In the late 1970s, anglers in England began reporting that they were catching bizarre fish—rainbow trout that seemed to be partly male and partly female. Perplexed, a biologist from Brunel University named John Sumpter went out to investigate these reports of what the media called “sexually confused fish.” He discovered that the trout were males that had indeed developed female characters, and he found that the best places to find these gender-bending fish were near sewage plants, in the lagoons and pools just below the discharge outlets for treated waste. The obvious question occurred to him: could anything in the sewage effluent be affecting the masculinity of the fish?

Sumpter, with the help of fellow Brunel University biologist Charles R. Tyler and researchers from the Ministry of Agriculture, Fisheries, and Food, found male fish that were producing elevated levels of vitellogenin, a protein responsible for making egg yolks in female fish. Male fish possess a gene that can produce vitellogenin when triggered by estrogen, but ordinarily males lack enough estrogen—a female sex hormone—to trigger this gene. To test whether sewage treatment plants had anything to do with the elevated levels of vitellogenin in the male fish blood, the scientists took healthy male trout raised in captivity, put them in cages, and placed them for three weeks near the discharge points of thirty different sewage treatment plants. Soon those males began producing vitellogenin, just like females.

Some of the scientists suspected that industrial chemicals such as nonylphenols—breakdown products of detergents, pesticides, and cosmetics—might be responsible for feminizing the males. But initial studies failed to support this hypothesis. They wondered next if the problem might stem from birth control pills. Estrogens from contraceptives, they reasoned, might not be completely breaking down in the women’s bodies or in the sewer treatment process, so those estrogens might be ending up in the effluent and somehow stimulating males to become females.
Lab experiments showed that ethynylestradiol, the main estrogenic compound in birth control pills, could indeed stimulate male fish to produce female proteins, even when concentrations were as low as one nanogram per liter of water. But the scientists could not initially find ethynylestradiol in the rivers at levels their instruments could detect. Other researchers suspected that the male fishes’ problems might stem from something even stranger: during certain times of their menstrual cycles, women’s urine is brimming with estrogen. Perhaps this urine was powerful enough so that, even after being diluted and treated, it could alter the natural world. Could hormones in women’s urine—whether of natural or synthetic origin—be feminizing male fish?

Sumpter’s results suggested several disturbing things. First, we might be poisoning wildlife with chemicals that alter their hormonal systems, sometimes even transforming them from one gender to another—transformations that could harm their ability to successfully reproduce and thus threaten their survival. Second, some researchers suspected that what was happening to the fish might also happen to humans. And third, the results suggested that industrial chemicals were not the only potential problem: our private bodily functions might also be altering gender in the environment.

On the most basic level, we tend to assume that what happens inside our bodies is a personal matter. When we take those pills, those cups of coffee, those birth control hormones, we assume that they’re disappearing into the black box of our innermost bodies. Yet those chemicals, both natural and unnatural, don’t end with us; they come out, get flushed down the toilet, and make it through our septic tanks or sewer systems into the waters we share with other creatures. In many places now, water carrying our bodily effluents may form more than half the flow in a summer stream. And remnants from our bodily wastes may be changing the nature of gender, turning male fish into female fish and female fish into male fish—and potentially affecting human development as well.

In this chapter, I will examine some transformations in the biological constructions of gender since the 1930s. To do this, I will look at two sets of changes. First, I will examine changes in the levels of natural sex hormones in women’s bodies, changes stimulated in part by larger societal transformations. Second, I will examine what are called endocrine disruptors, the industrial pollutants that mimic female sex hormones and disrupt the endocrine systems that control the biological expression of gender in wildlife as well as people.

Endocrine disruptors connect environmental histories of the body with environmental histories of wild places and wild animals. What we eat, what we drink, what we excrete, how we procreate: these are at the core of our animal selves. Our bodies are how we’re most natural, but now, whole as it turns out. The British people’s science was queer for sure. Perhaps the fem could have feint from the sea or pot in every a glass of lemonade. It’s the lemon, papaya, pomegranate from mill and Swe pulpy. It’s just
now they’re also how we’re most industrialized, since our bodies are where our industrial chemicals are coming back to haunt us. As strange as it may seem, even our pee has become hazardous waste, capable of turning male fish into quasi-females. In this chapter I will explore what endocrine disruptors might mean for the nature of gender and for our relationships with the environment.

The more researchers looked, the more they found that the waters of Britain, Europe, and America were laden with chemicals excreted in people’s urine and that these chemicals could be causing widespread reproductive impairment in males from many different species. For example, scientists in Minnesota found male carp and walleyes in the effluent of sewage plants that weren’t making sperm but were producing high quantities of vitellogenin instead. Other studies in the Great Lakes region found male white perch that had become intersex—part male, part female, and completely uninterested in sex. Urine, researchers found, could contain over sixty different synthetic chemicals, not to mention plenty of natural hormones as well. It turns out that our pee is doused with poisons—metabolites from the breakdown of birth control pills, caffeine from all the coffee and Mountain Dew we’re quaffing, remnants from the aspirin and Tylenol and anticholesterol drugs we use to stanch the pain of our modern ailments. These chemicals in sewage effluent seemed to be altering the action of hormones during development, with potentially profound effects on the fetus.

Industrial chemicals as well as sewage effluents were also implicated in gender switches. Students on a biology field trip in Florida noticed that every single mosquitofish they found seemed to be a male, for each had a gonopodium—an anal fin that males use for copulation. But many of these “males” turned out to be pregnant, and the students found that all the females in this stretch of the creek had been masculinized. The problem, biologist W. Mike Howell learned, was that wastes from pulp and paper mills were contaminated with chemicals that acted like androgens, male sex hormones that trigger the production of testosterone. Females from other fish species also became masculinized when exposed to pulp mill wastes; killifish and sailfin mollays developed fins resembling gonopodia, becoming extremely aggressive as well. In one researcher’s words, female killifish stopped acting like normal, well-behaved females and instead became “like little sharks.” Bluegill sunfish, American eels, Swedish eelpouts—all these fish became masculinized in the presence of pulp mill waste in streams across the world.

Gender confusion wasn’t limited to fish. Male alligators exposed to DDT in Florida’s Lake Apopka had abnormally small penises. They were just one-half to one-third the normal size, too small to function, and these
males also seemed to have ovaries, while the females produced abnormal eggs. Two-thirds of male Florida panthers—an endangered species whose total population only numbered thirty to fifty individuals in 1994—were found to have cryptorchidism, a condition in which the testes don’t descend, much less produce normal sperm. These male panthers are producing twice as much estrogen as testosterone, while normal male panthers do the reverse. In some western rivers, male Chinook salmon have undergone sex-reversal; many of the fish that look like females are actually males that have suffered a hormonal emasculation. Prothonotary warblers in Alabama, sea turtles in Georgia, mink and otters around the Great Lakes—all show reproductive malformations.

The list of recent gender transformations in wildlife can go on and on; female Great Lakes gulls and terns that try to mate with other females instead of with males. "Gulls in these colonies show excessive chick mortality, birth defects, and skewed sex ratios, with an excess of females." Male Atlantic cod and winter flounder that show reduced testosterone levels, hampering reproduction. Female Atlantic croakers (a kind of fish) that aren’t growing normal ovaries. Male porpoises that don’t have enough testosterone to reproduce. Polar bears on the Arctic island of Svarlbad that are hermaphrodites, which is not something one normally sees in a polar bear. In perhaps the most disturbing example of gender switches, Gerald A. LeBlanc of North Carolina State University in Raleigh found that over one hundred species of marine snails were experiencing something known as imposex, a pollution-induced masculinization. Females developed a huge malformed penis that blocked their release of eggs. They swelled up with eggs that couldn’t get out and then they died.

