ISSUE 16

Should DDT Be Banned Worldwide?

YES: Anne Platt McGinn, from "Malaria, Mosquitoes, and DDT," *World Watch* (May/June 2002)

NO: Donald R. Roberts, from Statement before the U.S. Senate Committee on Environment & Public Works, Hearing on the Role of Science in Environmental Policy-Making (September 28, 2005)

ISSUE SUMMARY

YES: Anne Platt McGinn, a senior researcher at the Worldwatch Institute, argues that although DDT is still used to fight malaria, there are other, more effective and less environmentally harmful methods. She maintains that DDT should be banned or reserved for emergency use.

NO: Donald R. Roberts argues that the scientific evidence regarding the environmental hazards of DDT has been seriously misrepresented by anti-pesticide activists. The hazards of malaria are much greater and, properly used, DDT can prevent them and save lives.

DDT is a crucial element in the story of environmentalism. The chemical was first synthesized in 1874. Swiss entomologist Paul Mueller was the first to notice that DDT has insecticidal properties, which, it was quickly realized, implied that the chemical could save human lives. It had long been known that more soldiers died during wars because of disease than because of enemy fire. During World War I, for example, some 5 million lives were lost to typhus, a disease carried by body lice. DDT was first deployed during World War II to halt a typhus epidemic in Naples, Italy. It was a dramatic success, and DDT was soon used routinely as a dust for soldiers and civilians. During and after the war, DDT was also deployed successfully against the mosquitoes that carry malaria and other diseases. In the United States cases of malaria fell from 120,000 in 1934 to 72 in 1960, and cases of yellow fever dropped from 100,000 in 1878 to none. In 1948 Mueller received the Nobel Prize for medicine and physiology because DDT had saved so many civilian lives.

DDT was by no means the first pesticide. But its predecessors—arsenic, strychnine, cyanide, copper sulfate, and nicotine—were all markedly toxic to humans. DDT was not only more effective as an insecticide, it was also less hazardous to users. It is therefore not surprising that DDT was seen as a beneficial substance. It was soon applied routinely to agricultural crops and used to control mosquito populations in American suburbs. However, insects quickly became resistant to the insecticide; see James E. McWilliams, *American Pests: The Losing War on Insects from the Colonial Times to DDT* (Columbia University Press, 2008). (In any population of insects, some will be more resistant than others; when the insecticide kills the more vulnerable members of the population, the resistant ones are left to breed and multiply. This is an example of natural selection.) In *Silent Spring* (Houghton Mifflin, 1962), marine scientist Rachel Carson demonstrated that DDT was concentrated in the food chain and affected the reproduction of predators such as hawks and eagles. In 1972 the U.S. Environmental Protection Agency banned almost all uses of DDT (it could still be used to protect public health). Other developed countries soon banned it as well, but developing nations, especially those in the tropics, saw it as an essential tool for fighting diseases such as malaria. Roger Bate, director of Africa Fighting Malaria, argues in "A Case of the DDTs," *National Review* (May 14, 2001) that DDT remains the cheapest and most effective way to combat malaria and that it should remain available for use.

It soon became apparent that DDT is by no means the only pesticide or organic toxin with environmental effects. As a result, on May 24, 2001, the United States joined 90 other nations in signing the Stockholm Convention on Persistent Organic Pollutants (POPs). This treaty aims to eliminate from use the entire class of chemicals to which DDT belongs, beginning with the "dirty dozen," pesticides DDT, aldrin, dieldrin, endrin, chlordane, heptachlor, mirex, and toxaphene, and the industrial chemicals polychlorinated biphenyls (PCBs), hexachlorobenzene (HCB), dioxins, and furans. Since then, 59 countries, not including the United States and the European Union (EU), have formally ratified the treaty. It took effect in May 2004. Fiona Proffitt, in "U.N. Convention Targets Dirty Dozen Chemicals," *Science* (May 21, 2004), notes, "About 25 countries will be allowed to continue using DDT against malaria-spreading mosquitoes until a viable alternative is found." These countries do, however, recognize the problems inherent in using pesticides such as DDT; see e.g., P. C. Abhilash and Nandita Singh, "Pesticide Use and Application: An Indian Scenario," *Journal of Hazardous Materials* (June 2009).

In the following selection, Anne Platt McGinn, granting that malaria remains a serious problem in the developing nations of the tropics, especially Africa, contends that although DDT is still used to fight malaria in these nations, it is far less effective than it used to be. She argues that the environmental effects are also serious concerns and that DDT should be banned or reserved for emergency use. In the second selection, Professor Donald R. Roberts argues that the scientific evidence regarding the environmental hazards of DDT has been seriously misrepresented by anti-pesticide activists. The hazards of malaria are much greater and, properly used, DDT can prevent them and save lives. Efforts to prevent the use of DDT have produced a "global humanitarian disaster."

YES

Anne Platt McGinn

Malaria, Mosquitoes, and DDT

This year, like every other year within the past couple of decades, uncountable trillions of mosquitoes will inject malaria parasites into human blood streams billions of times. Some 300 to 500 million full-blown cases of malaria will result, and between 1 and 3 million people will die, most of them pregnant women and children. That's the official figure, anyway, but it's likely to be a substantial underestimate, since most malaria deaths are not formally registered, and many are likely to have escaped the estimators. Very roughly, the malaria death toll rivals that of AIDS, which now kills about 3 million people annually.

But unlike AIDS, malaria is a low-priority killer. Despite the deaths, and the fact that roughly 2.5 billion people (40 percent of the world's population) are at risk of contracting the disease, malaria is a relatively low public health priority on the international scene. Malaria rarely makes the news. And international funding for malaria research currently comes to a mere $150 million annually. Just by way of comparison, that's only about 5 percent of the $2.8 billion that the U.S. government alone is considering for AIDS research in fiscal year 2003.

The low priority assigned to malaria would be at least easier to understand, though no less mistaken, if the threat were static. Unfortunately it is not. It is true that the geographic range of the disease has contracted substantially since the mid-20th century, but over the past couple of decades, malaria has been gathering strength. Virtually all areas where the disease is endemic have seen drug-resistant strains of the parasites emerge—a development that is almost certainly boosting death rates. In countries as various as Armenia, Afghanistan, and Sierra Leone, the lack or deterioration of basic infrastructure has created a wealth of new breeding sites for the mosquitoes that spread the disease. The rapidly expanding slums of many tropical cities also lack such infrastructure; poor sanitation and crowding have primed these places as well for outbreaks—even though malaria has up to now been regarded as predominantly a rural disease.

What has current policy to offer in the face of these threats? The medical arsenal is limited; there are only about a dozen antimalarial drugs commonly in use, and there is significant malaria resistance to most of them. In the absence of a reliable way to kill the parasites, policy has tended to focus on killing the mosquitoes that bear them. And that has led to an abundant use of synthetic pesticides, including one of the oldest and most dangerous: dichlorodiphenyl trichloroethane, or DDT.

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DDT is no longer used or manufactured in most of the world, but because it does not break down readily, it is still one of the most commonly detected pesticides in the milk of nursing mothers. DDT is also one of the "dirty dozen" chemicals included in the 2001 Stockholm Convention on Persistent Organic Pollutants [POPs]. The signatories to the "POPs Treaty" essentially agreed to ban all uses of DDT except as a last resort against disease-bearing mosquitoes. Unfortunately, however, DDT is still a routine option in 19 countries, most of them in Africa. (Only 11 of these countries have thus far signed the treaty.) Among the signatory countries, 31—slightly fewer than one-third—have given notice that they are reserving the right to use DDT against malaria. On the face of it, such use may seem unavoidable, but there are good reasons for thinking that progress against the disease is compatible with *reductions* in DDT use.

Malaria is caused by four protozoan parasite species in the genus *Plasmodium.* These parasites are spread exclusively by certain mosquitoes in the genus *Anopheles.* An infection begins when a parasite-laden female mosquito settles onto someone's skin and pierces a capillary to take her blood meal. The parasite, in a form called the *sporozoite,* moves with the mosquito's saliva into the human bloodstream. About 10 percent of the mosquito's lode of sporozoites is likely to be injected during a meal, leaving plenty for the next bite. Unless the victim has some immunity to malaria—normally as a result of previous exposure—most sporozoites are likely to evade the body's immune system and make their way to the liver, a process that takes less than an hour. There they invade the liver cells and multiply asexually for about two weeks. By this time, the original several dozen sporozoites have become millions of *merozoites*—the form the parasite takes when it emerges from the liver and moves back into the blood to invade the body's red blood cells. Within the red blood cells, the merozoites go through another cycle of asexual reproduction, after which the cells burst and release millions of additional merozoites, which invade yet more red blood cells. The high fever and chills associated with malaria are the result of this stage, which tends to occur in pulses. If enough red blood cells are destroyed in one of these pulses, the result is convulsions, difficulty in breathing, coma, and death.

As the parasite multiplies inside the red blood cells, it produces not just more merozoites, but also *gametocytes,* which are capable of sexual reproduction. This occurs when the parasite moves back into the mosquitoes; even as they inject sporozoites, biting mosquitoes may ingest gametocytes if they are feeding on a person who is already infected. The gametocytes reproduce in the insect's gut and the resulting eggs move into the gut cells. Eventually, more sporozoites emerge from the gut and penetrate the mosquito's salivary glands, where they await a chance to enter another human bloodstream, to begin the cycle again.

Of the roughly 380 mosquito species in the genus *Anopheles,* about 60 are able to transmit malaria to people. These malaria vectors are widespread

throughout the tropics and warm temperate zones, and they are very efficient at spreading the disease. Malaria is highly contagious, as is apparent from a measurement that epidemiologists call the "basic reproduction number," or BRN. The BRN indicates, on average, how many new cases a single infected person is likely to cause. For example, among the nonvectored diseases (those in which the pathogen travels directly from person to person without an intermediary like a mosquito), measles is one of the most contagious. The BRN for measles is 12 to 14, meaning that someone with measles is likely to infect 12 to 14 other people. (Luckily, there's an inherent limit in this process: as a pathogen spreads through any particular area, it will encounter fewer and fewer susceptible people who aren't already sick, and the outbreak will eventually subside.) HIV/AIDS is on the other end of the scale: it's deadly, but it burns through a population slowly. Its BRN is just above 1, the minimum necessary for the pathogen's survival. With malaria, the BRN varies considerably, depending on such factors as which mosquito species are present in an area and what the temperatures are. (Warmer is worse, since the parasites mature more quickly.) But malaria can have a BRN in excess of 100: over an adult life that may last about a week, a single, malaria-laden mosquito could conceivably infect more than 100 people.

Seven Years, Seven Months

"Malaria" comes from the Italian "mal'aria." For centuries, European physicians had attributed the disease to "bad air." Apart from a tradition of associating bad air with swamps—a useful prejudice, given the amount of mosquito habitat in swamps—early medicine was largely ineffective against the disease. It wasn't until 1897 that the British physician Ronald Ross proved that mosquitoes carry malaria.

The practical implications of Ross's discovery did not go unnoticed. For example, the U.S. administration of Theodore Roosevelt recognized malaria and yellow fever (another mosquito-vectored disease) as perhaps the most serious obstacles to the construction of the Panama Canal. This was hardly a surprising conclusion, since the earlier and unsuccessful French attempt to build the canal—an effort that predated Ross's discovery—is thought to have lost between 10,000 and 20,000 workers to disease. So the American workers draped their water supplies and living quarters with mosquito netting, attempted to fill in or drain swamps, installed sewers, poured oil into standing water, and conducted mosquito-swatting campaigns. And it worked: the incidence of malaria declined. In 1906, 80 percent of the workers had the disease; by 1913, a year before the Canal was completed, only 7 percent did. Malaria could be suppressed, it seemed, with a great deal of mosquito netting, and by eliminating as much mosquito habitat as possible. But the labor involved in that effort could be enormous.

That is why DDT proved so appealing. In 1939, the Swiss chemist Paul Müller discovered that this chemical was a potent pesticide. DDT was first used during World War II, as a delousing agent. Later on, areas in southern Europe, North Africa, and Asia were fogged with DDT, to clear malaria-laden mosquitoes

from the paths of invading Allied troops. DDT was cheap and it seemed to be harmless to anything other than insects. It was also long-lasting: most other insecticides lost their potency in a few days, but in the early years of its use, the effects of a single dose of DDT could last for up to six months. In 1948, Müller won a Nobel Prize for his work and DDT was hailed as a chemical miracle.

A decade later, DDT had inspired another kind of war—a general assault on malaria. The "Global Malaria Eradication Program," launched in 1955, became one of the first major undertakings of the newly created World Health Organization [WHO]. Some 65 nations enlisted in the cause. Funding for DDT factories was donated to poor countries and production of the insecticide climbed.

The malaria eradication strategy was not to kill every single mosquito, but to suppress their populations and shorten the lifespans of any survivors, so that the parasite would not have time to develop within them. If the mosquitoes could be kept down long enough, the parasites would eventually disappear from the human population. In any particular area, the process was expected to take three years—time enough for all infected people either to recover or die. After that, a resurgence of mosquitoes would be merely an annoyance, rather than a threat. And initially, the strategy seemed to be working. It proved especially effective on islands—relatively small areas insulated from reinfestation. Taiwan, Jamaica, and Sardinia were soon declared malaria-free and have remained so to this day. By 1961, arguably the year at which the program had peak momentum, malaria had been eliminated or dramatically reduced in 37 countries.

One year later, Rachel Carson published *Silent Spring,* her landmark study of the ecological damage caused by the widespread use of DDT and other pesticides. Like other organochlorine pesticides, DDT bioaccumulates. It's fat soluble, so when an animal ingests it—by browsing contaminated vegetation, for example—the chemical tends to concentrate in its fat, instead of being excreted. When another animal eats that animal, it is likely to absorb the prey's burden of DDT. This process leads to an increasing concentration of DDT in the higher links of the food chain. And since DDT has a high chronic toxicity—that is, long-term exposure is likely to cause various physiological abnormalities—this bioaccumulation has profound implications for both ecological and human health.

With the miseries of malaria in full view, the managers of the eradication campaign didn't worry much about the toxicity of DDT, but they were greatly concerned about another aspect of the pesticide's effects: resistance. Continual exposure to an insecticide tends to "breed" insect populations that are at least partially immune to the poison. Resistance to DDT had been reported as early as 1946. The campaign managers knew that in mosquitoes, regular exposure to DDT tended to produce widespread resistance in four to seven years. Since it took three years to clear malaria from a human population, that didn't leave a lot of leeway for the eradication effort. As it turned out, the logistics simply couldn't be made to work in large, heavily infested areas with high human populations, poor housing and roads, and generally minimal infrastructure. In 1969, the campaign was abandoned. Today, DDT resistance is widespread in *Anopheles,* as is resistance to many more recent pesticides.

Undoubtedly, the campaign saved millions of lives, and it did clear malaria from some areas. But its broadest legacy has been of much more dubious value. It engendered the idea of DDT as a first resort against mosquitoes and it established the unstable dynamic of DDT resistance in *Anopheles* populations. In mosquitoes, the genetic mechanism that confers resistance to DDT does not usually come at any great competitive "cost"—that is, when no DDT is being sprayed, the resistant mosquitoes may do just about as well as non-resistant mosquitoes. So once a population acquires resistance, the trait is not likely to disappear even if DDT isn't used for years. If DDT is reapplied to such a population, widespread resistance will reappear very rapidly. The rule of thumb among entomologists is that you may get seven years of resistance-free use the first time around, but you only get about seven months the second time. Even that limited respite, however, is enough to make the chemical an attractive option as an emergency measure—or to keep it in the arsenals of bureaucracies committed to its use.

