



Scared to Death

How Chemophobia
Threatens Public Health

Presented by the



AMERICAN COUNCIL
ON SCIENCE AND HEALTH

Written by **Jon Entine**

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Threatens Public Health

A Position Statement of
The American Council on Science and Health

By Jon Entine

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CHE•MO•PHO•BI•A:

the irrational fear of chemicals

Executive Summary

When Pamela Davis was pregnant with her daughter Meaghan, she started to worry about contamination from lead paint in her Hoboken, New Jersey home. She read stories about chemicals in baby dolls, pots, shower curtains and carpets. An article on the Internet warned that sippy cups were dangerous. A friend told her that the bright pink baby pajamas she had gotten as a gift were treated with toxic flame-retardants. Soon her entire nursery seemed to pose mysterious threats to her unborn baby. Pamela felt trapped.

If news stories and the Internet are to be believed, the dangers from chemicals are increasing, cancer stalks us at every turn and our children are vulnerable. Synthetic chemicals are essential for modern life, but our views of them are conflicted. We rely on chemicals to improve human health. Pharmaceuticals keep us healthy. Plastics are found in everything from toys to cars to medical supplies. Pesticides and herbicides boost food production and quality. It's impossible to conceive of life in the 21st century without the materi-

als and fuels that synthetic chemicals have made possible. But from soap to sunscreens, drugs to DDT, we are faced with an endless stream of confusing messages about the safety of chemicals we come in contact with every day. The synthetic ingredients that make up many products suggest the unknown, and like many of us, Pamela Davis processes that as fear. “Once you’re aware of one thing it just spreads and you start questioning everything,” she said. “You can drive yourself absolutely crazy trying to keep your baby healthy.”

Considering the conflicting narratives, the public has difficulty distinguishing between useful and benign substances in products and those that could pose dangers when misused. Highly publicized reports of environmental, chemical and pharmaceutical catastrophes—from the Exxon Valdez and BP oil spills to Bhopal to thalidomide—are mixed interchangeably with exaggerations and scare stories about chemicals found in common plastics or in our homes. Belief in the relative benefits of chemicals, trust in the industries that produce them and confidence in government regulators have never been lower. Corporations that produce chemicals are often portrayed as greedy and indifferent. Questions persist about the government’s ability to exercise its oversight responsibilities.

The perceived risk posed by common chemicals has grown even as research has raised doubts about the assumed links of many chemicals to cancer. Lifestyle factors like a lack of exercise, smoking, alcohol consumption and eating habits that lead to obesity contribute far more to the overwhelming majority of cancers, while the misuse of chemicals is believed to trigger only a few percent of the cases at most. Yet, the chemophobia epidemic keeps gaining momentum.

How does the public adjudge hazard, safety and risk? How safe is safe? Media perceptions and government regulations are often shaped by a fervor fed by misconceptions about the widespread dangers of common chemicals. An illusion has developed that chemicals can be divided into categories of “safe” versus “unsafe.” But any substance, even food and vitamins, can be harmful if we consume too much of it. Safety is relative, depending on the frequency, duration and magnitude of exposure. This obsession with chemicals is unhealthy. Serious health challenges need to be forcefully confronted,

but the resources devoted to challenging and removing relatively innocuous chemicals and developing substitutes—substances that have often not been scrutinized as much as the chemicals they would replace and thus confer an illusion of safety—divert us from addressing known health risks. This chemophobia can result in the opposite of what was intended: a decrease rather than an increase in public health.

Introduction

The public misunderstanding of chemicals and risk has arisen due to variety of factors: advances in analytical chemistry allowing the detection of ever smaller amounts of substances; evolution of the Internet and social media; emergence of environmental advocacy organizations staffed with committed activists but often few scientists; uncritical or outright biased reporting about claims that synthetic chemicals are inherently risky; industry capitulation to campaigns against their products; government inclination to respond to exaggerated claims in politically safe but scientifically unsound ways; and the erosion of public trust in authority, including of government, industry and the scientific community.

Chemical manufacturing is estimated to be a \$3 trillion global enterprise. The U.S. Environmental Protection Agency (EPA) estimates that there are 84,000 synthetic substances in use in the world today. Chemicals are used to make a wide variety of consumer goods, as well as products for the medical, agricultural, manufacturing, construction and service industries. The boom

started in the early 20th century and accelerated in the 1920s and '30s with advances in technology leading to the creation of new forms of plastics, including nylon and synthetic rubber, made from petrochemicals. The use of newly developed chemicals played an important role in the Allied victory in World War II.

In the postwar years, a country on the cusp of sustained prosperity embraced scientists and industry as architects of innovation. The 1950s brought affluence to more Americans, leading to an increased demand for consumer goods, from energy and detergents to plastic, rubber and fibers. A sophisticated pharmaceutical industry arose. Agribusiness grew rapidly in response to both public concern about feeding the world—the Green Revolution was made possible by the advent of pesticides and synthetic fertilizers—and the desire for fruits and vegetables year-round. It was an era of growing abundance and chemicals were viewed as essential components of this consumption revolution.

But the complexity of modern life gradually intervened. Dramatic growth laid bare the inadequacy of certain public protections. Corporations, the engines of progress, were also the main source of industrial pollutants that fouled our air, water and soil. Legitimate concerns emerged over the use of chemicals on farm products and in the making of consumer goods and drugs. Highly sophisticated detection techniques that measure minute levels of toxic chemicals in blood and urine helped fan anxiety. Fifty years ago, science could isolate a trace chemical from a capful dumped into a swimming pool; now we have instruments that can identify that same chemical in the parts per trillion in Lake Erie.

In response to the growing impact of chemicals, numerous federal agencies, most notably the EPA, which regulates chemicals in the environment, and the Food and Drug Administration (FDA), which regulates foods and drugs, were founded or expanded. The Centers for Disease Control (CDC) and the Occupational Health and Safety Administration (OSHA) also evaluate potentially hazardous chemicals, particularly those that cause, or might cause, cancer. These agencies have evolved in a climate of increasing public mistrust to address the growing complexity of modern production and con-

sumerism. Most industrial countries have comparable oversight bodies. Today, there are 170 synthetic chemicals or exposure circumstances that have been classified by one such agency, the International Agency for Research on Cancer (IARC), as known or probable human carcinogens.

Numerous chemicals—natural and synthetic—have been indentified in the environment as dangerous at elevated levels of exposure and for which genuine caution is warranted. For example, lead exposure can lead to neurological problems, including seizures, coma or death, which is why its use is tightly regulated. Many workers exposed to asbestos, another natural substance, developed lung disease and cancer because its toxic effects were not known, regulations were lax, ventilation systems were inadequate, and they did not wear protective clothing. Workers who handle almost any chemical in high enough concentrations need special protections. But even a highly toxic chemical should not necessarily be banned outright; that decision should be based on where and how a chemical is used and at what concentrations. Its potential risks must be balanced against its demonstrated benefits.

The public controversy, however, exists over relatively common chemicals found at minute levels supposedly lurking in our foods and in everyday consumer products. Lurid headlines, such as “Alarming Body Burden Results: Tests Reveal 300 Chemical Compounds in Newborn Babies” (Lance 2008) or “89 of 116 Chemicals Detected in Americans’ Blood and Urine” (Brown 2009), used alarmist language. Although advocacy groups play an important role in focusing public attention on potential environmental hazards, some NGOs (non-governmental organizations) consistently exaggerate the threats, going so far as to portray our houses, schools, hospitals and workplaces as toxic cauldrons. By their measure, questionable substances can be found in meats and fish, on fruits and vegetables. The bottled water industry, created because people feared contaminants endanger our tap water, now finds itself under scrutiny for selling water in plastic containers made with chemicals that modify our hormones. Cookware and plastic wrap, sippy cups and the cans used to package long-shelf life foods are portrayed as serious hazards. Danger looms in cosmetics, toothpaste and cleansers. Carpets, drapes and cabinetry are sources of alarm. The list goes on and on.

While scientists may scoff at this caricature of risk and the implication that chemicals are inherently dangerous, such stories are the calling card of many advocacy campaigns and are given credence in the media. Even as you read this, people are snapping up the latest scare treatise, *No More Dirty Looks*, which, according to *Time* magazine, “unmasks the toxic ingredients in mainstream chemicals.” (Walsh 2010)

Even as the hard evidence suggest Americans have never been safer when it comes to exposure to chemicals and drugs, many people mistakenly believe we face more environmental hazards now than at any point in history. That’s understandable. Over the years, the public has been traumatized by oil spills; the thousands of deaths and injuries associated with the methylmercury contamination of Minamata Bay in Japan by the Chisso Corporation from 1932 to 1968; the explosion at a Union Carbide pesticide plant in Bhopal in 1984; and occupational exposures to vinyl chloride, benzene and aniline dyes. The problems caused by the drug thalidomide, which was withdrawn in 1961, left deep scars. Numerous drugs have been withdrawn in recent years because of health concerns such as cardiovascular toxicity (e.g. Vioxx/Rofecoxib; fenfluramine, with fentermine called Fen-phen), liver damage (e.g. Trovan/Trovafloxacin) or other ill effects, some not sufficiently identified during trials.

Less clear-cut are controversies over exposure to environmental chemicals such as Agent Orange (a Vietnam-era defoliant that contained a dioxin compound), PCBs (polychlorinated biphenyls, found in industrial fluids) or the pesticide DDT (dichlorodiphenyltrichloroethane), in which scientists have modified or even reversed their assessments of toxicity. Equally problematic are reports about the purported dangers of chemicals that we encounter regularly in common products, such as BPA (bisphenol A) and phthalates used in plastics; the industrial surfactant PFOA (perfluorooctanoic acid also known as C8), PBDE (fire retardant compounds polybrominated diphenyl ethers) and atrazine, an herbicide.

Unfortunately, scientific literacy in the United States is abysmal. On the 200th anniversary of Charles Darwin’s birthday, a Gallup poll found that only 4 in 10 Americans believed in the science of evolution (Gallup 2009). Many journalists do not have the training or sophistication to put complex science

issues in context. Media stories and Web posts often demonize commonly used chemicals that scientists and regulators have found to be perfectly harmless. Unwarranted fears are intensified by the myth that “nontoxic” and “green” chemicals exist that can replace the allegedly risky ones. These narratives are bolstered by the mistaken belief that the presence of a synthetic chemical at any concentration is dangerous. The trace of a chemical in the air, water or even in our urine or blood is in itself not necessarily something to be concerned about. The Renaissance physician Paracelsus crystallized the central tenet of toxicology, loosely translated as, “The dose makes the poison.”¹ Our bodies and the environment are made up of thousands of chemicals, natural and synthetic, that theoretically could harm or kill us. Every chemical can be dangerous if the level of exposure is high enough. We need to weigh the benefits that a chemical might bring against its potential toxicity—and at what dose or level of exposure.

There are toxic threats in our environment and it’s important to identify them and take appropriate action, but the picture painted in some quarters far overstates the actual dangers. Regulation of chemicals is stricter and more effective than it’s ever been. There have been significant advances in technology and ways of handling chemicals by industry. Only a trickle of new drugs makes it to market each year. In the case of pesticides, for example, the crop chemical industry estimates that only one in 139,000 new compounds survive the gauntlet from the chemist’s laboratory to the farmers’ fields. Each potential product that makes it into production undergoes some 120 separate tests taking 8 to 10 years at a cost of as much as \$184 million (CropLife America 2010).

The politics of contested science can be a messy business for everyone. The motivations of industry and self-proclaimed environmental white knights are not always transparent. Intentions are difficult to deconstruct when ideology, financial incentives, academic reputations and public attention are in play. While scientists who accept private funding, even for a study of a sub-

¹ The German axiom, *Alle Ding’ sind Gift, und nichts ohn’ Gift; allein die Dosis macht, daß ein Ding kein Gift ist*, translates more directly as, “All things are poison and nothing is without poison, only the dose permits something not to be poisonous.”

stance that's not at issue, risk being labeled by advocacy groups and academic scientists as "corrupt," NGOs and university scientists who endorse exaggerated assessments of chemical risk are sometimes positioning themselves for government grants or publicity.

Chemophobia is rising even while the actual danger of chemical contamination or harm from everyday exposures, particularly in the workplace, has decreased sharply over the years. The very word "chemical" has become a hot button. A recent national poll by the University of Michigan found that the public rates "chemicals in the environment" almost as big a concern as teen pregnancy, alcohol abuse and child neglect, and far more dangerous than depression or school violence (University of Michigan Child Health Evaluation and Research Unit 2010). Yet, researchers have found that more than 70 percent of cancer cases can be linked to smoking and poor eating habits that lead to obesity, while exposure to chemicals causes only a few percent of the cases at most (Doll and Peto 1981). Perceptions about chemicals have become so distorted that many people are willing to forgo the unquestioned benefits of their use, such as in vaccines, because they believe that they could poison their children. The result is a society that is increasingly wary of chemicals and science in general, and supportive of the removal from the market of many useful and in some cases irreplaceable chemicals—even when there is no evidence that they pose serious risks and the substances that replace them are often untested. Moreover, out of political expediency, the government is often forced to respond to public scares by spending millions of dollars on amelioration, research and mitigation—money that often goes to organizations that have a financial incentive to maintain there are problems. If it's later perceived that this money was ill used, the credibility of both scientists and the government are compromised—and the public interest was not served.

The Rise of the Environmental Movement

In the early years after WWII, the benefits of industrial chemicals and the positive role of industry in general, especially in improving the quality of life, overshadowed environmental concerns. The agricultural revolution was transforming the world, bringing unanticipated levels of self-sufficiency and prosperity. Synthetic pesticides were hailed as modern miracles in the battle against pests, weeds and hunger.

However, public attitudes toward what were then called conservation issues began to change. Pollution emerged as a serious problem. A noxious mix of sulfur dioxide, carbon monoxide and metal and coal dust descended on the Pennsylvania town of Donora in 1948 and London in 1952, killed more than ten thousand and sickened more than 100,000. Los Angeles was regularly in the grip of a smoggy shroud. Fear of cancer—from pollution, radiation, agricultural chemicals, mysterious microbes in our food, water, whatever—escalated. It was the beginning of a long, gradual decline in the confidence of Americans in industry and the ability of government to protect them (American National Election Studies 2009).

Evolution of the FDA

Growing concerns in the 1950s spurred legislative action to amend the quarter-century-old Federal Food, Drug and Cosmetic Act (FDCA) from which the FDA had emerged. Congress had passed the FDCA in 1938 after the poisoning deaths of more than 100 patients who ingested sulfanilamide medication in which diethylene glycol was mistakenly used to dissolve the drug and make a liquid form. “Safe tolerances” had been established for “unavoidable poisonous substances” but the rules were vague because of the rudimentary science of the times. It became clear that the old laws did not adequately address the consequences of the surge in the use of complex chemicals on farms and in foods and their possible implications for human health.

In 1954, Congress passed the Miller Pesticide Amendment, which set safe tolerances for pesticide residue on raw fruits and vegetables. The Food Additives Amendment, passed four years later, in 1958, required premarketing clearances for substances intended to be added to food. Prior to that legislation, the FDA had to prove an additive was potentially harmful before it could obtain a court order banning its use. This law shifted the responsibility to prove safety to the manufacturer, even though “safety”—the absence of risk—cannot be “proven” by science.² The amendment included the Delaney clause that effectively banned any food additive that was shown to cause cancer in any species:

“No additive shall be deemed to be safe if it is found to induce cancer when ingested by man or laboratory animals or if it is found, after tests which are appropriate for the evaluation of the safety of food additives, to induce cancer in man or animals.” (Merill 1997)

This law broke new ground as it invoked science as the way to assess risk, but it was problematic for other reasons. The language of the clause implies that the results of cancer studies in nonhuman species, such as rodents, could

² The limit of detection always determines the extent of what we mean by safety, and we cannot prove the absence of something only its presence.

be assumed to apply to humans, thus resulting in bans when only minute levels are found. The Delaney clause also contradicts the central rule of toxicology: “the dose makes the poison.” It established the scientifically suspect notion that dose doesn’t always matter. In effect, the government legitimized the use of very high-dose studies in which animals were fed hundreds or even thousands of times more of a chemical than humans could possibly consume, without clear evidence that the effect on rodents correspond to the effect of low dose exposure on humans. (The Delaney clause remains operative today, but is followed only in part because evolving analytical techniques enable chemists to detect chemicals of interest in food or water at levels a billion to a trillion times lower than was possible in 1958. For example, if it’s shown that a regulated food additive does not cause cancer but contains a trace level impurity added during processing that does induce cancer when tested separately, the *de minimis* trace constituent would not result in the additive being banned.)

The new zero-tolerance legislation led to the country’s first national cancer panic. Only weeks before Thanksgiving 1959, miniscule traces of a synthetic herbicide that had been found to cause cancer in rodents exposed to high doses were detected in cranberries grown in Oregon and Washington. That set off a media scare. In the hysteria, the Secretary of Health, Education and Welfare announced:

“The Food and Drug Administration today urged that no further sales be made of cranberries and cranberry products produced in Washington and Oregon in 1958 and 1959 because of their possible contamination by a chemical weed killer, aminotriazole, which causes cancer in the thyroids of rats when it is contained in their diet...”

The sale of cranberries crashed that holiday season, devastating the industry. It was pointed out that one would need to eat 15,000 pounds of cranberries every day of one’s life to match the dose the rodents were fed, but reason was lost in the hysteria of the moment. The fears subsided when presidential hopefuls John Kennedy and Richard Nixon made a point of

eating cranberries and respected scientists spoke out to reassure the public (Life 1959).

The cranberry scare of 1959 was followed two years later by a legitimate crisis involving thalidomide, a sedative. Responding to one of the biggest medical tragedies of modern times, the government ordered the drug withdrawn from the market in 1961 after it was found to cause birth defects (Lenz 1998). The incident led to much stricter testing on pharmaceuticals and pesticides before they could be licensed and fed concerns that federal agencies might not be up to the task of overseeing potentially dangerous drugs and chemicals.

Silent Spring

The catalyzing event for the modern environmental movement was the publication of Rachel Carson's *Silent Spring* in 1962 (Carson 1962). Carson had worked for years at the U.S. Fish and Wildlife Service, eventually becoming the chief editor of that agency's publications. She argued in her book that uncontrolled and unexamined pesticide use was harming and even killing not only animals and birds, but also humans. She indicted industry and the federal government. The book kicked off a public dialogue about the affects of chemicals on wildlife and the environment.

Carson's primary target was dichlorodiphenyltrichloroethane (DDT), an insecticide then in widespread use in areas of the world where malaria was endemic, because of its effectiveness in controlling disease-carrying mosquitoes. Testing by the U.S. Public Health Service and the FDA's Division of Pharmacology had found no serious human toxicity from DDT, and the chemical's inventor was awarded the Nobel Prize in 1948. At the time of the book's publication, DDT had become an essential health weapon around the world, saving millions of lives each year. Carson alleged that DDT was harming eagle and falcon eggs by thinning shells, which could lead to fewer hatchlings. The title of her book was meant to evoke a spring season in which no bird songs could be heard because they had all vanished as a result of pesticide abuse.

In 1955 the American Cancer Society had predicted, “Cancer will strike one in every four Americans rather than the present estimate of one in five.” Seven years later, Rachel Carson would cleverly call her chapter on DDT and human cancer “One in Four.” Even people who did not care much about wildlife cared a lot about their own health and the health of their children. The greatest cancer threat, of course, is not from environmental chemicals but from cigarettes. One of Carson’s primary sources was Wilhelm Hueper, chief of environmental cancer research at the National Cancer Institute (NCI) and one of the leading researchers in this area. Hueper was so convinced that trace exposures to industrial chemicals were the major cause of cancer in humans that he focused far less attention on tobacco usage, which is now recognized as a far greater threat. The dangers of tobacco were addressed comprehensively in the 1964 report by the U.S. Surgeon General causally linking smoking to lung cancer (Public Health Service 1964). The tobacco industry responded defensively with a powerful disinformation campaign, further undermining the public’s trust in corporations. That helped give credence to one of the central arguments of the environmental movement: industry was putting profits ahead of the health of people and the planet.

Silent Spring may have been thin on the science of chemicals and cancer but it was a powerful and emotional *tour de force* for those who believed that environmental issues were being overlooked. The 1960s were marked by a growing sense that the government and “Corporate America” were aligned and indifferent to environmental challenges. A perception took hold that man himself as well as trees and wildlife were an endangered species. The cognoscenti began using an arcane term—ecology—in reference to a science of the environment, then still in its infancy.

As the decade drew to a close the Nixon Administration, already on the defensive because of Vietnam and a budding recession, found itself dealing with a number of high profile environmental challenges. When people witnessed on television the defoliation chemicals used in the jungles of Indochina, they became even more receptive to the environmental concerns advanced by Carson, consumer advocate Ralph Nader and others. Legiti-

mate concern over air and water pollution began spreading in widening eddies. Federal regulators faced increasing pressure from a skittish public to respond to concerns over the environment and public health even in cases where the science did not justify intervention.

What's now often referred to as the "cyclamate scare" is a case in point. The popular artificial sweetener cyclamate, which had been designated as GRAS (Generally Recognized as Safe) since the 1950s, came under scrutiny in 1969, when a study found that eight out of 240 rats fed a mixture of saccharin and cyclamates developed bladder tumors. The rats had been fed high-dose levels comparable to humans ingesting 350 cans of diet soda per day for months. No other labs could reproduce these findings, which are in themselves of questionable significance. But modest concerns erupted into a national scare when an FDA scientist went on network television displaying pictures of chick embryos that suffered from severe birth defects after being injected with cyclamates (Henahan 1977).

With the Delaney clause in effect, government regulators believed they had little wiggle room. "We recommend the cyclamate ban because of the law, not because there is any reason to believe that it causes cancer in man," said one of the reviewers (Science News 1969). Spurred by a public outcry orchestrated by consumer activists, including Nader's Public Interest Research Group, the FDA banned cyclamates (Price 1970). The success of the anti-cyclamate campaign led to the publication of the Nader-inspired book, *The Chemical Feast* (Turner 1970), which raked the FDA for not regulating "dangerous" food additives.

