Toxic Chemicals: Can What We Don’t Know Harm Us? 1,2

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The Chesapeake Bay Program began more than 20 years ago with assessments of a number of key areas, relying on measurements of habitats, plant and animal populations, and physical and chemical conditions. This approach used wildlife as indicators of Bay “health” and of potential threats to human health. The extent of toxic chemical contamination was one of the assessment endpoints in the original survey. When the initial assessment was completed in 1983, the results of Bay-wide surveys indicated that several specific waterways were contaminated. These waters, the Elizabeth River, Virginia, the James River, Virginia, and Baltimore Harbor, Maryland, were targeted for specific actions to address the problems of historical and ongoing pollution. Over the past 10 years or more, data on some toxic chemical releases into and levels in the environment have been collected, but these data are limited in scope. Furthermore, these data are not used to assess threats to human health or more generally to nonhuman endpoints. New and existing data on environmental levels of chemicals and effects at low concentrations provide evidence that toxic chemicals may threaten both human and nonhuman health in the wider Bay system.

Key Words: toxic chemicals; Chesapeake Bay; health; sentinels; endocrine disruptors.

INTRODUCTION

The symposium of which this paper is a part examines the relation between health of the Chesapeake Bay and that of its citizens; this paper focuses on toxic chemicals. Two compelling reasons prompt an investigation into the relationship between the ecosystem health and that of its human inhabitants. The same two reasons explain the widespread public support for Bay programs. First, the citizens of the Bay region have long been concerned with the health of “their” Bay as a resource, and rightly so. For centuries, the lives and livelihoods of the population have been intertwined with the Bay, dating back before colonization and continuing through the present. The Bay is a source of food, an avenue for transportation, an object of aesthetic appreciation, a part of the spiritual culture, and a site for recreation. The Bay is also a rich laboratory for scientific investigation. The Bay has been considered the most valuable resource of the region and an indicator of the health and condition of the environment in general. In this latter sense, the Bay is both the object of protection and an indicator of the condition of the greater environment.

The other reason that citizens are concerned over the Bay health is that they consider the Bay an indicator of their own health or an early warning system for more direct health threats to themselves. In this sense, the Bay may be thought of as an environmental “canary.” Citizens and regulatory agencies ask if ecosystems or components are showing signs of the effects of toxic chemicals for good reasons. The Bay is home to some substantial industrial complexes in the Baltimore, Maryland, Richmond, Virginia, and Norfolk, Virginia areas. Additionally, extensive farming and livestock operations in the Bay states, especially Pennsylvania and Virginia, use a wide range of pesticides. According to the Bay program (Toxics Fact Sheet October, 1997), approximately 7.2 million acres in the Bay states are under cultivation. Furthermore, population centers must treat both drinking water and wastewater; both types of facilities release toxic chemicals into the air and water and onto the land. The total use and release of toxic chemicals among all these large facilities is great enough that citizens and
government agencies need to know the location, disposition, and effects of these chemicals.

This paper examines the relationship between toxic chemicals in the Bay and effects on human health. We explore the perspective of the Bay as sentinel and as the resource being protected. This paper reviews the concept of indicators and sentinels, some of the historical information about toxic chemicals in the Bay area, and what is known about toxic chemicals in the Bay area now. Finally, the paper addresses the difficult question of whether the present information is sufficient to determine if toxic chemicals threaten human health and Bay health.

THE PROBLEM

The Chesapeake Bay and the Bay watershed ecosystems are too large and complex to address all aspects of toxic chemicals in either. Rather, this paper addresses the issue at the programmatic level, particularly the Chesapeake Bay Program, various monitoring programs, and recent initiatives and Bay-wide activities. From the data generated at the Bay level, we consider the extent to which the Bay and/or its constituent resources are indicators or sentinels for assessing human health. To be more efficient, the review necessarily focuses on key chemicals, defined elements in the ecosystem, and specific regions of the Bay. Traditionally, the toxic chemicals of greatest concern in the Bay area (EPA, 1983) include specific metals, organometals, and several types of organic compounds. The metals lead, mercury, cadmium, and chromium all derive from some of the industrial activities in the Bay area. Tributyltin (TBT) and tetraethyllead are two of the organometals that have posed contaminant problems in the Bay. The organic compounds include pesticides, especially chlorinated pesticides, and polynuclear aromatic hydrocarbons (PAHs) from combustion processes. Though not identified in 1983, dioxins presented contamination problems during the past decade (see for examples deFur, 1991). Several pesticides that are no longer in use, such as DDT and dieldrin, continue to contaminate surface and groundwater in many areas of the United States (Spuril et al., 1998; Kolpin et al., 1998), and DDT is one of the most commonly found contaminants in fish tissues (Hanson, 1997).

The Bay ecosystem system is composed of freshwater, estuarine, and marine habitats, plus the associated wetlands and riparian lands (see Schubel and Pritchard, 1987). The watershed includes the upland areas that drain into the Bay. The term ecosystem is often used synonymously with all living things that are not humans. Such is not, however, an accurate use of the term. The Bay ecosystem really consists of the living resources, the nonliving elements of the environment, and the interactions among all these parts. The key elements for the present discussion are the natural resources that may indicate something about toxic chemicals, those areas where toxic chemicals are most common (the "hot spots"), and some parts of the general Bay waters not in the "hot spots."

There are two obvious approaches to assessing toxic chemicals—the use of Bay health measures as indicators of human health in the Bay and the assessment of Bay health conditions in parallel with those for human health. In the first approach, Bay wildlife and ecosystem health is assumed to be related to human health in a fairly traditional sentinel fashion, much as the canary in the mineshaft was used. Society, largely the management and regulatory agencies, uses wildlife health because there is direct or indirect evidence that humans respond similarly to other animals. What happens to animals will happen to people. For the closest applicability, the animals need to be in the same place at the same time as people and subjected to the same conditions (exposures) as humans.

In the second approach, measuring Bay health conditions along with human health conditions, the wildlife health is used not as a direct sentinel but as an indicator of the larger system, of which humans are a part. In this approach, protecting human health and protecting health of the Bay, in whatever form that takes, are both valued and important endpoints, one does not have to be functionally related to the other. In this case, Bay species are protected because of their intrinsic importance and value. The intersection is important, but human health will not be ignored because an animal sentinel does not respond to the conditions that harm humans. By the same token, society would protect Bay species (e.g., bald eagles) even if human health were not directly and immediately at risk from the same conditions.

A third option, advocated by Suter (1997), a leading expert on ecological risk assessment, is to harmonize assessment of human health and ecological systems. Many EPA management and regulatory activities are carried out under risk assessment frameworks. Suter observed that the two fields would each improve by more closely collaborating and that human health risk assessors have much to learn from ecological risk assessment. He suggested merging the two approaches into a single effort, rather than treating humans and ecological endpoints as entirely separate. This same message was
heard from ecological risk management experts in a recent workshop held in Williamsburg (Bachman et al., 1998). The EcoRisk Management workshop concluded that the artificial separation of human health and ecological risk assessment may promote inefficiencies in managing real situations.