Malaria Taxes

In December 2000, the POPs Treaty negotiators convened in Johannesburg, South Africa, even though, by an unfortunate coincidence, South Africa had suffered a potentially embarrassing setback earlier that year in its own POPs policies. In 1996, South Africa had switched its mosquito control programs from DDT to a less persistent group of pesticides known as pyrethroids. The move seemed solid and supportable at the time, since years of DDT use had greatly reduced *Anopheles* populations and largely eliminated one of the most troublesome local vectors, the appropriately named *A. funestus* ("funestus" means deadly). South Africa seemed to have beaten the DDT habit: the chemical had been used to achieve a worthwhile objective; it had then been discarded. And the plan worked—until a year before the POPs summit, when malaria infections rose to 61,000 cases, a level not seen in decades. *A. funestus* reappeared as well, in KwaZulu-Natal, and in a form resistant to pyrethroids. In early 2000, DDT was reintroduced, in an indoor spraying program. (This is now a standard way of using DDT for mosquito control; the pesticide is usually applied only to walls, where mosquitoes alight to rest.) By the middle of the year, the number of infections had dropped by half.

Initially, the spraying program was criticized, but what reasonable alternative was there? This is said to be the African predicament, and yet the South African situation is hardly representative of sub-Saharan Africa as a whole.

Malaria is considered endemic in 105 countries throughout the tropics and warm temperate zones, but by far the worst region for the disease is sub-Saharan Africa. The deadliest of the four parasite species, *Plasmodium falciparum,* is widespread throughout this region, as is one of the world's most effective malaria vectors, *Anopheles gambiae.* Nearly half the population of sub-Saharan Africa is at risk of infection, and in much of eastern and central Africa, and pockets of west Africa, it would be difficult to find anyone who has not been exposed to the parasites. Some 90 percent of the world's malaria infections and deaths occur in sub-Saharan Africa, and the disease now accounts

for 30 percent of African childhood mortality. It is true that malaria is a grave problem in many parts of the world, but the African experience is misery on a very different order of magnitude. The average Tanzanian suffers more infective bites each *night* than the average Thai or Vietnamese does in a year.

As a broad social burden, malaria is thought to cost Africa between $3 billion and $12 billion annually. According to one economic analysis, if the disease had been eradicated in 1965, Africa's GDP would now be 35 percent higher than it currently is. Africa was also the gaping hole in the global eradication program: the WHO planners thought there was little they could do on the continent and limited efforts to Ethiopia, Zimbabwe, and South Africa, where eradication was thought to be feasible.

But even though the campaign largely passed Africa by, DDT has not. Many African countries have used DDT for mosquito control in indoor spraying programs, but the primary use of DDT on the continent has been as an agricultural insecticide. Consequently, in parts of west Africa especially, DDT resistance is now widespread in *A. gambiae.* But even if *A. gambiae* were not resistant, a full-bore campaign to suppress it would probably accomplish little, because this mosquito is so efficient at transmitting malaria. Unlike most *Anopheles* species, *A. gambiae* specializes in human blood, so even a small population would keep the disease in circulation. One way to get a sense for this problem is to consider the "transmission index"—the threshold number of mosquito bites necessary to perpetuate the disease. In Africa, the index overall is 1 bite per person per month. That's all that's necessary to keep malaria in circulation. In India, by comparison, the TI is 10 bites per person per month.

And yet Africa is not a lost cause—it's simply that the key to progress does not lie in the general suppression of mosquito populations. Instead of spraying, the most promising African programs rely primarily on "bednets"—mosquito netting that is treated with an insecticide, usually a pyrethroid, and that is suspended over a person's bed. Bednets can't eliminate malaria, but they can "deflect" much of the burden. Because *Anopheles* species generally feed in the evening and at night, a bednet can radically reduce the number of infective bites a person receives. Such a person would probably still be infected from time to time, but would usually be able to lead a normal life.

In effect, therefore, bednets can substantially reduce the disease. Trials in the use of bednets for children have shown a decline in malaria-induced mortality by 25 to 40 percent. Infection levels and the incidence of severe anemia also declined. In Kenya, a recent study has shown that pregnant women who use bednets tend to give birth to healthier babies. In parts of Chad, Mali, Burkina Faso, and Senegal, bednets are becoming standard household items. In the tiny west African nation of The Gambia, somewhere between 50 and 80 percent of the population has bednets.

Bednets are hardly a panacea. They have to be used properly and retreated with insecticide occasionally. And there is still the problem of insecticide resistance, although the nets themselves are hardly likely to be the main cause of it. (Pyrethroids are used extensively in agriculture as well.) Nevertheless, bednets can help transform malaria from a chronic disaster to a manageable public health problem—something a healthcare system can cope with.

So it's unfortunate that in much of central and southern Africa, the nets are a rarity. It's even more unfortunate that, in 28 African countries, they're taxed or subject to import tariffs. Most of the people in these countries would have trouble paying for a net even without the tax. This problem was addressed in the May 2000 "Abuja Declaration," a summit agreement on infectious diseases signed by 44 African countries. The Declaration included a pledge to do away with "malaria taxes." At last count, 13 countries have actually acted on the pledge, although in some cases only by reducing rather than eliminating the taxes. Since the Declaration was signed, an estimated 2 to 5 million Africans have died from malaria.

This failure to follow through with the Abuja Declaration casts the interest in DDT in a rather poor light. Of the 31 POPs treaty signatories that have reserved the right to use DDT, 21 are in Africa. Of those 21, 10 are apparently still taxing or imposing tariffs on bednets. (Among the African countries that have *not* signed the POPs treaty, some are almost certainly both using DDT and taxing bednets, but the exact number is difficult to ascertain because the status of DDT use is not always clear.) It is true that a case can be made for the use of DDT in situations like the one in South Africa in 1999—an infrequent flare-up in a context that lends itself to control. But the routine use of DDT against malaria is an exercise in toxic futility, especially when it's pursued at the expense of a superior and far more benign technology.

Learning to Live with the Mosquitoes

A group of French researchers recently announced some very encouraging results for a new anti-malarial drug known as G25. The drug was given to infected aotus monkeys, and it appears to have cleared the parasites from their systems. Although extensive testing will be necessary before it is known whether the drug can be safely given to people, these results have raised the hope of a cure for the disease.

Of course, it would be wonderful if G25, or some other new drug, lives up to that promise. But even in the absence of a cure, there are opportunities for progress that may one day make the current incidence of malaria look like some dark age horror. Many of these opportunities have been incorporated into an initiative that began in 1998, called the Roll Back Malaria (RBM) campaign, a collaborative effort between WHO, the World Bank, UNICEF, and the UNDP [United Nations Development Programme]. In contrast to the earlier WHO eradication program, RBM grew out of joint efforts between WHO and various African governments specifically to address African malaria. RBM focuses on household- and community-level intervention and it emphasizes apparently modest changes that could yield major progress. Below are four "operating principles" that are, in one way or another, implicit in RBM or likely to reinforce its progress.

1. Do away with all taxes and tariffs on bednets, on pesticides intended for treating bednets, and on antimalarial drugs. Failure to act on this front certainly undercuts claims for the necessity of DDT; it may also undercut claims for antimalaria foreign aid.

2. Emphasize appropriate technologies. Where, for example, the need for mud to replaster walls is creating lots of pothole sized cavities near houses—cavities that fill with water and then with mosquito larvae—it makes more sense to help people improve their housing maintenance than it does to set up a program for squirting pesticide into every pothole. To be "appropriate," a technology has to be both affordable and culturally acceptable. Improving home maintenance should pass this test; so should bednets. And of course there are many other possibilities. In Kenya, for example, a research institution called the International Center for Insect Physiology and Ecology has identified at least a dozen native east African plants that repel *Anopheles gambiae* in lab tests. Some of these plants could be important additions to household gardens.

3. Use existing networks whenever possible, instead of building new ones. In Tanzania, for example, an established healthcare program (UNICEF's Integrated Management of Childhood Illness Program) now dispenses antimalarial drugs—and instruction on how to use them. The UNICEF program was already operating, so it was simple and cheap to add the malaria component. Reported instances of severe malaria and anemia in infants have declined, apparently as a result. In Zambia, the government is planning to use health and prenatal clinics as the network for a coupon system that subsidizes bednets for the poor. Qualifying patients would pick up coupons at the clinics and redeem them at stores for the nets.

4. Assume that sound policy will involve action on many fronts. Malaria is not just a health problem—it's a social problem, an economic problem, an environmental problem, an agricultural problem, an urban planning problem. Health officials alone cannot possibly just make it go away. When the disease flares up, there is a strong and understandable temptation to strap on the spray equipment and douse the mosquitoes. But if this approach actually worked, we wouldn't be in this situation today. Arguably the biggest opportunity for progress against the disease lies, not in our capacity for chemical innovation, but in our capacity for *organizational innovation*—in our ability to build an awareness of the threat across a broad range of policy activities. For example, when government officials are considering loans to irrigation projects, they should be asking: has the potential for malaria been addressed? When foreign donors are designing antipoverty programs, they should be asking: do people need bednets? Routine inquiries of this sort could go a vast distance to reducing the disease.

Where is the DDT in all of this? There isn't any, and that's the point. We now have half a century of evidence that routine use of DDT simply will not prevail against the mosquitoes. Most countries have already absorbed this lesson, and banned the chemical or relegated it to emergency only status. Now the RBM campaign and associated efforts are showing that the frequency and intensity of those emergencies can be reduced through systematic attention to the chronic aspects of the disease. There is less and less justification for DDT, and the futility of using it as a matter of routine is becoming increasingly apparent: in order to control a disease, why should we poison our soils, our waters, and ourselves?

Donald R. Roberts

NO

Statement before the U.S. Senate Committee on Environment & Public Works, Hearing on the Role of Science in Environmental Policy-Making

Thank you, Chairman Inhofe, and distinguished members of the Committee on Environment and Public Works, for the opportunity to present my views on the misuse of science in public policy. My testimony focuses on misrepresentations of science during decades of environmental campaigning against DDT.

Before discussing how and why DDT science has been misrepresented, you first must understand why this misrepresentation has not helped, but rather harmed, millions of people every year all over the world. Specifically you need to understand why the misrepresentation of DDT science has been and continues to be deadly. By way of explanation, I will tell you something of my experience.

I conducted malaria research in the Amazon Basin in the 1970s. My Brazilian colleague—who is now the Secretary of Health for Amazonas State—and I worked out of Manaus, the capitol of Amazonas State. From Manaus we traveled two days to a study site where we had sufficient numbers of cases for epidemiological studies. There were no cases in Manaus, or anywhere near Manaus. For years before my time there and for years thereafter, there were essentially no cases of malaria in Manaus. However, in the late 1980s, environmentalists and international guidelines forced Brazilians to reduce and then stop spraying small amounts of DDT inside houses for malaria control. As a result, in 2002 and 2003 there were over 100,000 malaria cases in Manaus alone.

Brazil does not stand as the single example of this phenomenon. A similar pattern of declining use of DDT and reemerging malaria occurs in other countries as well, Peru for example. Similar resurgences of malaria have occurred in rural communities, villages, towns, cities, and countries around the world. As illustrated by the return of malaria in Russia, South Korea, urban areas of the Amazon Basin, and increasing frequencies of outbreaks in the United States, our malaria problems are growing worse. Today there are 1 to 2 million malaria deaths each year and hundreds of millions of cases. The poorest of the world's people are at greatest risk. Of these, children and pregnant women are the ones most likely to die.

U.S. Senate Committee on Environment and Public Works, September 28, 2005.

We have long known about DDT's effectiveness in curbing insect-borne disease. Othmar Zeidler, a German chemistry student, first synthesized DDT in 1874. Over sixty years later in Switzerland, Paul Müller discovered the insecticidal property of DDT. Allied forces used DDT during WWII, and the new insecticide gained fame in 1943 by successfully stopping an epidemic of typhus in Naples, an unprecedented achievement. By the end of the war, British, Italian, and American scientists had also demonstrated the effectiveness of DDT in controlling malaria-carrying mosquitoes. DDT's proven efficacy against insect-borne diseases, diseases that had long reigned unchecked throughout the world, won Müller the Nobel Prize for Medicine in 1948. After WWII, the United States conducted a National Malaria Eradication Program, commencing operations on July 1, 1947. The spraying of DDT on internal walls of rural homes in malaria endemic counties was a key component of the program. By the end of 1949, the program had sprayed over 4,650,000 houses. This spraying broke the cycle of malaria transmission, and in 1949 the United States was declared free of malaria as a significant public health problem. Other countries had already adopted DDT to eradicate or control malaria, because wherever malaria control programs sprayed DDT on house walls, the malaria rates dropped precipitously. The effectiveness of DDT stimulated some countries to create, for the first time, a national malaria control program. Countries with pre-existing programs expanded them to accommodate the spraying of houses in rural areas with DDT. Those program expansions highlight what DDT offered then, and still offers now, to the malaria endemic countries. As a 1945 U.S. Public Health Service manual explained about the control of malaria: "Drainage and larviciding are the methods of choice in towns of 2,500 or more people. But malaria is a rural disease. Heretofore there has been no economically feasible method of carrying malaria control to the individual tenant farmer or sharecropper. Now, for the first time, a method is available—the application of DDT residual spray to walls and ceilings of homes." Health workers in the United States were not the only ones to recognize the particular value of DDT. The head of malaria control in Brazil characterized the changes that DDT offered in the following statement: "Until 1945–1946, preventive methods employed against malaria in Brazil, as in the rest of the world, were generally directed against the aquatic phases of the vectors (draining, larvicides, destruction of bromeliads, etc. . . .). These methods, however, were only applied in the principal cities of each state and the only measure available for rural populations exposed to malaria was free distribution of specific drugs."

DDT was a new, effective, and exciting weapon in the battle against malaria. It was cheap, easy to apply, long-lasting once sprayed on house walls, and safe for humans. Wherever and whenever malaria control programs sprayed it on house walls, they achieved rapid and large reductions in malaria rates. Just as there was a rush to quickly make use of DDT to control disease, there was also a rush to judge how DDT actually functioned to control malaria. That rush to judgment turned out to be a disaster. At the heart of the debate—to the extent there was a debate—was a broadly accepted model that established a mathematical framework for using DDT to kill mosquitoes and eradicate malaria. Instead of studying real data to see how DDT

actually worked in controlling malaria, some scientists settled upon what they thought was a logical conclusion: DDT worked solely by killing mosquitoes. This conclusion was based on their belief in the model. Scientists who showed that DDT did not function by killing mosquitoes were ignored. Broad acceptance of the mathematical model led to strong convictions about DDT's toxic actions. Since they were convinced that DDT worked only by killing mosquitoes, malaria control specialists became very alarmed when a mosquito was reported to be resistant to DDT's toxic actions. As a result of concern about DDT resistance, officials decided to make rapid use of DDT before problems of resistance could eliminate their option to use DDT to eradicate malaria. This decision led to creation of the global malaria eradication program. The active years of the global malaria eradication program were from 1959 to 1969. Before, during, and after the many years of this program, malaria workers and researchers carried out their responsibilities to conduct studies and report their research. Through those studies, they commonly found that DDT was functioning in ways other than by killing mosquitoes. In essence, they found that DDT was functioning through mechanisms of repellency and irritancy. Eventually, as people forgot early observations of DDT's repellent actions, some erroneously interpreted new findings of repellent actions as the mosquitoes' adaptation to avoid DDT toxicity, even coining a term, "behavioral resistance," to explain what they saw. This new term accommodated their view that toxicity was DDT's primary mode of action and categorized behavioral responses of mosquitoes as mere adaptations to toxic affects. However this interpretation depended upon a highly selective use of scientific data. The truth is that toxicity is not DDT's primary mode of action when sprayed on house walls. Throughout the history of DDT use in malaria control programs there has always been clear and persuasive data that DDT functioned primarily as a spatial repellent. Today we know that there is no insecticide recommended for malaria control that rivals, much less equals, DDT's spatial repellent actions, or that is as long-acting, as cheap, as easy to apply, as safe for human exposure, or as efficacious in the control of malaria as DDT. . . . The 30 years of data from control programs of the Americas plotted . . . illustrate just how effective DDT is in malaria control. The period 1960s through 1979 displays a pattern of malaria controlled through house spraying. In 1979 the World Health Organization (WHO) changed its strategy for malaria control, switching emphasis from spraying houses to case detection and treatment. In other words, the WHO changed emphasis from malaria prevention to malaria treatment. Countries complied with WHO guidelines and started to dismantle their spray programs over the next several years. . . .