The alarmism served to reinforce the unscientific standard that high-dose studies on animals are automatically applicable to humans. It also legitimized the use of scientists to endorse politicized policy judgments, a disturbing but persistent pattern that undermines the confidence of the public in supposedly independent scientific experts. Cyclamates remain banned from food products in the United States, although the FDA has since publicly stated that a review of all available evidence does not implicate the sweetener as a carcinogen in mice or rats.

Birth of the EPA

Among the burning issues of the day were the alleged threats of DDT and the emerging concern that population growth posed a catastrophic threat to the future of the planet. One of the first of the new wave of environmental advocacy groups, the Environmental Defense Fund (now known as EDF or Environmental Defense), was founded in 1968 to specifically target DDT, and it helped launch legal actions against the use of the pesticide.

The bestselling 1968 book *The Population Bomb*, by entomologist Paul Ehrlich, blamed uncontrollable growth in what was then called “The Third World” as the seed of all environmental problems. He also railed against DDT. The issue of restricting population growth played into the debate over DDT in a disconcerting way. The public was confronted with Ehrlich’s (erroneous) conviction that hundreds of millions of people would starve to death in coming decades because of overpopulation. The issue of withdrawing anti-malarial programs as a means of population control was broadly discussed and debated. In his book, Ehrlich himself appeared to “blame” DDT for saving lives, exacerbating the overpopulation problem:

“The introduction of DDT in 1946 brought rapid control over the mosquitoes which carry malaria. As a result, the death rate on the island [of Ceylon] was halved in less than a decade. ... Death control [DDT use] did not reach Colombia until after World War II. ... Each child adds to the impossible burden of a family and to the despair of a mother.” (Ehrlich 1968)

However unintended, the exaggerated fears about population growth and environmental degradation led many conservationists to propose the unthinkable. They actively began debating Ehrlich over what he called a “death rate solution” to these combined problems. A debate erupted over banning DDT as a way to cull the world population through denying life-saving spraying of agricultural chemicals (Roberts 2010).

In response to growing public concern about a variety of environmental

challenges, the White House set up a Citizens' Advisory Committee on Environmental Quality in 1969. That was followed by the signing on January 1, 1970 of the National Environmental Policy Act, which led to the formation of the EPA. The agency assumed regulatory control of pesticides from the U.S.D.A. Not surprisingly, deciding the fate of DDT was the first task of the newly created EPA.

Scientists urged caution. The National Academy of Sciences reviewed the evidence in 1970, declaring, "In little more than two decades, DDT has prevented 500 million human deaths due to malaria, that would otherwise have been inevitable." The EPA hearing examiner, Judge Edmund Sweeney, who listened to eight months of scientific testimony about the risks of DDT, came to a similar conclusion about its benefits, found little scientific evidence of its potential harm and recommended against a ban. "DDT is not a carcinogenic hazard to man," he wrote:

"... DDT is not a mutagenic or teratogenic hazard to man. The uses of DDT under the registration involved here do not have a deleterious effect on freshwater fish, estuarine organisms, wild birds or other wildlife. The adverse effect on beneficial animals from the use of DDT under the registrations involved here is not unreasonable on balance with its benefit. The use of DDT in the United States has declined rapidly since 1959. The Petitioners have met fully their burden of proof. There is a present need for the continued use of DDT for the essential uses defined in this case. ... [N]ecessary replacements would in many cases have more deleterious effects than the harm allegedly caused by DDT." (EPA 1972b)

Two months after the Judge's hearings, EPA Administrator William Ruckelshaus, facing tremendous pressure from the media and NGOs, set aside the Judge's findings and announced a broad ban on DDT. He cited the results of high-dose studies in rodents and invoked the principles outlined in the Delaney clause, which until that time had only been used in assessing the carcinogenicity of food additives. The likelihood that a ban would cost lives, which

could have been assessed by cost-benefit or risk-risk analysis, was not considered. When it came to chemicals, perceptions and not scientific evidence was now driving the regulatory system. Today, 40 years after DDT was phased out, there is still no persuasive evidence that it is a human carcinogen or can be held responsible for widespread harm to wildlife.

Environmental Risk

The first Earth Day was held in 1970 shortly before the founding of the EPA. With pollution and the environment front and center in the public's mind, Congress responded by passing laws and launching new regulatory agencies. Key was the passage of the Toxic Substance Control Act (TSCA) of 1976. TSCA set up guidelines giving the government authority to determine if industrial chemicals present “an unreasonable risk of injury to health or the environment, and to take action with respect to chemical substances and mixtures which are imminent hazards” (EPA 2010). It specifically targeted polychlorinated biphenyls (PCBs). Over the years, the core statute has never been reauthorized or amended, but new oversight responsibilities have been added to regulate four additional chemicals: chlorofluorocarbons, dioxin, asbestos and hexavalent chromium. TSCA included a cost-benefit clause requiring that the government's authority should be exercised “in such a manner as not to impede unduly or create unnecessary economic barriers to technological innovation.”

In the late 1970s and early 1980s, two dramatic incidents—at Love Canal, New York, and at Times Beach, Missouri—focused the attention of the U.S. public on industrial chemicals in the environment. In 1978 the area around Love Canal, a neighborhood near Niagara Falls, was found to be contaminated by a variety of chemicals—21,000 tons of toxic waste buried by the Hooker Chemical Company. The public was soon inundated with stories that children born in the community had high rates of birth defects and cancer (Heath, et al. 1984). A subsequent state-of-the-art study by the CDC and two other national laboratories rejected the publicly accepted claim that the toxins caused serious genetic abnormalities or any marked rise in disease. “This [study] suggests that no specific relationship existed between exposure to chemical agents in the Love Canal area and increased frequency of chromosome damage,” the study asserted (Boffey 1983).

In 1982, the news was filled with reports that concentrated levels of dioxin had been discovered throughout the town of Times Beach. Later, PCBs were also found in the soil. Panic spread through the town, with every illness, miscarriage and death of an animal attributed to the chemicals. The EPA ordered an evacuation in 1983 and eventually declared it uninhabitable (Sun 1983). As concerns mounted, President Ronald Reagan formed a dioxin task force. At the time, dioxin, which was being blamed for a variety of illnesses in Vietnam veterans, was labeled as “the most toxic chemical synthesized by man,” based on high-dose studies in guinea pigs.

Subsequent research on the effects of dioxin on humans and other mammals led to a revised belief that its toxic effects are limited. No illnesses in Times Beach were ever linked to the presence of chemicals. Many experts question whether the razing of the town was necessary, citing the example of Seveso, Italy, the site of a disaster in 1976 that exposed residents to far higher levels of dioxin than those found in Times Beach and whose subsequent cleanup allowed the city to continue to exist. The Love Canal incident led directly to the 1980 passage of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), commonly known as Superfund (U.S. Congress 1980). Superfund not only sensitized people to the widespread nature of chemical contamination of soil and groundwater

but also led them to recognize that hazardous wastes were not only produced by industrial facilities but also by individuals in their homes, as a number of Superfund sites were local landfills. Battles over what to do with landfills have lasted years and in some cases decades. The designation of Superfund sites underscored a belief in the ineptness of government and inflamed the perception that the public was not being adequately protected.

Responding to growing concern about chemical contamination, some states and localities, convinced that the federal government was not acting proactively enough, took their own legislative actions. In the most striking example, in 1986 Californians voted for Proposition 65, the Safe Drinking Water and Toxic Enforcement Act, which ushered in a sweeping regulatory process for identifying and publicizing “toxic chemicals” (California Office of Environmental Health Hazard Assessment 2010). Proposition 65 requires the governor to publish a list of potentially dangerous chemicals. This list, which now includes hundreds of chemicals, many of which are not harmful at typical exposure levels, must be updated at least once a year. It has led to almost ubiquitous signs in gasoline filling stations, tire stores, workplaces, retail establishments (e.g. Macys, Home Depot) and even at airport boarding ramps warning that everyday products or chemicals are “known to the state of California to cause cancer, birth defects or reproductive harm.” The net effect initially was to stir anxiety among Californians and open up opportunities for class action suits, without any measurable benefits to public health.

Carcinogenic Risk

Until the 1960s, the standards used by the government to determine safety levels and manage risk were hopelessly imprecise and subjective. To establish safe levels for substances in the air, water or soil, regulators needed to move from the black/white qualitative approach of either allowing or banning a substance to a quantitative approach of determining how much of each substance might be allowable in each environmental situation. As the health focus on cancer and the fears associated with chemicals escalated, noted University of California at Berkeley chemist Bruce Ames invented a quick,

inexpensive test (now known as the Ames test) to evaluate toxicity. His test determines if any chemical of interest might cause mutations in the DNA of bacteria *in vitro* (in a controlled environment, such as in a test tube or Petri dish). If mutations were observed then that particular chemical was considered likely to be a carcinogen in lab animals.

The Ames test and the development of rodents modified to be cancer-prone led to an ultra-cautious toxicological evaluation system and chemical regulatory process. Over the years, what many scientists believe is a convoluted multi-stage model has been developed to extrapolate animal risk to people:

(1) Scientists do a biological assay (the Ames test) on some pesticide, food additive, preservative or other chemical to find out if it is mutagenic. It shows whether the DNA of the bacteria is altered in a significant way.

(2) If the chemical is confirmed as mutagenic, studies are then undertaken to determine what is called the “maximum tolerated dose” (MTD) of this chemical in rats or mice. The MTD is the amount of the chemical that almost kills a rodent (or almost achieves another parameter, such as suppressing body weight.) It is a dose that, depending on the particular chemical, can be thousands to millions of times higher than a human could ever ingest in a lifetime.

(3) Next, the rodents are fed just 10 percent less than the maximum tolerated dose daily for their entire one- to two-year lifetime.

(4) However, many chemicals cannot be fed to rodents because the substances are so noxious at the dosages given. So scientists often use *gavage* (forced feeding into the animal’s gut every day, often by injection), which is not how humans are exposed to the chemical, compromising the meaningfulness of the test.

(5) After a year or two, the rodents are sacrificed and scientists count up the tumors the animals accumulated in various organs. Most of the rodents in the control group, fed a normal diet, will have tumors anyway because they have been bred to be cancer prone. So, if the test group of rodents fed—or more likely injected with—some chemical at the highest dose has an average of, say, four tumors per animal in a particular organ, and the control group has an average of only one tumor per animal, then the chemical being tested

is said to increase cancer incidence by 300 percent (statistical significance is factored in). This does not mean that such a study proves a chemical will cause adverse effects in rats, let alone in humans exposed under more realistic conditions. Yet, this finding, designed as a first step in testing a hypothesis, often ends up in a headline or in a media release from one advocacy group or another attempting to use preliminary research to support a cause or movement.

(6) Next, and often under pressure from the energized media and environmental NGOs, a political body, such as the European Parliament or the U.S. Congress, or a regulatory body, such as the EPA, will classify and/or confirm this chemical as a likely human carcinogen, as if rodents were nothing more than miniature humans.

(7) These agencies then establish an “acceptable” level of the chemical—the EPA calls it “an upper estimate of the risk”—using what’s known as the “dose-response curve,” which includes a large margin-of-safety factor based on mathematical models. In moving to this new quantitative approach, government scientists began employing high-dose rodent studies and the same basic assumptions implicit in the Delaney clause: equating these studies to estimates of what might happen to humans exposed to the same chemicals at low doses. But there are no validated biological models that quantify the relationship between the high-dose animal results and low exposure levels experienced by humans.

Underscoring the relative arbitrariness of this process, the cutoff level is set differently by different agencies from country to country and even sometimes within a country. As in the case of the pesticide atrazine, these levels can vary by as much as 100 times. (The European safety cutoff level is 1 part per billion, while the World Health Organization sets it at 100 ppb.)

The result is that the scientific convention of setting one number to represent risk exaggerates the media and public perception of risk. Because only one number results from the assessment process, it is not surprising that, ignoring cautionary guidance by regulators, NGOs and the media select the country or agency with the tightest cutoff and then portrays this number as exact, as the best estimate of risk and as predictive of cancer incidence. But that misstates what a cutoff number means. As the EPA notes, “The actual

risk [from exposure to a chemical] may be significantly lower and may indeed actually be zero. It is important to recognize that the use of this model results in risk estimates that are protective, but not predictive of cancer incidence.” (EPA 1994)

Employing this model, a range of chemicals, including aminotriazole, DDT, cyclamates and Alar, at one time or another, have been in the crosshairs of environmental groups because of supposed cancer-causing effects on humans. Toxicology studies are important in public health because epidemiology is not very sensitive, as you cannot conduct experiments on humans. They serve as a basis for potency estimates and offer the opportunity to compare risks. However, the advantages of these studies must be balanced with their potential to exaggerate risk. High-dose effects do not necessarily occur at low doses and effects that occur in test species do not necessarily occur in humans exposed to the same agents.

Non-Carcinogenic Risk

In recent decades, there have been numerous claims linking chemical exposures to a wide variety of illnesses besides cancer: asthma, autism, attention deficit disorder, congenital malformations, sperm quality and quantity decline, diabetes, heart disease, Parkinson’s and dementia, among others (Safer Chemicals, Healthy Families 2010). To evaluate risks from chemicals that might cause effects other than cancer, the EPA has developed an evaluation model based on the general approach established by the Ames test. It assumes the direct applicability of high-dose laboratory animal tests to humans with subjective additional safety factors built in. The EPA then determines at what level a chemical causes an adverse reaction in the animal most sensitive to that chemical when it is fed the chemical over the course of set period of time. The “safe” human exposure limit is set 100 times (or more; California’s Proposition 65 uses 1,000 times) below the highest dose that is not expected to cause an adverse reaction if continuously exposed to a certain chemical. When the data are incomplete, regulators factor in the additional uncertainty by multiplying the safety factor, usually by 10 or even more, bringing the

safety margin, or margin of exposure, to 1,000 or more (10,000 times in the case of Proposition 65 listed chemicals; European regulators discuss a margin of exposure of 10,000 as sufficient for protection against “severe effects,” even carcinogenicity). So, for example, the safe level for adults would be set at 100 times lower than what has shown to adversely impact the most sensitive laboratory animal affected by that substance, while for children or pregnant women the safe dose level would be set 1,000 times or even 10,000 times lower to account for individual differences in humans.

The EPA calls this the Reference Dose (RfD). The term was originally known as the Acceptable Daily Intake (ADI), but it was criticized as potentially misleading as it wasn’t clear who was judging acceptability. Today, the meanings of RfD and ADI are synonymous. The RfD is the amount of a substance that a person at a specific weight can take orally every day over a lifetime without any appreciable health risk (with the exaggerated margin-of-error built in) (Barnes and Dourson 1988). Clearly, neither the RfD nor the ADI identifies the amount of exposure that is known to cause adverse effects. It’s an outer limit that assumes a lifetime of high-level exposure and is calculated by dividing no-effect doses from animal studies by 100, 1,000, 10,000 or more. These levels are protective in the extreme. But as with cancer exposure levels, advocacy groups and the media often use these safe dose figures as if they are precise levels that when exceeded by even the tiniest amount present a health danger.

Endocrine Disruptors

As toxicological research has become more refined, there has been an increasing focus on the effects of chemicals and drugs on human reproduction, pregnant women, infants and children. Our hormonal systems are acutely sensitive to change. This heightened concern traces back to the thalidomide tragedy in 1961, which was followed a decade later by the diethylstilbestrol (DES) debacle. From about 1940 to 1970, the synthetic nonsteroidal estrogen DES was given to pregnant women under the belief it could treat pregnancy complications and losses. The FDA subsequently withdrew DES from

use in pregnant women when it was shown to cause malformed uteruses and rare vaginal tumors in females who had been exposed to this drug in utero (Herbst, Ulfelder and Poskanzer 1971).

Although these were only two drugs among many thousands on the market, the seriousness of these problems fed a belief that the pharmaceutical industry could not be trusted, and the government was lax in its screening of drugs and chemicals and was not adequately exercising its regulatory authority. Unrealistic expectations that drugs (and all chemicals) should be risk-free have occasionally led to beneficial drugs being hastily removed from the marketplace. When reports circulate that someone, somewhere, has had an adverse reaction, there are reflexive calls for a ban and class action attorneys join the fray.

That's what happened in the case of Bendectin, a popular drug prescribed to treat nausea and vomiting during pregnancy. In 1983, an Australian researcher linked it to a variety of disorders, including fetal malformation. The release of the initial study touched off a media frenzy and demands by NGOs that the government withdraw the drug. Lawsuits mounted. Throughout the crisis the drug remained legal under the trade name Diclectin in Canada and Europe, which stood by studies that had found the drug safe. But the beleaguered manufacturer believed it had no choice but to pull it off the U.S. market. Soon after it discovered that William McBride, the scientist who claimed to have found teratogenic effects (which could alter the development of the embryo or fetus) from using the drug, had falsified his research. The FDA subsequently found no links to birth defects and no cause for alarm (Kutcher, et al. 2003) (Willhite and Mirkes 2005). Because of the negative publicity, however, the drug was not reintroduced in the United States.

During the 1990s, based on studies of fish and rodents, some university researchers began focusing on the potential impact of chemicals that appeared in laboratory tests to mimic or impede the effects of endogenous hormones such as estrogen. That's not in itself a cause for concern. Clover, some fruits, wheat and other flour and soy products (including fungal products at trace levels in wheat and other grains that are processed into bread, cereal pizza and even beer) can also potentially alter the way the hormones in our endocrine

system work. The natural chemicals that caused this effect were known objectively and innocuously as endocrine mediators.

By the early 1990s, some environmental activists and scientists began promoting a novel hypothesis: Low doses of certain chemicals might have a more severe impact than high doses. They argued that the reproductive system of animals, including humans, might not be subject to the classic dose response curve; there could be a non-monotonic response (Richter 2007). Looking to distinguish the similar hormonal effects caused by synthetic chemicals, they coined the term “endocrine disruptors,” and the label stuck. The term was chosen as a branding slogan, not unlike campaigners on abortion issues labeling themselves “prochoice” or “prolife.” Who would want to risk “disrupting” the development of a newborn? The novel notion was promoted in the best-selling book, *Our Stolen Future: Are We Threatening Our Fertility, Intelligence and Survival?* (Colburn, Dumanoski and Meyers 1996). The media and some scientists now use “endocrine disruption” interchangeably with the objective description “reproductive hazard,” even though it carries strong normative associations.

While some scientists believe there is persuasive evidence that certain common chemicals, such as the plastic additive BPA, can adversely affect human development, after more than fifteen years of research (Sharpe 2010) others believe endocrine disruption remains a hypothesis in search of data. The use of this novel paradigm has opened a new front against chemicals. Substances that have not been proven to be carcinogenic in humans at common levels of exposure—the pesticides DDT/DDE and dieldrin, dioxin, PCB, PBDE, and PFOA, for example—are now labeled potential endocrine disruptors even though the hypothesis itself remains in question (Kamrin, *The Low-Dose Hypothesis: Validity and Implications for Human Risk* 2007) (Kamrin, *Bisphenol A: A Scientific Evaluation* 2004).

The media and certain NGOs now carelessly link various substances to everything from human breast cancer to early puberty based on animal tests or trace levels found in the environment or in human blood and urine. No longer is it necessary for critics of chemicals to find evidence of actual harm; it is now sufficient to identify metabolic changes in laboratory animals in small-

scale hypothesis-driven studies to justify extensive and expensive new tests, which sometimes lead to onerous regulations. The federal government and chemical manufacturers are often portrayed as colluding to protect industry profits at the cost of human health.

Green Chemicals—Natural v. Synthetic

Many people who express concern about chemicals hold the mistaken belief that there are equivalent naturally occurring substitutes that are safer and as effective. Environmental groups have incorporated this argument in campaigns to ban various chemicals, proposing organic or “natural” substitutes. But little publicity is given to the limited effectiveness of many natural substances or the fact that many natural chemicals can also cause “endocrine disruption” or cancer in farm and laboratory animals.

Organic farming advocates maintain that so-called natural farming techniques result in more nutritious crops. There is no scientific research supporting that belief. The Agriculture Department pointedly “makes no claims that organically produced food is safer or more nutritious than conventionally produced food.” Scientists who systematically reviewed research over 50 years conclude that organically produced foods, including crops and livestock, are not more nutritious than those produced conventionally (Dangour, et al. 2010) (Rosen 2010). Not using herbicides or pesticides can, in some situations, result in increased stress on plants. If threatened by weeds, insects or poor weather, a plant’s inborn response is to generate protective natural chemicals, including mycotoxins, which can be quite toxic, and potent carcinogens.

Scientists have vigorously attempted to develop effective green chemicals—natural alternatives to synthetics known as biopesticides that can maintain the high yields and low prices upon so critical for mass food production. They have spent years researching the insecticidal properties of rosemary, thyme, clove and mint. According to Murray Isman, a leading researcher in this area from the University of British Columbia, herb-based pesticides have a broad range of action against bugs or weeds, in some cases killing them out-

right. But Isman says that claims that natural pesticides can replace synthetic chemicals are wildly exaggerated. Because the essential oils made from these herbs tend to evaporate quickly and degrade rapidly in sunlight, farmers need to apply them to crops more frequently than conventional pesticides—some persist for only a few hours, compared to days or even months—making the process labor intensive and expensive. As they are generally less potent than conventional pesticides, they must be applied in higher concentrations to achieve acceptable levels of pest control.

For example, environmental scientists looking at compounds used to combat soybean aphids, a major destroyer of that crop, discovered that “the organic products were much less effective than ... conventional pesticides at killing the aphids and they have a potentially higher environmental impact” (Bahlai, et al. 2010). Some biopesticides, such as the fungicide sulfur, may be more toxic or harmful than their synthetic counterparts. Natural pesticides also may be less selective in what they can kill while synthetic pesticides are developed to destroy only targeted pests. In sum, conventional pesticides remain the most effective and efficient way to control caterpillars, grasshoppers, beetles and other insects that feast on food crops (BBC 2009).

