If environmental hazards are to be regulated, why not regulate them consistently?

Regulating Environmental Hazards

By Richard Wilson

People seek answers to seemingly simple questions: Is the water safe to drink? Is there a safe level of radiation? But usually there is *not* a simple answer to such questions. For example, what is the safe speed of a car? A car travelling at only 1 mile an hour can kill a person by, say, pinning him to a wall. So we have to say that no driving speed—except, perhaps, zero—is absolutely safe. But in practice we do not strive for absolutely safe auto travel; we are willing to sacrifice some degree of safety for such other benefits as faster travel. Therefore, we usually think of a "safe" speed in such "practical" terms as 30 miles an hour on a residential street and 65 miles an hour on an interstate highway.

There seems to be a general reluctance to discuss "practical" safe levels for environmental hazards. The resulting debate about the regulation of environmental hazards is therefore unnecessarily acrimonious and inconsistent. Industrial chemicals that are toxic or suspected to be toxic garner more attention that toxic chemicals from natural sources; radiation from nuclear power plants garners more attention than radiation from natural sources. But a hazard is a hazard, regardless of its type. To the extent that the public demands regulation of environmental hazards, the regulation ought to be consistent with the regulation of other hazards and based on scientific evidence.

I begin with a new way of thinking about the exposure of populations to environmental hazards, then describe the relationship between exposure to such hazards and their effects on health. That leads me to an outline of alternative standards for limiting exposure to hazards. I conclude by suggesting a standard that could do much to make environmental regulation less controversial and more effective.

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A NEW WAY OF THINKING

The physician Paracelsus said several hundred years ago that "the dose makes the poison." Arsenic, for example, can kill promptly if given in large doses, but it does not kill at low doses. And low doses may, in some cases, be necessary for life. Thus, we usually think of a "safe dose" as an amount that will not cause a *prompt* death.

In the last 100 years we have become aware of many substances that can cause disease or death through constant exposure, even though they usually do not immediately cause disease or death. As life expectancy has improved markedly, and many major risks to life have been (almost) eliminated, we have come to pay more attention to smaller and smaller risks and risks that materialize late in life. We have been focused for a long time on cancers, first those attributed to radiation and then those attributed to chemicals and other substances. More recently, we have become concerned about air pollutants that cause lung problems.

Cancers, lung problems, and other diseases that are caused by environmental hazards are *chronic* effects. Whereas no member of a group will die from prompt effects if the group is fed less than a lethal dose of arsenic, some members of a group (or population) may suffer chronic effects if the group is constantly exposed to certain environmental hazards. Causality is often hard to establish in such cases because the effect of the exposure often is delayed for years and decades.

All we can do is estimate the probability of a chronic effect in a population, then decide whether and how to reduce that probability to an "acceptable" level.

Thus, just as there is no perfectly safe speed for a car, we cannot take it for granted that there is a "safe" threshold of exposure to environmental hazards. By the same token, it is meaningless to strive for "no risk" or perfect safety when it comes to environmental hazards. All we can do is estimate the probability of a chronic effect in a population, then decide whether and how to reduce that probability to an "acceptable" level.

THE RELATIONSHIP BETWEEN EXPOSURE AND EFFECT

HOW DOES THE PROBABILITY OF AN ADVERSE EFFECT change with the length and level of exposure to an environmental hazard? Here, we must distinguish between cellular and population effects.

Cellular Effect A biologist might ask about the probability that a substance will cause a cellular change that starts a chain of events that leads to a cancer, then ask how that probability varies with dose. In the 1920s Geoffrey Crowther suggested that the probability of initiating a cancer by exposure to radiation might be linear with dose. The idea of a linear, cellular dose-response relationship has since been extended to chemicals and other substances.

But even though the response of cells to exposure might be linear with dose, many biologists insist that the affected cells normally are repaired and that the repair mechanism fails only above a threshold of exposure to an agent. According to this idea, for every case of cancer in an individual, an agent must affect millions or billions of cells.

