

Precaution and the Lessons of History



Questions about risk, profit, and the burden of proof have troubled U.S. regulatory agencies ever since Harvey Washington Wiley called for a version of the precautionary principle in the early decades of the twentieth century. Since 1998 they have coalesced around the demand for a precautionary approach that would place the burden of proof on those who profit from toxic chemicals. That year, thirty-two scientists and physicians concerned about endocrine disruption published a consensus statement known as the Wingspread Statement on the Precautionary Principle. They wrote: “When an activity raises threats to the environment or human health, precautionary measures should be taken, even if some cause-and-effect relationships are not fully established scientifically. In this context, the proponent of an activity, rather than the public, should bear the burden of proof.”¹ Yet the precautionary principle is not easy to implement, for the environmental or health risks of a particular action are usually uncertain and occur in the future, while the costs of averting it are often immediate.

Precaution has a long history in public health. As Sonja Boehmer-Christiansen argues, a formal precautionary principle evolved out of the German concept of *Vorsorgeprinzip*, which developed in the legal tradition of the 1930s democratic socialism. *Vorsorgeprinzip* centered on the concept of good household management, a concept that justified state involvement in planning economic, technological, moral, and social initiatives.²

Precaution had been adopted well before this in public health efforts,

however. When the British physician John Snow recommended removing the handle from the Broad Street water pump in an attempt to stop London's 1854 cholera epidemic, that was a form of precaution. Scientists were still uncertain of the causes of cholera when Snow acted. He had found a correlation between polluted water and cholera five years earlier, but most scientists and physicians rejected his thesis as untenable, believing that airborne contaminants caused cholera. The biological mechanism underlying the link between polluted water and cholera was unknown. Yet even without firm proof, Snow had enough information to judge that the possible costs of inaction would probably be greater than the costs of action.³

Snow's vision of protecting the public through precautionary action continued as an important thread in public health. During the first decades of the twentieth century, Harvey Wiley and Walter Campbell argued that the federal government needed to use precaution as the basis of regulation. The Food and Drug Administration, they believed, needed to sift evidence from multiple perspectives, not just the industry standpoint, to find preliminary evidence that might suggest possible links between a compound and an adverse, potentially irreversible, outcome. This preliminary evidence might come from experiments on animals or from structural similarities between a given chemical with unknown effects and one with known effects. Precaution was justified, they believed, when the potential costs were high or irreversible compared to the benefits, when the person who bore the costs did not receive the benefits, and when preliminary evidence suggested a possible link between an action and a harm, even when the exact biological or chemical mechanisms underlying that link were still uncertain.⁴

Beginning in the 1970s, scientists and activists made efforts to extend the idea of precaution from the public health arena into broader environmental decision-making. In the 1970s, German foresters struggled to establish the causes of dying forests and developed a precautionary principle similar to that proposed by Wiley and Campbell. The 1985 report on the German Clean Air Act noted that a precautionary approach requires more than establishing the "level of proof needed to justify action to reduce hazards (the 'trigger' for action)." Other important elements include monitoring for early detection of hazards; promoting alternatives such as clean production and innovation in green chemistry and engineering; "the

proportionality principle, where the costs of actions to prevent hazards should not be disproportionate to the likely benefits”; and a commitment to take action “before full ‘proof’ of harm is available if impacts could be serious or irreversible.” The 1992 Rio Declaration on Environment and Development was explicitly grounded in precaution, and the principle has since become central to consumer and environmental protection policy in the European Union. In 2007 the European Union passed a law mandating that chemical companies must demonstrate that their products are safe before they can be placed on the market.⁵