Because plants (unlike synthetic pesticides) don’t need to be lab-tested in order to be sold, there’s never been much economic incentive to analyze plants for carcinogenicity. It’s almost understandable that a romantic view has developed that plants and organic production are naturally safer. Unfortunately, it’s not true. So great is humanity’s ability to shield itself from most natural threats and so powerful is the spiritual call of nature that we tend to forget that nature can be dangerous. The poisonous plants used as herbicides in organic farming didn’t evolve that way out of perversity. By the logic of Darwinian evolution, repelling something that can kill is a good way to live longer and pass on your seeds—especially if you’re a plant and can’t flee your enemies. Plants have been producing their own pesticides for hundreds of millions of years. Some biopesticides can present unique hazards. They are known as “microbial pesticides,” meaning that the pesticidal material is a fungus, or a virus or a bacterium, often with potential ill effects on humans (Muhawi 2004). As a result of attempts to promote the belief that any trace of a chemical that can cause cancer in animals

should be prohibited for human consumption, people cringe at the thought that produce might have some residues or that chemicals can be found in our blood and urine. Ironically, one of the original proponents of those scary characterizations was Bruce Ames, when he was a young scientist in the 1960s. After the development of his test in the 1960s, Ames became a favorite of environmental groups, who recruited him to help in campaigns to ban pesticides and herbicides. In later years, in part because of the discovery that many natural substances thought to be harmless were also mutagenic, he reversed his original position and now campaigns against chemophobia. Today Ames is known for his efforts to educate those who reflexively believe that anything natural must automatically be safer than anything synthetic.

As bioanalysis grew in sophistication, Ames turned his sights toward the natural world. He identified 52 natural pesticides, and evaluated them the same way artificial pesticides are tested, using high-dose rodent studies. Of the 52 natural pesticides, 27 caused cancer. The 52 pesticides Ames studied are only a fraction of all natural pesticides, and most plants contain a variety of pesticides. As Ames wrote in a letter to *Science* after the Alar apple incident, “[I]t is probable that almost every fruit and vegetable in the supermarket contains natural plant pesticides that are rodent carcinogens”—and could be subject to a ban under the Delaney clause.³ He developed a relative index of toxicity that expresses the human potency of a carcinogen as a percentage of its potency to laboratory rats and mice. Using this index, the hazard from Alar in a daily lifetime glass of apple juice came to 0.0017%. In comparison, the possible hazard from natural hydrazines of consuming one mushroom a day was 0.1%, and that from aflatoxin in a daily peanut butter sandwich was 0.03% (Ames and Gold 1989).

The public’s top concerns around eating are typically food poisoning, BPA, BSE (bovine spongiform encephalopathy or “mad cow” disease), growth hormones used in animals, animal feed, genetically modified (GM) food—and pesticides. But in today’s typical American diet, 99.99 percent of

³ Alar was used in apple production as a growth regulator. The Natural Resources Defense Council, an environmental group, helped stir public concern in 1989 that led to the withdraw of the chemical. See p. 44.

ingested chemicals (by weight) are natural. The average American eats 1 1/2 grams of natural pesticides a day—about 10,000 times more than the amount of artificial pesticides consumed. For example, roasted coffee contains 826 volatile chemicals. (Roasting causes the formation of new chemical compounds.) Twenty-one of those coffee chemicals have been tested on rodents, and 16 cause cancer. A cup of coffee includes 10 milligrams of carcinogens. Among the foods highest in natural pesticides are cabbage, broccoli, collard greens, Brussels sprouts, brown mustard (extremely high), black pepper (very high), nutmeg, jasmine tea, rosemary and apples (without Alar) (www.pnas.org/content/87/19/7777.full.pdf).

Some natural crops contain more pesticides than ones treated with synthetics. All potatoes naturally contain solanine to protect them against blight. Solanine is a fat-soluble toxin that in high concentrations can cause hallucinations, paralysis, jaundice and death. Conventional supermarket celery contains 800 parts per billion of the natural chemical psoralen. Created naturally when the celery is stressed, in high doses it's a poison that can damage DNA and tissue as well as cause extreme sensitivity to sunlight in humans. Organic celery, grown without the aid of artificial pesticides, can contain as much as 6,200 ppb psoralens—nearly eight times as much as celery harvested conventionally (Moalem and Prince 2007). Farm workers who handle large quantities of the organic celery develop skin rashes and burns. By any rational standard of risk assessment, supermarket celery is safer to harvest and eat than the organic alternative.

Does all this mean that we should give up organic celery or conventional apples or abandon a vegetarian diet altogether because we are exposed to high doses of natural pesticides? Not at all. The chemopreventive effects of the chemicals found in foods outweigh the carcinogenic impact of the natural pesticides. But it's also true that, as Ames has written, "the carcinogenic hazards from current levels of pesticide residue or water pollution are likely to be minimal relative to the background levels of natural substances. ... My own estimate for the number of cases of cancer or birth defects caused by man-made pesticide residues in food or water pollution—usually at levels hundreds of thousands or millions of times below that given to rats or mice—is

close to zero” (Ames and Gold 1989).

The cancer and chemical concerns ignited by Rachel Carson and Paul Ehrlich and perpetuated by some NGOs were definitively addressed in a 1996 report from the National Academy of Sciences, *Carcinogens and Anticarcinogens in the Human Diet* (National Academies Press 1996). The NAS concluded that levels of both synthetic and natural carcinogens are “so low that they are unlikely to pose an appreciable cancer risk.” Anticipating the debate over the relative merits of green chemicals, the NAS found more danger in organics: “Natural components of the diet may prove to be of greater concern than synthetic components with respect to cancer risk,” the scientists wrote.

If pesticides are banned after being said to be dangerous using high-dose rodent exposure studies, we are almost certainly trading a miniscule risk (cancer from artificial pesticide residues) for a more certain one. As well-tested artificial pesticides are phased out, there will be greater crop losses caused by insects, healthy fruit and vegetables will become more expensive, and some people will not be able to afford to eat them as often and will substitute carbohydrates. Overall health will suffer and some people in fact will develop serious complications from obesity, including diabetes. There is no such thing as a risk-free world. Every choice is a trade-off of one risk for another. Assessing environmental risk, particularly in our food supply, will remain a major challenge going forward (Krewski, et al. 2009). Toxicity testing and risk extrapolation remain matters of art as well as science.

Politics of the Precautionary Principle

Growing out of the environmental and Green movements in Sweden and Germany in the 1960s and '70s, the precautionary principle has become a key environmental regulatory standard in Europe and Canada. Although scientific advisory panels often resist applying the principle, its influence is growing year by year. It has flourished in international policy statements, conventions dealing with high-stakes environmental concerns in which the science is uncertain, and national strategies for sustainable development. Instead of acting against environmental risks after they have been assessed, it suggests that it is more appropriate to take regulatory action when there is only the hint of danger. It's a hazard standard, one that is gradually replacing the risk standard still used (but under assault) in the United States and in most of the rest of the world when it comes to chemical regulation.

The primary foundation of the precautionary principle and the basis for many globally accepted definitions emerged out of the work of the Rio Conference, or "Earth Summit," in 1992. Principle No. 15 of the Rio Declaration notes:

“In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation” (United Nations Environment Programme 1992).

Subsequently, a group of activists, the Science and Environmental Health Network (SEHN), met in 1998 at what was known as the Wingspread Conference to further lower the threshold from “threats of serious or irreversible damage” to “threats of harm.” As in the UNEP definition, and subsequently as it’s used today, lack of scientific evidence or certainty cannot be cited to block its invocation:

When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically (Science and Environmental Health Network 1998).

In its crudest application the precautionary principle has been invoked as a means of deciding whether to allow corporate activity and technological innovation that *might* have undesirable side effects on human health or the environment. In practice, the principle is strongly biased against the process of trial-and-error so vital to progress and the continued survival and well-being of humanity.

The notion is difficult to define, which presents challenges to regulators. It loosely suggests that if any human activity raises a perceived threat of harm, sanctions can be imposed even if no cause-effect relationship can be established scientifically. Some substances are held to be intrinsically dangerous at any level, even absent definitive risk data. It assumes as its formulative basis that concern over worst-case scenarios should drive regulation. Simply the possibility of a problem could be enough to justify its use. In its most extreme application, no trade-offs can be considered, such as whether the economic

costs of regulation outweigh the potential benefits of reducing far-fetched risk or marginal health or safety improvements.

Supporters of the principle view it as a necessary tool of risk management. While well-intended by many of its proponents, it inherently biases decision-making institutions toward the status quo. Critics also see it as an amorphous concept that lends itself to a reactive, excessively pessimistic view of technological progress and empirically based risk analysis. Applied cynically, it can be used as a thinly veiled tool to legitimize trade barriers under the cover of public policy. Indeed, over the past 10 years, the European Union has increasingly used the standard to support a variety of import bans—ranging from hormones in beef and milk, to aflatoxin in peanuts, to genetically engineered crops—leading to accusations of protectionism from the U.S. and other trade partners. While it can be applied in areas as different as climate change and anti-trust policy, a primary focus has been consumer products and food and the modern technologies used to produce them.

The move towards precautionary regulation accelerated in Europe in the 1990s because of a series of health scares, which contributed to the belief that traditional risk analysis methods and environmental policies had failed to adequately protect the public. Institutions, governments, politicians and scientists in Europe were eager to regain the public trust lost after outbreaks of BES in the United Kingdom and elsewhere, dioxins in Belgium and HIV-contaminated blood transfusions in France.

The precautionary principle has been the basis for that continent's ban on GM foods and many agricultural chemicals—in many cases without supporting data suggesting adverse health consequences in humans. Various shades of it have been integrated into the EU's regulatory system, REACH, which deals with the Registration, Evaluation, Authorization and Restriction of Chemical substances. The new law, entered into force in June 2007, justifies Europe's move away from risk-based calculations in all areas of science.

The EU uses the precautionary principle as a proactive tool of both risk assessment and risk management to be used in situations where science cannot provide definitive answers. In its February 2000 communiqué, the European Commission distinguished a “prudential approach,” declaring:

“... [A]pplication of the Precautionary Principle is part of risk management, when scientific uncertainty precludes a full assessment of the risk and when decision-makers consider that the chosen level of environmental protection or of human, animal and plant health may be in jeopardy” (EU Commission of the European Communities 2000).

By definition, risk assessment now includes a political dimension based on a chosen level of a perceived threat. Although the precautionary principle was not originally established to complement a scientific approach to risk, it has increasingly evolved to become a tool for the advancement of the views of more radical environment and health advocates.

The U.S. system for regulating chemicals relies primarily on peer-reviewed science and risk assessment using hazard and exposure data and a weight of evidence standard. But precautionary standards are reflected in the FDCA of 1938 and subsequent revisions, including the Delaney clause, as they required some measure of pre-market proof of safety. On an absolute basis, of course, this is scientifically impossible because everything, natural and synthetic, can be shown to be toxic.

As a consequence of this developing worldwide precautionary ethic, caution is now throttling the regulatory engine around the world. Lawmakers often respond to mere suggestions of potential harm with reckless proposals for bans or restrictions without any cost-benefit analysis or assessment of the unintended risks that such actions might impose on our health and economy. When scientists push back, the gridlock emboldens critics and heightens consumer anxiety both about the exaggerated dangers of what are often relatively harmless substances and the government’s apparent lack of ability to regulate these “harmful” chemicals. This standoff has become even more pronounced in recent years with the high-profile campaigns against phthalates, BPA and atrazine.

Even consumer labels and “green guides,” when misused, can undermine confidence in government oversight and demonize chemicals that have been tested and approved as safe. Advocacy groups promote these guides as a way

to help the consumer through the thicket of dangerous chemicals, when in truth they often inflame an irrational fear that synthetic substances are more harmful than natural ones. “A rose may be a rose. But that rose-like fragrance in your perfume may be something else entirely, concocted from any number of the fragrance industry’s 3,100 stock chemical ingredients, the blend of which is almost always kept hidden from the consumer,” asserts the Environmental Working Group in an online diatribe against the cosmetic industry (Environmental Working Group 2010). It writes that perfumes often contain what it calls “secret chemicals” not listed on labels that can trigger severe allergic reactions, cause cancer, impair neurological development or disrupt hormones, even at the minute levels these mystery chemicals are supposedly found in cosmetics. EWG provides no documentation for such exaggerated claims.

EWG, EDF and other NGOs propose labeling approved ingredients based on how rodents are affected when exposed at dosage levels a thousand or more times higher than what might be experienced by humans. So, for example, harmless perfumes made by Calvin Klein, Jennifer Lopez, Victoria’s Secret and other brands would be labeled as carcinogens or endocrine disruptors or neurotoxins (Environmental Working Group 2010). Such an addition, of course, would be equivalent to adding a skull-and-crossbones to the label, dooming a perfectly safe product and throwing a cloud over an entire industry. Yet this EWG report was approvingly disseminated through cyberspace and credulously featured by the mainstream media.

Environmental NGOs and the Media

The rise of the environmental movement and the fragmentation of the media in the age of the Web have led to a growing influence of advocacy organizations with the power to amplify almost any argument. Google has become the ultimate megaphone. Even the most discredited narrative can get a toehold in cyberspace, winding its way back into mainstream discourse and assuming a legitimacy that would have long-since disappeared in a more critical, linear age.

Many advocacy NGOs have become masters at this kind of information manipulation. They've capitalized on the erosion of trust in authority, raising their profile to play an outsized role in the national debate over our environmental future. Among the most adept and well funded are EDF, the NRDC, Greenpeace, National Wildlife Federation (NWF), Center for Science in the Public Interest (CSPI), Union of Concerned Scientists (UCS) and, more recently, EWG. They've also exploited advanced analytical techniques that measure very small levels of a chemical not only in the environment, but also in human tissues and fluids. NGOs now regularly provide their own interpretations of government studies, publicizing what they claim are understatements of danger (Environmental Working Group 2005) (Environmental Working Group 2010).

When chemical traces are found in our blood or urine, at whatever level, the narrative presented by interest groups is often one-sided. For example, advanced technological analyses of water samples have been used to show the presence of miniscule amounts of drugs or agricultural chemicals at levels far below what scientists believe can cause an effect on the most sensitive animals—with an additional 100-fold or 1,000-fold level of safety built in. That's why scientists conclude that these chemicals as normally encountered in the environment are not harmful—the exposure levels are just too low to be meaningful (Centers for Disease Control and Prevention 2010). Unfortunately, articles that demonize chemicals often prompt citizens and politicians to act hastily on the belief that the presence of a chemical at any level leads inexorably to an adverse health effect.

The NRDC campaign against Alar in 1989 is the paradigmatic example of how a NGO helped rewrite the narrative on a chemical once considered relatively innocuous. The NRDC worked with CBS's *60 Minutes* to promote its report on the dangers of Alar (the trade name for daminozide), a chemical sprayed on apples to regulate their growth and enhance their color. The February 1989 broadcast, largely based on the NRDC report "Intolerable Risk: Pesticides in Our Children's Food" told an audience of some 40 million people that Alar was a dangerous carcinogen. Then NRDC's public relations firm, Fenton Communications, which has since become a giant in the PR industry

by working with environmental campaigners, lobbied other major news organizations to feature the story.

David Fenton, the PR company's founder, struck gold when he got Meryl Streep, then one of Hollywood's hottest actresses, to front the story, even though she had no special knowledge of apples or Alar. Fenton teamed up with a long-time friend, David Gelber, a producer at *60 Minutes*, which aired a hysterical feature. Streep subsequently testified before Congress and toured TV talk shows. Not surprisingly, CBS's blockbuster report sent the public into a panic. School systems removed apples from their cafeterias, supermarkets took them off their shelves and orchard owners lost millions of dollars (Rosen 1990).

Backed into a corner by the controversy, the manufacturer pulled Alar from the market after the EPA wrote in a release, "[L]ong-term exposure to Alar poses unacceptable risks to public health," although the government cited no specific study. The high-dose research on which the EPA apparently based its hasty comments indicated that the only chance of human poisoning would come if a person ate thousands of apples a day for years. Since the infamous scare, virtually every reputable scientific body and leading scientist, including the National Cancer Institute, the American Medical Association, the World Health Organization (WHO), and the U.S. surgeon general have gone on record as saying that the use of Alar on apples never posed any serious risk.

The manufacturer's decision to withdraw Alar validated what is now the standard NGO campaign model: create scares (often working hand-in-glove with activist public relations agencies, such as Fenton, and compliant journalists, such as those at *60 Minutes*) to put industry on the defensive and embarrass government officials into making rash decisions based on public opinion rather than science. That cynical cycle has only exacerbated public mistrust of both industry and government.

Reforming the Toxic Substances Control Act

Considering the tenor of the public discourse about chemicals, it is understandable why there is increasing public concern about potential risks in

our food, air, water, soil and consumer products. The major anxiety within industry—and indeed of many scientists around the world—is that the weight of evidence deliberations that are the basis for most U.S. regulations will be usurped by politics. Environmental NGOs are targeting the 1976 Toxic Substances Control Act (EPA 2010), which they hope to evolve into the country’s central chemical oversight legislation.

Concern that developing embryos, infants and children are more sensitive to chemicals than adults led to the passage of the Food Quality Protection Act (FQPA) of 1996 (U.S. Congress 1996). Under the statute, the EPA was required to evaluate chemicals at a stricter level than TSCA, defining safety as a “reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue.” Costs and benefits could be a consideration for nonfood pesticide uses, but for food use only public health could be considered. In 1998, the EPA aggressively revised its approach to include an additional 10-fold safety factor for children (EPA 1998).

The latest battle over TSCA revolves around whether the U.S. will continue to embrace a risk-based view of chemicals (but modernized to reflect scientific data on non-carcinogenic effects) or a precautionary model grounded in fear of unknown or suspected hazards. Under the act, manufacturers must inform the EPA of their intent to manufacture a new chemical and present evidence about its risks and potential benefits. Regulators must weigh the costs of restrictions against the economic benefits of keeping the chemical in commerce. The act does not require assessment of the safety of thousands of chemicals previously evaluated and “grandfathered in” when the law was passed; nor does it apply to substances regulated under other legal frameworks, such as the FDCA or the Federal Insecticide, Fungicide and Rodenticide Act.

Other than screening new chemicals and regulating the five designated ones, the execution of TSCA’s mandate is vague, partially because Congress failed to define what constitutes a *reasonable* risk of injury and how to evaluate that risk. One prominent critic, Andy Igrejas, environmental-health campaign director for the Pew Charitable Trusts, maintains that the U.S. “has no real program to regulate industrial chemicals,” as a result of TSCA’s “deep flaws”

(Case, *The Real Story Behind Bisphenol A* 2009). There is pressure from environmental advocates to extend to TSCA provisions of the Delaney clause that now exist for synthetic food additives to other chemicals, such as bisphenol A (even though BPA is not believed to be carcinogenic in humans). According to the Delaney clause, if a synthetic food additive causes cancer in test animals at any dose it must be prohibited. If more widely adopted that would amount to a problematic precautionary test; people are not typically exposed to the high doses given to laboratory rodents and if the animals get cancer that does not guarantee that humans exposed to lower doses will suffer the same fate.

EPA administrator Lisa Jackson announced that reform of TSCA was high on her list of priorities when she assumed her position in January 2009. Senator Frank Lautenberg, Democrat of New Jersey, has proposed overhauling the whole system of regulating chemicals with the introduction of the Kid-Safe Chemical Act, which would require manufacturers to demonstrate their safety in order to introduce new chemicals or keep current ones on the market (U.S. Congress 2009). A House draft version of the bill would require the EPA to maintain a list of 300 priority chemicals to investigate “based on available scientific evidence, consideration of their risk relative to other chemical substances and mixtures, presence in biological and environmental media, use, production volume, toxicity, persistence, bioaccumulation, or other properties indicating risk.”

It’s unclear from the draft bill what criteria would be used to designate a chemical as “dangerous.” The recommendations are a hodge-podge, a mix of politics and precautionary-based notions. For example, in the proposed legislation, the non-carcinogenic BPA, found safe by all pertinent U.S. agencies and foreign scientific advisory boards, is grouped in the same category as lead, asbestos, cadmium and other known carcinogens (Willhite, Ball and McLellan 2008). The major concern is that the public bias against “all things chemical” will be incorporated in ill-conceived legislation that could undermine the long-standing regulatory commitment that relies on “best available data.”

President's Cancer Panel Annual Report for 2008-2009

These contradictions were borne out in the 2008-2009 report by the President's Cancer Panel, a three-person committee that advises the White House each year on national cancer strategy (National Cancer Institute 2010). It offers a jarring insight into just how endemic this new iteration of chemophobia has become in our society.

Nearly 1.5 million new cases of cancer are expected to be diagnosed in the U.S. each year; 562,000 Americans will die from the disease. Approximately 41 percent of people in the U.S. will be diagnosed with cancer at some point in their lives. The societal costs are staggering: an estimated \$243 billion each year. The Executive Summary reads as if exposure to exogenous chemicals were the primary cause of these cancers. The report is entirely devoted to environmental factors. It claims that the proportion of cancer cases triggered by chemicals in the environment has been "grossly underestimated," warning of "grievous harm" from chemicals and other hazards and "a growing body of evidence linking environmental exposures to cancer."

The report was scathingly and bewilderingly received by many cancer and chemical experts. The panel failed to invite scientists from the FDA, EPA, NAS, NIOSH, OSHA or the National Toxicology Program (NTP) to comment on environmental chemical risk, which raised doubts about the report's independence and scientific credibility. In an analysis entitled "Cancer Report Energizes Activists, Not Policy," Reuters' Health and Science editor noted, "[T]he report from the President's Cancer Panel ... has underwhelmed most mainstream cancer experts and drawn only a puzzled response from the White House. Even members of Congress who usually are eager to show they are fighting to protect the public have been mostly silent. Cancer experts say for the most part that we already know what causes most cases of cancer and it's not pollution or chemicals lurking in our water bottles" (Fox 2010).

Michael Thun, an epidemiologist from the American Cancer Society, wrote in an online response that the report was "unbalanced by its implication" and had presented an unproven theory on environmentally induced

cancers as if it were a fact. Suggesting that the risk is much higher when there is no proof diverts attention from things that are much bigger causes of cancer, like smoking, Dr. Thun said.

The consensus among cancer experts is that tobacco and diet (obesity) are the leading preventable causes of cancer, together making up half to two thirds of all cases. Infections are believed to cause 15-20 percent of the cancers with radiation, stress, lack of physical activity and environmental pollutants causing the rest. “Maybe up to 4 percent of cancer in the Western world is caused by contaminants and pollution and yet we are chasing new, unknown causes rather than focusing on acting on what we know,” said Graham Colditz, an epidemiologist at the Washington University School of Medicine in St. Louis and an adjunct professor at the Harvard School of Public Health. “Things like this report are making it harder to move the nation to a healthier lifestyle.”

