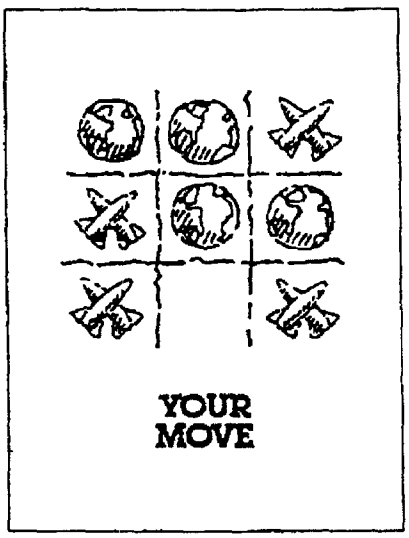
MAY/JUNE 1986 VOL 89 NO 4

EDITED AT THE MASSACHUSETTS INSTITUTE OF TECHNOLOGY

TechnologyReview



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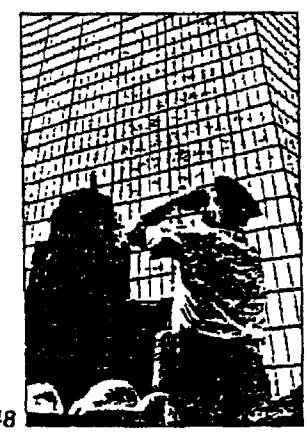

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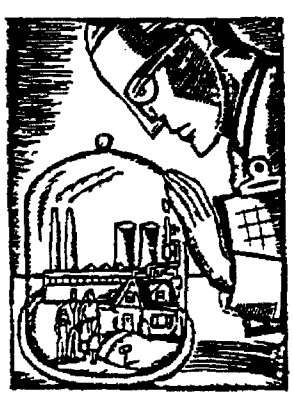

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COVER
Concept: Karen Craig
Design: Kathleen Sayre

Assessing Risks from Health Hazards: An Imperfect Science

BY DALE HATTIS AND DAVID KENNEDY

When William Ruckelshaus came back to run the embattled Environmental Protection Agency (EPA) in 1983, one of his primary goals was to separate the "science" of assessing health hazard risks from the "policy" of managing those risks. During the previous administration of Anne Burford Gorsuch, risk assessment had become so entangled with politics that many public observers felt that the EPA was acting as an advocate for the very industries it was supposed to regulate. For example, the EPA had concluded that there was no significant health risk to workers from exposure to formaldehyde, a chemical used to make particle board, plywood, and some permanent-press fabrics. The actual evidence on the health risks from formaldehyde was less reassuring than the EPA's position indicated.

