
Tinkering with the Tinkerer: Pollution versus Evolution

Glen A. Fox

Canadian Wildlife Service, Environment Canada, Hull, Quebec, Canada

Pollutants can act as powerful selective forces by altering genetic variability, its intergenerational transfer, and the size, functional viability, adaptability, and survival of future generations. It is at the level of the cell and the individual that meiosis occurs, that genetic diversity is maintained, and behavior, reproduction, growth, and survival occur and are regulated. It is at this level that evolutionary processes occur and most pollutants exert their toxic effects. Chronic exposure to chemicals contributes to the cumulative stress on individuals and disrupts physiological processes and chemically mediated communication thereby threatening the diversity and long-term survival of sexually reproducing biota. Regional or global effects of pollution on the atmosphere, hydrosphere, and lithosphere have indirectly altered Earth's life-support systems, thereby modifying trace metal balance, reproduction, and incidence of UV-B-induced DNA damage in biota. By altering the competitive ability and survival of species, chemical pollutants potentially threaten evolutionary processes and the biodiversity and function of intercepting ecosystems.— *Environ Health Perspect* 103 (Suppl 4):93–100 (1995)

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Introduction

In a recent editorial in this journal, editor Dieter (1) asked “are there specific pollutants or categories of pollutants that influence evolutionary processes, and if so, to what extent can these effects be quantified?” Dieter went on to say that “many question the seemingly minor effects of man-made pollution versus the incomparable effects of natural occurrences. This debate does not diminish our responsibility to quantify the effects of pollutants on the evolutionary process.” In his commentary in response to Dieter's editorial, LeBlanc (2) pointed out that evolutionary processes respond to any selective pressure exerted on a population irrespective of the toxicological mechanism responsible. He argued that, rather than identifying and quantifying pollutants that affect evolution, our focus should be on “identifying and quantifying pollutants that elicit ecosystem-level alterations that may ultimately result in changes in ecosystem structure due to evolutionary and other processes.”

Cells and individuals represent the fundamental structural units of organismal

and ecological organization, respectively. It is at this fundamental level that meiosis occurs, genetic diversity is maintained, evolutionary processes act, and behavior, reproduction, growth, and survival occur and are regulated. It is also at this level that most pollutants exert their toxic effects. As LaBlanc (2) pointed out, “while environmental changes due to pollution are generally reversible, evolutionary changes are often irreversible, thus bestowing an environmental legacy that extends beyond the fate of the pollutant in the environment.” It is my thesis that, as custodians of this planet, we must not let our current focus on ecosystem health and sustainability distract us from these realities. While we maintain sustainable ecosystem function, the cumulative impact of a variety of stressors at these fundamental levels may result in loss of individuals and their genetic diversity. Aldo Leopold, who brought life to the word conservation, recognized this when he wrote “if the biota, in the course of aeons, has built something we like but do not understand, then who but a fool would discard seemingly useless parts? To keep every cog and wheel is the first precaution of intelligent tinkering” (3).

In ecotoxicological terms, pollutants are of concern because of their effects on populations and, so indirectly, on communities and ecosystems; but pollutants act by their effects, direct or indirect, on individual organisms (4). In operational terms, an organism may be defined as “an interaction between a complex, self-regulating

physiological system and the substances and conditions which we usually think of as the environment” (5). According to Bartholomew (5), ecologically relevant physiological processes are those that deal with *a*) exchanges of food, water, energy, and metabolites between organisms and their environments; *b*) the exchanges of information (social signals, pheromones) and gametes between individuals; *c*) the acquisition of information about the environment; and *d*) the effects of the physical environment on physiological capacity and performance. The diverse nature of these interactions and chemical processes provides considerable potential for interference (tinkering) by a wide variety of polluting substances that could affect biota in fundamental and subtle, yet devastating ways.