Population Effect A population consists of healthy and sick people and young and old people, and each has a different exposure threshold. Thus, there can be a linear population-dose response to a chronic exposure, even though the exposure has no apparent effect on many members of the population because each individual has a threshold. We know that 30 percent of all people in the United States develop cancer in their lifetimes, mostly from natural causes, but it is not now possible—and, in theory, may be inherently impossible—to distinguish a cancer caused by an environmental pollutant from one caused naturally.

These observations led Crump, Hoel, Langley, and Peto to point out that millions or billions of cells have already been affected by natural causes and that a pollutant's effects on few extra cells can lead to a linear population-dose response. Crawford and Wilson pointed out that the argument is not unique to cancer and may well apply, for example, to lung problems caused by air pollution. These conclusions flow from the knowledge that a disease is already occurring in a population and the assumption that some members of the population are on the verge of developing the disease. Any exposure to an agent that causes the disease might then cause disease in a person who had been on the verge of developing it. Although linear with dose, the increase in disease rate from such an exposure could be very small and undetectable against the "background" level of the disease. Crawford and Wilson argue therefore that a linear dose response may be more common than usually assumed.

The concept of "collective dose" has been widely used to characterize the exposure of a population to radiation. The collective dose is the summed product of the dose and the number of persons exposed to that dose. If a linear dose response is assumed, the number of cancers produced by a pollutant is proportional to the collective dose no matter how that dose is distributed across a population.

But it must not be assumed that a linear relationship between exposure and adverse effect is unique to environmental exposures. On the contrary, Crawford and Wilson's argument merely brings environmental exposures to a par with other exposures in life. However hard we struggle to improve the safety of automobiles, for example, the probability of being killed by one increases with repeated exposure to the risk of being a driver, passenger, or pedestrian. This suggests to me that we should use the same practical methods of regulating environmental pollutants as we do for automobiles and other societal hazards.

EXPOSURE STANDARDS

GIVEN THE ELUSIVENESS OF ABSOLUTE SAFETY AND THE absence of a threshold below which an environmental hazard poses no risk to a population, on what basis can society set exposure standards?

Effective Threshold A possible standard is the "effective threshold"—the level below which it is not possible to detect an adverse effect in an epidemiological study. In

other words, if the background rate of a disease is 100 cases per million people per year, statisticians can estimate how many additional cases would have to occur each year to result in a statistically significant increase in the disease rate. If the predicted effect of an exposure were less than that critical figure, the exposure would then be acceptable. In practice, it is not possible to detect a lifetime risk of less than 1.5 percent, that is, an exposure that would kill 1.5 percent of the exposed group over a lifetime of 75 years.

Observations from workers or laboratory animals exposed to high levels of chemicals or radiation allow scientists to extrapolate downward to predict risks at lower, environmental exposures. Exposures associated with risks below 1.5 percent can therefore be accepted as practical exposure thresholds. The Health Physics Society and the American Nuclear Society, for example, have suggested a threshold for lifetime exposure to radiation of between 10 rems (roentgens of absorbed radiation) and 20 rems, which corresponds to a lifetime risk of 0.5 percent to 1 percent.

Table 1

Occupational Risks: Deaths per Year of Risky Activity[1]

Historically calculated risks[2]	
U.S. president	1 in 52
Metal miner	1 in 3,000
Policeman	1 in 3,000
Transportation worker	1 in 5,000
Quarry worker	1 in 5,000
Airline pilot[3]	1 in 10,000
Government office worker	1 in 11,000
Professor (frequently flying)	1 in 20,000
Risks involving uncertain dose response[4]	
Coal miner with black lung disease	1 in 200
Asbestos worker[5]	1 in 4,000
Airline pilot (cosmic ray exposure)	1 in 5,000
Hospital x-ray technician	1 in 10,000
Benzene worker	1 in 30,000

[1]Muliply by years of work to obtain lifetime risk. [2]Uncertainty about a factor of 2. [3]Risk of accident. [4]Uncertainty of slope about a factor of 3, plus the uncertainty of extrapolation.
[5]At threshold limit value of exposure one-fourth of the time.