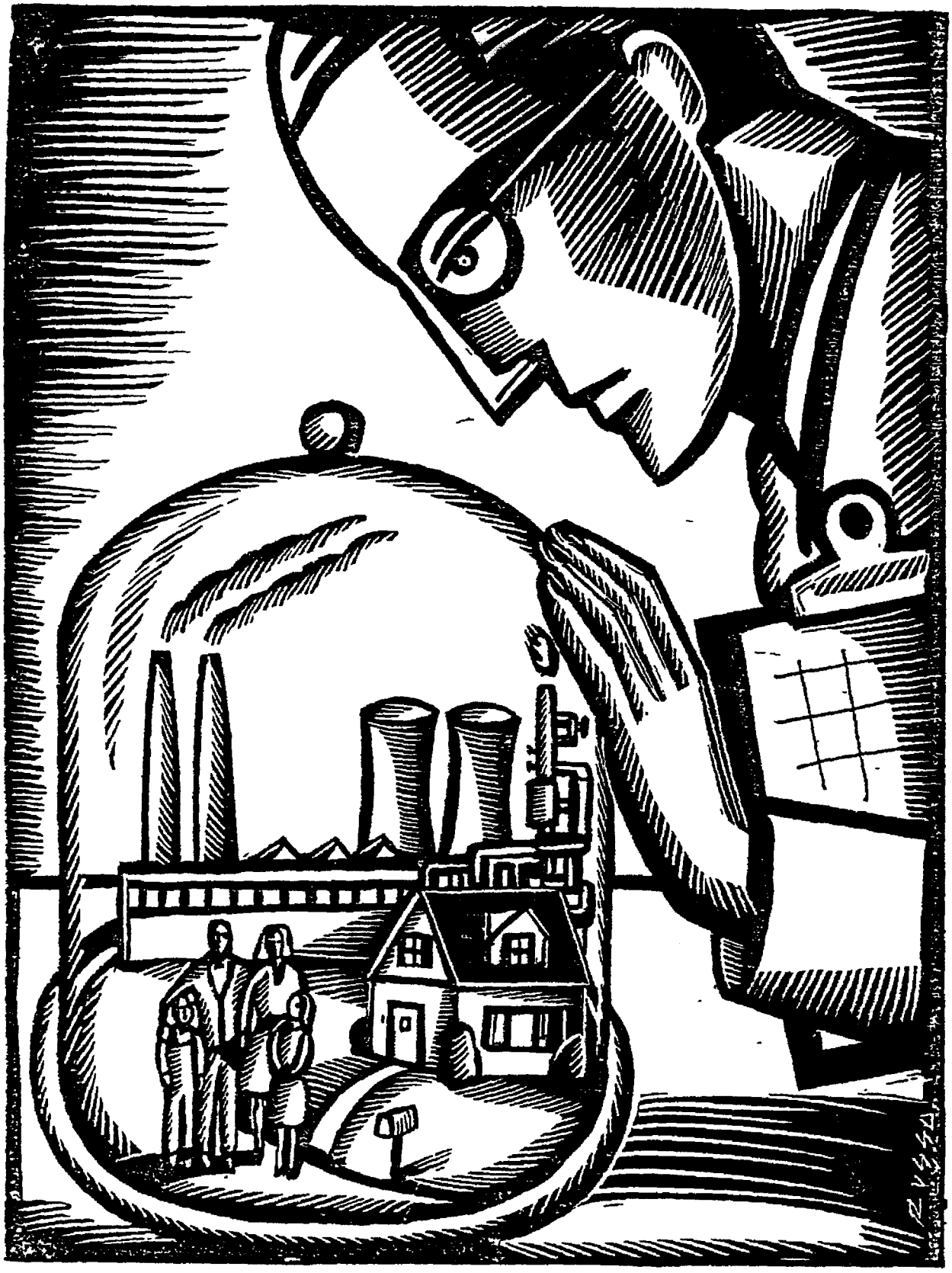
In an effort to make risk assessment more impartial, Ruckelshaus argued that scientists should first make an "objective" study of the extent of risk from exposure to a particular hazardous chemical or situation. How many people, for example, will die from cancer after 20 years of exposure to airborne arsenic emitted from a copper smelter in Tacoma, Wash.? Only after that assessment is made should governmental agencies move into the political realm and decide what to do about that risk.

Moreover, Ruckelshaus thought that his agency would better serve the public by being more explicit about the extent of risk from various environmental hazards. Potent carcinogens such as dioxin are very frightening, but just how dangerous is a very low concentration of the chemical? Should the EPA take great pains to reduce dioxin in waste sites to very low levels, or might the money spent on such cleanups save more lives if channeled toward controlling other kinds of pollution? Ruckelshaus believed that risk assessments comparing the threat from different hazards would help him, and the public, resolve such issues.

Ruckelshaus' intent was to create a special authority and credibility for risk assessment. He wanted to build a strong scientific foun-

Quantifying people's risks is a process rife with uncertainty. Analysts should make public those uncertainties so we can make more informed decisions about controlling hazards.

ILLUSTRATIONS ANTHONY RUSSO



The science behind risk assessment is simply not up to the challenge of consistently providing accurate answers.

dation upon which EPA and society at large could balance social, economic, and political concerns and reach sensible decisions about managing environmental risks.

Other policymakers shared his views. The National Research Council recommended in 1983 that regulatory agencies take steps to make the process of assessing risks more formal and scientific, and to maintain a clear conceptual distinction between risk assessment and risk management. Industry groups such as the American Industrial Health Council also insisted that "the scientific determination should be made separately from the regulatory determinations." Such industry groups have often suggested that environmental controversies be resolved by experts capable of critically evaluating specific facts.