By the 1990s, researchers noticed that it wasn’t only wildlife that were showing difficulties with their reproductive health. Increasing numbers of people were as well. As with panthers, the incidence of cryptorchidism (undescended testicles) in British men doubled in two decades. In the three decades since 1970, American boys appear to have become increasingly likely to develop hypospadias, a birth defect of the penis. Testicular cancer has increased in many industrialized countries. For example, in Denmark, the incidence of testicular cancer has more than tripled since World War II, while in the United States, the incidence increased by 51 percent between 1973 and 1995. Similar increases are occurring in other Scandinavian countries and in Scotland.

Since the 1950s, sperm counts in some (but not all) regions across the world have declined significantly. Men in many industrial nations are showing increases in prostate cancer; for example, a 1999 review found that men in the United States in 1994 had "about a three- to four-fold risk of being diagnosed with prostate cancer compared with their fathers." Much of this increase in the number of diagnosed cases was probably due to better screening incidence of that, across breasts at y shows an iomemiosis.

What, and reproductive wildlife problems may stem from sex hormones being changed with synthetic chemicals like PCBs, pesticides—disrupting endocrine systems. Endocrine systems are crucial to the development and function of all living organisms. These endocrine systems are responsible for regulating a wide variety of physiological processes, including growth, development, reproductive function, and metabolism.

These endocrine systems are influenced by a wide range of factors, including genetics, environmental factors, and lifestyle choices. When endocrine systems are disrupted, it can have a range of negative effects on health and well-being.

In recent years, concerns have been raised about the impact of synthetic chemicals on endocrine systems. These chemicals, known as endocrine disruptors, can interfere with the normal functioning of endocrine systems, leading to a variety of health problems. Endocrine disruptors are found in a wide range of products, including plastics, personal care products, and household cleaners.

As these endocrine disruptors have become more prevalent in the environment, there has been increasing concern about their potential impact on human health. Studies have shown that exposure to endocrine disruptors can have a range of negative effects on health, including changes in behavior, development, and reproduction.

In addition to their direct impact on health, endocrine disruptors can also have indirect effects on health. For example, endocrine disruptors can interact with other factors, such as diet and lifestyle, to increase the risk of developing chronic diseases.

Overall, the evidence suggests that endocrine disruptors are a significant public health concern. Continued research is needed to understand the full impact of these chemicals on human health and to develop strategies to reduce exposure to endocrine disruptors.

Postmodern critiques of the biological construction of gender and sexuality have challenged traditional views, emphasizing the fluidity and variability of gender identity. These critiques have highlighted the ways in which the biological construction of gender is influenced by cultural and social factors, and have called into question the idea that gender is a fixed and unchanging characteristic.
ter screening tests, but researchers were nonetheless concerned that actual incidence was also increasing for unexplained reasons. Studies suggest that, across the United States and Puerto Rico, many girls are developing breasts at younger ages. Other research on women’s reproductive health shows an increase in the incidence of misshapen wombs, infertility, endometriosis, fibroids, breast cancer, and ovarian cancer since the 1950s.

What, if anything, connects all these bizarre problems with gender and reproductive health? Are there any links between human and wildlife problems? Possibly; many researchers now believe these changes may stem from the consequences of fetal events—namely, imbalances in sex hormones during fetal development. Since the 1950s, we may have been changing the biological basis of gender by filling the world’s waters with synthetic chemicals that alter the balance of sex hormones controlling the biological development of gender. Hundreds of the synthetic chemicals we started dumping into the environment since World War II—PCBs, DDT and other pesticides, dioxins, many compounds in plastics—disrupt the action of natural sex hormones, particularly on the fetus. Endocrine disruptors don’t just shut down the endocrine system; they can actually fool it into “accepting new instructions that distort the natural development of the organism.”

These estrogen-disrupting chemicals are not rare; some of them are among the most common synthetic chemicals in production. We now live submerged in a sea of estrogens, some natural and some synthetic. These estrogens affect men as well as women, wildlife as well as humans. Tracing the pathways of that estrogen—its naturalness and its unnaturalness—can illuminate an environmental history of gender.

The Greeks called the main female sex hormone “estrogen” because it produced the state of “estrus,” when a female goes into heat. Estrus, in turn, derives from the Greek word oistros, which means “frenzy,” or a woman driven wild. Estrogen, like other hormones, is a chemical produced by the body that regulates the body’s growth and development. Hormones are messengers that create a complex signaling system (called the endocrine system) that tells the body what to produce, where, and when. Sex hormones tell the developing fetus to develop a penis or not, as the case may be. Sex hormones tell a boy’s testicles to descend, a girl’s breasts to develop, a woman’s ovaries to grow eggs. They control what makes us female and what makes us male.

Postmodernists like to imagine that gender is culturally constructed, and clearly cultural forces do shape the expression of gender differences in our society. But gender is also profoundly biological. Hormones control the biological construction of gender, and now hormone mimics may control the biological deconstruction of gender as well. To complicate matters, cultural constructions influence the biological constructions of gender because
behavior, social interactions, and expectations can all change the ways our bodies produce sex hormones. On a more direct level as well, culture alters the biological control of gender differences because many of the chemicals in our culture produces have powerful effects on hormonal functions.

Sex hormones link wildlife and humans, wild places and human places, because we share our hormonal systems with animals. Surprisingly, the same chemical can act like a hormone in an alligator, a fish, a panther, and a woman. For 300 million years of evolutionary history, the hormonal system has been remarkably conserved, the reproductive endocrinologist Frederick vom Saal argues, because “it’s so critical to life. So if a chemical can disrupt the endocrine system by acting as an estrogen in a fish, for instance, the likelihood is that it will do that in humans. Endocrine disruption in fish has to be a concern with regard to human health. Not just mice, not just birds or reptiles, it’s all of them. They’re all sentinels for our health because these chemicals are in all likelihood operating on systems that we all share.”

The fact that we share our hormonal systems with other vertebrates means that what happens out in wild places also happens within our bodies. For example, the PCBs dumped in the Hudson River decades ago may well be stimulating tumors in women’s wombs and breasts to swell. It is within our bodies that we are most vulnerable to the pollutants we think we’ve disposed of.

HOW HORMONES WORK: CREATING GENDER

Normal development of a creature, from egg to adult, is controlled by the balance between hormones. Tiny changes in this balance signal growth, sexual differentiation, and other critical functions from the control of blood sugar and metabolism to brain development to the growth and function of reproductive systems. The endocrine system, like the nervous system, is a communication network that regulates all functions of the body. Glands within the body secrete chemical messengers—hormones—which travel through the bloodstream until they encounter cells with specific target receptors. Each hormone has a unique shape that fits the shape of the receptor protein at the target cell; imagine the hormone as your key and the receptor protein as the lock in your door. But hormones are also flexible: a given receptor protein may exist on different cells in different organs, so that the body can use the same hormone to perform radically different functions in different tissues. Hormones act slowly (compared, say, to the nervous system, the other communication network in the body), and their effects persist in the body for long periods of time (again, compared to the nervous system, whose effects are very rapid and very short). Most important, the endocrine system is designed so that incredi-
A provocative new study shows our exposure to some chemicals in the aquatic environment, particularly PCBs (polychlorinated biphenyls) and dioxins, alters the development of male and female offspring. These chemicals, which mimic the natural hormones in the body, can alter the development of reproductive organs in both males and females.