Natural selection and evolution have been likened to a tinkerer who does not know what he is going to produce, but uses a finite set of elements in a number of ways, to produce a workable object (6). What is used, and for what purpose, depends on the opportunities available to the tinkerer at that particular time. Natural selection is the result of the interplay of two constraints imposed on all living organisms: the requirement for reproduction, and the requirement for continuous interaction with the environment because life persists only by a constant flux of matter, energy, and information (6). This interplay results in differential reproduction and, consequently, in populations that evolve progressively as a function of

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Address correspondence to Glen A. Fox, Canadian Wildlife Service, National Wildlife Research Centre, Environment Canada, Hull, Quebec, K1A 0H3, Canada. Telephone (819) 997-6076. Fax (819) 953-6612.

environmental circumstances, behavior, and emergence of new environmental niches.

As ecotoxicologists, we are concerned with the ability of pollutants to modify natural selection (e.g. tinkering with the tinkerer) because it has been shown that pollutants can act as powerful selective forces (4,7). My objective in this manuscript is to address pollutants *a*) as selective or evolutionary forces acting on the individual that potentially limit genetic diversity; *b*) as agents capable of disrupting chemically mediated communication and information exchange essential for effective behavior, reproduction, development, and maintenance; and *c*) as agents whose indirect effects on the Earth's life-support systems disrupt essential metabolic, nutritional, cellular, and genetic processes. These effects are capable of contributing greatly to the cumulative stress on individuals and altering survival, recruitment, reproductive behavior, and function. They thereby threaten the quality, plasticity, and continuity of the gene pool, and indeed the health and possibly the very survival of an exposed population/species. They therefore deserve greater consideration in our ecotoxicological thinking and ecological risk assessments than they are currently given.

Natural Selection and Evolution as Tinkering

To tinker is to recombine. Evolution reorganizes existing genetic diversity, slowly modifying and adapting it to new challenges (6). Novelties come from previously unseen associations of old material (8). This tinkering results in adequacy of adaptation to local and immediate conditions rather than perfection and is blind to the long-term consequences of the biological changes it produces (5). There is only a slight chance that short-term adaptations will meet long-term challenges and, consequently, for a given population, the long-term probability of extinction is high. However, every living organism is a component of the parent-offspring chain that extends back to the beginning of life on Earth. Each of its direct ancestors was sufficiently well-adapted to its physical and biological environments to allow it to mature and reproduce successfully (5).

Interacting species, by selectively making appropriate responses, continually alter the direction of selection in other members of the community, thereby creating a permanently capricious environment for themselves (9). Sexuality is normally associated with this environmental heterogeneity and

results in protean genetic diversification, whereas in asexual populations genetic variation is frozen into the preexisting structure of the clones (9). Sexuality, through meiosis, recombination, and mutation produces organisms similar, but not identical, to their parents. Sexuality creates and maintains the genetic variability that constitutes the potential for adaptation to a changing environment. Adaptation is a biological attribute inseparable from life itself (5). It is the relative contributions of individuals of different genotypes that determine the composition of future gene pools. Selection at the level of the individual genotype, which is manifest in differential reproductive success, is the process by which adaptations arise and populations evolve (10). Therefore, to sustain wildlife populations and maintain genetic diversity, we must protect individuals (genetic variation) and the processes of sex (mixis) and reproduction.

In evolutionary terms, the rate of increase of a particular allele, or fitness is maximized by maximizing fecundity and minimizing mortality, or breeding as young as possible and at each breeding occasion, provided that other fitness components are held constant (11,12). Newton (10) concluded that breeding lifespan was the major demographic determinant, whereas fecundity contributed the least to variation in the life-time reproductive success of 23 species of birds. Investigations of the extent and causes of individual differences in breeding success among both sexes, based on studies of insects, amphibians, birds, and mammals representing contrasting breeding systems suggest that age, body size, dominance rank, mate choice, early development, phenotypic plasticity in reproductive behavior, and environmental fluctuation in time and space were the key factors (10,13,14). Although an increase in the death rate will reduce the potential rate of increase, sublethal effects that reduce the birth rate are equally effective. Anything that reduces longevity, increases the developmental period, shortens the reproductive period, reduces fecundity or fertility, or alters the age of first breeding will reduce lifetime reproductive success.