TOXIC CHEMICALS AND HUMAN HEALTH IN THE BAY SYSTEM

The current approach to most environmental issues, especially concerning chemical contamination, is conceptually grounded in toxicology, the conceptual basis for risk assessment (NRC, 1983, 1996). Such an analysis consists of identifying the sources of chemicals, the exposures and exposure pathways, the hazardous effects of chemical exposures, and the relationships between dose and response (see NRC, 1983, 1993; Landis and Yu, 1998, for discussions). The toxicological approach provides a linear and sequential treatment of the situation, based on cause and effect relationships between chemical exposures and health outcomes (or ecological effects). The toxicological view can be greatly limited by the extent of current knowledge, leaving the outcome blind to novel or totally unforeseen situations (either effects or exposures). deFur (1999) argues that the current problems caused by endocrine-disrupting chemicals (see Colborn and Clement, 1992; Kavlock, 1996) are the consequence of such a limitation. The reproductive and developmental effects of chronic exposures were not in the usual range of effects (hazards) under review in traditional risk assessments. Hence, these effects were frequently overlooked. Alternatives to the toxicological approach include both a public health approach and an ecological approach; some aspects are discussed below.

While the term toxic chemicals refers to a great many substances (over 77,000 registered for commerce in the United States, according to EDSTAC, 1998), not all are considered here. The following are omitted from present consideration:

- Toxic effects of excess nutrients in the water
- Drugs
- Bacteria, viruses, and other microbes (especially diseases and their toxins)
- Nutritional status
- Ambient conditions that may be lethal, such as high temperatures and low oxygen

The focus here is on toxic chemical concentrations and releases in the Bay system and those cases where toxic chemicals are known to affect Bay species. This review and analysis cannot be comprehensive, but will take into account major sources of information on chemical releases and concentrations and on effects on wildlife and Bay species. T. Burke evaluates issues related to public health in the Bay area in another paper in this symposium.

ECOLOGICAL SYSTEMS AND COMPONENTS AS INDICATORS OR SENTINELS OF HUMAN HEALTH CONDITIONS

Indicators and sentinels are not identical terms and are not used interchangeably here. Indicator is the more general term for an animal or plant (or group or combination of plants and animals) whose presence or absence results from a change from the ideal or status quo. Indicator species have been used for many years in terrestrial and aquatic ecology to identify deteriorating conditions. Species that are tolerant of disturbance or poor conditions are indicators. Animals and plants that require high-quality conditions for their survival are also considered indicators because their absence indicates deteriorating conditions. Fish and crustaceans that require highly oxygenated waters are indicators of healthy habitats with high oxygen content and their absence is taken as an indication of poor conditions, i.e., low oxygen.

In an earlier survey of Baltimore Harbor, Pitzenmeyer (1971) found that the benthic fauna of the inner harbor was limited to pollution-tolerant species and the more sensitive forms, such as amphipods and isopods, were reduced or absent. Jensen et al. (1974) attributed the depauperate benthic fauna in Baltimore Harbor to acids and metals in the sediments in the vicinity of a steel mill.

The term indicators has an ecological meaning, but is also used outside the strict ecological setting, as in the present case. EPA (1992) applied current information to populations of wildlife in an attempt to determine characteristics of indicators and found that population-level changes were most effective in detecting changes caused by habitat alterations, trophic disturbances, and harvesting pressures that deplete populations. The least effective application of populations as indicators was for factors with unknown or uncertain causal factors, meaning that the population changes could not be attributed to intrinsic or extrinsic factors. Of eight cases examined, only elevated mortality of marine mammals was associated with possible toxic chemical contamination.

Allaby (1991) defines indicator species as those with narrow ecological amplitude regarding one or more environmental factors and thus, when present, indicate particular environmental conditions. Long-lived species, as with species presence over time,
represent integration of the temporal interaction between the species and the environment. This indicator may give a better biological assessment of habitat conditions than can a more precise physical measurement taken on any given day.

According to Pearson (1995), the characteristics of indicator species are:
1. Taxonomically well known and stable (e.g., birds).
2. Biology and general life history well understood.
3. Populations readily surveyed and manipulated—easy to identify, observe, and capture.
4. Higher taxa occupying a breadth of habitats and a broad geographical range.
5. Specialization of each population within a narrow habitat, e.g., tiger beetles < 1% occupying more than one obvious habitat type.
6. Patterns observed in the indicator taxon reflected in other related and unrelated taxa—showing patterns of response to factors, e.g., pollution and habitat degradation, that “presage” those of other members of the community or habitat.
7. Potential economic importance.

Indicators do not necessarily reflect the cause and effect relationships, as do sentinels. The examples mentioned thus far are more indicators than sentinels in demonstrating some change that might be but is not necessarily adverse. The changes expected from individuals or a population of indicator species might be:
- Spatial distribution of individuals or the population
- Abundance of the population
- Diversity of a community of organisms
- Presence or absence of a species
- Health or condition of the individuals in the population, including but not limited to size, age, development, growth rate, disease, behavior, reproduction
- Sex ratio of the population
- Population numbers over time

“Sentinels” are those species that have quite clear and quantifiable response(s) to a specific stimulus—in the present case, chemical exposure. In this regard, sentinels are more specific in their response to some environmental state. The canary in the mine was an effective sentinel animal because it was known to respond rapidly to carbon monoxide poisoning with respirator distress and failure. In present-day use, sentinels may also refer to populations of animals (or plants); egg shell thinning in birds is now considered a sentinel species response to organochlorine chemical exposure (NRC, 1991). This definition of sentinel is consistent with the NRC report in 1991 on the subject. The NRC gave four critical attributes of animal sentinel species:
1. Having a measurable response to the agent or agents in question.
2. Having a territory or home range that overlaps the area under investigation; the animal and the agents must occur in the same place and time.
3. Having individuals that are easily captured and enumerated.
4. Having sufficient population size and density to permit enumeration.

Ever since miners took canaries down into mine shafts (see NRC, 1991) as sensors of poisonous gas (especially carbon monoxide), humans have used and in some cases relied on other animals to protect their own health. Some animals have been recognized as more sensitive than humans to some rather poisonous chemicals. An even more fundamental and factual basis than casual observation of greater sensitivity for indicator or sentinel species lies in the commonality of underlying biological processes and mechanisms. In a recent essay, Mangum and Hochachka (1998) note that biological science has accepted the notion that all living things have a basically similar biochemical/cellular construct on which all life is based. This common biochemistry provides great opportunity for using animals as early warning systems, sentinels, or surrogates. Mangum and Hochachka (1998) also note the equally confounding notion that the millions of living species display a diversity that belies such a common cellular structure and function. But for the present essay, the common mechanism provides the basis for explaining both sentinels and indicators. The apparent paradox of incredible diversity is explained by the adaptive capacity of life in refining common mechanisms to fit specific conditions, such as low salinity estuaries that vary in physical and chemical conditions.

**BAY-SPECIFIC EXPERIENCES ASSESSING TOXIC CHEMICALS**

The initial investigation of Bay conditions included a survey of the distribution and concentration of toxic chemicals in the Bay (EPA, 1983). This survey covered the entire Bay, although at a rather coarse level, and included measuring water and sediment concentrations of a number of metals and organic compounds. Subsequently, Helz and Huggett (1987) reviewed the extent of toxic chemical contamination in the Bay generally and in the “hot spots.” Both EPA (1983) and Helz and Huggett
(1987) concluded that although toxic chemicals were found throughout the Bay, contamination was a threat to resources in specific areas of the Bay, rather than a widespread problem in most waters. The three contaminated areas identified in the 1980s and remaining so today are the ports of Norfolk, specifically the Elizabeth River, Virginia, and Baltimore, specifically the Patapsco River, Maryland, and the James River, Virginia (Fig. 1). The Anacostia River, in Washington, DC, is another site of discharges typical of industrial facilities, particularly from some industry and the Navy yard on the river.

