

Prevention of Environmental Pollution: Good for Our Health

Today, there is increasing recognition of the health costs, as well as the ecological and economic costs, of environmental contamination and the need to avert damage through pollution prevention. The shift from the strategy of controlling pollutants after they have been produced ("mopping up") to preventing their generation ("turning off the faucet") is already beginning. However, the transition will necessitate an integration of the two approaches and a continued reliance on established methods to estimate risks and set priorities. Indeed, scientists, environmentalists, and policy-makers are already working together to strengthen and reorient the current environmental program by incorporating new developments in toxicology and management without discarding methodologies that remain valid after decades of use.

At this point in the evolution of national environmental policy, it is important that we take stock of the achievements, as well as the failures, since the major environmental statutes were passed in the 1970s. Unfortunately, we often hear about the failures and not the gains, and some critics have been harsh indeed, pronouncing past and current environmental policy "the product of bad science and worse economics." They have dismissed as irrelevant laboratory testing systems used for decades to screen chemicals for toxicity, and they have discounted the health benefits of environmental protection. The "bottom line" in their view is that we have wasted vast amounts of money to fix trivial risks and that sweeping reform is needed. Although it represents an extreme position, this viewpoint is particularly damaging at a time when public understanding is essential to forward-looking initiatives such as efforts by EPA and other federal agencies to reduce the use of pesticides in this country.

What is the record on the health hazards of environmental pollutants? According to EPA, more than three and a half billion pounds of toxic chemicals are released each year directly to the air, surface water, land, or to underground injection wells. An additional one to two billion pounds of pesticides are used each year in the United States. A number of years ago a committee of the National Research Council estimated that few of the 50,000 chemicals in commerce and only 10% of pesticides and their inert ingredients had sufficient information on toxicity for a complete health-hazard evaluation. Even today we do not know enough about the potential of most of the chemicals in our environment to cause such serious effects as cancer, reproductive impairment, birth defects, harm to the developing child, neurologic disease, and damage to the immune system.

We do know, however, that most toxic chemicals exert multiple effects, and some, like the chlorinated hydrocarbons, are highly persistent in the environment and in body tissues, as illustrated by a recent study linking elevated serum levels of the major DDT metabolite with breast cancer (1). We have accumulated experimental and human data showing that more than 390 chemicals are cancer causing (2), 150 are reproductive and developmental toxicants (2), and several hundred are neurotoxic (3). Although limited in scope, our national monitoring network routinely detects hundreds of toxic chemicals in the air, drinking water, and food supply. Some naturally occurring substances, such as the regulated dietary contaminant aflatoxin, may also pose significant health hazards. However, comparisons between the risks of natural carcinogens in the diet versus the risks of synthetic pesticides are seriously limited by the inadequacy of data on the identity, concentrations and carcinogenic potency of both types of chemicals in the food supply.

Many researchers have shown that people vary widely in their susceptibility to toxicants. For example, we have recently reported a 70-fold variation in the levels of genetic damage in people similarly exposed to air pollution in an industrialized region of Eastern Europe (4). This finding is consistent with results from parallel studies of workers and cigarette smokers. Recognizing the importance of interindividual variability in susceptibility, a committee of the National Academy of Sciences recommended changes in the regulatory practices for pesticides in order to protect the health of children (5).

In some cases, pollution has resulted in disease epidemics which can't be missed: for example, it is estimated that asbestos exposure will cause several hundred thousand deaths in American workers by the year 2000 and that thousands of cases of neurologic disease have resulted from exposure to organic mercury, tri-orthocresyl phosphate, and Kepone. Recent studies indicate that as many as 60,000 deaths may occur each year in the United States as a result of fine particulate air pollution. There is growing evidence that environmental agents play a significant role in breast cancer causation (6). However, absent a rare "signal" effect, it has been extraordinarily difficult to establish a direct link between a particular environmental exposure and cases of chronic disease, especially those with long latencies such as cancer. Epidemiologic studies of human populations are of limited usefulness in prevention because they give answers only "after the fact" of illness or death.

Therefore, a decision was made decades ago by the nation's public health experts to screen chemicals using short-term and whole-animal test systems as a surrogate for humans. Under the established policy, the test results are then combined with all other relevant data as a basis for regulatory decisions in the absence of human data. Risks that are determined to be significant are then subject to regulation.

Are current toxicologic testing methods fatally flawed as some of the critics would have us believe? Unquestionably, the methodology that has drawn the most criticism has been the use of the animal bioassay to screen for potential human carcinogens. Epidemiologic studies have so far identified 50 human carcinogens, all of which, when properly tested, have been positive in animal bioassays. The majority were first identified in humans; even in the few cases where earlier animal tests had been positive, regulations were generally imposed only after the human cases appeared. Mindful of this experience, in the late 1970s Congress and regulators put in place the National Toxicology Program to include studies in which rodents are treated with test chemicals at and below the maximum tolerated dose in order to be able to detect effects within the animals' two- to three-year lifetimes.

This practice has drawn increasing fire from critics because of the possibility that the rodent tumors might result from high dose toxicity alone and could mislead about potential low-level effects in humans. While this is a serious concern and merits consideration, the record is reassuring in that a recent analysis of results from the National Toxicology Program indicates that the majority of carcinogens evaluated induce tumors at concentrations below the maximum dose given and that toxicity and carcinogenicity are not consistently correlated (7). A National Research Committee majority report recently reaffirmed the validity of this approach for identifying potential risks to humans (8). This does not mean that new, more informative, faster, or less costly testing procedures should not be developed. Indeed, important research is going on now at

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the National Institute of Environmental Health Sciences, academia, and elsewhere to develop and fine-tune testing and assessment procedures that incorporate recent advances in molecular biology. But there is broad agreement in the scientific and public health community that thorough toxicologic testing is central to disease prevention.

What successes can we claim? There have been important gains. We have prevented and are continuing to prevent millions of cases of environmental disease including lung cancer and mesothelioma from asbestos, damage to the developing nervous system of children from lead, liver cancer from vinyl chloride, and male sterility from dibromochloropropane, to name a few. For example, it is estimated that during the 7 years after the 1985 regulation of lead in gasoline, an estimated 10 million children were protected from the neurodevelopmental effects of lead. The vast majority of regulations have considered the costs of control as well as technological feasibility.

True, regulation is always undertaken in the face of considerable uncertainty concerning the health risks and the costs of regulation. Despite their apparent precision, quantitative estimates of the numbers of illnesses and deaths caused by toxic substances and the monetary estimates of costs and benefits of regulations are really only "guesstimates" derived using assumptions where data are missing. Even a herculean research effort will not resolve all of the unanswered questions. Recognizing this, President Clinton recently pledged to restrict emissions of greenhouse gases to reduce the serious threat of global warming. The same paradigm should apply to the risks of toxic substances.

The irony is that although environmentally related diseases are by definition preventable, they continue to impose major economic and human costs on society. They contribute to the spiralling costs of health care, which are expected to reach \$940 billion this year. For example, the health care costs of cancer now exceed \$100 billion dollars annually, or more than one-tenth of the nation's health care bill (9). The majority of cancer is believed to be preventable: a result of environmental factors such as cigarette smoke,

industrial pollutants, radiation, and diet. Even the most conservative estimates attribute 5–10% of cancer to pollutants in the workplace and ambient pollution, corresponding to 50,000–100,000 new cases of cancer each year in the United States, with their attendant health care costs. Taken together with the experience of the last three decades, these considerations support a strengthened and more cost-effective environmental policy centered on prevention.

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