There is only one problem with this call for authoritative, scientific risk assessment: such a commodity does not exist. In classical times, there was a great demand for the skills of soothsayers in reading entrails, and there is a similar amount of wishful thinking going on today. The fact is that the science behind risk assessment is not up to the challenge of consistently providing accurate answers about the degree of risk individuals or populations face from health hazards.

Scientific uncertainties remain in the process of assessing risks—uncertainties that some people find devastating. Vernon Houk, a senior official of the Centers for Disease Control (CDC), has said that the difference between risk assessment and a five-year weather forecast is that at least with the weather forecast, if you wait five years you find out whether you were right. Many risk assessments project numbers of cancers or deaths that, while large enough to arouse public concern, are too small to be definitively separated from those occurring normally in a given society. Furthermore, one usually has to wait decades—the length of time cancer often takes to emerge—before an unusually high incidence of disease can be confirmed.

There is also no way risk analysts at the EPA or other agencies can escape making value-laden choices in the course of their work—choices that render their results far less "scientific" and objective than Ruckelshaus envisioned. Even apparently neutral reports of what is known and not known generally reflect value-based assumptions about what matters and what does not.

Consider, for example, two recent analyses of daminozide, a chemical that regulates growth (apple growers use it to control the time when their crops are ready). At a hearing in Massachusetts on daminozide, Ian Nisbet, a consultant for the Massachusetts Department of Public Health, presented an analysis of the special risks the substance might pose to infants and young children. Young children generally eat more food relative to their body weight than adults, and they also seem to eat more processed apple products such as applesauce. Furthermore, there is good reason to expect that rapidly growing children with most of their lives ahead of them are more likely to develop cancer than adults. However, the very act of placing the daminozide analysis in this framework makes a value-laden statement: that the special sensitivity of young people should be considered in the public-policy process. By contrast, one of the authors of this article presented an analysis of daminozide's risk that made no special distinction regarding children. This analysis did include a broad range of possible hazards based on risks found in other chemicals in the same family of hydrazines. Both analyses were factual and "objective," but they clearly were not value neutral.

Since risk assessment cannot be wholly insulated from value judgments and is rife with uncertainty, it lacks the special credibility that some would claim for it. Therefore, agencies such as the EPA, the Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA) should not expect their risk analysts to come up with "bottom-line" answers to questions about whether use of a hazardous chemical or process should be banned or encouraged. Instead, these agencies should encourage their analysts to share the uncertainties involved in assessing a risk with policymakers and the public, who can then make more informed—albeit more complicated—decisions about regulating the risk.

Linking Cause and Effect: A Difficult Task

Risk assessment is a relatively new discipline, if it can be called a discipline at all, and there is no con-