Baltimore, Maryland, has served as an industrial port for many years and was the site of oil refining,
FIG. 2. Distribution of toxic metal contamination in sediments of the mainstem of the Bay and major tributaries (from EPA, 1983), based on a comparative scale that references measured concentrations of Cd, Cu, Cr, Ni, Pb, and Zn to predicted levels, based on sediment cores and rock weathering in the watershed. The highest concentrations are in the regions of the largest cities and at the mouths of the Rappahannock and Susquehanna Rivers.

Steel mills, metal processing, and food processing. These varied and diverse industrial activities produced both metal (Fig. 2) and organic (Fig. 3) contaminants. Various surveys addressed the nature and extent of toxic chemical contamination (see EPA, 1983; Helz and Huggett, 1987). Metals and organic compounds were reported in the harbor sediments, some from known sources (e.g., steel mills and sewage treatment plants) and other chemicals presumably from nonpoint sources or atmospheric deposition. PAHs are found in high concentrations in harbor sediments as a result of the burning of wood and coal in industrial processes. Polychlorinated biphenyls (PCBs) are also found in high levels in Baltimore Harbor; concentrations and number of PCBs decline with distance from the inner harbor. Clearly, these past practices have left a toxic legacy.

The Elizabeth River, Virginia, flowing through the port of Norfolk, was the site of industrial activity for many years and now faces the challenge of cleaning up afterward. Much of the accumulated toxic material is a result of the past practices of burning, creosote processing, and related activities. These have left a legacy of sediments contaminated with industrial by-products, especially PAHs (see Helz and Huggett, 1987). PAHs make up the greater part of creosote, which was used to treat wood to protect against rotting and boring insects. Shipyards still use a variety of toxic chemicals in treating ships and in cleaning and maintaining vessels. Among the toxic materials discharged from shipyards are a number of metals, including TBT, used as an antifouling treatment on ship hulls. TBT was banned from use on small vessels, but is still used on large ships and remains in sediments. TBT is also one of the compounds known to alter normal reproductive function and development in various marine animals, particularly snails (see Colborn et al., 1993).

The other well-documented and characterized site is the contamination of the James River with the pesticide Kepone in the 1970s (Bender and Huggett, 1987). The organochlorine pesticide chlordecone (trade name Kepone), was spilled into the James River at Hopewell, Virginia, and the contamination extended for about 30 miles to Newport News. The river was closed to all fishing from Hopewell to Newport News for many years, on the basis of contaminated fish, crabs, and oysters. The state and the EPA anticipated damage to health and the environment, principally from consumption of contaminated seafood, and direct toxicity, respectively. In recent years, the bans have been lifted, though the Kepone remains buried in about 30 miles of the river. The case supports the use of Bay species as sentinels or monitors of contamination. Crabs are quite sensitive to low levels of Kepone (Schimmel et al., 1979), and crabs, oysters, and fish all accumulate this compound. Fish continue to pick up Kepone in their tissues when they move into the river to spawn in the spring, albeit at low levels. Reevaluation of this contamination is now underway at Virginia Commonwealth University and is presented below.

Audet (1988) summarized U.S. Fish and Wildlife Service (USFWS) monitoring efforts of sediments in the Bay. The USFWS was interested in the distribution and concentration of toxic chemicals as potential threats to the natural resources of the Bay, as was the national NOAA survey of coastal waters. Given the experience with DDT, Kepone, and PCBs...
poisoning some Bay wildlife and habitats, it is logical that the USFWS would monitor sediment contamination. Audet (1988) noted that the levels of contaminants had declined generally and that the “hot spots” in the Bay were characterized and monitored by other programs. The report noted that the contaminants had the potential to impact wildlife in the Bay. Subsequent investigations on the relation between these contaminants and their effects on human health revealed that both people and wildlife are susceptible to the toxic effects of sediment contaminants (Ingersoll et al., 1998).

CHEMICALS IN THE BAY

Sources of Chemicals

The types of toxic chemicals of concern are pesticides, industrial chemicals, waste materials and products, and products of mining and associated materials processing. The Chesapeake Bay Program Office developed a list of toxic chemicals of concern (TOC) and another list of toxics of potential concern (TOPC) for the Bay area; these lists are supposed to be updated regularly (Table 1). The combined list is not meant to identify all of the toxic chemicals in the Bay system, but only those considered of concern due to concentration and location in an exposure path. Indeed, the current list contains only 14 chemicals on the TOC list and an additional 10 on the TOPC list. The list has been under revision for several years now; hence, the most recent approved list is nearly a decade old.

Known sources of toxic chemical inputs to the Bay system are too numerous to identify all here. The following categories of toxic chemical sources cover major source categories as listed by the EPA:

- Industrial manufacturing and processing

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<thead>
<tr>
<th>TOC list</th>
<th>TOPC list</th>
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<tbody>
<tr>
<td>Atrazine</td>
<td>Alachlor</td>
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<tr>
<td>Benz[a]anthracene</td>
<td>Aldrin</td>
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<td>Benzo[a]pyrene</td>
<td>Arsenic</td>
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<tr>
<td>Cadmium</td>
<td>Other PAHs</td>
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<td>Chlordane</td>
<td>Dieldrin</td>
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<td>Chromium</td>
<td>Fenvalerate</td>
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<td>Chrysene</td>
<td>Metolachlor</td>
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<td>Copper</td>
<td>Permethrin</td>
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<td>Fluoranthene</td>
<td>Toxaphene</td>
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<td>Lead</td>
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<td>Mercury</td>
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<td>Naphthalene</td>
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<td>PCBs</td>
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<td>Tributyltin</td>
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Note. The list is current as of the 1990 approval process but has not been finalized in recent years, hence the overdue nature of the approval.
- Waste disposal, storage, and transfer
- Automobile emissions and other internal combustion emissions and waste materials
- Agricultural use of pesticides
- Sewage treatment
- Home use of pesticides
- Golf course use of pesticides
- Military base activities

Some of these (e.g., manufacturing) are included on the EPA Toxic Release Inventory (TRI, see below). Other sources, such as household pesticide use, are not tracked or monitored in any way.

The question posed by this symposium is whether toxic chemicals in the Bay are affecting human health and the environment, especially as indicated by effects on wildlife or similar data. The information in question includes the full range of data discussed here—sources, exposure pathway, wildlife tissue levels, wildlife effects, human exposure pathways, and finally, human health effects. There are no cases that contain this full complement of data and show effects of contaminants that appear first in animals and then in humans.

Not all living systems in the Bay region, non-human and human alike, are equally exposed to or equally as sensitive to all chemicals. Humans are exposed to a range of chemicals that Bay system animals do not encounter or do not encounter at levels that produce effects. Gasoline vapors and automobile exhausts are excellent examples. Humans are exposed to the toxic chemicals in gasoline (e.g., benzene) and automobile fumes (e.g., carbon monoxide) from various sources and activities. Most, but not all, of the constituents of automobile-related emissions never reach the ground or water; humans are exposed through inhalation. There is no reason to expect an animal sentinel to show an effect or even an exposure from these sources, unless the chemicals persist and are deposited on land or water.