Industry has generally opposed efforts to extend precaution from medical to environmental policy, fearful that such an approach could stall innovation. The Business Roundtable was founded in 1972 to represent two hundred of the nation’s largest corporations, and this association has taken an increasingly active role in opposing environmental regulation. Gerald Markowitz and David Rosner argue that the association’s strategy has been to accentuate elements of complexity and uncertainty and then to argue that “economic interests should not be challenged until science has proven danger. Precaution is equated with economic and social stagnancy. . . . Progress, as defined by the industrial community, trumps precaution.”⁶ By the mid-1970s, as consumer concern over environmental pollution placed increasing pressure on industry, it responded with a “frontal assault on the public health ideals of prevention,” hiring product-defense firms, public relations agencies, and scientists who “systematically attacked environmentalists and labor activists as luddites determined to stifle our economy.”⁷ Industry advocates sought to portray precaution as a novel and reckless idea, rather than a long-held principle at the heart of public health. What was most daring about this campaign was industry’s largely successful effort to rewrite history in the public eye, portraying precaution as a new idea and indisputable proof of harm as a historical precedent.

Histories are not just academic exercises; they are political acts. Historians tend to be reluctant to work with policy makers for fear that they might be accused of the historian’s cardinal sin: presentism, or the error of judging past actions by the standards of the present. But we can and should learn from the experiences of the past, and we can do so without falling into presentism. Policy making is often based on arguments about the past, although rarely are those arguments as explicit as industry’s attempts to rewrite the history of the precautionary principle. Foresters, for example,

observe the ways forests have responded to particular ecological disturbances or silvicultural treatments in the past. They use their observations to derive hypotheses about how today's forests might respond to various changes, including logging, climate change, and suburban development. Hospital review boards examine past medical mistakes to avoid repeating them. But environmental policy makers rarely take a formal or structured approach to examining historical case studies to learn when evidence emerged about potential risks or when implementation of policies might have saved lives or suppressed innovation. As the political scientist Richard Neustadt and the historian Ernest May argue, policy makers need to be more explicit about framing hypotheses and testing them with historical evidence, rather than relying on anecdotes about the past.⁸

In 2001, a European Union team charged with implementing the precautionary principle examined fourteen case studies of historical hazards. The case studies involved an agent (such as mercury) that most contemporaries had regarded as harmless at prevailing levels of exposure until additional evidence about harmful effects emerged. The goal of the exercise was to identify when the first credible "early warnings" of potential harm emerged, determine how regulatory authorities responded (or failed to respond) to those warnings, and calculate the resulting costs and benefits of that inaction. The team came up with several lessons for policy makers that correspond closely with lessons we should learn from the case of diethylstilbestrol.⁹

One critical lesson discussed in the European Union case studies concerns the importance of first recognizing limits to knowledge and then accepting that continued uncertainty is no justification for inaction. As the European Union team writes, "No matter how sophisticated knowledge is, it will always be subject to some degree of ignorance. To be alert to — and humble about — the potential gaps in those bodies of knowledge that are included in our decision-making is fundamental. Surprise is inevitable." In *Seeing Like a State*, the political scientist and anthropologist James C. Scott describes the ideal land manager as one who is humble, experienced in making mistakes, willing to be wrong, and pragmatic about the limits of his or her knowledge. The same can be said of an ideal regulator, who should be willing to acknowledge the possibility of surprise. The European Union team notes that "acknowledging the inevitable limits of knowledge leads to greater humility about the status of the available sci-

ence, requiring greater care and deliberation in making the ensuing decisions. It also leads to a broadening of appraisals to include more scientific disciplines, more types of information and knowledge, and more constituencies.²¹⁰ The regulators involved with DES understood that their knowledge about the actions of synthetic hormones was limited, but when it came time to assess risks and make decisions, they seemed to lack the humility that a partial understanding requires.

Regulators need to look beyond direct impacts from a chemical and anticipate “as wide a range of conditions and effects as can reasonably be anticipated. Whilst accepting that even the broadest appraisal processes may still fail to foresee ‘surprises,’ there is much that can be done to guard against some of the consequences of the ubiquitous experience of ignorance and surprise.”²¹¹ Civil engineers learned this lesson a long time ago; it is now accepted engineering practice to assume a degree of ignorance and devise strategies to prevent outcomes that are by definition still unknown.