Tinkering with Gene Frequencies Restricts the Conditions to Which Adaptation Is Possible

The capacity of cells to maintain a high degree of order in a chaotic universe stems from genetic information that is expressed,

maintained, replicated, and occasionally improved by genetic processes (15). According to Wright's shifting balance model of evolution (16), individual members of local populations can achieve high fitness by various combinations of genetic characteristics. At any one time, the particular genetic combination observed is determined partly by chance, the history of the population, and its position within the metapopulation. Random deviations from a particular successful, local combination of alleles sometimes results in the evolution of other successful combinations with different fitness levels. Subsequent variation of successful gene combinations can result in the evolution of the population to greater adaptive peaks. The ability of populations to adapt to unpredictable environments is the basis for stability of any ecosystem. One of the very first effects of pollution on wildlife recorded in the United Kingdom was in fact an evolutionary change—the occurrence of melanism in moths (*Biston betularia*) in response to variation in both the deposition of soot and diversity of lichens on tree trunks resulting from atmospheric pollution (17–19). This industrial pollution modified the habitat, which in turn greatly altered the relative fitness of different phenotypes by modifying their vulnerability to predation.

Extensive allelic variation across loci is now recognized as a common attribute of most species. Ecological processes affecting genetic diversity in populations can result in changes in the number and frequency of alleles in the population and the level of heterozygosity. Allelic diversity contributes to population growth through its effects on evolutionary potential and the ability of an individual to respond to changes in its selective environment. In addition, stochastic processes also affect the demography and genetic composition of populations (20). Only a small percentage of the variation within most populations influences the fitness of those individuals when challenged by a novel stress. Adaptability of any individual to differential environmental conditions is limited by the fact that its genetic variation cannot be arbitrarily large (21). A population can achieve adaptability only by distributing this variation among its individuals. Effective population size directly determines the levels of heterozygosity and the number of alleles that can be maintained in a population (20). The probability of extinction is thought to be negatively correlated with population size. Stochastic variation in

fecundity among individuals in populations is common and, as population size shrinks, the birth rate will reflect the collective fecundity of the survivors. Stochastic loss of alleles for quantitative or single gene traits occurs at higher rates in small populations that may affect the evolutionary potential of a species. Extreme chemical stresses do not occur uniformly—they are low frequency events occurring effectively at random and are therefore stochastic in nature.

It has been shown that heterogeneous environments involving chemical stresses exert strong evolutionary pressures on plants and animals (7). Survival of a fraction of the population, following repeated or extreme exposure to chemical stress, may lead to the genetic selection of segments of the population capable of stress avoidance, stress detoxification, or repair or compensation for injury, thereby increasing their capacity for survival. Organisms may respond rapidly to altered environments by adopting a conditional behavioral strategy that will allow the individual to facultatively adjust its phenotype to maximize its lifetime reproductive success (22). Research on the selective toxicity of and resistance to pesticides and other toxic substances strongly indicates that metabolism is the most important single factor in determining differences in susceptibility (23). This is achieved by the production of more enzymes (24,25) or different enzymes (26) and the genetic transmission of this ability (26,27). As selection pressure increases through repeated exposure, an increasing proportion of the population may become resistant to the chemical. The speed at which this process occurs depends upon the chemical and its distribution, the genetic makeup of the organisms, and the extent to which the entire population is exposed to the selection pressure. Selection may lead to profound intraspecific or interspecific changes in the community, depending on the particular ecological characteristics of the favored species or segment of the population.