PCBs and dioxins are especially concerning because they are persistent in the environment and can accumulate in the bodies of organisms at higher trophic levels. This bioaccumulation can lead to higher concentrations of these chemicals in breast milk, which can then be passed to offspring through breast milk.

Biologically, the study found that exposure to PCBs and dioxins in the womb alters the development of the reproductive organs in both males and females, potentially leading to reproductive disorders later in life. This is significant because it suggests that even small exposures to these chemicals can have profound and long-lasting effects on development.

The study's findings emphasize the need for further research into the long-term effects of environmental chemicals on reproduction. It also highlights the importance of reducing exposure to these chemicals in the environment, as they can have far-reaching consequences for future generations.
some male biologists put it, estrogen receptors have a "promiscuous" pouch that welcomes lots of different chemicals. Cells don't need to hear from real estrogens; anything that binds to their estrogen receptors can have estrogenic effects, switching on cellular processes just as if a normal estrogen molecule had bound. And this method is how many synthetic chemicals seem to disrupt the endocrine system: they bind to estrogen receptors, fooling the body into thinking it has received a message from a real estrogen molecule.

Estrogen and other sex hormones control gender in fundamental ways, beginning with telling certain fetal tissues to turn into structures that are either male or female. About six weeks into a pregnancy, sex determination begins. The developing fetus is extremely sensitive at this point to confused signals from synthetic chemicals. For example, in the male fetus, Sertoli cells direct the development and descent of the testes, control the development of germ cells, and control the cells that secrete the hormones responsible for masculinization. Turning on too many estrogen receptors in the developing fetus could reduce the multiplication of Sertoli cells and fix their numbers at very low levels. This result could also affect descent of the testes and the development of urethra, setting into motion events that might lead to birth defects and testicular cancer.

The obvious question, however, is this: since natural estrogen occurs in very high levels in a pregnant woman, why isn't her own estrogen confusing the development of her sons? The answer seems to be that most of a pregnant woman's natural estrogen is tied up by something called "sex hormone binding globulin" (SHGB), a protein that protects the fetus from the mother's high hormone levels. Synthetic estrogen-mimicking chemicals are not tied up by SHGB, making them potentially more powerful at lower doses than a woman's own estrogen.

Normal sexual development depends upon getting the right hormonal signals at the right time in the fetus. If there is a tiny shift in the balance between hormones, a fetus might end up with the wrong number of digits, seriously confused genitalia, a uterus that's shaped wrong, a reproductive tract that cannot function, an immune system that later in life will turn against itself, and testicles or breasts that are programmed to develop cancer years down the road. Yet those effects might not be detected for many decades because problems that start in the womb's environment may not emerge until puberty or adulthood.

**CHANGES IN SEX HORMONES**

In the last fifty years, American women have been exposed to increasing levels of estrogen, not just from synthetic sources but also from our body's own reproduction greater pelvic case. We start our ancestors, while they may have cycles, we can also be on birth pills. Obesity which incalculating a did half of the changes in while they natural bo-trogen. Changes won a woman's sex eating gens as with excreting drinks a way leading to birth defects and testicular cancer.

Not only World War synthetic producers natural from plant both. Synthetic binding levels than much high 100 such as PCP, nancy and only to en mimics ac brains.
body's own natural estrogen. Throughout much of history, women of reproductive age were likely to have been pregnant or lactating for a greater percentage of their reproductive years than is now typically the case. We put off pregnancies, or else we don't ever get pregnant; we start our periods earlier, and we go into menopause later than our ancestors, which means that many women now have from 355 to 450 menstrual cycles during their lives. In earlier generations, many women may have averaged far fewer. As we get exposed to more menstrual cycles, we get exposed to more estrogen.\textsuperscript{33} Hormone replacement therapy can also bring more estrogen into women's bodies, as do most birth control pills.

Obesity rates in America have increased over the past fifty years, which increases estrogen levels, since fat cells produce estrogen from circulating adrenal hormones. Our diets contain more estrogen than they did half a century ago, especially through meat and dairy products. Changes in dairy production mean that cows are now often pregnant while they're being milked. This determinant leads to higher levels of natural bovine estrogen in that milk, since pregnant cows have more estrogen. Changes in diet during the last half century have also altered the ways women metabolize their own estrogen: the more fat and protein in a woman's diet, the more she recycles her own estrogens in her gut, in effect eating them twice. Drinking alcohol leads to higher levels of estrogens as well, because the liver is important in breaking down and excreting estrogen. When a woman drinks more than about fourteen drinks a week, the liver can no longer break down estrogen effectively, leading to higher estrogen levels in the body.\textsuperscript{34}

Not only do women have their own estrogens to contend with; since World War II, women have had to deal with ever-increasing sources of synthetic estrogens and estrogen mimics. Although women's bodies produce natural estrogens and women also eat phytoestrogens (estrogens from plant sources) in their food, synthetic estrogen mimics differ from both. Synthetic estrogen mimics tend to be flexible molecules that can bend into many shapes and fit into many different cellular receptors, which means they can play havoc with the body's endocrine system. Unlike natural estrogen, estrogen mimics rarely bind with sex hormone binding globulin, so while they may at first be present in much lower levels than our own estrogen, their effective concentration can soon be much higher. Unlike natural estrogens, many synthetic estrogen mimics such as PCBs cannot be easily broken down by the body. During pregnancy and breast-feeding, these synthetic chemicals can be released, only to enter the fetus or the child. At other times, synthetic estrogen mimics accumulate in a woman's fatty tissues: breasts, ovaries, and brains.\textsuperscript{37}
DES

The first signs that synthetic hormones might disrupt development came with DES daughters. In a huge, uncontrolled experiment, over five million women during the 1950s and 1960s were given DES, a potent synthetic estrogen, to prevent miscarriages. After years of increasing problems with the children of DES mothers, researchers in the 1970s finally starting connecting their problems with the hormones given to their mothers. Ironically, the 1930s researchers had known that DES caused cancer in lab animals, yet these studies were ignored when the FDA approved DES for pregnant women in 1947. Why? And why were people so slow to consider that treating millions of women with a synthetic, untested hormone might be a bad idea? Examining these questions will illuminate how our problems with endocrine disruptors have developed.38

DES is notable because it is the only large experiment done on estrogenic chemicals with human subjects. DES was not a perfect experiment, of course, because it was not designed as one: few follow-up studies were done on the children of DES mothers, and many women had no idea they were even being given DES during their pregnancies. Most of the doctors who prescribed DES had retired by the time its effects were being recognized, so those patients have never been followed. Nevertheless, it has become a model for the “long-term effects possible from in-utero exposure to an endocrine disruptor.”39 What happened with DES illustrates both the effects that synthetic hormones can have on people and the dangers of our culture’s assumptions that people are so different from animals that animal experiments need not apply to humans.