The report does acknowledge that there is no hard evidence that environmental factors play a significant role in causing cancer—200 pages in. After sensational speculation about the potential dangers of certain chemicals the report concedes, “At this time we do not know how much environmental exposures influence cancer risk.” The dearth of evidence did not stop the authors from proposing that the government actively restrict chemicals based on consumer concerns, even absent evidence of actual harm and despite the costs of such regulation.

Case Study: Bisphenol A— Precautionary Regulation

The President's Cancer Panel report contains numerous overstatements and inaccuracies, which reflect the panel's reliance on the perspective of advocates and select scientists rather than a broad representation of scientists most familiar with studies on the chemicals commented upon. One primary target about which the panel gets considerable information wrong is bisphenol A, an industrial chemical used to add strength and flexibility to many plastics and to make the epoxy resins that are used to line canned goods to prevent contamination. In the opening letter to the president, the panel notes, "bisphenol A (BPA) is still found in many consumer products and remains unregulated in the United States, despite the growing link between BPA and several diseases, including various cancers." The panelists urge the government to take precautionary measures to restrict its usage.

The controversy surrounding bisphenol A dramatically illustrates the virulence of chemophobia and the new forms it is taking. BPA is one of the most ubiquitous chemicals in the world. It has been in use for more than 50

years in the manufacture of polycarbonate plastics and epoxy resins in dentistry; in thermal paper production; and as a polymerization inhibitor in the formation of some polyvinyl chloride plastics. It is found in electronics, DVDs, car dashboards, eyeglass lenses, and microwavable plastic containers. Approximately 6 billion pounds are produced globally each year. When used as a building block in polycarbonate plastic products, BPA makes them stronger—hard enough to replace steel and transparent enough to substitute for glass. Polycarbonate can withstand high heat and has high electrical resistance. At present, alternatives for many of its uses—such as in the protective coating of metal can liners, where it does not affect taste, helps prevent bacterial contamination and extends shelf life at a relatively low cost—do not exist for most foods (Layton 2010).

Campaigns Against BPA

BPA is also one of the world's most studied chemicals—it has been subject to literally thousands of studies. In 1982, the National Cancer Institute and the National Toxicology Program cleared it as a potential carcinogen (National Toxicology Program 1982), and a review by the EPA endorsed its safety in 1988 (EPA 1988). Twenty years later, in 2008, the FDA reviewed the studies to date and declared BPA safe at estimated levels of human exposure (U.S. Food and Drug Administration 2008). A year later, in 2009, under pressure from advocacy groups that had sharply criticized the findings as an example of the Bush administration's alleged anti-science bias, the Obama Administration announced the FDA would reassess the 2008 review.

For the past four years, BPA has been under constant attack by select environmental groups, journalists and some social scientists campaigning to ban the chemical outright or restrict its use in products handled by infants and children (Case, *The Real Story Behind Bisphenol A* 2009) (Vogel 2009). The point organization for much of this criticism is EWG, which has been actively lobbying for a ban since 2007. EWG is most noted for its work lobbying for a ban of phthalates. EWG does not have any scientists with targeted expertise in plastics. That does not deter it from regularly seeding the Web with sen-

sational, simplistic and often-misleading interpretations of complex studies. For example, in November 2009, as the environmental community anxiously awaited the FDA's decision regarding BPA, EWG posted a report on the *Huffington Post* with the headline, "BPA Wrecks Sex, Fouls Food—and Probably Worse" (Shannon 2009).

The public campaign conducted by EWG and other advocacy organizations has led to thousands of stories by mainstream news organizations and on the web. The *Milwaukee Journal Sentinel* alone has published no fewer than 50 stories—for which it has won a bushel of journalism awards—excoriating the government for not restricting or banning the use of BPA. It consistently frames the issue using what can only be characterized as sensational tactics. In what it calls a "Watchdog Report," the *Journal Sentinel* warned that BPA could cause, in humans, "cancers of the breast, brain and testicles; lowered sperm counts, early puberty and other reproductive system defects; diabetes; attention deficit disorder, asthma and autism" (Milwaukee Journal Sentinel 2010)—none of which is supported by scientific studies or international regulatory agencies.

A feedback loop has developed among news organizations and select environmental groups and consumer advocates promoting the view that BPA is unsafe. In its December 2009 issue, *Consumer Reports* repeated unfounded allegations that "BPA has been linked to a wide array of health effects including reproductive abnormalities, heightened risk of breast and prostate cancers, diabetes, and heart disease" in humans—erroneous claims that subsequently turned up in the President's Cancer report but which have been rejected by the NTP, risk assessments by the FDA and the European Union. Rejecting the findings of research authorities, the magazine urged the FDA to revise its "inadequate and out of date" standards. (Consumer Reports 2009) The *Consumer Reports* article inspired panic-inducing reaction stories at ABC News, the *Los Angeles Times*, Fox News and *The New York Times*, as well as hundreds of other articles in smaller publications and on the web. The Susan G. Komen Foundation was so overwhelmed and alarmed by calls from frightened women, it consulted with a top expert in the field, Melissa Bondy, an epidemiologist at the University of Texas MD Anderson Cancer Center. "[T]here is

no evidence to suggest a link between BPA and risk of breast cancer,” Bondy concluded in a summary alert still posted on the foundation’s website (Susan G. Komen for the Cure 2010).

Considering the change in ideological complexion at the head of the FDA, ban proponents were taken aback in January 2010 when the agency announced it was standing by its 2008 conclusion that BPA is safe as used. It declared the chemical posed “negligible” or “minimal” concern for most adults and “is not proven to harm children or adults,” concluding, “[s]tudies employing standardized toxicity tests used globally for regulatory decision making thus far have supported the safety of current low levels of human exposure to BPA.” (Food and Drug Administration 2010) When asked directly if adults or children faced any real health dangers, Joshua Sharfstein, M.D., the FDA’s principal deputy commissioner, minced no words: “If we thought it was unsafe, we would be taking strong regulatory action” (National Institutes of Health 2010). While reaffirming there were no dangers, the FDA report recommended ways to limit exposure to BPA and said it is funding more studies.

In its study, released four months after the FDA report, the White House Cancer Panel ignored the FDA’s conclusion that BPA was safe for adults and infants and that families should not change their use of infant formula or food. Instead, the report cited selective and out of context elements of the FDA statement to reinforce the belief that BPA is unsafe. The panelists also claimed—erroneously—that the NTP had said “there is cause for concern” about the chemical’s link with reproductive abnormalities, when the NTP in fact concluded there was “negligible concern” for reproductive effects.

If the FDA had taken action and supported restrictions, it would have come as a shock to regulators worldwide. BPA has undergone comprehensive reviews by 10 other regulatory bodies in Europe, North America, Asia, Australia and New Zealand (Butterworth 2009). In what is considered the most comprehensive and definitive review to date, in 2006, the European Union’s European Food Safety Authority (EFSA) certified that BPA is safe for use in products handled by adults and infants (EFSA 2006).

The EFSA took up the issue once again in 2010 after the French and Danish government decided to ban BPA in food-contact products for infants and

toddlers based on what they saw as uncertainties raised by a recent report of BPA's neurotoxic effects on rodents, known as the Stump study (Stump, et al. 2010). The EFSA panel of 21 scientists consulted with international risk assessment authorities, including the FDA, Health Canada and the WHO, and conducted a comprehensive review of the Stump study and all research on BPA toxicity through July 2010. On September 30, the EFSA reasserted there is no "convincing evidence" of neurobehavioral toxicity of BPA, concluding, "[T]hese studies have many shortcomings" and are not relevant to human health (EFSA 2010).

Once again, what is most notable is that even though obligated to assess chemical exposures on precautionary grounds, EFSA has continued to find that the low-dose rodent studies are not methodologically or statistically convincing. Its conclusion: BPA is safe as used by adults, infants and pregnant women.

How does it happen that a White House panel of supposed experts glibly endorses regulating BPA in the U.S. as Europe regulates it in the belief that the EU would restrict its use under the precautionary principle—but is so sloppy in its work that it does not know that European regulators have consistently come to the same conclusion as U.S. regulators, that BPA is harmless? How does it happen that a substance consistently deemed safe by reviewing bodies and scientific studies remains in the crosshairs of campaigning journalists, politicians and environmentalists? What does this controversy suggest about how scientific decisions are made in a highly charged political environment?

Low Dose Theory

Researchers generally agree BPA is neither mutagenic nor a likely human carcinogen (Haighton, et al. 2002). There is disagreement, however, about whether the chemical presents any other danger to children or infants. The controversy results from the newer ways scientists are attempting to evaluate chemical risk. Some scientists and NGOs have zeroed in on evidence that trace levels of BPA can leach from the plastic, that this produces a laboratory response on estrogen-responsive cancer cells (Krishnan, et al. 1993). It's been

labeled an “endocrine disruptor.” Such a finding is not necessarily, or even likely, a cause for concern. As previously noted, many natural substances that alter the way the hormones in our endocrine system work are potent and present at levels comparable to or higher than BPA.

The studies on BPA do indicate serious hormonal effects on rodents when BPA is injected or consumed at levels at least 500,000 times greater than humans consume (Dekant and Völkel 2008). How meaningful are these findings for humans, who are exposed to only the tiniest fraction of the chemical injected into rats?

Chemicals tested on animals rarely have identical effects on humans at comparable dosages, and sometimes have no discernible effect because of inherent flaws in studies and significant differences between the species in biochemistry, physiology and other metabolic systems. Other doubts have been raised because of what scientists call non-reproducibility—estrogenic effects and reproductive impacts shown in one laboratory cannot be confirmed in others (Kamrin, *Bisphenol A: A Scientific Evaluation* 2004).

It’s also important to distinguish whether an experiment on BPA was carried out using oral studies or by injections. The reproducible studies have been almost all been experiments in which BPA has been administered by injection. But humans are not exposed to BPA through injections. In humans, BPA is ingested; 99 percent of exposure is through our diet. Consequently, regulatory agencies do not put much stock in tests in which a substance is introduced to subjects in a different way from that to which humans are exposed. The European Food Safety Authority, Health Canada, WHO, the FDA, the NTP and every regulatory body that has systematically assessed the risks of BPA either reject studies of injected BPA outright or gives strong preference to those in which animals receive BPA orally. While studies in which rodents were injected with BPA have shown some (but often contradictory) effects, the results from experiments in which rats receive the chemical orally have proved biologically implausible and not reproducible (Howdeshell, et al. 2008).

Why would that be the case? BPA taken orally is rapidly detoxified, first in the gastrointestinal tract and then in the liver (Doerge, et al. 2010). Enzymes

transform BPA into a water-soluble chemical known as BPA-glucuronide, which repeated studies have shown is harmless. Within a few hours of being ingested, it's not chemically active and does not accumulate in tissues. Rapidly excreted in urine, this substance has a half-life of just six hours (Völkel, et al. 2002). Even when used in dental sealants, BPA exits the system in fewer than 24 hours (Joskow, et al. 2006). Regulators are thus faced with a dilemma. The injection studies on BPA are contradictory and often were not carried out using Good Laboratory Practices (GLP); ingestion studies, when positive, have generally been of questionable quality and not reproducible; and studies on oral ingestion of BPA make it clear that BPA, taken orally, is soon rendered innocuous and excreted.

There is a common, and seemingly damning, allegation against BPA, that turns up repeatedly in media reports and even some academic studies: BPA has been found in the urine of more than 93 percent of people over six years old (Calafat, et al. 2007). That assertion even appears in the President's Cancer report.

That makes for a sensational headline, but what does it mean? Not much. Advanced bioanalysis ensures we can find many chemicals in nanogram levels even in pure water used for high-performance liquid chromatography. To put these findings in perspective, tests by the CDC have also found dietary estrogens (called phytoestrogens)—known hormone “disruptors” that occur naturally in a vast array of products such as nuts, seeds, soy, tofu, wheat, berries, bourbon and beer—in the urine of more than 90 percent of people, with some at levels 100 times higher than traces of BPA. Moreover, the miniscule amount of BPA or dietary estrogens that might somehow be found in urine are considered harmless, as it is pharmacologically inactive and doesn't bioaccumulate. The White House report got it wrong when it stated that the CDC had found biologically active BPA in 93 percent of Americans, when the CDC had actually found that 98 percent was biologically inactive (Centers for Disease Control and Prevention 2010).

Time and again, the CDC has weighed in on this point, only to be ignored by the media. “In animal and human studies, bisphenol A is well absorbed orally,” the CDC notes (citing numerous studies) in its latest report on BPA,

released in July 2010. “Finding a measurable amount of bisphenol A in the urine does not mean that the levels of bisphenol A cause an adverse health effect. ... In humans, little free bisphenol A circulates after oral absorption due to the high degree of glucuronidation by the liver. The glucuronidated bisphenol A is excreted in the urine within 24 hours with no evidence of accumulation.”

The only significant science-based question is whether a particular substance is harmful at the trace levels to which humans are exposed. The debate over BPA has been riddled with distortions over what levels might be toxic. NGOs jumped on a study from China suggesting that Chinese workers who handled BPA in bulk in unsafe conditions had lower sperm counts (Kaiser Permanente Division of Research 2009). The EWG disseminated the story and the *Los Angeles Times*, *Milwaukee Journal Sentinel* and other organizations played it up with outrageous, out-of-context headlines. But the study was extremely preliminary. Only a fraction of the workers at the plant agreed to participate in that study, which did not correct for other confounders, such as whether the workers with low sperm counts smoked (more than 68 percent of the workers at the plant smoked, and smoking is a proximate cause of low sperm count).

Incidents of occupational exposure to BPA are incredibly rare and prior research suggests that workers handling it at high concentrations and without protective equipment may not be in harm’s way (Guobing, et al. 2005). Moreover, research on workers exposed to level hundreds or thousands of times higher than consumers might face (even in extreme circumstances) provides no insight as to its potential to harm as the chemical is normally encountered. The NTP has reported “negligible concern” that men exposed at non-occupational capacities—in other words, men who are exposed to BPA from using plastic containers or consuming canned foods—would experience reproductive effects (Center for the Evaluation of Risks to Human Reproduction 2008).

Ideological Regulation

The scientific community appears divided into two conflicting camps when it comes to assessing BPA's risks. Regulatory authorities and scientists, who rely on long-established study protocols, including GLP, are on one side, and they have concluded, almost unanimously, that BPA presents no serious harm. They represent the majority, but their views are often downplayed or even ridiculed by advocacy groups and a small faction of university-based scientists who embrace precautionary notions and the low dose, endocrine disruptor paradigm. These disputes have turned acrimonious on occasion at academic conferences, where shouting matches have broken out, and in premier journals, where the shouting is in ink. Over the summer, *Nature* published a long "Letter to the Editor" by two distinguished FDA toxicologists taking the journal to task for what they claimed was "biased" reporting for trying to explain away why low-dose BPA studies are yielding contradictory results that regulators consistently find wanting (Lorentzen and Hattan 2010).

One of the major differences between the two approaches is that the studies by university scientists are hypothesis-driven: they are usually small studies asking targeted questions, designed to challenge existing paradigms. Free of regulatory responsibilities, they often trumpet their findings to a general press that is ideologically sympathetic. The majority of the state-of-the-art larger studies—that follow GLP and upon which the FDA and other regulators rely—have shown few consistent effects from BPA. The government sometimes mandates these larger GLP studies, and industry is required to fund them. That presents an easy target for critics, including activist academics, NGOs and journalists, although there is no evidence that any "industry-funded" data has been manipulated or compromised. In essence, there is a clash of cultures between academic research scientists, who are testing new hypotheses and have serious concerns about the hormonal and epigenetic (i.e. non-genetic factors that cause an organism's genes to behave or express themselves differently) effects of BPA and regulatory scientists, who must weigh a range of risks and unintended consequences before enacting or changing regulations.

These differences reappear every time a new study comes out. In 2001, the NTP released an independent study of the evidence for and against the novel hypothesis. In its conclusion, the report says, “The Subpanel is not persuaded that a low dose effect of BPA has been conclusively established as a general or reproducible finding,” although it did recommend further review (National Toxicology Program 2001). Numerous studies followed, including one by the Harvard Center for Risk Analysis (Gray, et al. 2004) (Goodman, et al. 2006). All of them raised doubts about the validity of the low-dose hypothesis and the reproducibility of findings based on tests performed on animals injected with BPA. Nevertheless, after each of these studies, the authors were attacked. Frederick S. vom Saal, an expert in animal neurobiology at the University of Missouri who has emerged as the most vocal critic of BPA, argued that these reports all failed to take into account the “latest knowledge” in endocrinology, developmental biology, and estrogen-receptor research (vom Saal and Hughes 2005).

To respond to the consensus of BPA’s comparative safety, in 2006, vom Saal coordinated a conference that brought together dozens of skeptical scientists, 38 of whom signed a statement endorsing the low-dose endocrine-disruptor hypothesis. These committed signees are the scientists noted by the President’s Cancer Panel and many media reports as “independent.” Considering the lack of dissenting viewpoints, their summary conclusion, known as the Chapel Hill Consensus Statement, was hardly surprising. It found BPA associated with “organizational changes in the prostate, breast, testis, mammary glands, body size, brain structure and chemistry, and behavior of laboratory animals” (vom Saal, et al. 2007). Using inflammatory language uncharacteristic of science, vom Saal summed up their conclusion: “The science is clear and the findings are not just scary, they are horrific. When you feed a baby out of a clear, hard plastic bottle, it’s like giving the baby a birth control pill” (University of Missouri College of Arts and Sciences 2005).

The “consensus” statement was widely disseminated in the worldwide media and led to hearings in many countries, where the debate took on a decidedly ideological edge. Public concerns sparked a review by Health Canada. When Mark Richardson, the chief scientist and head of the study, unofficially

concluded the evidence showed that the dangers of BPA were “so low as to be totally inconsequential” and compared its estrogenic effects to tofu, activists and the media, led by *The Globe and Mail* of Toronto, mounted an attack on his credibility that led to his reassignment (Mittelstaedt 2007). Months later, when the official report was finally issued, Health Canada echoed Richardson’s findings and rejected claims that BPA was unsafe. “The current research tells us the general public need not be concerned,” Health Canada declared after reviewing hundreds of studies. “Bisphenol A does not pose a risk to the general population, including adults, teenagers and children” (Government of Canada 2008).

Nonetheless, the precautionary principle is embodied in the law in Canada (and in the EU, where it is applied differently, but not yet in the U.S.). Considering the anxiety generated and absent convincing scientific evidence, Canadian officials felt compelled to ban polycarbonate baby bottles (although other infant products containing BPA were deemed safe). “Even though scientific information may be inconclusive,” Health Canada wrote, “decisions have to be made to meet society’s expectations that risks be addressed and living standards maintained.” Activists now regularly and disingenuously (or out of ignorance) cite the Canadian ban, arrived at through fear rather than based on scientific evidence, as “proof” that regulatory bodies are now finding BPA harmful.

The stage then shifted to Europe, which has slightly different precautionary standards. In a stunning turn of events, health authorities in France rejected the opportunity to follow in Canada’s footsteps. “Canadian authorities banned BPA under public pressure and without any serious scientific study,” Minister of Health Roselyne Bachelot said during an inquiry at the National Assembly in March 2009. “The precautionary principle is a principle of reason and under no circumstances a principle of emotion,” she concluded, noting, “It applies when there are no reliable studies. Here, there are reliable studies, which conclude, with current scientific data, that baby bottles containing this chemical compound are innocuous” (Rimondi 2009).

In late spring 2010, after a renewed campaign by activists using the now discredited Stump study, the French Senate and Assembly put aside the scien-

tific findings and the recommendations of its health minister and approved a ban on infant bottles containing BPA. A precautionary ban also went into effect in Denmark in July 2010. Both the French and Danish bans remain in effect even though the study that fed the concerns was dismissed as inadequate and unpersuasive in the latest EFSA review.

FDA and EPA Weigh In

In recent years, the U.S. government has committed tens of millions of dollars, and promises to spend an additional \$30 million under the stimulus bill, in an attempt to resolve remaining questions about the potential danger of BPA. In the government's first major review after the "consensus" statement, the FDA's National Toxicology Program released an extensive peer-reviewed analysis in 2008 of the various studies of BPA and again concluded there was no reason for serious concern about its effects on human reproduction or development in adults or children (NTP, HHS, and NIEHS). The NTP used the term "some concern" to characterize the possible effects of BPA on fetuses. The term has never been defined, but in practice it's been used when the agency did not consider a chemical harmful or worthy of restrictions or health warnings; in effect, scientists say, it's been used as a code phrase to suggest further study. The NTP pointedly reached that qualified conclusion because the rodent studies were not "experimentally consistent"—some showed no problems and test results could not be replicated in many instances.

The EPA subsequently funded two additional multigenerational analyses. Both studies failed to support the low-dose hypothesis. The most recent analysis, which appeared in November 2009 in *Toxicological Sciences*, a leading scientific journal, was particularly definitive. Carried out at the EPA's Office of Research and Development in Research Triangle, North Carolina, it was specifically designed to cover a wide range of BPA doses. L. Earl Gray Jr. and his colleagues concluded that BPA is an extremely weak estrogen not worthy of being called an "endocrine disruptor." BPA was found to be so weak that even at levels of exposure 4,000 times higher than the maximum exposure of humans in the general population there were no discernible effects (Ryan

2010). Gray's research mirrored findings by regulatory agencies around the world. The hodgepodge of low-dose endocrine disruptor studies is "inadequate," "not replicable," and "extremely limited" in value, Gray's team wrote, concluding, "BPA did not display any estrogenicity" (Gray Jr. 2010).

The first comprehensive FDA-sponsored study of pharmacokinetics of BPA in primates, which are biologically closer to humans than rodents, reached much the same conclusion. Among the findings of the University of Georgia and FDA researchers, published in the October 2010 issue of *Toxicology and Applied Pharmacology* (Doerge, et al. 2010):

- BPA does not accumulate in the body;
- BPA is efficiently metabolized by adult monkeys after oral exposure;
- The capability of neonatal monkeys to metabolize BPA is equivalent to adult monkeys, which suggests that neonates may not be more sensitive to the potential effects of BPA; and
- Primate results suggest that studies in rodents may over-predict health risks associated with BPA ingestion.