Ignorance can sometimes be intentional. An industry might prefer not to find out about the potential harm its product might cause because continued uncertainty means continued profits. Without monitoring of potential hazards, we are almost guaranteed to be more ignorant than we need be. Yet as DES consumer groups found out, inducing the federal government to monitor industry is difficult, because the political pressures on regulators can be overwhelming.

Several key uncertainties abounded in the DES research, and these foreshadow the uncertainties that haunt today’s endocrine-disruptor policies. The significance of laboratory-animal experiments for people, the boundaries between synthetic and natural processes, the risks of low levels of exposure, and the significance of environmental influences on the developing fetus were all uncertain in the 1930s. They remain uncertain today, not because of lack of research effort but because of the complexity of endocrine systems. Using this complexity as a justification for continuing to expose people and environments to synthetic chemicals has proven to be a useful strategy for industry, but it is not one that is likely to protect Americans’ health or the environment.

The regulatory agencies’ willingness to approve DES was partly derived from the unwillingness of clinicians to pay heed to experimental evidence from laboratory animals. Karl John Karnaky, for example, insisted that the animal studies showing that DES caused fetal harm did not

apply to people. In one publication, Karnaky noted that numerous lab studies had shown that DES was damaging to the fetus. And yet even after summarizing all the reports that DES harmed the fetus or prevented implantation of the fertilized egg, Karnaky went on to state that women are not laboratory animals, and thus there was no reason to believe that DES was harmful to women.¹²

For ethical and logistical reasons, it is impossible to gather experimental evidence on human subjects, but it is vital that we not delay regulatory action until human evidence is available. Nevertheless, when environmentalists and regulators attempt to limit human exposure because of findings from animal evidence, industry representatives argue that such precaution is absurd because rats are not people. Many doctors and lawmakers remain unwilling to believe that a compound's harmful effects on laboratory animals or wildlife can be extrapolated to humans. Frederick vom Saal noted in the 1990s that "before DES was used on five million women in the U.S., it was clear from animal studies that DES would be damaging to fetuses. But we have this absolutely bizarre notion that humans are separate from the rest of life on Earth. You will hear physician after physician say, 'But that's an animal. What relevance does that have to humans?'"¹³ One clear message from the DES story is that we should not assume that research on laboratory animals has no meaning for people.

For decades, scientists and regulators debated the possible significance of low levels of exposure to synthetic chemicals. Even when researchers agreed that high levels of estrogens might cause harm, significant disputes remained about what those results might mean at the low levels common in the environment. Traditional toxicological models of risk posited dose-response models, where the dose makes the poison; in this model, low levels beneath a given threshold value would not be expected to cause harm. Industry advocates argued that these threshold values were based in sound science, but a careful reading of history reveals that they were often the result of political negotiation.

The boundary between natural and synthetic was also a continuing source of uncertainty. The drug companies argued that because bodies naturally produced estrogens, levels of additional estrogens that were just a fraction of the highest levels of the natural estrogens would not have a toxic effect. When Karnaky argued that DES treatment during pregnancy was safe, he pointed to the fact that a woman's body naturally produced

high levels of estrogens during pregnancy, making the additional amounts from DES insignificant. Drug companies promoting DES manipulated the concept of naturalness, with its attendant implications of purity and safety. These same arguments remain potent today in debates over the safety of steroid hormones given to livestock.¹⁴

Another critical issue focused on the limits of technology and knowledge. If technology did not exist to measure a residue, did that mean the residue did not exist? If an effect could not be measured, was the effect therefore nonexistent? Industry initially argued that only effects and residues that were measurable existed. Scientists consulting with the FDA disputed this, arguing that an inability to detect liver damage from DES, for example, could mean that liver damage did not exist. But it might mean that available tests lacked the sensitivity to show slow, chronic changes. Initially, the FDA regulators agreed with this idea, refusing to assume that an inability to detect a residue or an effect meant the chemical was safe. Yet by 1947, this idea had been discarded, as the FDA joined the industry in arguing that if something could not be measured by available technologies, it did not exist.

