Methods for and Approaches to Evaluating Susceptibility of Ecological Systems to Hazardous Chemicals

Joanna Burger

Environmental and Occupational Health Sciences Institute and Graduate Program in Ecology and Evolution, Rutgers University, Piscataway, New Jersey

Differences in genetic susceptibility to hazardous chemicals affect individuals of both human and nonhuman populations. In both cases, differences in response to chemicals or general ill health result as a function of these differences in genetic susceptibility. However, ecological systems are a compilation of hundreds or even thousands of different species, resulting in structural and functional characteristics that are themselves affected by differences in susceptibility. Although individual and population differences in susceptibility to hazardous chemicals underlie effects at the community and the ecosystem level, they do not account for all differences. I propose a two-tiered approach to evaluating susceptibility to ecological systems: a general susceptibility as a function of ecosystem type (based on structure and function of that system) and a differential in susceptibility within broad ecosystem types as a function of biotic and abiotic factors. In terrestrial ecosystems, the two factors that most affect overall susceptibility are species diversity and hydrology; evaluation of the effects of hazardous chemicals involves measuring species diversity and water movement. This same methodological approach can be applied to aquatic ecosystems and to highly altered ecosystems such as agriculture, forestry, fisheries, and urbanization.

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Introduction

Ecological systems have several levels of organization, from the component individuals, populations, and communities to the full ecosystems. Recently, the importance of landscape scale considerations has moved to the fore, as we realize that ecological processes cannot always be easily compartmentalized (1–4). Understanding the effects of chemicals and measuring or assessing the effects of chemicals at the different levels of organization in ecological systems requires understanding the factors that affect exposure and response (5,6). Differences in individual physiology, behavior, and ecology influence the extent to which individuals and populations are exposed to hazards, as well as how they respond (7).

In this paper I examine what exposure and susceptibility mean within the different levels of ecological systems, provide examples of the factors that affect susceptibility (given equal potential for exposure), and derive a conceptual model for the differential susceptibility of different terrestrial ecosystems. Thus I propose an overall method for evaluating the effect of chemicals on ecological systems.

Exposure, Response, and Susceptibility

Assessment of the effects of hazardous chemicals on human health has concentrated on the individual, and on organ, cellular, and subcellular causes of illness (5,8). This has led to investigations aimed at identifying the mechanisms of action of the causative agents. A great deal of attention has been devoted to assessing biomarkers of exposure, effects, and susceptibility (9–11). Presumably such biomarkers will allow earlier and more cost-effective detection of factors that predispose to or cause ill health. The overall goal is preserving, maintaining, or achieving health of the individual.

Health professionals recognize that people vary in their susceptibility to diseases and chemicals, and that understanding the causes of these differences is critical to understanding and predicting the effects of chemicals on humans (12,13). Susceptibility differences in humans can be genetic or phenotypic, and understanding differences in genetic susceptibility to toxic chemicals is a growing challenge for the health sciences. Lifestyle can modify these susceptibilities, including factors such as diet, exercise regime, and occupation.

Individual susceptibility as a function of genetic makeup also occurs in non-human biota and significantly influences the impact at the population, community, and ecosystem level (8). Repeated or continued stresses can serve as evolutionary selective forces that change the character of organisms within ecosystems (14,15), but this aspect of response has received relatively little direct attention (16). Ecologists and ecotoxicologists have examined individual differences that affect response, but have not considered their data within the framework of susceptibility.

For example, in typical ecotoxicological studies conducted in the laboratory, test organisms of a similar sex and age class are exposed to a chemical and the response is recorded. More recent ecotoxicological treatments have recognized the hierarchical nature of ecological systems (17). Often the outcomes of traditional ecotoxicological studies were expressed as LD50 or ED50 values, which reflects variation in susceptibility among individuals because the exposure has been carefully controlled. It is internal factors, including genetic differences in susceptibility, that determine the range of responses. Some individuals die sooner than others or die at lower doses.

