CHAPTER 26

Basics of Environmental Toxicology

GERALD A. LEBLANC

26.1 INTRODUCTION

Industrial and agricultural endeavors are intimately associated with the extensive use of a wide array of chemicals. Historically chemical wastes generated through industrial processes were disposed of through flagrant release into the environment. Gasses quickly dispersed into the atmosphere; liquids were diluted into receiving waters and efficiently transported away from the site of generation. Similarly pesticides and other agricultural chemicals revolutionized farm and forest productivity. Potential adverse effects of the application of such chemicals to the environment were viewed as insignificant relative to the benefits bestowed by such practices. Then in 1962, a science writer for the US Fish and Wildlife Service, Rachel Carson, published a book that began by describing a world devoid of birds and from which the title *The Silent Spring* was inspired. In her book Ms. Carson graphically described incidents of massive fish and bird kills resulting from insecticide use in areas ranging from private residences to national forests. Further she inferred that such pollutant effects on wildlife may be heralding similar incipient effects on human health.

The resulting awakening of the general public to the hazards of chemicals in the environment spurred several landmark activities related to environmental protection, including Earth Day, organization of the US Environmental Protection Agency, and the enactment of several pieces of legislation aimed at regulating and limiting the release of chemicals into the environment. Appropriate regulation of the release of chemicals into the environment without applying unnecessarily stringent limitation on industry and agriculture requires a comprehensive understanding of the toxicological properties and consequences of release of the chemicals into the environment. It was from this need that modern environmental toxicology evolved.

Environmental toxicology is defined as the study of the fate and effects of chemicals in the environment. Although this definition would encompass toxic chemicals naturally found in the environment (i.e., animal venom, microbial and plant toxins), environmental toxicology is typically associated with the study of environmental chemicals of anthropogenic origin. Environmental toxicology can be divided into two subcategories:

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environmental health toxicology and ecotoxicology. Environmental health toxicology is the study of the adverse effects of environmental chemicals on human health, while ecotoxicology focuses upon the effects of environmental contaminants upon ecosystems and constituents thereof (fish, wildlife, etc.). Assessing the toxic effects of chemicals on humans involves the use of standard animal models (i.e., mouse and rat) as well as epidemiological evaluations of exposed human populations (i.e., farmers and factory workers). In contrast, ecotoxicology involves the study of the adverse effects of toxicants on myriad of organisms that compose ecosystems ranging from microorganisms to top predators. Further, comprehensive insight into the effects of chemicals in the environment requires assessments ancillary to toxicology such as the fate of the chemical in the environment (Chapter 27), and toxicant interactions with abiotic (nonliving) components of ecosystems. Comprehensive assessments of the adverse effects of environmental chemicals thus utilize expertise from many scientific disciplines. The ultimate goal of these assessments is elucidating the adverse effects of chemicals that are present in the environment (retrospective hazard assessment) and predicting any adverse effects of chemicals before they are discharged into the environment (prospective hazard assessment). The ecological hazard assessment process is discussed in Chapter 28.

Historically chemicals that have posed major environmental hazards tend to share three insidious characteristics: environmental persistence, the propensity to accumulate in living things, and high toxicity.

26.2 ENVIRONMENTAL PERSISTENCE

Many abiotic and biotic processes exist in nature that function in concert to eliminate (i.e., degrade) toxic chemicals. Accordingly many chemicals released into the environment pose minimal hazard simply because of their limited life span in the environment. Chemicals that have historically posed environmental hazard (i.e., DDT, PCBs, TCDD) resist degradative processes and accordingly persist in the environment for extremely long periods of time (Table 26.1). Continued disposal of persistent chemicals into the environment can result in their accumulation to environmental levels sufficient to pose toxicity. Such chemicals can continue to pose hazard long after their disposal into the environment has ceased. For example, significant contamination of Lake Ontario by the pesticide mirex occurred from the 1950s through the 1970s. Mass balance studies performed 20 years later revealed that 80% of the mirex deposited into the lake

Table 26.1 Environmental Half-lives of Some Chemical Contaminants

Contaminant	Half-life	Media
DDT	10 Years	Soil
TCDD	9 Years	Soil
Atrazine	25 Months	Water
Benzoperylene (PAH)	14 Months	Soil
Phenanthrene (PAH)	138 Days	Soil
Carbofuran	45 Days	Water

persisted. One decade following the contamination of Lake Apopka, Florida, with pesticides including DDT and diclofol, populations of alligators continued to experience severe reproductive impairment. Both biotic and abiotic processes contribute to the degradation of chemicals.

26.2.1 Abiotic Degradation

A plethora of environmental forces compromise the structural integrity of chemicals in the environment. Many prominent abiotic degradative processes occur due to the influences of light (photolysis) and water (hydrolysis).

Photolysis. Light, primarily in the ultraviolet range, has the potential to break chemical bonds and thus can contribute significantly to the degradation of some chemicals. Photolysis is most likely to occur in the atmosphere or surface waters where light intensity is greatest. Photolysis is dependent upon both the intensity of the light and the capacity of the pollutant molecules to absorb the light. Unsaturated aromatic compounds such as the polycyclic aromatic hydrocarbons tend to be highly susceptible to photolysis due to their high capacity to absorb light energy. Light energy can also facilitate the oxygenation of environmental contaminants via hydrolytic or oxidative processes. The photooxidation of the organophosphorus pesticide parathion is depicted in Figure 26.1.

