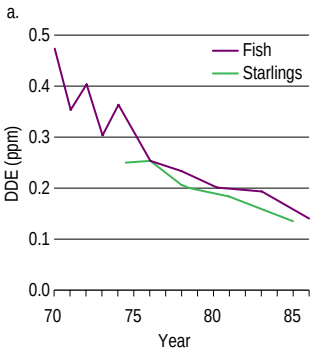


Fig. 12. Concentrations of organochlorine chemicals in U.S. freshwater fishes, 1970–1986 (U.S. Fish and Wildlife Service, National Contaminant Biomonitoring Program, unpublished data).



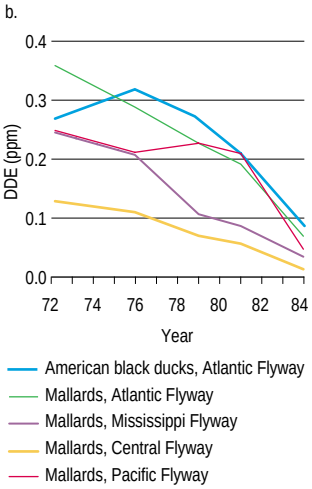


Fig. 13. Mean concentrations of DDE, the most stable metabolite of DDT, in U.S. Fish and Wildlife Service monitoring networks, 1970–1986: a) in freshwater fish and European starlings collected nationwide and b) in mallards and American black ducks from the four major flyways (from Schmitt and Bunck 1995).

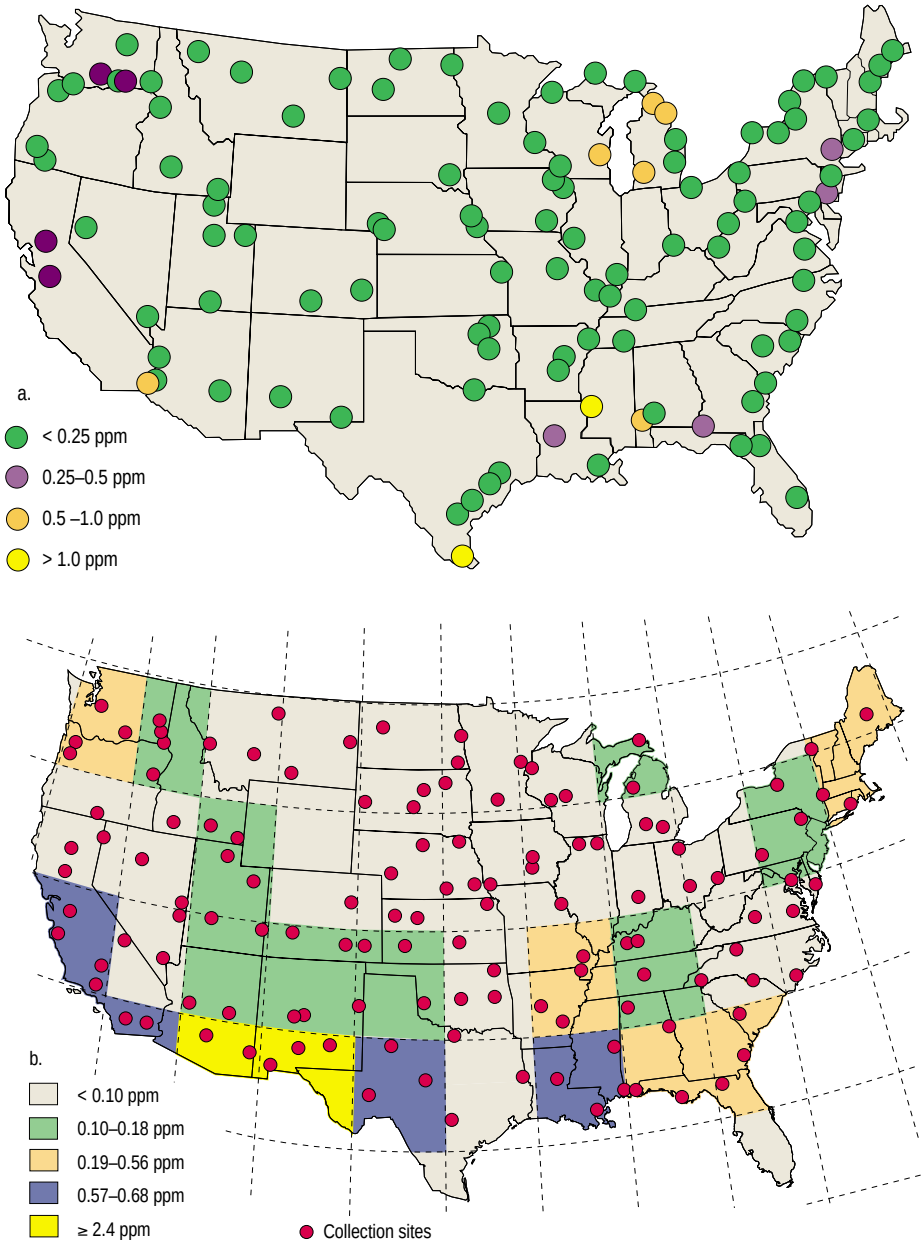


Fig. 14. Geographic distribution of DDE residues in the conterminous United States: a) in freshwater fish collected in 1986 from the indicated sites and b) in European starlings collected in 1985 from the indicated sites. Also shown are boundaries of the 5-degree (latitude and longitude) sampling blocks (dashed lines) and collection sites (red dots; from Schmitt and Bunck 1995).

Endocrine-Disrupting Compounds in the Environment

One of the most recent concerns associated with environmental contaminants is that some of these toxic chemicals may interfere with animal hormones. These compounds are referred to as endocrine disruptors because they disrupt normal functioning of the endocrine system. Many of the chemicals that may pose this threat have been in the environment for a long time (such as DDT), but only recently has this toxic mode of action been identified. The number of observations of aberrant endocrine functions in both human and wildlife populations that could be explained by contaminants working as endocrine disruptors is especially disconcerting (Colborn et al. 1993).

Some of the effects that have been attributed to endocrine disruptors have been dramatic enough to be described in the news media, including male American alligators with underdeveloped sex organs, vitellogenin (an egg and yolk protein normally found only in females) in male animals, and male Florida panthers with undescended testes and abnormal sex hormone ratios (Begley and Glick 1994; Raloff 1994). These examples typify estrogenic effects of endocrine disruptors, xenobiotic chemicals that, because of their chemical structures, can mimic estrogen, a female sex hormone. When the body's hormone receptors recognize the contaminant as estrogen, they respond as they would to the hormone, and the result is feminization of the exposed organism. Some of these effects may be more subtle than physical abnormalities and may manifest themselves as behavioral changes (Fox et al. 1978), such as aberrant behavior of birds during nesting, which can have significant effects on their nesting success.

Endocrine disruptors also may work in ways other than as estrogen mimics. Some compounds are thought to alter the activity of other sex hormones (androgens and progesterone), thyroxine, glucocorticoids, and possibly others. Endocrine disruptors may act not only by mimicking a hormone, as in the case of the estrogens previously described, but may also act as inhibitors (Kelce et al. 1995). Much current evidence points to early development (embryo, fetus, juvenile) as the most sensitive stages for exposure, although the effects on exposed young are often not apparent until an organism reaches sexual maturity, complicating the demonstration of cause and effect (Colborn et al. 1993).

Although some startling observations have been made in wildlife populations, epidemiological data from humans heighten concern about the endocrine disruption theory. There is evidence of a significant decrease in sperm counts in men over the last 50 years (Carlsen et al. 1992), with the exposure to endocrine-disrupting compounds a possible cause. Also, increasingly frequent human cancers over the last 20 years include those of the breast, ovaries, testes, and prostate (Reis et al. 1994), all tissues that are sensitive to sex hormones. One of the confounding factors in the theory of endocrine-disrupting contaminants as responsible for some of these observations is that naturally occurring compounds in some fruits and vegetables can also have estrogenic activity. Some scientists believe the amount of estrogenic activity an individual would receive from these naturally occurring chemicals in the diet is far greater than that from environmental contaminants.

These observations, and the implications of involvement of manufactured chemicals as a cause, prompted a national workshop sponsored by the U.S. Environmental Protection Agency in April 1995. This workshop brought together experts from human and ecological health research to discuss what was known about the potential for endocrine disruption due to environmental exposure, and to suggest what research is most urgently needed. While it appears that there is not enough scientific evidence to adequately address the health risks of endocrine disruptors, a dedicated research effort needs to be undertaken because of the magnitude of the potential impacts and the plausibility of this toxic mode of action.

See end of chapter for references

Author

Michael J. Mac
USGS National Center
Mailstop 300
12201 Sunrise Valley Drive
Reston, Virginia 20192

delta waters of the Gulf of Mexico (Calder and Means 1987). Concentrations also remain slightly elevated in other areas where pesticide use was historically heavy, such as the California Central Valley (Saiki and Schmitt 1986) and the Yakima Valley, Washington (Johnson et al. 1988; Schmitt et al. 1990). Levels of DDT remain highest near sites of its former production and formulation (O'Shea et al. 1980; Calder and Means 1987; U.S. Environmental Protection Agency 1992; Schmitt and Bunck 1995; Turgeon and Robertson 1995).

Toxaphene concentrations also declined (Schmitt et al. 1990; Fig. 12), but residues of this insecticide may still affect biota. Although used most extensively on cotton in the South and Southwest, toxaphene is transported atmospherically to the Great Lakes (Rice and Evans 1984; Rice et al. 1986), where it accumulates in lake trout (Ribick et al. 1981). Toxaphene is a complex mixture of many compounds; it is especially toxic to fish. Although it degrades more rapidly than DDT and many other organochlorine compounds, the most toxic components resist degradation (Harder et al. 1983). Concentrations of these toxic components remain elevated in Great Lakes lake trout (Gooch and Matsamura 1987).

Elevated but declining concentrations of some organochlorine insecticides remain from agricultural use in the Corn Belt. They also persist near urban areas, where compounds such as aldrin, dieldrin, and chlordane were used to control termites; levels are especially high in Hawaii (Schmitt et al. 1990). Like DDT, concentrations of these insecticides also remain highest near sites of former manufacture and

formulation (U.S. Environmental Protection Agency 1992).

Many bird populations are recovering from the effects of eggshell thinning; for example, the U.S. Fish and Wildlife Service has proposed changing the status of the bald eagle from endangered to threatened in most of the conterminous states, the exception being the Southwest (U.S. Fish and Wildlife Service 1994), and herring gull populations are increasing in the Great Lakes (Weseloh et al. 1994). In parts of the Southwest, DDE concentrations remain somewhat elevated (Schmitt and Bunck 1995; Fig. 14), possibly from the continuing use of dicofol, an organochlorine miticide (Table 1) that may contain traces of DDT and DDE (Jarman et al. 1986). Historically, the accumulation of pesticides by migratory birds wintering outside of the United States was a concern (for example, Henny et al. 1982). Recently reported concentrations of DDE in migrating peregrine falcons sampled in southern Texas are low relative to the past (Mora 1995); nevertheless, migratory species are still exposed to organochlorine and other pesticides on their wintering grounds. Concentrations of DDE and other pesticides remain elevated in parts of the Rio Grande Valley (International Boundary and Water Commission 1994), however, and the peregrine falcon population of the trans-Pecos region is declining because of poor reproduction (McKinney 1994). Organochlorine insecticides have been shown to impair bird reproduction through mechanisms other than eggshell thinning in many species (for example, Fry and Toone 1981; Colborn et al. 1993). These insecticides also persist in soils, tending to accumulate in soil invertebrates (for example, Beyer and Gish 1980). Collectively, these findings indicate that organochlorine pesticides remain a threat to U.S. biological diversity.

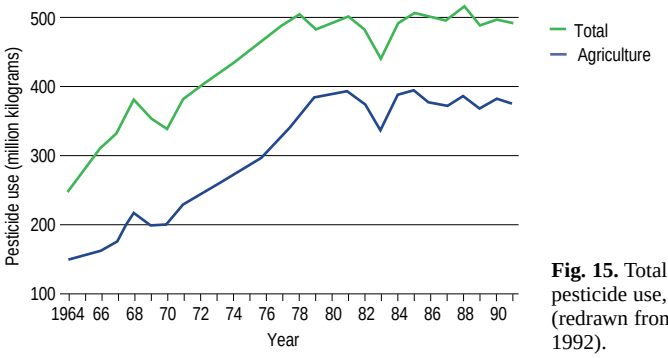


Fig. 15. Total and agricultural U.S. pesticide use, 1964–1991 (redrawn from Aspelin et al. 1992).

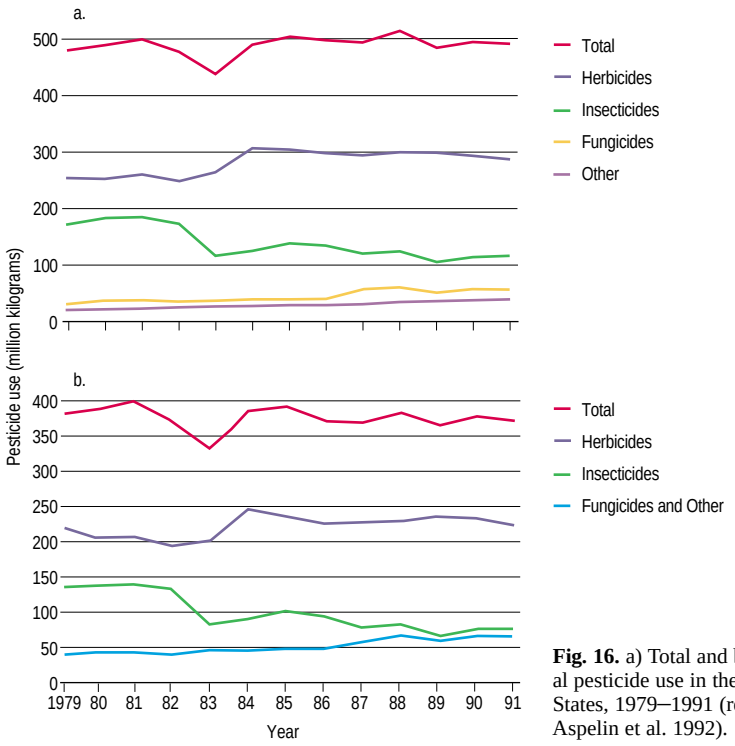


Fig. 16. a) Total and b) agricultural pesticide use in the United States, 1979–1991 (redrawn from Aspelin et al. 1992).

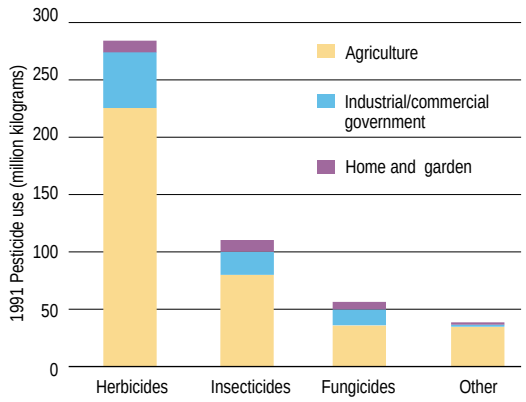


Fig. 17. United States pesticide use in three sectors, 1991 (redrawn from Aspelin et al. 1992).