I find it amazing that many who oppose the use of DDT describe its earlier use as a failure. Our own citizens who suffered under the burden of malaria, especially in the rural south, would hardly describe it thus.

Malaria was a serious problem in the United States and for some localities, such as Dunklin County, Missouri, it was a very serious problem indeed. For four counties in Missouri, the average malaria mortality from 1910 to 1914 was 168.8 per 100,000 population. For Dunklin County, it was 296.7 per 100,000, a rate almost equal to malaria deaths in Venezuela and actually greater than the

mortality rate for Freetown, Sierra Leone. Other localities in other states were equally as malarious. Growing wealth and improved living conditions were gradually reducing malaria rates, but cases resurged during WWII. The advent of DDT, however, quickly eradicated malaria from the United States.

DDT routed malaria from many other countries as well. The Europeans who were freed of malaria would hardly describe its use as a failure. After DDT was introduced to malaria control in Sri Lanka (then Ceylon), the number of malaria cases fell from 2.8 million in 1946 to just 110 in 1961. Similar spectacular decreases in malaria cases and deaths were seen in all the regions that began to use DDT. The newly formed Republic of China (Taiwan) adopted DDT use in malaria control shortly after World War II. In 1945 there were over 1 million cases of malaria on the island. By 1969 there were only 9 cases and shortly thereafter the disease was eradicated from the island and remains so to this day. Some countries were less fortunate. South Korea used DDT to eradicate malaria, but without house spray programs, malaria has returned across the demilitarized zone with North Korea. As DDT was eliminated and control programs reduced, malaria has returned to other countries such as Russia and Argentina. Small outbreaks of malaria are even beginning to appear more frequently in the United States.

These observations have been offered in testimony to document first that there were fundamental misunderstandings about how DDT functioned to exert control over malaria. Second, that regardless of systematic misunderstandings on the part of those who had influence over malaria control strategies and policies, there was an enduring understanding that DDT was the most cost-effective compound yet discovered for protecting poor rural populations from insect-borne diseases like malaria, dengue, yellow fever, and leishmaniasis. I want to emphasize that misunderstanding the mode of DDT action did not lead to the wholesale abandonment of DDT. It took an entirely new dimension in the misuse of science to bring us to the current humanitarian disaster represented by DDT elimination.

The misuse of science to which I refer has found fullest expression in the collection of movements within the environmental movement that seek to stop production and use of specific man-made chemicals. Operatives within these movements employ particular strategies to achieve their objectives. By characterizing and understanding the strategies these operatives use, we can identify their impact in the scientific literature or in the popular press.

The first strategy is to develop and then distribute as widely as possible a broad list of claims of chemical harm. This is a sound strategy because individual scientists can seldom rebut the scientific foundations of multiple and diverse claims. Scientists generally develop expertise in a single, narrow field and are disinclined to engage issues beyond their area of expertise. Even if an authoritative rebuttal of one claim occurs, the other claims still progress. A broad list of claims also allows operatives to tailor platforms for constituencies, advancing one set of claims with one constituency and a different combination for another. Clever though this technique is, a list of multiple claims of harm is hardly sufficient to achieve the objective of a ban. The second strategy then is to mount an argument that the chemical is not needed and propose

that alternative chemicals or methods can be used instead. The third strategy is to predict that grave harm will occur if the chemical continues to be used.

The success of Rachel Carson's *Silent Spring* serves as a model for this tricky triad. In *Silent Spring*, Rachel Carson used all three strategies on her primary target, DDT. She described a very large list of potential adverse effects of insecticides, DDT in particular. She argued that insecticides were not really needed and that the use of insecticides produces insects that are insecticide resistant, which only exacerbates the insect control problems. She predicted scary scenarios of severe harm with continued use of DDT and other insecticides. Many have written rebuttals to Rachel Carson and others who have, without scientific justification, broadcast long lists of potential harms of insecticides. . . .

[T]ime and science have discredited most of Carson's claims. Rachel Carson's descriptions of inappropriate uses of insecticides that harmed wildlife are more plausible. However, harm from an inappropriate use does not meet the requirements of anti-pesticide activists. They can hardly lobby for eliminating a chemical because someone used it wrongly. No, success requires that even the proper use of an insecticide will cause a large and systematic adverse effect. However, the proper uses of DDT yield no large and systematic adverse effects. Absent such adverse actions, the activists must then rely on claims about insidious effects, particularly insidious effects that scientists will find difficult to prove one way or the other and that activists can use to predict a future catastrophe.

Rachel Carson relied heavily on possible insidious chemical actions to alarm and frighten the public. Many of those who joined her campaign to ban DDT and other insecticides made extensive use of claims of insidious effects. These claims were amplified by the popular press and became part of the public perception about modern uses of chemicals. For example, four well-publicized claims about DDT were:

1. DDT will cause the obliteration of higher trophic levels. If not obliterated, populations will undergo reproductive failure. Authors of this claim speculated that, even if the use of DDT were stopped, systematic and ongoing obliterations would still occur.
2. DDT causes the death of algae. This report led to speculations that use of DDT could result in global depletion of oxygen.
3. DDT pushed the Bermuda petrel to the verge of extinction and that full extinction might happen by 1978.
4. DDT was a cause of premature births in California sea lions.

Science magazine, the most prestigious science journal in the United States, published these and other phantasmagorical allegations and/or predictions of DDT harm. Nonetheless, history has shown that each and every one of these claims and predictions were false.

1. The obliteration of higher trophic levels did not occur; no species became extinct; and levels of DDT in all living organisms declined precipitously after DDT was de-listed for use in agriculture. How could the prediction have been so wrong? Perhaps it was so wrong because the paper touting this view used a predictive model based on an assumption of no DDT degradation. This was a startling assumption even at the time as *Science* and other journals had previously published papers that showed DDT was ubiquitously degraded in the environment and in living creatures. It was even more startling that *Science* published a paper that flew so comprehensively in the face of previous data and analysis.
2. DDT's action against algae reportedly occurred at concentrations of 500 parts per billion. But DDT cannot reach concentrations in water higher than about 1.2 parts per billion, the saturation point of DDT in water.
3. Data on the Bermuda petrel did not show a cause-and-effect relationship between low numbers of birds and DDT concentrations. DDT had no affect on population numbers, for populations increased before DDT was de-listed for use in agriculture and after DDT was de-listed as well.
4. Data gathered in subsequent years showed that "despite relatively high concentrations [of DDT], no evidence that population growth or the health of individual California sea lions have been compromised. The population has increased throughout the century, including the period when DDT was being manufactured, used, and its wastes discharged off southern California."

If time and science have refuted all these catastrophic predictions, why do many scientists and the public not know these predictions were false? In part, we do not know the predictions were false because the refutations of such claims rarely appear in the literature.

When scientists hear the kinds of claims described above, they initiate research to confirm or refute the claims. After Charles Wurster published his claim that DDT kills algae and impacts photosynthesis, I initiated research on planktonic algae to quantify DDT's effects. From 1968–1969, I spent a year of honest and demanding research effort to discover that not enough DDT would even go into solution for a measurable adverse effect on planktonic algae. In essence, I conducted a confirmatory study that failed to confirm an expected result. I had negative data, and journals rarely accept negative data for publication. My year was practically wasted. Without a doubt, hundreds of other scientists around the world have conducted similar studies and obtained negative results, and they too were unable to publish their experimental findings. Much in the environmental science literature during the last 20–30 years indicates that an enormous research effort went into proving specific insidious effects of DDT and other insecticides. Sadly, the true magnitude of such efforts will never be known because while the positive results of research find their way into the scientific literature, the negative results rarely do. Research on insidious actions that produce negative results all too often ends up only in laboratory and field notebooks and is forgotten. For this reason, I place considerable weight on a published confirmatory study that fails to confirm an expected result.

The use of the tricky triad continues. A . . . recent paper . . . published in *The Lancet* illustrates the triad's modern application. Two scientists at the National

Institute of Environmental Health Sciences, Walter Rogan and Aimin Chen, wrote this paper, entitled "Health risks and benefits of bis(4-chlorophenyl)-1,1, 1-trichloroethane (DDT)." It is interesting to see how this single paper spins all three strategies that gained prominence in Rachel Carson's *Silent Spring*.

The journal *Emerging Infectious Diseases* had already published a slim version of this paper, which international colleagues and I promptly rebutted. The authors then filled in some parts, added to the claims of harm, and republished the paper in the British journal, *The Lancet*. To get the paper accepted by editors, the authors described studies that support (positive results) as well as studies that do not support (negative results) each claim. Complying with strategy number 1 of the triad, Rogan and Chen produce a long list of possible harms, including the charge that DDT causes cancer in nonhuman primates. The literature reference for Rogan and Chen's claim that DDT causes cancer in nonhuman primates was a paper by Takayama et al. Takayama and coauthors actually concluded from their research on the carcinogenic effect of DDT in nonhuman primates that "the two cases involving malignant tumors of different types are inconclusive with respect to a carcinogenic effect of DDT in nonhuman primates." Clearly, the people who made the link of DDT with cancer were not the scientists who actually conducted the research.

The authors enacted strategy number two of the triad by conducting a superficial review of the role of DDT in malaria control with the goal of discrediting DDT's value in modern malaria control programs. The authors admitted that DDT had been very effective in the past, but then argued that malaria control programs no longer needed it and should use alternative methods of control. Their use of the second strategy reveals, in my opinion, the greatest danger of granting authority to anti-pesticide activists and their writings. As *The Lancet* paper reveals, the NIEHS scientists assert great authority over the topic of DDT, yet they assume no responsibility for the harm that might result from their erroneous conclusions. After many malaria control specialists have expressed the necessity for DDT in malaria control, it is possible for Rogan and Chen to conclude that DDT is not necessary in malaria control only if they have no sense of responsibility for levels of disease and death that will occur if DDT is not used.

Rogan and Chen also employ the third strategy of environmentalism. Their list of potential harms caused by DDT includes toxic effects, neurobehavior effects, cancers, decrements in various facets of reproductive health, decrements in infant and child development, and immunology and DNA damage. After providing balanced coverage of diverse claims of harm, the authors had no option but to conclude they could not prove that DDT caused harm. However, they then promptly negated this honest conclusion by asserting that if DDT is used for malaria control, then great harm might occur. So, in an amazing turn, they conclude they cannot prove DDT causes harm, but still predict severe harm if it is used.

Rogan and Chen end their paper with a call for more research. One could conclude that the intent of the whole paper is merely to lobby for research to better define DDT harm, and what's the harm in that? Surely increasing knowledge is a fine goal. However, if you look at the specific issue of the relative need for

research, you will see that the harm of this technique is great. Millions of children and pregnant women die from malaria every year, and the disease sickens hundreds of millions more. This is an indisputable fact: impoverished people engage in real life and death struggles every day with malaria. This also is a fact: not one death or illness can be attributed to an environmental exposure to DDT. Yet, a National Library of Medicine literature search on DDT reveals over 1,300 published papers from the year 2000 to the present, almost all in the environmental literature and many on potential adverse effects of DDT. A search on malaria and DDT reveals only 159 papers. DDT is a spatial repellent and hardly an insecticide at all, but a search on DDT and repellents will reveal only 7 papers. Is this not an egregiously disproportionate research emphasis on non-sources of harm compared to the enormous harm of malaria? Does not this inequity contribute to the continued suffering of those who struggle with malaria? Is it possibly even more than an inequity? Is it not an active wrong?

Public health officials and scientists should not be silent about enormous investments into the research of theoretical risks while millions die of preventable diseases. We should seriously consider our motivations in apportioning research money as we do. For consider this: the U.S. used DDT to eradicate malaria. After malaria disappeared as an endemic disease in the United States, we became richer. We built better and more enclosed houses. We screened our windows and doors. We air conditioned our homes. We also developed an immense arsenal of mosquito control tools and chemicals. Today, when we have a risk of mosquito borne disease, we can bring this arsenal to bear and quickly eliminate risks. And, as illustrated by aerial spray missions in the aftermath of hurricane Katrina, we can afford to do so. Yet, our modern and very expensive chemicals are not what protect us from introductions of the old diseases. Our arsenal responds to the threat; it does not prevent the appearance of old diseases in our midst. What protects us is our enclosed, screened, air-conditioned housing, the physical representation of our wealth. Our wealth is the factor that stops dengue at the border with Mexico, not our arsenal of new chemicals. Stopping mosquitoes from entering and biting us inside our homes is critical in the prevention of malaria and many other insect-borne diseases. This is what DDT does for poor people in poor countries. It stops large proportions of mosquitoes from entering houses. It is, in fact, a form of chemical screening, and until these people can afford physical screening or it is provided for them, this is the only kind of screening they have.

DDT is a protective tool that has been taken away from countries around the world, mostly due to governments acceding to the whims of the anti-pesticide wing of environmentalism, but it is not only the anti-pesticide wing that lobbies against DDT. The activists have a sympathetic lobbying ally in the pesticide industry. As evidence of insecticide industry working to stop countries from using DDT, I am attaching an email message dated 23rd September and authored by a Bayer official. . . . The Bayer official states

> "[I speak] Not only as the responsible manager for the vector control business in Bayer, being the market leader in vector control and pointing out by that we know what we are talking about and have decades of

> experiences in the evolution of this very particular market. [but] Also as one of the private sector representatives in the RBM Partnership Board and being confronted with that discussion about DDT in the various WHO, RBM et al circles. So you can take it as a view from the field, from the operational commercial level—but our companies [sic] point of view. I know that all of my colleagues from other primary manufacturers and internationally operating companies are sharing my view."
>
> The official goes on to say that
>
> "DDT use is for us a commercial threat (which is clear, but it is not that dramatical because of limited use), it is mainly a public image threat."
>
> However the most damning part of this message was the statement that
>
> "we fully support EU to ban imports of agricultural products coming from countries using DDT"

[There is] . . . clear evidence of international and developed country pressures to stop poor countries from using DDT to control malaria. This message also shows the complicity of the insecticide industry in those internationally orchestrated efforts.

Pressures to eliminate spray programs, and DDT in particular, are wrong. I say this not based on some projection of what might theoretically happen in the future according to some model, or some projection of theoretical harms, I say this based firmly on what has already occurred. The track record of the anti-pesticide lobby is well documented, the pressures on developing countries to abandon their spray programs are well documented, and the struggles of developing countries to maintain their programs or restart their uses of DDT for malaria control are well documented. The tragic results of pressures against the use of DDT, in terms of increasing disease and death, are quantified and well documented. How long will scientists, public health officials, the voting public, and the politicians who lead us continue policies, regulations and funding that have led us to the current state of a global humanitarian disaster? How long will support continue for policies and programs that favor phantoms over facts?

POSTSCRIPT

Should DDT Be Banned Worldwide?