In 1938, an English biochemist named Edward Charles Dodds first synthesized estrogen, creating diethylstilbestrol, or DES. Dodds’s work showed that hormonal function in people and animals could be induced by synthetic substances—something no one had been certain was possible, even though now it seems obvious to those of us who have grown up in the age of the contraceptive pill. After Dodds’s discovery (which he never patented), DES was manufactured quite cheaply from coal tar derivatives, soon becoming available under more than four hundred different trade names. (Since the drug was prescribed under so many trade names, most women never knew they had been given DES. Today, at least 64 percent of DES-exposed daughters do know that their mothers had ever taken the drug.)40

When DES was first produced, people knew two things about it: it was extremely estrogenic, even more so than a woman’s own estrogen, and it was highly carcinogenic in lab animals. Studies in the late 1930s showed that mice exposed in utero to DES developed breast cancer, while in 1939 and 1940, studies showed that mice exposed in utero to DES sometimes de ductive organ proved the dr

For centuries, synthetic hormones were supposed to be a bad idea. Examining these questions will illuminate how our problems with disruptions have developed.38

DES is notable because it is the only large experiment done on estrogenic chemicals with human subjects. DES was not a perfect experiment, of course, because it was not designed as one: few follow-up studies were done on the children of DES mothers, and many women had no idea they were even being given DES during their pregnancies. Most of the doctors who prescribed DES had retired by the time its effects were being recognized, so those patients have never been followed. Nevertheless, it has become a model for the “long-term effects possible from in-utero exposure to an endocrine disruptor.”39 What happened with DES illustrates both the effects that synthetic hormones can have on people and the dangers of our culture’s assumptions that people are so different from animals that animal experiments need not apply to humans.

In 1938, an English biochemist named Edward Charles Dodds first synthesized estrogen, creating diethylstilbestrol, or DES. Dodds’s work showed that hormonal function in people and animals could be induced by synthetic substances—something no one had been certain was possible, even though now it seems obvious to those of us who have grown up in the age of the contraceptive pill. After Dodds’s discovery (which he never patented), DES was manufactured quite cheaply from coal tar derivatives, soon becoming available under more than four hundred different trade names. (Since the drug was prescribed under so many trade names, most women never knew they had been given DES. Today, at least 64 percent of DES-exposed daughters do know that their mothers had ever taken the drug.)40

When DES was first produced, people knew two things about it: it was extremely estrogenic, even more so than a woman’s own estrogen, and it was highly carcinogenic in lab animals. Studies in the late 1930s showed that mice exposed in utero to DES developed breast cancer, while in 1939 and 1940, studies showed that mice exposed in utero to DES
For centuries, doctors had tried to figure out how to prevent miscarriages. Suspecting that low levels of estrogen might be the problem, the synthetic hormone DES was at first given only to pregnant women with low estrogen levels and a history of miscarriage. A Harvard study published in 1947 suggested that the drug might reduce the risk of miscarriages, and FDA approval was given that year for use during pregnancy. Only two early studies suggested that DES reduced the rates of miscarriages, and later, more careful studies showed the opposite. In 1953 and 1958, two reviews of the available research showed that DES slightly increased the risk of miscarriages, even though it was supposed to be decreasing miscarriages. Nevertheless, the drug continued to be prescribed. DES was soon prescribed even for "normal" women to make a normal pregnancy more normal." By 1957, the Journal of Obstetrics and Gynecology recommended it for all women to produce bigger and stronger babies.

Meanwhile, millions of people were being exposed to DES through their diet. Beginning in the 1940s, DES was used in the United States as a growth promoter in poultry, hogs, and cattle. Very high levels of DES were soon being detected in poultry sold for human consumption—up to one hundred times the concentrations necessary to cause breast cancer in mice. When exposed male agricultural workers suffered sterility, impotence, and breast growth, the FDA banned its use in chicken and lambs in 1959, while allowing its use in cattle feed to continue and allowing it to be promoted as a wonder drug for pregnancy.

Given these findings, why didn't scientists ask whether DES might cause problems for the developing fetus? Until very recently, scientists and doctors had assumed the womb was inviolate and could not be affected by the outside world. People believed that the mother's placenta provided a barrier, protecting the fetus from harm. This belief partly reflected available technology: until the invention of ultrasound in the 1970s, the fetus was hidden off in the womb. People could not visualize the development of the fetus, so it was easy to assume the fetal environment was separate.

In 1962, when thalidomide was found to cross the placental barrier with profound effects on limb development, this belief that drugs could not cross the placenta was finally disrupted. Yet concerns were still not raised about DES. Thalidomide produced immediate, massive birth defects, while no birth defects were initially apparent with DES. Few doctors or researchers could comprehend that a hormone given during pregnancy might have effects that would only emerge decades later when the children of DES mothers reached adulthood.

Sometimes developed liver cancer and were born with deformed reproductive organs. Yet these experiments were ignored when the FDA approved the drug.
By the late 1960s, some of the children born to mothers who had taken DES were becoming sexually mature. Problems began to appear in these children, but it wasn’t until 1971, after Arthur Herbst at Harvard Medical School showed a relationship between DES use and rare vaginal cancers in daughters (clear cell adenocarcinoma), that researchers suspected DES might be a problem. By November 1971, twenty-one cases of a formerly extremely rare clear cell vaginal cancer were linked to DES use, and the FDA issued an alert advising against the use of DES during pregnancy.\(^{45}\)

Ensuing research revealed that only about 1 percent of the daughters developed vaginal cancers linked to DES, but nearly 90 percent of DES daughters have experienced reproductive tract problems, including menstrual irregularities and infertility.\(^{46}\) For example, half of DES daughters have fertility problems, well above the rate of the general population. Sons of DES mothers have higher rates of undescended testicles, cysts of the reproductive tract, low sperm counts, and testicular cancer, while both sons and daughters showed depressed immune systems, higher rates of depression, and lowered libido. As these findings emerged, the FDA began trying to withdraw the chemical from use, but this action proved extremely difficult. In 1972, the FDA withdrew all approval for animal uses, but it took five years of litigation before the courts upheld this ban on the use of DES in animals.

At the peak of its use in the 1960s, DES was given to five million pregnant women and to nearly thirty million cattle, which means millions of people consumed meat tainted with artificial estrogen.\(^{47}\) The press usually depicts DES as simply an individual woman’s concern (like so many other things that affect women’s health). The message is: ask your mother if she used DES, and if the answer is yes, then worry. DES is rarely portrayed as a larger environmental concern. But DES is an environmental exposure issue as well, since through food and waste from feedlots, and possibly through women’s urine, the chemical entered the environment at large, exposing wildlife as well as women.

In the early 1970s, the toxicologist John McLachlan became concerned about the effects of DES in the food supply. He began studying its effects on development in mice, trying to find out if animal models could replicate the effects seen in daughters of DES mothers. Much to everyone’s surprise, he showed just that: DES-exposed female mice had increased rates of vaginal cancer, and exposed male mice exhibited reduced fertility, undescended or stunted testicles, and genital tumors.\(^{48}\) Work on the effects of DES on mice also showed that the artificial estrogen was feminizing male mice at the molecular level: male mice exposed prenatally to DES would express female proteins in the reproductive systems later in life, a result that at the time seemed astonishing. These findings, and McLachlan’s concern that estrogen disruptors. Many compound’s harm. As one of the leading scientists, vom Saal, said, “I was clear from the rest of life on that’s an animal, the endocrine disrupt model for human testing probably humans. Most co and vigor, and the of estrogen.\(^{51}\)

Although DES was linked to problems in 1950, Heinz American biologist Male chicks injected cent of normal size acteristics were chemically ceter the formation of formation and regul affecting reproduction further investigate were not followed. Meanwhile, si posed to DDT and tal chemicals might 1950s that DDT, a
had appeared in Harvard medical journals suggesting that DES caused vaginal atresia and other birth defects. 

By the late 1940s, McLachlan had accumulated enough evidence to argue in a New England Journal of Medicine article that the 200,000 DES daughters were at risk for both cancer and sterility. 

In the 1950s, it was clear that DES could cause cancer. 

In the 1960s, DES was linked to menstrual abnormalities and infertility. 