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Address correspondence to Dr. J. Burger, Graduate Program in Ecology and Evolution, Nelson Hall, Rutgers University, Piscataway, NJ 08855-1059. Telephone: (908) 445-4318. Fax: (908) 445-5870. E-mail: burger@biol-ogy.rutgers.edu

Abbreviation used: NRC, National Research Council of the U.S. National Academy of Sciences.
To some extent, variations in individual susceptibility in ecotoxicology experiments can be inferred from the variance around the mean. That is, any measure of variance in an experiment with otherwise similar organisms exposed to the same dose is a measure of population susceptibility. Moreover, dose–response curves can be used to examine individual susceptibility when the response is the number of organisms affected (5). There are many examples of differences of individual susceptibility (which ecologists call fitness) and several examples will suffice.

Barber et al. (15) examined clonal variation in general responses of Daphnia magna to toxic stress in the laboratory. They found wide genetic differences in susceptibility to cadmium in both acute tolerance and chronic stress tolerance. These experiments illustrate the importance of laboratory tests in demonstrating genetic differences in susceptibility of relatively small organisms that are low on the food chain.

Weis (14,18–20), in a long series of elegant experiments with killifish (Fundulus heteroclitus), showed that the teratogenicity of methylmercury was dependent on the previous toxic environment of parents. That is, eggs from fish living in a site that had been contaminated with mercury for many years were less susceptible to the effects of mercury than those from a relatively clean site, even though in the laboratory both were exposed to the same dose of mercury. Presumably this involved genetic changes in the population beginning in the early 1970s when the mercury exposure occurred.

In this paper I concentrate on methods and approaches to evaluate the effects on high levels of organization.

**Assessment of Ecological Systems Compared to Humans**

Although individuals in populations of human and nonhuman biota can exhibit the same types and ranges of variation in genetic susceptibility (13), ecological systems are subject to a far greater range of susceptibilities because of their greater complexity (21–23). Even relatively simple ecological systems contain hundreds of different species, each with their own range of susceptibilities to chemicals or other adverse agents or conditions. Moreover, ecological systems are not just a sum of the individual organisms, but have both structural and functional relationships critical to a healthy ecosystem (24). It is the structural and functional relationships that make examining susceptibility difficult in ecological systems. Methods for evaluating effects at higher levels of organization are less well studied than those for individual species.

Susceptibility at any level has to be considered in terms of some agreed outcome. At every level of ecological organization, from the individual to ecosystems, there are indicators or measures (biomarkers or bioindicators) of well-being (5,6). At the population level, population stability is the end point of concern (27). In the case of ecosystems, methods to measure effects include changes in species numbers and diversity, loss of rare and endangered species, loss of primary productivity, disruption of nutrient cycles, and habitat loss and degradation, to name but a few (6,25–27).

Some other general aspects affect susceptibility of ecosystems, including ecosystem size, fragmentation, and landscape pattern. That is, even though a given ecosystem is largely undisturbed within its borders, its size and placement with respect to other ecosystems influences its susceptibility. For example, some interior-nesting birds and large predatory cats require large forests to maintain stable populations. Smaller forest patches, though otherwise pristine, do not provide sufficient space to maintain enough territories for an interbreeding population. Similarly, if a small forest patch is surrounded by agricultural land, there may be no way for organisms to move in and out of that forest.

This suggests that the boundaries between ecosystem types are particularly vulnerable to chemical stresses (or physical or biological stresses) because these regions are where species exchange occurs. In addition, there are many species that thrive in the ecotones between different ecosystem types. Since the destruction or contamination of either adjacent ecosystem could affect survival of the populations residing there, they are particularly vulnerable to chemical contamination.

I propose that there are two major approaches to examine susceptibility of ecological systems, at least above the individual and population level: overall susceptibility of ecosystems (how much or how likely they are to change when stressed) and factors that affect susceptibility within the overall vulnerability of each ecosystem or biotope. In other words, some ecosystem types (as a class) are more susceptible to hazardous chemicals than others. There are, however, a number of factors that result in different susceptibilities within these broad classes of ecosystems. By types of ecosystems, I refer to generally recognizable systems such as tundra, desert, temperate rainforest, or tropical rainforest. I will discuss each of these types of susceptibilities in turn for terrestrial ecosystems.

The factors that affect aquatic ecosystems and highly managed ecosystems are clearly different. In aquatic systems both the size of the system and the degree of water movement or turnover are critical factors affecting susceptibility. For example, the Exxon Valdez oil spill in Prince William Sound, Alaska, had a much greater effect on that marine ecosystem than did the Sea Empress spill between England and Wales, largely because in the former there is little water movement, and the oil remained within the sound for much longer without dispersing to the ocean where it could be diluted (28).