New-Generation Pesticides: Organophosphate, Carbamate, and Synthetic Pyrethroid Compounds

Organochlorine pesticides began to be replaced in the late 1960's, first with organophosphorus and then carbamate and synthetic pyrethroid compounds. As these so-called soft pesticides, which degrade rapidly and do not accumulate, became more widely used from the mid-1960's through the late 1970's, pesticide use in the United States increased steadily (Fig. 15). Total pesticide use has remained at about 500 million kilograms annually since 1979, but total and agricultural insecticide use declined (Aspelin et al. 1992; Fig. 16). Agriculture accounted for 76%–77% of total United States pesticide use through 1990–1991 (Figs. 16 and 17), with corn and soybeans accounting for 62% of all pesticides used on crops (Gianessi 1986). The counties with the greatest total 1987 expenditures for agricultural chemicals were in the West, Midwest, mid-South, Texas, and Florida (Fig. 18). Highest values were in counties where fresh fruits and vegetables are grown for human consumption, in rice- and cotton-producing areas,

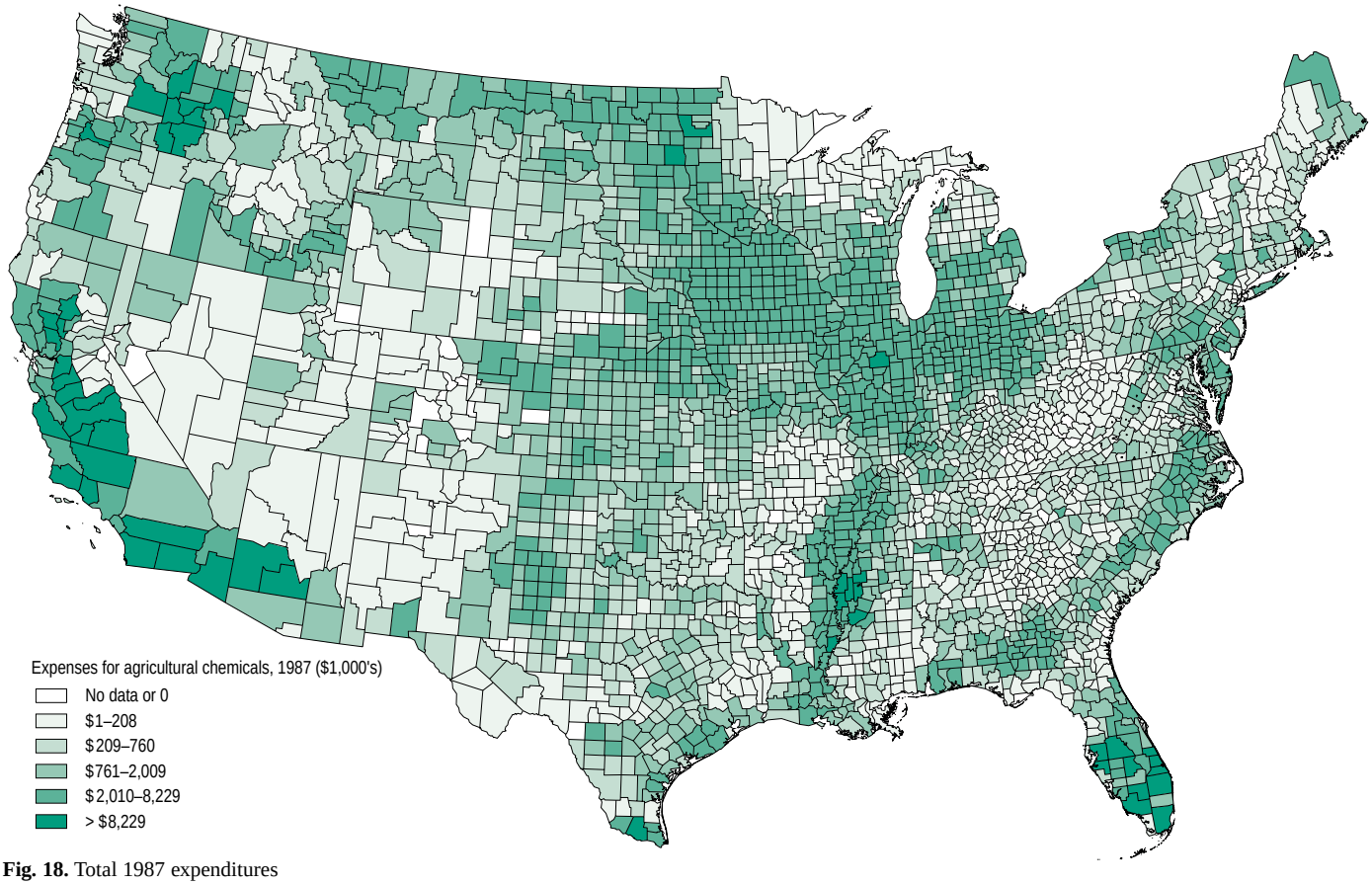


Fig. 18. Total 1987 expenditures for farm chemicals other than fertilizers, by county, in the conterminous United States (from Battaglin and Goolsby 1995).

and in the Corn Belt (Fig. 18). Among soft pesticides, the most heavily used compounds include methyl parathion, butylate, methomyl, carbaryl, terbufos, fonofos, and azinphos-methyl, all organophosphates and carbamates (Smith 1987; Table 1). Carbofuran (Table 1) was also heavily used in agriculture (Smith 1987), but its registration has been canceled (U.S. Environmental Protection Agency 1989).

Although soft pesticides are shorter-lived than organochlorines, do not accumulate, and the total amount in use has declined (Fig. 16), the high toxicity and fast action of many newer pesticides have resulted in increasing incidences of fish and wildlife kills (Glaser 1995).

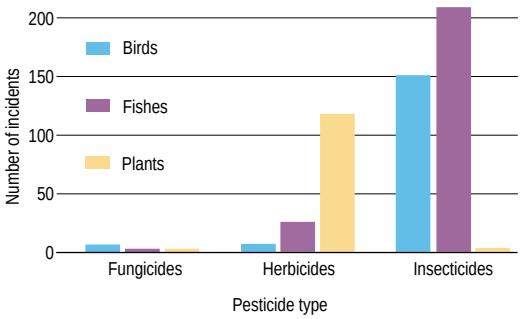


Fig. 19. Reported incidences of kills of birds, fishes, and plants from three classes of pesticides, 1988–1993 (U.S. Environmental Protection Agency, Office of Pesticide Programs, Ecological Effects Branch, unpublished data).

Insecticides account for most of these incidents (Fig. 19). Some 270 species of birds have been represented in more than 500 kills reported from 1988 to 1993. Incidents vary in size from a few to thousands of birds. Songbirds are most often affected; carbamate pesticides have been implicated most frequently as the cause in kills of songbirds and raptors, and organophosphates most often involved in kills of fish and waterfowl (U.S. Environmental Protection Agency, Office of Pesticide Programs, unpublished data). As described earlier for oil spills, exact numbers of organisms affected in pesticide-poisoning incidents are difficult to determine. Incident investigations are neither systematically nor uniformly conducted, and the reports are therefore difficult to interpret (for example, Greig-Smith 1994). Laboratory and field investigations have shown that delayed mortality and altered reproductive and predator-avoidance behavior occur, and losses attributable to these effects are not represented in kill numbers (for example, Mineau and Peakall 1987). Consequently, numbers of dead organisms reported are probably far lower than the true totals. Despite these difficulties, pesticides, primarily from agriculture, were estimated to have caused the deaths of 6–14 million of the 141 million fish that died annually in fish kills from

1977 to 1987, and 672 million birds are directly exposed to high concentrations of pesticides each year in the United States; 67 million of these birds die (Pimentel et al. 1992). Carbofuran was estimated to have killed 1–2 million birds annually in the United States during the early 1980's (U.S. Environmental Protection Agency 1989). Recent declines in some wildlife populations, including the endangered Indiana bat in Missouri (R. Clawson, Missouri Department of Conservation, unpublished 1994 annual survey data), have raised new concerns about currently used agricultural chemicals.

Soft pesticides are highly toxic to terrestrial and aquatic invertebrates, including beneficial predatory insects and those that pollinate plants. Insect losses from pesticide use can affect populations of insectivorous birds, mammals, and other wildlife (for example, Seidel and Whetmore 1995). No estimates of the cumulative effects of pesticides on invertebrate biological diversity or secondary effects on insect-dependent wildlife are available; however, annual U.S. economic losses attributed to the effects of pesticides on insect pollination (including losses of commercial honey bees) and other beneficial insects are valued at $840 million (Pimentel et al. 1992). Recent evidence indicates that new-generation pesticides may affect the homing behavior of honey bees at sublethal concentrations (Vandame et al. 1995); the total losses due to pesticides may therefore be much greater. Soft pesticides are also highly toxic to aquatic invertebrates, including pearlymussels and the larvae of many aquatic insects. A recent kill of endangered tar spinymussels in Nash County, North Carolina, was attributed to soft pesticides (Fleming et al. 1995). Pearlymussel die-offs have been reported with increasing frequency since the 1980's throughout the United States (Neves 1987), but the involvement of pesticides or other contaminants in these incidents has not been fully investigated. In general, exposure of organisms to soft pesticides is difficult to gauge because most compounds neither persist nor accumulate.

Herbicides

Stimulated by high fuel costs, chemical weed control became increasingly common in United States agriculture during the 1970's. Consequently, much of the increase in United States agricultural pesticide use since the 1960's is attributable to agricultural herbicide increases (Figs. 15, 16, and 17). Corn (45%), soybeans (18%), pasture (6%), cotton (6%), sorghum (5%), wheat (4%), and rice (3%) account for most U.S. agricultural herbicide use (Gianessi and Puffer 1991). Atrazine, alachlor, metolachlor, trifluralin, cyanazine, metribuzin, glyphosate, and 2,4-D (Table 1) are the most heavily used compounds (Gianessi and Puffer 1991; Aspelin et al. 1992). Herbicide use is heaviest in the Midwest, South, and mid-Atlantic regions, and in Florida, California, and Oregon (Battaglin and Goolsby 1995). Until recently, herbicides and defoliants containing arsenic were used heavily in orchards and in cotton production; consequently, arsenic concentrations in soils and in waters draining extensively treated areas can remain elevated (Sheets 1980). Arsenical herbicides remain registered for use in cotton and for weed control in turf, including lawns.

Although there are exceptions (for example, Eisler 1990; Fig. 19), most currently used herbicides do not accumulate in biota nor are they acutely toxic to animals at environmental concentrations (Hoffman 1990, 1994). Herbicides are highly toxic to nontarget terrestrial and aquatic plants (Kemp et al. 1983, 1985; Cunningham et al. 1985), however, which has raised new concerns. Because of their widespread use—and for some compounds, persistence—herbicide contamination of surface and groundwaters in agricultural areas has become common (for example, Paulson et al. 1993). Atrazine is heavily used in the Midwest and in the Chesapeake Bay watershed for weed control in corn (Fig. 20). Some 235,000 kilograms of atrazine were estimated to have been exported from the Mississippi River to the Gulf of Mexico in 3 months during 1991 (Goolsby et al. 1991). The effects of chronic herbicide pollution on aquatic vegetation in the Midwest (where concentrations are highest overall) and in the Gulf of Mexico have not been investigated. In Chesapeake Bay, atrazine and other herbicides were estimated to have contributed, along with nutrient enrichment and sedimentation, to the decline of submerged aquatic vegetation (Kemp et al. 1983; Sanders 1987). Aquatic vegetation has recovered somewhat in the brackish regions of Chesapeake Bay since the early 1980's but not in the freshwater areas of the upper bay (Pendleton 1995). The involvement of herbicides in declines of aquatic vegetation elsewhere has received less study.

A recent, comprehensive ecological risk assessment of atrazine revealed that atrazine application rates have declined somewhat over the last decade, and that concentrations in surface waters are not sufficiently high enough to represent a significant risk except in small reservoirs and streams receiving agricultural runoff (Solomon et al. 1996). Nevertheless, the long-term, cumulative risks of herbicide pollution have not been fully assessed. The distributions of aquatic and terrestrial animals are highly dependent on plant distributions (for example, Chick and McIvor 1994). Consequently,

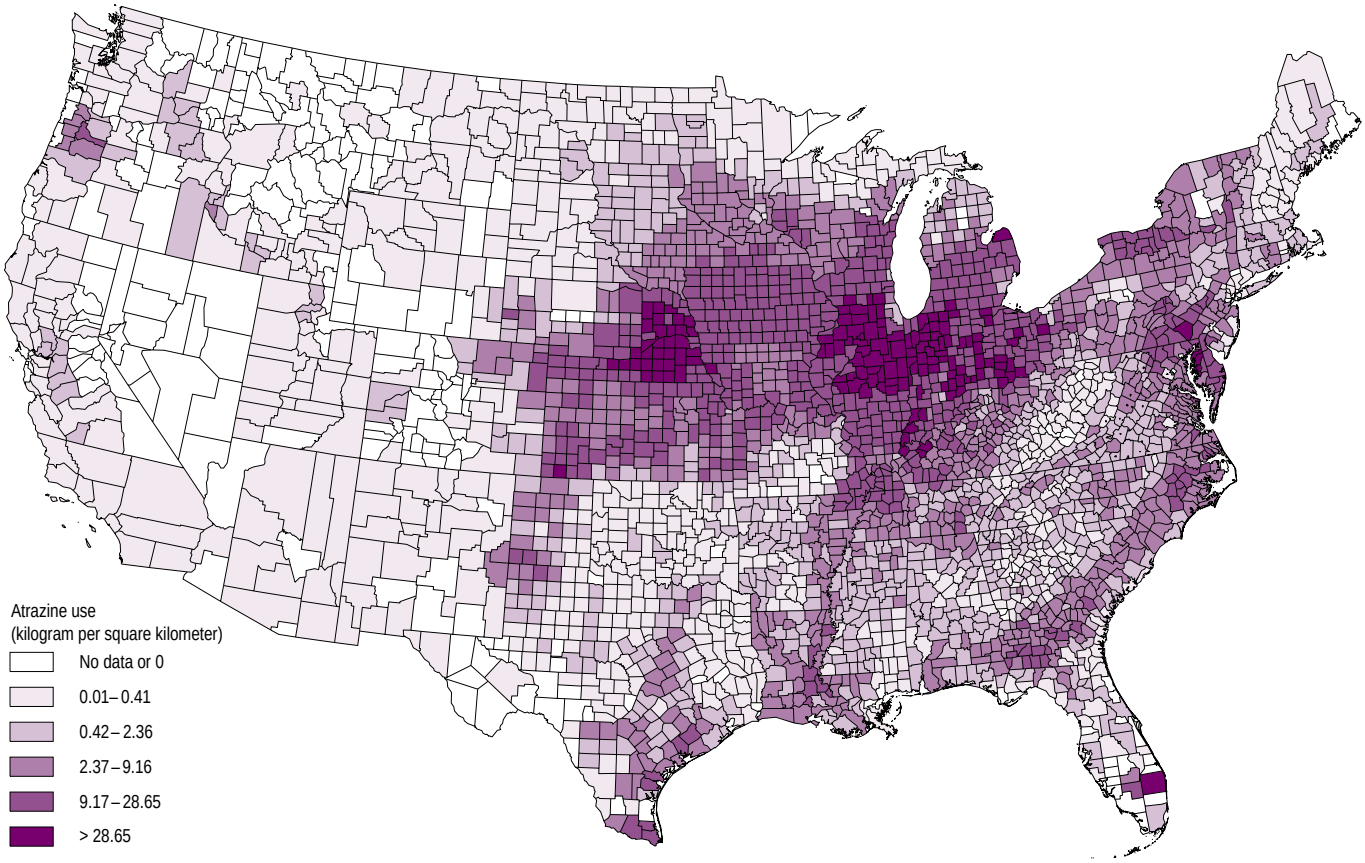


Fig. 20. Use of atrazine, by county, in the conterminous United States in 1987 (from Battaglin and Goolsby 1995).

chronic, low-level herbicide pollution may alter the structure of plant communities, which, in turn, can influence the distribution of animals (for example, Cooke 1977). Because the use of herbicides at current levels is unprecedented, long-term effects on nontarget vegetation and secondary effects on animal populations are unknown.

Fig. 21. Aerial photograph of Bridgeport, Connecticut, an urban–industrial estuary.