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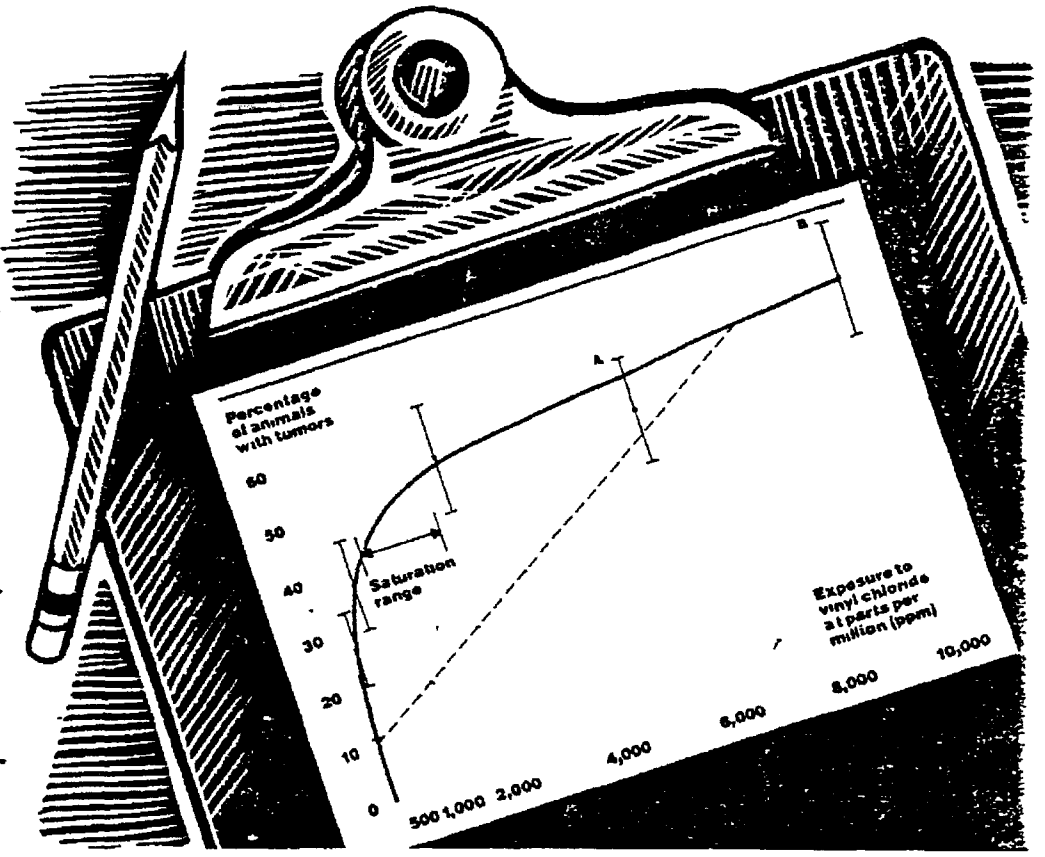
sensus on which basic rules and procedures to apply in solving particular problems. Although government analysts have been assessing risks of various kinds for decades, the call for quantitative risk assessment—i.e., expressing the extent of risk in numerical form—is relatively recent. Until the 1970s, risk was perceived as a simpler, more black-and-white problem: is or is not DDT carcinogenic and mutagenic? The answer is an unequivocal yes. However, data on the risks of many other toxic substances are more ambivalent, and the risk to people remains unclear. In such cases, policymakers are more likely to take into account the economic consequences of restricting the chemical in question. More sophisticated detection technologies also allow scientists to measure the effect of these substances in smaller and smaller amounts, producing results that are not as clearcut as previous tests were.

Evaluating an environmental hazard to see how many people might be at risk is difficult because analysts must follow that hazard through whatever twists and turns it takes in the real world. Analysts have to determine how potential threats are released into, and move through, the environment. They have to figure out how much of the substance people might eat, breathe, or otherwise take up, and then estimate how much of it they would absorb. Finally, analysts must determine just how much of a hazard the absorbed level of the substance poses.

Figuring out how much of a toxic chemical reaches people—the degree of exposure—can be quite challenging. For example, analysts generally use computer models to assess how a toxic plume of different-sized particles of arsenic disperses through the air from a smelting plant, or how a chemical would be carried through the ground to wells supplying water. Yet it is difficult to incorporate all the important information into these models. In most cases groundwater flows more readily in horizontal directions (out from a waste site) than vertically. But measurements of the way water flows in different directions may not be available, making it hard to predict when wells at different depths and distances from the waste site might be affected. Even when data of this sort are available, the models themselves often oversimplify the system in question. One EPA model of smelter emissions into air assumed that the smelting plant was on a flat plain when in fact it was on a steep hill. As a result, EPA scientists misstated the wind patterns and initially overestimated the concentrations of arsenic that would affect the nearby town.

Even if analysts know how much of a substance is in the environment, they can't necessarily predict how much people will actually absorb. People breathe at different rates depending on their level of activity: workers laboring heavily at a construction site, garment workers sitting at sewing machines, and people sleeping in the surrounding community will all receive different doses of an airborne contaminant. Individuals also have widely different breathing rates and dietary habits, profoundly affecting the doses of specific substances they receive from air and food. Finally, people absorb substances in varying amounts depending on the thickness of their skins and the properties of their nasal mucous, and even on whether they tend to breathe through their noses or their mouths.