**A Model for Differential Susceptibility of Different Terrestrial Ecosystems to Hazardous Chemicals**

Ecosystems vary in their vulnerability to hazardous chemicals, given equal exposure, largely because of three factors: a) differences in fate and transport of chemicals through the system, b) differences in the complexity of the system, and c) differences in individual responses of the component species populations.

Differences in individual species responses is critical because any given community or ecosystem could have few or many species that are susceptible. However, not all species in an ecosystem are equally important to the function and stability of the system; some have a keystone role.

A keystone species refers to the relative role a species has in community structure and function (29). That is, if some species disappeared from the system, there would be little effect. However, if keystone species are removed, by exposure to hazardous chemicals or other factors, their loss would have multiple direct and indirect effects. For example (Figure 1), removing a certain predator A may have the direct effect of allowing prey populations B and C to increase (because they are no longer being eaten by A); and the indirect effect of increasing other predators (who no longer have to compete with A), thereby decreasing the populations of prey populations D and E because they now have to compete with larger populations of populations B and C, and decreasing the food sources (plant or animal populations) of population...
Figure 1. Diagram of keystone species effects. The top indicates the normal predator–prey and competitive interactions that can occur in a community (before). The bottom indicates the possible changes in population levels following decreases or extirpation of species A by hazardous chemicals (or any other perturbation). Direct effects (shown in box) occur because prey populations can increase, but there are indirect effects on other predators (increases in X due to loss of competition), on other prey (due to both increases in predator X and increases in competitors B and C), and on the food organisms of B and C (decreases due to increases in B and C).

B and C. This example could go on, because there are many cascading effects that result from removing a keystone species (30). In the past, ecologists have concentrated only on the effect on its prey. A good example of a keystone organism is Morin’s (31) study of competition between aquatic insects and vertebrates. It indicates that both abundance and biomass of anurans can be influenced by the presence of specific insects.

Although Mills et al. (32) have expressed a contrary view, clearly the role of some species in particular ecosystems is critical to its functioning. For example, cord grass (Spartina alterniflora) is a critical component of salt marshes in the eastern United States, and its removal would result in a general collapse of the system. Thus methods to evaluate the effects of chemicals such as oil on cord grass have been key in understanding susceptibility of that system.

Combining the relative susceptibility of individual species to a hazardous chemical, with the relative severity of the keystone response (or of their keystone function) results in a given susceptibility of an ecosystem (Figure 2). Ecosystems or habitat types that contain many species that are susceptible to a given chemical, and many species that have a high keystone function, result in greater overall susceptibility.

There are many other factors that render an ecosystem vulnerable or susceptible to hazardous chemicals. Two factors, however, stand out as having a greater effect than all others: hydrology and species diversity.

Differences in fate and transport of chemicals through the system is critical to susceptibility because when chemicals move rapidly through a system, a higher proportion of the species are exposed and receive a higher acute dose than in systems where movement is slow. If it takes months or years for the chemical to be transported through the system, the effects will be less because of decreasing dose, and excretion or modification. Thus, in terrestrial systems where chemicals move rapidly, as happens in systems with wide, fast-moving rivers, there is a high potential for exposure for most organisms living in this system. On the other hand, in the arctic tundra, where water movement is slow, it may take years for the chemicals to disperse very far from the source, reducing the potential effects.

The hydrologic regime is critical because water is the medium in which chemicals can be dispersed most easily and quickly through an ecosystem. That is, if a chemical is spilled in the desert, it may percolate through the soil, but may have little impact for many years because it is not mobilized into the food web. However, if a chemical spills in a stream or river, it is quickly carried wherever the river flows. If a chemical spills in a forest, followed by a heavy rainstorm, the chemical will be carried through the soil and to the waterways. In both cases, the movement of the hazardous chemical through the ecosystem will be both rapid and ubiquitous. Hydrological regime can be used as an indication of speed of movement through a system. In this sense I am using hydrology as a surrogate for fate and transport, but any aspect of fate and transport could be used in the model.