Hydrolysis. Water, often in combination with light energy or heat, can break chemical bonds. Hydrolytic reactions commonly result in the insertion of an oxygen atom into the molecule with the commensurate loss of some component of the molecule. Ester bonds, such as those found in organophosphate pesticides (i.e., parathion; Figure 26.1), are highly susceptible to hydrolysis which dramatically lowers the environmental half-lives of these chemicals. Hydrolytic rates of chemicals are influenced by the temperature and pH of the aqueous media. Rates of hydrolysis increase with increasing temperature and with extremes in pH.

26.2.2 Biotic Degradation

While many environmental contaminants are susceptible to abiotic degradative processes, such processes often occur at extremely slow rates. Environmental degradation of chemical contaminants can occur at greatly accelerated rates through the action of microorganisms. Microorganisms (primarily bacteria and fungi) degrade chemicals in an effort to derive energy from these sources. These biotic degradative processes are enzyme mediated and typically occur at rates that far exceed abiotic degradation. Biotic degradative processes can lead to complete mineralization of chemicals to water, carbon dioxide, and basic inorganic constituents. Biotic degradation includes those processes associated with abiotic degradation (i.e., hydrolysis, oxidation) and processes such as the removal of chlorine atoms (dehalogenation), the scission of ringed structures (ring cleavage), and the removal of carbon chains (dealkylation). The process by which microorganisms are used to facilitate the removal of environmental contaminants is called bioremediation.

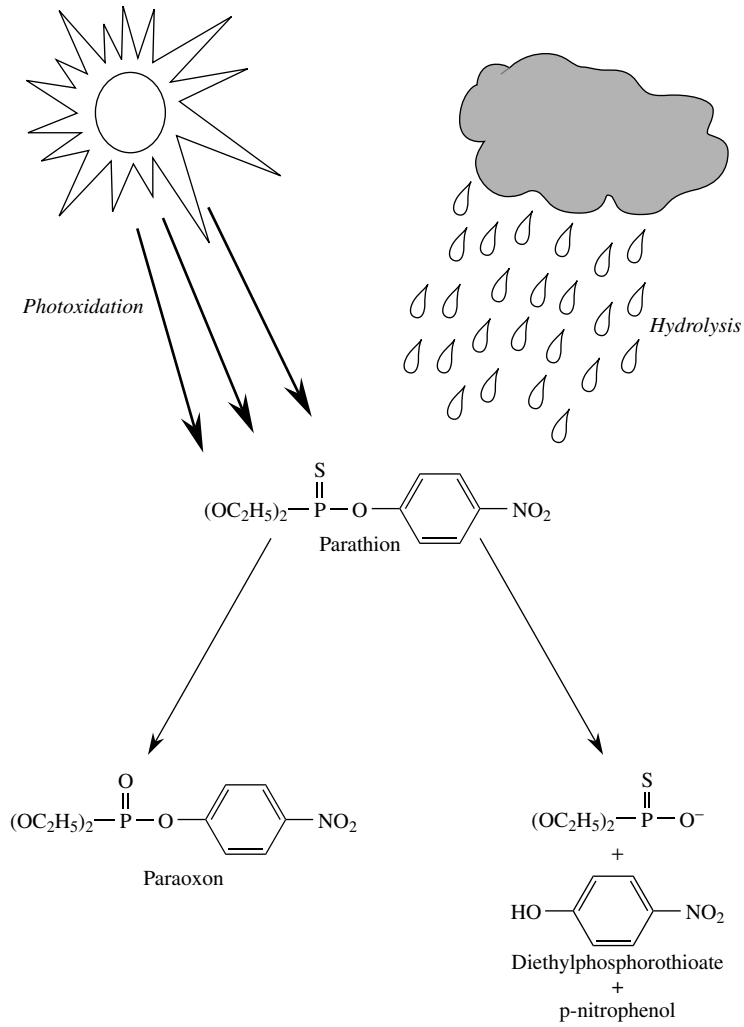

Figure 26.1 The effect of sunlight (photooxidation) and precipitation (hydrolysis) on the degradation of parathion.

26.2.3 Nondegradative Elimination Processes

Many processes are operative in the environment that contribute to the regional elimination of a contaminant by altering its distribution. Contaminants with sufficiently high vapor pressure can evaporate from contaminated terrestrial or aquatic compartments and be transferred through the atmosphere to new locations. Such processes of global distillation are considered largely responsible for the worldwide distribution of relatively volatile organochlorine pesticides such as lindane and hexachlorobenzene. Entrainment by wind and upper atmospheric currents of contaminant particles or dust onto which the contaminants are sorbed also contribute to contaminant redistribution. Sorption of contaminant to suspended solids in an aquatic environment with commensurate sedimentation can result with the removal of contaminants from the water

column and its redistribution into bottom sediments. Sediment sorption of contaminants greatly reduces bioavailability, since the propensity of a lipophilic chemical to partition from sediments to organisms is significantly less than its propensity to partition from water to organism. More highly water soluble contaminants can be removed and redistributed through runoff and soil percolation. For example, the herbicide atrazine is one of the most abundantly used pesticides in the United States. It is used to control broadleaf and weed grasses in both agriculture and landscaping. Atrazine is ubiquitous in surface waters due to its extensive use. A study of midwestern states revealed that atrazine was detectable in 92% of the reservoirs assayed. In addition atrazine has the propensity to migrate into groundwater because of its relatively high water solubility and low predilection to sorb to soil particles. Indeed, field studies have shown that surface application of atrazine typically results in the contamination of the aquifer below the application site. A more detailed account of the fate of chemicals in the environment is presented in Chapter 27.