Determining how much of a hazard the absorbed substance poses is another complicated problem. For example, evidence of cancer or other toxic effects in humans attributable to a specific cause would seem to constitute the ideal basis for regulation. A high incidence of lead poisoning in people living near lead smelters would seem to indicate the need for better pollution controls on those plants. Unfortunately, such clear-cut instances are rare, because epidemiological studies are notoriously insensitive in de-



Extrapolating the results of animal studies to humans can be complicated. Animals are exposed to large amounts of chemicals so that toxic effects will appear at significant levels. However, at these doses, the animal enzymes that convert the chemicals to cancer-causing substances become saturated and cannot make any more toxic by-products. This effect occurred when rats were exposed to increasing doses of vinyl chloride. The curve showing how many animals developed tumors leveled off shortly after the saturation point.

Risk assessors usually apply only the data from the two highest dose points (A and B) to weigh human risk. If they had done so in this case, the curve would have been a straighter, more gradual line, greatly underestimating the human risk at low doses.

tecting health effects from relatively low levels of exposure. As David Ozonoff, chief of the Environmental Health Section at the Boston University School of Public Health, has said, "A good working definition of a catastrophe is an effect so large that even an epidemiological study can detect it."

The problem is that the rates of specific illnesses from a given hazard often must be several times above average before one can conclude that they aren't simply random fluctuations. In one celebrated case now being tried, a group of parents from Woburn, Mass., is suing two chemical companies for dumping toxic wastes in the neighborhood. The parents claim that these wastes leaked into local drinking water and caused an unusually high incidence of leukemia among their children. This high incidence, which has already resulted in the deaths of five children, could indeed be due to the companies' toxic-waste dumping, or it could be a random fluctration. It could also be the result of a completely different and unknown phenomenon.

Another complication is the fact that unless scientists perform special monitoring measures at the time of exposure, there is rarely good information about how heavily certain populations have been exposed to a chemical. Nor is information about "confounding" factors such as smoking, alcohol use, and the toxic's interactions with other environmental hazards readily available. These difficulties do not always render epidemiological information useless: solid positive results can provide a good indication of a specific level of risk from a given substance. For example, studies among workers exposed to arsenic, and among residents exposed to arsenic compounds in well water, have unequivocally revealed the carcinogenic properties of inorganic arsenic. However, a negative result is usually not proof that there is no risk, but rather an indication that the risk, if any, is less than the study is capable of detecting.

The Trouble with Animal Studies

In the great majority of cases where the epidemiological evidence is incomplete or ambiguous, using animal studies to make projections may make more sense. However, such studies suffer from their own serious uncertainties. Experimental animals are generally exposed to high concentrations of chemicals to ensure that if there are any toxic effects, they will appear at statistically significant levels. A mathematical model called a "dose-response curve" has to be fitted to the resulting data to assess the probability that people will get cancer at the much lower levels they might realistically encounter. However, such high doses can complicate the interpretation of results in a number of different ways.

For example, molecular biologists have discovered the existence of certain enzymes in cells that convert chemicals to more toxic metabolites, beginning the march toward cancer. At very high doses of the toxic

ILLUSTRATION ANTHONY RUSSO, DIAGRAM OMNIGRAPHICS

Epidemiological studies are notoriously insensitive in detecting health effects from relatively low levels of exposure.

substance, these enzymes are fully occupied and cannot generate toxic by-products at a greater rate. Thus, increased doses do not necessarily lead to more cancers. In extrapolating downward to realistic levels of human exposure, risk assessors often don't take this saturation effect into account and, in effect, underestimate the risk to humans.

Ideally, the dose-response relationship would be derived from a detailed theory about how the chemical actually works to produce cancers in animals and in humans. Such a theory would be based on knowledge about how the chemical is absorbed, metabolized, and excreted from animal and human systems. Risk assessors do not generally attempt to draw together much of the latest knowledge available, and as a result their models do not capture the details of cause-and-effect relationships. Instead, these analysts generally fit one of a number of statistical formulas to the data on tumor formation.