Professor Roberts is not alone in his disapproval of the efforts to halt the use of DDT. Alexander Gourevitch, "Better Living Through Chemistry," *Washington Monthly* (March 2003), comes close to accusing environmentalists of condemning DDT on the basis of politics or ideology rather than of science. Angela Logomasini comes even closer in "Chemical Warfare: Ideological Environmentalism's Quixotic Campaign Against Synthetic Chemicals," in Ronald Bailey, ed., *Global Warming and Other Eco-Myths: How the Environmental Movement Uses False Science to Scare Us to Death* (Prima, 2002). Her admission that public health demands have softened some environmentalists' resistance to the use of DDT points to a basic truth about environmental debates: over and over again, they come down to what we should do first: Should we meet human needs regardless of whether or not species die and air and water are contaminated? Or should we protect species, air, water, and other aspects of the environment even if some human needs must go unmet? What if this means endangering the lives of children? In the debate over DDT, the human needs are clear, for insect-borne diseases have killed and continue to kill a great many people. Yet the environmental needs are also clear. The question is one of choosing priorities and balancing risks. See John Danley, "Balancing Risks: Mosquitoes, Malaria, Morality, and DDT," *Business and Society Review* (Spring 2002). It is worth noting that John Beard, "DDT and Human Health," *Science of the Total Environment* (February 2006), finds the evidence for the ill effects of DDT more convincing and says that it is still too early to say it does not contribute to human disease.

The serious nature of the malaria threat is well covered in Michael Finkel, "Bedlam in the Blood: Malaria," *National Geographic* (July 2007). Mosquitoes can be controlled in various ways: Swamps can be drained (which carries its own environmental price), and other breeding opportunities can be eliminated. Fish can be introduced to eat mosquito larvae. And mosquito nets can be used to keep the insects away from people, although bednets must be subsidized in the poorest areas and people must be taught how to use them properly; see Phoebe Williams, et al., "Malaria Prevention in Sub-Saharan Africa: A Field Study in Rural Uganda," *Journal of Community Health* (August 2009). See also Jeffrey Marlow, "Malaria: New Methods and New Hope in Battling an Old Scourge," *World Watch* (May/June 2008). But these (and other) alternatives do not mean that there does not remain a place for chemical pesticides. In "Pesticides and Public Health: Integrated Methods of Mosquito Management," *Emerging Infectious Diseases* (January–February 2001), Robert I. Rose,

an arthropod biotechnologist with the Animal and Plant Health Inspection Service of the U.S. Department of Agriculture, says, "Pesticides have a role in public health as part of sustainable integrated mosquito management. Other components of such management include surveillance, source reduction or prevention, biological control, repellents, traps, and pesticide-resistance management." "The most effective programs today rely on a range of tools," says McGinn in "Combating Malaria," *State of the World 2003* (W. W. Norton, 2003). Still, some countries see DDT as essential. See Tina Rosenberg, "What the World Needs Now Is DDT," *New York Times Magazine* (April 11, 2004). However when the World Health Organization (WHO) endorsed the use of DDT to fight malaria, there was immediate outcry; see Allan Schapira, "DDT: A Polluted Debate in Malaria Control," *Lancet* (December 16, 2006). So far, however, the outcry seems fruitless; see Apoorva Mandavilli, "DDT Is Back," *Discover* (January 2007).

It has proven difficult to find effective, affordable drugs against malaria; see Ann M. Thayer, "Fighting Malaria," *Chemical and Engineering News* (October 24, 2005), and Claire Panosian Dunavan, "Tackling Malaria," *Scientific American* (December 2005). A great deal of effort has gone into developing vaccines against malaria, but the parasite has demonstrated a persistent talent for evading all attempts to arm the immune system against it. See Z. H. Reed, M. Friede, and M. P. Kieny, "Malaria Vaccine Development: Progress and Challenges," *Current Molecular Medicine* (March 2006). A newer approach is to develop genetically engineered (transgenic) mosquitoes that either cannot support the malaria parasite or cannot infect humans with it; see George K. Christophides, "Transgenic Mosquitoes and Malarial Transmission," *Cellular Microbiology* (March 2005), and M. J. Friedrich, "Malaria Researchers Target Mosquitoes," *JAMA: Journal of the American Medical Association* (May 23, 2007). On the other hand, Nicholas J. White, "Malaria—Time to Act," *New England Journal of Medicine* (November 9, 2006), argues that rather than wait for a perfect solution, we should recognize that present tools—including DDT—are effective enough now that there is no excuse to avoid using them.

It is worth stressing that malaria is only one of several mosquito-borne diseases that pose threats to public health. Two others are yellow fever and dengue. A recent arrival to the United States is West Nile virus, which mosquitoes can transfer from birds to humans. However, West Nile virus is far less fatal than malaria, yellow fever, or dengue, and a vaccine is in development.

It is also worth stressing that global warming means climate changes that may increase the geographic range of disease-carrying mosquitoes. Many climate researchers are concerned that malaria, yellow fever, and other now mostly tropical and subtropical diseases may return to temperate-zone nations and even spread into areas where they have never been known. See Atul A. Khasnis and Mary D. Nettleman, "Global Warming and Infectious Disease," *Archives of Medical Research* (November 2005). Sarah DeWeerdt, "Climate Change, Coming Home," *World Watch* (May/June 2007), notes that "by 2020 human-triggered climate change could kill 300,000 people worldwide every year," in large part because of the spread of diseases such as malaria.

ISSUE 17

Do Environmental Hormone Mimics Pose a Potentially Serious Health Threat?

YES: Michele L. Trankina, from "The Hazards of Environmental Estrogens," *The World & I* (October 2001)

NO: Michael Gough, from "Endocrine Disrupters, Politics, Pesticides, the Cost of Food and Health," *Daily Commentary* (December 15, 1997)

ISSUE SUMMARY

YES: Professor of biological sciences Michele L. Trankina argues that a great many synthetic chemicals behave like estrogen, alter the reproductive functioning of wildlife, and may have serious health effects—including cancer—on humans.

NO: Michael Gough, a biologist and expert on risk assessment and environmental policy, argues that only "junk science" supports the hazards of environmental estrogens.

Following World War II there was an exponential growth in the industrial use and marketing of synthetic chemicals. These chemicals, known as "xenobiotics," were used in numerous products, including solvents, pesticides, refrigerants, coolants, and raw materials for plastics. This resulted in increasing environmental contamination. Many of these chemicals, such as DDT, PCBs, and dioxins, proved to be highly resistant to degradation in the environment; they accumulated in wildlife and were serious contaminants of lakes and estuaries. Carried by winds and ocean currents, these chemicals were soon detected in samples taken from the most remote regions of the planet, far from their points of introduction into the ecosphere.

Until very recently most efforts to assess the potential toxicity of synthetic chemicals to living things, including human beings, focused almost exclusively on their possible role as carcinogens. This was because of legitimate public concern about rising cancer rates and the belief that cancer causation was the most likely outcome of exposure to low levels of synthetic chemicals.

Some environmental scientists urged public health officials to give serious consideration to other possible health effects of xenobiotics. They were generally ignored because of limited funding and the common belief that toxic effects other than cancer required larger exposures than usually resulted from environmental contamination.

In the late 1980s Theo Colborn, a research scientist for the World Wildlife Fund who was then working on a study of pollution in the Great Lakes, began linking together the results of a growing series of isolated studies. Researchers in the Great Lakes region, as well as in Florida, the West Coast, and Northern Europe, had observed widespread evidence of serious and frequently lethal physiological problems involving abnormal reproductive development, unusual sexual behavior, and neurological problems exhibited by a diverse group of animal species, including fish, reptiles, amphibians, birds, and marine mammals. Through Colborn's insights, communications among these researchers, and further studies, a hypothesis was developed that all of these wildlife problems were manifestations of abnormal estrogenic activity. The causative agents were identified as more than 50 synthetic chemical compounds that have been shown in laboratory studies to either mimic the action or disrupt the normal function of the powerful estrogenic hormones responsible for female sexual development and many other biological functions.

Concern that human exposure to these ubiquitous environmental contaminants may have serious health repercussions was heightened by a widely publicized European research study, which concluded that male sperm counts had decreased by 50 percent over the past several decades (a result that is disputed by other researchers) and that testicular cancer rates have tripled. Some scientists have also proposed a link between breast cancer and estrogen disrupters.

In response to the mounting scientific evidence that environmental estrogens may be a serious health threat, the U.S. Congress passed legislation requiring that all pesticides be screened for estrogenic activity and that the Environmental Protection Agency (EPA) develop procedures for detecting environmental estrogenic contaminants in drinking water supplies; see the EPA's Endocrine Disruptor Screening Program Web site at http://www. epa.gov/scipoly/oscpendo/index.htm. In April 2009, the EPA released its list of chemicals for initial screening.

In the following selections, Michele L. Trankina argues that a great many synthetic chemicals behave like estrogen, alter the reproductive functioning of wildlife, and may have serious health effects—including cancer—on humans. She insists that regulatory agencies must minimize public exposure. Michael Gough argues that only "junk science" supports the hazards of environmental estrogens. Expensive testing and regulatory programs can only drive up the cost of food, he says, which will make it harder for the poor to afford fresh fruits and vegetables. Furthermore, health protection will not be increased.

YES

Michele L. Trankina

The Hazards of Environmental Estrogens

What do Barbie dolls, food wrap, and spermicides have in common? And what do they have to do with low sperm counts, precocious puberty, and breast cancer? "Everything," say those who support the notion that hormones are disrupting everything from fish gender to human fertility. "Nothing," counter others who regard the connection as trumped up, alarmist chemophobia. The controversy swirls around the significance of a number of substances that behave like estrogens and appear to be practically everywhere—from plastic toys to topical sunscreens.

Estrogens are a group of hormones produced in both the female ovaries and male testes, with larger amounts made in females than in males. They are particularly influential during puberty, menstruation, and pregnancy, but they also help regulate the growth of bones, skin, and other organs and tissues.

Over the past 10 years, many synthetic compounds and plant products present in the environment have been found to affect hormonal functions in various ways. Those that have estrogenic activity have been labeled as environmental estrogens, ecoestrogens, estrogen mimics, or xenoestrogens (*xenos* means foreign). Some arise as artifacts during the manufacture of plastics and other synthetic materials. Others are metabolites (breakdown products) generated from pesticides or steroid hormones used to stimulate growth in livestock. Ecoestrogens that are produced naturally by plants are called phytoestrogens (*phyton* means plant).

Many of these estrogen mimics bind to estrogen receptors (within specialized cells) with roughly the same affinity as estrogen itself, setting up the potential to wreak havoc on reproductive anatomy and physiology. They have therefore been labeled as disruptors of endocrine function.

Bizarre Changes in Reproductive Systems

Heightened concern about estrogen-mimicking substances arose when several nonmammalian vertebrates began to exhibit bizarre changes in reproductive anatomy and fertility. Evidence that something was amiss came serendipitously in 1994, from observations by reproductive physiologist Louis Guillette of the University of Florida. In the process of studying the decline of alligator populations at Lake Apopka, Florida, Guillette and coworkers noticed that many male alligators had smaller penises than normal. In addition, females superovulated,

with multiple nuclei in some of the surplus ova. Closer scrutiny linked these findings to a massive spill of DDT (dichloro-diphenyl-trichloroethane) into Lake Apopka in 1980. Guillette concluded that the declining alligator population was related to the effects of DDT exposure on the animals' reproductive systems.

Although DDT was banned for use in the United States in the early 1970s, it continues to be manufactured in this country and marketed abroad, where it is sprayed on produce that is then sold in U.S. stores. The principal metabolite derived from DDT is called DDE, a xenoestrogen that lingers in fat deposits in the human body for decades. Historically, there have been reports of estrogen-mimicking effects in various fish species, especially in the Great Lakes, where residual concentrations of DDT and PCBs (polychlorinated biphenyls) are high. These effects include feminization and hermaphroditism in males. In fact, fish serve as barometers of the effects of xenoestrogen contamination in bodies of water. An index of exposure is the presence of vitellogenin, a protein specific to egg yolk, in the blood of male fish. Normally, only females produce vitellogenin in their livers, upon stimulation by estrogen from the ovaries.

Ecoestrogens are further concentrated in animals higher up in the food chain. In the Great Lakes region, birds including male herring gulls, terns, and bald eagles exhibit hermaphroditic changes after feeding on contaminated fish. Increased embryo mortality among these avians has also been observed. In addition, evidence from Florida links sterility in male and female panthers to their predation on animals exposed to pesticides with estrogenic activity.

The harmful effects of DDT have also been observed in rodents. Female rodents treated with high concentrations of DDT become predisposed to mammary tumors, while males tend to develop testicular cancer. These observations raise the question of whether pharmacological (that is, low) doses of substances with estrogenic activity translate into physiological effects. Usually they do not, but chronic exposure may be enough to trigger such effects.

Dangers to Humans

If xenoestrogens cause such dramatic reproductive effects in vertebrates, including mammals, what might be the consequences for humans? Nearly a decade ago, Frederick vom Saal, a developmental biologist at the University of Missouri Columbia, cautioned that mammalian reproductive mechanisms are similar enough to warrant concern about the effects of hormone disruptors on humans.

A 1993 article in *Lancet* looked at decreasing sperm counts in men in the United States and 20 other countries and correlated these decreases with the growing concentration of environmental estrogens. The authors—Niels Skakkenbaek, a Danish reproductive endocrinologist, and Richard Sharpe, of the British Medical Research Council Reproductive Biology Unit in Scotland—performed a meta-analysis of 61 sperm-count studies published between 1938 and 1990 to make their connection.

"Nonbelievers" state that this interpretation is contrived. Others suggest alternative explanations. For instance, the negative effects on sperm counts

could have resulted from simultaneous increases in the incidence of venereal diseases. Besides, there are known differences in steroid hormone metabolism between lower vertebrates (including nonprimate mammals) and primates, so one cannot always extrapolate from the former group to the latter.

Even so, the incidence of testicular cancer, which typically affects young men in their 20s and 30s, has increased worldwide. Between 1979 and 1991, over 1,100 new cases were reported in England and Wales—a 55 percent increase over previous rates. In Denmark, the rate of testicular cancer increased by 300 percent from 1945 to 1990. Intrauterine exposure to xenoestrogens during testicular development is thought to be the cause.

Supporting evidence comes from Michigan, where accidental contamination of cattle feed with PCBs in 1973 resulted in high concentrations in the breast milk of women who consumed tainted beef. Their sons exhibited defective genitalia. Furthermore, observers in England have noted increased incidences of cryptorchidism (undescended testes), which results in permanent sterility if left untreated, and hypospadias (urethral orifice on the underside of the penis instead of at the tip). The one area of agreement between those who attribute these effects to ecoestrogens and those who deny such a connection is that more research is needed.

Perhaps one of the most disturbing current trends is the alarming increase in breast cancer incidence. Fifty years ago, the risk rate was 1 woman in 20; today it is 1 in 8. Numerous studies have implicated xenoestrogens as the responsible agents. For instance, high concentrations of pesticides, especially DDT, have been found in the breast tissue of breast cancer patients on Long Island. In addition, it has long been known that under certain conditions, estrogen from any source can be tumor promoting, and that most breast cancer cell types have estrogen receptors.

Precocious Puberty

If that were not enough, the growing number of estrogen mimics in the environment has been linked to early puberty in girls. The normal, average age of onset is between 12 and 13. A recent study of 17,000 girls in the United States indicated that 7 percent of white and 27 percent of black girls exhibited physical signs of puberty by age seven. For 10-year-old girls, the percentages increased to 68 and 95, respectively. Studies from the United Kingdom, Canada, and New Zealand have shown similar changes in the age of puberty onset.

It is difficult, however, to elucidate the exact mechanisms that underlie these trends toward precocious puberty. One explanation, which applies especially to cases in the United States, points to the increasing number of children who are overweight or obese as a result of high-calorie diets and lack of regular exercise. Physiologically, an enhanced amount of body fat implies reproductive readiness and signals the onset of puberty in both boys and girls.

For girls, more body fat ensures that there is enough stored energy to support pregnancy and lactation. Young women with low percentages of body fat caused by heavy exercise, sports, or eating disorders usually do not experience the onset of menses until their body composition reflects adequate fat mass.

Many who study the phenomenon of premature puberty attribute it to environmental estrogens in plastics and secondhand exposure through the meat and milk of animals treated with steroid hormones. An alarming increase in the numbers of girls experiencing precocious puberty occurred in the 1970s and '80s in Puerto Rico. Among other effects, breast development occurred in girls as young as one year. Premature puberty was traced to consumption of beef, pork, and dairy products containing high concentrations of estrogen.