By the same token, species diversity is a surrogate for complexity in ecosystem structure and function. In general, when there are few species, ecosystems are more susceptible to chemicals or other perturbations because there are fewer links for dissipation of chemical effects. It is more likely that all organisms will be affected, and less likely that the system can recover quickly. This is particularly true in agricultural, forestry, or other managed ecosystems where species diversity is typically low. Such systems are very vulnerable to insect scourges, as well as chemical perturbations. On the other hand, systems with many different species have a lower potential for disruption because of the variety of potential pathways.

These two factors, hydrology and species diversity, allow for the possible development of a conceptual model for the differential susceptibility of different major ecosystems to hazardous chemicals (Figure 3). This model gives an indication of where the effects might be expected to be greatest, given equal exposures to the same toxic chemicals. The model predicts that arctic lakes will be the most susceptible, while tropical habitats will be the least susceptible.

Using this model for differences in susceptibility among ecosystem types, there are several methods for measuring variations in these two factors that would predict relative susceptibility. For hydrology, the speed and volume of water movement through a system could be measured. In the case of river and streams, this would be speed of movement through the channel. In the case of terrestrial systems, percolation could be measured. There are well-established methods for both of these parameters.

Species diversity can be measured by simply counting the number of species in the system. A more sophisticated methodology would include counting the number of species native to that ecosystem, and counting the number of exotic species. Total species diversity would be the sum of these two, but for examining the effects of chemicals, the number of native species is a better indication of lack of disruption. Species diversity can be interpreted only with an understanding of what species diversity should be in a relatively pristine system.
To demonstrate that these factors can affect relative susceptibility of communities and ecosystems, I have chosen to list specific effects on individual species or types of species. However, it is critical to remember that if a factor renders a given species more or less susceptible to a hazardous chemical, then the effect on this species population will in all likelihood affect the other species it depends on and that depend on it, and other similar species may likewise be affected (Figure 1). Previous exposure of an ecosystem to hazardous chemicals, either the same or a different one, can affect the overall response of the ecosystem because some of the organisms may have experienced selection for tolerance. That is, with low-level exposure to a toxic substance, organisms predisposed to deal with the toxic substance survive and reproduce, while others less able to do so perish. Gradually the genetic makeup of the population shifts toward lower susceptibility. This has been demonstrated both in laboratory experiments (15) and in the field (20).

Fish exposed to methylmercury for many years undergo selection such that eggs from these regions are less susceptible to mercury than those from other regions (14,19). This is one of the best examples of genetic changes in susceptibility at the population level. Although less clearly demonstrated from laboratory experiments, there is some evidence that invertebrates exposed to chronic oil pollution are better able to survive continued oil pollution than are organisms from pristine habitats (33).

The mere presence of some hazardous chemicals changes the susceptibility of many organisms, despite similar exposures (Table 1). For example, susceptibility of fish and birds to mercury is reduced by the presence of selenium (34). This apparently operates at the absorption or uptake level (35), but the effects on populations and communities are clear.

Energy relationships of different trophic levels affect bioaccumulation, and thus long-term effects of hazardous chemicals (36). Similarly, the level of organic matter present in an ecosystem affects the release rate and bioavailability of chemicals, such as mercury (37). The relationships between the accumulation of organic matter and ecosystem susceptibility is a fruitful area for further research.

There are a variety of abiotic features of communities or ecosystems which result in differential susceptibilities within major types of ecosystems. That is, when considering temperate estuaries, susceptibility of

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**Figure 3.** Model for overall ecosystem susceptibility, given equivalent exposure, to hazardous chemicals.