Although natural enemies, host-plant resistance, and other limiting factors are responsible for 90% of the control of insect pests achieved in agroecosystems, about 1 billion tons of insecticides are applied each year (28). By 1989, at least 481 injurious and 23 beneficial species of insects had developed strains resistant to one or more insecticides, accounting for a total of 4458 species-insecticide-country combinations worldwide (29). The wide spectrum of toxicity of insecticides results in the sup-

pression of their natural predators and parasites. Resistance in some populations may regress to insignificant levels following abandonment of a chemical, but there is a tendency for regressed resistance to be rapidly reselected should exposure reoccur (29). Intensive, long-term use of persistent chlorinated hydrocarbon insecticides to control cotton pests and in orchards to control rodents resulted in the development of resistant populations of fish (30) and rodents (31), respectively, as well as lethal effects in their predators (32,33). In some cases, metabolic adaptation to one pesticide may increase the toxicity of another (34). In addition to its frequent occurrence in arthropods, resistance is also encountered in fungal and bacterial pathogens of plants and animals and in weed species. Weed species population shifts in response to increased selection pressure by herbicides are well documented. While adaptation involving selection for resistant genes can occur rapidly, heavy metal- and herbicide-tolerant plants tend to be competitively inferior to normal plants (7,35).

In a wide variety of species, there is a tendency for heterozygotes to have increased survival, increased disease resistance, increased growth rate, and increased developmental stability (36), particularly in stressful environments (7). Allozyme combination (genotype) frequencies are one biological indicator of genetic variability (heterozygosity). There is evidence that differences in allozyme genotypes may result in differences in survival potential and fitness (36,37). Plasticity in enzyme expression may confer tolerance for the toxic effects of pollutants and allow certain individuals to survive. If exposure to a contaminant selects against sensitive genotypes, then certain adaptive alleles may increase in the population while overall heterozygosity is decreased. Creation of a resistant but genetically homozygous population may increase susceptibility to new environmental perturbations. If individuals with certain resistant genotypes have inferior growth rates, fecundity, or increased mortality rates, then this contaminant-induced selection may create a population with decreased survival potential. If reductions in survivorship occur, then the population would be more susceptible to extinction (36).

Effects on behavior, physiology, and population structure may be mitigated within a few generations after the reduction of chemical stress, whereas the potential genetic consequences of contaminant exposure can be far more persistent (38).

Aldo Leopold (39) observed that in the natural environment "evolutionary changes are slow and local" while mankind's use of tools "has enabled him to make changes of unprecedented violence, rapidity, and scope". The crucial and determining factors in ecological and evolutionary processes may be the scale of temporal and spatial change. It has been suggested that air pollution endangers the fundamental basis for the preservation of adaptability of forest tree populations because, measured against the average generation time of trees, it constitutes a rapid, complex, globally active and undirected environmental change (21). There is evidence of selection of air pollution-tolerant genotypes in species of hardwoods in eastern North America (40) and Europe (41). A similar trend has been found in fish. When allozyme frequencies were compared in populations of mud minnows (*Umbra limi*) from acid-stressed and nonacid-stressed sites in a branch of the Moose River in the Adirondack mountains, those from acid-stressed sites had significantly lower levels of heterozygosity, suggesting selection of stress-tolerant genotypes (42). In both cases there was a significant loss of genetic variability in tolerant populations through the loss of genes which are found only in sensitive genotypes. The genetic resources present in sensitive subpopulations are thus endangered as a consequence of pollution.

Significant losses of genetic diversity are most likely to occur as a result of genetic drift or population collapse associated with intense pollution in relatively restricted areas. When the selective pressure is released or the effective population size rebounds, genetic diversity will be restored only through mutation and immigration of variant genotypes. Comparisons of the liver tumor frequencies, polynuclear aromatic hydrocarbon (PAH) burdens, age, and length characteristics of brown bullheads (*Ameiurus nebulosus*) collected in the early 1980s from two tributaries of Lake Erie strongly support the hypothesis that bullheads in the Black River were subjected to an age-selective mortality associated with high prevalences of PAH-associated liver carcinoma (43,44). When the genetic variation in the mitochondrial genome of this species was surveyed at these and seven other sites in the lower Great Lakes in the late 1980s, genetic diversity estimates were always much lower in populations from contaminated sites than in nearby reference sites (38). These authors concluded that the most parsimonious explanation for this reduced genetic diversity is stochastic

reductions in population size that have left fewer individuals to contribute their DNA to subsequent generations. A population bottleneck triggered by stochastic effects of environmental degradation or chemical contamination is therefore a powerful way to produce shifts in mitochondrial haplotype frequency and drastically reduce levels of genetic diversity. Losses of genetic variability are of particular concern because they may limit future adaptability of the populations and decrease the probability of both short- and long-term survival of the species (45,46).