26.3 BIOACCUMULATION

Environmental persistence alone does not render a chemical problematic in the environment. If the chemical cannot enter the body of organisms, then it would pose no threat of toxicity (see Chapter 6). Once absorbed, the chemical must accumulate in the body to sufficient levels to elicit toxicity. Bioaccumulation is defined as the process by which organisms accumulate chemicals both directly from the abiotic environment (i.e., water, air, soil) and from dietary sources (trophic transfer). Environmental chemicals are largely taken up by organisms by passive diffusion. Primary sites of uptake include membranes of the lungs, gills, and gastrointestinal tract. While integument (skin) and associated structures (scales, feathers, fur, etc.) provide a protective barrier against many environmental insults, significant dermal uptake of some chemicals can occur. Because the chemicals must traverse the lipid bilayer of membranes to enter the body, bioaccumulation potential of chemicals is positively correlated with lipid solubility (lipophilicity).

The aquatic environment is the major site at which lipophilic chemicals traverse the barrier between the abiotic environment and the biota. This is because (1) lakes, rivers, and oceans serve as sinks for these chemicals, and (2) aquatic organisms pass tremendous quantities of water across their respiratory membranes (i.e., gills) allowing for the efficient extraction of the chemicals from the water. Aquatic organisms can bioaccumulate lipophilic chemicals and attain body concentrations that are several orders of magnitude greater that the concentration of the chemical found in the environment (Table 26.2). The degree to which aquatic organisms accumulate xenobiotics from the environment is largely dependent on the lipid content of the organism, since body lipids serve as the primary site of retention of the chemicals (Figure 26.2).

Chemicals can also be transferred along food chains from prey organism to predator (trophic transfer). For highly lipophilic chemicals, this transfer can result in increasing concentrations of the chemical with each progressive link in the food chain (biomagnification). As depicted in Figure 26.3, a chemical that bioaccumulates by a factor of 2 regardless of whether the source of the contaminant is the water or food would have the potential to magnify at each trophic level leading to high levels in the birds of

Table 26.2 Bioaccumulation of Some Environmental Contaminants by Fish

Chemical	Bioaccumulation Factor[a]
DDT	127,000
TCDD	39,000
Endrin	6,800
Pentachlorobenzene	5,000
Lepthophos	750
Trichlorobenzene	183

Source: Data derived from G. A. LeBlanc, 1994, *Environ. Sci. Technol*. **28**: 154–160.

[a]Bioaccumulation factor is defined as the ratio of the chemical concentration in the fish and in the water at steady-state equilibrium.

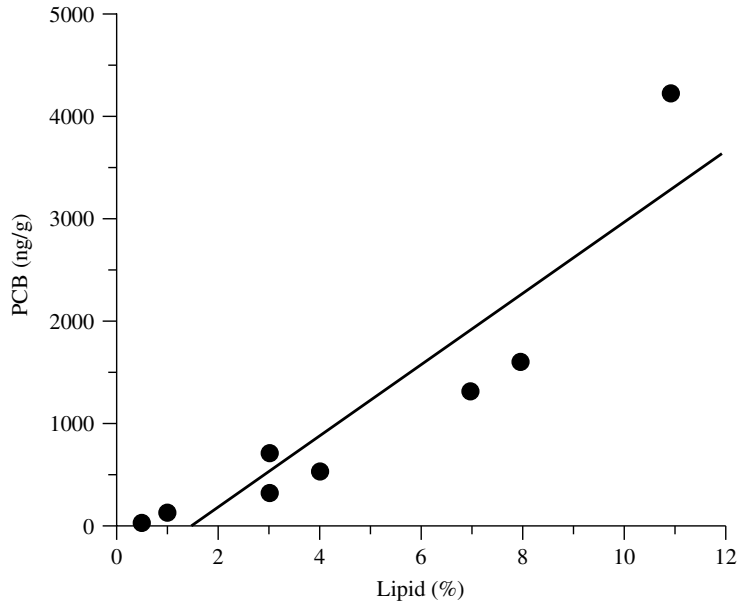

Figure 26.2 Relationship between lipid content of various organisms sampled from Lake Ontario and whole body PCB concentration (Data derived from B. G. Oliver and A. J. Niimi, *Environ. Sci. Technol*. **22**: 388–397, 1988.)

prey relative to that found in the abiotic environment. It should be noted that bioaccumulation is typically much greater from water than from food, and it is unlikely that an organism would accumulate a chemical to the same degree from both sources. The food-chain transfer of DDT was responsible for the decline in many bird-eating raptor populations that contributed to the decision to ban the use of this pesticide in the United States.