**Table 1.** Factors that affect susceptibility of communities and ecosystems to hazardous chemicals.

| Factor                | Example of effect                                                                 | Reference source |
|-----------------------|-----------------------------------------------------------------------------------|------------------|
| Previous exposure     | Increased tolerance of fish embryo from mercury-contaminated habitats             | (14)             |
|                       | Exposure to low-level toxins can cause shifts in tolerance or resistance of species | (45)             |
| Toxic environment     | Presence of some metals affects susceptibility to or uptake of others: selenium     | (35)             |
|                       | effects lowered by presence of mercury, copper, and cadmium                        |                  |
|                       | Uptake of mercury lowered by presence of selenium in fish                          | (34)             |
|                       | Treatment by pesticides changes invertebrates to alter community structure         | (46)             |
|                       | Changes in community structure after acidification                                 | (47)             |
| Acidification         | Acidification of freshwater affects geochemistry and bioavailability of aluminum   | (48)             |
|                       | and mercury                                                                       |                  |
|                       | Fish in low alkalinity lakes more susceptible to mercury                            | (49)             |
|                       | Changes in community structure after acidification                                 | (50)             |
| Seasonality           | Differential tolerance of fish embryos to mercury relative to date of lunar spawning | (19)             |
|                       | Differential body condition due to winter starvation                                | (51)             |
|                       | influences mercury effects in birds                                               |                  |
| Salinity              | Differential fertilization of eggs in fish                                         | (52)             |
|                       | Lowering salinity decreased the teratogenicity of methylmercury in fish eggs       | (19)             |
| Temperature           | Low temperatures cause increased toxicity of selenium in fish                      | (53)             |
|                       | Reduction in temperatures causes increase in teratogenicity of methylmercury in    | (18)             |
|                       | fish eggs                                                                          |                  |
| Organic matter levels | Amount of organic matter affects release rate and bioavailability of methylmercury  | (37)             |
|                       | Increased nest density increases mortality in chicks                                 | (54)             |
|                       | exposed to lead through mechanism of decreased parental recognition                |                  |
| Longevity             | Differences in longevity of organisms affect potential for bioaccumulation of all  | (55)             |
|                       | toxicis                                                                            |                  |

**Factors Affecting Susceptibility within Ecosystem Types**

Given the general susceptibility of major ecosystem types because of their structure and function, there are other factors which predict additional differences in ecological susceptibility to hazardous chemicals. These will be discussed below. Although the list is not exhaustive, it is meant to give an overview of the kinds of factors that affect susceptibility (Table 1).
the systems will vary depending upon acidification, salinity, temperature, and seasonality (Table 1). While each factor affects susceptibility predictably for many hazardous chemicals, understanding how these multiple factors interact will be challenging, but will contribute to our ability to predict ecosystem effects from hazardous chemicals.

Finally, there are a variety of biological factors that affect ecosystem susceptibility, in addition to accumulation of organic matter. Social factors have been largely overlooked, but deserve attention. Epidemiologists and human health risk assessors are well aware that population dispersion and density can affect well-being and health (38), yet these aspects are often ignored in ecological systems.

Burger and Gochfeld (7,22) showed that young herring gulls were differentially susceptible to the effects of lead depending upon nesting density. That is, chicks that were in dense areas of the colony were more likely to be killed by neighbors (through the mechanism of delayed recognition of their parents and neighbors) in areas where neighbors were close.

In the recent controversy over environmental estrogens (39), it is likely that social factors are acting as one filter for differences in susceptibility. That is, environmental chemicals lead to a feminization of males in gulls and alligators, with decreased heterosexual pairing and abnormal sexual development (40–43). However sex ratios within the population partially determined the relative susceptibility of individuals: where sex ratios were heavily skewed toward females, there were more female–female gull pairs (44). The effects of environmental estrogens, it seems to me, have the potential to be modified by the social environment, affecting susceptibility of individuals and populations.