Another study from Puerto Rico revealed higher concentrations of phthalate —a xenoestrogen present in certain plastics—in girls who showed signs of early puberty, compared with controls.

It may be that excess body fat and exposure to estrogenic substances operate in concert to hasten puberty. Body fat is one site of endogenous estrogen synthesis. Exposure to environmental estrogens may add just enough exogenous hormone to exert the synergistic effect necessary to bring on puberty, much like the last drop of water that causes the bucket to overflow.

Although most xenoestrogens produce detrimental effects, at least one subgroup—the phytoestrogens—includes substances that can have beneficial effects. Phytoestrogens are generally weaker than natural estrogens. They are found in various foods, such as flax seeds, soybeans and other legumes, some herbs, and many fruits and vegetables. Some studies suggest that soy products may offer protection against certain cancers, including breast, prostate, uterus, and colon cancers. On the other hand, high doses of certain phytoestrogens—such as coumestrol (in sunflower seeds and alfalfa sprouts)—have been found to adversely affect the fertility and reproductive cycles of animals.

Unlike artificially produced xenoestrogens, phytoestrogens are generally not stored in the body but are readily metabolized and excreted. Their health effects should be evaluated on a case-by-case basis, considering such factors as the individual's age, medical and family history, and potential interactions with medications or supplements.

Plastics, Plastics Everywhere

It has been estimated that perhaps 100,000 synthetic chemicals are registered for commercial use in the world today and 1,000 new ones are formulated every year. While many are toxic and carcinogenic, little is known about the chronic effects of the majority of them. And there is growing concern about their potential hormone-disrupting effects. The problem of exposure is complicated by numerous carrier routes, including air, food, water, and consumer products.

Consider certain synthetics that turn up in familiar places, including food and consumer goods. Such seemingly inert products as plastic soda and water bottles, baby bottles, food wrap, Styrofoam, many toys, cosmetics, sunscreens, and even spermicides either contain or break down to yield xenoestrogens. In addition, environmental estrogens are among the byproducts created by such processes as the incineration of biological materials or industrial waste and chlorine bleaching of paper products.

In April 1999, Consumers Union confirmed information previously reported by the Food and Drug Administration regarding 95 percent of baby bottles sold in the United States. The bottles, made of a hard plastic known as polycarbonate, leach out the synthetic estrogen named bisphenol-A, especially when heated or scratched. Studies verifying the estrogenic activity of bisphenol-A were published in *Nature* in the 1930s, but it did not arouse much concern then. A 1993 report published in *Endocrinology* showed that bisphenol-A produced estrogenic effects in a culture of human breast cancer cells.

Additional studies published by vom Saal in 1997 and '98 have shown that bisphenol-A stimulates precocious puberty and obesity in mice. Others have detected leaching of bisphenol-A from polycarbonate products such as plastic tableware, water cooler jugs, and the inside coatings of certain cans (used for some canned foods) and bottle tops. Autoclaving in the canning process causes bisphenol-A to migrate into the liquid in cans.

Spokespersons from polycarbonate manufacturers have stated that they cannot replicate vom Saal's results, but he counters that industry researchers have not done the experiments correctly.

DEHA (di-[2-ethylhexyl] adipate) is a liquid plasticizer added to some plastic food wraps made of polyvinyl chloride (PVC). Scientific studies have shown that the fat-soluble DEHA can migrate into foods, especially luncheon meats, cheese, and other products with a high fat content. For a 45-pound child eating cheese wrapped in such plastic, the limit of safe intake is 1.5 ounces by European standards or 2.5 ounces by Environmental Protection Agency criteria. Studies conducted by Consumers Union indicate that DEHA leaching from commercial plastic wraps is eight times higher than European directives allow. Fortunately, alternative wraps—such as Handi-Wrap™ and Glad Cling Wrap—are made of polyethylene; chemicals in them do not appear to leach into foods.

Barbie dolls manufactured in the 1950s and '60s are made of PVC containing a stabilizer that degrades to a sticky, estrogen-mimicking residue and accumulates on the dolls' bodies. This phenomenon was noted by Danish museum officials in August 2000. Yvonne Shashoua, an expert in materials preservation at the National Museum of Denmark, warns that young children who play with older Barbie and Ken dolls expose themselves to this estrogenic chemical, and she suggests enclosing the dolls with xenoestrogen-free plastic wrap. Storing the dolls in a cool, dark place also helps prevent the harmful stabilizer from oozing out. Who would have thought that these models of glamour would become health hazards?

Another consumer nightmare is a group of chemical plasticizers known as phthalates. They have been associated with various problems, including testicular depletion of zinc, a necessary nutrient for spermatogenesis. Zinc deficiency results in sperm death and consequent infertility. Many products that previously contained phthalates have been reformulated to eliminate them, but phthalates continue to be present in vinyl flooring, medical tubing and bags, adhesives, infants' toys, and ink used to print on food wrap made of plastic and cardboard. They have been detected in fat-soluble foods such as infant formula, cheese, margarine, and chips.

Some environmental estrogens can be found in unusual places, such as contraceptive products containing the spermicide nonoxynol-9. This chemical degrades to nonylphenol, a xenoestrogen shown to stimulate breast cancer cells. Nonylphenol and other alkylphenols have been detected in human umbilical cords, plastic test tubes, and industrial detergents.

Various substances added to lotions (including sunscreens) and cosmetics serve as preservatives. Some of them, members of a chemical family called parabens, were shown to be estrogen mimics by a study at Brunel University in the United Kingdom. The researchers warned that "the safety of these chemicals should be reassessed with particular attention being paid to . . . levels of systemic exposure to humans." Officials of the European Cosmetic Toiletry and Perfumery Association dismissed the Brunel study as "irrelevant," on the grounds that parabens do not enter the systemic circulation. But they ignored the possibility of transdermal introduction.

Additional questions have been raised about the safety—in terms of xenoestrogen content—of recycled materials, especially plastics and paper. Because it is unlikely that a moratorium on chemical synthesis will occur anytime soon, such questions will continue to surface until the public is satisfied that regulatory agencies are doing all they can to minimize exposure. Fortunately, organizations such as the National Institutes of Health, the National Academy of Sciences, the Environmental Protection Agency, the Centers for Disease Control, many universities, and other institutions are involved in efforts to monitor and minimize the effects of environmental estrogens on wildlife and humans.

Michael Gough

NO

Endocrine Disrupters, Politics, Pesticides, the Cost of Food and Health

Environmentalists and politicians and federal regulators have added environmental estrogens or endocrine disrupters to the "concerns" or scares that dictate "environmental health policy." That policy, from its beginning, has been based on ideology, not on science. To provide some veneer to the ideology, its proponents have spawned bad science and junk science that claims chemicals in the environment are a major cause of human illness. There is no substance to the claims, but the current policies threaten to cost billions of dollars in wasted estrogen testing programs and to drive some substantial proportion of pesticides from the market.

Rachel Carson, conjuring up a cancer-free, pre-industrial Garden of Eden launched the biggest environmental scare of all in the 1960s. She charged that modern industrial chemicals in the environment caused human cancers. It mattered not at all to her or to her readers that cancers are found in every society, pre-industrial and modern. What mattered were opinions of people such as Umberto Safiotti of the National Cancer Institute, who wrote:

> I consider cancer as a social disease, largely caused by external agents which are derived from our technology, conditioned by our societal lifestyle and whose control is dependent on societal actions and policies.

When Saffioti said "societal actions and policies," he meant government regulations.

By 1968, environmental groups and individuals—including some scientists—appeared on TV and on the floors of the House and Senate to say, over and over again, "The environment causes 90 percent of cancers." They didn't have to say "environment" meant pollution from modern industry and chemicals—especially pesticides—everyone already knew that. Saffioti and others had told them.

In the 1970s, the National Cancer Institute [NCI] released reports that blamed elevated rates for all kinds of cancers on chemicals in the workplace or in the environment. The institute did not have evidence to link those exposures to cancer. It didn't exist then, and it doesn't, except for a limited number

of high exposures in the workplace, exist now. So what? The reports were gobbled up by the press, politicians, and the public.

In our ignorance of what causes most cancers, the "90 percent" misstatement provided great hope. If the carcinogenic agents in the environment could be identified and eliminated, cancer rates should drop. NCI scientists said so, and they said success was just around the corner if animal tests were used to identify carcinogens. Congress responded. It created the Environmental Protection Agency [EPA] and the Occupational Safety and Health Administration [OSHA]. Both agencies have lots of tasks, but both place an emphasis on controlling exposures to carcinogens. Congress passed and amended law after law. The Clean Air Act, the Clean Water Act, the Safe Drinking Water Act, amendments to the Fungicide, Insecticide, and Rodenticide Act—the euphonious FIFRA—the Toxic Substances Control Act, and the Resource Recovery and Control Act poured forth from Capitol Hill.

And in return, EPA and OSHA, to justify their existence, generated scare after scare. They are aided by all kinds of people eager for explanations about their health problems or for government grants and contracts for research or other work or for money to compensate for health effects or other problems that could be blamed on chemicals.

In 1978, we had the occupational exposure scare. Astoundingly, according to a government report, six workplace substances caused 38 percent of all the cancers in the United States. It was nonsense, of course, and many scientists ridiculed the report, but the government never retracted it. The government scientists who contributed to it never repudiated it.

At about the same time, wastes disposed in Love Canal near Niagara Fall, NY, spewed liquids and gases into a residential community. The chemicals were blamed as the cause of cancers, birth defects, miscarriages, skin diseases, you name it. None of it was true, but waste sites around the country were routinely identified as "another Love Canal" or a "Love Canal in the making," and Congress gave the nation the Superfund Law. Since its passage, Superfund has enriched lawyers and provided secure employment to thousands who wear moon suits and dig up, burn, and rebury wastes, and done nothing for the nation's health. For those who doubt the importance of politics in the environmental health saga, it's worth recalling that every state had two waste sites on the first list of sites slated for priority cleanup under Superfund.

By the 1980s, EPA was chucking out the scares. We had the 2,4,5-T scare, the dioxin scare, the 2,4-D scare, the asbestos in schools scare, the radon in homes scare, the Alar scare, the EMF scare. I've left some out, but the common thread that linked the scares together was cancer. Each scare prompted investigations by affected industries and non-government scientists. Each scare fell apart, revealed as a house of cards jerry-rigged from bad science, worse interpretations of the science, and terrible policy.

In fact, by the late 1970s, there was ample evidence that the much-talked about "cancer epidemic" and the 90 percent statement were simply wrong. Cancer rates were not increasing and rates for some cancers were higher in industrial countries and rates for others were higher in non-industrial countries. The U.S. fell in the middle of countries when ranked by cancer rates.

Sure, there are some carcinogenic substances in the nation's workplaces, but the best estimates are that they cause four percent or less of all cancers, and the percentage is decreasing because the biggest occupational threat, asbestos, is gone. Environmental exposures might cause two or three percent of cancer—on the outside—and they might cause much less.

The research into causes of cancer—not stories designed to bolster the chemicals cause cancer myth—did reveal that there are preventable causes of cancer. Not smoking is a good idea, as is eating lots of fruits and vegetables, not gaining too much weight, restricting the number of sexual partners, and, for those who are fair-skinned, being careful about sun exposures. It's not a lot different from what your mother or grandmother told you.

The government can take a nanny role in urging us to behave, but that's not where the big bucks are. The big bucks are in regulation, and regulation doesn't seem to have much to do with cancer.

In any case, cancer death rates began to fall in 1990, they've fallen since, and the fall is growing steeper. Maybe that information is blunting the cancer scare. I somehow doubt it. I think that the public has become numb to the cancer scare or that it fatalistically accepts the notion that "everything causes cancer." In any case, the environmentalists and the regulators needed a new scare.

The collapse of the cancer scare wasn't good news to everyone. Government bureaucrats and scientists in the anti-carcinogen offices and programs at EPA and elsewhere have secure jobs. Congress easily finds the will to write laws establishing environmental protection activities, but it lacks the will or patience to examine those activities to see if they've accomplished anything. And, let's face it, Congress doesn't eliminate established programs. But the growth of programs slows, and money can become scarce, and that can squeeze researchers who depend on EPA grants and contracts to fund their often senseless surveys and testing programs. Moreover, the fading of scares doesn't benefit environmental organizations that utter shrill cries about scares and coming calamities in their campaigns for contributions.

Here's an example of just how disappointed some people can be that cancer isn't on the rise. Dr. Theo Colborn, a wildlife biologist working for the Conservation Foundation in the late 1980s, was convinced that the chemicals in the Great Lakes were causing human cancer. She set out to prove it by reviewing the available literature about cancer rates in that region. She couldn't. In fact, she found that the rates for some cancers in the Great Lakes region were lower than the rates for the same cancers in other parts of the United States and Canada. . . .

Failing to find cancer slowed her down but didn't stop her. She knew that those chemicals were causing something. All she had to do was find it.

And find it, she did. She collected every paper that described any abnormality in wildlife that live on or around the Great Lakes, and concluded that synthetic chemicals were mimicking the effects of hormones. They were causing every problem in the literature, whether it was homosexual behavior among gulls, crossed bills in other birds, cancer in fish, or increases or decreases in any wildlife population.

The chemicals that have those activities were called "environmental estrogens" or "endocrine disrupters." There was no more evidence to link them to every abnormality in wildlife than there had been in the 1960s to link every human cancer to chemicals. The absence of evidence wasn't much of a problem. Colborn and her colleagues believed that chemicals were the culprit, and the press and much of the public, nutured on the idea that chemicals were bad, didn't require evidence.

Even so, Colborn had a problem that EPA faced in its early days. Soon after EPA was established, the agency leaders realized that protecting wildlife and the environment might be a good thing, but that Congress might not decide to lavish funds on such activities. They were sure, however, that Congress would throw money at programs that were going to protect human health from environmental risks. Whether Colborn knew that history or not, she apparently realized that any real splash for endocrine disrupters depended on tying them to human health effects.

Using the same techniques she'd used to catalogue the adverse effects of endocrine disrupters on wildlife, she reviewed the literature about human health effects that some way or another might be related to disruption of hormone activity. The list was long, including cancers, birth defects, and learning disabilities, but the big hitter on the list was decreased sperm counts. According to Colborn and others' analyses of sperm counts made in different parts of the world under different conditions of nutrition and stress and at different time periods, sperm counts had decreased by 50 percent in the post–World War II period.

If there's anything that catches the attention of Congress, it's risks to males. Congress banned leaded gasoline after EPA released a report that said atmospheric lead was a cause of heart attacks in middle-aged men. The reported decrease in sperm counts leaped up for attention, and attention it got. Congressional hearings were held, magazine articles were written, experts opined about endocrine disrupters and sexual dysfunctions.

And then it fell apart. Scientists found large geographical variations in sperm counts that have not changed over time. Those geographical variations and poor study designs accounted for the reported decrease. That scare went away, but endocrine disrupters were here to stay.

Well-organized and affluent women's groups are convinced that breast cancer is unusually common on Long Island. . . . We know that obesity, estrogen replacement therapy, and late child-bearing or no child-bearing, all of which are more common in affluent women, are associated with breast cancer. Nevertheless, from the very beginning, environmental chemicals have been singled out as the cause of the breast cancer excess. The insecticide aldicarb, which is very resistant to degradation was blamed, but subsequent studies failed to confirm the link. A well-publicized study found a link between DDT and breast cancer, but larger, follow-up studies failed to confirm it. But there're lots of environmental chemicals, and no evidence is required to justify a suggestion of a link between the chemicals and cancer.