Unfortunately, the different mathematical models produce widely varying results. "Multistage" models, favored by molecular biologists, are based on the belief that cancer occurs as the end result of a series of genetic changes in specific cells. If a toxic chemical interferes with the copying of the genetic information in a cell's DNA, the errors, or mutations, will usually be passed on to the descendent "daughter" cells. Even if the error is confined to a single nucleotide base on the DNA code, the results can be severe if important genetic information is altered.

For example, in some cases a cancer-causing gene known as an oncogene becomes active because of a single mutation: one specific nucleotide is replaced with another. That one change instructs the gene to produce a specific amino acid instead of another. Chains of amino acids link together to form proteins, the basic molecules that help cells function. In this case, the single change in this amino acid produces a protein that begins the process of producing cancer, although researchers do not yet know how. The crucial implication of this model is that even the tiniest amount of a toxic substance that can affect DNA has some chance of inducing cancer. In principle, there is no exposure threshold below which the toxic substance does not pose a risk.

In contrast, "probit" models imply that individual organisms do in fact have a specific tolerance level, or threshold, below which exposure to a toxic chemical is safe. This type of model is traditionally favored by pharmacologists and toxicologists, who view biological processes as complex webs of processes, exquisitely balanced so that modest perturbations in the system will prompt corrective actions to restore normal functioning. As long as the biological insult the system suffers is not too great—i.e., is below a certain threshold—the system ought to be able to repair any damage that may be temporarily produced. The implication is that there is no benefit from regulating toxic substances when exposure falls below such a threshold.

Many scientists believe that this type of model is perfectly appropriate when applied to traditional types of acute toxic insult, such as the lung damage from methyl isocynate that occurred on such a devastating scale in Bhopal. However, some scientists find this model more questionable for carcinogens that seem to act directly or indirectly on DNA. Furthermore, given the difficulties of extrapolating human exposure from high-dose animal experiments, there is rarely, if ever, any way to specify actual thresholds for people.

Despite these reservations, risk analysts continue to use very different models depending upon their professional ideology. And the results continue to differ dramatically. In a recent experiment, Alice Whitemore, an epidemiologist at Stanford University School of Medicine, fit different models to experimental data for male rats exposed to the carcinogenic pesticide ethylene dibromide (EDB). Widely used to fumigate grain, EDB has recently been found in bread, cereal, cake mixes, and the like. Whitemore found that depending on the model chosen, the likelihood that an individual will get cancer from low-level exposure to EDB can differ by a factor of one million.

Given these enormous discrepancies and what is at stake for industry, workers, and the general public, there is considerable controversy as to which models are most appropriate for assessing risks. Analysts at EPA and OSHA tend to use the more conservative multistage model, favored by molecular biologists, or a similar model. For instance, OSHA used this model in a recent assessment of the risk of workers exposed to EDB. This assessment, which was based on extrapolation from an experiment with rodents, showed that the risk is very high indeed: after 45 years of intermittent exposure to 20 parts per million of EDB in the air—the maximum amount then permitted under OSHA standards—the chance that a worker would develop one common form of

Risk analysts should try to eliminate, or at least disclose, biases in their thinking.

cancer was pegged at between 38 and 59 percent. However, very few workers were actually exposed at this level or for this duration; the calculation was done to show that there would be a "significant risk" to workers if they were exposed over a working lifetime to the permitted levels. OSHA, however, has not yet reduced its maximum level of EDB exposure in the workplace.

When results from the multistage model lead an agency to ban or severely restrict the use of a chemical, fireworks often result. For instance, the EPA's recent ban on the use of EDB as a pesticide prompted a vociferous protest from companies that manufacture the chemical. However, the ban has stood.

Other serious difficulties plague the process of interpreting animal studies and extrapolating them to humans. Because animals and humans metabolize substances differently, the level of the test chemical that reaches various parts of the animal and the human body can vary widely. Hence, animals and humans may suffer from different health effects. For example, bis-chlormethyl ether, used as a laboratory chemical, tends to produce nasal tumors in rats but lung tumors in people. The contrast may stem from differences in how deeply the chemical penetrates into the respiratory system before being absorbed.