Other chemicals simply do not cause a toxic effect at the levels in the general environment, yet may cause effects in humans or in isolated places in the environment. PCBs, dioxins, and some other metals fall in this category. PCBs are found primarily in sediments because they are highly fat-soluble and not water-soluble. Humans are exposed through the food chain by eating animals, notably fish that have accumulated PCBs from the sediments. In some cases, the concentrations of PCBs are increased through food chain accumulation and biomagnification as one animal eats another that already contains PCBs. The exposure of humans to PCBs in the atmosphere.

Exposure Pathways

The exposure aspect of toxic chemical control and investigation has generated substantial regulatory and policy discussion. In toxicology, exposure is one of the major factors considered in evaluating human health impacts from chemicals. The explanation that without exposure there is no effect has a logical simplicity that cannot be denied. If a toxic material never comes into contact with a living system, that system can never be affected by the chemical. This approach is more applicable to humans, occupying only part of the total ecosystem, than to all other living elements combined. Compounds released into the environment will eventually make contact with a living organism. Identifying and eliminating exposures and exposure pathways is a critically important component of public health intervention programs.

The major exposure pathways for toxic chemicals in the Bay system are taken from both traditional toxicology and modern environmental toxicology. The major pathways are respiratory uptake through breathing, dietary uptake in food or water, and absorption through the skin by direct contact or from air. We can also add gestational and lactational exposures to the fetus and neonate. The latter exposure pathways have been recognized as more important than previously considered, principally due to the recent investigation of endocrine-disrupting chemicals (see Colborn et al., 1993).

In the Bay region, the two pathways most commonly associated with the Bay and pollution are uptake in seafood and from water, either in drinking water or from absorption of chemicals across the skin. By far the most important exposure is uptake in food, especially for chemicals that are found in the Bay itself.

Food uptake of toxic chemicals is a well-recognized exposure path that is under some examination at the national level (EPA). Basically, the problem is that some of the most worrisome chemicals are not readily soluble in water (hydrophobic), but do dissolve in fats and oils (lipophilic). As a result, these compounds migrate into fats and oils and remain until the fat or oil is mobilized or released.

Effects in the Bay System

The Bay effort has included resource monitoring in general terms and in a specific sense since the
onset of the program. The resources most studied are those of substantial economic significance (e.g., crabs, oysters, striped bass) as well as ecological significance (e.g., sea grasses or submerged aquatic vegetation [SAV], wetlands, benthic communities, oyster bars). Catch records for the commercial species provide valuable sources of information on population fluctuations and other Bay-wide or river-specific trends in these important species. Only in recent years have more intense efforts turned to monitoring noncommercial living resources (e.g., SAV).

Oyster catches have been declining for more than a few decades, according to both historical and recent data. One of the first surveys of oyster populations was conducted by Baylor over concern that catches were falling, especially catch per unit effort (Haven, 1987). Indeed, more than a century ago Brooks (1891) reported on the declining oyster harvest and attributed the decline to overharvesting and mismanagement. Recently, when the state of Maryland sought a consensus strategy to recover oyster populations and harvest, overharvesting and management problems were recognized as but two causes for oyster declines. Oyster parasitic infections (Dermo and MSX) take a heavy toll, and the contributions from deteriorating water quality and toxic chemical poisoning are now acknowledged, but largely unexamined (Maryland Oyster Roundtable Report, 1995).

Sea grasses in the 1980s were the object of investigation because of the realization that some seagrass areas were now sparse or had been depleted of grasses entirely. Indeed, in the Bay assessment (EPA, 1987) the EPA recorded dramatic declines in sea grasses, and the term submerged aquatic vegetation came into popular use in the Bay program. SAV surveys continued for many years, and even today, as a way of charting progress toward recovery. The cause of the SAV decline was not apparent at first. Following some investigation, it was concluded that overenrichment of the aquatic system and excess turbidity from sediment both contributed to obscuring sunlight and limiting the photosynthetic capability of SAV. This was coupled with increased deposition of sediment on the plant surface, causing growth of a variety of microorganisms, some of which challenged the plants systems and increased the disease incidents. But it was fairly clear that no direct or even indirect effects of chemicals were responsible for the decline in SAV.

During the early years of the Bay cleanup, and even in the preceding years, bald eagle, osprey, and peregrine falcon populations declined throughout much of the East Coast and the Great Lakes (see Cade, 1990 for review). The cause turned out to be the pesticide DDT impairing eggshell formation in these birds. Eagle eggs especially were too thin to support the weight of the adult birds incubating the eggs and thus were crushed. DDT was found to cause eggshell thinning in some bird species and high DDT residues in several species of piscivorous birds confirmed the nature of the problem. High DDT residues were also found in osprey and peregrine falcons, contributing to the decline of these populations as well. Falcons were eventually extirpated from the East Coast and only through federal agency efforts were reintroduced from animals in other parts of the country.

Two critically important conclusions came from the work on bald eagles and other raptors on the East Coast. One conclusion was that wildlife species can serve as effective sentinel species, as noted by the NRC (1991). This conclusion supported the creation of programs to monitor wildlife populations for the presence or effects of contaminants in the environment. The other important point from the work on bald eagles and other species affected by DDT is that acute poisoning is not the only factor contributing to population declines, especially in regional declines of substantial magnitude. Eggshell thinning was a serious affect not observed as acute mortality of the exposed animals. The offspring suffered in much the same way that scientists recognize that endocrine-disrupting chemicals affect reproductive and developmental processes. Nor was chemical poisoning the sole impact on eagle populations. Human activities encroaching on eagle nesting sites resulted in nest abandonment and limitations on reproduction at the population level. Through this research, scientists and resource managers came to realize the potential adverse effects of habitat degradation and reduction or loss.

Another case in which the effects of toxic chemical contamination have been shown to affect populations of Bay species is in the Elizabeth River, Virginia. This river system is heavily impacted from long use for various industrial activities. One major chemical problem is high levels of PAHs in the sediment, primarily from past industrial combustion using coal, from coal processing, and from old creosote plants on the river (Helz and Huggett, 1987). Although the sources are long closed, the creosote residues remain in the sediment, along with a number of other contaminants, such as metals and PCBs. While the metals and the PCBs in the river sediments are known to affect human health and both are taken up and passed along to humans via...
seafood, PAHs are not treated the same way by most marine animals. Most PAHs are not passed along and are not toxic in their immediate form, but the metabolic breakdown products are quite toxic. Initial surveys of fish in the vicinity revealed a high incidence of tumors (Klauda and Bender, 1987). Researchers have continued to monitor and investigate this heavily contaminated site. The latest findings (Van Veld et al., 1997) indicate that the fish Fundulus still is able to reproduce in the Elizabeth River, despite lethal incidences of liver tumors in the population, because reproduction occurs prior to death. Furthermore, enzyme levels in this population are altered to accommodate the high concentrations of toxic PAHs in the sediment.