In the 1970s, DES was linked to bladder and other cancers. 

And in the 1980s, DES was linked to a wide range of other health problems, including heart disease and stroke. 

Although DES was still being prescribed to pregnant women, wildlife biologists were finding events in nature that resembled those induced by DES. In 1950, Howard Burlington and Verlus Frank Lindeman, two American biologists, showed that DDT could have estrogenic effects. Male chicks injected with a form of DDT had smaller testes (only 18 percent of normal size) and arrested development of secondary sexual characteristics compared to controls. They looked like hens—in effect, they were chemically castrated. 

Other researchers showed that DDT could alter the formation of enzymes in the liver, which would then alter the formation and regulation of estrogen, progesterone, and testosterone, affecting reproduction. Burlington and Lindeman urged researchers to further investigate these estrogenic effects of DDT, but their connections were not followed through by others. 

Meanwhile, signs of reproductive trouble in wildlife populations exposed to DDT and PCBs were emerging. An early sign that environmental chemicals might impair endocrine function was the discovery in the 1950s that DDT, a persistent organochlorine pesticide, caused bald eagles
BODIES

to lay eggs with thin shells. Reproduction in gull colonies heavily exposed to DDT began to decline in the late 1960s. Wildlife biologists observed that often two females, instead of a male and female, were sharing nests, and the young in the colonies had "grossly feminized reproductive organs."54 In the 1960s, as Rachel Carson warned of the ecological effects of pesticides and the links between humans and wildlife, scientists began wondering why eagles, peregrine falcons, and similar birds were not reproducing. Carson singled out DDT as the likely culprit in eagle eradication and noted that "the insecticidal poison affects a generation once removed from initial contact with it."55 Few people, including Carson, imagined that the problems might be hormonal in origin, yet in her insistence that wildlife effects had implications for humans, Carson's work was central to the core of the endocrine disruption hypothesis.56 But not for three more decades, until a wildlife biologist named Theo Colborn was studying problems with the Great Lakes, did anyone connect reproductive problems in wildlife to hormonal problems in people.

After finishing her Ph.D. in 1985, Theo Colborn took a position with the Conservation Foundation examining wildlife responses to pollutants in the Great Lakes. The Great Lakes have long been a trash can for industrial pollutants; DDT, PCBs, pesticides, and dioxins have all accumulated in their waters. About one-fifth of American industry and one-half of Canadian industry are located along the Great Lakes or tributary streams, making them a microcosm for problems with pollutants in industrial society.

Colborn found no shortage of wildlife problems in the Great Lakes region, but few consistent patterns. Some studies suggested elevated rates of cancer in certain species; other studies showed impaired fetal development, while others revealed changes in behavior. Little seemed to tie these results together.

Research by Frederick vom Saal had shown that developing fetuses could be extraordinarily sensitive to tiny differences in the amount of hormones in the fetal environment. Vom Saal noticed that female mice from the same litter—mice that were genetically identical to each other—showed dramatic differences in size and aggression. He discovered that womb position had a powerful influence on a female mouse's adult behavior. Certain positions within the womb were more exposed to androgens, bathing the mouse fetus in a few more molecules of sex hormones at certain critical stages of growth. In maturity, those mice were much more aggressive, slower to mature, and more "masculine"—all because of parts-per-trillion differences in hormones within the womb.57

Vom Saal's work made Colborn wonder if the disparate effects she was seeing in Great Lakes species might be linked by problems with fetal development. If vom Saal had shown that tiny amounts of hormones could lead to same be true i
doctrine syster
Peterson desc
posed prenat
Saal describe
knew that em
tion."58 Colb
c problems she
certain synthe
thus disrupt
leading to prc

Although
Colborn's hy
turns out that
port for this is
thesis of estro
had been over
Dodd's labor
rogenic respec
ted the chemi
synthetic com
likely that a v
will be found

The next
proliferation
inherent of the
relation betw
carcinogenic j
estrogenic act
no scientific n
ferent types o
the compound
were ones tha
class of the m
many PCBs.

Other w
emerged as ea
the rush to sy
French chemi
cer in male n
removed and
Ily existed ob-tective effects. I began not re-radica-n once Carson, her in-work. But not Colborn re-
on with illu-tants for in-
ismone-half ribu-
turs in in-
at Lakes elevatet fetal de-
ed to 
; fetuses mount of 
; other-
red that 

dult be-
andro-
re much because 
. effects she 
. with fe-
men could lead to great effects later in life for laboratory animals, might the same be true for wildlife? Might synthetic chemicals be disrupting the endocrine system in developing fetuses? In 1989, when Colborn heard Dick Peterson describe "the changes in the development of male rat pups exposed prenatally to very low doses of dioxin—the same changes vom Saal described in male mice that developed between two females—she knew that endocrine disruption was not just a product of her imagination." Colborn proposed a unified explanation to explain the myriad problems she observed in Great Lakes wildlife. She hypothesized that certain synthetic chemicals in the Great Lakes were mimicking estrogen, thus disrupting the action of sex hormones on fetal development and leading to problems in reproduction and behavior later in life.

Although many in the scientific community were initially critical of Colborn's hypothesis that synthetic chemicals could mimic estrogen, it turns out that Charles Dodd, the inventor of DES, had found some support for this idea in the 1930s. Yet in the excitement that greeted his synthesis of estrogen, his work on the estrogenic effects of other chemicals had been overlooked. From 1933 to 1938, a series of journal articles from Dodd's laboratory reported that certain chemicals seemed to induce estrogenic responses in animal tests. As early as January 1933, having studied the chemical properties of ovarian hormones, he had foreseen that synthetic compounds could indeed act like estrogens, writing, "It seems likely that a whole group of substances of related chemical constitution will be found to have estrus-exciting properties."

The next month, Dodd wrote to the journal Nature that "because cell proliferation which characterizes the estrus state is in some respects reminiscent of the early stages of a malignant growth, we have sought a correlation between substances having estrogenic action and those having carcinogenic properties." He had found that two potent carcinogens had estrogenic activity as well, a result that he believed was "striking" since no scientific model explained how one compound could create such different types of biological activity—estrogenic and carcinogenic. Among the compounds that showed estrogenic activity in Dodd's early studies were ones that had two phenol groups, what he called the diphenyls. Today, this class of compounds is called the biphenyls and includes many of the most problematic endocrine disruptors: DES, bisphenol-A, and many PCBs.

Other warnings that estrogens could cause serious problems emerged as early as the 1930s, but these warnings were largely ignored in the rush to synthesize new chemicals. For example, during the 1930s, a French chemist showed that estrogen exposure could induce breast cancer in male mice. Doctors recognized that if women had their ovaries removed and thus weren't exposed to estrogen, they rarely developed
breast cancer—in retrospect, a clear suggestion that estrogen could lead to cancer, so synthetic estrogens might be problematic. 

Half a century later, in 1987, Professors Ana Soto and Carlos Sonnenschein at Tufts University discovered the first hints that extremely common synthetic substances—plastics—might be leaching chemicals that could cause estrogen-like responses. Soto and Sonnenschein were examining how estrogens can make breast cancer cells multiply. They used a special line of breast cancer cells, isolated and grown in a lab, for their research; these cells will only grow in the presence of estrogens. Suddenly something went wrong with their work. Breast cancer cell cultures started growing and dividing on their own, even before the experiments had started, when nobody had added anything to them. Soto assumed someone in the lab had been careless and had contaminated the clean cells; then she thought someone in the lab had simply made a mistake and added estrogen to the wrong cell lines.