Industrial and Urban Development

Many of the most environmentally significant contaminants that emanate from industries and population centers have already been discussed in the context of other human activities. These include contaminants associated with mineral and energy exploitation: lead, mercury, other metals, arsenic, and selenium—all from industrial and municipal discharges, fossil fuel combustion, and solid waste disposal (that is, landfills and incinerators); PAH's and other organic compounds from fuels, lubricants, solvents, and industrial processes; combustion gases (SO_2, NO_x, CO, CO_2) from domestic, industrial, and automotive fuel consumption; nutrients from wastewater treatment plants and urban runoff; and pesticides from manufacturing and formulation facilities and from runoff in urban and suburban areas (Fig. 21). Certain industries, including those already noted—pesticide manufacturing and formulation (pesticides and precursors), petrochemicals and petroleum refining (numerous industrial and commercial chemicals, PAH's and related compounds, metals), chlor-alkali production (mercury), and base and precious metal reduction and manufacturing (metals, PAH's, PCB's)—

were historically responsible for the release of large quantities of contaminants to the environment. The forest products industry, which includes pulp-making, paper-making, and wood-treatment facilities, released PCB's, dioxins, mercury, wood preservatives including creosote and pentachlorophenol (Table 1), and other toxic by-products at numerous locations throughout North America.

Relatively few of the thousands of industrial and consumer chemicals manufactured and released into the environment have been thoroughly tested and evaluated. Of those that have, even fewer (other than the contaminants already discussed) have been found to be toxic at environmental concentrations. Among toxic industrial chemicals, the industrial organochlorine compounds have been the most injurious to biota. These chemicals are structurally similar to organochlorine pesticides, with which they also share many toxicological and environmental fate properties. This group includes PCB's, chlorinated dioxins, and other structurally similar compounds (Table 1), some of which bioaccumulate and are highly toxic to fishes and wildlife. The group also includes chlorofluorocarbons (CFC's) and volatile organic compounds (VOC's)—air pollutants involved in global climate change and thinning of the Earth's protective ozone layer (Table 1).

Polychlorinated Biphenyls, Dioxins, and Dibenzofurans

Polychlorinated biphenyls (PCB's), dioxins, and dibenzofurans are families made up of many structurally similar compounds that vary greatly in toxicity and persistence. Among them are some of the most toxic and persistent chemicals yet investigated (for example, Safe 1990). Chlorinated dioxins were first discovered as toxic by-products of chlorophenols, which are feedstocks for the synthesis of many products (U.S. Environmental Protection Agency 1992; Table 1). Early formulations of the wood preservative pentachlorophenol and of the herbicide 2,4,5-T (Table 1) were also contaminated with chlorinated dioxins; wood treatment and herbicide manufacturing facilities synthesizing, formulating, or using these materials were significant sources of these compounds to the environment (U.S. Environmental Protection Agency 1992). Chlorinated dioxins were also produced by bleaching processes used historically in paper manufacturing and are released into the atmosphere from many combustion sources including municipal waste incinerators and cigarette smoke (Muto and Takizawa 1989; U.S. Environmental Protection Agency 1992).

Trace quantities of chlorinated dioxins occur widely in North America because of their persistence and diversity of sources. Concentrations tend to be highest in waters that received direct discharges from point sources, such as sites of herbicide and hexachlorophene synthesis and formulation. Examples include the Passaic River and Newark Bay, New Jersey; the Kanawha River, West Virginia; Bayou Meto, Arkansas; the Brazos River, Texas; and tributaries of Saginaw Bay, Michigan. Concentrations are also elevated in waters that received effluents from bleached kraft paper mills, which are widely distributed throughout North America (Kuehl et al. 1989; U.S. Environmental Protection Agency 1992).

Relatively small amounts of chlorinated dioxins and dibenzofurans occur in the environment as impurities and by-products of other substances. In contrast, large quantities of PCB's were manufactured for use in many applications (Table 1). First synthesized in 1881, some 635 million kilograms were produced in the United States alone from the beginning of commercial production in 1929 until the use, manufacture, and distribution of PCB's were discontinued in 1979 (U.S. Environmental Protection Agency 1979). Because of their longevity and widespread use in consumer products, contamination by PCB's is widespread, and concentrations tend to be elevated near all human population centers (Schmitt et al. 1990; U.S. Environmental Protection Agency 1992). Concentrations are generally highest near former point sources, primarily facilities that manufactured electrical and hydraulic equipment, paper mills that either produced or recycled carbonless copy paper, and metallurgical facilities, including foundries. Such point sources initially contaminated many waterways, including the Housatonic, Hudson, St. Lawrence, and Ohio rivers; New Bedford Harbor, Massachusetts; Waukeegan Harbor and Green Bay on Lake Michigan; Saginaw Bay on Lake Huron; and Lake Ontario.

Despite being manufactured and distributed since 1929, PCB's were not recognized as environmental contaminants until the 1960's, when they were discovered in fish and wildlife from the Baltic Sea (Jensen 1966). Soon after, PCB's were found to have accumulated in Lake Michigan coho salmon (Johnson and Pecor 1969), which caused birth defects and early kit mortality in ranch mink raised on salmon (Aulerich et al. 1971; Aulerich and Ringer 1977). Subsequently, PCB's and chlorinated dioxins have been among the most intensively and extensively investigated pollutants. Because some dioxins and PCB's are highly toxic and bioaccumulate, they are especially hazardous to predatory fishes and fish-eating birds and mammals. Significant ecological effects attributable to the reproductive toxicity of these and related

compounds have either been documented or are suspected to have occurred in fishes, reptiles, mammals, and birds, especially in the Great Lakes basin (Colborn 1991). Affected bird populations in the Great Lakes basin include the endangered Forster's terns of Green Bay, Wisconsin, in which reduced reproductive success and a high rate of deformities in hatchlings have been documented (Kubiak et al. 1989; Ankley et al. 1993); double-crested cormorants, in which hatching success is negatively correlated with dioxinlike activity of PCB's in eggs (Fig. 22); and bald eagles, in which coastal populations that eat fish with access to the Great Lakes reproduce less successfully than inland populations (Colborn 1991; also see chapter on Great Lakes). Reproductive impairment or population declines attributable to organochlorine chemicals—including PCB's and dioxins—have also been documented in Great Lakes populations of lake trout, coho and chinook salmon, snapping turtles, black-crowned night-herons, Caspian terns, common terms, herring gulls, ring-billed gulls, ospreys, mink, and river otters (Johnson and Pecor 1969; Mineau et al. 1984; Colborn 1991; Mac and Edsall 1991; Spitsbergen et al. 1991; Tillitt et al. 1991a,b, 1992; Wren 1991; Mac et al. 1993). The white whale population of the Saguenay–St. Lawrence estuary has also been affected (Colborn 1991). Elsewhere, PCB's were suspected of having caused a precipitous decline in the abundance of mink in the Upper Mississippi River corridor during the 1960's, from which the population has since partially recovered (Wiener et al. 1995). Dioxins and PCB's are suspected of inhibiting reproduction in the bald eagle population nesting along the Hood Canal, Washington (U.S. Fish and Wildlife Service, Division of Environmental Contaminants, unpublished data). Delayed egg maturation and reduced reproductive success in the starry flounder population of San Francisco Bay have also been attributed to PCB's (Spies and Rice 1988). Additionally, dioxins and PCB's are among the contaminants implicated in recent die-offs of bottlenose dolphins on the Atlantic and gulf coasts (Kuehl et al. 1991; Hutchinson and Simmonds 1994); the mid-Atlantic population has been declining over the last decade (Kuehl et al. 1991).

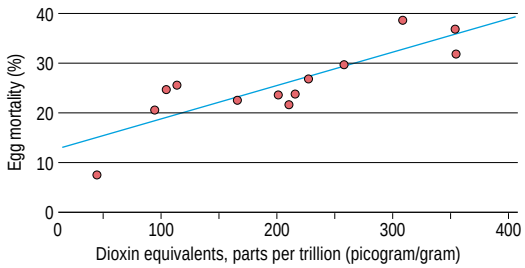


Fig. 22. Egg mortality in Great Lakes colonies of double-crested cormorants versus PCB's in eggs as dioxin-equivalent concentrations, 1987–1988 (Tillitt et al. 1991b); each point represents the mean for a colony. A similar relation between dioxin equivalents and hatching success has been demonstrated for United States bald eagles (D. E. Tillitt, U.S. Geological Survey, unpublished data; Tillitt et al. 1991a).

Restrictions on the sale and use of PCB's since the mid-1970's have resulted in reduced PCB concentrations in biota. Nationwide, PCB concentrations declined in freshwater fishes (Fig. 12) and European starlings through the mid-1980's (Bunck et al. 1987; Schmitt et al. 1990; Schmitt and Bunck 1995). Substantial declines occurred in heavily contaminated areas such as the Great Lakes and the Hudson and Ohio rivers (Mineau et al. 1984; St. Amant et al. 1984; Baumann and Whittle 1988; Hesselberg et al. 1990; Schmitt et al. 1990). Residues of PCB's nevertheless remain widespread because of their persistence and dispersion in water and air. Concentrations remain highest in the industrialized areas of the Northeast and Midwest (Fig. 23). The sediments of heavily contaminated waterways, which can be redistributed by natural processes as well as by human activities such as dredging and dredge spoil disposal, remain a significant and continuing source of PCB's, dioxins, and other contaminants to the environment, as do PCB's in consumer products either still in circulation or deposited in landfills. In addition, the toxicity of the PCB's, dioxins, and related compounds remaining in the environment may not be declining because the most toxic components among them may be selectively accumulated and resistant to degradation (for example, Kuehl et al. 1987; Tanabe et al. 1987; Kannan et al. 1989; Smith et al. 1990; Tillitt et al. 1992). Therefore, upper-level predators in marine and aquatic food chains—predatory fishes, fish-eating birds, and marine mammals—remain at risk. Because of the great difficulty and expense of analyzing dioxins or individual PCB's, there are no U.S. trend data for them.

Chlorofluorocarbons and Volatile Organic Compounds

Chlorofluorocarbons (CFC's) and volatile organic compounds (VOC's) are pollutants that are not generally perceived as directly toxic to plants or animals at environmental concentrations. Rather, CFC's and VOC's have the potential to affect biological diversity indirectly by altering the composition of the atmosphere. The VOC's have been used widely in industrial and

consumer applications (Table 1). Chloroform, one of the most widespread VOC's, can also be produced by chlorination of water and sewage. The VOC's are common groundwater pollutants, but concentrations in surface waters are generally low (Schwartzenbach and Giger 1985). The CFC's (Table 1) comprise widely used gases (Cumberland et al. 1982). In the troposphere (lower atmosphere), CFC's and VOC's contribute to the greenhouse effect and global warming. Through a complex series of chemical reactions, they also contribute to smog and ozone production, which damage vegetation.

Total CFC emissions have declined since the mid-1970's, primarily because of restrictions on their use in open-cell foams and as propellants; however, CFC releases from other applications, including refrigeration, continued to increase through the early 1990's (Boden et al. 1994). The long-term fate of CFC's and their breakdown products, which accumulate in the stratosphere (upper atmosphere), is unknown. In the stratosphere, CFC's, VOC's, and their degradation products contribute to ozone depletion, which enhances the penetration of damaging ultraviolet solar radiation (that is, UV-B). The DNA of plants and animals can be damaged by UV-B (van der Leun and de Gruijl 1993), which also interferes with the immune function in animals (Fabacher et al. 1994). The ecological risks associated with UV-B exposure vary for two primary reasons: because species differ in their sensitivity (for example, Little and Fabacher 1994), and because the distribution of UV-B is not geographically uniform (Madronich 1993). Aquatic and marine organisms that congregate near the water surface, as well as high-elevation plants and animals, are particularly vulnerable to UV-B. Among sensitive high-elevation organisms is the Lahontan cutthroat trout, a threatened species (Little and Fabacher 1994). Recent declines in California populations of western toads and Cascades frogs have been attributed to the inability of these species to repair UV-B-induced DNA damage (Blaustein et al. 1994).

Current stratospheric concentrations of CFC's and related compounds are the highest ever recorded (Anderson et al. 1991), and ozone depletion, formerly observed only over Antarctica, now also occurs periodically over North America (Kerr and McElroy 1993). The distribution and abundance of many organisms, including those most responsible for the primary and secondary productivity of the oceans, may currently be limited by UV-B irradiance (Calkins 1982; Häder 1993). If so, forecasts of UV-B increases in North America as a consequence of stratospheric ozone depletion will influence biological diversity over a wide area.

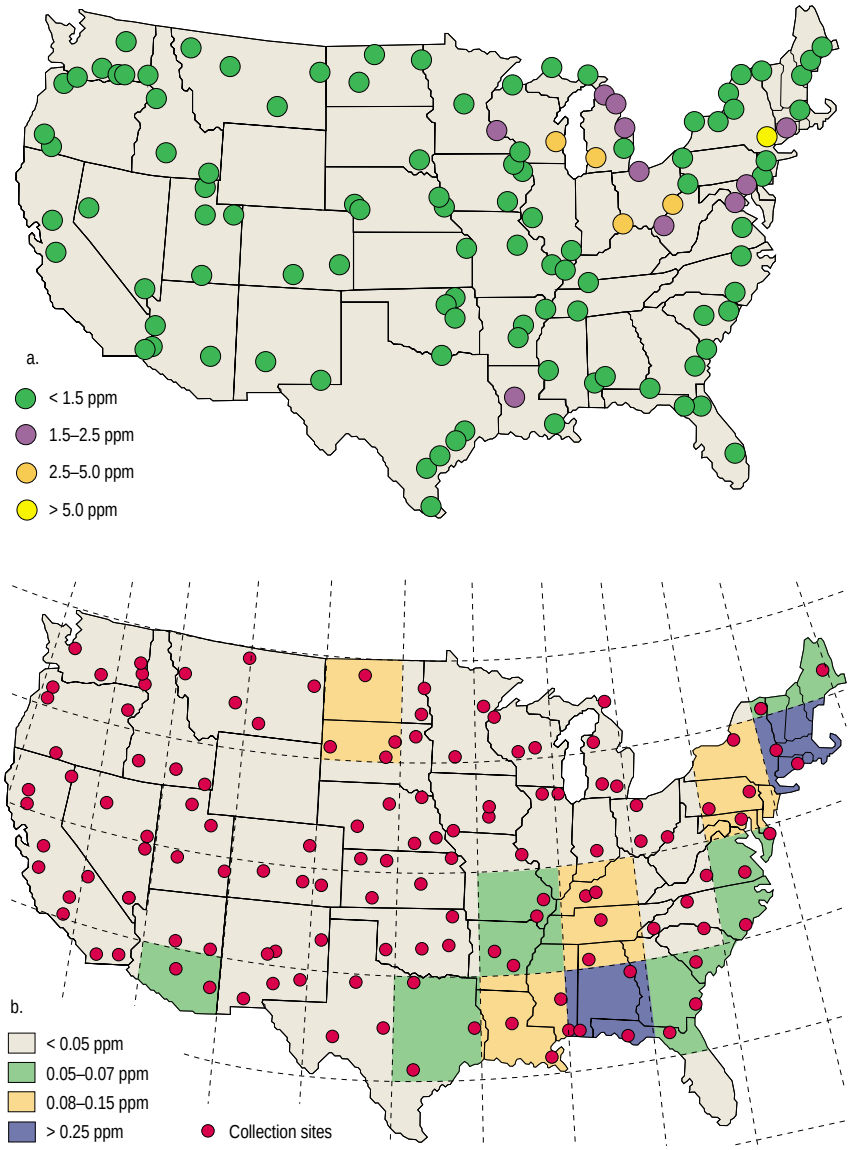


Fig. 23. Geographic distribution of PCB residues in the conterminous United States: a) in freshwater fishes collected in 1986 from the indicated sites and b) in European starlings collected in 1985 from the indicated sites. Also shown are boundaries of the 5-degree (latitude and longitude) sampling blocks (dashed lines) and collection sites (red dots; from Schmitt and Bunck 1995).