Bioaccumulation can lead to a delayed onset of toxicity, since the toxicant may be initially sequestered in lipid deposits but is mobilized to target sites of toxicity

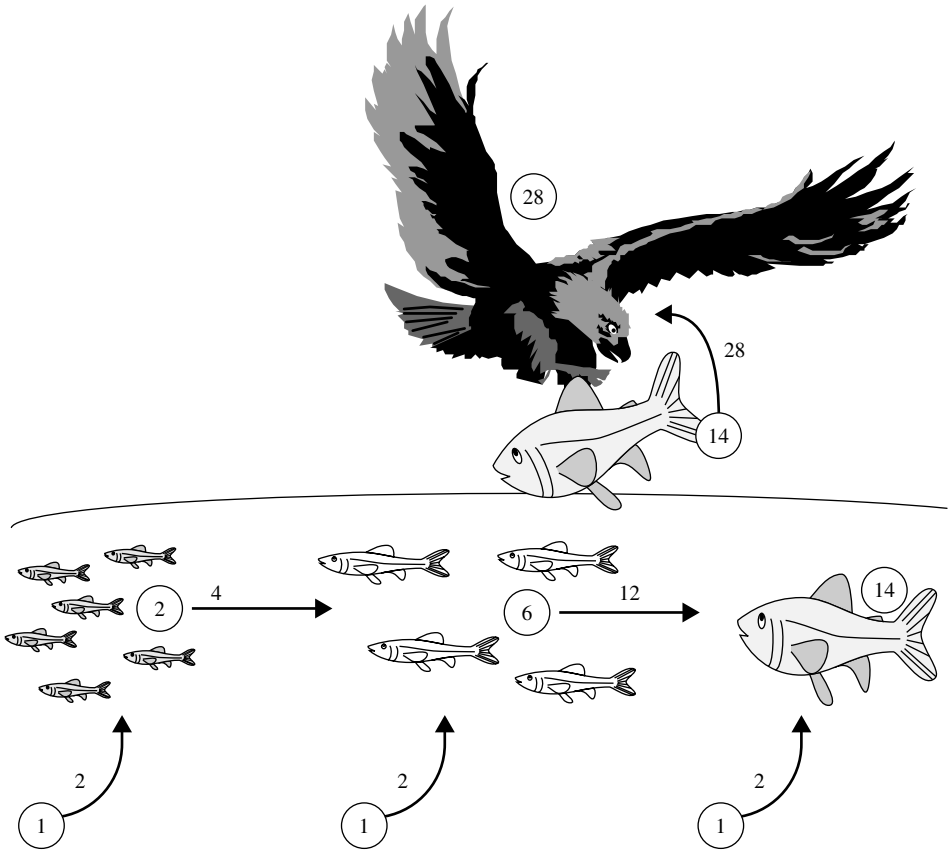

Figure 26.3 Bioaccumulation of a chemical along a generic food chain. In this simplistic paradigm, the amount of the chemical in the water is assigned an arbitrary concentration of 1, and it is assumed that the chemical will bioaccumulate either from the water to the fish or from one trophic level to another by a factor of 2. Circled numbers represent the concentration of chemical in the respective compartment. Numbers associated with arrows represent the concentration of chemical transferred from one compartment to another.

when these lipid stores are utilized. For example, lipid stores are often mobilized in preparation for reproduction. The loss of the lipid can result in the release of lipophilic toxicants rendering them available for toxic action. Such effects can result in mortality of adult organisms as they approach reproductive maturity. Lipophilic chemicals also can be transferred to offspring in lipids associated with the yolk of oviparous organisms or the milk of mammals, resulting in toxicity to offspring that was not evident in the parental organisms.

26.3.1 Factors That Influence Bioaccumulation

The propensity for an environmental contaminant to bioaccumulate is influenced by several factors. The first consideration is environmental persistence. The degree to

Table 26.3 Measured and Predicted Bioaccumulation Factors in Fish of Chemicals That Differ in Susceptibility to Biotransformation

Chemical	Susceptibility to Biotransformation	Bioaccumulation Factor	
		Predicted	Measured
Chlordane	Low	47,900	38,000
PCB	Low	36,300	42,600
Mirex	Low	21,900	18,200
Pentachloro-phenol	High	4,900	780
Tris(2,3-dibromo-propyl)phosphate	High	4,570	3

Source: Predicted bioaccumulation factors were based upon their relative lipophilicity as described by, D. Mackay, *Environ. Sci. Technol.* 1982, **16**: 274–278.

which a chemical bioaccumulates is dictated by the concentration present in the environment. Contaminants that are readily eliminated from the environment will generally not be available to bioaccumulate. An exception would be instances where the contaminant is continuously introduced into the environment (i.e., receiving water of an effluent discharge).

As discussed above, lipophilicity is a major determinant of the bioaccumulation potential of a chemical. However, lipophilic chemicals also have greater propensity to sorb to sediments, thus rendering them less available to bioaccumulate. For example, sorption of benzo[a]pyrene to humic acids reduced its propensity to bioaccumulate in sunfish by a factor of three. Fish from oligotrophic lakes, having low suspended solid levels, have been shown to accumulate more DDT than fish from eutrophic lakes that have high suspended solid contents.

Once absorbed by the organism, the fate of the contaminant will influence its bioaccumulation. Chemicals that are readily biotransformed (Chapter 7) are rendered more water soluble and less lipid soluble. The biotransformed chemical is thus less likely to be sequestered in lipid compartments and more likely to be eliminated from the body. As depicted in Table 26.3, chemicals that are susceptible to biotransformation, bioaccumulate much less than would be predicted based on lipophilicity. Conjugation of xenobiotics to glutathione and glucuronic acid (Chapter 7) can target the xenobiotic for biliary elimination through active transport processes thus greatly increasing the rate of elimination (Chapter 10). Differences in chemical elimination rates contribute to species differences in bioaccumulation.