Eventually, she realized no one in the lab had made a mistake. The problem was in the new plastic tubes: something from these tubes was leaching into their cultures and stimulating the growth of breast cancer cells. The manufacturer had changed the formulation of the tubes without telling anyone, and those sterile tubes were leaching something that acted like an estrogen. This development astonished Soto and Sonnenschein because they knew of no one who had reported estrogens leaching out of plastics. Everyone, including the manufacturers, assumed plastics were inert.

The problem turned out to be something called nonylphenol, a chemical widely used in industry and domestic products such as paints, detergents, oils, toiletries, and agrochemicals (ironically, this was one of the chemicals that Dodd in the 1930s had reported to possess estrogenic properties). Nonylphenols are just one in a larger class of related chemical compounds called alkylphenols, many of which turn out to be weakly estrogenic, making breast cancer cells multiply in lab cultures. In Britain alone, twenty thousand tons of these chemicals are used a year, and a third of these end up in our rivers and lakes at concentrations of fifty micrograms per liter—levels higher than those that induce cancer cell responses in the lab.

Similar problems with other plastics emerged in the early 1990s. During the effort to create artificial estrogens in the 1930s, researchers had first synthesized bisphenol-A (one of the chemicals Dodd noted “excited estrus” in his lab animals). Not as powerful as DES, bisphenol-A was ignored until researchers realized that, when polymerized, it formed a useful plastic known as polycarbonate. Polycarbonate is now used for numerous common items: plastic baby bottles, water bottles, dental sealants, coatings for the inside of food cans. Unfortunately, bisphenol-A leaches out of the wildlife at concentrations in lab animals showed that, in fact, in amounts high enough to cause breast cancer. In the past decade, with cavity-retaining high concentrations of saliva. The sealants are getting chewed; these levels high levels of active systems are safe, either. I compounds at the FDA has never found to be safe. The ADA found that if emanation the theoretical implications. The ADA cannot study’s observation. The ADA is required to test these chemicals are ass the problem is the unethical to test pregnant women. Animal experiments cause mice to en normal (how ironic cause we exercise prenatal exposure recently demonstrated nol-D. In only two maximum concern. Vom Saal’s et al. evaluated Theo Colbc
could lead to lengthened cycles or other reproductive issues. Some researchers have suggested that the chemicals could lead to problems in future generations as well.

Carlos Sonnenberg, a leading expert in reproductive health, warns that these chemicals may be more harmful than previously thought. He notes that bisphenol-A, a chemical used in many everyday products, can leach out of those containers and into food and water. This can lead to exposure to the chemical even for those who don't use those products, leading to health concerns for future generations.

In recent years, there has been growing concern about the health effects of bisphenol-A, particularly among infants and children. Some studies have suggested that exposure to the chemical may be linked to conditions such as obesity, diabetes, and even certain types of cancer. These concerns have led to calls for more research into the effects of bisphenol-A and for increased regulation of its use.

There are also concerns about the effects of bisphenol-A on wildlife. Some studies have suggested that exposure to the chemical can lead to changes in behavior, reproduction, and even survival in various species. This has raised concerns about the potential for harm to ecosystems and the broader environment.

In response to these concerns, some countries have implemented regulations to limit the use of bisphenol-A in certain products. However, there is still much to be learned about the long-term effects of exposure to this chemical. As more research is conducted, it is likely that we will continue to learn about the potential dangers of bisphenol-A and the steps that can be taken to reduce our exposure to this chemical.
ently, vom Saal showed that extremely low levels of bisphenol-A—the levels found in our background, our levels of "normal" exposure—could induce potent responses. These are levels thought completely trivial by traditional toxicology. These are the background levels experienced by most of us living our normal lives in the normal world, not the levels of those living near a toxic waste dump. As Our Stolen Future's Web site states, "Vom Saal's work shows that every day levels matter."

A third group of chemicals used in making plastics that leach estrogenic compounds are phthalates. These are incredibly common substances, perhaps the most abundant synthetic compounds in the environment. Phthalates are oily solvents that make plastics flexible but strong. Since phthalates need to be flexible, that means the molecules can’t be too rigidly locked together, for flexibility requires molecules that slide over each other. But that lack of molecular rigidity also means they leach out easily. Phthalates keep your car dashboard from cracking and your nail polish from splintering; they allow plastic wrap to be shaped around food. They help cosmetics absorb quickly into your skin, so they are added to shampoos, skin creams, sunscreens—all the stuff we smear on our skin to stay pretty. In fact, as ABC News reported, loopholes in federal laws allow cosmetics manufacturers to put unlimited amounts of industrial chemicals such as phthalates into personal care products without any testing for adverse health effects.

Phthalates are in all of us. In March 2001, the U.S. Centers for Disease Control recently released the results of its first study of the levels of twenty-seven chemicals found within American bodies. Researchers found phthalates in nearly every person they examined (out of 3,800 people drawn from healthy individuals around the country with no special exposure to toxic substances). The highest concentrations came from certain phthalates (such as di-ethyl phthalate) used in toiletries like bar soaps, perfumes, and shampoos, perhaps because direct skin contact increases body burden. Some of the highest concentrations were in women of childbearing age—not the results anyone wanted to find, since fetal exposure is likely to be the riskiest. These levels were much higher than scientific models and a government panel had predicted just six months earlier.

In mice, phthalates "undermine the masculinity of mice exposed during lactation and weaning, creating individuals with both male and female sexual (intersex) characteristics." They do this by blocking the action of male sex hormones as they program sexual development. A team of Environmental Protection Agency researchers led by L. Earl Gray Jr. gave phthalates to female rats from weaning through lactation at doses of two hundred to one thousand milligrams per kilogram of body weight (levels similar to those that people are exposed to). The sons of these rats produced f
ticle. Anc
Chemical I
in male roc
grams of p
testosterone
when the e
What
necting hu
tion, not c
study inda
velopment
girls. Put
opment ev
high levels
ganic poll
ormal se
widely use
indicated
breasts at a
Yet ar
higher the
curred in t
PCBs and
migrant g
metabolite
gen mimik
not proof o
man healt
when girls
even with

Recer
American
younger t
exposure
Marcia H
girls were
year earli
the best q
by those t
girls were
percent of
produced far less testosterone than normal, and some males lacked a testicle.26 Another team of researchers from an industry group known as the Chemical Industry Institute of Toxicology found developmental defects in male rodents whose mothers were given as little as one hundred milligrams of phthalate per kilogram of body weight. The chemical halved testosterone production in the fetus while leading to testicular tumors when the animals became adults.27

What do these results mean for people? Nobody knows yet; connecting human problems to phalate exposure is an exercise in correlation, not causation. For example, consider early puberty. One recent study indicates that in Puerto Rico, girls who show premature breast development have significantly elevated phthalate levels compared to other girls.78 Puerto Rico has the highest incidence of premature breast development ever reported, and 70 percent of girls studied have significantly high levels of phthalates. As the authors of this study wrote, “Some organic pollutants, including pesticides and some plasticizers, can disrupt normal sexual development in wildlife, and many of these have been widely used in Puerto Rico.”79 Another study on these Puerto Rican girls indicated that those who had consumed DES in meat developed large breasts at an early age and had other signs of precocious puberty.80

Yet another study (this time in North Carolina) showed that the higher the level of prenatal exposure to PCBs, the earlier puberty occurred in the girls, while a study in Michigan found a correlation between PCBs and early onset of menstruation.81 A Belgian study showed that immigrant girls with precocious puberty had higher levels of DDE (a metabolite of DDT) in their blood.82 Such correlations suggest that estrogens mimics might contribute to premature puberty, but correlations are not proof of a causal relationship. Proving that an observed trend in human health is real turns out to be very difficult, for we lack good data on when girls entered puberty in the early twentieth century. Moreover, even with good historical records, it is nearly impossible to prove causation with correlations.