Summary, Conclusions, and Information Needs

Environmental contaminants are generally perceived as a twentieth-century problem. To the contrary, some of the most significant contaminants are biogenic (for example, petroleum), are natural constituents of the Earth's crust (arsenic, selenium, and metals), or result from the combustion of biogenic materials (PAH's). Accumulation to harmful concentrations may result naturally but occurs primarily when human activities such as agriculture, mining, logging, and dredging accelerate rates of natural processes. Once released into the environment, contaminants are distributed with water, air, soil, and sediment—the same

processes and media that govern the distribution of nutrients and other substances. Consequently, contaminants may affect biota long distances from their sources. Many of our most serious contaminant problems originated from the application of old-world mineral and energy extraction technologies at unprecedented rates. Subsequent problems developed as unforeseen consequences of accelerated energy and mineral extraction and of the industries, technologies, and human population centers they engendered. Many of these processes and problems predated environmental concerns and the technology and scientific knowledge base necessary to detect and interpret them, and their effects may have been masked by other natural and anthropogenic factors.

The comparatively recent regulation of toxic pollutants has lowered United States concentrations of many of these pollutants to the lowest levels since measurements began. Regulation of surface and subsurface mining and of direct discharges of pollutants to waterways and to the atmosphere, and the replacement of most bioaccumulative toxins with environmentally kinder alternatives have collectively reduced inputs of many pollutants to North American ecosystems. Concentrations of many toxins have declined accordingly. Reclamation has reduced the extent and magnitude of acid mine drainage from historically and actively mined lands, but whether effects on biota have been successfully mitigated has not been determined. Problems related to regulated contaminants persist, but they are primarily associated with historically contaminated sites, the atmospheric and waterborne redistribution of pollutants already released into the environment, and continuing use of these products outside North America. With the reductions in emissions, the sediments of industrialized waterways and the atmosphere have become important contaminant sources. The continuing discovery of potentially significant ecological effects attributable to regulated contaminants at environmental concentrations previously thought safe implies that further reductions may be warranted. With traditional contaminant sources regulated, future reductions in environmental concentrations will have to result from the control of more diffuse sources. These include agricultural soil erosion, urban runoff, and the atmospheric and waterborne transport of contaminants from artificial and natural sinks—industrial and mining waste disposal sites, landfills, and contaminated sediments.

In eastern North America, there is some evidence that recent restrictions on SO_2 emissions can reduce the acidity of surface waters (Lettenmaier et al. 1991; Clair and Ehrman 1995). Nevertheless, recent global increases in fossil fuel consumption increased atmospheric CO_2 concentrations by 12.8% from 1959 to 1992 (Boden et al. 1994), much of which was attributable to coal and oil combustion. Because fossil fuel combustion also releases mercury, selenium, and other elements into the atmosphere, it may be a source of the increasing concentrations of these elements observed in some parts of North America over the last decade. North American and worldwide electric power generation is expected to increase. Total emissions of toxic elements, PAH's, and SO_2 could increase correspondingly, offsetting anticipated future decreases at individual sources. Similarly, expected increases in petroleum consumption, especially in the transportation sector (Greene and Santini 1993), are expected to increase total pollutant emissions despite reductions from individual sources. Thus, increases in NO_x emissions from transportation sources could partly offset gains in rainfall pH made by reducing SO_2 emissions from stationary sources such as power plants. In addition, global economics determine the rate at which domestic energy supplies are exploited and, consequently, the rate at which energy-associated pollutants are released into North America. At present, U.S. oil demand is met largely by foreign sources. Should economic and political factors favor the large-scale exploitation of domestic energy sources such as oil shale and coal-based fuels, large quantities of toxins would be released. Mineral and energy exploitation effects would most likely occur in those parts of the West where biological diversity is already limited by water quality and quantity.

Environmental toxicology is a relatively new field that has focused primarily on the study of individual contaminants and on the distribution and abundance of toxins. Far less is known about the cumulative effects that the many chemicals released into the environment have had on biota, especially against the background of other human-induced perturbations. Although the need to link toxicology and population biology has been recognized (for example, Kendall and Lacher 1994), relatively few investigations of long-term effects of contaminants on populations have been undertaken. At the population level, evaluation of chemical or other effects requires information on the extent and magnitude of the stresses and losses to which populations are subjected and the size and status of the populations (for example, Pulliam 1994). This latter information is largely unknown for most species. Still more difficult to evaluate are sublethal effects on such aspects of species biology as reproductive and migratory behavior, which can be affected by pesticides and other contaminants at low concentrations. In addition, many new-generation pesticides are

short-lived and do not accumulate, making it difficult to document exposure. Collectively, these difficulties confound efforts to estimate the extent and population-level significance of losses from contaminants. The lack of this information is the greatest impediment to a full understanding of the significance of chemical exposure; that is, considerably more is known about chemicals and what they can do than about what they have actually done (Gilbertson and Schneider 1991). And finally, the advent of widespread contamination by synthetic chemicals is recent and unprecedented. These chemicals therefore represent a source of selective pressure to which long-lived organisms and ecosystems may not have adapted. Long-term study will be necessary to discriminate the effects of contaminants from those of other environmental stresses and natural variability if problems are to be foreseen and remedied before irreversible ecological harm occurs.

Acknowledgments

I thank R. Kendall and the graduate students of The Institute of Wildlife and Environmental Toxicology, Clemson University, and two anonymous reviewers for comments on an earlier version of the manuscript; E. Callahan, P. Greene, M. Laustrup, and J. Thomas for assistance with illustrations; and L. Jacobs for library and information services. I also thank P. Baumann, C. Brussard, D. Chapman, S. Finger, T. Haines, D. Hardesty, J. Layzer, G. Lear, D. Lemly, E. Little, B. McKinney, H. Ohlendorf, M. Saiki, D. Tillitt, and Normandeau Associates, Inc. for contributing maps, information, and photographs.

Author

Christopher J. Schmitt
U.S. Geological Survey
Biological Resources Division
Environmental and Contaminants
Research Center
4200 New Haven Road
Columbia, Missouri 65201

Cited References

Adams, L., M. G. Hanavan, N. W. Hosley, and D. W. Johnston. 1949. The effects on fish, birds, and mammals of DDT used in the control of forest insects in Idaho and Wyoming. Journal of Wildlife Management 13:245–254.

Albers, P. H. 1977. Effects of external applications of fuel oil on hatchability of mallard eggs. Pages 158–167 *in* D. A. Wolfe, editor. Fate and effects of petroleum hydrocarbons in marine organisms and ecosystems. Pergamon Press, New York.

Amhad, M. U. 1974. Coal mining and its effects on water quality. Pages 49–56 *in* R. A. Deju, editor. Extraction of minerals and energy: today's dilemmas. Ann Arbor Science, Mich.

Anderson, J. G., D. W. Tooney, and W. H. Brune. 1991. Free radicals within the Antarctic vortex: the role of CFC's in Antarctic ozone loss. Science 251:39–46.

Anderson, R. B., and W. H. Everhart. 1966. Concentrations of DDT in landlocked salmon (*Salmo salar*) at Sebago Lake, Maine. Transactions of the American Fisheries Society 95:160–164.

Andreasen, J. K., and R. W. Spears. 1983. Toxicity of Texan petroleum well brine to the sheepshead minnow (*Cyprinodon variegatus*), a common estuarine fish. Bulletin of Environmental Contamination and Toxicology 30:277–283.

Andrilenas, P. 1974. Farmers' use of pesticides in 1971—quantities. U.S. Department of Agriculture, Economic Research Service, Agricultural Economics Report 252. 56 pp.

Ankley, G. T., G. J. Niemi, K. B. Lodge, H. J. Harris, D. L. Beaver, P. D. Jones, J. P. Giesy, D. E. Tillitt, T. R. Schwartz, and C. Hagley. 1993. Bioaccumulation of planar, polychlorinated dibenzofurans and dibenzo-*p*-dioxins by birds nesting in the lower Fox River and Green Bay, Wisconsin, USA. Archives of Environmental Contamination and Toxicology 24:332–334.

Appalachian Regional Commission. 1969. Acid mine drainage in Appalachia. U.S. Congressional House Document 91-180. Volumes 1–3. Appendix C.

Armstrong, H. W., K. Fucik, J. W. Anderson, and J. M. Neff. 1979. Effects of oilfield brine effluent on sediments and benthic organisms in Trinity Bay, Texas. Marine Environmental Research 2:55–69.

Aspelin, A. L., A. H. Grube, and R. Toria. 1992. Pesticide industry sales and usage, 1990 and 1991 market estimates. Pesticides and Toxic Substances (H.7503W), U.S. Environmental Protection Agency, Office of Pesticide Programs, Biological and Economic Analysis Division, Washington, D.C.

Aulerich, R. J., and R. K. Ringer. 1977. Current status of PCB toxicity to mink and effect on their reproduction. Archives of Environmental Contamination and Toxicology 6:279–292.

Aulerich, R. J., R. K. Ringer, H. L. Seagran, and W. G. Youatt. 1971. Effects of feeding coho salmon and other Great Lakes fish on mink reproduction. Canadian Journal of Zoology 49:611–616.

Baker, D. H., Jr., and R. B. Bhappu. 1974. Specific problems associated with the processing of minerals. Pages 77–94 *in* R. A. Deju, editor. Extraction of minerals and energy: today's dilemmas. Ann Arbor Science, Mich.

Baker, J., and C. Schofield. 1980. Aluminum toxicity to fish as related to acid precipitation and Adirondack surface water quality. Pages 292–293 *in* D. Drablos and A. Tollan, editors. Acid precipitation—effects on forest and fish project. Proceedings of the International Conference on the Ecological Impacts of Acid Precipitation, Aas, Norway.

Baker, J. P. 1991a. Biological effects of changes in surface water acid-base chemistry. Pages 119–123 *in* P. M. Irving, editor. Acid deposition: state of science and technology. Report 13. Summary report of the National Acid Precipitation Assessment Program, Washington, D.C.

Baker, L. A. 1991b. Current status of surface water acid-base chemistry. Pages 89–101 *in* P. M. Irving, editor. Acid deposition: state of science and technology. Report 9. Summary report of the National Acid Precipitation Assessment Program, Washington, D.C.

Barnard, J. E., and A. A. Lucier. 1991. Changes in forest health and productivity in the United States and Canada. Pages 135–138 *in* P. M. Irving, editor. Acid deposition: state of science and technology. Report 16. Summary report of the National Acid Precipitation Assessment Program, Washington, D.C.

Barton, P. 1978. The acid mine drainage. Pages 313–358 *in* J. O. Nriagu, editor. Sulfur in the environment. John Wiley & Sons, New York.

Battaglin, W. A., and D. A. Goolsby. 1995. Spatial data in geographic information system format on agricultural chemical use, land use, and cropping practices in the United States. U.S. Geological Survey, Water-Resources Investigations Report 94-4176. Denver, Colo. 87 pp.

Baumann, P. C. 1989. PAH, metabolites, and neoplasia in feral fish populations. Pages 269–289 *in* U. Varanasi, editor. Metabolism of polycyclic aromatic hydrocarbons in the aquatic environment. Chemical Rubber Company Press, Boca Raton, Fla.

Baumann, P. C., and R. B. Gillespie. 1986. Selenium bioaccumulation in gonads of largemouth bass and bluegill from three power plant cooling reservoirs. Environmental Toxicology and Chemistry 5:695–701.

Baumann, P. C., J. C. Harshbarger, and K. J. Hartman. 1990. Relationship between liver tumors and age in brown bullhead populations from two Lake Erie tributaries. Science of the Total Environment 94:71–87.

Baumann, P. C., and T. W. May. 1984. Selenium residues in fish from inland waters of the United States. Pages 7–1 to 7–16 *in* Workshop proceedings: the effects of trace elements on aquatic ecosystems. Electric Power Research Institute, Palo Alto, Calif.

Baumann, P. C., and D. M. Whittle. 1988. The status of selected organics in the Laurentian Great Lakes: an overview of DDT, PCB's, dioxins, furans, and aromatic hydrocarbons. Aquatic Toxicology 11:241–257.

Bencala, K. E., D. M. McNight, and G. W. Zellwager. 1987. Evaluation of natural tracers in an acidic and metal-rich stream. Water Resources Research 23:827.

Bender, M. E., W. J. Hargis, Jr., R. J. Huggett, and M. H. Roberts. 1988. Effects of polynuclear aromatic hydrocarbons on fishes and shellfish: an overview of research in Virginia. Marine Environmental Research 24:237–241.

Beyer, W. N., and C. D. Gish. 1980. Persistence in earthworms and potential hazards to birds of soil applied DDT, dieldrin, and heptachlor. Journal of Applied Ecology 17:295–307.

Biglane, K. E., and R. A. Lafleur. 1967. Notes on estuarine pollution with emphasis on the Louisiana gulf coast. Pages 690–692 *in* G. H. Lauff, editor. Estuaries. American Association for the Advancement of Science Publication 83, Washington, D.C.

Black, J. J., and P. C. Baumann. 1991. Carcinogens and cancers in freshwater fishes. Environmental Health Perspectives 90:27–33.

Blancher, P. J. 1991. Acidification: implications for wildlife. Transactions of the North American Wildlife and Natural Resources Conference 56:195–204.

Blaustein, A. R., P. D. Hoffman, D. G. Hokit, J. M. Kesecker, S. S. Walls, and J. B. Hays. 1994. UV repair and resistance to solar UV-B in amphibian eggs: a link to population declines? Proceedings of the U.S. National Academy of Sciences 91:1791–1795.

Boden, T. A., D. P. Kaiser, R. J. Sepanski, and F. W. Stoss. 1994. Trends '93: a compendium of data on global change. Carbon Dioxide Information Analysis Center (CDIAC), Oak Ridge National Laboratory, Oak Ridge, Tenn. CDIAC Communications 20:8–10.

Bradford, D. F., M. S. Gordon, D. F. Johnson, R. D. Andrews, and W. B. Jennings. 1994. Acidic deposition as an unlikely cause for amphibian population declines in the Sierra Nevadas, California. Biological Conservation 69:155–161.

Buhl, K. J., and S. J. Hamilton. 1990. Comparative toxicology of inorganic contaminants released by placer mining to early life stages of salmonids. Ecotoxicology and Environmental Safety 20:325–342.

Bunck, C. M., R. M. Prouty, and A. J. Krynitsky. 1987. Residues of organochlorine pesticides and polychlorinated biphenyls in starlings (*Sturnus vulgaris*) from the continental United States, 1982. Environmental Monitoring and Assessment 8:59–75.

Burdick, G. E., E. J. Harris, H. J. Dean, T. M. Walker, J. Skea, and D. Colby. 1964. The accumulation of DDT in lake trout and the effect on reproduction. Transactions of the American Fisheries Society 93:127–136.

Calder, J. A., and J. C. Means. 1987. Summary. Pages 73–75 *in* Progress report and preliminary assessment of findings of the Benthic Surveillance Project—1984. National Status and Trends Program for Marine Environmental Quality. National Oceanic and Atmospheric Administration, National Ocean Service, Office of Oceanography and Marine Assessment, Rockville, Md.

Calkins, J., editor. 1982. The role of solar ultraviolet radiation in the marine environment. Plenum Press, New York. 724 pp.

Carline, R. F., and G. J. Jones. 1993. Assessment of aquatic animal communities in the vicinity of the Palmerton, Pennsylvania zinc smelter. Environmental Toxicology and Chemistry 12:1661–1670.

Carline, R. F., W. E. Sharpe, and C. J. Gagen. 1992. Changes in fish communities and trout management in response to acidification of streams in Pennsylvania. Fisheries 17:33–38.

