

P R E F A C E



In 2000, I invited Maria, a graduate student at the University of Wisconsin–Madison, to address my undergraduate environmental studies seminar. Maria had grown up along the Fox River in Wisconsin, where paper mills lined the shore. During her childhood, the stench from the mill waste in the river had been so bad that the city of Green Bay had dumped perfume in the water. But perfume could not mask the toxic contamination. In the 1960s the paper companies had manufactured carbonless copy paper coated with industrial chemicals known as polychlorinated biphenyls (PCBs). Few scientists had suspected the potent hormonal effects that PCBs could have on developing fetuses and children, and the chemicals had gone essentially unregulated. Many of the PCBs used by the paper companies had made their way into the Fox River, where they had accumulated in the fatty tissues of fish.

Every Friday night, Maria's family had participated in the Wisconsin tradition of the fish fry, going to a tavern to eat their fill of local fish. On hot summer days they had splashed in the cool waters of Green Bay, where the Fox River empties into Lake Michigan. And now, decades later, the river was a Superfund site where various groups contested responsibility for cleaning up the PCBs, chemicals that had become notorious for their toxic properties, particularly their ability to disrupt hormone systems.

Although Maria was training as an environmental scientist, she did not talk to my class about the technical details of her research. She did not dwell on the hormonal effects of parts-per-billion of PCBs. She did not describe

the ways that PCBs changed thyroid hormone function or the ways that the chemicals altered brain development and the immune system. Instead, she talked with us about her young child. Should she breastfeed her daughter, she asked us. Maria knew from her research that, as an infant, her child was particularly vulnerable to chemicals that had accumulated into more concentrated and toxic forms. Breastfeeding would reduce the concentration of PCBs in Maria's own body, accumulated over decades lived along the Fox River. But she would pass on those chemicals to her daughter, with unknown and potentially tragic effects. Knowing that her own body was a toxic waste site, how could she breastfeed her child? At the same time, knowing that breast milk offered many health benefits to babies, how could she deny those to her daughter?

Like the rest of my class, I was haunted by Maria's dilemma. The thought that we have saturated rivers, wildlife, and ourselves with synthetic chemicals with potentially toxic effects began to trouble me. Maria's story gave a human face to the accumulating data suggesting that reproductive problems are increasing across a broad range of animals, from Great Lakes fish to people. Many researchers suspect that the culprits are synthetic chemicals that disrupt hormonal signals, particularly in the developing fetus. In the past decade, thousands of experimental studies have shown that synthetic chemicals can alter hormones in laboratory animals and wildlife, while numerous human studies have found correlations between exposure to industrial chemicals and reproductive problems. Endocrine-disrupting chemicals are not rare; they include the most common synthetic chemicals in production. Since World War II, synthetic chemicals in plastics, pharmaceuticals, and pesticides have permeated bodies and ecosystems throughout the United States, often with profound health and ecological effects, yet the government has largely failed to regulate them. How has this massive regulatory failure come about? Given what has been known about the risks of endocrine-disrupting chemicals since the 1940s, why have federal regulatory agencies done so little to protect public and environmental health?

Industry advocates argue that government bureaucracies have held back progress by overregulating chemicals, banning or limiting their use without scientific proof of harm. Environmentalists counter that both laws and ethics forbid the human experiments that would provide that proof. In the absence of complete knowledge, environmentalists argue,

what is known as the precautionary principle should guide the regulation of toxic chemicals: if an action might cause severe or irreversible harm to complex systems, the burden of proof should be on the industry to show that it is safe, rather than on affected communities to show that it is harmful.

What can history teach us about scientific uncertainty and the precautionary principle that can help guide the regulation of endocrine disruptors? In *Toxic Bodies*, I examine the histories of several key endocrine-disrupting chemicals, including the synthetic estrogen diethylstilbestrol (DES), various pesticides such as DDT (Dichloro-Diphenyl-Trichloroethane), and several compounds found in common plastics. In each of these cases, scientists had substantial cause for health concerns when the chemicals were introduced, yet in each case, federal agencies were slow to protect public health.