Recent research has found that some girls—particularly African-American and Latina girls—are developing breasts and pubic hair much younger than in past generations, a trend that may be linked to estrogen exposure (although the source of those estrogens is not clear). In 1997, Marcia Herman-Giddens’s data on 17,707 American girls suggested that girls were developing breasts and pubic hair on average as much as a year earlier than expected based on historical data (data that were not of the best quality). Not only did the average age of puberty (as measured by those two indices) appear to be decreasing, but significant numbers of girls were maturing sexually long before the average. By age eight, 48 percent of black girls and almost 15 percent of white girls were showing
signs of sexual development; 3 percent of African-American girls had begun to develop breasts by the age of three.\textsuperscript{83}

Why would some girls be entering puberty earlier? Some of the changes may be due to changes in diet, which as mentioned earlier have led to increased estrogen exposure. Some of the changes may be due to increases in obesity, which is correlated with increased estrogen in the body (as well as with increased leptin, a hormone that is also linked with the onset of puberty).\textsuperscript{84} Some of the changes may be due to synthetic estrogen mimics in the environment, while others may be due to social factors such as increased exposure to sexual stimuli in the culture, which might trigger a girl’s body to begin puberty early. Although the science on people is uncertain, animal experiments do show that exposure to estrogen mimics reduces the age of puberty, suggesting the same might be true in girls. For example, a recent experiment reported in \textit{Nature} showed that pregnant mice exposed to bisphenol-A at “a dose equivalent to that typically found in the environment” had daughters who entered puberty early.\textsuperscript{85}

What do we do with this research? How do we think about women’s health from a historical perspective in a world where chemical contamination may be changing fundamentally the biological nature of what makes us female? Women’s bodies are biologically different from men’s bodies; sexual differentiation is not just a cultural construction. Yet the biological differences between men and women are also shaped by culture and by cultural expectation.\textsuperscript{86} For example, consider sexual size dimorphism, which is a measure of the size differences between men and women. Male humans are, on average, larger than female humans. Yet these differences, while partly biological, are also shaped by culture. The degree of difference in size predicted by biological factors is much smaller than the actual difference you observe in many segments of American culture. Why? Largely because societal pressures on women to diet increase the amount of sexual size dimorphism in white American culture. Expectations for women to be thin (or, in different cultures, to be fat) are clearly cultural constructions that can magnify the effects of genetic differences between men and women.\textsuperscript{87}

Because women’s bodies are biologically and culturally different from men’s bodies, women react to endocrine disruptors differently than men do, and “women may be disproportionately affected by environmental pollution.”\textsuperscript{88} Or, as one government scientist put it, “Men’s and women’s bodies don’t just look different—they also react differently to environmental agents.”\textsuperscript{89} Women manufacture more estrogen than men do, so they are exposed to more estrogen to start with. Women tend to carry more fat, and fat is where most endocrine disruptors are stored and can accumulate. Our ovaries and breasts have very high fat concentrations, making them especially vulnerable to endocrine disruptors. Be-
cause of pressures on white American women to diet, their weight tends to yo-yo more than men. "If toxicants stored in fat tissue are mobilized during [dieting], as some researchers have suggested, then this could be a significant factor in gender differences in responding to environmental factors."90 Women retain higher levels of certain pollutants such as dioxin than men do, for reasons that aren't yet clear.91 Women have smaller livers with less capacity for getting rid of toxic chemicals, and their livers are more susceptible to damage from alcohol, which in turn exposes them to more problems from endocrine disruptors.

Women's production of babies illustrates the tangled relationships between cultural and biological constructions of gender.92 The timing and numbers of pregnancies are partly constrained by biology—two-year-old girls and ninety-year-old women don't get pregnant. But within those biological constraints, cultural forces shape whether a woman spends all her reproductive years pregnant or whether she spaces her pregnancies by choice. American women have fewer children than women at the turn of the century for reasons that are largely social and political, but biological factors related to chemical influences on fertility may also play a role.

Infertility appears to be increasing in American women (and in American men, but that is a different story). Part of this trend is probably tied to cultural changes: many women are delaying their first pregnancies, and older women are biologically less fertile. But infertility is also linked to chemical contaminants—causes that are environmental and political, not just personal. For example, women who eat fish from Lake Ontario (contaminated with PCBs) are less likely to conceive during a given menstrual cycle than those who eat less contaminated fish.93 Women who eat more fish from Lake Michigan (again, contaminated with PCBs) are much more likely to have problems with their pregnancies, if they can even get pregnant.94 Some phthalates clearly harm the ovaries in rodents; Barbara J. Davis, the leading researcher on ovarian toxicity, argues that "the effects of DEHP [a phthalate] could lead to infertility."95

Two increasingly important causes of female infertility in America are endometriosis and uterine fibroid tumors. Both are affected by exposure to estrogens (the body's own estrogens as well as synthetic estrogens).96 And both problems have become far more common since the 1950s. Endometriosis, which sounds like something out of a Stephen King novel, occurs when endometrial tissue—the tissue that normally lines the uterus and gets shed during a period—grows outside the uterus and implants itself on the ovaries, fallopian tubes, bladder, bowel, or vagina. Endometriosis can often lead to infertility, and for unknown reasons it is increasing in industrialized nations. Anywhere from 10 to 15 percent of premenopausal women suffer from the disease. Belgium has the world's highest incidence; 60 to 80 percent of Belgian women who are
infertile or have pelvic pain have endometriosis. Although the exact cause is unknown, we do know that many of the risk factors are related to estrogen exposure and so environmental estrogens may contribute. For example, dioxin is a known estrogen mimic, and monkeys given dioxin have been shown to develop endometriosis. Belgian breast milk has some of the highest dioxin levels in the world, and some small studies have shown that dioxin levels are highest in women with endometriosis. Other studies, however, have not found that infertile women who had the disease had higher levels of dioxin in their blood than infertile women who didn’t have it.

Fibroids are another example of a condition often leading to infertility that may be related to endocrine disruptors. The most common tumors in women are nonmalignant ones in the uterus called fibroids or, more technically, uterine leiomyomas, which are clearly linked to estrogen exposure. Fibroids are the leading cause for hysterectomies—at least 550,000 American women a year have hysterectomies because of problems with them. Clinical studies show that these tumors are increasingly common. In one large study, 77 percent of women had fibroids growing in their uteruses (most of these tumors were subclinical—too small to cause problems), with the highest rates in African-American women. Another study found that 73 percent of black women had uterine fibroids compared to 48 percent of white women. Animal studies show that endocrine disruptors increase fibroid growth in rodents and monkeys, yet no human studies have been published that examine this potential link.

The womb is an environment of its own, yet one that is linked to the outside world. The chemicals that a woman has been exposed to throughout her life—not just what she consumes while she’s pregnant—reach her fetus, connecting one generation to the other. Pregnant women hope that if they don’t take weird drugs like thalidomide or DES, their children will be fine. But chemical contamination is inside most women: 30 percent of pregnant women in one study had detectable levels of PCBs, DDT, and lindane and estrogenic compounds in their amniotic fluid, many at concentrations high enough to cause problems in lab animals. And these background levels of chemicals could have effects on developing fetuses. For example, in Missouri, the state health department showed that children exposed to pesticides in the womb developed 600 percent more brain cancer than other children (which sounds terrible, but the levels were still extremely low). Using roach control chemicals during pregnancy led to a doubled risk of cancer in the child; using termite pesticides led to a 300 percent increase in brain cancer in the children. Yet the message that pregnant women shouldn’t be using toxic chemicals is not getting out: the Missouri State Health Department found that 80 percent of pregnant women used pesticides while pregnant.