Table 2

Some Commonplace Public Risks: Action or Type of Exposure and Lifetime Risk

Historically calculated risks[1]	
All cancers	1 in 4
Cigarette smoking	1 in 3
Motor vehicle accident (motorist or passenger)	1 in 80
Motor vehicle accident (pedestrian)	1 in 400
Home accidents	1 in 120
Electrocution	1 in 3,000
Being hit by meteorite	1 in 25,000
Being hit by falling aircraft	1 in 200,000
Risks involving uncertain dose-response relationship[2]	
Air pollution (eastern United States)	1 in 50
Drinking water with EPA limit of chloroform	1 in 50,000
Drinking water with EPA limit of arsenic	1 in 100
School with U.S. average of asbestos	less than 1 in a million

[1]Uncertainty about a factor of 2. [2]Uncertainty a factor of 3 or more, plus the uncertainty of extrapolation.

Negligible Risk Some scientists and members of the public prefer to be even more cautious, reasoning that a practical threshold of 1 percent may be taken as an invitation to increase pollution from where it may now be to a higher level, as long as it remains below the threshold. That concern has led to an emphasis on "negligible risk." But what is "negligible" and how can one detect such a low risk?

Regulatory agencies have established a "virtually safe dose" (VSD) for carcinogens, which corresponds to 1 additional cancer in a million people exposed to that dose every day for their lifetimes. Most people accept VSD as a negligible risk. But can society consistently regulate to meet such a stringent negligible risk criterion? As Tables 1 and 2 suggest, individuals and the public at large routinely accept much greater risks than 1 in a million. I argue therefore that regulatory attempts to meet a stringent negligible risk criterion are arbitrary and capricious.

Cost per Life Saved None of the standards I have discussed thus far addresses the cost of reducing the level of exposure. The U.S. Nuclear Regulatory Commission (NRC) was probably the first government agency to address the issue of cost. It suggested that exposures to radiation should be reduced if that can be done at a cost of $1,000 per person-rem—about $2,000 per person-rem in today's dollars. That is about $4 million per cancer averted in the population, or what economists call a statistical life. (In keeping with the usual practice of accepting greater occupational than public risks, the independent National Council for Radiation Protection suggested that exposures for medical and dental personnel should be reduced if that can be done at a cost of $10 to $1,000 per person-rem.) Notably, in 1998 the U.S. Environmental Protection Agency (EPA) proposed draft guidelines that suggested the use of $4 million per statistical life in cost-benefit analyses of proposed regulations.

CONCLUSION

IF WE TAKE $4 MILLION PER STATISTICAL LIFE AS A STANDARD for regulatory action, some substances are overregulated and others are underregulated; for example:

- According to a study by the Harvard Center for Risk Analysis, NRC regulations for radioactive waste from a reactor mandate a cost of $800 million per statistical life saved.

- A recent article in *Health Physics* suggested that whereas the federal program to cap uranium mine tailings was justified at a cost about $500,000 per statistical life, extension of the program to more remote mines at a cost of a billion dollars per life saved was unjustified.

- Some specialists and I have argued that in the United States air pollution causes about 50,000 deaths a year. If we accept that a life saved is worth $4 million, the government should be spending about $200 billion annually to reduce exposure to pollutants in the air. Clearly, we are not spending that much.

The recent decision in the *ata* case, in which the U.S. Court of Appeals for the District of Columbia set aside EPA's standards for fine particles, underscores the need for Congress to establish a consistent, scientifically based standard for the regulation of environmental hazards. The adoption of a standard, such as $4 million per life saved, would help to ensure that public moneys are used efficiently and not wasted in the pursuit of unattainable goals. Such a standard might also remove some of the emotion from debates about environmental regulation, allowing legislators and the public to focus on the real issues of risk and cost.

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