The Kepone contamination of the James River was discovered when three seemingly unrelated events took place in Hopewell, Virginia; monitoring for effects was not involved at all. The three events were: (1) the failure of sewage treatment processes due to death of the microbial flora that process industrial wastes at the waste treatment facility in Hopewell, Virginia; (2) the illegal midnight dumping of barrels of industrial waste in the wetlands; and (3) the admittance of workers with acute neurotoxicity symptoms to medical facilities (Taylor et al., 1978).

A reexamination of this contamination problem in 1997–1998 by Virginia scientists led to further understanding of contamination in the James River (Garman et al., 1998). This work originally focused on Kepone levels in larger, older catfish of several species. The complete study included measurements of 26 toxic chemicals (metals, pesticides, PCBs, etc.) in fish tissues, the standard EPA protocol for measuring fish tissue contamination. Kepone was not found above the analytical detection limit in catfish from the James River, but other toxic compounds were found in sufficiently high concentrations to warrant concern. PCBs, organotin compounds, mercury, and DDT and metabolites were found in catfish tissues and many of the catfish, especially males, appeared to exhibit arrested or indeterminate sexual development. Problems with sexual development associated with or caused by contamination from several of these compounds have been reported in snails, male fish, and alligators (see Colborn and Clement, 1992; Colborn et al., 1993; Kavlock et al., 1996). As of this writing, the EPA and the Virginia Department of Environmental Quality are examining the threat to human health and the need for cleaning up the contaminated sediment. Clearly, a biomonitoring effort using fish as indicators has promise for this river system.

These catfish are fished recreationally and by subsistence fishers on the James River. In this case, the sources are industrial and municipal discharges, likely organotin leaching off the hulls of large ships on the James River and nonpoint source runoff from the land. Runoff includes atmospheric deposition of chemicals such as PCBs that can be transported long distances in air. Runoff is also the most likely explanation for the presence of some of the banned pesticides, such as DDT and chlordane.

**MONITORING PROGRAMS IN THE BAY**

United States Geological Survey

The USGS has for some years monitored groundwater and surface water quality through the National Ambient Water Quality Assessment Program (NAWQA; U.S. Geological Survey, 1996; Lindsey et al., 1998; Spruill et al., 1998). This program provides some of the best and most well-documented data on the occurrence and distribution of pesticides, metals, and industrial compounds in both types of water. USGS data are available in hard copy and from The internet Web site as well (see Lindsey et al., 1998). The program assesses water quality on a watershed or basin level, based on the USGS determinations of 50 major watersheds and basins throughout the nation. These study areas are called Study Units and cover about half of the U.S. land mass and about 70% of the water supply for the United States. Not all of the Study Units are under the same level of investigation simultaneously; comprehensive investigations are underway in about one-third. Recent data are presented here for the Potomac and Susquehanna Study Units in the Chesapeake Bay area (Fig. 4).

A range of organic chemicals and metals are found in streambed sediments in the lower Susquehanna River basin (Figs. 5 and 6). The highest concentrations occur in areas with mixed agricultural and industrial uses, compared to concentrations in areas of forest and forest mixed with agriculture (Fig. 5). The PAHs, phthalates, and phenols detected in streambed sediments are products associated with industrial practices; some are widespread in other waterways as well (Hale et al., 1999) and may be found throughout the United States. PAHs in urban/industrial areas were high enough to raise some concerns for human health and aquatic life. Both fish tissues and sediments also accumulate chlorinated organic compounds, including pesticides and PCBs (Fig. 6). As with the chlorinated organic compounds, all three groups of compounds were highest in industrial-use areas. Chlordanes, PCBs, and DDT
were all high enough in fish tissues to raise concerns for human health, although, according to Lindsey et al. (1998), the appropriate Pennsylvania agencies did not find a need to issue health warnings for the species sampled (white sucker and small mouth bass).

A number of pesticides occurred in both well water and streams (Fig. 7), and the same six most frequently detected pesticides were the same in both wells and streams. More than one pesticide was detected in almost all cases, and the most commonly detected pesticides were herbicides used on corn (atrazine, metolachlor, simazine, prometon, alachlor, and cyanazine). Atrazine, one of the most commonly used and detected herbicides across the nation, was found in 98% of the stream samples and 74% of the well samples in the Lower Susquehanna River basin (Lindsey et al., 1998). The presence of chemicals in either water source is related to use patterns (e.g., Figs. 5 and 6), as well as to the regional geological characteristics and chemical properties of the compounds. Some soils are more prone to leaching (Fig. 8), particularly limestone, and some compounds, such as atrazine, are more mobile and have a higher leaching potential.

Compounds such as chlordane and some of the other chlorinated organic compounds break down slowly under natural conditions and therefore remain in stream sediments for many years, even after use and input to the system has long since stopped. DDT, another lipophilic and persistent compound, was present at 23 of 26 sites. Concentrations of chlordane in fish tissues and sediments are directly related (e.g., chlordane, Fig. 9), due to the properties of the chemicals in question. These lipophilic chemicals adsorb to organic particles and dissolve readily in fats, not in water, and the major route of uptake is via food ingestion, either in the food or as sediment is taken in incidental to feeding.

The levels of many of these compounds are below the point at which traditional testing reveals either acute or chronic effects. For some of the compounds, including mercury, lead, chlordane, PCBs, and DDT, aquatic life is at risk from the adverse effects at the levels found in streams in the Potomac River basin (Figs. 10 and 11). According to the USGS, sediment at 14 of 23 sites had concentrations high enough to pose a risk to aquatic life (Lindsey et al., 1998) and at 6 sites, there was a high likelihood of harm to aquatic life. The screening levels are based on laboratory toxicity tests and adjustments made to account for variation among species; hence, the screening level is not able to conclusively identify what species might be affected. Clearly, these data point to the need to complete more thorough field and laboratory examinations and to explore the possibilities of remedial actions to protect both human health and the environment.

It is clear from the USGS data that pesticides and industrial chemicals are widely distributed in the Bay region and are found in inland as well as tidal waters. The most commonly occurring pesticides are DDT and other chlorinated organic compounds, primarily because they were widely used and are highly persistent. Additionally, PCBs are found in many sites and are highly persistent and
Concentrations (in parts per billion) of PAHs, phthalates, and phenols in stream sediments in the Lower Susquehanna River basin, according to land use of the surrounding land. Four sites on large rivers were excluded from this comparison because the sites had characteristics dissimilar to those of the other sites. The numbers of sites are given below the land-use type.

Selected organochlorine pesticides (chlordane and DDT) and PCBs in sediment and fish (white sucker) tissues in the Lower Susquehanna River basin, according to land use of the surrounding land.

Transportable in air and in water, though much more so in the former. Lead and mercury, for much the same reasons, persist in waters, sediments, and fish tissues. One of the greatest unknowns is whether the combination of these and other toxic chemicals has effects on human health or the environment.

Toxic Release Inventory

The Toxic Release Inventory (TRI) is a database of quantities of chemicals released into the environment from or stored on-site at certain facilities and is mandated by federal law. Reports are filed annually with the EPA and reported publicly in several forms. One readily accessible location is the WWW site of the EPA for the TRI data (www.epa.gov). These reporting requirements cover specific types of facilities and chemical compounds that are released in excess of a certain amount. The TRI reports for the Bay states of Virginia, Maryland, and Pennsylvania and for Washington, DC are summarized from the internet data for all chemicals reported (Table 2).