26.4 TOXICITY

26.4.1 Acute Toxicity

Acute toxicity is defined as toxicity elicited as a result of short-term exposure to a toxicant. Incidences of acute toxicity in the environment are commonly associated with accident (i.e., derailment of a train resulting in leakage of a chemical into a river) or imprudent use of the chemical (i.e., aerial drift of a pesticide to nontarget areas). Discharge limits placed upon industrial and municipal wastes, when adhered to, have been generally successful in protecting against acute toxicity to organisms in waste-receiving areas. As discussed in Chapter 11, the acute toxicity of a chemical is commonly quantified as the LC50 or LD50. These measures do not provide any insight

Table 26.4 Ranking Scheme for Assessing the Acute Toxicity of Chemicals to Fish and Wildlife

Fish LC50 (mg/L)	Avian/Mammalian LD50 (mg/kg)	Toxicity Rank	Example Contaminant
>100	>5000	Relatively nontoxic	Barium
10–100	500–5000	Moderately toxic	Cadmium
1–10	50–500	Very toxic	1,4-Dichlorobenzene
<1	<50	Extremely toxic	Aldrin

into the environmentally acceptable levels of contaminants (a concentration that kills 50% of the exposed organisms is hardly tolerable). However, LC50 and LD50 values do provide statistically sound, reproducible measures of the relative acute toxicity of chemicals. LC50 and LD50 ranges for aquatic and terrestrial wildlife, respectively, and their interpretation are presented in Table 26.4.

Acute toxicity of environmental chemicals is determined experimentally with select species that serve as representatives of particular levels of trophic organization within an ecosystem (i.e., mammal, bird, fish, invertebrate, vascular plant, algae). For example, the US Environmental Protection Agency requires acute toxicity tests with representatives of at least eight different species of freshwater and marine organisms (16 tests) that include fish, invertebrates, and plants when establishing water quality criteria for a chemical. Attempts are often made to rank classes of organisms by toxicant sensitivity; however, no organism is consistently more or less susceptible to the acute toxicity of chemicals. Further the use of standard species in toxicity assessment presumes that these species are "representative" of the sensitivity of other members of that level of ecological organization. Such presumptions are often incorrect.

26.4.2 Mechanisms of Acute Toxicity

Environmental chemicals can elicit acute toxicity by many mechanisms. Provided below are example mechanisms that are particularly relevant to the types of chemicals that are more commonly responsible for acute toxicity in the environment at the present time.

Cholinesterase Inhibition. The inhibition of cholinesterase activity is characteristic of acute toxicity associated with organophosphate and carbamate pesticides (see Chapter 11 for more detail on cholinesterase inhibition). Forty to 80% inhibition of brain cholinesterase activity is typically reported in lethally poisoned fish. Acute toxicity resulting from cholinesterase inhibition is relatively common among incidents of acute poisoning of fish and birds due to the high volume usage of organophosphates and carbamates in applications such as lawn care, agriculture, and golf course maintenance. Cholinesterase inhibition in fish may occur following heavy rains in aquatic habitats adjacent to areas treated with the pesticides and subject to runoff from these areas. Acute toxicity to birds commonly occurs in birds that feed in areas following application of the pesticides.

Narcosis. A common means by which industrial chemicals elicit acute toxicity, particularly to aquatic organisms, is through narcosis. Narcosis occurs when a chemical accumulates in cellular membranes interfering with the normal function of the membranes. Typical responses to the narcosis are decreased activity, reduced reaction to external stimuli, and increased pigmentation (in fish). The effects are reversible, and nonmoribund organisms typically return to normal activity once the chemical is removed from the organism's environment. Prolonged narcosis can result in death. Approximately 60% of industrial chemicals that enter the aquatic environment elicit acute toxicity through narcosis. Chemicals that elicit toxicity via narcosis typically do not elicit toxicity at specific target sites and are sufficiently lipophilic to accumulate in the lipid phase or the lipid-aqueous interface of membranes to sufficient levels to disrupt membrane function. Chemicals that induce narcosis include alcohols, ketones, benzenes, ethers, and aldehydes.

Physical Effects. Perhaps most graphic among recent incidents of environmental acute toxicity is the physical effects of petroleum following oil spills. Slicks of oil on the surface of contaminated waters results in the coating of animals, such as birds and marine mammals, that frequent the air-water interface. Such a spill of unprecedented magnitude and consequence in the United States occurred on March 24, 1989, when the hull of the Exxon Valdez was ruptured on Bligh Reef in Prince William Sound, Alaska. Nearly 11 million gallons of crude oil spilled onto the nearshore waters killing more wildlife than any prior oil spill in history. Thousands of sea birds and mammals succumbed to the acute effects of the oil.

Hypothermia is considered a major cause of death of oiled marine birds and mammals. These organisms insulate themselves from the frigid waters by maintaining a layer of air among the spaces within their coat of fur or feathers. The oil penetrates the fur/feather barrier and purges the insulating air. As a result the animals rapidly succumb to hypothermia. In addition to hypothermia, these animals can also experience oil toxicosis. Inhalation of oil, as well as ingestion through feeding and preening, can result in the accumulation of hydrocarbons to toxic levels. Toxicity to sea otters has been correlated to degree of oiling and is characterized by pulmonary emphysema (bubbles of air within the connective tissue of the lungs), gastric hemorrhages, and liver damage.