Carr, R. S. 1993. Sediment quality assessment survey of the Galveston Bay system. U.S. Environmental Protection Agency, National Estuary Program. Galveston Bay National Estuary Program Report GBNEP-30. 101 pp.

Carson, R. 1962. Silent spring. Houghton-Mifflin, Boston, Mass. 368 pp.

Chick, J. H., and C. C. McIvor. 1994. Patterns in the abundance and composition of fishes among beds of different macrophytes: viewing a littoral zone as a landscape. Canadian Journal of Fisheries and Aquatic Sciences 51:2873–2882.

Clair, T. A., and J. M. Ehrman. 1995. Acid precipitation-related chemical trends in 18 rivers of Atlantic Canada—1983 to 1992. Environmental Monitoring and Assessment 35:165–179.

Clark, D. R., Jr. 1981. Bats and environmental contaminants: a review. U.S. Fish and Wildlife Service Special Scientific Report—Wildlife 225. 27 pp.

Clark, D. R., Jr. 1987. Selenium accumulation in mammals exposed to contaminated California irrigation drainwater. Science of the Total Environment 66:147–168.

Clark, D. R., Jr., P. A. Ogasawara, G. J. Smith, and H. M. Ohlendorf. 1989. Selenium accumulation by raccoons at Kesterson National Wildlife Refuge, California, 1986. Archives of Environmental Contamination and Toxicology 18:787–794.

Clark, R. C., Jr., and D. W. Brown. 1977. Petroleum: properties and analyses in biotic and abiotic systems. Pages 1–90 *in* D. C. Malins, editor. Effects of petroleum on arctic and subarctic marine environments and organisms. Volume 1. Nature and fate of petroleum. Academic Press, New York.

Clark, R. C., Jr., and J. S. Finley. 1977. Effects of oil spills in arctic and subarctic environments. Pages 411–476 *in* D. C. Malins, editor. Effects of petroleum on arctic and subarctic marine environments and organisms. Volume 2. Biological effects. Academic Press, New York.

Clark, R. C., Jr., and W. D. MacLeod, Jr. 1977. Inputs, transport mechanisms, and observed concentrations of petroleum in the marine environment. Pages 91–224 *in* D. C. Malins, editor. Effects of petroleum on arctic and subarctic marine environments and organisms. Volume 1. Nature and fate of petroleum. Academic Press, New York.

Clarkson, T. W. 1991. Inorganic and organometal pesticides. Pages 497–584 *in* W. J. Hayes, Jr., and E. R. Laws, Jr., editors. Handbook of pesticide toxicology. Academic Press, San Diego, Calif.

Clawson, R. L., and D. R. Clark, Jr. 1989. Pesticide contamination of endangered gray bats and their food base in Boone County, Missouri, 1982. Bulletin of Environmental Contamination and Toxicology 42:431–437.

Colborn, T. 1991. Epidemiology of Great Lakes bald eagles. Journal of Toxicology and Environmental Health 33:395–453.

Colborn, T., F. S. vom Sall, and A. M. Soto. 1993. Developmental effects of endocrine-disrupting chemicals in wildlife and humans. Environmental Health Perspectives 101:378–384.

Connell, D. W. 1982. An approximate petroleum hydrocarbon budget for the Hudson Raritan Estuary—New York. Marine Pollution Bulletin 13:89–93.

Cooke, A. S. 1973. Shell thinning in avian eggs by environmental pollutants. Environmental Pollution 4:85–152.

Cooke, A. S. 1977. Effects of field applications of the herbicides diquat and dichlobenil on amphibians. Environmental Pollution 12:43–50.

Cooper, K. 1991. Effects of pesticides on wildlife. Pages 463–496 *in* W. J. Hayes, Jr., and E. R. Laws, Jr., editors. Handbook of pesticide toxicology, Volume 1. Academic Press, San Diego, Calif.

Cope, O. B. 1961. Effects of DDT spraying for spruce budworm on fish in the Yellowstone River system. Transactions of the American Fisheries Society 90:239–251.

Cordone, A. J., and D. W. Kelley. 1961. The influences of inorganic sediment on the aquatic life of streams. California Fish and Game 47:189–228.

Cosby, B. J., P. F. Ryan, J. R. Webb, G. M. Hornberger, and J. N. Galloway. 1991. Mountains of West Virginia. Pages 297–318 *in* D. E. Charles, editor. Acidic deposition and aquatic ecosystems. Springer-Verlag, New York.

Cronk, J. K., W. J. Mitsch, and R. M. Sykes. 1990. Effective modelling of a major inland oil spill on the Ohio River. Ecological Modelling 51:161–192.

Cumberland, J. H., J. R. Hibbs, and I. Hoch. 1982. The economics of managing chlorofluorocarbons. Stratospheric ozone and climate issues. Resources for the Future, Washington, D.C. 511 pp.

Cunningham, J. J., W. M. Kemp, M. R. Lewis, and J. C. Stevenson. 1985. Temporal responses of the macrophyte, *Potamogeton perfolatus* L., and its associated autotrophic community to atrazine exposure in estuarine microcosms. Estuaries 7:519–530.

Desgrangies, J.-L., and C. Gagnon. 1994. Duckling response to changes in the trophic web of acidified lakes. Hydrobiologia 279/280:207–221.

Diamond, A. W. 1989. Impacts of acid rain on aquatic birds. Environmental Monitoring and Assessment 12:245–254.

Drent, P. J., and J. W. Woldendorp. 1989. Acid rain and eggshells. Nature (London) 339:431.

Driscoll, C. T., J. P. Baker, J. J. Bisogni, and C. L. Schofield. 1980. Effects of aluminum speciation on fish in dilute acidified water. Nature (London) 284:161–164.

Driscoll, C. T., C. Yan, C. L. Schofield, R. Munson, and J. Holsapple. 1994. The mercury cycle and fish in the Adirondack lakes. Environmental Science & Technology 28:136A–143A.

Duffy, L. K., R. T. Bowyer, J. W. Testa, and J. B. Farrow. 1993. Differences in blood haptoglobin and length-mass relationships in river otters (*Lutra canadensis*) from oiled and nonoiled areas of Prince William Sound, Alaska. Journal of Wildlife Diseases 29:353–359.

D'Unger, C., D. Chapman, and R. S. Carr. 1996. Discharge of oilfield-produced water in Nueces Bay, Texas: a case study. Environmental Management 20: 143–150.

Dvorak, A. J., and B. G. Lewis. 1978. Impacts of coal-fired power plants on fish, wildlife, and their habitats. U.S. Fish and Wildlife Service Biological Services Program FWS/OBS-78/29. 260 pp.

Eadie, B. J. 1984. Cycling of polynuclear aromatic hydrocarbons in the Great Lakes ecosystem. Pages 213–238 *in* J. O. Nriagu and M. S. Simmons, editors. Toxic contaminants in the Great Lakes. John Wiley & Sons, New York.

Eichers, T. R., P. A. Andrilenas, and T. W. Anderson. 1978. Farmers' use of pesticides in 1976. U.S. Department of Agriculture, Economics, Statistics, and Cooperatives Service, Agricultural Economics Report 418. 58 pp.

Eichers, T., R. Jenkins, and A. Fox. 1971. DDT used in farm production. U.S. Department of Agriculture, Economic Research Service, Agricultural Economics Report 158. 7 pp.

Eisler, R. 1990. Paraquat hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildlife Service Biological Report 85(1.22). 28 pp.

Eisler, R. 1991. Cyanide hazards to fish, wildlife, and invertebrates: a synoptic review. U.S. Fish and Wildlife Service Biological Report 85(1.23). 55 pp.

Ellenberger, S. A., P. C. Baumann, and T. W. May. 1994. Evaluation of effects caused by high copper concentrations in Torch Lake, Michigan, on reproduction in yellow perch. Journal of Great Lakes Research 20:531–536.

Elson, P., A. Meister, J. Saunders, R. Saunders, J. Sprague, and V. Zitko. 1973. Impact of chemical pollution on Atlantic salmon in North America. Pages 83–110 *in* M. Smith and W. Carter, editors. Proceedings of the international symposium on the Atlantic salmon: management, biology and survival of the species. International Atlantic Salmon Federation, Special Publication 4(1). St. Andrews, New Brunswick, Canada.

Exxon Valdez Trustee Council. 1994. Five years later. 1994 status report on the *Exxon Valdez* oil spill. Anchorage, Alaska. 19 pp.

Fabacher, D. L., J. M. Besser, C. J. Schmitt, J. C. Harshbarger, P. H. Peterman, and J. A. Lebo. 1991. Contaminated sediments from tributaries of the Great Lakes: chemical characterization and carcinogenic effects in medaka (*Orizias latipes*). Archives of Environmental Contamination and Toxicology 20:17–34.

Fabacher, D. L., E. E. Little, S. B. Jones, E. C. DeFabo, and L. J. Webber. 1994. Ultraviolet-B radiation and the immune response of rainbow trout. Pages 205–217 *in* J. Stolen and T. C. Stolen, editors. Modulators of fish immune responses: models for environmental toxicology/biomarkers and immunostimulators. Volume 1. SOS Publications, Fair Haven, N.J.

Ferguson, D. E., D. D. Culley, W. D. Cotton, and R. P. Dodds. 1964. Resistance to chlorinated hydrocarbon insecticides in three species of fresh water fish. BioScience 14:43–44.

Finlayson, B. J., and K. M. Verrue. 1980. Estimated safe zinc and copper levels for chinook salmon, *Oncorhynchus tshawytscha*, in the upper Sacramento River, California. California Fish and Game 66:68–82.

Finlayson, B., and D. C. Wilson. 1979. Acid mine waste—how it affects king salmon in the upper Sacramento River. Outdoor California 1979:8–12.

Fiss, F. C., and R. F. Carline. 1993. Survival of brook trout embryos in three episodically acidified streams. Transactions of the American Fisheries Society 122:268–278.

Fleming, W. J., T. P. Augspurger, and J. A. Alderman. 1995. Freshwater mussel die-off attributed to antichlolinesterase poisoning. Environmental Toxicology and Chemistry 14:877–879.

Fox, A., T. Eichers, P. Andrilenas, R. Jenkins, and H. Blake. 1968. Extent of farm pesticide use on crops in 1966. U.S. Department of Agriculture, Economic Research Service, Agricultural Economics Report 147. 23 pp.

Freda, J. 1986. The influence of acidic pond waters on amphibians: a review. Water, Air and Soil Pollution 30:439–450.

Fry, D. M., and C. K. Toone. 1981. DDT-induced feminization of gull embryos. Science 213:922–924.

Fucik, K. W., T. J. Bright, and K. S. Goodman. 1984. Measurements of damage, recovery, and rehabilitation of coral reefs exposed to oil. Pages 115–133 *in* J. Cairns, Jr., and A. L. Buikema, Jr., editors. Restoration of habitats impacted by oil spills. Butterworth Publishers, Boston, Mass.

Garrett, G. P., and C. R. Inman. 1984. Selenium-induced changes in fish populations of a heated reservoir. Proceedings of the annual conference. Southeastern Association of Fish and Wildlife Agencies 38:612–624.

Garrott, R. A., L. L. Eberhardt, and D. M. Burn. 1993. Mortality of sea otters in Prince William Sound following the *Exxon Valdez* oil spill. Marine Mammal Science 9:343–359.

Geraci, J. R., and T. G. Smith. 1977. Consequences of oil fouling on marine mammals. Pages 399–410 *in* D. C. Malins, editor. Effects of petroleum on arctic and subarctic marine environments and organisms. Volume 2. Biological effects. Academic Press, New York.

Getter, C. D., G. Cintron, B. Dicks, R. R. Lewis III, and E. D. Seneca. 1984. The recovery and restoration of salt marshes and mangroves following an oil spill. Pages 65–114 *in* J. Cairns, Jr., and A. L. Buikema, Jr., editors. Restoration of habitats impacted by oil spills. Butterworth Publishers, Boston, Mass.

Ghering, P. J., R. J. Nolan, P. G. Wantabe, and A. M. Schumann. 1991. Solvents, fumigants, and related compounds. Pages 637–730 *in* W. J. Hayes, Jr., and E. R. Laws, Jr., editors. Handbook of pesticide toxicology. Volume 2. Academic Press, San Diego, Calif.

Gianessi, L. P. 1986. A national pesticide usage database. Quality of the Environment Division, Resources for the Future. Washington, D.C.

Gianessi, L. P., and C. Puffer. 1991. Herbicide use in the United States. Quality of the Environment Division, Resources for the Future. Washington, D.C. 128 pp.

Gilbertson, M., and R. S. Schneider. 1991. Preface to special issue: International Joint Commission workshop on cause–effect linkages. Journal of

Toxicology and Environmental Health 33:v–x.

Girts, M. A., and R. L. P. Kleinmann. 1986. Constructed wetlands for treatment of acid mine drainage: a preliminary review. Pages 165–171 *in* National symposium on mining, hydrology, sedimentology, and reclamation. University of Kentucky, Lexington.

Glaser, L. C. 1995. Wildlife mortality attributed to organophosphorus and carbamate pesticides. Pages 416–418 *in* E. T. LaRoe, G. S. Farris, C. E. Puckett, P. D. Doran, and M. J. Mac, editors. Our living resources: a report to the nation on the distribution, abundance, and health of U.S. plants, animals, and ecosystems. U.S. Department of the Interior, National Biological Service, Washington, D.C.

Gooch, J. W., and F. Matsamura. 1987. Toxicity of chlorinated bornane (toxaphene) residues isolated from Great Lakes lake trout (*Salvelinus namaycush*). Archives of Environmental Contamination and Toxicology 16:349–355.

Goolsby, D. A., R. C. Coupe, and D. J. Markovchick. 1991. Distribution of major herbicides in the Mississippi River and its tributaries, April through June 1991. U.S. Geological Survey, Water-Resources Investigations Report 91-4163. 35 pp.

Graham, R. J. 1960. Effects of forest insect spraying on trout and aquatic insects in some Montana streams. Pages 62–65 *in* C. M. Tarzwell, editor. Biological problems in water pollution. Transactions of the 1959 seminar. U.S. Department of Health, Education, and Welfare, Technical Report W60-3. Public Health Service, Cincinnati, Ohio.

Grau, C. R., T. Roudybush, J. Dobbs, and J. Wathen. 1977. Altered yolk structure and reduced hatchability of eggs from birds fed single doses of petroleum oils. Science 195:779–781.

Graveland, J., R. van der Wal, J. H. van Balen, and A. J. Noordvijk. 1994. Poor reproduction in forest passerines from decline of snail abundance on acidified soil. Nature (London) 368:446–448.

Great Lakes Water Quality Board. 1985. Report on Great Lakes water quality. 1985 report to the International Joint Commission (IJC). IJC, Great Lakes Regional Office, Windsor, Ontario, Canada. 212 pp.

Greene, D. L., and D. J. Santini, editors. 1993. Transportation and global climate change. American Council for an Energy Efficient Economy, Washington, D.C. 357 pp.

Greig-Smith, P. W. 1994. Understanding the impact of pesticides on wild birds by monitoring incidents of poisonings. Pages 301–319 *in* R. J. Kendall and T. E. Lacher, Jr., editors. Wildlife toxicology and population modeling. Integrated studies of agroecosystems. Lewis Publishers, Boca Raton, Fla.

Gundlach, E. R., and M. O. Hayes. 1978. Vulnerability of coastal environments to oil spill impacts. Journal of the Marine Technical Society 12:18–28.

Häder, D.-P. 1993. Effects of enhanced solar ultraviolet radiation on aquatic ecosystems. Pages 155–192 *in* M. Tevini, editor. UV-B radiation and ozone depletion. Effects on humans, animals, plants, microorganisms, and materials. Lewis Publishers, Boca Raton, Fla.