Each time regulators reached the limits of their knowledge about the effects of a chemical exposure, they decided to move ahead and allow people to be exposed. Each time they vowed to use that new exposure as an experiment that would be monitored, so that policy makers could learn from the experiment. The toxic chemicals were released with the underlying assumption that “any major problems will emerge in good time for corrective action.”¹⁵ The corollary often cited was that if no major problems emerged, the compound must be safe. Yet when no monitoring is being done, that fundamental assumption is wrong. People may be dying in increased numbers from a particular chemical exposure, yet if their death rates are not being monitored, industry will continue to insist its products are safe. Time and again the federal agencies failed to learn from their own histories — sometimes because they lacked the funding and political power to insist on monitoring, and sometimes because they refused to pay attention to results.

The continuing failure of the FDA to regulate DES and the continuing insistence of physicians on prescribing the drug were closely linked to particular social constructions of diseases and treatments. As the medical historian Robert Bud argues about antibiotics, drugs “came to stand for

the technical solution to infection, replacing control through prevention.” Similarly, for many, DES represented a technical solution to menopause, then to miscarriage, and eventually to grain shortages. Advocates of progress tended to override concerns based in precaution. Rosner and Markowitz show how during Depression-era debates over the safety of lead paint, the lead industry “sought to co-opt the growing public health movement by identifying lead with modernity and health. . . . The themes of order, cleanliness, and purity that were hallmarks of the efforts to reform and sanitize American life were quickly incorporated into the promotional materials developed by the industry.”¹⁶ A similar pattern emerged for DES. Rather than addressing the larger ecological issues of “accidents of pregnancy,” DES seemed to promise a technical solution that was cheap and, above all, modern. The pharmaceutical companies played upon these themes in their promotions of the synthetic hormone.

A crucial lesson from the DES history is that science alone cannot solve our chemical problems. Like many people trained in science, I had assumed that additional research on chemical and biological mechanisms would resolve policy conflicts. If we found more evidence about bisphenol A's effects on gene expression, then surely federal agencies would restrict the chemical. This has proven no more true than the hope in the 1960s that DES would be banned as soon as researchers linked DES use to cancer in women. With both chemicals, research findings alone did not lead to action. As the history of DES makes clear, the call for “more research!” has often become a way of delaying action, keeping profitable drugs and chemicals on the market as long as possible.¹⁷

In *The Secret History of the War on Cancer*, the epidemiologist Devra Davis describes how industry lobbyists have manipulated scientific uncertainty and risk-assessment protocols to delay action against toxic chemicals, particularly those that can cause cancer. Many industries have used a systematic approach to magnify doubt and delay regulatory action. “The first step is to feign blindness to a problem induced by a chemical by making sure that no records are kept concerning the health of the workers handling the chemical in question (without data, there is no proof). The second step is to create evidence systematically which refutes any possibility of a problem. Then sponsor carefully designed studies in low-risk populations which will confuse. Then fund yet more studies to suggest that doubt remains even after the dangers are clearly defined. Finally, use

litigation, political lobbying and confidentiality clauses to delay publication of results for years or decades.”¹⁸ The DES histories show how successful these strategies can be.

The history of tobacco regulation offers a useful parallel. In *The Cigarette Century*, the public health historian Alan Brandt argues that the failure of U.S. doctors and regulators to respond to the dangers posed by tobacco was the result of a deliberate strategy to manufacture doubt. As Davis writes, “Working first with medical experts . . . the tobacco strategists counted on their ability to hire leading scientists who did not want to believe that smoking was harmful. With such an impressive front line, tobacco sympathizers carefully crafted doubt about what evidence is required before we can say that a given agent truly is a true threat to human health.”¹⁹ Strategies to promote pesticides and other endocrine disruptors were similar. In 1996, the Food Quality Protection Act (HR 1627) was passed, eviscerating the Delaney Clause and replacing it with the standard of “reasonable certainty that no harm will result from aggregate exposure to pesticide residue.”²⁰ In other words, processed foods would be allowed to contain residues of carcinogenic pesticides. Zero tolerance was replaced with a risk-assessment standard that allows carcinogens to be present in processed foods if they create a “negligible risk” of causing cancer. This policy rested on the belief that scientists can indeed “assign accurate risks to the likelihood that a given quantity of a chemical will cause cancer.”²¹