The head researcher, Daniel Doerge, a chemist at the EPA's National Center for Toxicological Research in Arkansas and a staff member on the EPA Science Advisory Board, supports no known horse in this race. In three papers released this year, he and his colleagues have found that newborns and infants can metabolize BPA much like adults do, that rats injected with BPA (as opposed to being fed it) overestimate human exposure and that current estimates of human exposures to BPA, which are exceedingly low, are likely to be accurate. His findings are a direct rebuke of the key assumptions underpinning the endocrine disruptor hypothesis.

In a reasonable world, the stream of comprehensive EPA and FDA reviews and studies, backed by consistent evaluations of BPA's relative safety by European health authorities, should quell concern over the low-dose, endocrine-disruptor, precautionary principle-fed hypothesis. But we don't live in a reasonable world. The renewed focus is now political. Both the House and

Senate are entertaining bills banning the use of BPA in products handled by infants, and numerous states and localities have passed restrictions, including Minnesota, Maryland, Wisconsin, Connecticut, Washington, Vermont, New York, Albany County and the cities of Schenectady and Chicago.

Case Study: Atrazine — Weighing Risks and Benefits

Farmers have been known to say that the most important invention in the history of agriculture besides the plow is the herbicide atrazine.

The odorless white powder is applied on farms to control a wide range of broadleaf and yield-robbing grassy weeds. Manufactured by the Swiss-based agrichemical company Syngenta and licensed in the United States since 1958, atrazine is part of the chemical family of triazine herbicides used on many fruits and vegetables, including nuts, citrus and grapes. It was among the first of what are called “selective herbicides,” which destroy weeds that would otherwise choke a crop and starve it of nutrients, but do not harm the crop itself. In combination with other products, it can help boost the efficacy of other weed killers. Yet it is considered so comparatively gentle by farmers that it can be applied even after a crop’s first shoots appear above the ground.

Almost half of the atrazine in use is applied in the U.S., where it is used on dozens of crops, including more than half of the country’s corn crop, 90 percent of its sugar cane and two-thirds of its sorghum. More than 160 million pounds

of atrazine is produced annually. Although regulatory agencies have consistently determined that atrazine is safe as used, it has come under relentless attack by anti-pesticide groups and some university scientists, who are convinced that it poses potential health threats for aquatic animals such as frogs and, by extension, to humans. They are concerned that it might affect human reproduction and hormonal activity—that it's an “endocrine disruptor”—making it equivalent to a ticking chemical time bomb.

Atrazine fits a variety of farming systems. It is credited as being a key factor in the transformation of farming from the relatively low-yield, massively labor-intensive activity that prevailed into the first half of the 20th century and through the dust-bowl Thirties into the advanced, high-technology industry it has become today. It is the most widely used herbicide in conservation tillage systems, which are designed to prevent soil erosion. It has become a critical tool in the no-plow revolution that is helping to cut carbon pollution.

Atrazine conserves water because the stalks, husks and other crop residue from previous harvests are left on the ground and the soil is not plowed up. Less plowing means less use of oil-hungry farm machinery. Not turning over the earth to kill weeds also keeps huge amounts of carbon dioxide trapped in the ground, limiting CO₂ emissions. According to the U.S. Department of Energy, the adoption of no-till and other conservation methods around the world could result in the recovery 40-50 billion tons of carbon—about two-thirds of the carbon lost over time as a result of conventional agricultural practices, which is remarkable. As a reference, it's estimated that approximately six billion tons of carbon are released from fossil fuels each year in the United States alone (U.S. Energy News).⁴

Some analysts estimate that 10 to 40 percent of sugar cane yield could be lost without atrazine. An EPA study concluded that atrazine boosts yields by 6 percent or more, saving corn farmers as much as \$28 per acre—more than \$2

⁴ According to the DOE, “Researchers estimate that the extensive adoption of no-till agriculture, diversified rotations, cover crops, fertility management, erosion control and irrigation management can lead to the recovery of two thirds of the carbon that has been lost from the soil due to conversion of native ecosystems to agriculture and the use of conventional management practices.”

billion in direct economic benefits, which could be the difference between solvency and bankruptcy for many (EPA 2002). Another study looking at combined data from 236 university cornfield trials from 1986 to 2005 found that crops treated with atrazine yielded an average of 5.7 bushels more per acre than those treated with alternative herbicides (Fawcett 2008).

Not everyone agrees with those estimates, however. Tufts University economist Frank Ackerman, who has campaigned for tighter restrictions on atrazine and other chemicals and works closely with atrazine critics, wrote a controversial analysis in 2007 challenging the EPA study, claiming atrazine increases yields by as little as one percent (Ackerman 2007). In contrast, a recent analysis conducted for Syngenta by University of Chicago economist Don Coursey concluded that a ban on atrazine could cost corn farmers between \$26 and \$58 per acre. He estimated that as many as 21,000 to 48,000 farm and farm-related jobs could be lost, and the negative economic impact to the U.S. economy could reach as high as five billion dollars a year (Coursey 2010).

Studies and Regulation

Atrazine is one of the most assessed and regulated agricultural chemicals in history. There have been more than 6,000 studies on the herbicide, compared to the 100 to 200 safety studies generally required by the EPA before registering a product. It has long been considered safe because it has a short half-life, does not bio-accumulate in organisms, and reportedly induces abnormalities and deformities only at very high doses (UK Rapporteur Monograph 1996) (Solomon, et al. 1996).

Atrazine has been approved as safe in regulatory reviews throughout the world. No country has ever discontinued the use of atrazine based on evidence of health dangers—including the member states of the European Union. In 1996, when the EU first formally evaluated atrazine, its scientific reviews were positive: “It is expected that the use of atrazine, consistent with good plant protection practice, will not have any harmful effects on human or animal health or any unacceptable effects on the environment,” the regulators concluded (UK Rapporteur Monograph 1996). However, in 2003, faced with

arguments that there were lingering uncertainties about the hidden dangers of chemicals, EU officials reexamined the evidence under the precautionary principle. Although they could find no evidence that atrazine caused any harm, EU officials eventually concluded that water-monitoring data were insufficient to guarantee that trace levels of atrazine in water would not surpass the agreed-upon level that had been set by EU member states for all pesticides based on precautionary arguments, not proof of harm. Atrazine is not on any list of banned chemicals and could be re-registered if the necessary monitoring data could be provided to show that it was found in drinking water at the levels deemed safe by the EU (Brussels: Health and Consumer Protection Directorate-General 2003).

Other regulatory bodies, even those that incorporate precautionary standards, have not recommended that it be banned. In 2004 Canada, which has restricted BPA under a narrow interpretation of the precautionary principle, found atrazine safe (Health Canada 2004). The World Health Organization concluded in 1999 that atrazine does not cause cancer in humans (International Agency for Research on Cancer Monographs 1999) and reaffirmed the finding of its relative safety in 2010. Based on recent data reaffirming the relatively innocuous hazard profile of atrazine, the WHO dramatically revised the exposure threshold level, setting it 100 times higher than the obsessively cautious EU. (World Health Organization 2010). After an extensive review of the data in 2008, the Australian government concluded that it “continues to be satisfied that [atrazine] can be safely used ... subject to those conditions outlined on product labels” (Australian Pesticides and Veterinary Medicines Authority 2010). In 2010, faced with another claim that atrazine may be associated with birth defects, the Australian government examined the latest research and reaffirmed its safety designation. It wrote on its Chemicals in the News website:

“Every year, a number of epidemiological studies describing correlations between certain human health or environmental findings and pesticide use are published. Because of the relatively low rate of occurrence of birth defects, epidemiological

studies of this type offer some useful information and hypotheses. In the regulatory context, any causal link has to be established by more extensive investigations and targeted follow-up studies” (Australian Pesticides and Veterinary Medicines Authority 2010).

Atrazine has faced the most intense scrutiny in the U.S., where it has been almost continuously evaluated for decades. Although regulatory authorities that rely on long-established study protocols consistently had concluded that it presents no serious harm as utilized, aggressive campaigns by anti-chemical NGOs such as the NRDC, EWG, and the Pesticide Action Network (PAN) prompted another review in 2005. After one of the most intense analyses of any substance in history, the EPA formally relicensed it in 2006, declaring it safe when properly used.

Ban proponents, emboldened by the EU action, did not give up, however. The NRDC had sued the EPA in 2004 under provisions of several federal laws that the group claimed should have long ago led to a ban, but it eventually lost. When the Obama administration took office in 2009, the NRDC saw an opening to again press its case. In August of that year, it issued a scathing, well-publicized critique, accusing the agency of ignoring the presence of atrazine in drinking water and in natural watersheds across the Midwest (Natural Resources Defense Council 2009). The media gave the report enormous attention, reinvigorating advocacy blogs and stirring politicians.

In October 2009—barely three years after the EPA had completed one of the most exhaustive scientific investigations of a commercial product ever undertaken—the agency announced it would evaluate atrazine once again, citing the NRDC report as its reason. “Our examination of atrazine will be based on transparency and sound science, including independent scientific peer review,” said the head of the *Office of Prevention, Pesticides and Toxic Substances* (EPA 2009). The EPA subsequently convened a series of “scientific advisory panels” (SAPs), composed of yet another team of independent scientists, to reexamine the chemical on an accelerated schedule.

Harm Versus Risk

Atrazine is one of many hundreds of compounds that can be detected in water. Every year an estimated 495,000 pounds of the herbicide become airborne and fall with rain, sometimes hundreds of miles from the source. Although it breaks down quickly, it has nonetheless been detected at infinitesimal levels—measured in parts per billion (ppb)—in lakes, streams and other waterways as well as in drinking-water systems in agricultural areas.

Does atrazine at the residue levels found in drinking water in the U.S., Europe and elsewhere pose genuine threats to human health, as is sometimes reported? The controversy revolves around perceptions of chemicals and risk. The mere presence of a compound in water does not constitute a threat. Scientists have long used the “weight of evidence” approach to assess potential toxicity, which requires balancing complex and often conflicting evidence. They attempt to discover the exposure level at which a chemical does not harm an animal—the “no effect” level—and then set human safe exposure standards that are tens, hundreds or thousands of times lower than this “no effect” amount. This built-in safety cushion ensures with a huge margin that no one is exposed to harmful levels of a regulated substance. This is the ultra-high threshold standard used by the EPA and regulatory bodies to assess chemicals, including atrazine.

The gap between the public’s perception of harm and scientific determinations of risk is often significant, as a 2008 “investigation” by the Associated Press that went awry illustrates. In a widely circulated article, the news organization found a vast array of pharmaceuticals in the drinking water of at least 41 million Americans. That investigation touched off a panic of sorts in New York City, long proud of its pristine drinking water, and prompted a study by the city’s Department of Environmental Protection. Released in May 2010, the city report indeed noted that investigators found traces of chemicals—but the levels were harmless, measured mostly in the parts per trillion (New York City Department of Environmental Protection 2010). One part per trillion is equivalent to one drop of water in 26 Olympic-size swimming pools, officials noted. “Just because you detect something doesn’t mean that it’s a problem,” said Cas Holloway, commissioner of the DEP (Saul 2010).

Each regulatory body sets its own exposure standard for the annual average concentration of a chemical. The standards are somewhat arbitrary. The EU sets the cut off for any agricultural at 1 ppb regardless of its chemical properties or hazardous potential. The U.S. EPA sets the atrazine standard at 3 ppb based its classification as a carcinogen, which scientists now believe it is not. Canada's standard is 5 ppb, the United Kingdom's is 15 ppb and Australia's is 40 ppb. In October 2010, after an extensive review of the various international standards and the latest scientific data on atrazine, WHO concluded its standard was far too restrictive, and revised it to 100 ppb (World Health Organization 2010).

On occasion, atrazine has been detected in drinking water in various communities at very low concentrations. A 2006 U.S. Geological Survey reported that approximately 75 percent of untreated stream water and about 40 percent of all groundwater samples from selected agricultural areas from 1992-2001, mostly in the corn-growing Midwest, contained miniscule traces of atrazine that occasionally spiked for short time periods at over 3 ppb (Gilliom 2006). Some NGOs cited the report in sensational news releases as evidence of atrazine's dangers. But that is not what the study showed, according to scientists. It concluded that "[C]oncentrations of pesticides detected in streams and wells were usually lower than human-health benchmarks, indicating that the potential for effects on drinking-water sources probably is limited to a small proportion of source waters."

The EPA's 3 ppb annual standard for treated drinking water was derived using a one thousand-fold safety factor that sets a level shown to have no health effects in laboratory animal studies. To put this in perspective, it is estimated that even if a person were to drink thousands of gallons of water containing 3 ppb of atrazine every day for a lifetime, he would still not be exposed to amounts shown to have effects in lab studies. Said in another way, the 2006 survey found miniscule erosion in the huge safety cushion. Using the standards in place in the U.S., Canada, Australia or under the new WHO guidelines, the concerns expressed by NGOs appear alarmist

Under an agreement with the EPA, Syngenta conducts weekly testing during the growing season of any drinking-water system that has been found to

contain annual atrazine and metabolite levels above 2.6 ppb (which is equivalent to an annual atrazine level of 1.6 ppb). In general, the already low levels of the herbicide found in water have been trending down over the course of the last 10 to 15 years. According to the EPA, concentrations in raw water declined significantly between 1994 and 2006 at 103 frequently monitored sites (Sullivan, et al. 2009). However, in its 2009 report, the NRDC crunched the raw data and found that three local water systems—two in Illinois and one in Indiana—in previous years had, on occasion, temporarily exceeded the 3 ppb EPA limit by fractional amounts. In each of the three cited cases, the annual averages in these communities did not exceed the EPA’s 3 ppb annual limit.

Those findings, noted in press releases and widely disseminated, created the misleading belief that these drinking water systems were somehow unsafe. That’s not the case. The EPA was aware of the occasional spikes. Based on decades of tests on atrazine, it did not consider these occasional spikes a safety threat for either short-term (acute) or long-term (chronic) potential exposure. However, in its sensational report, the NRDC characterized the spikes as “particularly alarming,” claiming that “potential adverse effects [are] associated with even short exposures to atrazine” (Natural Resources Defense Council 2009)—an opinion, while sensational and widely circulated, has not been confirmed in any study or accepted by the EPA. And again, in the context of the latest scientific data, as incorporated in the new WHO standard, the NRDC’s position comes across as alarmist.

Steve Bradbury, deputy director in the Office of Pesticide Programs at the EPA, said the monitoring program has never found atrazine levels approaching the 90-day or one-day maximums (Souder 2009). A cumulative risk assessment for triazine pesticides (the family of chemicals that includes atrazine) published by the EPA in 2006 concluded, “Risk assessments for cumulative exposures to triazine residues via drinking water based on currently registered uses of atrazine and simazine are not of concern” (USEPA Office of Pesticide Programs, Health Effects Division 2006).

The “Endocrine Disruptor” Hypothesis Controversy

As in the case of BPA, atrazine’s comparatively benign toxicological profile has long posed a challenge for its critics. University of California herpetologist (research focus on amphibians) Tyrone Hayes is the most ardent. The Berkeley professor began studying atrazine in the 1990s with research funded by Syngenta, as part of its due diligence. Hayes and the company parted ways in the late 1990s. He claims he came to suspect that atrazine was interfering with the natural production of hormones, and he decided to pursue his studies independently.

In 2002, Hayes published a study that ban proponents had been hoping for. His team focused on amphibian populations, which have been in worldwide decline for decades, baffling scientists. In lab experiments that exposed clawed frogs to lower doses of atrazine, the researchers produced males with ambiguous genitalia and squeaky, soprano-like croaks—hermaphrodites. “We hypothesize that atrazine induces aromatase [a protein that spurs the production of the female hormone estrogen] and promotes the conversion of testosterone to estrogen,” the Hayes team wrote (Hayes, et al. 2002).

Hayes’s study set off an immediate firestorm. It was released at the same time as another team, in a much larger study funded by Syngenta but also operating independently, found no meaningful link between atrazine exposure and abnormalities. Keith Solomon of the University of Guelph, Ontario, Canada, found that lower levels of atrazine did not induce aromatase, a result that, if true, would undermine Hayes’s conclusion (Renner 2002). The controversy, which persists today, was fully engaged.

Whereas precautionary thinking is easy to grasp and plays into our instinctual fear of the unknown, the concept of relative risk is very hard for most nonscientists, including many journalists, to get their minds around. Branding any chemical as a toxic “endocrine disruptor” is about as useful as describing a car as “fast.” Relative to what? Under what conditions? The question for regulators remains: how much of a substance causes a deleterious effect? To put this in perspective, vitamin D—an essential vitamin for life—has about

the same toxicity as arsenic. The 2005 Dietary Guidelines for Americans recommends that healthy older adults consume 1000 IU/day, whereas in adults, taking 50,000 IU/day for several months can produce toxicity. This 50:1 ratio would surely confound regulators, if the chemical were not essential to human life.

Knowing the effect and the dose at which that effect can occur is the evidence-based standard used by the EPA to regulate chemicals. The precautionary principle, on the other hand, asks only for effect and then demands action without the context of exposure. The only significant science-based question is whether a particular substance is harmful at the trace level at which it is present in the human body. Many synthetic chemicals labeled endocrine disruptors are millions of times less potent than estrogen or testosterone and simply do not have the “punch” to affect the endocrine system very much. For atrazine, the relevant factor is potency relative to estrogen or testosterone. Studies that apply classic risk analysis have consistently shown that “a risk to human health [from atrazine is] essentially nonexistent” (Cooper, et al. 1996 is one of numerous studies).

The case against atrazine rests largely on the integrity of the central body of research by its chief critic, Dr. Hayes. For example, a widely circulated joint polemic issued in January 2010 by the Land Stewardship Project and the Pesticide Action Network cites Hayes more than 50 times and includes a question-and-answer section with him in which he outlines his allegations (Land Stewardship Project and Pesticide Action Network 2010). Although his reports have been widely criticized, no mention is made of alternate perspective, conveying the false impression that Dr. Hayes’s views are widely embraced by mainstream scientists.

Many independent scientists have raised doubts about the reliability of his data and his conclusions, viewing him more as an activist than an objective researcher. “Atrazine has been used widely in South Africa for the past 45 years, and our studies showed that *Xenopus* [a genus of highly aquatic frogs native to Sub-Saharan Africa] are doing equally fine in agricultural and nonagricultural areas,” zoologist Louis du Preez of North-West University in South Africa noted in response. African clawed frogs do not appear to be suffering

from the herbicide in their native habitats. “If atrazine had these adverse effects on *Xenopus* in the wild, surely we would have picked it up by now” (Biello 2010).

The EPA and scientists on the government’s independent SAPs have doggedly tried to replicate Hayes’s findings, but to no avail. In 2005, the agency published a 95-page white paper, concluding that his work and many other studies drawing similar conclusions about atrazine’s impact on amphibians were “scientifically flawed.” Anne Lindsay, then the deputy director of the Office of Pesticides, testified that the EPA “has never seen either the results from any independent investigator published in peer-reviewed scientific journals or the raw data from Dr. Hayes’ additional experiments that confirm Dr. Hayes’ conclusions.” According to Lindsay, “The existing data are insufficient to demonstrate that atrazine causes such effects [aromatase induction]” (Statement of EPA’s Anne E. Lindsay, Minnesota House of Representatives 2005).

The controversy did not fade, however, as advocacy groups continued to cite Hayes’s findings and press regulators to ban atrazine. Facing intense public scrutiny stirred by the media, the EPA required Syngenta to fund extensive additional independent laboratory studies carried out in two separate labs in the United States and Germany—the most extensive reviews ever undertaken on atrazine. Both studies refuted Hayes’s conclusions. Biologist Werner Kloas of Humboldt University in Berlin found no impact on clawed frogs at concentrations comparable to those investigated by Hayes. He questioned the single exposure level used by Hayes in his study and the lack of measurement of female hormone levels in the affected frogs. Kloas’ findings are particularly noteworthy because he has publicly expressed his view that a chemical should be banned for precautionary reasons if there is evidence, however incomplete, questioning its safety (Biello 2010).

After a SAP review of all the data, in 2007, the EPA concluded, “There is no compelling reason to pursue additional testing” (EPA 2007). But that definitive assessment did not deter critics. Although the two Syngenta-funded studies were conducted under the strictest application of EPA’s GLP Standards and were thoroughly audited and inspected data point by data point by the EPA, advocacy groups dismissed them as inherently not credible—as

they have all studies in which the industry participated or funded.

That sweeping denunciation illustrates a lack of understanding of the process of evaluating and approving chemicals, notes Amy Kaleita, an agricultural and biosystems engineer at Iowa State University. Chemical companies fund large-scale studies not to mollify the media but because they are necessary to meet federal guidelines. In the case of atrazine, the Federal Insecticide, Fungicide, and Rodenticide Act places the burden of proving safety on pesticide companies. For a chemical such as atrazine to be approved, it must undergo a battery of tests designed by the EPA and often carried out by independent laboratories, which follow rigorous, internationally recognized Quality Assurance Protocols. The data is available to EPA auditors, who often review the study methodology and conclusions in fine detail. If the EPA determines that the study protocol is in any way deficient, it requires companies to fund additional tests.

By contrast, the peer review process is not very efficient in sorting out quality from bad peer-reviewed papers. Journal articles do not require editorial oversight or government audit. A manuscript often contains only a few paragraphs explaining the methodology behind the study and little information, if any, about quality assurance procedures. Reviewers rarely have access to the raw data summarized in the paper, and study authors decide for themselves whether to respond to reviewer comments and questions, let alone dialogue with them. Atrazine, Kaleita says, highlights “[t]he absurdity of dismissing industry funded studies in favor of peer review.” (Kaleita, 2010)

Hayes’ work has been peer reviewed for journal articles, but the data remain in a black box to regulators and independent scientists. Because of the storm of controversy fanned by the NRDC and other advocacy groups, in 2008 the Australian government’s Department of Environment, Water, Heritage and the Arts reviewed all of Hayes’ studies. Its conclusion: “Atrazine is unlikely to have an adverse impact on frogs at existing levels of exposure” (Australian Pesticides and Veterinary Medicines Authority 2010). That same year, in experiments that closely replicated Hayes’s study outline, endocrinologist Taisen Iguchi at the Okazaki Institute for Integrative Bioscience (Japan) and colleagues raised tadpoles in various concentrations of atrazine and found no

hermaphroditic frogs (Oka, et al. 2008). After reviewing the data, endocrinologist Robert Denver of the University of Michigan, well-recognized for his independence, commented that the experiments “appear to be carefully executed and the data thoughtfully interpreted. Overall, this appears to be a sound study that does not support the view that atrazine adversely affects amphibian gonadal development through an estrogenic action” (Renner 2008).