REFERENCES

1. Forman RR, ed. Pine Barrens, Ecosystems and Landscape. New York:Academic Press, 1979.
2. Forman RR, Godron M. Landscape Ecology. New York:Wiley & Sons, 1986.
3. Dunning JB, Danielson BJ, Pulliam HR. Ecological processes that affect populations in complex landscapes. Oikos 65:169–175 (1991).
4. Karr JR. Protecting ecological integrity: an urgent societal goal. Yale J Int Law 18:297–306 (1993).
5. Peckall D. Animal Biomarkers as Pollution Indicators. Cornwall, England:Chapman & Hall, 1992.
6. Linthurst RA, Bourdeau P, Tardiff RG. Methods to Assess the Effects of Chemicals on Ecosystems. Chichester, England:Wiley & Sons, 1995.
7. Burger J. A risk assessment for lead in birds. J Toxicol Environ Health 45:369–396 (1995).
8. Vouk VB, Butler GC, Hoel DG, Peckall DB. Methods for Estimating Risk of Chemical Injury: Human and Non-human Biota and Ecosystems. Chichester, England:Wiley & Sons, 1985.
9. National Research Council. Biological Markers in Pulmonary Toxicology. Washington:National Academy Press, 1989.
10. National Research Council. Biological Markers in Reproductive Toxicology. Washington:National Academy Press, 1989.
11. National Research Council. Biological Markers in Immunotoxicology. Washington:National Academy Press, 1992.
12. Omenn GS. Predictive identification of hypersusceptible individuals. J Occup Med 24:369–374 (1982).
13. Woodhead AD, Bender MA, Leonard RC. Phenotypic Variation in Populations. New York:Plenum Press, 1988.
14. Weis JS, Weis P, Heber M, Vaidya S. Investigations into mechanisms of heavy metal tolerance in killifish (Fundulus heteroclitus) embryos. In: Physiological Mechanisms of Marine Pollution Toxicity (Vernberg W, Calabrese A, Thurberg F, Vernberg FJ, eds). New York:Academic Press, 1982;311–330.
15. Baird DJ, Barber I, Calow P. Clonal variation in general responses of Daphnia magna to toxic stress. II: Chronic life-history effects. Funct Ecol 4:399–408 (1990).
16. Callow P, Sibly RM. A physiological basis of population processes: ecoxenological implications. Funct Ecol 4:283–388 (1990).
17. Newman MC, Jagoe CH. Ecotoxicology: A Hierarchical Treatment. Boca Raton, FL:Levis Publishers, 1996.
18. Weis JS, Weis P, Ricci JL. Effects of cadmium, zinc, salinity, and temperature on the teratogenicity of methylmercury to the killifish (Fundulus heteroclitus). Rapp Reun Cons Explor Mer 178:64–70 (1981).
19. Weis JS, Weis P, Heber M. Variation in response to methylmercury by killifish (Fundulus heteroclitus) embryos. In: Aquatic Toxicology Hazard Assessment Fifth Conference, American Society for Testing and Materials (Pearson J, Foster R, Bishop WR, eds), 1982;109–119.
20. Weis JS, Weis P. Effects of environmental pollutants on early fish development. Aquatic Sci 1:45–73 (1989).
21. Burger J. How should success be measured in ecological risk assessment? The importance of "predictive accuracy." J Toxicol Environ Health 42:367–376 (1994).
22. Burger J, Gochfeld M. Lead and behavioral development in young herring gulls: effects of timing of exposure on individual recognition. Fundam Appl Toxicol 21:187–195 (1993).
23. Gochfeld M, Burger J. Evolutionary consequences for ecological risk assessment and management. Environ Monit Assess 28:161–168 (1993).
24. Burger J, Gochfeld M. Behavioral impairment of lead-exposed herring gulls in nature. Fundam Appl Toxicol 23:553–561 (1996).
25. Sheehan PJ, Miller DR, Butler GC, Bourdeau P, eds. Effects on community and ecosystem structure and dynamics. In: Effects of Pollutants at the Ecosystem Level. Chichester, England:Wiley & Sons, 1986;23–50.
26. Suter GW, II: Endpoints for regional ecological risk assessment. Environ Manage 14:9–23 (1990).
27. Hunsaker C, Carpenter D, Messer J. Ecological indicators for regional monitoring. Bull Ecol Soc Amer 71:165–172 (1990).
28. Burger J, Oil Spills. New Brunswick, NJ:Rutgers University Press, 1997:262 pp.
29. Paine RT. Food web complexity and species diversity. Am Nat 100:65–75 (1966).
30. Abrams PA. Arguments in favor of higher order interactions. Am Nat 121:887–891 (1983).
31. Morin, PJ, Lawler SP, Johnson EA. Competition between aquatic insects and vertebrates: interaction strength and higher order interaction. Ecology 69:1401–1409 (1988).