A group of toxicologists with the American Health Foundation lobbied particularly hard for the repeal of the Delaney Clause. One toxicologist, J. H. Weisburger, published a series of papers contending that the Delaney Clause was pointless because cancer was genetic, not environmental, in origin. In 1996, Weisburger argued that the Delaney Clause “was based on the hypothesis held in the 1950s that human cancers are due to environmental chemicals. This is clearly not true for the great majority of cancers and therefore, the Delaney Clause as framed has not saved any lives, is obsolete, and should be eliminated.”²² Weisburger and other staff at the American Health Foundation were key authors of a position statement on carcinogens in foods that was endorsed by the North American Society of Toxicologic Pathologists in 1995. In this position statement they argued that the Delaney Clause was essentially “irrational” because after the clause was passed in 1962, “major progress has been made in understanding mechanisms of cancer induction and in recognizing causes

of human cancer. The Clause in conjunction with its present legal interpretation and implementation does not provide for rational, scientific evaluation of carcinogens.” The position statement declared that animal studies were often inapplicable for humans, and chemicals in food should be monitored only on the basis of human, not animal, experiments.²³

The American Health Foundation also worked under contract with tobacco companies, promoting research into “safe” cigarettes. We can trace abundant parallels between the tobacco lobbyists and the firms hired to slow regulation of global warming and toxic chemicals. Not only are the strategies the same; the people, funding sources, consultants, and public relations firms are often the same as well. The historian Naomi Oreskes argues that one key political tactic involves manufacturing a fake debate to undercut emerging scientific consensus. This tactic has been used against the consensus that sulfur and nitrogen emissions cause acid rain, the consensus that chlorofluorocarbons cause the hole in the ozone layer, the consensus that cigarette smoking causes cancer, the consensus on endocrine disruptors, and particularly the growing consensus on global warming. These efforts follow a similar pattern. First, deniers argue that the science is uncertain. Then they argue that the scientific concerns are exaggerated and the true risks are small, particularly compared to natural risks already existing in the environment. Finally, they state that technology will solve the problem, eliminating the need for government interference. The campaigns against environmental and public health regulation involve the same institutions, run by the same people, funded by the same sources.²⁴

David Michaels and Celeste Monforton describe in detail the ways that the tobacco industry promoted scientific uncertainty to delay regulatory action. They write that “the tobacco industry recognized the value of magnifying the debate in the scientific community on the cause-and-effect relationship between smoking and lung cancer. In the 1960s, the Tobacco Institute published a journal entitled *Tobacco and Health Research*, aimed at physicians and scientists. The criteria for publishing articles in the journal were straightforward: ‘The most important type of story is that which casts doubt on the cause-and-effect theory of disease and smoking.’ In order to ensure that the message was clearly communicated, the public relations firm advised that headlines ‘should strongly call out the point—Controversy! Contradiction! Other Factors! Unknowns!’”²⁵ While many

people feel that this strategy has marked the tobacco industry as unique, Michaels and Monforton point out that there is nothing unusual about tobacco companies. The same firms are involved in the same activities today, particularly to block regulation of bisphenol A and other common chemicals that offer profits to some people and risks to millions.

The current Endocrine Disruptor Screening Program illustrates the problems with the argument that more science is the only rational solution. As part of the Food Quality Protection Act, Congress ordered the EPA to begin screening and testing chemicals and pesticides for endocrine-disrupting effects by 1999. The Endocrine Disruptor Screening Program would consist of a series of tests on laboratory animals designed to discover whether synthetic chemicals are endocrine disruptors. This sounded rational. With more than a hundred thousand chemicals on the market today, a coordinated screening program might help consumers and regulators know which chemicals interfere with hormones and which do not.