Keith Solomon, by then head of the Centre for Toxicology at the University of Guelph, reviewed more than 130 recent studies on atrazine for *Critical Reviews in Toxicology*, a well-regarded international journal. The team’s conclusion, published in 2008: Most studies found atrazine had no significant effects, and even in cases where effects were found, they were not substantial enough to warrant concern:

“We have brought the results and conclusions of all of the relevant laboratory and field studies together in this critical review. . . . Based on a weight of evidence analysis of all of the data, the central theory that environmentally relevant concentrations of atrazine affect reproduction and/or reproductive development in fish, amphibians, and reptiles is not supported by the vast majority of observations. The same conclusions also hold for the supporting theories such as induction of aromatase, the enzyme that converts testosterone to estradiol. For other responses, such as immune function, stress endocrinology, parasitism, or population-level effects, there are no indications of effects or there is such a paucity of good data that definitive conclusions cannot be made” (K. Solomon 2008).

Although a massive meta-analysis published in fall 2009 raised some concerns about the effects of atrazine, it pointedly noted that Hayes and only Hayes has found that atrazine increased aromatase and that no study has found it affects vitellogenin levels, a protein that should be present if atrazine was seriously affecting the endocrine system. Its conclusion: “These data do not support the hypothesis that atrazine is strongly estrogenic to fish” (Rohr and McCoy 2010).

Most recently, in March 2010, Hayes was the lead author on a paper published by the National Academy of Sciences arguing that atrazine demasculinized frogs throughout all life stages, from tadpole to adult, when they were exposed to a single dose below 3 ppb. Hayes and his team speculated that the atrazine was absorbed through the frogs' skin and turned on a gene that in male frogs should stay off—it converted testosterone into estrogen, flooding the frog's body with the wrong chemical signal (Hayes, et al. 2010). No other research team, independent or industry funded, has found similar effects. Australian officials reviewed the new study, found it wanting, and said there was not sufficient evidence to reconsider its current conclusion that atrazine is safe as currently used (Australian Pesticides and Veterinary Medicines Authority 2010).

The EPA has been eager to review the data from Hayes' studies, but the Berkeley scientist has steadfastly refused to cooperate with regulators. After years of frustration, in a May 2010 letter, the agency's Donald Brady, director of the EPA's Environmental Fate and Effects Division, Office of Pesticide Programs, issued a highly unusual rebuke to Hayes in a response to an inquiry from Illinois state representative Dave Winters, who had contacted the EPA after the Berkeley scientist testified before the state legislature urging a ban on the pesticide:

“As with most reviews conducted by the EPA, the analysis of data and studies is not limited to a single individual [at EPA] but rather involves interdisciplinary scientific teams and multiple rounds of peer review. You [Winter] asked whether EPA was in agreement with Dr. Hayes' findings. . . . I regret that the EPA science staff in the Office of Pesticide Programs' EFED could not properly account for the sample sizes and study design reportedly used by the Berkeley researchers. As a result, we were unable to complete any independent analysis to support the study's conclusions” (Letter from U.S. EPA's Donald Brady to Illinois State Representative Dave Winters 2010).

One would think that questions raised about Hayes' studies by internationally respected toxicology laboratories and regulatory agencies would

make headlines at least comparable to the scare stories that regularly appear after the publication of each of his controversial papers—but they didn't. Why have journalists refused to provide a balanced perspective on atrazine in particular and chemicals in general? Simply stated, many reporters are poorly schooled in science. They often do not have the sophistication or inclination to apply weight of evidence criteria or critically parse science from ideology. While new claims that one product or another contains harmful chemicals often results in a sensational front-page story, because of the journalist's default mindset, a study that shows a chemical is safe or has few effects is often ignored or relegated to the back pages. What is the news value in the headline "Atrazine Found Safe; Scientists Conclude Fears Overblown"?

A Precautionary Future?

The scientific evidence strongly suggests that atrazine does not present a serious danger to aquatic wildlife, let alone humans. Unable to make headway on the science, atrazine opponents have turned to politics and litigation. Lawsuits have been filed against Syngenta and other corporations that market and manufacture products containing atrazine. Farmers face ongoing activist campaigns intended to pressure U.S. regulators into adopting more precautionary policies. If the EPA imports and implements this precautionary model, atrazine and other chemicals found safe by classic weight of evidence risk assessment studies would be subject to what would amount to a political review of their acceptability. Such a seismic shift in regulatory standards could lead to restrictions based on suspicions and fears rather than scientific evidence. Trade-offs, such as the higher food costs and the damage to America's farming economy and international competitiveness that a ban would inflict, could be downplayed or ignored. If the precautionary view prevails, the unintended consequences could include more soil erosion, less sustainable farming, more environmental degradation—and a hungrier world.

Implications for Public Health

Policymakers use what is called risk-risk analysis to evaluate chemicals. They consider two key questions. At what levels could a substance cause harm? What would be the possible unintended consequences if a useful chemical were pulled off the market? The only justification for banning BPA or any chemical would be if it could be shown, based on empirical science, that current risks outweigh established benefits.

Benefits of a Chemical Exceed Risks

When asked in January 2010 whether the low estrogenic impact of BPA warranted further restrictions, FDA Deputy Commissioner Sharfstein responded as a scientist, carefully balancing costs and benefits. “FDA does support the use of bottles with BPA because the benefit of nutrition outweighs the potential risk of BPA,” he said. (Strictly speaking, the FDA does not consider benefits in its analyses of food packaging, like polycarbonate containers;

packaging should be approved as long as it meets safety standards and regardless of the benefits of the product it contains.) As he noted, restricting BPA could have the opposite effect; its benefits would be lost while resources that could otherwise be devoted to addressing established health risks would be wasted on trying to eliminate low-potential risks.

It is important to do risk-benefit and risk-risk analyses—balancing the actual and potential risks of various chemicals with their utility against potential harms. But reflexively responding to public or NGO fears by banning or otherwise limiting the use of certain chemicals that have not been demonstrated to pose actual risks to humans will not improve public health. In some cases, an untested chemical may end up replacing a relatively innocuous substance, such as BPA. Undoubtedly some replacements could end up causing actual harm while the original chemical only posed theoretical harm based on experiments using animals in high-dose studies. Some regulations do not address actual scientific and health risks, but have been put in place almost solely in response to advocacy campaigns.

For example, the accumulation of oil in the Gulf of Mexico in the wake of the BP disaster has led to widespread concerns that fish are contaminated while tests indicate only limited areas have been seriously affected. People just can't shake their fear of chemicals. The problem has been encouraged in part because of a history of government "consumption advisories," which warn the public about eating fish containing low levels of chemicals, such as PCBs or mercury, for which little evidence exists that they cause harm to humans at low levels. In general, the health benefits of eating fish, particularly in preventing the nation's biggest killer, heart disease, are demonstrated and significant, far outweighing the miniscule potential dangers (Mozaffarian and Rimm 2006).

The paradigmatic example of an overreaction is what happened to DDT, the insecticide targeted by Rachel Carson. DDT remains the totemic villain of the environmental movement, but it has saved more lives from malaria and other insect-borne diseases than any other chemical. In retrospect, the ban on DDT has proven to be a mistake of tragic proportion. In the early 1960s, several developing countries had nearly wiped out malaria. After they stopped using the insecticide, other control methods had only modest success and ma-

laria came raging back. In one of many examples, in Sri Lanka (then Ceylon), DDT spraying had reduced malaria cases from 2.8 million in 1948 to 17 by 1963.

After spraying was stopped in the wake of the uproar after the publication of *Silent Spring*, the number of cases exploded to 2.5 million. Malaria still kills about one million people a year, mainly children, and primarily in Africa, despite the decades-long effort to eradicate it without DDT. Many scientists and some environmental groups, including the Sierra Club and the EDF, have recently urged that the use of pesticide be reconsidered, because its effectiveness is unrivaled and it causes minimal collateral damage when properly applied. In 2006, after millions of preventable deaths, the World Health Organization reversed course and endorsed the use of the insecticide as one effective way to control malaria (Roberts 2010).

Given the state of the science at the time Carson wrote her book, one might generously make the case that her concerns about the potentially unknown effects of synthetic chemicals on human health were not unwarranted. Some key facts were unclear. But after four decades chasing the potential risks of DDT and certain other chemicals without measurably improving world health, and in some cases degrading it, her followers in the environmental movement bear the responsibility of wasting billions of dollars and destroying millions of lives.

Risks of Replacement or Amelioration Exceed Benefits

There were also other unintended consequences of banning DDT. At the time of the ban, William Ruckelshaus noted that methyl parathion would be the primary replacement. That decision was a lethal mistake. After several deaths linked to the chemical, the EPA in 1999 acknowledged that parathion is “hazardous to workers,” even to those wearing protective clothing, and accepted voluntary cancellation of many of its registered uses. The EPA, when confronted by scientifically naïve if well-meaning activists, had put expediency over saving lives.

The effort to remove asbestos from the walls of schools has addressed dangers but created others. Asbestos had been shown to cause lung cancer and mesothelioma in workers who had installed it (National Cancer Institute 1995). When asbestos was found in many public buildings, widespread concern erupted (Mossman, et al. 1990). The EPA jettisoned traditional risk analysis based on quantitative levels of exposure. Under the Asbestos Hazard Emergency Response Act of 1986 (U.S. Congress 1986), the EPA required all public school districts and private schools to inspect school buildings for asbestos and develop amelioration plans in a timely fashion. Because school districts, fearing suits, took the directive as an order for removal, in effect the EPA took the expensive and potentially dangerous position that the presence of any asbestos in any part of a school constituted an unacceptable hazard. As the EPA now notes on its website, “intact, undisturbed asbestos-containing materials generally do not pose a health risk.” Although the EPA now says removing the asbestos could cause more harm to workers and the general public than leaving it in place, NGOs and tort lawyers continue to harangue public officials to remove all traces of asbestos, regardless of the financial or environmental costs.

The movement to replace chlorine with chloramine has also proved misguided in some cases. Chlorination reduces microbial agents of disease. Environmental activists in Washington, D.C., citing high-dose animal studies on rats and mice, claimed it was harmful and had it removed from the water system (International Joint Commission 2003). There is no question that high dose chloroform can cause liver damage and is a precursor to liver cancer, but to suggest the trace levels cause cancer in humans is irresponsible and incites needless public fears. Moreover, chloramine causes the lead scale on pipes to dissolve into the water, creating a genuine neurotoxic hazard (Switzer, et al. 2006). Thus, a hypothetical danger was replaced by a real risk.

A campaign by consumer groups to remove diacetyl, a natural byproduct of fermentation found in butter, from artificially flavored buttered popcorn after it was found to cause a rare, serious lung disease in a small number of production workers who inhaled it in large quantities has led to unintended consequences. The European Food Safety Authority has evaluated its health

effects on popcorn-eating consumers and found it safe (EFSA 2004). Instead of focusing on the actual threat, the occupational hazard, many activists warn consumers in overheated Web posts to be suspicious of scientific assertions that eating popcorn flavored with diacetyl is safe. Why? Because the FDA and even physicians use lax standards in evaluating chemical exposure, says the Environmental Working Group. “No one knows how many chemicals with potential dangers lurk in the everyday objects we use and foods we eat,” it writes in an ominous story on diacetyl (Environmental Working Group 2010). Before its campaign against BPA, the *Milwaukee Journal Sentinel* focused its ire on diacetyl. “Snack could be toxic,” it sensationalized in a headline in one of numerous stories. In fact, the only consumer case known to date involves one Colorado man who reportedly ate at least two bags of buttery microwave popcorn almost daily for more than 10 years was diagnosed with the same disorder (Rutledge 2007). Facing the prospect of a consumer backlash, manufacturers began replacing diacetyl with an untested substitute, pentanedione. Now new studies show pentanedione is worse than diacetyl, which is actually harmless unless abused. (Hubbs, et al. 2010)

Psychology of Risk Perception

In the face of human irrationality and recklessness, can anything be done to restore balance to the discourse about chemicals? Why are so many people, who are educated and otherwise rational, so deathly afraid of chemicals? Reporters do not take to the cyberwaves to expound on the latest discovery that fruits and vegetables are nutritious and safe. It’s bad news, all the time, and it creates paranoia and chemophobia. As the New Jersey mother mentioned in the Introduction, Pamela Davis, remarked, “Once you’re aware of one thing it just spreads and you start questioning everything. You can drive yourself absolutely crazy trying to keep your baby healthy.” But even the relentless noise of the 24/365 media machine cannot completely account for the persistent fear that even the tiniest concentration of a synthetic chemical poses serious dangers. Clearly, our minds have a difficult time weighing rational versus irrational risks.

By now most people are familiar with the sadly comical DHMO scare. A controversy erupted in the 1990s when it was circulated on the Internet that the chemical dihydrogen monoxide had been linked to a range of medical and environmental problems, including excessive sweating and vomiting, with confirmed reports that it had been found in tumors of terminal cancer patients. A website, www.dhmo.org, documented its many dangers: It's a ubiquitous chemical and a major component in acid rain that could cause severe burns in its gaseous state, prove fatal if accidentally inhaled, contribute to erosion, and decrease the effectiveness of automobile brakes. There were proposals to "ban this toxic substance" in Australia and in localities in the United States. For the scientifically literate, of course, DHMO is the chemical formula for water. The biggest driver of fear is the unknown and that's what some activists prey upon, be they from NGOs, academic laboratories or social networking sites.

There is also a gap between perceived and actual risk. Risks that are unfamiliar or under someone else's control or are hidden—How much pesticide residue is on my child's broccoli?—are considered far more dangerous and frightening than perceivable hazards, even when they are less threatening. Former professional football coach and broadcaster John Madden refuses to fly but regularly drives cross-country in his trailer home, which is a more dangerous way to travel. As the science journalist David Ropeik has written, it's helpful to acknowledge that the process of assessing risks is not logical. People make mental shortcuts to deal with information overload, the challenge of processing conflicting risks. For example, those whom he calls "pure food obsessives" believe that "everything God (or Nature) designed is good for you." They often default to irrational beliefs, even to their peril. He cites the case of people who drink raw milk despite evidence that the "all natural" version occasionally contains deadly *E. coli* mixed with a daily dose of calcium (Ropeik 2010). For whatever reason, many people are hard-wired to believe that risks in nature are somehow less threatening than the ones created by man.

Trust in Scientists and Science

Public anxiety over perceived environmental risks threatens to over-

whelm sound scientific analysis, leading to poor public policy decisions and creating a serious obstacle to innovation and the necessity to rapidly commercialize scientific advances. How do we elevate the discussion so the public is best served when it comes to understanding the risks and benefits of chemicals? There are no easy answers. Justified or not, confidence that government officials and corporations will serve the public interest is extremely low. From restrictions on stem cell research to “crackdowns” on agricultural chemicals, politicians have often put personal, religious and ideological views ahead of science. In that light, restoring a measure of balance in the discussion of the role of science and chemicals in our society is a daunting challenge.

Although most of us regard science as an invaluable tool for protecting and enhancing life, those in the grip of chemophobia often consider it a tool of greedy corporations empowered by institutional indifference. The cynicism is not entirely unjustified. There have been numerous environmental catastrophes marked by corporate recklessness, with government asleep at the switch, from Minamata Bay to mines in West Virginia to oil exploration and safety problems. It’s no wonder, in this context, that conspiracy theories and misinformation about the alleged dangers of chemicals have found a fertile home in cyberspace, media reports and in the minds of so many people.

As recently as the 1980s, the public relied on a limited stream of respected sources when it came to making sense of their health concerns: doctors and medical professionals; the mainstream media, including TV networks and local stations, major newspapers and key magazines; and government agencies staffed by what we assumed were independent, career scientists. Today, there are tens of thousands of “news generators,” many of them eager to get attention by presenting alarmist views.

Alternative medicine is flourishing and oversight agencies are often perceived as incompetent, corrupted or corruptible. Scientists may retain a measure of the public’s trust, but there are concerns that many of them are captive to industry or are otherwise compromised.

Another driver is the U.S. litigation system, in which tort lawyers troll for potentially lucrative class action suits. Lawyers comb the news trying to identify an industry or company that could pay for the consequences of contract-

ing an alleged disease. These are tempting targets, especially in key jurisdictions notoriously sympathetic to class action litigation.

Educators do a poor job of teaching biology, chemistry, math, physics, and risk analysis essential to an understanding of science and technology. Americans are bombarded by stories about pesticides, air pollutants and the like, but they are not educated to the risky hazards of daily life, from over-eating to unsafe sex. We are not providing students with the skills to differentiate between theoretical dangers, such as those embodied in cancer risk assessments from chemical exposures, and real (actuarial) risk, such as the odds of contracting cardiovascular disease from a fatty diet. Consequently, our educational system remains under constant attack by conservatives and liberals intent on shaping science to their personal ideologies.

Irrationality is an inherent part of the human condition. People believe what they want to believe. Even the well-educated embrace cherished dogmas, like “natural is always safer and better.” This extreme precautionary perspective fails to assess natural and human threats on the same basis. People tend to routinely ignore the potential benefits of technology, in effect favoring nature over humanity. Many people do not appreciate that the risks created by technological stagnation are often at least as real as those caused by technological advancement.

One way to at least start the process of better understanding may be for scientists and the organizations that represent them to aggressively engage in a vigorous and coordinated public dialogue about uncertainty and risk. To assess how scientists perceive the risks from exposure to commonly-encountered chemicals, the Society of Toxicology teamed with George Mason University’s Center for Health and Risk Communication and its affiliated Statistical Assessment Service (STATS) to survey more than 900 toxicologists. In contrast to public opinion, only 33 percent ascribed significant risks to food additives and just one-in-four to cosmetics. By and large, toxicologists challenged the alarmist views of some environmental activists about which chemicals or exposures are most dangerous. Phthalates were considered high risk by 11 percent; BPA by 9 percent; and Teflon by 3 percent. Smoking (89 percent); second-hand smoke (44 percent); mercury (37 percent); aflatoxin,

a naturally occurring fungus found in peanut butter, (29 percent); and exposure to sunlight (26 percent) were all considered far more dangerous. Fewer than one out of four believed that regulation should be guided by the precautionary principle and three-quarters said that the U.S. system for evaluating chemicals is superior to the European system. (STATS 2009)

Scientists are most concerned by the politicization of research. Two-thirds believe the peer review process has become too politicized; three-fourths believe scientists should restrict public statements to their areas of expertise; and a solid majority fault both the media and regulators for not doing a balanced job in explaining chemical risk to the public. The findings questioning media credibility were echoed by a recent poll of more than 2,500 members of the American Association for the Advancement of Science by the Pew Research Center, 76 percent of whom believed that news reports fail to distinguish between scientific findings that are well founded and those that are not (Pew 2009). Some 48 percent say reporters regularly oversimplify science issues. Few journalists seem to be able to distinguish between the concepts of actual dangers and potential risks.

Most scientists are aware of the widespread misrepresentation of risk by the media and the policy problems that it causes, but do not speak out. Scientists have largely remained silent when the public discussion turns to the trade-off of benefits and risks from chemicals. They are often unwilling to engage controversial issues that could endanger their funding and research. The consequences of not challenging this misinformation are severe. The public interprets the unwillingness of scientists to engage those who campaign against chemicals as an implicit validation of their dangers. Those who do speak out are often left isolated or branded as industry apologists. Maybe the best we can hope for is that brave scientists, scientifically literate journalists and government officials who are responsible for translating science into regulatory policy will take the public's best interest into account. This perspective needs to be presented to legislators so they have information necessary to resist the irrational and often regressive impulses stirred by the scare tactics that are so common today.

Throughout history, scientific innovations and discoveries have been sub-

ject to criticism and resistance. It is primarily the fear of the unknown that fuels this sentiment. This is not to say that reasonable concerns regarding scientific innovations should be ignored. Appropriate safeguards should be implemented while adopting the latest technology. But we have to recognize, and educate the public and public officials, that most activities involving technology will have undesired effects as well as desirable ones. Fear of the unknown and exaggerated precautions shouldn't be invoked to impede scientific progress. Had it not been for a stream of scientific innovations throughout history, the world today would not be able to support seven billion people living in dynamic and complex community systems. Science and technology have improved our lives in more ways than we can imagine, and chemicals have played a key role. Let's hope that continues.

Appendix:

Common Myths and Facts About Chemicals

Myth #1: A chemical-free world would be safer and healthier.

A chemical-free world is not possible. Everything—people, plants, animals, rocks, cars, air—is made up of chemicals. Some of these chemicals occur in their natural state and others are produced by combining naturally occurring chemicals.

Chemicals are everywhere—in living things, in inanimate parts of the environment and in the products vital to our health and quality of life. The natural world operates through the interactions of a vast array of chemicals. For example, humans need the chemical oxygen to survive. Plants, on the other hand, need carbon dioxide to grow and flourish. Thus, the chemical waste product of one form of life is the raw material for another. Even beneficial chemicals are dangerous at high levels. We need some 20 percent oxygen in air, but humans exposed to 100 percent oxygen for more than 24 hours will suffer massive lung damage.

Humans depend on many other types of chemicals including proteins,

carbohydrates, fats, metals and vitamins. These are supplied by food. The chemicals in the food we eat are utilized as raw materials for our growth and functioning. However, because humans are so complex, some of the chemical processes needed for these activities can malfunction. As a result, humans are subject to a variety of diseases that reflect excesses or deficiencies in these essential chemicals. For example, diabetes can result from the lack of production of the chemical insulin. Fortunately, it is now possible to make insulin synthetically and add this chemical to humans to counteract the effects of diabetes.

Thus, we are dependent on synthetic, as well as natural, chemicals for treating disease and improving both longevity and the quality of life. Both natural and synthetic chemicals are integral to all aspects of modern life. For example, natural chemicals in petroleum power cars, trucks and other vehicles, providing us with mobility and access to foods and goods from faraway places. Synthetic chemicals are critical to the functioning of the cornucopia of electronic devices, including computers and cell phones, giving us the ability to communicate around the globe instantaneously. There is no such thing as a chemical-free product and, indeed, chemicals are essential to human life and to our standard of living. Not only is a chemical-free world unachievable, it would be undesirable if it were possible.