Moreover, the metabolite or by-product of the test chemical, rather than the chemical itself, is often the toxic substance, and animal systems can differ from human ones in the type and concentration of metabolites they produce. For example, dogs and people primarily succumb to bladder cancer from some aromatic amines such as benzidine (used in the manufacture of dyes), while rodents get cancer of the liver. This is apparently because the different mammalian systems form metabolites that react at different sites in the body. As the National Research Council, which often advises the federal government on scientific issues, points out in a recent congressionally commissioned report, correcting for these differences is not easy because researchers often lack enough information about human and animal systems.

Disclosing Bias in Risk Assessment

It would be easy, but mistaken, to look at this litany of problems as a wholesale indictment of the entire concept of risk assessment. William Ruckelshaus himself came close to drawing such a conclusion in 1985 after leaving the EPA. He wrote that risk analysis "is a kind of pretense; to avoid paralysis of protective action that would result from waiting for 'definitive' data, we assume that we have greater knowledge than scientists actually possess and make decisions based on those assumptions."

Although now more aware of its limitations, Ruckelshaus still believes in risk assessment, and Lee Thomas, the current EPA administrator, is pursuing his predecessor's vision of higher-quality risk assessments. And properly so, since some form of risk assessment is essential in dealing with environmental hazards. There is real value in encouraging a conceptual and professional separation between risk analysts and risk managers. Such a separation will never produce the kind of ironclad and unimpeachable scientific analyses some would like. However, it will reinforce the preeminent duty of analysts to eliminate inappropriate biases from their thinking and work.

The sort of thing to avoid is the EPA's handling of formaldehyde under Gorsuch. In 1981 the agency in effect took an advocacy position to support a decision that regulation was not necessary. Its assessment suggested that there was no significant risk because its analysts lacked epidemiological evidence that the substance is carcinogenic. The analysts relied instead on arguments about thresholds to conclude that low-level exposure is safe. Most risk assessors know how slippery epidemiological data are, and they are familiar with the vigorous debate regarding the viability of thresholds for carcinogenicity. The EPA ignored such ambiguities. Norton Nelson, a prominent environmental scientist, said that the EPA took "an extreme position" in deciding that the data on health risks of formaldehyde were not very significant.

Risk analysts should be encouraged to cultivate an approach—an ideology, if you will—that provides the public with exactly the opposite of extreme, advocacy positions. Since risk assessors often cannot answer the question "exactly how much risk does some hazard pose?" they must tackle the question they can answer: how much do we know about a particular hazard, and what are the important uncertainties in that picture?

The objective of this approach would be to help policymakers and the public make informed choices based upon the available information. Risk assessors of this stamp will have to be particularly open and

sensitive in choosing which methods of analysis are appropriate in each situation. They will also need to draw on insights from various disciplines. Take, for example, the original studies assessing the risk of workers exposed to airborne lead. Since workers exposed to less lead didn't have correspondingly reduced blood lead levels, some scientists originally suggested that much of the lead found in workers' blood must have been introduced by some mechanism other than breathing. However, pharmacologists know that lead is stored in bone when blood is saturated, and can be released into the blood if blood lead levels fall. The original scientists didn't take that important fact into account and came up with the wrong assumption that workers were somehow eating the lead.

Risk assessors should be intimately familiar with such effects and be able to apply the insights and techniques of different specialties where appropriate. Unfortunately, risk asessment practices today tend to prevent such interdisciplinary familiarity. Many government risk assessors are not trained in toxicology, biochemistry, or other disciplines. Thus, they often do not have the background to understand and include in their assessments the detailed processes that produce disease. Instead, risk analysts usually review the research of other specialists and use statistical methods to draw quantitative conclusions.

Specialists from different disciplines such as pathology, toxicology, and chemistry often form teams to perform risk assessments, which is a step in the right direction. But there's still a tendency to maintain strong disciplinary boundaries on such teams—to separate, for example, the analysis of the dose people are exposed to from an analysis of how that dose correlates with actual health effects.