Haines, T. A. 1981. Acid precipitation and its consequences for aquatic ecosystems: a review. Transactions of the American Fisheries Society 110:669–707.

Haines, T. A. 1992. New England's rivers and Atlantic salmon. Pages 131–139 *in* R. H. Stroud, editor. Stemming the tide of coastal fish habitat loss. National Coalition for Marine Conservation, Savannah, Ga.

Hall, L. W., Jr., and D. T. Burton. 1982. Effects of power plant coal pile and coal waste runoff and leachate on aquatic biota: an overview with research recommendations. Chemical Rubber Company, Critical Reviews in Toxicology 10:287–301.

Hall, L. W., Jr., S. E. Finger, and M. C. Ziegenfuss. 1991. A review of in situ and on-site striped bass contaminant and water quality studies in Maryland waters of the Chesapeake Bay watershed. American Fisheries Society Symposium 14:3–15.

Hall, L. W., Jr., A. E. Pinckney, L. O. Horseman, and S. E. Finger. 1985. Mortality of striped bass larvae in relation to contaminants and water quality in a Chesapeake Bay tributary. Transactions of the American Fisheries Society 114:861–868.

Hallock, R. J., and L. L. Hallock, editors. 1993. Detailed study of irrigation drainage in and near wildlife management areas, west-central Nevada, 1987–90. Part B. Effect on biota in Stillwater and Fernley Wildlife Management areas and other nearby wetlands. U.S. Geological Survey, Water-Resources Investigations Report 92-4024B. 84 pp.

Hamilton, S. J., and B. Waddell. 1994. Selenium in eggs and milt of razorback suckers (*Xyrauchen texanus*) in the middle Green River, Utah. Archives of Environmental Contamination and Toxicology 27:195–201.

Haramis, G. M., and D. S. Chu. 1987. Acid rain effects on waterfowl: use of black duck broods to assess food resources of experimentally acidified wetlands. Pages 173–181 *in* A. W. Diamond and F. L. Fillion, editors. The value of birds. International Council for Bird Preservation, Technical Publication 6. Cambridge, England.

Harder, H. W., T. V. Carter, and T. F. Bidleman. 1983. Acute effects of toxaphene and its sediment-degraded products on estuarine fish. Canadian Journal of Fisheries and Aquatic Sciences 40:2119–2125.

Hardie, M. G., J. C. Jennett, E. Bolter, B. Wixson, and N. Gale. 1974. Water resources problems and solutions associated with the new Lead Belt of S.E. Missouri. Pages 109–122 *in* R. F. Hadley and D. T. Snow, editors. Water resources problems related to mining. American Water Resources Association, Proceedings 18. Minneapolis, Minn.

Hayes, W. J., Jr. 1991. Introduction. Pages 1–33 *in* W. J. Hayes, Jr., and E. R. Laws, Jr., editors. Handbook of pesticide toxicology. Volume 1. Academic Press, San Diego, Calif.

Heck, W. W., and C. E. Anderson. 1980. Effects of air pollutants on plants. Pages 133–160 *in* F. E. Guthrie and J. J. Perry, editors. Introduction to environmental toxicology. Elsevier, New York.

Heit, M. 1985. The relationship of a coal fired power plant to the levels of polycyclic aromatic hydrocarbons (PAH) in the sediment of Cayuga Lake. Water, Air, and Soil Pollution 24:41–61.

Helz, G. R., and R. J. Huggett. 1987. Contaminants in Chesapeake Bay: the regional perspective. Pages 270–297 *in* S. K. Majumdar, L. W. Hall, Jr., and H. M. Austin, editors. Contaminant problems and management of Chesapeake Bay resources. Pennsylvania Academy of Science, Easton.

Henley, D. E., and D. G. Rauschuber. 1981. Freshwater needs of fish and wildlife resources in the Nueces–Corpus Christi Bay area, Texas: a literature synthesis. U.S. Fish and Wildlife Service, Office of Biological Services, FWS/OBS-80/10. 410 pp.

Henny, C. J., L. J. Blus, D. J. Hoffman, and R. A. Grove. 1994. Lead in hawks, falcons and owls downstream from a mining site on the Coeur d'Alene River, Idaho. Environmental Monitoring and Assessment 29:267–288.

Henny, C. J., L. J. Blus, D. J. Hoffman, R. A. Grove, and J. S. Hatfield. 1991. Lead accumulation and osprey production near a mining site on the Coeur d'Alene River, Idaho. Archives of Environmental Contamination and Toxicology 21:415–424.

Henny, C. J., F. R. Ward, K. E. Riddle, and R. M. Prouty. 1982. Migratory peregrine falcons, *Falco peregrinus*, accumulate pesticides in Latin America during winter. Canadian Field-Naturalist 96:333–338.

Herlihy, A. T., P. R. Kaufmann, M. E. Mitch, and D. D. Brown. 1990. Regional estimates of acid mine drainage impact on streams in the mid-Atlantic and southeastern United States. Water, Air, and Soil Pollution 50:91–107.

Herman, S. G., R. L. Garrett, and R. L. Rudd. 1969. Pesticides and the western grebe. Pages 24–53 *in* M. W. Miller and G. G. Berg, editors. Pesticide fallout. Thomas, Springfield, Ill.

Hesselberg, R. J., J. P. Hickey, and D. A. Nortrup. 1990. Contaminant residues in the bloater (*Coregonus hoyi*) of Lake Michigan, 1969–1986. Journal of Great Lakes Research 16:121–129.

Hodgins, H. O., B. B. McCain, and J. M. Hawkes. 1977. Marine fish and invertebrate diseases, host disease resistance, and pathological effects of petroleum. Pages 95–173 *in* D. C. Malins, editor. Effects of petroleum on arctic and subarctic marine environments and organisms. Volume 2. Biological effects. Academic Press, New York.

Hoffman, D. J. 1979. Embryotoxic and teratogenic effects of petroleum hydrocarbons in mallards (*Anas platyrhynchos*). Journal of Toxicology and Environmental Health 5:835–844.

Hoffman, D. J. 1990. Embryotoxicity and teratogenicity of environmental contaminants to bird eggs. Reviews of Environmental Contamination and Toxicology 115:40–89.

Hoffman, D. J. 1994. Measurements of toxicity and critical stages of development. Pages 47–67 *in* R. J. Kendall and T. E. Lacher, Jr., editors. Wildlife toxicology and population modeling. Integrated studies of agroecosystems. Lewis Publishers, Boca Raton, Fla.

Hoiland, W. K., F. W. Rabe, and R. C. Biggam. 1994. Recovery of macroinvertebrate communities from metal pollution in the South Fork and mainstem of the Coeur d'Alene River, Idaho. Water Environment Research 66:84–88.

Holmes, W. N., and J. Cronshaw. 1977. Biological effects of petroleum on marine birds. Pages 359–398 *in* D. C. Malins, editor. Effects of petroleum on arctic and subarctic marine environments and organisms. Volume 2. Biological effects. Academic Press, New York.

Huckabee, J. W., C. P. Goodyear, and R. D. Jones. 1975. Acid rock in the Great Smokies: unanticipated impact on aquatic biota of road construction in regions of sulfide mineralization. Transactions of the American Fisheries Society 105:677–684.

Hutchinson, J. D., and M. P. Simmonds. 1994. Organochlorine contamination in pinnipeds. Review of Environmental Contamination and Toxicology 136:123–167.

Ide, F. P. 1957. Effect of forest spraying with DDT on aquatic insects of salmon streams. Transactions of the American Fisheries Society 86:208–219.

International Boundary and Water Commission. 1994. Binational study regarding the presence of toxic substances in the Rio Grande/Rio Bravo and its tributaries along the boundary portion between the United States and Mexico. Final report, International Boundary and Water Commission, El Paso, Tex. 247 pp.

Jagoe, C. H., T. A. Haines, and F. W. Kircheis. 1984. Effects of reduced pH on three life stages of Sunapee char *Salvelinus alpinus*. Bulletin of Environmental Contamination and Toxicology 33:430–438.

Jarman, W. M., A. M. Springer, W. A. Walker II, and W. G. Hunt. 1986. A metabolic derivation of DDE from kelthane. Environmental Toxicology and Chemistry 5:13–19.

Jensen, S. 1966. Report on a new chemical hazard. New Science 32:612.

Johnson, A., D. Norton, and B. Yake. 1988. Persistence of DDT in the Yakima River drainage, Washington. Archives of Environmental Contamination and Toxicology 17:289–297.

Johnson, H. E., and C. Pecor. 1969. Coho salmon mortality and DDT in Lake Michigan. Transactions of the North American Wildlife Natural Resources Conference 34:157–166.

Jordan, M. J. 1975. Effects of zinc smelter emissions and fire on a chestnut–oak woodland. Ecology 56:78–91.

Kannan, N., S. Tanabe, M. Ono, and R. Tatsukawa. 1989. Critical evaluation of polychlorinated biphenyl toxicity in terrestrial and marine mammals: increasing impact of non-*ortho* and mono-*ortho* coplanar polychlorinated biphenyls from land to ocean. Archives of Environmental Contamination and Toxicology 18:850–857.

Kemp, W. M., W. R. Boynton, J. J. Cunningham, J. C. Stevenson, T. W. Jones, and J. C. Means. 1985. Effects of atrazine and linuron on photosynthesis and growth of the macrophytes *Potamogeton perfoliatus* L. and *Myriophyllum spicatum* L. in an estuarine environment. Marine Environmental Research 16:255–280.

Kemp, W. M., W. R. Boynton, R. R. Twilley, J. C. Stevenson, and J. C. Means. 1983. The decline of submerged vascular plants in upper Chesapeake Bay: summary of results concerning possible causes. Marine Technology Society Journal 17:78–89.

Kendall, R. J., and T. E. Lacher, Jr., editors. 1994. Wildlife toxicology and population modeling. Integrated studies of agroecosystems. Lewis Publishers, Boca Raton, Fla. 576 pp.

Kerr, J. B., and C. T. McElroy. 1993. Evidence for large upward trends of ultraviolet-B radiation linked to ozone depletion. Science 262:1032–1034.

Kiffney, P. M., and W. H. Clements. 1993. Bioaccumulation of heavy metals by benthic invertebrates at the Arkansas River, Colorado. Environmental Toxicology and Chemistry 12:1507–1517.

King, K., and C. A. Lefever. 1979. Effects of oil transferred from incubating gulls to their eggs. Marine Pollution Bulletin 10:319–321.

Kinney, E. C. 1964. Extent of acid mine pollution in the United States affecting fish and wildlife. U.S. Fish and Wildlife Service Fish and Wildlife Circular 191.

Kooyman, G. L., R. W. Davis, and M. A. Castellini. 1977. Thermal conductance of immersed pinniped and sea otter pelts before and after oiling with Prudhoe Bay crude. Pages 151–157 *in* D. A. Wolfe, editor. Fate and effects of petroleum hydrocarbons in marine organisms and ecosystems. Pergamon Press, New York.

Kubiak, T., H. Harris, L. Smith, T. Schwartz, D. Stalling, J. Trick, L. Sileo, D. Docherty, and T. Erdman. 1989. Microcontaminants and reproductive impairment of the Forster's tern on Green Bay, Lake Michigan—1983. Archives of Environmental Contamination and Toxicology 706–721.

Kuehl, D. W., B. C. Butterworth, A. McBride, S. Kroner, and D. Bahnick. 1989. Contamination of fish by 2,3,7,8-tetrachlorodibenzo-*p*-dioxin: a survey of fish from major watersheds in the United States. Chemosphere 18:1997–2014.

Kuehl, D. W., P. M. Cook, A. R. Betterman, and B. C. Butterworth. 1987. Bioavailability of polychlorinated dibenzo-*p*-dioxins and dibenzofurans from contaminated Wisconsin River sediments to carp. Chemosphere 16:667–679.

Kuehl, D. W., R. Haebler, and C. Potter. 1991. Chemical residues in dolphins from the U.S. Atlantic coast including Atlantic bottlenose obtained during the 1987/88 mass mortality. Chemosphere 22:1071–1084.

Layzer, J. B., and R. M. Anderson. 1992. Impacts of the coal industry on rare and endangered aquatic organisms of the upper Cumberland River basin. Final report to the Kentucky Department of Fish and Wildlife Resources and the Tennessee Wildlife Resources Agency. National Biological Service, Tennessee Cooperative Fishery Research Unit, Cookville. 118 pp.

Layzer, J. B., M. E. Gordon, and R. M. Anderson. 1993. Mussels: the forgotten fauna of regulated rivers. A case study of the Caney Fork River. Regulated Rivers Research and Management 8:63–71.

Leduc, G. 1984. Cyanides in water: toxicological significance. Pages 153–224 *in* L. J. Weber, editor. Aquatic toxicology. Volume 2. Raven Press, New York.

Lemly, A. D. 1985. Ecological basis for regulating aquatic emissions from the power industry: the case with selenium. Regulatory Toxicology and Pharmacology 5:465–486.

Lemly, A. D., S. E. Finger, and M. K. Nelson. 1993. Sources and impacts of irrigation drainwater contaminants in arid wetlands. Environmental Toxicology and Chemistry 12:2265–2279.

Lemly, A. D., and G. J. Smith. 1987. Aquatic cycling of selenium: implications for fish and wildlife. U.S. Fish and Wildlife Service Fish and Wildlife Leaflet 12. 10 pp.

Leppäkoski, E. J., and L. S. Lindström. 1978. Recovery of benthic macrofauna from chronic pollution in the sea area off a refinery plant, southwest Finland. Journal of the Fisheries Research Board of Canada 35:766–775.

Lettenmaier, D. P., E. R. Hooper, C. Waggoner, and K. B. Faris. 1991. Trends in stream water quality in the continental United States, 1878–1987. Water Resources Research 27:327–339.

Likens, G. E., and F. H. Bormann. 1974. Acid rain: a serious environmental problem. Science 184:1176–1179.

Little, E. E., and D. L. Fabacher. 1994. Comparative sensitivity of rainbow trout and two threatened salmonids, Apache trout and Lahontan cutthroat trout, to ultraviolet-B radiation. Archiv fuer Hydrobiologie 43:217–226.

Lloyd, D. S., J. P. Koenings, and J. D. LaPerriere. 1987. Effects of turbidity in fresh waters of Alaska. North American Journal of Fisheries Management 7:18–33.

Longcore, J. R., H. Boyd, R. T. Brooks, G. M. Haramis, D. K. McNicol, J. R. Newman, K. A. Smith, and F. Stearns. 1993. Acidic deposition: effects on wildlife and habitats. Wildlife Society Technical Review 93-1. 42 pp.

Mac, M. J., and C. C. Edsall. 1991. Environmental contaminants and the reproductive success of lake trout in the Great Lakes: an epidemiological approach. Journal of Toxicology and Environmental Health 33:375–394.

Mac, M. J., T. R. Schwartz, C. C. Edsall, and A. M. Frank. 1993. Polychlorinated biphenyls in Great Lakes lake trout and their eggs: relations to survival and congener composition 1979–1988. Journal of Great Lakes Research 19:752–765.

Madronich, S. 1993. UV radiation in the natural and perturbed atmosphere. Pages 17–69 *in* M. Tevini, editor. UV-B radiation and ozone depletion. Effects on humans, animals, plants, microorganisms, and materials. Lewis Publishers, Boca Raton, Fla.

Malins, D. C., M. M. Krahn, M. S. Meyers, L. D. Rhodes, D. W. Brown, C. A. Krone, B. B. McCain, and S.-L. Can. 1984. Toxic chemicals in sediments and biota from a creosote-polluted harbor: relationships with hepatic neoplasms and other hepatic lesions in English sole (*Parophrys vetulus*). Carcinogenesis 6:1463–1469.

Mann, K. H., and R. B. Clark. 1978. Long-term effects of oil spills on marine intertidal communities. Journal of the Fisheries Research Board of Canada 35:791–795.