Senator Al D'Amato is from Long Island, and he shares his constituents' concerns. During a hearing about the Clean Water Act, Senator D'Amato

heard testimony by Dr. Anna Soto from Tufts University about her "E-Screen." According to Dr. Soto, her quick laboratory test could identify chemicals that behave as environmental estrogens or endocrine disrupters for $500 a chemical. Since environmental estrogens seem to some people to be a likely cause of breast cancer, Soto's test appeared to be a real bargain.

Senator D'Amato pushed for an amendment to require E-Screen testing of chemicals that are regulated under the Clean Water Act, but he was unsuccessful. Later in 1995, a senior Senate staffer, Jimmy Powell, took the E-Screen amendment to a very junior Senate staffer and told her to incorporate it into the Safe Drinking Water Act as an "administrative amendment." She did, it passed the Senate, and, for the first time, there was a legislative requirement for endocrine testing.

In the spring of 1996, House Committees were considering legislation to amend the Safe Drinking Water Act and new legislation related to pesticides in food. Aware of the Senate's action, some members of the House committees were eager to include endocrine testing in their legislation, but there was resistance as well. Chemical companies viewed the imposition of yet another test as certain to be an expense, unlikely to cost as little as $500 a chemical, and bound to raise new concerns about chemicals that would require far more extensive tests and research to understand or discount.

Furthermore, so far as food was concerned, there was a general conviction that all the safety factors built into the testing of pesticides and other chemicals that might end up in food provided adequate margins of protection. That conviction was shattered by rumors that reached the House in May 1996. According to the rumors, Dr. John McLachlan and his colleagues at Tulane University had shown that mixtures of pesticides and other environmental chemicals such as PCBs were far more potent in activating estrogen receptors, the first step in estrogen modulation of biochemical pathways than were single chemicals. In the most extreme case, two chemicals at concentrations considered safe by all conventional toxicity tests were 1600 times as potent as estrogen receptor activators as either chemical by itself. The powerful synergy raised new alarms.

In May, everyone concerned about pesticides knew that EPA had a draft of the Tulane paper, and EPA staff were drifting around House offices, but they refused to answer questions about the Tulane results. The silence signaled the expected significance of the paper. A month later, in June, the paper appeared in *Science*. It was a big deal. *Science* ran a news article about the research with a picture of the Tulane researchers. It also ran an editorial by a scientist from the National Institutes of Health who offered some theoretical explanations for how combinations of pesticides at very low levels could affect cells and activate the estrogen receptors. *The New York Times, The Washington Post,* other major newspapers, and news magazines and TV reported the news. If there was ever any doubt that FQPA [Food Quality Protection Act] would require tests for endocrine activity, the flurry of news about the Tulane results erased them.

While the House was drafting the FQPA, Dr. Lynn Goldman, an assistant administrator at EPA, established a committee called the "Endocrine Disrupter

Screening and Testing Advisory Committee" (EDSTAC) [which is now] considering tests for all of the 70,000 chemicals that it estimates are present in commerce, and it's not limiting its recommendations to tests for estrogenic activity. It's adding tests for testosterone and thyroid hormone activity as well as for anti-estrogenic, anti-testosterone, and anti-thyroid activity. The relatively simple E-Screen, which is run on cultured cells, is to be supplemented by some whole animal tests. Tests on single compounds will have to be complemented by tests of mixtures of compounds. The FQPA requires that "valid" tests be used. None of the tests being considered by EDSTAC has been validated; many of them have never been done.

EDSTAC's estimate of 70,000 chemicals in commerce is on the high side—some of those chemicals are used in such small amounts and under such controlled conditions that there's no exposure to them. Dr. Dan Byrd has estimated that 50,000 is a more realistic number. He's also looked at the price lists from commercial testing laboratories to see how much they would charge for a battery of tests something like EDSTAC is considering. Some of the tests haven't been developed, but assuming they can be, Dr. Byrd estimates testing each chemical will cost between $100,000 and $200,000. The total cost would be between $5 and $10 billion. . . .

The Tulane results played some major role in the passage of FQPA, the focus on endocrine disrupters, and Dr. Goldman's establishment of EDSTAC. The Tulane results are wrong. Several groups of scientists tried to replicate the Tulane results. None could. At first, Dr. McLachlan insisted his results were correct. He said that the experiments he reported required expertise and finesse and suggested that the scientists who couldn't repeat his findings were at fault, essentially incompetent. That changed. In July 1997, just 13 months after he published his report, he threw in the towel, acknowledging that neither his laboratory nor anyone else had been able to produce the results that had created such a stir.

Whether the initial results were caused by a series of mistakes or a willful desire to show, once and for all, that environmental chemicals, especially pesticides are bad, bad, bad, we don't know. We do know that the results were wrong.

No matter, EPA now assumes as a matter of policy that synergy occurs. Good science, repeatable science that showed the reported synergy didn't occur has been brushed aside. In its place, we have bad science or junk science. If the Tulane results were the products of honest mistakes, they're bad science; if they flowed from ideology, they're junk science. The effect is the same, but the reasons are different.

The estrogenic disrupter testing under FQPA is going to cost a lot of money and cause a lot of mischief. But the effects of that testing are off somewhere in the future. More immediately, a combination of ideology-driven science and congressional misreading of that science threatens to drive between 50 and 80 percent of all pesticides from the market.

In 1993, a committee of the National Research Council spun together the facts that childhood development takes place at specific times as an infant matures into a toddler and then a child, that infants, toddlers, and children

eat, proportionally, far larger amounts of foods such as apple juice and apple sauce and orange juice than do adults, and that pesticides can be present in those processed foods. From those three observations, the committee concluded that an additional safety factor should be included in setting acceptable levels for pesticides in those foods. Left out from the analysis was any evidence that current exposures cause any harm to any infants, toddlers, or children. No matter.

Most people who worry about pesticides expected EPA and the Food and Drug Administration to react to the NRC recommendation by reducing the allowable levels of pesticides on foods that are destined for consumption by children. Maybe they would have. We'll never know. In the FQPA, Congress directed that a new ten-fold safety factor be incorporated into the evaluation of the risks from pesticides.

Safety factors are a fundamental part in the evaluation of pesticide risks. Pesticides are tested in laboratory animals to determine what concentrations to cause effects on the nervous, digestive, endocrine, and other systems. At some, sufficiently low dose that varies from pesticide to pesticide, the chemical does not cause those adverse effects. That dose, called the "No Observed Adverse Effect Level" (or NOAEL), is then divided by 100 to set the acceptable daily limit for human ingestion of the chemical. The FQPA requires division by another factor of 10, so the acceptable daily limit will be the NOAEL divided by 1000 instead of 100. Acceptable limits will be ten-fold less.

Dr. Byrd has estimated that up to 80 percent of all currently permitted uses of pesticides would be eliminated by an across the board application of the 1000-fold safety factor. He cites another toxicologist who estimates that 50 percent of all pesticides would be eliminated from the market. The extent to which these draconian reductions will be forced remains to be seen, but pesticide manufacturers and users can look forward to a period of even-greater limbo as EPA sorts through it new responsibilities and decides how to implement FQPA.

There's no convincing evidence that pesticides in food contribute to cancer causation and none that they cause other adverse health effects. Restrictions on pesticides in food will not have a demonstrable effect on human health. On the other hand, the estrogen testing program and the new safety factor will drive pesticide costs up and pesticide availability down.

Some manufacturers may lose profitable product lines; some may even lose their businesses. Farmers will pay more. They will pass those costs onto middlemen and processors, who, in turn, will pass them onto consumers. Increases in the costs of fruits and vegetables won't change the food purchasing habits of the middle class, but they may and probably will affect the purchases of the poor. The poor are already at greater risks because of poor diets, and the increased costs can be expected to further decrease their consumption of fresh fruits and vegetables.

POSTSCRIPT

Do Environmental Hormone Mimics Pose a Potentially Serious Health Threat?

Stephen H. Safe's "Environmental and Dietary Estrogens and Human Health: Is There a Problem?" *Environmental Health Perspectives* (April 1995) is often cited to support the contention that there is no causative link between environmental estrogens and human health problems. He draws a cautious conclusion, calling the link "implausible" and "unproven." Gough's belief that the battle against environmental estrogens is motivated by environmentalist ideology rather than facts is repeated by Angela Logomasini in "Chemical Warfare: Ideological Environmentalism's Quixotic Campaign Against Synthetic Chemicals," in Ronald Bailey, ed., *Global Warming and Other Eco-Myths: How the Environmental Movement Uses False Science to Scare Us to Death* (Prima, 2002). Some caution is certainly warranted, for the complex and variable manner by which different compounds with estrogenic properties may affect organisms makes projections from animal effects to human effects risky.

Sheldon Krimsky, in "Hormone Disruptors: A Clue to Understanding the Environmental Cause of Disease," *Environment* (June 2001), summarizes the evidence that many chemicals released to the environment affect—both singly and in combination or synergistically—the endocrine systems of animals and humans and may threaten human health with cancers, reproductive anomalies, and neurological effects. He cautions that the regulatory machinery is likely to move very slowly, adding that we cannot wait for scientific certainty about the hazards before we act. See also M. Gochfeld, "Why Epidemiology of Endocrine Disruptors Warrants the Precautionary Principle," *Pure & Applied Chemistry* (December 1, 2003).

Theo Colborn, a senior scientist with the World Wildlife Fund, first drew public attention to the potential problems of environmental estrogens with the book *Our Stolen Future* (Dutton, 1996), coauthored by Dianne Dumanoski and John Peterson Myers. Colborn clearly believes that the problem is real; she finds the evidence that extensive damage is being done to wildlife by synthetic estrogenic chemicals convincing and thinks it likely that humans are experiencing similar health problems. Recent data reinforce her and Krimsky's points; see Rebecca Renner, "Human Estrogens Linked to Endocrine Disruption," *Environmental Science and Technology* (January 1, 1998) and Ted Schettler, et al., *Generations at Risk: Reproductive Health and the Environment* (MIT Press, 1999). Julia R. Barrett, "Phthalates and Baby Boys," *Environmental Health Perspectives*, (August 2005), report effects of phthalate plastic-softeners on the development

of the human male reproductive system. Laboratory work has found that at least one phthalate reduces the number of sperm-generating cells in fetal testis tissue; see Romain Lambrot, et al., "Phthalates Impair Germ Cell Development in the Human Fetal Testis in Vitro without Change in Testosterone Production," *Environmental Health Perspectives* (January 2009). When researchers added synthetic estrogen to a Canadian lake, they found that the reproduction of fathead minnows was so severely affected that the population nearly died out; see Karen A. Kidd, et al., "Collapse of a Fish Population After Exposure to a Synthetic Estrogen," *Proceedings of the National Academy of Sciences of the United States of America* (May 22, 2007). In 1999 the National Research Council published *Hormonally Active Agents in the Environment* (National Academy Press), in which the council's Committee on Hormonally Active Agents in the Environment reports on its evaluation of the scientific evidence pertaining to endocrine disruptors. The National Environmental Health Association has called for more research and product testing; see Ginger L. Gist, "National Environmental Health Association Position on Endocrine Disrupters," *Journal of Environmental Health* (January–February 1998).

Elisabete Silva, Nissanka Rajapakse, and Andreas Kortenkamp, in "Something From 'Nothing'—Eight Weak Estrogenic Chemicals Combined at Concentrations Below NOECs Produce Significant Mixture Effects," *Environmental Science and Technology* (April 2002), find synergistic effects of exactly the kind dismissed by Gough. Also, after reviewing the evidence, the U.S. National Toxicology Program found that low-dose effects had been demonstrated in animals (see Ronald Melnick, et al., "Summary of the National Toxicology Program's Report of the Endocrine Disruptors Low-Dose Peer Review," *Environmental Health Perspectives* [April 2002]). Some researchers say there is reason to think endocrine disruptors are linked to the epidemic of obesity in the U.S. and to a decline in the proportion of male births in the U.S. and Japan (Julia R. Barrett, "Shift in the Sexes," *Environmental Health Perspectives*, June 2007). Retha R. Newbold, et al., "Effects of Endocrine Disruptors on Obesity," *International Journal of Andrology* (April 2008), review the literature on the obesity link and conclude that the evidence is strong enough to warrant expanding "the focus on obesity [and consequent diabetes and heart disease] from intervention and treatment to include prevention and avoidance of these chemical modifiers."

The view that environmental hormone mimics or disruptors have potentially serious effects continues to gain support. Evidence continues to accumulate, and changes are beginning to happen. Plastic bottles, which contain the hormone disruptor bisphenol-A, are being replaced by metal bottles; see Nancy Macdonald, "Plastic Bottles Get the Eco-Boot," *Maclean's* (October 15, 2007). Medical plastics, softened by phthalates, are being reformulated; see "Medical Devices to Get Phthalate Substitution Rule," *European Environment & Packaging Law Weekly* (June 1, 2007).

ISSUE 18

Is the Superfund Program Successfully Protecting Human Health from Hazardous Materials?

YES: Robert H. Harris, Jay Vandeven, and Mike Tilchin, from "Superfund Matures Gracefully," *Issues in Science & Technology,* Summer 2003, Vol. 19, Issue 4

NO: Randall Patterson, from "Not in Their Back Yard," *Mother Jones* (May/June 2007)

ISSUE SUMMARY

YES: Environmental consultants Robert H. Harris, Jay Vandeven, and Mike Tilchin argue that although the Superfund program still has room for improvement, it has made great progress in risk assessment and treatment technologies.

NO: Journalist Randall Patterson argues that the Superfund Program is not applied to some appropriate situations, largely because people resist its application.

The potentially disastrous consequences of ignoring hazardous wastes and disposing of them improperly burst upon the consciousness of the American public in the late 1970s. The problem was dramatized by the evacuation of dozens of residents of Niagara Falls, New York, whose health was being threatened by chemicals leaking from the abandoned Love Canal, which was used for many years as an industrial waste dump. Awakened to the dangers posed by chemical dumping, numerous communities bordering on industrial manufacturing areas across the country began to discover and report local sites where chemicals had been disposed of in open lagoons or were leaking from disintegrating steel drums. Such esoteric chemical names as dioxins and PCBs have become part of the common lexicon, and numerous local citizens' groups have been mobilized to prevent human exposure to these and other toxins.

The expansion of the industrial use of synthetic chemicals following World War II resulted in the need to dispose of vast quantities of wastes laden with organic and inorganic chemical toxins. For the most part, industry adopted a casual attitude toward this problem and, in the absence of regulatory restraint, chose the least expensive means of disposal available. Little attention

was paid to the ultimate fate of chemicals that could seep into surface water or groundwater. Scientists have estimated that less than 10 percent of the waste was disposed of in an environmentally sound manner.

The magnitude of the problem is truly mind-boggling: Over 275 million tons of hazardous waste is produced in the United States each year; as many as 10,000 dump sites may pose a serious threat to public health, according to the federal Office of Technology Assessment; and other government estimates indicate that more than 350,000 waste sites may ultimately require corrective action at a cost that could easily exceed $500 billion.

Congressional response to the hazardous waste threat is embodied in two complex legislative initiatives. The Resource Conservation and Recovery Act (RCRA) of 1976 mandated action by the Environmental Protection Agency (EPA) to create "cradle to grave" oversight of newly generated waste, and the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), commonly called "Superfund," gave the EPA broad authority to clean up existing hazardous waste sites. The implementation of this legislation has been severely criticized by environmental organizations, citizens' groups, and members of Congress who have accused the EPA of foot-dragging and a variety of politically motivated improprieties. Less than 20 percent of the original $1.6 billion Superfund allocation was actually spent on waste cleanup. In 2009, the U.S. Inspector General issued a report accusing the EPA of mismanagement; see "Inspector General's Report Faults EPA for Management of 819 Superfund Accounts," *BNA's Environmental Compliance Bulletin* (April 6, 2009) (for EPA commentary, visit http://www.epa.gov/oig/reports/2009/20090318-09-P-0119_glance.pdf).