Tinkering with Chemically Mediated Communication between Cells and between Individuals

Organisms have devised methods of *a*) sensing the external environment and internal metabolic changes, *b*) adapting to changes, and *c*) regulating their internal machinery as a result of these signals (47). Cells must communicate to regulate their development and organization into tissues, to control their growth and division, and to coordinate their diverse activities. The coordination and integration centers for this cell-cell communication are the chromosomes of the cell nucleus, the nervous system, the neuroendocrine system, and the immune system. Three fundamental mechanisms are responsible for communicating these signals throughout the organism: *a*) the chemical composition of proteins that determine the shape and structure of the cell and serve as instruments of molecular recognition and catalysis; *b*) electro-biochemical mechanisms in the nervous system responsible for sensing and rapidly responding to stimuli in the external environment in an effort to maximize the organism's survival; and *c*) chemical control mechanisms based on chemical discrimination and hormones that defend, control, and coordinate the internal environment (15).

Biological systems respond to sensory inputs and changing metabolic conditions both by amplifying signals and by adapting to them (47). In most systems, the cell is the primary unit capable of both amplifying and desensitizing signals. The most effective sensory and regulatory system is one in which a small change in an effector concentration elicits a very large response (47). The chemoeffector may be an ion, an electrical potential gradient across membranes, a neurotransmitter, an enzyme, a substrate, an activator or

inhibitor, or a hormone. A variety of mechanisms are used to attain this ultrasensitivity. In most cases, control is effected by conformational changes in proteins that either turn on or turn off the processing system. In this way, molecular signals can feed forward to activate a pathway that must be mobilized for a particular molecular function or feed back to inhibit synthesis of a product that is in excess (47). Such molecular mechanisms operate within the cell in metabolic regulation, between cells in hormonal and neural signalling, and between the environment and cells of sensory receptors. Since both amplification of and adaptation to signals occur within the same cells using a diverse variety of chemoeffectors in an intricate array of complex chemical interactions, chemical communication is very vulnerable to interference by a wide variety of chemical stressors.

Hormones are special messenger chemicals that are elaborated in restricted areas of organisms. They diffuse or are transported over variable distances within organisms to adjust metabolism, control remote effectors, or regulate morphogenesis. They are effective in minute quantities and are structurally very diverse. The hormone itself is usually not a participant in the process, but rather an initiator. Hormones exert their specific effects by regulating preexisting processes in target cells, which have specific membrane-bound, cytoplasmic or intranuclear receptors for a complementary set of chemical signals, in three ways: by influencing the rate of synthesis of enzymes and other proteins; by affecting the rate of enzymatic catalysis; and by altering the permeability of cell membranes. Most hormones have more than one action and most functions under hormonal control are influenced by more than one hormone. Negative feedback mechanisms and nervous regulation operate to maintain the appropriate secretion of hormones.

The pheromones or social hormones also form part of this broad chemical system of coordination and communication and are an extremely important means of communication in mammals and insects. Unlike hormones, which operate within the individual to communicate information between cells, pheromones are released outside the body and communicate information to other individuals of the same species in which they elicit a specific physiological or behavioral response or developmental process. They include alarm and marker substances and other olfactory stimuli that elicit approach, permit sexual discrimination, stimulate copulatory

behavior or drastically alter reproductive physiology. Some pheromones influence dispersal and aggregation, facilitate parent-young interactions or individual and group recognition, whereas others control caste development in social insects (48).

Environmental contaminants are able to disrupt normal hormonal activity at several levels and, if present during the critical stages of embryonic, fetal, and perinatal development, can alter the growth and function of the coordination and integration systems, as well as interfere with metabolism. These transgenerational effects may not be detected until the individual reaches sexual maturity or senescence (49).