The most detailed case study in this book focuses on DES, the first synthetic chemical to be marketed as an estrogen and one of the first synthetic chemicals identified as an endocrine disruptor. Beginning in the 1940s, millions of women were prescribed DES by their doctors, at first to treat the symptoms of menopause. In 1947 the Food and Drug Administration (FDA) approved DES for pregnant women with diabetes, and drug companies advertised it widely, promoting the use of DES in *all* pregnancies as a way to reduce the risk of miscarriage. Although no evidence ever supported this claim, millions of pregnant woman took the drug.

Meanwhile, millions of Americans were also being exposed to DES through their diet. Beginning in 1947, DES was approved in the United States as a steroid to promote growth, first in poultry and then in cattle. High levels of DES were soon detected in poultry sold for human consumption—up to a hundred times the concentration necessary to cause breast cancer in mice. Concern over DES's effects soon grew among women who used the drug, farmers who handled treated livestock, and workers who manufactured the material. Federal agencies initially dismissed these concerns as unfounded, but eventually, after exposed male agricultural workers suffered sterility, impotence, and breast growth, the FDA banned the use of DES implants in chicken in 1959, while allowing its continued use in cattle feed and for pregnant women.

The chemical became an environmental issue as well as a personal health issue. By the 1950s, farmers gave cattle the hormone to promote rapid

weight gain, which was a key factor enabling the rapid expansion of industrialized feedlots. As cattle excreted waste, the metabolic byproducts of DES moved from feedlots into broader ecosystems, exposing a wide range of wildlife to the hormone. Chemical residues in the food supply changed the internal ecosystems of humans, livestock, and wildlife, interconnecting their bodies with their environment in increasingly troubling ways.

In 1971 researchers in Boston reported a cluster of extremely rare vaginal cancers in young women whose mothers had taken DES while they were pregnant. These problems had not been apparent at birth; they emerged only at puberty or young adulthood, sometimes decades after fetal exposure. Mothers and children exposed to DES organized to call for research into the drug, and eventually consumers, scientists, and concerned congressional representatives forced the FDA to ban the chemical for most uses.

The full dimensions of the health and environmental disaster that resulted from widespread DES use are only now becoming apparent. By 2002, DES had emerged in toxicological studies as a carcinogen and developmental toxicant so potent that the toxicity of other chemicals is often measured against it. Of the two to five million children who were exposed to DES prenatally, nearly 95 percent of those sampled have experienced reproductive-tract problems, including menstrual irregularities, infertility, and higher risks of a variety of reproductive cancers. At the peak of its use in the 1960s, DES was given to nearly 95 percent of feedlot cattle in the United States, which meant that millions of people consumed meat tainted with the artificial estrogen, and the estrogenic wastes from feedlots made their way into aquatic ecosystems, with unknown effects.

Why did the FDA approve the drug? Even before the agency approved DES in 1941, researchers knew that it caused cancer and problems with sexual development in laboratory animals. These concerns initially led FDA commissioner Walter Campbell to reject the drug in 1940, arguing that regulators must follow what he called the “conservative principle,” essentially adopting the precautionary principle sixty years before that term came into common usage. Yet a year later, the FDA abandoned its position of precaution, and by 1947 the agency was telling critics of DES that it was up to them to prove that DES had caused harm, rather than up to the drug companies to show that it was safe. When companies applied for approval to use DES in livestock and for pregnant women, the same

pattern unfolded twice more. Each time, the agency refused approval, citing the need for precaution given the known risks of the drug. But each time, it quickly gave way to industry pressure.

Why were regulators unable or unwilling to resist industry pressure? To begin with, they tended to share certain cultural and conceptual beliefs that industry lobbyists were quick to exploit. Contemporary scientific models of toxicology and development generally did not allow for the possibility that very low levels of synthetic chemicals could influence hormonal actions in the body. Indeed, emerging research that showed the harmful effects of various synthetic chemicals often seemed to violate the standard toxicological paradigms of the era, making it difficult for regulators to interpret scientific results. Even when experimental evidence from laboratory animals seemed to provide compelling proof of harm, uncertainty about the validity of animal studies in assessing risks for people made it difficult for regulators to defend principles of precaution in court.