At first, enthusiasm was high within environmental and research communities. The journalists Susanne Rust, Meg Kissinger, and Cary Spivak write that “the EPA convened a committee of scientists from academia, the government and the chemical industry to lay the groundwork for testing these chemicals. They came up with a way to identify and test chemicals for the risks and get the information to the public. . . . Then-EPA administrator Carol Browner said in 1998 that her agency would begin fast-tracking efforts to screen these compounds by the end of that year. ‘Some 15,000 chemicals used in thousands of common products, ranging from pesticides to plastics,’ would be screened, Browner said.” But a decade and nearly \$80 million later, debates over protocols meant that not a single chemical had been screened.²⁶

Delays and calls for more science are not the only ways the Endocrine Disruptor Screening Program has favored business as usual. Critics say that Stephen Johnson, the EPA administrator who served under the Bush administration from 2005 to 2009, favored industry in myriad ways, for example by allowing the Endocrine Disruptor Screening researchers to conduct lab tests that use a strain of rat that is essentially unresponsive to known hormone-disrupting chemicals.²⁷ In addition, the EPA allowed the study rats to be fed soy-based chow high in phytoestrogens that can mask the effect of endocrine-disrupting chemicals. The rats that were supposed to be controls could not be true controls after eating endocrine-disrupting

phytoestrogens for dinner. The tests also used a dosage range that made it difficult to detect low-dose effects, which are the ones of greatest concern. And finally, the Endocrine Disruptor Screening Program did not require that tests be conducted on prenatal exposure, even though the fetus is the most vulnerable to exposure. “If your objective is not to find anything, that’s the perfect way to do it,” notes Frederick vom Saal.²⁸

The reasons for most miscarriages, stillbirths, and premature births were a mystery during the DES era, and they remain a mystery today. Some miscarriages occur because of chromosomal abnormalities. Others come about because of hormonal problems such as diabetes, which was the problem for which DES was originally prescribed before its expansion into all cases of repeated miscarriage and then all cases of pregnancy. Many other “accidents of pregnancy” are intimately connected to what Sandra Steingraber calls the ecology of pregnancy: the larger web of relationships that interconnect a woman and her family with social, biophysical, and cultural environments.

Poverty, poor nutrition, stress, exposure to smoke, and exposure to environmental pollutants may all be involved in fetal death. Some of these involve individual choices that the mother makes, such as what she drinks, smokes, or eats. Historically, much of the emphasis on prenatal care has been on the mother’s individual choices. Miscarriage is often portrayed as due to a flaw of the mother’s. She had bad genes or she ate the wrong thing, she took a drink, or her body was inadequate for the task of fulfilling her female duty. As the authors of a 2004 health publication suggest, work to reduce the rate of low-birth-weight infants should focus on “improving maternal lifestyle choices.”²⁹

In 2003, a flurry of media attention greeted the publication of two studies that appeared to show that treatment with the hormone progesterone might reduce the risk of preterm babies.³⁰ Like the initial DES studies, these two studies were limited in extent, did not test to see whether the hormone reduced perinatal morbidity and mortality, and did not follow the babies into adulthood to find out whether the prenatal exposure affected their later health. As with DES, the mechanisms of the hormone’s activity were unclear. The studies did not attempt to show whether progesterone was safe for fetal development, nor were they able to address the multitude causes that lead to preterm births — factors such as poverty, poor

nutrition, air pollution, and obesity. A few researchers urged caution, pointing out the parallels to the DES history. Nevertheless, many interpreted the results just as an earlier generation had interpreted the early DES studies. One newspaper headline promised “A Shot of Hope” with progesterone. What the *New York Times* called “the toll of anguish” of premature labor might be solved with an inexpensive pill.³¹