32. Mills LS, Soule ME, Doak DF. The keystone-species concept in ecology and evolution. BioScience 43:219–224 (1993).
33. Burger J, ed. Before and After the Oil Spill: the Arthur Kill. New Brunswick, NJ:Rutgers University Press, 1993.
34. Klairekampf JF, Hodigins DA, Lutz A. Selenium toxicity and mercury-selenium interactions in juvenile fish. Arch Environ Contam Toxicol 12:405–413 (1983).
35. Brisbin IL Jr., Newman MC, McDowell SG, Peters EL. Prediction of contaminant accumulation by free-living organisms: application of a sigmoidal model. Environ Toxicol Chem 9:141–149 (1990).
36. Gneozi GP, Montague CL. Influence of the energy relationships of trophic levels and of elements on bioaccumulation. Ecotoxicol Environ Safety 30:203–218 (1995).
37. Zillioux EJ, Porcella DB, Benoit JM. Mercury cycling and effects in freshwater wetland ecosystems. Environ Toxicol Chem 12:2245–2264 (1993).
38. Last JM. Housing and health. In: Public Health and Preventive Medicine (Last JM, Wallace RB, eds). Norwalk, CT:Appleton & Lange, 1992:671–675.
39. Colborn T, Clement C. Chemically-induced Alterations in Sexual and Functional Development: the Wildlife/Human Connection. Princeton, NJ:Princeton Scientific Publ, 1992.
40. Fox GA, Gilman AP, Peakall DB, Anderka FW. Behavioral abnormalities of nesting lake Ontario herring gulls. J Wild Manage 42:477–483 (1978).
41. Fry DM, Toone CK. DDT-induced feminization of gull embryos. Science 213:922–924 (1981).
42. Guillette LJ Jr. Endocrine-disrupting environmental contaminants and reproduction: lessons from the study of wildlife. In: Women's Health Today: Perspectives on Current Research and Clinical Practice (Popkin DR, Peddle LJ, eds). New York:Parthenon Publ Group, 1994:201–207.
43. Gillette LJ Jr., Gross TS, Masson GR, Matter JM, Percival HR, Woodward AR. Developmental abnormalities of the gonad and abnormal sex hormone concentrations in juvenile alligators from contaminated and control lakes in Florida. Environ Health Perspect 102:680–688 (1994).
44. Fox GA. Epidemiological and pathobiological evidence of contaminant-induced alterations in sexual development in free-living wildlife. In: Chemically-induced Alterations in Sexual and Functional Development: the Wildlife/Human Connection (Colborn T, Clement C, eds). Princeton, NJ:Princeton Scientific Publ, 1992:147–158.
45. Van-Loveren H, Vos JG, Reijnders JH, Osterhaus ADME. Impairment of immune function in harbor seals (Phoca vitulina) feeding on fish from polluted waters. Ambio 23:155–159 (1994).
46. Grieg-Smith P, Frampton G, Hardy T. Pesticides, cereal farming and the environment. In: The Boxworth Project. London:HMSO, 1992:288–292.
47. Adkisson PL, Niles GA, Walker JK, Bird LS, Scott, HB. Controlling cotton's insect pests: a new system. Science 216:19–22 (1991).
48. Nelson WO, Campbell PGC. The effects of acidification on the geochemistry of Al, Cd, Pb and Hg in freshwater environments: a literature review. Environ Pollut 71:91–130 (1991).
49. Spry DJ, Wiener JG. Metal bioavailability and toxicity to fish in low-alkalinity lakes: a critical review. Environ Pollut 71:243–304 (1991).
50. Schinder DW, Mills KH, Malley DF, Findlay DL, Shearer JA, Davies IJ, Turner MA, Linsey GA, Cruikshank DR. Long-term ecosystem stress: the effects of years of experimental acidification on a small lake. Science 228:1395–1401 (1985).
51. Molen EJ, van Blok AA, deGraff GD. Winter starvation and mercury intoxication in grey herons (Ardea cinerea) in the Netherlands. Ardea 70:173–184 (1982).
52. Bush CP, Weis JS. Effects of salinity on fertilization success in two populations of Fundulus heteroclitus. Biol Bull 164:406–417 (1983).
53. Lemly AD. Metabolic stress during winter increases the toxicity of selenium to fish. Aquatic Toxicol 27:133–158 (1993).
54. Burger J, Gochfeld M. Temporal scales in ecological risk assessment. Arch Environ Contam Toxicol 23:484–488 (1992).
55. Burger J, Gochfeld M. Ecological and human health risk assessment: a comparison. In: Interconnections Between Human and Ecosystem Health (DiGiulio RT, Monosson E, eds). London:Chapman & Hall, 1996:127–148.