Myth #2: Synthetic chemicals are dangerous; natural chemicals are safe.

All chemicals, whether synthetic or natural, have the potential to cause harm to people under the right circumstances. There are no nontoxic chemicals. Chemicals differ only in the types of toxicity they can cause and the exposure level at which these effects occur.

Many natural chemicals are toxic at high doses, including those in the food we eat and the water we drink. For example, a number of chemicals that occur naturally in our diet have been shown to be carcinogenic to rodents at high doses. Others, such as compounds found in soy products, can cause effects similar to those of human hormones. Thus, natural chemicals that are

critical for life may also cause harm if humans are exposed to them under certain conditions. Similarly, other natural chemicals, such as arsenic, have been shown to cause adverse effects in humans when found in high levels in drinking water. The toxicology literature is rich with stories of “endemic diseases” caused by natural food ingredients.

The same types of effects that are produced by exposure to natural chemicals, such as carcinogenicity and hormonal effects, also can occur from exposure to synthetic chemicals. In almost all cases, these effects occur only at high doses and so, as a group, synthetic chemicals are no more toxic than natural ones. The potency of a chemical does not depend on whether it is natural or synthetic; some of the most toxic chemicals are natural and some of the least toxic are synthetic. Indeed, there are a number of natural chemicals that are very highly toxic; these include the toxins that cause botulism and tetanus.

Both synthetic and natural chemicals can be toxic and present risks. Whether a chemical should or should not be used should be based on its risks and benefits, and how or if it should be used. For example, a synthetic chemical used as a pesticide may be very important for destroying insects that carry dangerous diseases but may also cause toxicity at high doses. Chemicals naturally occurring in gasoline, a product critical for transportation, may also cause toxicity if exposures are high. In both cases, these chemicals are valuable because their benefits outweigh their risks.

Myth #3: Synthetic chemicals are the cause for the rising incidence of many serious diseases, including cancer

First, over the past few decades there has been a decrease, not an increase, in the rate at which new cancers are diagnosed and the rate at which people die from cancer. Second, while there have been reported increases in the incidence of other diseases, the causes for such increases are not known.

Cancer is a disease that causes dread because of the toll it takes on victims and their families. Because cancer is a disease that becomes more common as we age, the number of cancers has been increasing as we live longer. This in-

crease in number gives the perception that cancer is becoming more common at all ages. However, when the incidence and death rates for cancers are calculated for each age group, it can be seen that they are decreasing. For example, if we looked at the rate of cancer in 80 year olds today, we would find that it is lower than it was in 80 year olds 10 years ago.

Cancer is not the only health problem that is of serious concern. Diseases that affect children, such as autism and asthma, also have been in the public eye because of reported increases in the numbers of cases of these illnesses. Careful studies of the reasons for these increases suggest that in many cases they are apparent, not real. This can occur due to changes in diagnostic practices, greater availability of diagnostic and treatment services, earlier age at diagnosis, and greater public awareness. The scientific evidence does not support claims that these diseases are due to chemical exposures.

Further, when overall health indicators — rather than the incidence of individual diseases — are examined, it is clear that the health of the American population has been continually improving. Longevity has increased significantly during the last 50 years, a period marked by a tremendous increase in the types and amounts of chemicals in everyday use. In addition, people are staying healthy longer, so that the quality of life as well as our average lifespan has improved in recent generations.

Thus, the myth that there has been a rising incidence of serious illnesses and that these are due to the increased use of synthetic chemicals does not stand up to scrutiny. It is very clear that public health has improved significantly over the recent past, due in large part to the contributions of synthetic chemicals to the diagnosis and treatment of a wide variety of diseases. Careful analysis reveals that many claimed increases in diseases are not real. In addition, in-depth assessments of the causes of existing cases of these illnesses do not demonstrate a connection between the diseases and environmental chemicals.

Myth #4: Detection of a chemical in the environment or a sample of blood or urine means that people are in danger of adverse effects.

People are exposed to thousands of natural and synthetic chemicals each day without evidence of harm. Thus, the detection of a chemical in the environment or in a sample of blood does not imply that toxic effects are occurring.

Because natural and synthetic chemicals occur in the environment around us, people are exposed to these agents each day in the air they breathe, the water they drink and the food they eat. Therefore, it is not surprising that these chemicals can be found in samples of human blood and/or urine. Indeed, reports about the variety of chemicals found in such samples are common in the media. In some cases, reporters have written stories on analyses of their own blood or urine to dramatize the findings. In other instances, reports feature the results of large-scale government studies on the blood and/or urine levels of environmental chemicals.

What does the discovery of these chemicals in human fluids mean? First, human blood and urine normally contain a wide variety of natural chemicals. Blood contains nutrients that are carried throughout the body, but it also transports unwanted waste products resulting from normal body processing of these nutrients. These products go to the kidneys where they are excreted in urine. Many of these waste compounds can cause serious effects in people if they build up to high levels as can happen when the kidneys do not function properly.

Similarly, a number of environmental chemicals, both natural and synthetic, can be found in the blood and urine. The human body has the ability to excrete these just as it excretes its own unwanted waste products. The presence of such chemicals does not imply that any adverse effects are occurring, just as the presence of the body's waste products does not mean that the humans carrying them are suffering toxicity. Only if these environmental chemicals build up to high levels is there a likelihood of harm.

Careful analysis by government scientists of the levels of these environ-

mental chemicals in blood and/or urine demonstrates that they are almost always present at very low levels, often called trace levels. These levels are not high enough to cause any harm; just because they are present does not mean that there is a risk involved. These analyses tell us only if people have been exposed to the chemicals studied — not if any effects are likely. Additional information, such as how often exposure has occurred, for how long and at what levels, is necessary to determine the possibility of toxic effects.

Myth #5: Chemicals used in food, consumer products and agriculture have not been shown to be safe.

Since all chemicals, natural or synthetic, can cause toxicity at some dose, none of them are absolutely safe. Indeed, there is no way to show that any chemical is absolutely safe at any dose since you can always imagine other tests that could be performed to look for more and more obscure and unlikely effects.

Since absolute safety is not a possibility, the question is whether these food, consumer and agricultural chemicals have undergone enough testing so there is a reasonable likelihood that they will cause no harm when used properly. While it has been claimed that adequate testing and evaluation have not been performed—and thus that our food and consumer products are unsafe—a careful analysis shows that this is not the case.

The claims of insufficient testing are of two types. The first is based on the idea that the current toxicity tests are not appropriate in the light of new knowledge. A good example of this is the assertion that chemicals can show no effects at high doses but still produce significant toxicity at much lower doses. Those who espouse this view say it demonstrates that traditional testing done at high doses may miss toxic effects. That's a controversial hypothesis that has, as yet, limited support among scientists.

The second type of claim is that not enough testing has been done or that it has been performed and/or evaluated in a biased way. Generally, the incomplete or biased testing results are linked to industry. While it is true that much

of the toxicity testing of products in commerce is performed by industry, this is because the federal regulatory system requires such evaluations. This approach has been very successful in almost all cases, as evidenced by the overall safety of the food supply and the very small number of chemicals in consumer products that have been shown to cause any toxicity, even in sensitive individuals, when used as intended.

Hence, the belief that chemicals have not been adequately tested before the public is exposed to them does not hold up under careful scrutiny. It is based on two assertions, neither of which is supported by the evidence. The first, that current test methods are inadequate, is based on assertions of scientists who do not represent the scientific consensus and the second, that industry testing is insufficient and/or biased, is not supported by the safety records of foods and consumer products.

Myth #6: If there is any evidence that a chemical might cause harm, it should be taken off the market.

As stated previously, all chemicals, both natural and synthetic, are toxic at some exposure level so applying this principle would lead to the removal of all chemicals, whether beneficial or not. This approach would deny people the benefits of drugs that cure serious diseases, disinfectants that protect citizens against microorganisms, pesticides that protect us against insect-borne diseases, and a host of life-saving medical devices.

Those who believe that chemicals should be removed from the market whenever there is the slightest evidence that they may cause harm base this view on the “better safe than sorry” precautionary principle. However reasonable this principle may seem on the surface, this approach is unlikely to make you safer and, instead, could very well increase risk.

Why is this? For one, devoting resources to taking a chemical — and products containing it — off the market and replacing it means that these same resources will not be available to assess other risks. If there is little evidence that this product causes serious harm, then it is unlikely there will be any reduc-

tion in risk from removing it. On the contrary, since this action would divert resources from known risks to public health, it is more likely that there would be a net decrease in safety.

In addition, the replacement of a product in common use has environmental consequences since it would require the use of significant amounts of energy to collect and dispose of the banned substance, and to develop, produce, market and distribute a replacement. Generating the energy needed for these steps would be associated with pollution and the potential for adverse effects in people exposed to these pollutants. Thus, the replacement process itself entails risks that must be considered.

It is often the case that at least some of the benefits of the product being replaced are lost. This happens because many products, such as plastics in medical devices, are in use because of unique properties that cannot be exactly duplicated. So, in addition to a significant possibility of increased risk from banning a chemical of unproven harm, there is also the likelihood of a loss of benefits.

Because all chemicals are toxic, it is quite likely that there will be some toxicity associated with the replacement. It is often not clear until a product has been in use for a long time what this toxicity is and how many people it may affect. It is quite possible that the replacement chemical, and products containing it, will be associated with at least as much risk as the original chemical. The application of the principle of "better safe than sorry" can result in the replacement of an unsubstantiated risk with an unknown one.

The seemingly prudent step of taking chemicals off the market when there is the slightest suggestion of toxicity is unlikely to accomplish what is intended. Because there is no solid evidence of harm, it is not clear that any reduction in risk will occur. It is much more likely that there will be an overall increase in risk, because the substitution process incurs other risks, as well as a loss of benefits if the chemical and products containing this chemical are taken off the market. The really prudent step is to make the best scientific evaluation of the risk from the product as compared to the risks and loss of benefits associated with removing it from the market before any actions are taken.

Myth #7: Claims by advocacy groups are objective and based on the best science.

Although advocacy groups often assert that their claims of danger from chemicals are based on science, close examination reveals that these assertions often do not reflect the best or most complete science. In some cases, they do not reflect any science at all; they rely on the belief that the presence of a chemical is equivalent to risk.

Advocacy groups, as their name implies, advocate for particular positions. In the process, they marshal the best arguments they can make to support their position. This often entails citing evidence that is most conducive to their case, no matter how valid, and ignoring evidence that is contrary to it. Further, they often try to portray scientists who have an opposing view as biased while asserting that they are objective. Relying on the tendency of many media sources to publicize dramatic findings, they are often able to dominate the headlines.

However, a close scientific examination of advocacy group claims reveals they are often based on studies by scientists who do not reflect the expert consensus or a balanced treatment of the available evidence. Instead, they tend to emphasize the worst possible interpretation of the data. Yet, in the absence of solid evidence, such groups suggest that restricting or eliminating particular chemicals is necessary. This position is based on the conviction that it is prudent to take chemicals off the market even if there is only the slightest support for the contention that they pose a risk to the public.

Unfortunately, a number of factors contribute to the public's willingness to accept blanket claims by advocacy groups. The media give excessive attention to the views of NGOs that are sensational and critical of industry. Industry responds to the barrage of negative publicity by removing the attacked products. That often leads government officials to pass restrictive legislation. This attack and withdraw cycle, repeated again and again, contributes to a public perception that the original allegations were scientifically valid. However, this is often not the case. These predictable reactions by the media, industry and government are shaped by the desire for publicity at any cost, or

by policy and economic considerations, not on an assessment of the scientific validity of the claims.

Yes, environmental disasters have occurred due to corporate greed or indifference and government incompetence. There are examples in which corporations have exercised their influence to bend policy to their needs, and the public has paid the price. But for the most part, the desires of corporations and the public coincide. Businesses that break the trust of their customers don't prosper let alone survive. And in all but a very few instances, the regulatory machinery, however inefficient, does identify new drugs, improved ways to grow and preserve foods, and enhance the quality of our food and water. We can improve the system, in some cases significantly, but the evidence doesn't support the cynical belief encouraged by many activists that corporations are out to fleece their customers and government is corrupt or hopelessly inefficient.

Science needs to rest on a solid body of independently verified evidence. Any evidence is not equivalent to valid evidence. When scrutinized, many claims by advocacy groups are not scientifically sound. They reflect a selective use of facts and often rely on scientists with a demonstrable (and sometimes avowed) bias. These groups often rely on popular but mistaken beliefs to bolster their positions: that it is possible to have chemical-free products; that synthetic chemicals are more dangerous than natural ones; that some chemicals are nontoxic; that synthetic chemicals are responsible for increases in disease; that detection of a chemical is equivalent to a toxic effect; and that it is prudent to take useful and desirable products off the market even in the absence of solid scientific evidence of harm. That's not science.

Bibliography

- Ackerman, F. "The Economics of Atrazine." *International Journal of Occupational and Environmental Health* 13, no. 4 (October/December 2007): 437–45.
- American Council on Science and Health. "America's War on Carcinogens." 2005.
- . "Biomonitoring: Measuring Levels of Chemicals in People and What the Results Mean." ACSH, New York, 2005.
- . "Endocrine Disrupters: A Scientific Perspective." ACSH, New York, 1999.
- . "Facts Versus Fears (Fourth Edition): A Review of the Greatest Unfounded Health Scares of Recent Times." ACSH, New York, 2004.
- . "Sugar Substitutes and Your Health." ACSH, New York, 2006.
- . "Traces of Environmental Chemicals in the Human Body: Are They a Risk to Health?" ACSH, New York, 2003.
- American National Election Studies. "ANES Guide to Public Opinion and Electoral Behavior. Trust in the Federal Government 1958-2004." ANES. 2009. http://www.electionstudies.org/nesguide/toptable/tab5a_1.htm.
- Ames, B., and L. S. Gold. "Pesticides, Risks, and Applesauce." *Science* 244 (May 1989): 755-757.

- Australian Pesticides and Veterinary Medicines Authority. "Chemicals in the News: Atrazine." 2010 19-March. http://202.125.15.27/news_media/chemicals/atrazine.php.
- . "Chemicals in the News: Atrazine." 2010 31-May. http://www.apvma.gov.au/news_media/chemicals/atrazine.php#amphibians.
- Bahlai, Christine, Yingen Xue, Cara M. McCreary, Arthur W. Schaafsma, and Rebecca H. Hallett. "Choosing Organic Pesticides over Synthetic Pesticides May Not Effectively Mitigate Environmental Risk in Soybeans." *PLoS one*. 2010 22-June. <http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0011250>.
- Barnes, D. G., and M. Dourson. "Reference Dose (RfD): Description and use in health risk assessment." *Reg. Toxicol. Pharm*, 1988: 471-486.
- BBC. "Herbs 'Can Ben Natural Pesticides.'" <http://news.bbc.co.uk/2/hi/science/nature/8206045.stm>. . 2009 17-August.
- Biello, David. "Sex-Changing Weed Killer." *Scientific American*. 2010 May. <http://www.scientificamerican.com/article.cfm?id=sex-changing-weed-killer>.
- Boffey, P. "Study of Love Canal Finds No Direct Link to Genetic Diseases." *The New York Times*, 1983 18-May.
- Brown, L. "What's Your Chemical Burden." *Illinois Wesleyan University Wellness Center*. 2009 31-March. http://www.iwu.edu/~wellness/environment_folder/body_burden.htm.
- Brussels: Health and Consumer Protection Directorate-General, Scientific Committee on Plants, European Commission. *Opinion of the Scientific Committee on Plants on Specific Questions from the Commission concerning the Evaluation of Atrazine in the Context of Council Directive 91/414/EEC*. SCP/ATRAZINE/002-Final, 2003.
- Butterworth, T. "New Independent Study by EPA Refutes BPA Risk." *Statistical Assessment Service*. 2009 30-October. http://stats.org/stories/2009/breaking_news_bpa_oct30_09.html.
- Calafat, A. M, Xiaoyun Ye, Lee-Wang Wong, John A. Reidy, and Larry L. Needham. *Environmental Health Perspectives* 116 (2007).
- California Office of Environmental Health Hazard Assessment. "Proposition 65 in Plain Language!" *State of California, Sacramento, CA*. 2010 March. <http://www.oehha.org/prop65/background/p65plain.html>.

- Carson, R. *Silent Spring*. New York: Houghton Mifflin, 1962.
- Case, D. "The Real Story Behind Bisphenol A." *Fast Company*, February 2009. <http://www.fastcompany.com/magazine/132/the-real-story-on-bpa.html?page=0%2C0>.
- Center for the Evaluation of Risks to Human Reproduction. "NTP-CERHR Monograph on the Potential Human Reproductive and Developmental Effects of Bisphenol A." 2008 September. <http://cerhr.niehs.nih.gov/chemicals/bisphenol/bisphenol.pdf>.
- Centers for Disease Control and Prevention. "Bisphenol A: Cas no. 80-05-7. National Report on Human Exposure to Environmental Chemicals," 2010 11-10. <http://cdc.gov>.
- Clean Water Action. "Fighting Drugs In Drinking Water." <http://www.cleanwateraction.org/programinitiative/fightingdrugsdrinkingwater>.
- Colburn, T., Dianne Dumanoski, and J. P. Meyers. *Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival?—A Scientific Detective Story*. New York: Penguin, 1996.
- Consumer Reports*. "Concern over Canned Foods: Our Tests Find Wide Range of Bisphenol A in Soups, Juice, and More." *Consumer Reports*. 2009 December. <http://www.consumerreports.org/cro/magazine-archive/december-2009/food/bpa/overview/bisphenol-a-ov.htm>.
- Cooper, R. L., T. E. Stoker, J. M. Goldman, M. B. Parish, and L. Tyrey. "Effect of atrazine on ovarian function in the rat." *Reprod Toxicol*, no. 10 (1996): 257–264.
- Coursey, Don. "Sound Science, Not Politics, Should Guide Atrazine Legislation." *Des Moines Register*, 2010 7-July.
- CropLife America. "Pesticide Regulation." 2010. <http://www.croplifeamerica.org/crop-protection/pesticide-regulation>.
- Dangour, A. D., K. Lock, A. Hayter, A. Aikenhead, E. Allen, and R. Uauy. "Nutrition-related health effects of organic foods: a systematic review." *The American Journal of Clinical Nutrition* 92, no. 1 (July 2010): 203-210.
- Dekant, W. and W. Völkel. "Human Exposure to Bisphenol A by Biomonitoring: Methods, Results and Assessment of Environmental Exposures." *Toxicology and Applied Pharmacology* 228, no. 1 (April 2008): 114-34.
- Doerge, Daniel R., Nathan C. Twaddle, Kellie A. Woodling, and Jeffrey W. Fisher. "Pharmacokinetics of bisphenol A in neonatal and adult rhesus monkeys." *Toxicology and Applied Pharmacology* 248, no. 1 (October 2010): 1-11.

- Doll, R., and R. Peto. "The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today." *J. Natl. Cancer Inst.*, 1981: 66(6):1191308.
- E.U. Commission of the European Communities. "Communication From The Commission on the precautionary principle." February 2000.
- Ehrlich, Paul. *The Population Bomb*. Amereon Limited, 1968.
- Environmental Working Group. "Body Burden: The Pollution in Newborns." 2005. <http://www.ewg.org/reports/bodyburden2/execsumm.php>.
- . "PCBs in Farmed Salmon." 2003. <http://www.ewg.org/reports/farmedpcbs>.
- . "Skin Deep Cosmetic Safety Database." 2010. <http://www.cosmeticsdatabase.com>.
- . "What We Don't Know About the Toxic Stuff Around Us." April 19, 2010. <http://www.ewg.org/kid-safe-chemicals-act-blog/2010/04/what-we-don%E2%80%99t-know-about-the-toxic-stuff-around-us/>.
- European Food Safety Authority. "Opinion of the Scientific Panel on Food Additives, Flavourings, Processing Aids and Materials in Contact with Food (AFC) on a request from the Commission related to Flavouring Group Evaluation 11 (FGE.11)." *EFSA Journal*, no. 166 (2004).
- . "Opinion of the Scientific Panel on Food Additives, Flavourings, Processing Aids and Materials in Contact with Food (AFC) Related to 2,2-Bis (4-Hydroxyphenyl)Propane." *EFSA Journal* 428 (2006): 1-75.
- . "Scientific Opinion on Bisphenol A: evaluation of a study investigating its neurodevelopmental toxicity, review of recent scientific literature on its toxicity and advice on the Danish risk assessment of Bisphenol A." September 23, 2010. <http://www.efsa.europa.eu/en/press/news/cef100930.htm>.
- Fawcett, Richard. "Twenty Years of Corn Yield with and without Atrazine." *North Central Weed Science Society*, 2008. <http://www.ncwss.org/proceed/2008/abstracts/137.pdf>.
- Food and Drug Administration. *Update on Bisphenol A (BPA) for Use in Food Contact Applications*. Washington, D.C.: 111th Cong., 2d sess, 2010.
- Fox, M. "Cancer Report Energizes Activists, Not Policy." *Reuters*, 2010 10-May.
- Gallup. "On Darwin's Birthday, only 4 in 10 Believe in Evolution." February 11, 2009. <http://www.gallup.com/poll/114544/darwin-birthday-believe-evolution.aspx>.