The *New York Times* medical writer Jane Brody acknowledged that failed pregnancy is complicated: “Poverty, lack of prenatal care and chronic stress raise the risk as well. Women who smoke raise their risk by 20 percent to 30 percent. As many as 15 percent of all preterm births are attributable to smoking during pregnancy. Body weight also matters: the risk rises if a woman is too thin before pregnancy or gains too little or too much during the pregnancy. . . . Women with certain health problems are more likely to deliver prematurely. These include diabetes, whether it developed in pregnancy or beforehand; high blood pressure, either before or during pregnancy; and serious infections like bacterial pneumonia, kidney infection, acute appendicitis or a sexually transmitted disease.”³² But instead of addressing poverty, pollution, and racism, the media response raised hopes that with progesterone doctors could treat high-risk pregnancies with a pill. Yet as the DES tragedies illustrate, the ecology of a healthy pregnancy is intimately linked to the larger ecosystems, not just to the individual genes or choices a woman makes.

Industrial chemicals are abundant artifacts of a society that was brought into being within a highly specific cultural infrastructure against a deeper historical backdrop of evolution that occurred without their presence. And yet increasingly they are a part of the natural world, and as persistent chemicals, many of them will continue to be a part of our bodies far into the future.³³ Although we cannot remove the traces of these chemicals from our bodies or ecosystems, governments can decide to act with precaution.

Steingraber and the biologist Mary O’Brien offer alternative approaches to environmental regulation that recognize the need to make difficult decisions in the face of continuing uncertainty. Rather than precluding all action until everything is known about a chemical, these approaches advocate that anyone proposing to use a chemical should first compare a wide range of alternatives, then choose the least toxic alternative available for the given situation. Under the National Environmental Policy Act of 1970, federal land agencies are required to conduct a similar comparison of

alternatives. Before creating a new forest-management plan, for example, the Forest Service must first provide an Environmental Impact Statement that describes the possible outcomes from a wide variety of reasonable alternatives. Theoretically, these may range from “do nothing” to “do everything.” Comparing a wide range of alternatives can help decision makers realize that better approaches exist — particularly since the National Environmental Policy Act requires that federal land agencies involve the public in the planning process. As anybody connected with forest planning can tell you, the process is not perfect, but alternatives assessment does offer a way to bring diverse voices into the regulatory process and a way to search for the least toxic alternative.³⁴

The most important lesson of the DES tragedy is the need for intelligent regulation to protect public health and the environment. Among the free market enthusiasts of the George W. Bush administration, regulation was a dirty word. During the eight years of anti-regulatory fervor between 2000 and 2008, environmental and health agencies were eviscerated. Political appointees chosen to head the agencies were often people who had spent their careers battling regulation rather than trying to make it more effective. Little wonder that the Food and Drug Administration and the Environmental Protection Agency are both in chaos. While it is impossible to say what changes President Obama’s administration might bring to environmental policy, a respect for scientific evidence and a pragmatic understanding of the need for careful regulation and oversight should be among the highest priorities.

The financial chaos of fall 2008 illustrates the perils of ideology-driven deregulation. After years of deregulation and the abdication of oversight responsibilities, financial systems threatened to collapse. Advocates of deregulation had insisted that financial systems were resilient, able to absorb whatever was tossed at them, yet the chaos of 2008 finally made pundits rediscover the virtues of regulation. Why were so many powerful groups able to ignore the growing signs of instability and stress? As the historian Peter Perdue notes, “Governing elites resist looking too closely into historical roots of current crises; they suppress evidence and manipulate historical narratives to legitimate themselves. The fact that financial crises, or environmental crises, have reoccurred repeatedly even in recent memory doesn’t guarantee that anyone will really want to address the fundamental causes. Historians have to recognize, and tell their readers, that

impulses to denial, willful blindness, and ideological distortion are just as powerful as rational analysis in causing social change.³⁵ Historians must play exactly this role. We might not be able to prevent the powerful from willfully manipulating historical narratives to stay in power. But as the DES histories make clear, we can and should provide counternarratives that push back against these manipulations.

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