In addition, confusion about the boundaries between natural and synthetic chemicals made it difficult for agency staff to understand why synthetic chemicals might cause harm even though natural estrogenic chemicals, which were relatively common in the food supply, did not. Cultural assumptions about gender differences also shaped the ways that scientists, regulators, and consumers understood hormones and their effects on the body. Finally, many regulators shared with industry staff a modernist worldview that combined faith in scientific expertise with the belief that technological progress could and should control nature. These beliefs often made regulators more skeptical of consumer claims of harm than they were of industry claims of safety. And while individual staff members within the federal agencies worked hard to protect public health, political appointees who headed the agencies often seemed more responsive to industry concerns about profits than to their own staff's concerns about risks.

Political, cultural, and scientific pressures all shaped these repeated retreats from precaution, and the echoes of those decisions still haunt us today. Menopausal and pregnant women no longer take DES, and livestock are no longer fattened with it, but Americans still face problems posed by chemicals with similar hormonal effects. Livestock continue to be treated with steroids, while pesticides continue to proliferate in the food supply. Plastics such as bisphenol A leach chemicals with hormonal activity into the drinking water, and every month brings new reports of

intersex fish and cancer-ridden whales. And the effects of DES exposure still confront many people, including the women who took the drug, their sons and daughters who were exposed to it in the womb, and the people who ate meat tainted with its residues.

As I worked on this book, I found myself exploring a parallel toxic history of my own possible exposure to DES. Like 20 to 80 percent of American women, I had uterine leiomyomas, tumors commonly known as fibroids. Estrogen stimulates these tumor cells, and fetal exposure to DES has been linked to fibroid growth. Midway through the research, my formerly benign fibroids suddenly became much more problematic. Doctors poked me up and down, debating whether the unusually rapid tumor growth meant I had uterine cancer rather than just fibroids. They speculated about whether my cervical dysplasia, deformed uterus, and tumors might be signs of DES exposure. After a hysterectomy, when the pathologist's report indicated that "no normal uterine tissue" had been found in my "grossly malformed" uterus, I realized that a book I had thought was about other people's experience might indirectly be about my own.

My typical bias is to be skeptical. I have been trained, first as a scientist and then as a historian, to distrust correlations as causal explanations and to be leery of those who see disaster everywhere. But my perspective shifted slightly as I began thinking about my own family's cancers and the toxic chemicals present in the places where I grew up on the East Coast and where I now make my home in Wisconsin.

I started wondering how many of my family's cancers and reproductive problems might be linked to toxic exposures. My mother had colon cancer; my grandmother died of melanoma; a great aunt had breast cancer; another aunt died of pancreatic cancer. Of the five women in my immediate family, all have had a string of reproductive issues: miscarriages, infertility, endless fibroids, diseased fallopian tubes, ectopic pregnancies, cervical dysplasia, hysterectomies, excised ovaries, suspicious mammograms and breast biopsies, and two cases of suspected uterine cancer. Each of these is linked, in laboratory studies on animals and in epidemiological studies on women, to endocrine disruptors. But not a single one of us can point to a specific reproductive problem and pinpoint a specific exposure as the cause. Any given reproductive failure could have been random bad luck. Any given exposure was probably harmless. Nevertheless, we have somehow created a world in which mushrooming chemical exposures go

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hand in hand with reproductive chaos, partly because no one can prove that an individual chemical caused a particular health problem, and so regulators have largely failed to act.

In this book I examine the landscape of exposure that begins in our own bodies and connects us across generations, across species, and across ecosystems. Failures of regulation are expressed not just in hearings and court cases, but also inside our own bodies. How did Americans persuade themselves after World War II that it was a good idea to release millions of tons of chemicals known to be toxic into the environment? What assumptions about scientific expertise, the role of experts, and vulnerability to natural fluctuations drove the postwar generation's faith in better living through chemistry? How did we come to accept the increasingly toxic saturation of our bodies and our environments? How have cultural constructions of sex and gender shaped scientific and policy responses to endocrine disruptors? Diethylstilbestrol has largely been banned, but thousands of other endocrine disruptors remain in common use. Learning the lessons of DES can help us address current disputes over regulating today's endocrine-disrupting chemicals.