26.4.3 Chronic Toxicity

Chronic toxicity is defined as toxicity elicited as a result of long-term exposure to a toxicant. Sublethal end points are generally associated with chronic toxicity. These include reproductive, immune, endocrine, and developmental dysfunction. However, chronic exposure also can result in direct mortality not observed during acute exposure. For example, chronic exposure of highly lipophilic chemicals can result in the eventual bioaccumulation of the chemical to concentrations that are lethal to the organisms. Or as discussed previously, mobilization of lipophilic toxicants from lipid compartments during reproduction may result in lethality. It is important to recognize that, while theoretically, all chemicals elicit acute toxicity at a sufficiently high dose, all chemicals are not chronically toxic. Chronic toxicity is measured by end points such as the highest level of the chemical that does not elicit toxicity during continuous, prolonged exposure (no observed effect level, NOEL), the lowest level of the chemical that elicits

Table 26.5 Acute and Chronic Toxicity of Pesticides Measured from Laboratory Exposures of Fish Species

Pesticide	LC50 (μg/L)	Acute Toxicity	Chronic Value (μg/L)	ACR	Chronic Toxicity
Endosulfan	166	Extremely toxic	4.3	39	Yes
Chlordecone	10	Extremely toxic	0.3	33	Yes
Malathion	3,000	Very toxic	340	8.8	No
Carbaryl	15,000	Moderately toxic	378	40	Yes

toxicity during continuous, prolonged exposure (lowest observed effect level, LOEL), or the chronic value (CV) which is the geometric mean of the NOEL and the LOEL. Chronic toxicity of a chemical is often judged by the acute : chronic ratio (ACR), which is calculated by dividing the acute LC50 value by the CV. Chemicals that have an ACR of less than 10 typically have low to no chronic toxicity associated with them (Table 26.5).

The following must always be considered when assessing the chronic toxicity of a chemical: (1) Simple numerical interpretations of chronic toxicity based on ACRs serve only as gross indicators of the potential chronic toxicity of the chemical. Laboratory exposures designed to establish chronic values most often focus upon a few general endpoints such as survival, growth, and reproductive capacity. Examination of more subtle end points of chronic toxicity may reveal significantly different chronic values. (2) Laboratory exposures are conducted with a few test species that are amenable to laboratory manipulation. The establishment of chronic and ACR values with these species should not be considered absolute. Toxicants may elicit chronic toxicity in some species and not in others. (3) Interactions among abiotic and biotic components of the environment may contribute to the chronic toxicity of chemicals, while such interactions may not occur in laboratory assessments of direct chemical toxicity. These considerations are exemplified in the following incidence of chronic toxicity of chemicals in the environment.

26.4.4 Species-Specific Chronic Toxicity

Tributyltin-Induced Imposex in Neogastropods. Scientists noted in the early 1970s that dogwhelks inhabiting the coast of England exhibited a hermaphroditic-like condition whereby females possessed a penis in addition to normal female genitalia. While hermaphroditism is a reproductive strategy utilized by some molluscan species, dogwhelks are dioecious. This pseudohermaphroditic condition, called imposex, has since been documented worldwide in over 140 species of neogastropods. Imposex has been implicated in reduced fecundity of neogastropod populations, population declines, and local extinction of affected populations.

The observation that imposex occurred primarily in marinas suggested causality with some contaminant originating from such facilities. Field experiments demonstrated that neogastropods transferred from pristine sites to marinas often developed imposex. Laboratory studies eventually implicated tributyltin, a biocide used in marine paints, as the cause of imposex. Tributyltin is toxic to most marine species evaluated in the

Table 26.6 Toxicity of Tributyltin to Aquatic Organisms

Species	Acute Toxicity (LC50, μg/L)	Chronic Toxicity (LOEL, μg/L)	Imposex (μg/L)
Daphnid	1.7	—	—
Polychaete worm	—	0.10	—
Copepod	1.0	0.023	—
Oyster	1.3	0.25	—
Dogwhelk	—	—	≤0.0010

laboratory at low parts-per-billion concentrations (Table 26.6). However, exposure of neogastropods to low parts-per-trillion concentrations can cause imposex (Table 26.6). Thus neogastropods are uniquely sensitive to the toxicity of tributyltin, with effects produced that were not evident in standard laboratory toxicity characterizations.

Atrazine-Induced Hermaphroditism in Frogs. The herbicide atrazine historically has been considered environmentally safe for use since the material has proved to be only slightly to moderately toxic in standard fish and wildlife toxicity evaluations. Measured atrazine levels in surface waters rarely exceed 0.04 mg/L. The acute and chronic toxicities of atrazine to aquatic organisms are typically in excess of 1 mg/L. Thus ample safety margins appear to exist for this compound. Recent studies with frogs have revealed that exposure to 0.0001 mg/L atrazine through the period of larval development caused the frogs to develop both a testis and an ovary. The toxicological significance of this chemical-induced hermaphroditic condition is not known. However, environmentally relevant levels of the herbicide appear to have the potential to adversely impact reproductive success of these organisms.