Amendments designed to close RCRA loopholes were enacted in 1984, and the Superfund Amendments and Reauthorization Act (SARA) added $8.6 billion to a strengthened cleanup effort in 1986 and an additional $5.1 billion in 1990. While acknowledging some improvement, both environmental and industrial policy analysts remain very critical about the way that both RCRA and Superfund/SARA are being implemented. Efforts to reauthorize and modify both of these hazardous waste laws have been stalled in Congress since the early 1990s. But the work went on. The Superfund program continued to identify hazardous waste sites that warranted cleanup and to clean up sites; see http://www.epa.gov/superfund/ for the latest news. In 2004, it even declared that the infamous Love Canal site was finally safe.

In the following selections, Robert H. Harris, Jay Vandeven, and Mike Tilchin argue that the Superfund program has had to struggle with unexpectedly large cleanup tasks such as mines, harbors, and the ruins of the World Trade Center in New York; is subject to greater resource demands than ever before; and still has room for improvement. But, they say, the program has made great progress in risk assessment and treatment technologies. Journalist Randall Patterson makes it clear that not all places made dangerous to human health by chemicals are industrial sites. They can be suburbs such as El Dorado Hills, CA, where ordinary construction activities produce hazardous waste of another sort, in this case dust containing asbestos. He argues that El Dorado Hills has not been labeled a Superfund site largely because people deny there is a problem and resist the EPA's intervention.

YES

Robert H. Harris, Jay Vandeven, and Mike Tilchin

Superfund Matures Gracefully

Superfund, one of the main programs used by the Environmental Protection Agency (EPA) to clean up serious, often abandoned, hazardous waste sites, has been improved considerably in recent years. Notably, progress has been made in two important areas: the development of risk assessments that are scientifically valid yet flexible, and the development and implementation of better treatment technologies.

The 1986 Superfund Amendments and Reauthorization Act (SARA) provided a broad refocus to the program. The act included an explicit preference for the selection of remediation technologies that "permanently and significantly reduce the volume, toxicity, or mobility of hazardous substances." SARA also required the revision of the National Contingency Plan (NCP) that sets out EPA's rules and guidance for site characterization, risk assessment, and remedy selection.

The NCP specifies the levels of risk to human health that are allowable at Superfund sites. However, "potentially responsible parties"—companies or other entities that may be forced to help pay for the cleanup—have often challenged the risk assessment methods used as scientifically flawed, resulting in remedies that are unnecessary and too costly. Since SARA was enacted, fundamental changes have evolved in the policies and science that EPA embraces in evaluating health risks at Superfund sites, and these changes have in turn affected which remedies are most often selected. Among the changes are three that collectively can have a profound impact on the selected remedy and attendant costs: EPA's development of land use guidance, its development of guidance on "principal threats," and the NCP requirement for the evaluation of "short-term effectiveness."

Before EPA's issuance in 1995 of land use guidance for evaluating the potential future public health risks at Superfund sites, its risk assessments usually would assume a future residential use scenario at a site, however unrealistic that assumption might be. This scenario would often result in the need for costly soil and waste removal remedies necessary to protect against hypothetical risks, such as those to children playing in contaminated soil or drinking contaminated ground water, even at sites where future residential use was highly improbable. The revised land use guidance provided a basis for selecting more realistic future use scenarios, with projected exposure patterns that may allow for less costly remedies.

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Potentially responsible parties also complained that there was little ... to tailor remedies to the magnitude of cancer risk at a site, and that ... costly remedies would be chosen for sites where the cancer risks ma... by several orders of magnitude. However, EPA's guidance on principal ... essentially established a risk-based hierarchy for remedy selection. For ex... ple, if cancer risks at a site exceed 1 in 1,000, then treatment or waste remov... or both might be required. Sites that posed a lower lifetime cancer risk could be managed in other ways, such as by prohibiting the installation of drinking water wells, which likely would be far less expensive than intrusive remedies.

Revisions to the NCP in 1990 not only codified provisions required by the 1986 Superfund amendments, but also refined EPA's evolving remedy-selection criteria. For example, these revisions require an explicit consideration of the short-term effectiveness of a remedy, including the health and safety risks to the public and to workers associated with remedy implementation. EPA had learned by bitter experience that to ignore implementation risks, such as those associated with vapor and dust emissions during the excavation of wastes, could lead to the selection of remedies that proved costly and created unacceptable risks.

Although these changes in risk assessment procedures have brought greater rationality to the evaluation of Superfund sites, EPA still usually insists on the use of hypothetical exposure factors (for example, the length of time that someone may come in contact with the site) that may overstate risks. The agency has been slow in embracing other methodologies, such as probabilistic exposure analysis, that might offer more accurate assessments. Thus, some remedies are still fashioned on risk analyses that overstate risk.

Technological Evolution

Cleanup efforts in Superfund's early years were dominated by containment and excavation-and-disposal remedies. But over the years, cooperative work by government, industry, and academia have led to the development and implementation of improved treatment technologies.

The period from the mid-1980s to the early 1990s was marked by a dramatic increase in the use of source control treatment, reflecting the preference expressed in SARA for "permanent solutions and alternative treatment technologies or resource recovery technologies to the maximum extent practicable." Two types of source control technologies that have been widely used are incineration and soil vapor extraction. Although the use of incineration decreased during the 1990s because of cost and other factors, soil vapor extraction remains a proven technology at Superfund sites.

Just as early source control remedies relied on containment or excavation and disposal offsite, the presumptive remedy for groundwater contamination has historically been "pump and treat." It became widely recognized in the early 1990s that conventional pump-and-treat technologies had significant limitations, including relatively high costs. What emerged to fill the gap was an approach called "monitored natural attenuation" (MNA), which makes use of a variety of technologies, such as biodegradation, dispersion,

dilution, absorption, and volatilization. As the name suggests, monitoring the effectiveness of the process is a key element of this technology. And although cleanup times still may be on the order of years, there is evidence that MNA can achieve comparable results in comparable periods and at significantly lower costs than conventional pump-and-treat systems. EPA has taken an active role in promoting this technology, and its use has increased dramatically in recent years.

As suggested by the MNA example, what may prove an even more formidable challenge than selecting specific remedies is the post-remedy implementation phase—that is, the monitoring and evaluation that will be required during coming decades to ensure that the remedy chosen is continuing to protect human health and the environment. Far too few resources have been devoted to this task, which will require not only monitoring and maintaining the physical integrity of the technology used and ensuring the continued viability of institutional controls, but also evaluating and responding to the developing science regarding chemical detection and toxicity.

Coming Challenges

In recent years, the rate at which waste sites are being added to the National Priorities List (NPL) has been decreasing dramatically as compared with earlier years. In fiscal years 1983 to 1991, EPA placed an average of 135 sites on the NPL annually. The rate dropped to an average of 27 sites per year between 1992 and 2001. Although many factors have contributed to this trend, three stand out:

- There was a finite group of truly troublesome sites before Superfund's passage, and after a few years most of those were identified.
- The program's enforcement authority has had a profound impact on how wastes are managed, significantly reducing, although not eliminating, the types of waste management practices that result in the creation of Superfund sites.
- A range of alternative cleanup programs, such as voluntary cleanup programs and those for brownfields, have evolved at both the federal and state levels. No longer is Superfund the only path for cleaning up sites.

But such programmatic changes are about more than just site numbers. In 1988, most NPL sites were in the investigation stage, and the program was widely criticized as being too much about studies and not enough about cleanup. Superfund is now a program predominantly focused on the design and construction of cleanup remedies.

This shift reflects the natural progress of sites through the Superfund pipeline, the changes in NPL listing activity, and a deliberate emphasis on achieving "construction completion," which is the primary measure of achievement for the program as established under the Government Performance and Results Act. It is a truism in regulatory matters that what gets done is what gets measured, and Superfund is no exception.

In the late 1990s, many observers believed that the demands on Superfund were declining and that it would be completed sometime in the middle of the first decade of the new century. But this is not proving to be true. Although expenditures have not been changing dramatically over time, the resource demands on the program are greater today than ever before.

Few people would have predicted, for example, that among the biggest technical and resource challenges facing Superfund at this date would be the cleanup of hard-rock mining sites and of large volumes of sediments from contaminated waterways and ports. These sites tend to be very costly to clean up, with the driver behind these great costs weighted more toward the protection of natural resources than of human health. In mapping the future course of the program, Congress and EPA must address the question of whether Superfund is the most appropriate program for cleaning up these types of sites.

There are other uncertainties, as well. The substantial role that Superfund has played in emergency response in the aftermath of 9/11, the response to the anthrax attacks of October 2001, and the program's role in the recovery of debris from the crash of the space shuttle Columbia were all totally unforeseeable. Although many valuable lessons have been learned over the past 20 years of the program, there remain substantial opportunities for improvement as well as considerable uncertainty about the kinds of environmental problems Superfund will tackle in the coming decade.

Randall Patterson

NO

Not in Their Back Yard

When the EPA discovered asbestos in their Little League fields, the residents of idyllic El Dorado Hills rushed to protect themselves—from reality.

The land in question is a high, rolling hillside, where California's Central Valley slopes into the Sierra foothills. Many have been drawn here, and after the first stampede, for gold in 1849, most left disappointed. Then the land lay virtually empty for many years until, as the state capital grew, its emptiness became valuable. In the late 1970s, as the local Chamber of Commerce tells it, a developer was traveling along U.S. Highway 50 when, 25 miles east of Sacramento, he looked up and had a vision.

Grand homes started to appear, followed by fine schools, lush golf courses, and a central shopping district modeled after a Tuscan village. The entire hillside was transformed into a patchwork of gated communities, and over the past 15 years, El Dorado Hills, as developers dubbed the spot, has been one of the fastest-growing areas in California. "High per capita income along with low crime rates have attracted many new families to this vibrant and desirable destination of the future," says the Chamber of Commerce's website. "As the name 'El Dorado' translates, this is a land of golden opportunity!"

Some residents of the bedroom community like to think of it as the next Silicon Valley, but the president of the Chamber of Commerce admits there are few jobs, and "the one thing very different about El Dorado Hills" is that no one has to be here. Almost no one leaves either, which is surprising when you consider that what lies beneath these hills isn't gold but something potentially deadly.

Housing developers and real estate agents don't like talking about the dark side of paradise, so I had to rely on Terry Trent to give me an unofficial tour. Trent, a 50-ish retired construction consultant, no longer lives in El Dorado County but admits that his former home has become something of an obsession. "There are some really dangerous things in the ground," he said as we drove down the main street, El Dorado Hills Boulevard. Talking nonstop, Trent pointed to the hillside that forms the spine of the town, which, according to his map from the state's Division of Mines and Geology, contains a vast deposit of naturally occurring asbestos.

There are similar deposits all over California; serpentine, a primary source of asbestos, is the state rock. It's not dangerous—unless it becomes airborne dust and gets in your lungs. The trouble is, in El Dorado Hills, dust isn't just a

fact of life but a sign of progress. "Here in the mountains, we don't let no rocks stand in our way," Trent said. Over the past 10 years, as the town's population has doubled to around 35,000, hundreds of bulldozers have scraped over this ground, clearing land for thousands of new homes. In that earth is a form of asbestos—amphibole—that's significantly more toxic than the type commonly used commercially.

Trent's map of the amphibole deposits was made 50 years ago. Still, most residents were unaware of the asbestos until 11 years ago, when Trent uncovered a huge, glistening vein of it in his yard. He went to the local papers with the news, and there was a brief outcry. But the construction went on, and most of El Dorado Hills has been built in the years since.

Turning onto Harvard Way, Trent stopped before a grassy embankment that he claims is shot through with asbestos. "You can see how they carved through it to build this road," he said. He recalled watching as construction crews gouged out a spot for a new middle school nearby, crushing the excavated rock and transporting the gravel to become the foundation for the new community center. A half-mile stretch of Harvard Way was covered in dust, he said, for months.

Nine years ago, tests commissioned by the *Sacramento Bee* found high concentrations of amphibole fibers inside Trent's home. He moved soon afterward. To this day, he still can't understand why few of his former neighbors seem concerned about the threat beneath them, or why the town is still fully occupied, its homes still worth hundreds of thousands of dollars. "I don't like to be mean," he said, "but I'm a little cranky with these people. It's like they're in shock. They don't want this stuff to exist; therefore, they put their heads in the sand. They say, 'Well, it's not here, and it's not bad, and here, I'll prove that it's not here and it's not bad.'"

In the fall of 2004, agents from the Environmental Protection Agency descended on El Dorado Hills in respirators and protective suits and headed for a town park. There, they began playing as children would—tossing baseballs, kicking soccer balls, biking, and running. All the while, they took air samples, and all the while they were quietly watched by the citizens of El Dorado Hills.

The civic leaders of El Dorado Hills had spent many months trying to stave off these tests, scrambling to protect the community not from potentially toxic substances, but from the EPA's potentially toxic information. Taking the lead was Vicki Barber, the superintendent of schools. A stout woman with compressed lips and an unwavering gaze, she recently won an award for being "a person who does not accept the word 'no' when it comes to what is good for students." After asbestos was found during the construction of a high school soccer field in 2002, Barber questioned a costly EPA-mandated cleanup. When a citizen formally asked the EPA to test the town's public areas for asbestos in 2003, Barber quickly emerged as the agency's most determined local foe. Before the study was even under way, she began writing to the EPA as well as to senators and congressmen, questioning whether the agency had the "legal and scientific authority" to conduct what she called a "science experiment" with "limited benefit to the residents." At least four state legislators and one

congressman responded by putting pressure on the EPA, which in turn agreed not to declare El Dorado Hills a Superfund site, regardless of what it might find there.

In May 2005, the EPA announced its findings: Almost every one of more than 400 air samples collected at the park, as well as along a hiking trail and in three school yards, contained asbestos fibers. Most of the samples contained amphibole fibers known as tremolite—long, thin strands that, when inhaled, tend to wedge in the lungs for much longer and at higher concentrations than other forms of asbestos, even at very low exposure levels.

The correlation between amphibole asbestos and lung disease has been demonstrated around the world, from Canada to Cyprus, and perhaps most convincingly in the mining town of Libby, Montana, where hundreds of residents have been diagnosed with asbestos-related diseases and many have died. After reviewing the EPA's findings in El Dorado Hills, a Canadian epidemiologist told the *Fresno Bee* that the area's exposure levels were comparable to those in towns where mining had gone on for a century. "You can certainly say people are going to die, and there are going to be increased cases of cancer," he said. "I wouldn't live there. I wouldn't want my family to live there."

The EPA, however, avoided making such dire statements. Its report neither specified how toxic the samples were nor quantified the health risk to the residents of El Dorado Hills. Rather, it simply noted that the exposure levels were "of concern." Jere Johnson, who oversaw the study, explained that the agency was trying not to sound alarmist. "We wanted to inform the community without scaring them," she said.

But local officials received the EPA's findings almost as a declaration of war on El Dorado Hills and its way of life. Jon Morgan, the director of the county's environmental management department, thundered to a local television station that the tremolite announcement "may unnecessarily scare the living daylights out of every man, woman, and child in El Dorado County and could possibly devastate the county for years to come."

When I visited El Dorado Hills, more than a year after the EPA had run its tests, I was told over and over that the biggest threat facing the town was big-government intrusion. "The issue is not asbestos," declared James Sweeney, chairman of the county Board of Supervisors. "The issue is the EPA." Superintendent Barber said that El Dorado Hills is like any other community in California, "and yet why is it that the EPA has decided to focus on only here?" One county supervisor claimed that an EPA official had told her he was going to make El Dorado Hills "a poster child for asbestos." "What I heard," town general manager Wayne Lowery said, "is that the Libby, Montana, project is being wrapped up and they have all these employees with training, and they're looking for a place to keep them employed."