Matter, W. J., J. J. Ney, and O. E. Maughan. 1978. Sustained impact of abandoned surface mines on fish and benthic invertebrate populations in headwater streams of southwestern Virginia. Pages 203–216 *in* D. E. Samuel, J. R. Stauffer, C. H. Hocutt, and W. T. Mason, editors. Surface mining and fish/wildlife needs in the eastern United States. U.S. Fish and Wildlife Service, Biological Services Program FWS/OBS-78/81.

May, T. W., and G. L. McKinney. 1977. Cadmium, lead, mercury, arsenic, and selenium concentrations in freshwater fish, 1976–77—National Pesticide Monitoring Program. Pesticides Monitoring Journal 15:14–38.

Mayer, F. L., Jr., and M. R. Ellersieck. 1986. Manual of acute toxicity: interpretation and data base for 410 chemicals and 66 species of freshwater animals. U.S. Fish and Wildlife Service Resource Publication 160. 506 pp.

McGurk, M. D., H. D. Warburton, T. B. Parker, and M. Litke. 1990. Early life history of Pacific herring: 1989 Prince William Sound egg incubation experiment. National Technical Information Service Technical Report NTIS/PB90-265885.

McGurk, M. D., H. D. Warburton, T. B. Parker, M. Litke, and J. B. Marliave. 1992. Effects of the *Exxon Valdez* oil spill on survival of Pacific herring eggs and viability of their larvae. Pages 255–257 *in* E. G. Baddaloo, S. Ramamoorthy, and J. W. Moore, editors. Proceedings of the nineteenth annual aquatic toxicity workshop, 4–7 October 1992, Edmonton, Alberta. Canadian Technical Report of Fisheries and Aquatic Sciences 1942.

McKinney, B. R. 1994. Black Gap Wildlife Management Area peregrine falcon (*Falco peregrinus anatum*) research project (distribution, population trends and seasonal movements of peregrine falcons in trans-Pecos, Texas and Northern Mexico, 1994 field season). Texas Parks and Wildlife Department, Austin. 27 pp.

Meyers, P. A. 1984. Petroleum contaminants in the Great Lakes. Pages 147–162 *in* J. O. Nriagu and M. S. Simmons, editors. Toxic contaminants in the Great Lakes. John Wiley & Sons, New York.

Mielke, J. E. 1990. Oil in the ocean: the short- and long-term impacts of a spill. Congressional Research Service, Library of Congress, Report for Congress SPR 90-356. 34 pp.

Minchew, C. D., and J. D. Yarbrough. 1977. The occurrence of fin rot in mullet (*Mugil cephalus*) associated with crude oil contamination of an estuarine–pond ecosystem. Journal of Fish Biology 10:319–323.

Mineau, P., G. A. Fox, R. J. Norstrom, D. V. Weseloh, D. J. Hallett, and J. A. Ellenton. 1984. Using the herring gull to monitor levels and effects of organochlorine contamination in the Canadian Great Lakes. Pages 425–452 *in* J. O. Nriagu and M. S. Simmons, editors. Toxic contaminants in the Great Lakes. John Wiley & Sons, New York.

Mineau, P., and D. B. Peakall. 1987. An evaluation of avian impact assessment techniques following broad scale forest insecticide sprays. Environmental Toxicology and Chemistry 6:781–791.

Mining Policy Center. 1994. Burden of gilt. The legacy of abandoned mines. Washington, D.C.

Missouri Department of Conservation. 1992. Rare and endangered species of Missouri checklist. Jefferson City, Mo. 43 pp.

Mix, M. C. 1986. Cancerous diseases in aquatic animals and their association with environmental pollutants: a critical literature review. Marine Environmental Research 20:1–141.

Moore, J. N., S. N. Luoma, and D. Peters. 1991. Downstream effects of mine effluent on an intermontane riparian system. Canadian Journal of Fisheries and Aquatic Sciences 48:222–232.

Moore, R., and T. Mills. 1977. An environmental guide to Western surface mining. Part 2. Impacts, mitigation and monitoring. U.S. Fish and Wildlife Service, Western Energy and Land Use Team, Office of Biological Services FWS/OBS-78/04.

Mora, M. A. 1995. Residues and trends of organochlorine pesticide and polychlorinated biphenyls in birds from Texas, 1965–1988. National Biological Service, Fish and Wildlife Research 14. 26 pp.

Muto, H., and Y. Takizawa. 1989. Dioxins in cigarette smoke. Archives of Environmental Health 44:171–174.

Nance, J. 1991. Effects of oil/gas field produced water on the macrobenthic community in a small gradient estuary. Hydrobiologia 220:189–204.

National Acid Deposition Program/National Trends Network. 1995. NADP/NTN Coordination Office, Fort Collins, Colo.

Neff, J. M. 1979. Polycyclic aromatic hydrocarbons in the aquatic environment. Sources, fates, and environmental effects. Applied Science Publishers, London, England. 262 pp.

Neves, R. J. 1987. Recent die-offs of freshwater mussels in the United States: an overview. Pages 7–18 *in* R. J. Neves, editors. Proceedings of a workshop on die-offs of freshwater mussels in the United States. U.S. Fish and Wildlife Service and Upper Mississippi River Conservation Committee, Rock Island, Ill.

Norcross, B. L. 1992. Responding to an oil spill: reflections of a fishery scientist. Fisheries 17:4–5.

Nriagu, J. O., editor. 1984. Environmental impacts of smelters. John Wiley & Sons, New York. 608 pp.

Nriagu, J. O. 1990. The rise and fall of leaded gasoline. Science of the Total Environment 92:13–28.

Ohlendorf, H. M., D. J. Hoffman, M. K. Saiki, and T. W. Aldrich. 1986. Embryonic mortality and abnormalities of aquatic birds: apparent impacts of selenium from irrigation drainwater. Science of the Total Environment 52:49–63.

Ohlendorf, H. M., R. Hothem, and T. W. Aldrich. 1988. Bioaccumulation of selenium by snakes and frogs in the San Joaquin Valley. Copeia 1988:704–710.

Olive, J. H., J. L. Jackson, D. Keller, and P. Wetzel. 1992. Effects of oil field brines on biological activity of two tributaries of the Little Muskingum River, southeastern Ohio. Ohio Journal Science 92:139–146.

O'Shea, T., W. J. Fleming III, and E. Cromartie. 1980. DDT contamination at Wheeler National Wildlife Refuge. Science 209:509–510.

Pastorok, R. A., D. C. Peek, J. R. Sampson, and M. A. Jacobsen. 1994. Ecological risk assessment for river sediments contaminated by creosote. Environmental Toxicology and Chemistry 13:1929–1941.

Paulson, R. W., E. B. Chase, J. S. Williams, and D. W. Moody, editors. 1993. National

water summary, 1990–1991. Hydrologic events and stream water quality. U.S. Geological Survey Water-Supply Paper 2400. 590 pp.

Pendleton, E. 1995. Natural resources in the Chesapeake Bay watershed. Pages 263–267 *in* E. T. LaRoe, G. S. Farris, C. E. Puckett, P. D. Doran, and M. J. Mac, editors. Our living resources: a report to the nation on the distribution, abundance, and health of U.S. plants, animals, and ecosystems. U.S. Department of the Interior, National Biological Service, Washington, D.C.

Perry, J. J. 1980. Oil in the biosphere. Pages 198–248 *in* F. E. Guthrie and J. J. Perry, editors. Introduction to environmental toxicology. Elsevier, New York.

Phillips, D. J. H. 1987. Toxic contaminants in the San Francisco Bay–Delta and their possible biological effects. San Francisco Bay–Delta Aquatic Habitat Institute, Richmond, Calif. 413 pp.

Phillips, J. C., and F. C. Lincoln. 1930. American waterfowl. Their present situation and outlook for the future. Houghton-Mifflin, Boston, Mass. 312 pp.

Piatt, J. F., C. J. Lensink, W. Butler, M. Kendziorek, and D. R. Nysewander. 1990. Immediate impact of the *Exxon Valdez* oil spill on marine birds. Auk 107:387–397.

Pimentel, D., H. Acquay, M. Biltonen, P. Rice, M. Silva, J. Nelson, V. Lipner, S. Giordano, A. Horowitz, and M. D'Amore. 1992. Environmental and economic costs of pesticide use. BioScience 42:750–760.

Placet, M. 1991. Emissions involved in acidic deposition processes. Report 1. Pages 25–33 *in* P. M. Irving, editor. Acidic deposition: state of science and technology. Summary report of the U.S. National Acid Precipitation Program. Washington, D.C.

Presser, T. S., M. A. Sylvester, and W. H. Low. 1994. Bioaccumulation of selenium from natural geologic sources in the western states and its potential consequences. Environmental Management 18:423–436.

Preston, W. L. 1981. Vanishing landscapes: land and life in the Tulare Lake basin. University of California Press, Berkeley. 278 pp.

Prouty, R. M., and C. M. Bunck. 1986. Organochlorine residues in adult mallard and black duck wings, 1981–82. Environmental Monitoring and Assessment 6:49–57.

Pulliam, H. R. 1994. Incorporating concepts from population and behavioral ecology into models of exposure to toxins and risk assessment. Pages 13–26 *in* R. J. Kendall and T. E. Lacher, Jr., editors. Wildlife toxicology and population modeling. Integrated studies of agroecosystems. Lewis Publishers, Boca Raton, Fla.

Quilliam, M. A., M. S. Lant, C. Kaiser-Farel, D. R. McCalla, C. P. Sheldrake, A. A. Kerr, J. N. Lockington, and E. S. Gibbons. 1985. Identification of polycyclic aromatic compounds in mutagenic emissions from steel casting. Biomedical Mass Spectrometry 12:143–150.

Ramirez, P., Jr. 1993. Contaminants in oilfield produced waters discharged into the Loch Katrine wetland complex, Park County, Wyoming, and their bioconcentration in the aquatic bird food chain. U.S. Fish and Wildlife Service, Region 6 Contaminants Program, Cheyenne, Wyo. 37 pp.

Ray, D. E. 1991. Pesticides derived from plants and other organisms. Pages 585–636 *in* W. J. Hayes, Jr., and E. R. Laws, Jr., editors. Handbook of pesticide toxicology. Academic Press, San Diego, Calif.

Ribick, M. A., G. R. Dubay, J. D. Petty, D. L. Stalling, and C. J. Schmitt. 1981. Toxaphene residues in fish: identification, quantification, and confirmation at part per billion levels. Environmental Science & Technology 16:310–318.

Rice, C. P., and M. S. Evans. 1984. Toxaphene in the Great Lakes. Pages 163–194 *in* J. O. Nriagu and M. S. Simmons, editors. Toxic contaminants in the Great Lakes. John Wiley & Sons, New York.

Rice, C. P., P. J. Samson, and G. E. Noguchi. 1986. Atmospheric transport of toxaphene to Lake Michigan. Environmental Science & Technology 20:1109–1116.

Richins, R. T., and A. C. Risser, Jr. 1975. Total mercury in water, sediment, and selected aquatic organisms, Carson River, Nevada—1972. Pesticides Monitoring Journal 9:44–54.

Roach, R. W., R. S. Carr, C. L. Howard, and B. W. Cain. 1993. An assessment of produced water impacts in the Galveston Bay system. U.S. Fish and Wildlife Service, Houston, Tex. 56 pp.

Robotham, P. W. J., and R. A. Gill. 1989. Input, behavior, and fates of petroleum hydrocarbons and their residues. Pages 41–80 *in* J. Green and M. W. Trett, editors. The fate and effects of oil in fresh water. Elsevier, London, England.

Roline, R. A. 1988. The effects of heavy metals pollution of the upper Arkansas River on the distribution of aquatic macroinvertebrates. Hydrobiologia 160:3–8.

Rosseland, B., O. Skogheim, and I. Sevalrud. 1986. Acid deposition and effects in Nordic Europe. Damage to fish populations in Scandinavia continue to apace. Water, Air, and Soil Pollution 30:65–74.

Russell, R. W., S. J. Hecnar, and G. D. Haffner. 1995. Organochlorine pesticide residues in southern Ontario spring peepers. Environmental Toxicology and Chemistry 14:815–817.

Safe, S. 1990. Polychlorinated biphenyls (PCB's), dibenzo-*p*-dioxins (PCDD's), dibenzofurans (PCDF's), and related compounds: environmental and mechanistic considerations which support the development of toxic equivalency factors. Critical Reviews in Toxicology 21:51–88.

Saiki, M. K. 1986. A field example of selenium contamination in an aquatic food chain. Pages 67–76 *in* Proceedings of the first annual environmental symposium on selenium in the environment. California Agricultural Technology Institute, Publication CAT1/860201, Fresno.

Saiki, M. K., M. R. Jennings, and S. J. Hamilton. 1991. Preliminary assessment of the effects of selenium in agricultural drainage on fish in the San Joaquin Valley. Pages 369–385 *in* A. Dinar and D. Zilberman, editors. The economics and management of water and drainage in agriculture. Kluwer, Boston, Mass.

Saiki, M. K., and C. J. Schmitt. 1986. Organochlorine chemical residues in bluegills and common carp from the irrigated San Joaquin Valley floor, California. Archives of Environmental Contamination and Toxicology 15:357–366.

Sanders, J. G. 1987. Contaminant effects on primary producers in Chesapeake Bay. Pages 394–416 *in* S. K. Majumdar, L. W. Hall, Jr., and H. M. Austin. Contaminant problems and management of Chesapeake Bay resources. Pennsylvania Academy of Science, Easton.

Schmitt, C. J., and W. G. Brumbaugh. 1990. National Contaminant Biomonitoring Program: concentrations of arsenic, cadmium, copper, lead, mercury, selenium, and zinc in freshwater fishes of the United States. Archives of Environmental Contamination and Toxicology 19:41–72.

Schmitt, C. J., and C. M. Bunck. 1995. Persistent environmental contaminants in fish and wildlife. Pages 413–416 *in* E. T. LaRoe, G. S. Farris, C. E. Puckett, P. D. Doran, and M. J. Mac, editors. Our living resources: a report to the nation on the distribution, abundance, and health of U.S. plants, animals, and ecosystems. U.S. Department of the Interior, National Biological Service, Washington, D.C.

Schmitt, C. J., J. L. Zajicek, and P. L. Peterman. 1990. National Contaminant Biomonitoring Program: residues of organochlorine chemicals in freshwater fishes of the United States, 1976–1984. Archives of Environmental Contamination and Toxicology 19:748–782.

Schofield, C. 1976. Acid precipitation: effects on fish. Ambio 5:228–230.

Schreiber, R. K. 1995. Acidic deposition (acid rain). Pages 418–420 *in* E. T. LaRoe, G. S. Farris, C. E. Puckett, P. D. Doran, and M. J. Mac, editors. Our living resources: a report to the nation on the distribution, abundance, and health of U.S. plants, animals, and ecosystems. U.S. Department of the Interior, National Biological Service, Washington, D.C.

Schwartzenbach, R. P., and W. Giger. 1985. Behavior and fate of halogenated hydrocarbons in groundwater. Pages 446–471 *in* C. H. Ward, W. Giger, and P. L. McCarty, editors. Groundwater quality. John Wiley & Sons, New York.