26.4.5 Abiotic and Biotic Interactions

Chlorofluorocarbons–Ozone–UV-B Radiation–Amphibian Interactions. The atmospheric release of chlorofluorocarbons has been implicated in the depletion of the earth's stratospheric ozone layer which serves as a filter against harmful ultraviolet radiation. Temporal increases in UV-B radiation have been documented and pose increasing risks of a variety of maladies to both plant and animal life.

Commensurate with the increase in UV-B radiation levels at the earth's surface has been the decline in many amphibian populations. Multiple causes may be responsible for these declines including loss of habitat, pollutants, and increased incidence of disease; however, recent studies suggest that increases in UV-B radiation may be a major contributor to the decline in some populations. Field surveys in the Cascade Mountains, Oregon, revealed a high incidence of mortality among embryos of the Cascades frog and western toad. Incubation of eggs, collected from the environment, in the laboratory along with the pond water in which the eggs were collected resulted in low mortality, suggesting that contaminants in the water were not directly responsible for the mortality. Furthermore placement of UV-B filters over the embryos, incubated under ambient environmental conditions, significantly increased viability of the embryos.

Several amphibian species were examined for photolyase activity. This enzyme is responsible for the repair of DNA damage caused by UV-B radiation. A more than

80-fold difference in photolyase activity was observed among the species examined. Photolyase activity was appreciably lower in species known to be experiencing population decline as compared to species showing stable population levels. Recent studies have also suggested that ambient UV-B radiation levels can enhance the susceptibility of amphibian embryos to mortality originating from fungal infection.

These observations suggest that chlorofluorocarbons may be contributing to the decline in amphibian populations. However, this toxicological effect is the result of abiotic interactions (i.e., chlorofluorocarbons depleting atmospheric ozone levels, which increase UV-B radiation penetration resulting in embryo mortality) (Figure 26.4). In addition abiotic (UV-B) and biotic (fungus) interactions may also be contributing to the toxicity. Such effects would not be predicted from direct laboratory assessments of the toxicity of chlorofluorocarbons to amphibians and highlight the necessity to consider possible indirect toxicity associated with environmental contaminants.

Masculinization of Fish due to Microbial Interactions with Kraft Pulpmill Effluent. Field surveys of mosquito fish populations in the state of Florida revealed populations containing females that exhibited male traits such as male-type mating behavior and the modification of the anal fin to resemble the sperm-transmitting gonopodium of males. Masculinized females were found to occur downstream of kraft pulpmill effluents suggesting that components of the effluent were responsible for the masculinizing effect. Direct toxicity assays performed with the effluent did not produce such effects. However, the inclusion of microorganisms along with the effluent resulted in masculinization. Further studies revealed that phytosterols present in the kraft pulpmill effluent can be converted to androgenic C19 steroids by microorganisms and these steroids are capable of masculinizing female fish (Figure 26.5).

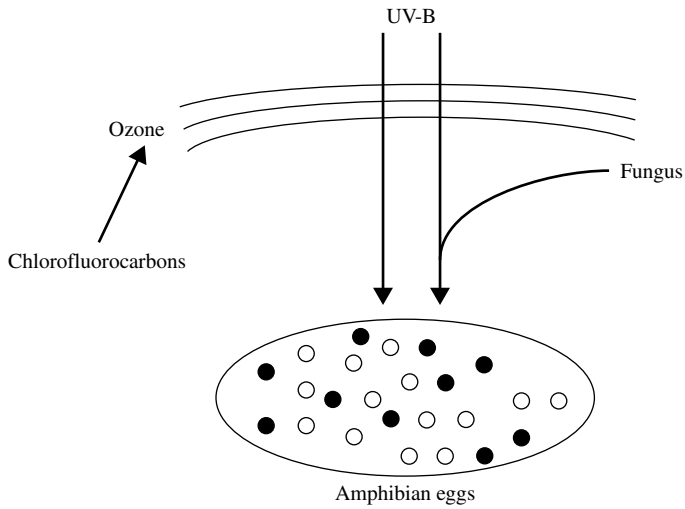

Figure 26.4 Abiotic and biotic interactions leading to the indirect toxicity of chlorofluorocarbons to amphibians. Atmospheric release of chlorofluorocarbons causes the depletion of the stratospheric ozone layer (abiotic-abiotic interaction). Depleted ozone allows for increased penetration of UV-B radiation (abiotic-abiotic interaction). UV-B radiation alone and in combination with fungus (abiotic-biotic interaction) causes increased mortality of amphibian embryos.