A few trends are apparent in the data for all chemicals and all facilities by state. The District of Columbia, with only three facilities, makes no substantial contribution to the total and is not considered further. Air emissions dominate the list of total releases, but the total amounts of on-site waste management and energy recovery (using the waste as fuel for another process) greatly exceed the amounts released from all categories. The distribution of emissions and releases is not even among the Bay states, especially when considered on the basis...
of pounds released per facility. On this basis, pounds released per facility, Virginia has proportionately more air emissions (114,116 lbs/facility) and Pennsylvania has proportionately more water discharges (18,997 lbs/facility). Virginia facilities have about twice the total on-site releases in lbs/facility as the other two states. The most dramatic difference is the pounds released per member of the population (lbs/person): Virginia = 8.6; Maryland = 2.6; Pennsylvania = 5.6 (Table 2).

Individual state summaries give data on the top 10 chemicals across all release categories for each of the states (Tables 3-5). A few toxic compounds dominate the total emissions reported under TRI—methanol, ammonia, and toluene. Methanol and ammonia occur in nature yet are the by-products of a large number of industrial and municipal processes, including organic waste treatment, such as sewage treatment (ammonia). Toluene is a solvent used in various extraction and distillation processes. Volatile organic compounds (including methanol) make up a substantial fraction of the releases into air and water; these are solvents and industrial materials that may be used in various processes (e.g., production of styrene), and, of course, metals and metal compounds are found in the releases into all media.

Some of the differences among the states simply reflect the different industrial bases and population centers. Virginia and Pennsylvania have petrochemical facilities and Maryland is home to steel production in Baltimore. Pulp and paper mills, cement factories, waste incinerators, and chemical manufacturing all have different combinations and amounts of chemical use and release. These differences in facility processes explain the variations in the chemical constituents on the top 10 list for each state (Tables 3-5). It is more difficult to explain why the pounds released per facility is so variable from one state to the next. One explanation is that each state varies considerably in the administration of the permit programs for water, air, and solid waste releases.

While all of these compounds are listed because they are toxic and released in excess of a certain amount, the matter of threats to human health and the environment remains undetermined from these data. Looking at the effects question toxicologically,
FIG. 9. Chlordane in sediments and tissues of three fish species for selected sites in the Potomac River basin. ND, not detected.

Releases are but one element; the delivered dose and potential effects must also be taken into account. Of the amounts of TRI chemicals released, there are no estimates of their fate at a regional level. Some efforts estimate fate and transport of emissions or discharges for regulatory purposes, but there is no overall effort to determine what happens to the millions of pounds released into the air, water, and land of the Bay states.

Interestingly enough, at least some of the metals must wind up in the Bay waters and sediments, because the surveys by the EPA (1983), the NOS (Hanson, 1997), and the USFWS (1988) report metals in waters, sediments, and wildlife tissues. Furthermore, these same metals (e.g., lead, chromium, zinc, manganese, and nickel) are known threats to human health when the exposure (dose) is sufficient (ATSDR, 1997).

State Agencies

State agencies conduct water quality monitoring through two programs: the discharge permits program and water quality monitoring. Any facility that discharges into waters of the state must first secure a National Pollution Discharge Elimination System (NPDES) permit. In the Bay, these permit programs are operated by the state agencies, under authority from the EPA, as described in the Clean Water Act.

Discharge permit holders are required to file monthly reports on the materials found in the discharge, for those chemicals listed in the permits. State agencies must also report to the EPA on the state of the water quality and whether rivers, lakes, and other bodies of water can be used for swimming, fishing, and other uses. To accurately report to the EPA, agencies need to conduct some sort of monitoring program to provide the necessary data. These monitoring programs assess primarily temperature, pH, bacteria levels, oxygen levels, suspended sediment, and odor.

National Oceanographic and Atmospheric Administration (NOAA) National Ocean Survey (NOS)

Beginning in 1984, NOAA conducted a survey of coastal waters to determine the extent of contamination from land-based sources. The survey consisted of measuring the concentrations of a series of metals and organic chemicals in sediment samples, fish tissue, and mussel or oyster tissue. The survey sampled from all coasts, including several sites in the Bay region, both industrialized (Elizabeth River) and rural (York River). This effort documented some trends that were expected, to say the least. Major harbors and rivers were the most contaminated sites, and locations with no industrial or other...
human activities had no or little measurable contaminants. Metals, industrial chemicals, and pesticides were found commonly in coastal sediments. One of the few surprises was the presence of relatively high concentrations of DDT and its by-products and PCBs, even though both were banned by the mid-1970s.

Hanson (1997) used the results of the ongoing Benthic Surveillance Project of the NOS to identify relationships between sediment and liver contaminant concentrations in fish. Hanson used a large number of contaminants (organic and metals) from more than two dozen sites to determine major trends that might be obscured in smaller data sets. Sediment and fish liver samples were collected from Maine to Texas; Hanson compared the North Atlantic coast (north of Raritan Bay, NJ) with the south Atlantic and Gulf coasts. The more contaminated sites in the northeast region resulted in elevated liver contaminants compared with the mid-Atlantic and Gulf regions. Hanson concluded that when analyzed properly, the liver contaminants were an effective indicator of sediment pollution.

One of the important conclusions from the NOAA survey efforts is the confirmation that sediments and fish tissue do contain measurable levels of toxic metals and organic compounds. Some of these chemicals are present in fairly high levels, consistent with the results of the USGS surveys.

CONCLUSIONS

The available data provide little complete information on the relationship between Bay and human...
FIG. 11. Concentrations of five contaminants (lead, mercury, DDT, PCBs, and chlordane) from sediments at selected sites in the Potomac River basin shown in Fig. 10.

health effects from toxic chemical exposures. The data do show the release of large quantities of toxic chemicals into the Bay ecosystem, including air, water, and land. Hot spots of contaminated sediments around the areas of Baltimore, Washington, and Norfolk remain.

Biological systems are sufficiently conservative in structure and function that Bay animals and systems can and should be used as sentinels for health of a wide array of animals, including humans. Animals in the Bay system have great potential for serving as indictors and sentinels for monitoring both human health and Bay health conditions. Examples include fish in the Elizabeth and James Rivers and animals living in the bottom of Baltimore Harbor. But the Bay program and state agencies make insufficient use of indicators and monitors. The overlap between human health and ecological health has not been as fully examined and used in the Bay as it might be.

While animal sentinels or indicators are viable and useful, with many known examples of the connection between the effects of contaminant exposures on the health of wildlife and those on the health of humans, the relationship is not sufficiently tight to rely on its ability to satisfactorily protect human health. One of the limitations is the diversity of chemicals found in and released into the Bay; the other is the diversity of biological systems in the region. Some wildlife species are more sensitive than humans and therefore are excellent candidates for indicator or sentinel species. In other cases, the use of animal sentinels has simply not been developed with scientific data.