What were officials supposed to do with the EPA's information, wondered Debbie Manning, the president of the Chamber of Commerce. "Are you just going to make it a ghost town?" There was no need to rush to judgment until all the facts had been determined. "I'm sure you know that asbestos is a complicated issue," she told me, explaining that there had been a locally led effort to get the information out about asbestos. Fortunately, El Dorado Hills

is a highly educated community, she said, and you can trust that "with good information, people can make proper decisions."

One of those struggling to do just that was Vicki Summers, a 47-year-old mom who said that her chief duty was to protect her two children from "any type of health issues." Surrounded by stuffed animals, she sat on a couch in her spacious living room, her eyes darting as she spoke of the dangers they faced. "Every day in the newspaper, there's something new," she said—chemicals in the water, mad cow disease, bird flu. She often felt overwhelmed, especially when experts said different things. Summers had guzzled green tea for its anti-cancer benefits until she heard it caused colon cancer. She had gorged on fish during her first pregnancy, thinking it was "brain food," only to be told, while expecting her second child, that mercury could cause brain damage.

The same sort of thing had happened to her dad. When she was a girl, he had made a point of buying margarine because he wanted to protect his family from cholesterol. "And now he's had three strokes," she explained, "and they say it's the plaque buildup from the hydrogenated vegetable oil." So much conflicting information was almost paralyzing. "It would be better to be ignorant," she said, "because then you wouldn't have to be stressed out by all this."

Summers called her home her "dream house," and said she had long considered El Dorado Hills "heaven on Earth," but when she heard about the asbestos, her first thought was, "If this is Love Canal, and there's going to be a mass exodus, I don't want to be the last one out."

But no exodus from El Dorado Hills ever began. Many residents seemed oddly comforted by the apparent uncertainty of the situation. Just after the EPA report came out, Summers joined a thousand other citizens in the town gym, looking for answers. There, school superintendent Barber reassured the crowd that the town was "deeply committed to maintaining public health and safety." Then she ripped into the EPA report for offering no solid "risk information." She pointed out there had been no abundance of "pulmonary cases" in the area. In other words, with nobody getting sick or dying, there was no real evidence of any hazard, so why worry? "Risk is a part of all of our lives," Barber said. "But we also need to keep it in perspective." She received enthusiastic applause.

Scientists, however, are more certain about the dangers of asbestos. There is no known safe exposure level to asbestos, whether it is in a commercial or a natural form; even low doses can cause malignant mesothelioma, a rare lung cancer. And the evidence keeps mounting. In the summer of 2005, researchers from the University of California-Davis Department of Public Health Sciences announced the findings of a study comparing the addresses of 2,900 Californians suffering from malignant mesothelioma against a geological map of the state. They concluded that the risk of developing lung cancer was directly related to how close the patients had lived to areas of rock associated with naturally occurring tremolite asbestos. (Likewise, the odds of getting mesothelioma drop 1 percent for every mile one moves away from an asbestos source.) As one of the authors explained, "We showed that breathing asbestos in your community is not magically different from breathing asbestos in an industrial setting."

All of this passed over El Dorado Hills like clouds in the blue sky. County supervisor Helen Baumann's reaction to the UC Davis report was that "there are studies counter to that." Local government would "err on the side of public health and safety," she said, "until some better science comes forward."

So began the effort to live in El Dorado Hills without coming into contact with the earth. In the park and the school playing fields where the EPA had collected samples, workers trucked in clean soil to replace two feet of topsoil, as the agency had recommended. The county banned using leaf blowers on town property, "except for emergency situations." Residents were encouraged to take precautions such as removing their shoes before entering their homes, driving slowly with the windows up on unpaved roads, and "limiting time spent on dirt." Home-builders agreed to spray down building sites and rinse off their trucks' tires after work. Any sightings of "fugitive dust" were to be reported to a new "dust enforcement" team.

Life in El Dorado Hills under the new regime was "manageable," said Baumann. That seemed to be the point of the show: If you took a few precautions, there was no need to panic. She proudly pointed out that the dust hot line rarely received a call. Gerri Silva, the interim director of the county department of environmental management, told me the measures had worked so well that in all of El Dorado Hills, "there is no dust."

In the town's main coffee shop, Bella Bru, languor pervaded as well. An engineer named Matt Parisek said that asbestos was "pretty muck a fake issue." "It's pretty obvious [the EPA] selected this county because of our conservative Republican reputation," he said. And, wondered retiree Carole Gilmore, "If asbestos is all over California, I don't know why they zero in on El Dorado Hills."

Real estate broker Charles Hite, who is the current resident of Terry Trent's old house, pointed out that none of his neighbors had died. "If it is such a health hazard," he asked, "why are people still buying and building? Why are real estate prices going up? Why doesn't government shut the city down?"

There was a circular logic at work. The local government had told everyone to stay calm, and so residents weren't afraid. And if no one was scared, then perhaps there was nothing to fear. "The vast, vast majority" trusted that they were safe, said Baumann. But, she added, there remained "a very, very small centralized group that keeps pushing issues and pushing issues to the extreme." She specifically mentioned Terry Trent, who to this day sends regular, dire emails to residents, reporters, and scientists. The group of extremists who "do not represent our community" also included, in Baumann's view, some members of a community group that meets monthly to discuss asbestos.

Vicki Summers had joined the Asbestos Community Advisory Group as part of her effort to educate herself. When someone would mention the low body count as evidence that nothing was wrong, she could now cite the UC Davis study, or note that the latency period for asbestos-related diseases is about 30 years. Who lived in these hills back then, she asked, "a dozen ranchers?" Other parents assumed that if they couldn't see asbestos in the air, it wasn't there; Summers could picture invisible fibers burrowing into her kids' lungs. "It never disappears," she said, wide-eyed. "That's the scary thing. And

it's a known carcinogen. It's a known carcinogen. And if we know that, we know it's not good for our kids."

Summers said she was thinking only of her family when, at a community meeting, she passed a note to an EPA official, asking, "Should I move?" After that was reported by a newspaper, she received an answer in an anonymous, late-night phone call: "Vicki Summers, I think you should move!"

Short of evacuating, there are only two ways to solve El Dorado Hills' asbestos problem: Either pave over every asbestos deposit in town—an impossible task—or make the asbestos magically disappear. Soon after the EPA report came out, Superintendent Barber got in touch with the National Stone, Sand and Gravel Association, an industry trade group, which commissioned the R.J. Lee Group to scrutinize the EPA's data. In 2000, it was reported that R.J. Lee's president, Richard Lee, had been paid about $7 million for testifying more than 250 times on behalf of the asbestos industry. When the *Seattle Post-Intelligencer* found asbestos in Crayola crayons, it was Lee who did a study that discovered none. When people began getting sick and dying in Libby, Montana, R.J. Lee found that local asbestos levels had been overstated by the EPA.

As in Libby, R.J. Lee declared that what the EPA had been calling asbestos in El Dorado Hills was, in fact, not asbestos at all. Some of the fibers were not the proper size; others contained a bit too much aluminum. The EPA had, in other words, completely goofed.

Supervisor Baumann hailed "a study that has profound information in it." Barber bustled off to Washington, D.C., at taxpayer expense, carrying news of "this startling scientific development." And Wayne Lowery, the town manager, admitted it was hard to get excited about controlling asbestos "when you've got two different interpretations of the same data."

At a meeting of the Asbestos Community Advisory Group in the fire station last winter, Summers and a handful of other residents met with the EPA's Jere Johnson to try to understand how asbestos could vanish before their eyes. Two builders were also at the table. Baumann seemed to have been thinking of them when, at an earlier county supervisors' meeting, she had issued a "public thank you" to "the very, very smart people attending those meetings, making sure another thought process is heard."

One of the builders introduced R.J. Lee's report and started to expound on "cleavage fragments," mineral composition, microns, and the like. "I don't want to get bogged down with these semantics," a fireman interrupted. "We know we have it here." The group's chairman shushed him, though, saying he didn't want to get bogged down in semantics either, "but I think we almost have to." The builder persisted with his mantra of scientific uncertainty. "We all wish science to be this nice black-and-white thing," he said, "and sometimes it isn't." The debate seemed essentially over by the time the EPA's Johnson clarified the topic: It was not geology but public health. "The body can't tell the difference between a fragment and a fiber," Johnson said. Whatever you called it, it would still get stuck in your lungs.

For Summers, the starkness of the issue had again faded to gray. When she started looking for a safer place to live, she was alarmed at first to realize

there were cancer clusters and crime everywhere, and then she had taken comfort in danger. If nowhere on Earth was completely safe, she reasoned, why should she leave El Dorado Hills? Perhaps it was as Vicki Barber had said: Risk is a part of all of our lives. Her family could stay, she decided, as long as she took precautions, such as wiping down her home with damp rags, and kept pushing to get all the facts. And if new studies revealed the asbestos to be "just fragments and dust," well, she admitted, she'd be "thrilled to death."

But she knew otherwise, didn't she? She knew that asbestos was here, and she knew that it was bad? The question silenced her. "I don't want to know it, though," she finally answered. "I don't want to know it. That's when I can't sleep. I mean, I love it here. I love it here. That's why it's hard. Part of me wants to be in denial."

After reviewing R.J. Lee's report on El Dorado Hills, the EPA declared last spring that the company had violated "generally accepted scientific principles." R.J. Lee, in turn, discovered "a number of important differences of opinion as well as factual misstatements" in the EPA's response. The EPA at last asked the U.S. Geological Survey to step in and do its own testing. In December, the USGS announced that its analysis confirmed that "material that can be classified as tremolite asbestos is" in El Dorado Hills. But the geologists were uncomfortable assessing the health risks, and so the controversy continues.

Supervisor Baumann thought the EPA should just leave El Dorado Hills be. Her constituents had "really calmed down, been educated." Why, she wondered, would anyone want to excite them again? The public health debate seemed to have come full circle when Sweeney, the chairman of the Board of Supervisors, griped about the "extreme cost" of dust controls, saying, "We're putting our public at risk by telling them to do things that are absolutely unnecessary."

Up on the ridge, behind the high school, Terry Trent stood beside a driveway with a piece of tremolite in his hand. As he tossed the rock back to the ground, there was the sound of a small engine starting. "Watch this," Trent said, and a smile broke over his face as a gardener passed by with a leaf blower, dust billowing all around.

POSTSCRIPT

Is the Superfund Program Successfully Protecting Human Health from Hazardous Materials?

Superfund cleanups, when those responsible for contaminated sites could not pay or could not be found, were to have been funded by taxes on industry (e.g., the Crude Oil Tax, the Chemical Feedstock Tax, the Toxic Chemicals Importation Tax, and the Corporate Environmental Income Tax). These taxes expired in 1995, and Congress has so far refused to reauthorize them, but the Obama Administration's 2010 budget presumes that the tax will be reinstated soon; see "Obama Budget Would Reinstate Superfund Tax," *Chemical Week* (March 2, 2009). Meanwhile, the Environmental Protection Agency has received $600 million of economic stimulus funds to clean up 50 superfund sites. Some of these sites will also benefit from federal, state, and private funds designed to turn "brownfields" into new industrial and commercial developments; see JoAnn Greco, "Brown Is the New Green," *Planning* (March 2009).

The Competitive Enterprise Institute objects that taxes such as the superfund taxes are an assault on consumer pocketbooks, as is the Comprehensive Environmental Response, Compensation, and Liability Act's (CERCLA's) "joint and several liability" clause, which can make minor contributors to toxics sites liable for large cleanup costs even when they acted according to all laws and regulations in force at the time. However, in May 2009, the U.S. Supreme Court ruled that some contributors may not be liable for cleanup costs; see "US Supreme Court Ruling Limits Superfund Liability," *Oil Spill Intelligence Report* (May 7, 2009).

Meanwhile, the hazardous waste problem takes new forms. Even in the 1990s, an increasingly popular method of disposing of hazardous wastes was to ship them from the United States to "dumping grounds" in developing countries. Iwonna Rummel-Bulska's "The Basel Convention: A Global Approach for the Management of Hazardous Wastes," *Environmental Policy and Law* (vol. 24, no. 1, 1994) describes an international treaty designed to prevent or at least limit such waste dumping. But eight years later, in February 2002, the Basel Action Network and the Silicon Valley Toxics Coalition (http://svtc.org) published *Exporting Harm: The High-Tech Trashing of Asia*. This lengthy report documents the shipping of electronics wastes, including defunct personal computers, monitors, and televisions, as well as circuit boards and other products rich in lead, beryllium, cadmium, mercury, and other toxic materials. Some 50 to 80 percent of the "e-waste" collected for recycling in the western United States is shipped to destinations such as China, India, and Pakistan, where recycling and disposal methods lead to widespread human and environmental

contamination. An updated version of this report, *Poison PCs and Toxic TVs: E-Waste Tsunami to Roll Across the US: Are We Prepared?*, was released in February 2004. In August 2005, Greenpeace released K. Brigden, et al., *Recycling of Electronic Wastes in China and India: Workplace and Environmental Contamination* (http://www.e-takeback.org/press_open/greenpeace.pdf). Currently the problem of electronic waste continues to grow; see Elisabeth Jeffries, "E-Wasted," *World Watch* (July/August 2006).

Among the solutions that have been urged to address the hazardous waste problem are "take-back" and "remanufacturing" practices. Gary A. Davis and Catherine A. Wilt, in "Extended Product Responsibility," *Environment* (September 1997), urge such solutions as crucial to the minimization of waste and describe how they are becoming more common in Europe. Brad Stone, "Tech Trash, E-Waste: By Any Name, It's an Issue," *Newsweek* (December 12, 2005), describes their appearance in the United States, where some states are making take-back programs mandatory for computers, televisions, and other electronic devices. After *Exporting Harm* was published and drew considerable attention from the press, some industry representatives hastened to emphasize such practices as Hewlett Packard's recycling of printer ink cartridges. See Doug Bartholomew's "Beyond the Grave," *Industry Week* (March 1, 2002), which also stressed the need to minimize waste by intelligent design. The Institute of Industrial Engineers published in its journal *IIE Solutions* Brian K. Thorn's and Philip Rogerson's "Take It Back" (April 2002), which stressed the importance of designing for reuse or remanufacturing. Anthony Brabazon and Samuel Idowu, in "Costing the Earth" *Financial Management (CIMA)* (May 2001), note that "take-back schemes may [both] provide opportunities to build goodwill and [help] companies to use resources more efficiently."

ISSUE 19

Should the United States Reprocess Spent Nuclear Fuel?

YES: Phillip J. Finck, from Statement before the House Committee on Science, Energy Subcommittee, Hearing on Nuclear Fuel Reprocessing (June 16, 2005)

NO: Charles D. Ferguson, "An Assessment of the Proliferation Risks of Spent Fuel Reprocessing and Alternative Nuclear Waste Management Strategies," from Testimony before the U.S. House of Representatives Committee on Science and Technology Hearing on Advancing Technology for Nuclear Fuel Recycling: What Should Our Research, Development and Demonstration Strategy Be? (June 17, 2009).

ISSUE SUMMARY

YES: Phillip J. Finck argues that by reprocessing spent nuclear fuel, the United States can enable nuclear power to expand its contribution to the nation's energy needs while reducing carbon emissions, nuclear waste, and the need for waste repositories such as Yucca Mountain.

NO: Charles D. Ferguson, Philip D. Reed senior fellow for science and technology at the Council on Foreign Relations, argues that even though reprocessing can help reduce nuclear waste management problems, because as currently practiced it both poses a significant risk that weapons-grade material will fall into the wrong hands and raises the price of nuclear fuel (compared to the once-through fuel cycle), it should not be pursued at present.

As nuclear reactors operate, the nuclei of uranium-235 atoms split, releasing neutrons and nuclei of smaller atoms called fission products, which are themselves radioactive. Some of the neutrons are absorbed by uranium-238, which then becomes plutonium. The fission product atoms eventually accumulate to the point where the reactor fuel no longer releases as much energy as it used to. It is said to be "spent." At this point, the spent fuel is removed from the reactor and replaced with fresh fuel.