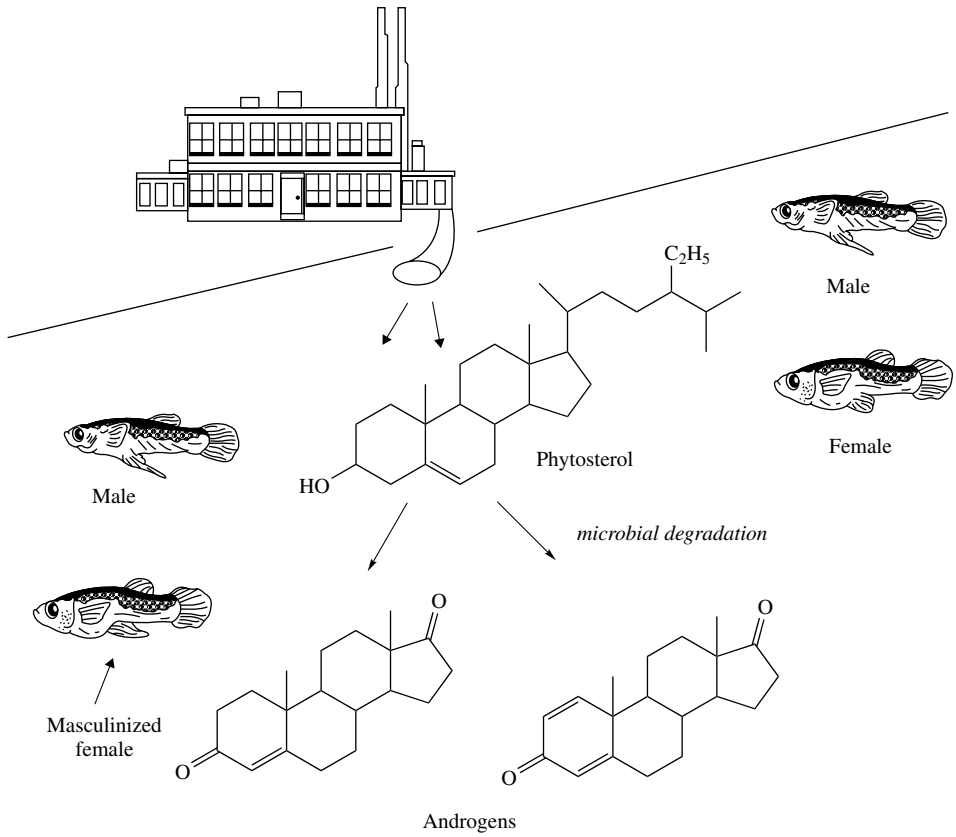

Figure 26.5 Indirect toxicity of kraft pulpmill effluent to mosquito fish. Phytosterols in the mill effluent are converted to C19 steroidal androgens through the action of microorganisms in the environment. These androgens masculinize both anatomy and behavior of female mosquito fish. An arrow identifies the modified anal fin on the masculinized female.

Thus abiotic (phytosterols) : biotic (microorganisms) interactions in the environment must occur before this occult toxicity associated with the kraft pulpmill effluent is unveiled.

Environmental Contaminants and Disease among Marine Mammals. Massive mortality have occurred over the past 20 years among populations of harbor seals, bottlenose dolphins, and other marine mammals worldwide. In many instances this mortality has been attributed to disease. For example, nearly 18,000 harbor seals died in the North, Irish, and Baltic seas in the late 1980s due to phocine distemper virus. Incidences of the disease outbreak were highest in areas containing high levels of pollutants, and seals that succumbed to the disease were found to have high tissue levels of polychlorinated biphenyls (PCBs). PCBs and other organochlorine chemicals such as DDT, hexachlorobenzene, and dieldrin have been shown to immunosuppress laboratory animals, and accumulation of these chemicals by the seals may have increased their susceptibility to the virus. This hypothesis was tested by feeding fish, caught either from a relatively pristine area or from a polluted coastal area, to seals for 93 weeks then

assessing the integrity of the immune system in the seals. Seals fed the contaminated fish did indeed have impaired immune responses lending credence to the hypothesis that organochlorine contaminants in the marine environment are rendering some species immunodeficient. Mortality occurs, not as a direct result of chemical toxicity, but due to increased susceptibility to pathogens.

26.5 CONCLUSION

Environmental toxicologists have learned a great deal about the effects of chemicals in the environment and the characteristics of chemicals that are responsible for the hazards they pose. Much of the information gained has been due to retrospective analyzes of the environmental consequences of the deposition of chemicals into the environment. Such analyzes have resulted in curtailing the release of demonstrated hazardous chemicals into the environment and provide benchmark information upon which the regulation of chemicals proposed for release into the environment can be based. The recognition that environmentally hazardous chemicals commonly share characteristics of persistence, potential to bioaccumulate, and high toxicity has resulted in development and use of chemicals that lack one or more of these characteristics yet fulfill societal needs previously served by hazardous chemicals. For example, recognition that persistence and propensity to bioaccumulate were largely responsible for the environmental hazards posed by many organochlorine pesticides led to the development and use of alternative classes of pesticides such as organophosphates, carbamates, and pyrethroids. While these chemicals all possess the toxicity necessary to function as pesticides, their lack of persistence and reduced propensity to bioaccumulate makes them more suitable for use in the environment.

Such advances in our understanding of the fate and effects of chemicals in the environmental does not imply that the role of environmental toxicologists in the twenty-first century will diminish. A dearth of information persist in areas vital to continued protection of natural resources against chemical insult. These include understanding (1) the unique susceptibilities of key species to the toxicity of different classes of chemicals, (2) the interactions of chemical contaminants with abiotic components of the environment that lead to increased toxicity, (3) the toxicological consequences of exposure to complex chemical mixtures, and (4) the consequences of toxicant effects on individuals with respect to ecosystem viability. Additionally continued research is needed to develop molecular and cellular biomarkers of toxicant exposure and effect that could be used to predict dire consequences to ecosystem before such effects are manifested at higher levels of biological organization. The role of the environmental toxicologist undoubtedly will increase in prospective activities aimed at reducing the risk associated with chemical contaminants in the environments before problems arise, and hopefully will decrease with respect to assessing damage caused by such environmental contaminants.

SUGGESTED READING

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