Some of the toxic chemicals in the Bay system have characteristics that do not permit quick and
TABLE 2
Toxics Release Inventory (TRI) Summary Data for the Chesapeake Bay States, Based on Data Extracted June 10, 1998

<table>
<thead>
<tr>
<th></th>
<th>Virginia</th>
<th>Maryland</th>
<th>Pennsylvania</th>
<th>Washington, DC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population</td>
<td>6,187,358</td>
<td>4,781,468</td>
<td>11,881,643</td>
<td>606,900</td>
</tr>
<tr>
<td>Number of facilities</td>
<td>444</td>
<td>195</td>
<td>1,189</td>
<td>3</td>
</tr>
<tr>
<td>On-site releases of air emissions</td>
<td>50,667,719</td>
<td>8,574,878</td>
<td>41,419,631</td>
<td>3,910</td>
</tr>
<tr>
<td>Surface water discharges</td>
<td>1,190,262</td>
<td>1,845,508</td>
<td>22,588,120</td>
<td>300</td>
</tr>
<tr>
<td>Underground injection</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Releases to land</td>
<td>1,068,761</td>
<td>1,920,064</td>
<td>2,563,778</td>
<td>5,000</td>
</tr>
<tr>
<td>Total on-site releases</td>
<td>52,926,752</td>
<td>12,340,450</td>
<td>66,571,529</td>
<td>9,210</td>
</tr>
<tr>
<td>On-site waste management recycling</td>
<td>178,353,208</td>
<td>18,543,556</td>
<td>387,088,578</td>
<td>0</td>
</tr>
<tr>
<td>Energy recovery</td>
<td>29,707,489</td>
<td>13,397,901</td>
<td>50,287,872</td>
<td>0</td>
</tr>
<tr>
<td>Treatment</td>
<td>317,701,825</td>
<td>34,012,979</td>
<td>149,234,860</td>
<td>1,695</td>
</tr>
<tr>
<td>Total on-site waste management</td>
<td>317,701,825</td>
<td>65,954,436</td>
<td>586,611,310</td>
<td>1,695</td>
</tr>
</tbody>
</table>

Note: All chemical amounts are reported in pounds. For all releases estimated as a range, the midpoint of the range was used in these calculations.

reliable predictions of effects based on the initial estimates of exposures. These chemicals are persistent, bioaccumulative, frequently found in or associated with soils and sediments, and exert toxic effects after some time period. Some of these chemicals (e.g., PCBs, TBT, dioxins) exert effects at low concentrations, and perhaps in the next generation of animals.

Humans are exposed to conditions that Bay animals and plants do not experience, and therefore human health protection needs to include monitoring and assessment activities not specifically included in the Bay programs. Air quality is the most obvious area in which enhanced monitoring is needed to protect human health, especially in population centers.

Few or no data exist on the multiple exposures that humans and the living resources in the Bay system face. No one knows whether exposure to 500 chemicals at low levels will have or is having some harmful effect on humans or on the Bay. It is not clear whether the multiple stresses faced by organisms living in estuarine conditions normally are protective against the additional stresses caused by human activities or instead serve to make the responses more severe and therefore more serious.

The delay or lag time present in many Bay programs means that by the time some problems are identified, the conditions will have altered, some for the worse. The most toxic chemicals are persistent and many are accumulative, as with DDT. Damage from activities that were halted years or decades ago

TABLE 3
Virginia Toxics Release Inventory Summary 1996: Top 10 Chemicals for Total Releases

<table>
<thead>
<tr>
<th>Chemical name</th>
<th>Air emissions discharges</th>
<th>Surface water</th>
<th>Releases to land</th>
<th>Total releases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Methanol</td>
<td>13,574,709</td>
<td>40,915</td>
<td>35,108</td>
<td>13,650,732</td>
</tr>
<tr>
<td>Toluene</td>
<td>7,196,850</td>
<td>566</td>
<td>154</td>
<td>7,197,570</td>
</tr>
<tr>
<td>Ammonia</td>
<td>5,726,628</td>
<td>219,025</td>
<td>11,483</td>
<td>5,957,136</td>
</tr>
<tr>
<td>Hydrochloric acid (1995 and after: &quot;acid aerosols&quot; only)</td>
<td>3,718,621</td>
<td>0</td>
<td>0</td>
<td>3,718,621</td>
</tr>
<tr>
<td>Methyl ethyl ketone</td>
<td>2,823,289</td>
<td>628</td>
<td>1,780</td>
<td>2,825,697</td>
</tr>
<tr>
<td>Xylene (mixed isomers)</td>
<td>2,489,318</td>
<td>0</td>
<td>355</td>
<td>2,489,673</td>
</tr>
<tr>
<td>Dichloromethane</td>
<td>1,434,825</td>
<td>0</td>
<td>0</td>
<td>1,434,825</td>
</tr>
<tr>
<td>Sulfuric acid (1994 and after: &quot;acid aerosols&quot; only)</td>
<td>1,422,153</td>
<td>0</td>
<td>19</td>
<td>1,422,172</td>
</tr>
<tr>
<td>N-Hexane</td>
<td>964,473</td>
<td>0</td>
<td>0</td>
<td>964,473</td>
</tr>
<tr>
<td>N-Butyl alcohol</td>
<td>879,184</td>
<td>1,600</td>
<td>0</td>
<td>880,784</td>
</tr>
</tbody>
</table>

Note: Results are based on data extracted on June 10, 1998. All chemical amounts are reported in pounds. For all releases estimated as a range, the midpoint of the range was used in these calculations. Underground injection was equal to zero for all top 10 chemicals and therefore is not included in the table.
TABLE 4


<table>
<thead>
<tr>
<th>Chemical name</th>
<th>Air emissions</th>
<th>Surface water</th>
<th>Releases to land</th>
<th>Total releases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>discharges</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitrate compounds</td>
<td>1</td>
<td>21,512,168</td>
<td>0</td>
<td>21,512,169</td>
</tr>
<tr>
<td>Toluene</td>
<td>6,899,085</td>
<td>1,296</td>
<td>1,623</td>
<td>6,902,004</td>
</tr>
<tr>
<td>Dichloromethane</td>
<td>4,364,672</td>
<td>76</td>
<td>0</td>
<td>4,364,748</td>
</tr>
<tr>
<td>Methanol</td>
<td>4,088,085</td>
<td>18,619</td>
<td>3,710</td>
<td>4,110,414</td>
</tr>
<tr>
<td>Hydrochloric acid</td>
<td>3,136,780</td>
<td>500</td>
<td>0</td>
<td>3,137,280</td>
</tr>
<tr>
<td>Methyl ethyl ketone</td>
<td>2,885,929</td>
<td>5,178</td>
<td>0</td>
<td>2,891,107</td>
</tr>
<tr>
<td>Ammonia</td>
<td>2,412,609</td>
<td>193,565</td>
<td>1,414</td>
<td>2,607,588</td>
</tr>
<tr>
<td>Xylene (mixed isomers)</td>
<td>2,578,002</td>
<td>630</td>
<td>14</td>
<td>2,578,646</td>
</tr>
<tr>
<td>Trichloroethylene</td>
<td>1,759,544</td>
<td>363</td>
<td>0</td>
<td>1,759,907</td>
</tr>
<tr>
<td>Styrene</td>
<td>1,562,802</td>
<td>102</td>
<td>12,965</td>
<td>1,575,869</td>
</tr>
</tbody>
</table>

Note. Results are based on data extracted on June 10, 1998. All chemical amounts are reported in pounds. For all releases estimated as a range, the midpoint of the range was used in these calculations.

persists for years and could have been prevented or at least lessened through expeditious monitoring and assessment programs.

Is There Harm?