Take exposure to formaldehyde. The substance can affect respiratory tissue in a number of ways. The formaldehyde can itself react with DNA and begin the process that ultimately can lead to cancer. It can also inhibit the enzymes responsible for repairing DNA. Finally, at high doses, it can kill cells and thereby stimulate cell replication to replace those that are lost. Enhanced cell replication reduces the time available for repair of DNA lesions, increasing the chance that permanent genetic changes will occur in the exposed cells. Given these multiple effects, there is some reason to suspect that short-term exposure to high doses of formaldehyde might cause more damage than longer term exposure to the same total amount. Hence, OSHA and EPA should express their results in terms of the amount of time people spend at specific exposure levels. Unfortunately, these agencies now express exposure levels as an average over time.

Some of this tunnel vision can be blamed on the narrow and often uncoordinated focus of the risk assessors. But the agencies themselves must take the blame for not acting quickly enough on important new information; pressure from agencies such as the White House Office of Management and Budget, and the potential for lawsuits from industry and citizen groups are two reasons for such lethargy. Some officials are reluctant to try innovative approaches to risk assessment because they may be more likely to be shot down in the tortuous process leading to a regulatory decision.

Risk assessors also must do a better job of deciding what the scope of their analyses ought to be. For example, they have to decide whether to include the possibility that a toxic chemical may interact dangerously with other chemicals in the environment, even though there are no hard data available on that interaction and its result. Risk assessors also have to decide who to consider when analyzing a chemical's toxicity. Take the case of ozone, a pollutant formed when hydrocarbon fumes evaporate from automo-

An informed public will have to accept responsibility for making painful choices among competing goods.

ile exhausts, gasoline, nd paint solvents and eact with sunlight. hould analysts focus on ie risk to the majority elatively healthy people, r to the relatively few elerly and asthmatic peo-le who might be articularly susceptible o ozone smog?

Analysts must recognize that such choices are aden with value judgnents and make an effort o avoid or least state hose judgments. Yet all oo often, as John Holdren of the University of California at Berkeley has written, risk assessors tend to omit issues that they have decided are too uninteresting, difficult to quantify, speculative, or likely to be "misinterpreted." To prevent this kind of preselection, we should ensure that analysts publicly disclose the choices they make.

A Cure for the "Bottom-Line" Illness

Finally, risk assessors must do a better job of identifying and assessing uncertainties and communicating them to policymakers and the public. Given the many difficulties with the science that risk assessment draws on, analysts should take care that it is never said of them, as it has been of the State Department: "They're never right, but they're always sure." The goal here should be, in the words of Nicholas Ashford, associate professor of technology and policy at M.I.T., to "bound the set of not clearly incorrect answers," rather than to focus solely on the most likely answer statistically.

Risk analysts should never present a "best estimate" of risk without some accompanying statement of statistical uncertainties and other ambiguities. Policymakers often suffer from "bottom-line illness"; all they want is the number at the end of the study. Risk assessors should carefully avoid that disease. Instead of one bottom-line estimate, they should present a range of likely estimates, including their different consequences.

We could formulate many other such prescriptions for analysts, but the basic principle is that they should communicate their findings so that policymakers and the public can fully understand the issues and uncertainties. Interested observers should be able to comprehend the important assumptions, data gaps, and choices almost as if they themselves had gone through the process. This is all the more important at a time when the U.S. has limited resources for making its environment "safe" from hazards. As Ruckelshaus said recently, we have to "abandon the impossible goal of perfect security and accept the responsibility for making difficult and painful choices among competing goods."

In a more paternalistic society, an elite group of leaders might weigh competing costs and benefits and set safety limits for the rest of us according to the elite's view of what is best. But we live in a democracy and have a right to participate in the dialogue concerning which risks should be controlled and to what degree. Moreover, we have to consider the fact that the cleanup itself is not generally risk free: workers can become injured and nearby residents can be exposed to air pollution from bulldozers cleaning up a site.

Risk assessment can help inform this important social and political dialogue. It can also help raise the quality of that dialogue, by revealing where science is—and is not—likely to be of service in resolving doubts about the nature and magnitude of environmental risks. □