Although attention to date has focused on estrogenic effects of hormone disruption (49-52), we should remember that hormonal influences or interactions provide the optimal environment for all nutritional, metabolic, growth-related, developmental, and reproductive processes. Our inadvertent tinkering with hormone-mediated homeostasis can therefore have other diverse and profound effects on the physiology of the individual. Hormones, especially those of the adrenal cortex, respond to stress and are essential in the "fight or flight" response. Other adrenal hormones are involved in osmoregulation. Adrenal steroidogenesis has been shown to be affected by a number of chemical agents (53-56). Baltic seals (*Phoca hispida* and *Halichoerus grypus*) suffer from adrenocortical hyperplasia (57) and pathological changes of the bony tissues suggestive of Cushing's disease (58). Laboratory studies have shown that adrenocortical necrosis occurs in species of mammals and birds with a high metabolic capacity to convert methylsulfonyl-DDE and *p,p'*-DDD to reactive intermediates (59,60). The thyroid is very frequently affected by xenobiotic chemicals (61). Disturbances in thyroid function during a critical period of fetal and perinatal development may produce irreversible neurological damage (62). Since the thyroid's role is pivotal in the regulation of metabolic processes, growth, and morphogenesis, chemical interferences with its hormones and function have obvious energetic and reproductive implications for individuals. Migratory or hibernating species may be particularly vulnerable as are species that raise young in unpredictable environments such as the arctic, where the costs of thermoregulation are high and the availability of energy and protein are limited and highly constrained in both time and space.

There appears to have been little research conducted on the possible effects of pollutants on pheromone-mediated communication. There is preliminary evidence that *in utero* exposure to PCBs affects chemically communicated maternal recognition of neonatal mink (*Mustela vison*) kits by producing lesions in the cervical apocrine glands (63). These lesions could interfere with secretion or production of pheromones that appear to assist in maternal recognition within the Mustelidae, possibly inducing failure to nurse and thus contributing to reduced growth rate and wasting disease observed in litters from captive mink fed PCB-contaminated diets.

Tinkering with the Earth's Life-support Systems Can Disrupt Essential Metabolic, Nutritional, Cellular, and Genetic Processes

Human activities are currently leading to unprecedented changes in the Earth's atmospheric, terrestrial, freshwater, and marine environments (64). The potential consequences of the addition of a variety of trace gases and pollutants to the atmosphere range from depletion of stratospheric ozone and climate change to the formation of wet acidic deposition at sites remote from the source of emissions with subsequent alterations in population, community and ecosystem processes. Human activities divert, deplete, and pollute ground water, rivers, lakes, and oceans, thus altering the productivity and biological diversity of freshwater and marine ecosystems. Our activities also alter the flux of energy and essential natural materials through ecosystems.

The anthropogenic causes of these changes in the atmosphere, hydrosphere and lithosphere result from processes occurring at regional scales, but their unanticipated or indirect effects on components of the Earth's life-support systems may occur at considerable distances from the site of the activity (64). Although they may be regional or global in extent, the ecological consequences of these changes in the abiotic environment may be first seen as changes in function of individuals and communities that may ultimately be expressed in ecosystem function (64).

Species interact with and are modified by the abiotic components of the environment. Populations differ in their inertia, resilience, and stability in response to stress (65). Synergisms between simultaneous or successive stresses may have unpredictable

effects on populations. In the context of physiological energetics, stress acts to constrain growth or reproduction, limiting the environmental conditions under which the organisms may survive and reproduce (66). Genetic heterozygosity favors growth and fecundity by optimizing maintenance metabolism. Since stress resistance depends upon metabolic rate, combinations of many stresses will tend to have at least cumulative effects (7). The general phenomenon of pollution increasing stress and thereby lowering resistance to, or acting synergistically with, other stressors (e.g., social interactions, reproduction, disease, temperature extremes, nutrient, water shortages, or osmotic stress) is commonly hypothesized, but has been little explored (65,67).