- Garte, S. "Our Chemophobia Conundrum." *The American*. 2010 2-February. <http://www.american.com/archive/2010/february/our-chemophobia-conundrum>.
- Gilliom, R.J, et. al. "The Quality of Our Nation's Waters: Pesticides in the Nation's Streams and Ground Water, 1992-2001." U.S. Geological Survey Circular 1291, 2006.
- Goodman, Julie E., et al. "An Updated Weight of the Evidence Evaluation of Reproductive and Developmental Effects of Low Doses of Bisphenol A. ." *Critical Reviews in Toxicology*, no. 36 (2006): 387-457.
- Government of Canada. "Chemical Substances: Bisphenol A, Fact Sheet." 2008 17-10. <http://www.chemicalsubstanceschimiques.gc.ca/fact-fait/bisphenol-a-eng.php>.
- Gray Jr., L. E. "Letter to the editor." *Toxicological Sciences*, March 2010.
- Gray, George M., et al. "Weight of the evidence evaluation of low-dose reproductive and developmental effects of bisphenol A." *Human and Ecological Risk Assessment*, September 2004: 875-921.
- Guobing, X., S. Junling, H. Guohua, Z. Lixing, W. Tongjun, and Z. Zhijun. "Investigation into serum BPA and sex hormone levels of workers in epoxy resin manufacturing." *J. Environ. Occup. Med.*, no. 22 (2005): 295-298.
- Haighton, L. A., J. J. Hlywka, J. Doull, R. Kroes, and B. S. Lynch. "An Evaluation of the Possible Carcinogenicity of Bisphenol A to Humans." 2002 April. <http://dx.doi.org/doi:10.1006/rtph.2001.1525>.
- Hayes, T. B., et al. "Hermaphroditic, Demasculinized Frogs after Exposure to the Herbicide Atrazine at Low Ecologically Relevant Doses." *PNAS* 99, no. 8 (April 2002): 5476-80.
- Hayes, Tyrone B., et al. "Atrazine induces complete feminization and chemical castration in male African clawed frogs (*Xenopus laevis*)." 2010 1-March. <http://www.pnas.org/content/early/2010/02/12/0909519107.abstract>.
- Health Canada. "Re-evaluation Decision Document: Atrazine." 2004 25-May. http://www.hc-sc.gc.ca/cps-spc/pubs/pest/_decisions/rrd2004-12/index-eng.php.
- Heath, C. W., M. R. Nadel, M. M. Zack Jr., A. T. L. Chen, M. A. Bender, and R. J. Preston. "Cytogenetic Findings in Persons Living Near the Love Canal." *JAMA* 251, no. 11 (1984): 1437-1440.

- Henahan, John F. "Whatever Happened to the Cranberry Crisis?" *The Atlantic Online*. March 1977. <http://www.theatlantic.com/past/docs/issues/95dec/lead/hencran.htm>.
- Herbst, A. L., H. Ulfelder, and D. C. Poskanzer. "Adenocarcinoma of the vagina. Association of maternal stilbestrol therapy with tumor appearance in young women." *N Engl J*. 15 (1971): 878-81.
- Howdeshell, Kembra L., Jonathan Furr, Christy R. Lambright, Vickie S. Wilson, Bryce C. Ryan, and L. Earl Gray. "Gestational and Lactational Exposure to Ethinyl Estradiol, but Not Bisphenol A, Decreases Androgen-Dependent Reproductive Organ Weights and Epididymal Sperm Abundance in the Male Long Evans Hooded Rat." *Toxicological Sciences* 102, no. 2 (2008): 371-82.
- Hubbs, A. F., et al. "Airway Epithelial Toxicity of the Flavoring Agent, 2,3-Pentanedione." *The Toxicologist CD* 114, no. S-1 (March 2010).
- International Agency for Research on Cancer Monographs. "Atrazine." 1999. <http://monographs.iarc.fr/ENG/Monographs/vol73/mono73-8.pdf>.
- International Joint Commission. "Risks and Benefits of Drinking Water Disinfection." 2003. http://www.ijc.org/rel/pdf/05_waterdisfall2003.pdf.
- Joskow, R., D. B. Barr, J. R. Barr, A. M. Calafat, L. L. Needham, and C. Rubin. "Exposure to bisphenol A from bis-glycidyl dimethacrylate-based dental sealants." *Journal of the American Dental Association* 137, no. 3 (2006): 353-62.
- Kaiser Permanente Division of Research. "Workplace BPA Exposure Increases Risk of Male Sexual Dysfunction: First Human Study to Measure Effects of BPA on Male Reproductive System." 2009 11-November. http://www.eurekalert.org/pub_releases/2009-11/kp-wbe110309.php.
- Kaleita, Amy. "Sound Science on Trial." *The Daily Caller*. June 24, 2010. <http://dailycaller.com/2010/06/04/sound-science-on-trial/2/>.
- Kamrin, M. A. "Bisphenol A: A Scientific Evaluation." *Medscape General Medicine*, 2004 3-September.
- . "The Low-Dose Hypothesis: Validity and Implications for Human Risk." *Intl. J. Toxicol* 26 (2007): 13-23.
- Krewski, D., M. E. Andersen, E. Mantus, and L. Zeise. "Toxicity testing in the 21st century: implications for human health risk assessment." *Risk Anal* 29 (2009): 474-479.

- Krishnan, A. V., P. Stathis, S. F. Permuth, L. Tokes, and D. Feldman. "Bisphenol-A: An Estrogenic Substance Is Released from Polycarbonate Flasks during Autoclaving." *Endocrinology* 132 (1993): 2279–86.
- Kutcher, J. S., A. Engle, J. Firth, and S. H. Lamm. "Bendectin and birth defects II: Ecological analyses." *Birth Defects Research Part A: Clinical and Molecular Teratology* 67, no. 2 (2003): 88-97.
- Lance, J. "Alarming Body Burden Results: Tests Reveal 300 Chemical Compounds in Newborn Babies." *Eco Child's Play*. 2008 13-October. <http://ecochildsplay.com/2008/10/13/alarming-body-burden-results-tests-reveal-300-chemical-compounds-in-newborn-babies/> .
- Land Stewardship Project and Pesticide Action Network. "The Syngenta Corporation and Atrazine: The Cost to the Land, People and Democracy." 2010 January. <http://www.landstewardshipproject.org/pdf/AtrazineReportJan2010.pdf>.
- Layton, Lyndsey. "Alternatives to BPA Containers Not Easy for U.S. Foodmakers to Find." *Washington Post*, 2010 23-February.
- Lenz, W. "A short history of thalidomide embryopathy." *Teratology*, 1998: 203-215.
- "Letter from U.S. EPA's Donald Brady to Illinois State Representative Dave Winters." 2010 17-May. <http://agsense.org/epa-responds-to-illinois-rep-concerning-hayes-atrazine-based-frog-gonad-assertions/> .
- Life. "Mercy Ma! No Cranberries!" 1959 23-November.
- Lorentzen, Ronald, and D. G. Hattan. "Response to Nature Editorial: The weight of evidence." *Nature*. April 22, 2010. <http://www.nature.com/nature/journal/v464/n7292/full/4641103b.html>.
- Merill, R. A. "Food Safety Regulation: Reforming the Delaney Clause." *Annual Review of Public Health*, 1997: 313-40.
- Milwaukee Journal Sentinel. "Chemical Fallout." 2010. <http://www.jsonline.com/watchdog/34405049.html>.
- Mittelstaedt, M. "Scientist's Endorsement of Bisphenol A under Review." *Toronto Globe and Mail*, 2007 20-June.
- Moalem, S., and J. Prince. *Survival of the Sickest: A Medical Maverick Discovers Why We Need Disease*. William Morrow, 2007.
- Mossman, B. T., J. Bignon, M. Corn, A. Seaton, and J. B. L. Gee. "Asbestos: Scientific Developments and Implications for Public Policy." *Science* 247, no. 4940 (1990): 294-301.

- Mozaffarian, D., and E. B. Rimm. "Fish Intake, Contaminants, and Human Health: Evaluating the Risks and the Benefits." *JAMA* 296, no. 15 (2006): 1885-1899.
- Muhawi, D. "Safe Pesticides? Natural vs. Synthetic Pesticides." *EcoWorld*. 2004 25-June. <http://www.ecoworld.com/animals/safe-pesticides.html>.
- National Academies Press. "Carcinogens and Anticarcinogens in the Human Diet: A Comparison of Naturally Occurring and Synthetic Substances." (National Research Council) 1996.
- National Cancer Institute. "Cancer Facts: Questions and Answers About Asbestos Exposure." Washington, D.C., 1995.
- . "President's Cancer Panel Annual Report for 2008-2009." 2010. <http://deainfo.nci.nih.gov/advisory/pcp/pcp.htm>.
- National Institutes of Health. "Health Agencies Express Concern over BPA." *Medline Plus of the U.S. National Library of Medicine and NIH*. 2010 15-January. http://www.nlm.nih.gov/medlineplus/news/fullstory_94170.html.
- National Toxicology Program. "NTP Technical Report on the Carcinogenesis Bioassay of Bisphenol A (CAS No. 80-0507) in F344 Rats and B6C3F1 Mice (Feed Study)." 1982, 82-1771.
- National Toxicology Program, HHS and the NIEHS. "National Toxicology Program's Report of the Endocrine Disruptors Low-Dose Peer Review." 2001 August. <http://ntp.niehs.nih.gov/ntp/htdocs/liason/LowDosePeerFinalRpt.pdf>.
- Natural Resources Defense Council. "Atrazine: Poisoning the Well: How the EPA Is Ignoring Atrazine Contamination in the Central United States." 2009 22-August. <http://www.nrdc.org/health/atrazine/default.asp>.
- New York City Department of Environmental Protection. "DEP Study Shows No Risk from Pharmaceuticals and Personal Care Products in NYC Drinking Water." 2010 27-May. http://www.nyc.gov/html/dep/html/press_releases/10-55pr.shtml.
- Oka, T., et al. "Effect of Atrazine on Metamorphosis and Sexual Differentiation in *Xenopus Laevis*." *Aquatic Toxicology* 7, no. 4 (2008): 215-26.
- Pew Research Center for the People & the Press. "Public Praises Science; Scientists Fault Public, Media." <http://pewresearch.org/pubs/1276/science-survey>. 2009 9-July.
- Pielke, R.A., Jr. *The Honest Broker: Making Sense of Science in Policy and Politics*. Cambridge: Cambridge University Press, 2007.

- Price, J. M. "Bladder tumors in rats fed cyclohexylamine or high doses of a mixture of cyclamate and saccharin." *Science*, 1970: 1131-1132.
- PRWeb. "Levels of Trace Pharmaceuticals in Drinking Water Too Low to Impact Human Health According to Water Research Foundation Study." *PRWeb, Ferndale, WA*. 2009. http://www.dwsd.org/announce/releases/pr2009_03_04_Pharmaceuticals_in_Water.pdf.
- Public Health Service. "Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service." Public Health Service Publication, Washington, D.C., 1964, 64-1103.
- Public Health Service. "Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service." Public Health Service Publication 64-1103, 1964.
- Renner, Rebecca. "Atrazine Effects in *Xenopus* Aren't Reproducible." *Environmental Science and Technology*, 2008 15-May: 3491-93.
- . "Controversy Lingers over Herbicides' Link to Frog Deformities." *Science*, 2002 1-November: 938-39.
- Richter, C. et al. "In Vivo Effects of Bisphenol A in Laboratory Rodent Studies." *Reproductive Toxicology*. 2007 August-September. <http://dx.doi.org/doi:10.1016/j.reprotox.2007.06.004>.
- Rimondi, L. "Bachelot: des études ont conclu à l'innocuité des biberons contenant du Bisphénol-A [Bachelot: Studies Concluded Feeding-Bottles Containing Bisphenol A Are Harmless]." Agence France Presse. 2009 31-March. <http://www.lepoint.fr/actualites-sciences-sante/2009-03-31/bachelot-des-etudes-ont-conclu-a-l-innocuite-des-biberons/919/0/330807>.
- Roberts, D. et al. *The Excellent Powder: DDT's Political and Scientific History*. Indianapolis: Dog Ear Publishing, 2010.
- Rohr, J. R., and K. A. McCoy. "A Qualitative Meta-Analysis Reveals Consistent Effects of Atrazine on Freshwater Fish and Amphibians." *Environmental Health Perspective* 118, no. 1 (October 2010): 20-32.
- Ropeik, D. "It's Irrational to Say that People Who Get Risk Wrong Are Irrational." *Psychology Today Blog*. 2010 20-July. <http://www.psychologytoday.com/blog/how-risky-is-it-really/201007/it-s-irrational-say-people-who-get-risk-wrong-are-irrational>.
- Rosen, J.D. "Much Ado about Alar." *Issues in Science and Technology*, 1990 fall: 85-90.

- Rosen, J.D. "A Review of the Nutrition Claims Made by Proponents of Organic Food." *Comprehensive Reviews in Food Science and Food Safety* 2010: 270-277.
- Rutledge, Raquel. "Snack could be toxic." *Milwaukee Journal Sentinel*, September 5, 2007.
- Ryan, B. C. et al. "In Utero and Lactational Exposure to Bisphenol A, in Contrast to Ethinyl Estradiol, Does Not Alter Sexually Dimorphic Behavior, Puberty, Fertility, and Anatomy of Female LE Rats." *Toxicological Sciences* 114, no. 1 (2010): 133-48.
- Safer Chemicals, Healthy Families. "The Health Case for Reforming the Toxic Substances Control Act." 2010.
- Safer Chemicals, Healthy Families. "The Health Case for Reforming the Toxic Substances Control Act." 2010.
- Saul, Michael Howard. "City's Water Tests Clean." *Wall Street Journal*. 2010 27-May. http://online.wsj.com/article/SB10001424052748704032704575268983421433118.html?mod=googlenews_wsj.
- Science and Environmental Health Network. "The Wingspread Consensus Statement on the Precautionary Principle, The Wingspread Conference on the Precautionary Principle." 1998. <http://www.sehn.org/state.html#w/>.
- Science News. "Cyclamates banned." 1969: 96 (17): 369.
- Shannon, E. "BPA Wrecks Sex, Fouls Food—and Probably Worse." *Huffington Post*. 2009 12-November. http://www.huffingtonpost.com/elaine-shannon/bpa-wrecks-sex-fouls-food_b_354268.html.
- Sharpe, R. M. "Is It Time to End Concerns over the Estrogenic Effects of Bisphenol A?" *Toxicological Sciences* 114, no. 1 (2010): 1-4.
- Solomon, K. R., et al. "Ecological Risk Assessment of Atrazine in North American Surface Waters." *Environmental Toxicology and Chemistry*, 1996: 31-76.
- Solomon, Keith. "Effects of Atrazine on Fish, Amphibians, and Aquatic Reptiles: A Critical Review." *Critical Reviews in Toxicology* 38, no. 9 (2008): 721-72.
- Souder, William. "As EPA Re-Evaluates Safety of Herbicide Atrazine, Minnesota Conducts Its Own Review." *MinnPost.com*. 2009 28-October. www.minnpost.com/stories/2009/10/28/12903/as_epa_re-evaluates_safety_of_herbicide_atrazine_minnesota_conducts_its_own_review.
- "Statement of EPS's Anne E. Lindsay, Minnesota House of Representatives." 2005 16-February. http://www.atrazine.com/atrazine/images/Lindsay_written.pdf.

- STATS-Statistical Assessment Service and Center for Health and Risk Communications at George Mason University. "Toxicologists' Opinions on Chemical Risk: A Survey of the Society of Toxicology. 2009 21-May. http://stats.org/stories/2009/are_chemicals_killing_us.html.
- Sullivan, Daniel J., Aldo V. Vecchia, Daniel L. Lonenz, Robert J. Gilliom, and Jeffrey D. Martin. "Trends in Pesticide Concentrations in Corn-Belt Streams, 1996–2006." USGS Scientific Investigations Report 2009-5132. 2009. <http://pubs.usgs.gov/sir/2009/5132>.
- Sun, M. "Missouris costly dioxin lesson." *Science News*, 1983 28-January: 367-368.
- Switzer, J. A., V. V. Rajasekharan, S. Boonsalee, E. A. Kulp, and E. W. Bohannon. "Evidence that Monochloramine Disinfectant Could Lead to Elevated Pb Levels in Drinking Water." *Environ. Sci. Technol.*, 2006: 40: 3384-3387.
- Turner, J. S. *The Chemical Feast: Ralph Nader's Study Group Report on the Food and Drug Administration*. N.Y.: Grossman Publishers, 1970.
- U.S. Congress. "Asbestos Hazard Emergency Response Act (AHERA)." 1986.
- . "BPA-Free Kids Act of 2009, S. 753, 111th Cong., 1st sess." 2009. <http://www.govtrack.us/congress/bill.xpd?bill=s111-753>.
- . "CFR Title 42—the Public Health and Welfare, Chapter 103: Comprehensive Environmental Response, Compensation, and Liability." Washington, D.C., 1980.
- . "P.L. 104-170 - Food Quality Protection Act of 1996." Washington, D.C., 1996.
- . "S. 3209: Safe Chemicals Act of 2010." 2009. <http://www.govtrack.us/congress/bill.xpd?bill=s111-3209> (accessed 27 2010, 2010).
- U.S. Energy News. "Less Is More: No-Till Agriculture Helps Mitigate Global Warming." September 2009. <http://www.Eurekaalert.org/features/doe/2005-09/dnnl-lim091605.php>.
- U.S. Environmental Protection Agency. "Bisphenol A. (CASRN 80-05-7)." *Integrated Risk Information System*. 1988. <http://www.epa.gov/ncea/iris/subst/0356.htm> (accessed 2010 17-March).
- . "Consolidated DDT Hearing: Hearing Examiner Edmund M. Sweeney's Findings, Conclusions and Orders." 1972a. http://www.epa.gov/history/topics/ddt/1972_EPA_DDT_hearing.PDF.
- . "Consolidated DDT Hearing: Opinion and Order of the Administrator." Washington, D.C., 1972b.

- “Environmental Progress.” <http://www.epa.gov/earthday/history.htm>.
- “EPA Begins New Scientific Evaluation of Atrazine.” *EPA News Release*. 2009 7-October. <http://yosemite.epa.gov/opa/admpress.nsf/8b770facf5edf6f185257359003fb69e/554b6abea9d0672f85257648004a88c1!OpenDocumnt>.
- “EPA Guidance for Assessing Chemical Contamination Data for Use in Fish Advisories.” Washington, D.C., 1994.
- “Fact Sheet for Schools: Caulk containing PCBs may be present in older schools and buildings.” 2009. <http://www.epa.gov/pcbsincaulk/caulkschoolkit.pdf>.
- U.S. Environmental Protection Agency. *FQPA Safety Factor (10X)*, Staff Paper #2, TRAC 5/2829/98. Washington, D.C.: U.S. EPA, 1998.
- “Pesticide News Story: Scientific Advisory Panel to Discuss Amphibian Gonadal Development; White Paper Posted to Docket.” 2007 24-September. http://www.epa.gov/oppfead1/cb/csb_page/updates/2007/sap-mtg-atrazine.htm.
- “Summary of the Toxic Substances Control Act.” <http://www.epa.gov/lawsregs/laws/tsc.html>.
- “Summary of the Toxic Substances Control Act.” 2010. <http://www.epa.gov/lawsregs/laws/tsc.html>.
- Biological and Economic Analysis Division. “Assessment of Potential Mitigation Measures for Atrazine.” 2002. <http://www.btny.purdue.edu/ppp/ABA-02-03.pdf>.
- “Assessment of Potential Mitigation Measures for Atrazine.” 2002. <http://www.btny.purdue.edu/ppp/ABA-02-03.pdf>.
- U.S. Food and Drug Administration. “Draft Assessment of Bisphenol A for Use in Food Contact Applications, 110th Cong., 2d sess.” 2008 August. http://www.fda.gov/ohrms/dockets/AC/08/briefing/2008-0038b1_01_02_FDA_BPA_Draft_Assessment.pdf.
- UK Rapporteur Monograph. “Atrazine: Report and Proposed Decision of the United Kingdom Made to the European Commission under Article 7(1) of Regulation 3600/92 Council Directive 91/414/EEC Regulation 3600/92.” United Kingdom Pesticide Directorate, 1996.
- United Nations Environment Programme. “Rio Declaration on Environment and Development.” 1992 14-June. <http://www.unep.org/Documents.multilingual/Default.asp?DocumentID=78&ArticleID=1163>.

- University of Michigan Child Health Evaluation and Research Unit. "National Poll on Children's Health." August 16, 2010.
- University of Missouri College of Arts and Sciences. "Evidence Mounts against Chemical Used Widely in Everyday Plastic Products." 2005 5-April. <http://rcp.missouri.edu/articles/>.
- USEPA Office of Pesticide Programs, Health Effects Division. "Triazine Cumulative Risk Assessment." 2006.
- Völkel, W. et al. "Metabolism and Kinetics of Bisphenol A in Humans at Low Doses Following Oral Administration." *Chemical Research in Toxicology* 15, no. 10 (2002): 1281-87.
- Völkel, W., T. Colnot, G. A. Csanady, J. G. Filser, and W. Dekant. "Metabolism and Kinetics of Bisphenol A in Humans at Low Doses Following Oral Administration." *Chemical Research in Toxicology* 15, no. 10 (2002): 1281-87.
- Vogel, S. "The Politics of Plastics: The Making and Unmaking of Bisphenol A 'Safety'." *American Journal of Public Health* 99, no. S3 (November 2009): S559-66.
- vom Saal, F. S., and Claude Hughes. "An Extensive New Literature Concerning Low-Dose Effects of Bisphenol A Shows the Need for a New Risk Assessment." *Environmental Health Perspectives* 113, no. 8 (2005): 926-33.
- vom Saal, F. S., S. M. Belcher, L. J. Guillette, R. Hauser, J. P. Myers, and G. S. Prins. "Chapel Hill Bisphenol A Expert Panel Consensus Statement: Integration of Mechanisms, Effects in Animals and Potential to Impact Human Health at Current Levels of Exposure." *Reproductive Toxicology* 24, no. 2 (2007): 131-38.
- Walsh, B. "Going Green: About Face." *Time*, 2010: 176(3): 45.
- WebMD. *Multiple Chemical Sensitivity*. 2010. <http://www.webmd.com/atozguides/multiplechemicalsensitivity>.
- Willhite, C. C., and P. Mirkes. "Developmental toxicology." In *Encyclopedia of Toxicology*, by P. Wexler, 742-780. Oxford: Elsevier, 2005.
- Willhite, Calvin C., Gwendolyn L. Ball, and Clifton J. McLellan. "Derivation of a Bisphenol a Oral Reference Dose (RfD) and Drinking-Water Equivalent Concentration." *Journal of Toxicology and Environmental Health*, no. Part B, 11:2 (February 2008): 69-146.
- World Health Organization. "Chemical hazards in drinking water - atrazine. Background and summary statements for the 4th edition." *Water Sanitation and Health*. 2010. http://www.who.int/water_sanitation_health/dwq/chemicals/atrazine/en/.

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