Seidel, G. E., and R. C. Whetmore. 1995. Effects of dimilin application on

white-footed mouse populations in a central Appalachian forest. Environmental Toxicology and Chemistry 14:793–799.
Settle, D. M., and C. C. Patterson. 1980. Lead in albacore: guide to lead pollution in Americans. Science 207:1167–1176.
Shales, S., B. A. Thake, B. Frankland, D. H. Khan, J. D. Hutchinson, and C. F. Mason. 1989. Biological and ecological effects of oil. Pages 81–172 *in* J. Green and M. W. Trett, editors. The fate and effects of oil in freshwater. Elsevier, London, England.
Sheets, T. J. 1980. Agricultural pollutants. Pages 24–33 *in* F. E. Guthrie and J. J. Perry, editors. 1980. Introduction to environmental toxicology. Elsevier, New York.
Sisterson, D. L. 1991. Deposition monitoring: methods and results. Report 6. Pages 65–74 *in* P. M. Irving, editor. Acidic deposition: state of science and technology. Summary report of the U.S. National Acid Precipitation Program. Washington, D.C.
Smith, A. G. 1991. Chlorinated hydrocarbon insecticides. Pages 731–916 *in* W. J. Hayes, Jr., and E. R. Laws, Jr., editors. Handbook of pesticide toxicology. Academic Press, San Diego, Calif.
Smith, G. J. 1987. Pesticide use and toxicology in relation to wildlife: organophosphorous and carbamate compounds. U.S. Fish and Wildlife Service Resource Publication 170. 171 pp.
Smith, L. M., T. R. Schwartz, and K. Feltz. 1990. Determination and occurrence of AHH-active polychlorinated biphenyls, 2,3,7,8, -tetrachloro-*p*-dioxin and 2,3,7,8-tetrachlorodibenzofuran in Lake Michigan sediment and biota. The question of their relative toxicological significance. Chemosphere 21:1063–1085.
Smith, R. A., R. B. Alexander, and M. G. Holman. 1987. Water quality trends in the nation's rivers. Science 235:1607–1615.
Smith, R. M., and C. F. Cole. 1970. Chlorinated hydrocarbon insecticide residues in winter flounder, *Pseudopleuronectes americanus*, from the Weeweantic River estuary, Massachusetts. Journal of the Fisheries Research Board of Canada 27:2374–2380.
Smith, S. B., M. A. Blouin, and M. J. Mac. 1994. Ecological comparisons of Lake Erie tributaries with elevated incidence of fish tumors. Journal of Great Lakes Research 20:701–716.
Solomon, K. R., D. B. Baker, R. P. Richards, K. R. Dixon, S. J. Klaine, T. W. LaPoint, R. J. Kendall, C. P. Weisskopf, J. M. Giddings, J. P. Giesy, L. W. Hall, Jr., and W. M. Williams. 1996. Ecological risk assessment of atrazine in North American surface waters. Environmental Toxicology and Chemistry 15:31–76.
Spaulding, W. M., and R. D. Ogden. 1968. Effects of surface mining on the fish and wildlife resources of the United States. U.S. Fish and Wildlife Service Resource Publication 68. 51 pp.
Spies, R. B., and D. W. Rice, Jr. 1988. Effects of organic contaminants on reproduction of the starry flounder, *Platichthys stellatus*, in San Francisco Bay. II. Reproductive success of fish captured in San Francisco Bay and spawned in the laboratory. Marine Biology 98:191–200.
Spitsbergen, J. M., M. K. Walker, J. R. Olson, and R. E. Peterson. 1991. Pathologic alterations in early life stages of lake trout *Salvelinus namaycush*, exposed to 2,3,7,8- tetrachlorodibenzo-*p*-dioxin as fertilized eggs. Aquatic Toxicology 19:41–72.
St. Amant, J. R., M. E. Pariso, and T. B. Sheffy. 1984. Polychlorinated biphenyls in seven species of Lake Michigan fish, 1971–1981. Pages 311–319 *in* J. O. Nriagu and M. S. Simmons, editors. Toxic contaminants in the Great Lakes. John Wiley & Sons, New York.
Steiner, A. J. 1984. Midwinter Waterfowl Inventory—Atlantic Flyway 1954–1984 trend analysis. U.S. Fish and Wildlife Service, Newton Corner, Mass. 284 pp.
Stephens, D. W., B. Waddell, L. A. Peltz, and J. B. Miller. 1992. Detailed study of selenium and selected elements in water, bottom sediment, and biota associated with irrigation drainage in the middle Green River basin, Utah, 1988–90. U.S. Geological Survey, Water-Resources Investigations Report 92-4084. Salt Lake City, Utah. 164 pp.
Stevens, J. T., and D. D. Sumner. 1991. Herbicides. Pages 1317–1408 *in* W. J. Hayes, Jr., and E. R. Laws, Jr., editors. Handbook of pesticide toxicology. Academic Press, San Diego, Calif.
Swartz, R. C., P. F. Kemp, D. W. Schults, G. R. Ditsworth, and R. J. Ozretich. 1989. Acute toxicity of sediment from Eagle Harbor, Washington, to the infaunal amphipod *Rheopoxinius abronius*. Environmental Toxicology and Chemistry 8:215–222.
Tanabe, S., N. Kannan, A. Subriminian, S. Wantabe, and R. Tatsukawa. 1987. Highly toxic coplanar PCB's: occurrence, source, persistency and toxic implications to wildlife and humans. Environmental Pollution 47:147–163.
Tennessee Valley Authority. 1991. Status of the Ocoee Reservoirs. An overview of reservoir conditions and uses. Resource Group, River Basin Operations, Water Resources Division, TVA/WR/WQ—91/4. Chattanooga, Tenn. 18 pp.
Thompson, S. P., and K. L. Merritt 1988. Western Nevada wetlands—history and current status. Nevada Public Affairs Review 1:40–45.
Thrane, K. E. 1987. Deposition of polycyclic aromatic hydrocarbons (PAH) in the surroundings of primary aluminum industry. Water, Air, and Soil Pollution 33:385–393.
Tillitt, D. E., G. T. Ankley, D. Verbrugge, and J. P. Giesy. 1991a. H4IIE rat hepatoma cell bioassay-derived 2,3,7,8-tetrachloro-*p*-dioxin equivalents in colonial fish-eating waterbird eggs from the Great Lakes. Archives of Environmental Contamination and Toxicology 21:91–101.
Tillitt, D. E., J. P. Giesy, and G. T. Ankley. 1991b. Characterization of the H4IIE rat hepatoma cell bioassay as a tool for assessing toxic potency of planar halogenated hydrocarbons. Environmental Science & Technology 25:87–92.
Tillitt, D. E., J. P. Giesy, J. P. Ludwig, H. Kurita-Matsuba, D. V. Weseloh, P. S. Ross, C. A. Bishop, L. Sileo, K. Stromborg, J. Larson, and T. J. Kubiak. 1992. Polychlorinated biphenyl residues and egg mortality in double-crested cormorants from the Great Lakes. Environmental Toxicology and Chemistry 11:1281–1288.
Trautmann, M. B. 1957. The fishes of Ohio. Ohio State University Press, Columbus. 782 pp.
Turgeon, D., and A. Robertson. 1995. Contaminants in coastal fish and mollusks. Pages 408–412 *in* E. T. LaRoe, G. S. Farris, C. E. Puckett, P. D. Doran, and M. J. Mac, editors. Our living resources: a report to the nation on the distribution, abundance, and health of U.S. plants, animals, and ecosystems. U.S. Department of the Interior, National Biological Service, Washington, D.C.
Twomey, A. C., and S. J. Twomey. 1936. Selenium and duck sickness. Science 83:470–471.
Tyre, G. L., and R. G. Barton. 1986. Treating critical areas in the Tennessee Copper Basin. Journal of Soil and Water Conservation 41:381–382.
U.S. Environmental Protection Agency. 1977. Heavy metal pollution from spillage at ore smelters and mills. Office of Research and Development, Industrial Environmental Research Laboratory, Cincinnati, Ohio. EPA-600/2-77-171. 109 pp.
U.S. Environmental Protection Agency. 1979. Polychlorinated biphenyls 1929–1979: final report. Office of Toxic Substances, Washington, D.C. EPA-560/6-79-004.
U.S. Environmental Protection Agency. 1985. Report to Congress. Wastes from the extraction and beneficiation of metallic ores, phosphate rock, asbestos, overburden from uranium mining, and oil shale. Office of Solid Waste, Washington, D.C. EPA/530-SW-85-033.
U.S. Environmental Protection Agency. 1989. Carbofuran: a special review. Technical support document. Office of Pesticides and Toxic Substances, Washington, D.C. EPA-540/9-89-027.
U.S. Environmental Protection Agency. 1992. National study of chemical residues in fish. Volume 1. Office of Science and Technology, Standards and Applied Science Division. EPA-823/R-92-008a.
U.S. Environmental Protection Agency. 1993. National emissions inventory of mercury and mercury compounds: interim final report. Office of Air and Radiation, Office of Air Planning and

Standards, Research Triangle Park, N.C. EPA-453/R-93-048.
U.S. Environmental Protection Agency. 1995. Effluent limitations guidelines, pretreatment standards, and new source performance standards: oil and gas extraction point source category, coastal subcategory; proposed rule, 40 CFR Part 435. Federal Register 60:9427–9481.
U.S. Fish and Wildlife Service. 1994. Endangered and threatened wildlife and plants; reclassify the bald eagle from endangered to threatened in most of the lower 48 states; proposed rule, 50 CFR Part 17. Federal Register 59:35584–35594.
Vandame, R., M. Meled, M.-E. Colin, and L. P. Belzunces. 1995. Alteration of the homing flight in the honey bee *Apis mellifera* L. exposed to sublethal dose of deltamethrin. Environmental Toxicology and Chemistry 14:855–860.
van der Leun, J. C., and F. R. de Gruijl. 1993. Influences of ozone depletion on human and animal health. Pages 95–123 *in* M. Tevini, editor. UV-B radiation and ozone depletion. Effects on humans, animals, plants, microorganisms, and materials. Lewis Publishers, Boca Raton, Fla.
Vaughan, G. L., A. Talak, and R. R. Anderson. 1978. The chronology and character of recovery of aquatic communities from the effects of strip mining for coal in east Tennessee. Pages 119–125 *in* D. E. Samuel, J. R. Stauffer, C. H. Hocutt, and W. T. Mason, editors. Surface mining and fish/wildlife needs in the eastern United States. U.S. Fish and Wildlife Service, Biological Services Program FWS/OBS-78/81.
Wallace, G. J., W. P. Nickell, and R. F. Bernard. 1961. Bird mortality in the Dutch elm disease program in Michigan. Cranbrook Institute of Science Bulletin 41:1–44.
Warner, K., and O. C. Fenderson. 1962. Effects of DDT spraying for forest insects on Maine trout streams. Journal of Wildlife Management 26:87–93.
Watt, W. 1986. The case for liming some Nova Scotia salmon rivers. Water, Air, and Soil Pollution 31:775–789.
Welch, L. 1994. Contaminant burdens and reproductive rates of bald eagles breeding in Maine. M.S. thesis, University of Maine, Orono. 87 pp.
Weseloh, D. V. C., P. J. Ewins, J. Struger, P. Mineau, and R. J. Norstrom. 1994. Geographic distribution of organochlorine contaminants and reproductive parameters in herring gulls on Lake Superior in 1993. Environmental Monitoring and Assessment 29:229–252.
West, R. L., and E. Snyder-Conn. 1987. Effects of Prudhoe Bay reserve pit fluids on water quality and macroinvertebrates of arctic tundra ponds. U.S. Fish and Wildlife Service Biological Report 87(7). 48 pp.
White, D. H., and A. J. Krynitsky. 1986. Wildlife in some areas of New Mexico and Texas accumulate elevated DDE residues, 1983. Archives of Environmental Contamination and Toxicology 15:149–157.
Wiemeyer, S. N., C. M. Bunck, and C. J. Stafford. 1993. Environmental contaminants in bald eagle eggs—1980–84—and further interpretations of relationships to productivity and shell thickness. Archives of Environmental Contamination and Toxicology 24:213–227.
Wiener, J., T. Naimo, C. Korschgen, R. Dahlgren, J. Sauer, K. Lubinski, S. Rogers, and S. Brewer. 1995. Biota of the Upper Mississippi River ecosystem. Pages 236–239 *in* E. T. LaRoe, G. S. Farris, C. E. Puckett, P. D. Doran, and M. J. Mac, editors. Our living resources: a report to the nation on the distribution, abundance, and health of U.S. plants, animals, and ecosystems. U.S. Department of the Interior, National Biological Service, Washington, D.C.
Wigington, P. J., Jr. 1991. Episodic acidification of surface water acid-base chemistry. Report 12. Pages 115–118 *in* P. M. Irving, editor. Acid deposition: state of science and technology. Summary report of the National Acid Precipitation Assessment Program, Washington, D.C.
Wilhm, J. L., and T. C. Dorris. 1968. Biological parameters for water quality criteria. BioScience 18:477–481.
Williams, J. E., J. E. Johnson, D. A. Hendrickson, S. Contreras-Balderas, J. D. Williams, M. Navarro-Mendoza, D. E. McAllister, and J. E. Deacon. 1989. Fishes of North America endangered, threatened, or of special concern, 1989. Fisheries 14:2–20.
Winger, P. V. 1978. Fish and benthic populations of the New River, Tennessee. Pages 190–216 *in* D. E. Samuel, J. R. Stauffer, C. H. Hocutt, and W. T. Mason, editors. Surface mining and fish/wildlife needs in the eastern United States. U.S. Fish and Wildlife Service, Biological Services Program FWS/OBS-78/81.
Woodin, B. R., and J. J. Stegeman. 1993. Elevated P4501A protein in intertidal fish in Prince William Sound associated with the *Exxon Valdez* oil spill. Marine Environmental Research 35:203–204.
Woodward, D. F., W. G. Brumbaugh, A. J. DeLonay, E. E. Little, and C. E. Smith. 1994. Effects on rainbow trout fry of a metals-contaminated diet of benthic invertebrates from the Clark Fork River, Montana. Transactions of the American Fisheries Society 123:51–62.
Woodward, D. F., and R. G. Riley. 1983. Petroleum hydrocarbon concentrations in a salmonid stream contaminated byoil field discharge water and effects on macrobenthos. Archives of Environmental Contamination and Toxicology 12:327–334.
Wren, C. D. 1991. Cause–effect linkages between chemicals and populations of mink (*Mustela vison*) and otter (*Lutra canadensis*) in the Great Lakes basin. Journal of Toxicology and Environmental Health 33:549–585.
Wright, R., and E. Snevik. 1978. Acid precipitation: chemistry and fish populations in 700 lakes in southernmost Norway. Internationale Vereinigung fuer Theoretische und Angewandte Limnologie Verhandlungen 20:765–775.

A History of Aquatic Toxicology

Carson, R. 1962. Silent spring. Houghton-Mifflin, Boston, Mass. 368 pp.

Endocrine-Disrupting Compounds in the Environment

Begley, S., and D. Glick. 1994. The estrogen complex. Newsweek. March 21, 1976–77.
Carlsen, E., A. Giwereman, N. Keiding, and N. E. Skakkebaek. 1992. Evidence for decreasing sperm quality of semen during the past 50 years. British Medical Journal 304:609–613.
Colborn, T., F. S. vom Saal, and A. M. Soto. 1993. Developmental effects of endocrine-disrupting chemicals in wildlife and humans. Environmental Health Perspectives 101:378–384.
Fox, G. A., A. P. Gilman, D. B. Peakall, and F. W. Anderka. 1978. Aberrant behavior of nesting gulls. Journal of Wildlife Management 42:477–483.
Kelce, W. R., C. R. Stone, S. L. Laws, L. E. Gray, J. A. Kemppaainen, and E. M. Wilson. 1995. Persistent DDT metabolite p,p'–DDE is a potent androgen receptor antagonist. Nature 375:581–585.
Raloff, J. 1994. The gender benders—are environmental "hormones" emasculating wildlife? Science News 145:24–27.
Reis, L. A. G., G. A. Miller, B. F. Hankey, C. L. Kosary, A. Harras, and B. K. Edwards, editors. 1994. SEER cancer statistics review, 1973–1991: tables and graphs. National Institute of Health Publication 94–2789, National Cancer Institute, Bethesda, Md.