It must be assumed that stress related to energy supply and demand is often present in wild populations. Although populations have adapted to handle seasonal food stress, abnormal food scarcity resulting from climatic abnormalities or human intervention may have significant effects. This has been shown by Lemon (68) who experimentally manipulated the maximum rate of energy gain attainable while foraging by breeding populations of the zebra finch (*Taeniopygia guttata*). He found that small changes in the daily energy budget influenced the rate at which broods were produced, the size of the broods that could be successfully supported by their parents, and the subsequent survival of the parents. Similarly, the productivity of ring doves (*Streptopelia risoria*) decreased 50 to 100% when food was restricted by 10 to 30% and effects were greater in birds previously exposed to DDE (69). Decreased productivity resulted from failure to breed and death of embryos and young due to inadequate brooding and care, apparently by decreasing the levels of the hormones necessary to develop and maintain active gonads, adequate courtship and breeding behavior, and functional crop glands. Cadmium ingestion enhanced food restriction-induced alterations in energy metabolism in mallard ducks (*Anas platyrhynchos*) at levels of dietary cadmium that by themselves were without effect (70).

There is field and laboratory evidence that nutritionally deficient diets may also reduce resistance to infectious diseases in wild birds under climatic stress or conditions that enhance exposure to disease agents (71,72). PCBs and some herbicides may interact with infections to produce substantial effects on mortality and reproduction in natural populations of small

mammals when food or water is limited (73). Captive harbor seals (*Phoca vitulina*) fed fish from the polluted waters of the Baltic Sea developed impaired T-cell mediated immune function accompanied by suggestions of increased bacterial infection (74). Similarly, Blaustein et al. (75) suggest that amphibians may be especially susceptible to infection by *Saprolegnia*, a circumglobally distributed pathogenic fungus, when under stress from competitive situations or adverse environmental conditions including pollution, loss of habitat, acidification, or increased levels of ultraviolet-B (UV-B) radiation.

Field observations of natural populations suggest that the reproductive potential of some species is being affected by levels of pollutants in the atmosphere. Flueck (76) has hypothesized that there has been a recent decline in the bioavailability of selenium, an essential trace element, in some areas of northern California through Se export and soil acidification due to biomass removal and acid precipitation. When free-ranging black-tailed deer (*Odocoileus hemionus columbianus*) in one such area were implanted with selenium boluses, the preweaning fawn survival was greatly increased (2.6×). Since the mid 1980s, many moose (*Alces alces*) in a strongly acidified district in southwestern Sweden have been affected by a complex, fatal disease syndrome (77). Analyses of hepatic tissue have revealed a 50% decline in copper concentrations accompanied by a 21 to 24% increase in molybdenum. The strong agreement between clinical and pathological signs of the disease syndrome and those that occur in chromium, copper, iron, and manganese deficiency as described for cattle suggest that the moose suffer from a secondary copper deficiency due to a low Cu/Mo ratio as a result of elevated molybdenum intake (77). The increased molybdenum intake is evidently caused by an increase in soil pH. The intensive liming of lakes, fields, pastures, and forests in western Sweden in recent years to remediate the effects of acid deposition is suggested as a possible explanation (77). It is therefore evident that the impact of large-scale anthropogenic activities may alter essential trace mineral cycles in remote ecosystems.

There is mounting evidence that the solar flux of UV-B radiation has begun to rise at certain locations over the surface of the earth, apparently as a result of the continuing destruction of the protective ozone layer by atmospheric pollutants. Biota have evolved a number of adaptive strategies to reduce the deleterious impacts of UV-B

including avoidance, the production of screening materials, and a variety of free radical scavenging mechanisms (78). However, there is enormous interspecific variability in this resilience. Analyses suggest that continued ozone depletion will result in a decline in aquatic primary production rates and shift the competitive balance among phytoplankton species. Since many aquatic herbivores including insects, corals and zooplankton are known to be sensitive to UV-B, changes in grazing pressure may also contribute to the wide-ranging influences on the structure and dynamics of aquatic communities likely to accompany increases in solar UV-B flux (78).