The short answer is that no one knows for sure, but citizens want to know and state and federal agencies should be looking. The recent realization that compounds can act at low levels on reproduction and development in a variety of animals (Colborn and Clement, 1992; Kavlock et al., 1996) has prompted a reexamination of the adequacy of toxicity testing and evaluation. Recently, Roe et al. (1997) found that the majority of chemicals in high production volume in the United States had only rudimentary toxicity data available on which to base regulatory decisions. Roe et al. (1997) based their work on a random sample of 100 chemicals on the high production volume list, but the conclusions held across a larger group of the more than 75,000 chemicals registered for use in commerce. EPA has now instituted a program, albeit a voluntary one, to correct this deficiency in chemical registration and testing programs.

State environmental and health departments are charged with the responsibility of monitoring conditions related to human and environmental health, reporting results to the public, and taking appropriate actions. The discovery of toxic chemicals (PCBs, DDT, TBT, chlordane, and mercury) in sediments and fish tissues in the James River, Virginia, below Hopewell (Garman et al., 1998) points to the need for increased diligence by state agencies in maintaining

TABLE 5

Maryland Toxics Release Inventory Summary 1996: Top 10 Chemicals for Total Releases

<table>
<thead>
<tr>
<th>Chemical name</th>
<th>Air emissions</th>
<th>Surface water</th>
<th>Releases to land</th>
<th>Total releases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>discharges</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nitrate compounds</td>
<td>10</td>
<td>1,516,774</td>
<td>80,267</td>
<td>1,597,051</td>
</tr>
<tr>
<td>Manganese compounds</td>
<td>34,093</td>
<td>77,306</td>
<td>1,460,610</td>
<td>1,572,009</td>
</tr>
<tr>
<td>Carbonyl sulfide</td>
<td>1,357,000</td>
<td>0</td>
<td>0</td>
<td>1,357,000</td>
</tr>
<tr>
<td>Methanol</td>
<td>1,057,563</td>
<td>0</td>
<td>0</td>
<td>1,057,563</td>
</tr>
<tr>
<td>Toluene</td>
<td>780,436</td>
<td>18</td>
<td>0</td>
<td>780,454</td>
</tr>
<tr>
<td>Xylene (mixed isomers)</td>
<td>636,312</td>
<td>9</td>
<td>0</td>
<td>636,321</td>
</tr>
<tr>
<td>Styrene</td>
<td>601,006</td>
<td>0</td>
<td>0</td>
<td>601,006</td>
</tr>
<tr>
<td>Sulfuric acid (1994 and after “acid aerosols” only)</td>
<td>469,230</td>
<td>0</td>
<td>0</td>
<td>469,230</td>
</tr>
<tr>
<td>Ammonia</td>
<td>388,160</td>
<td>24,886</td>
<td>17,745</td>
<td>430,791</td>
</tr>
<tr>
<td>Certain glycol ethers</td>
<td>427,589</td>
<td>0</td>
<td>5</td>
<td>427,594</td>
</tr>
</tbody>
</table>

Note. Results are based on data extracted on June 10, 1998. All chemical amounts are reported in pounds. For all releases estimated as a range, the midpoint of the range was used in these calculations.
monitoring efforts, even for banned or restricted chemicals.

Next Steps: Monitoring for More Information

As extensive as local and even national efforts to monitor and assess the condition of the Bay have been, these efforts have been limited and unevenly successful. The original assessment of the Bay, completed more than 15 years ago in 1983 and launching the cleanup effort (EPA, 1983), was a lengthy and fairly comprehensive assessment. This assessment was based on existing literature and field surveys completed over a period of years. When the cleanup began in more earnest, workgroups were formed, including one on toxic chemicals. Much of the monitoring and cleanup effort was relegated to the state level where agencies were charged with enforcing discharge limits and other control measures. Since 1983, the Bay Program has continued some attention on toxic chemicals, but toxic chemicals have not been a priority or a focus of the Bay Program. The inability of the Bay Program toxic chemicals workgroup to finalize the 1996 Toxics of Concern list, as of the writing of this paper, is evidence for this conclusion.

The EPA undertook a more elaborate and far-reaching program to assess environmental conditions, presumably across the entire country, and began at the East Coast in the mid-Atlantic region. This program, the Environmental Monitoring and Assessment Program (EMAP), included measures of the relationships among ecological and human health indicators. One goal was to understand some of the question posed in this symposium—the relationships between environmental and human health. Laudable as the goals were, EMAP met with mixed reviews at best. In a recent evaluation, the National Research Council (NRC, 1995) concluded that the undertaking was too grand and not sufficiently well focused to achieve meaningful results in comparison to the resources EPA devoted to EMAP. In the wake of EMAP, EPA and state agencies are not monitoring water quality with respect to contamination from toxic chemicals through either water, sediment, or fish tissue assessment programs.

Policy Options: Risk Assessment and the Precautionary Principle

The present regulatory approach in the United States has increasingly used risk-based programs (NRC, 1983, 1996). These programs have had mixed success and the most difficulty in dealing with toxic chemicals at low levels that exert effects that are less visually obvious. The risk-based approach makes a number of assumptions, not the least of which is that the problems are amenable to a toxicological analysis (e.g., exposure, effects, dose response, characterization). In addition, the toxicological approach is much more applicable to cases of the known than to cases of the unknown or the predicted. Finally, the risk-based approach does not account for pollution prevention or for taking a whole-systems approach that includes widespread use of sentinel or indicator species.

Four Principles for Policy

An alternative to a risk-based approach could be incorporated into new policies to protect the environment and human health from the effects of chemicals released into the environment (deFur, 1999).

I. Every action needs to improve the environment. Keeping the status quo is not sufficient. The assumption that many actions taken in modern society were relatively environmentally neutral has simply not proven true. Products, chemicals, and energy use have changed the local, regional, and global environment. Now, the next generation of decisions must promote and implement environmental recovery from many past actions.

II. Every act has an effect. The laws of thermodynamics apply here and for every action there will be an equal and opposite reaction. Creating, using, and releasing chemicals will have some response in the “environmental system.” The lesson from chemicals that act on hormonal systems (e.g., see Colborn et al., 1993) is that even “neutral” chemicals have unexpected effects—no one looked for these effects at first. New policies must anticipate the unexpected by assuming that chemicals have toxic effects.

III. The timing makes the poison. Past toxicological practice has been that the dose makes the poison. But now it is clear that some chemicals exert an effect only when administered at precise times, and perhaps without an effects threshold. It is not possible to control the timing of an exposure to chemicals present in the environment; therefore, guarantees of protection, safety, or even estimated risk are simply not possible.

IV. Shift the burden of proof to the applicant and when in doubt, do not proceed. This policy assumes that one more chemical, one more exposure, or even more of the same chemical will cause harm. The
burden of proof must shift to the applicant, the proponent, the activity, or the facility to demonstrate no harm. The automatic default must not allow further releases, emissions, or uses when we cannot predict the consequences. Society can no longer allow activities to go forward, as a matter of default action because of insufficient data to “prove” that it will cause harm. A corollary to this point is that less contamination is better: The fewer toxic chemicals put into the environment, the better. With time and normal ecological processes, some past errors will be covered and no longer accessible; others will be biologically processed, but slowly. Lead will not degrade, and dioxin only degrades slowly.

REFERENCES


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