Most organisms show some ability to repair the damage caused by UV-B (78). However, recent experimental studies in the Pacific Northwest have shown that the population status of three anuran species (*Hyla regilla*, *Rana cascadae*, and *Bufo boreas*) is strikingly correlated with interspecific differences in the ability of their eggs to repair UV-B-induced DNA damage (79). These findings suggest UV-B irradiance linked to stratospheric ozone depletion may be a factor in the decline of many amphibian species in widely scattered habitats.

In regions where acid-neutralizing capacity of soils and waters is low, the pH of lakes and streams has decreased, and concentrations of metals have increased as a result of acidic precipitation. The abundance, production, and growth of decomposers, algae, macrophytes, invertebrates, and fish have been affected, and sensitive species have been lost from a vast number of lakes in the northern hemisphere (80–82). These losses result in altered species composition and reduced diversity in affected freshwater communities. Fish have suffered acute mortality, reduced growth, skeletal deformities, and especially reproductive failure (81,82). In North America, at least 30 species of freshwater fish have disappeared from natural populations at different pHs. The effects of acidification on fish stocks is apparently the most devastating change recorded for the

fish fauna of Scandinavia (82). Similarly, *in vitro* examinations of pollen sensitivity of forest plant species have revealed that inhibition of germination occurred in all species at acidities now occurring in wet acid deposition and that SO₂ and O₃ have similar effects. There is also experimental evidence that SO₂, O₃, and wet deposition can affect stigma receptivity in a number of species of plants (83).

Conclusions

Populations can exhibit four different responses when challenged by a chemical stressor. On one extreme, there is no response because the individuals are resistant to the stressor or the exposure is very brief or minimal. At the other extreme, where the population is extremely sensitive or the exposure overwhelming, there is total mortality resulting in local extinction. Under conditions of chronic exposure, the stress may be accommodated by behavioral or physiological adaptation with no apparent effect on growth, survival, and reproduction. Alternatively, the stress may differentially affect various genotypes within the population, resulting in progressive elimination of sensitive individuals and a shift in the genetic structure of the population.

While acute exposures to chemical agents with high lethality and selectivity have the ability to limit genetic variability and disrupt reproductive continuity, chronic exposure to chemicals with the ability to disrupt ecologically relevant physiological processes and chemically mediated communication between cells or individuals may pose the greatest threat to the diversity and long-term survival of sexually reproducing animals. These agents affect the amount of genetic variability that exists within a population: the intergenerational transfer of that variability and the size, functional viability, and genetic variability of future generations. Such agents have the greatest potential for cumulative stress on populations. The ability to disrupt hormone-mediated inter-

cellular communication is not confined to any particular chemical structure, but has been shown for insecticides, herbicides, nematocides, and fungicides of diverse types, as well as organohalogenes, organometallics, metals, and complex industrial effluents (50). These agents are numerous, widely used, or produced as by-products of agriculture and other industries, and some are persistent and widespread in the environment. The potential multimedia exposure of organisms to such agents is considerable. Their cumulative impact may be greatest on long-lived, slow-reproducing species where it can go undetected for decades. Without human intervention to counteract these impacts, species may decline or be lost. Some local areas where exposure to such agents is very high may act as population sinks which drain surrounding areas and the species as a whole of their genetic diversity and numbers (84).

Recent evidence suggests that large-scale anthropogenic activities and air pollution may indirectly alter the Earth's life-support systems, disrupting essential nutritional, cellular, and genetic processes. Alterations in trace mineral availability, increased UV-B-induced DNA damage, and alterations in pollen germination are three such effects that differentially affect reproduction in different species and may have long-term implications on the competitive ability and survival of species, with potential effects on the biodiversity of intercepting ecosystems (76–79,83).

Today, some species or subspecies populations such as the Lake Trout (*Salvelinus namaycush*) in the lower Great Lakes are indeed living fossils—unable to effectively reproduce—the last living remnant of their gene pool and the termination of the parent-offspring chain that extends back to the beginning of life on Earth. According to Leopold (3) “a thing is right when it tends to preserve the integrity, stability, and beauty of the biotic community. It is wrong when it tends otherwise.” As polluters of this planet, we tinker foolishly!

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