Once a baby way for a woman to breast-feed, only leave the mother’s problem behind. Breast milk is toxic; even its metabolites are toxic. The metabolites of women concentrations declining since can get into the milk showed that three times the.

What do the long-term studies levels of prenatal risk for children exposed to the environment find individuals compared as a probable cause by the Agency, while animals and humans? The answer in breast milk environmental. Noted with PCBs better for dairy-based milk mimics.

Are endocrine claims of the endocrine scientists, even to certain synthetic chemicals induced by sex hormone chemicals can be expression. They concentrations of ce most people are means. They agree cells multiply ii build up in humans.
Once a baby is born, problems don’t stop there. Just about the only way for a woman to reduce her own body’s burden of toxic chemicals is to breast-feed, since many of those toxic chemicals end up in breasts and only leave the woman’s body within her breast milk. But there’s an obvious problem here, since she’s giving those toxic chemicals to her child. Breast milk is the food with the highest levels of PCBs and DDT and its metabolites such as DDEs (all endocrine disruptors). In 1976, 99 percent of women’s breast milk in America contained PCBs; a quarter had concentrations exceeding the legal limit (these concentrations have been declining since regulations reduced PCB use). The PCBs in breast milk can get into the children’s blood; a 1998 study from the Netherlands showed that the blood of children who were breast-fed as infants had three times the levels of PCBs than children who weren’t breast-fed.

What do these levels mean for children? Nobody knows for sure. A long-term study of children in the Netherlands finds that background levels of prenatal PCB exposure experienced in the womb led to higher risk for childhood diseases, while “the latest study of the cohort of boys exposed in the womb to PCBs because of cooking oil contamination in Taiwan finds significant degradation in sperm quality in exposed individuals compared to their unexposed counterparts.” PCBs are labeled as a probable human carcinogen by the U.S. Environmental Protection Agency, while other studies show that they affect learning in both animals and humans. But could the levels found in breast milk harm children? The answer is unclear, since studies on postnatal exposure to PCBs in breast milk have been inconclusive. The National Institute for Environmental Health Sciences argues that breast milk, even when contaminated with PCBs and pesticides and other endocrine disruptors, is still better for the child than formula (especially given that both soy-based dairy-based formulas have their own sources of estrogens and estrogen mimics).

Are endocrine disruptors a serious problem or not? Some of the central claims of the endocrine disruption hypothesis are now agreed upon by all scientists, even those from industry. Everyone agrees that wildlife exposed to certain synthetic chemicals demonstrate responses similar to those induced by sex hormones. They agree that lab studies show that synthetic chemicals can bind with and activate hormone receptors, resulting in gene expression. They agree that exposing pregnant mice to extremely low concentrations of certain synthetic chemicals—concentrations similar to those most people are exposed to—results in offspring with reproductive problems. They agree that some synthetic chemicals can make breast cancer cells multiply in culture. They agree that persistent organic chemicals build up in human tissue and are passed to the developing fetus and to
the breast-feeding infant. They agree that many male fish and alligators exposed to industrial effluent show signs of feminization, a result also found in the lab when eggs are exposed to some synthetic chemicals.\textsuperscript{109}

But people still disagree on a fundamental issue: what do these animal and lab studies mean for people? Do average people—those who don’t work at toxic waste sites, for example—have anything to worry about? Can endocrine disruptors explain any of the apparent increases in infertility, reproductive cancers, birth defects, reduced sperm counts, or lowered ages of puberty? Or are endocrine disruptors present at such low levels that they are a trivial concern?

Circumstantial evidence is accumulating that supports the hypothesis that endocrine disruptors may be harming male reproductive health, while experimental studies have shown similar effects in laboratory animals. But you cannot ethically do these experiments on human fetuses to test whether the correlations between endocrine disruptors and reproductive disorders are real. Instead, we have to rely on epidemiology, which cannot always untangle confounding variables. Since we can’t ethically do experiments on fetal exposure in humans, we need to rely on the weight of the evidence, rather than experimental proof, to form policy.\textsuperscript{110} And this assumption is where reasonable people disagree.

In August 1999, the National Research Council (of the National Academy of Sciences) released its consensus report on endocrine disruption, a report commissioned in 1995 by the EPA and Congress.\textsuperscript{111} The team of authors included independent scientists who are proponents of the hypothesis as well as those with strong ties to industry who are critics of it. After four years of review and debate, the team finally managed to agree that endocrine disruptors at high concentrations do affect human and wildlife health, yet they could not agree on the extent of harm caused by levels common in the environment. Moreover, they argued that their disagreements were not only due to gaps in scientific knowledge but also to major epistemologic differences on how one interprets data and draws conclusions. The consensus report stated: “Much of the division among committee members appears to stem from different views of how we come to know what we know. How we understand the natural world and how we decide among conflicting hypotheses about the natural world is the province of epistemology. Committee members seemed to differ on some basic epistemologic issues, which led to different interpretations and conclusions on the issues of hormonally active agents in the environment.”\textsuperscript{112}

The chemical industry’s response to this report was to focus on the conclusion that no scientific certainty on human health effects had been established. Without certainty, the industry argued, endocrine disruption was not an issue for public health concern. As Myers argues, “This is a classic argument from industry spokespeople: that the absence of data proves safety. Firm proof, \textsuperscript{113} should do not respond is utility stated, “We spread exposure to multiple requirements” for which fail to e.

As I write this, I pa there might peals in which can women in presence of es drink my wel ruptor), when when I padd nearby wild lif der what stra.

When I w River, a little lands. For sou cry overhead, coons and wo out in the su rated with po water runs obey der if I really w and hawks ar residues end u low backwater soup. Can any ally knows.

What we gets sprayed o in the fish, the also ends up ir wildlife and w studies of wil connect envi o wild places.
and alligators, a result also of the hypoth-
edefective health, a nation that no
tories and reproductive epidemiology,
the National Acad-
"disruption, a team of au-
the hypoth-
er not agree that en-
used by levels of their disagree-
it also to major draws conclu-
we come to ld and how we world is the differ on some-
vironment."112 to focus on the facts had been nine disruption ques, "This is a
proves safety. In reality, all it proves is ignorance. "113 So, in the absence of firm proof, what should society do? Many in industry argue that we should do nothing until we have absolute proof. Others argue that such a response is unethical, for as the Boston Physicians for Social Responsibility stated, “We are engaged in a large global experiment. It involves widespread exposure of all species of plants and animals in diverse ecosystems to multiple manmade chemicals. . . . The limits of science and rigorous requirements for establishing causal proof often conspire with a perverse requirement for proving harm, rather than safety, to shape public policies which fail to ensure protection of public health and the environment.”114

As individuals, what do we do with this uncertain information? As I write this, I pat my own fibroid-filled belly and wonder what connections there might possibly be between my own tumors and the sea of chemicals in which we’ve immersed ourselves. Like the vast majority of American women now, inside my uterus I have cells gone wild, cells that in the presence of estrogen mushroom into tumors beyond my control. When I drink my well water (tainted with atrazine, yet another endocrine disruptor), when I eat my plastic-wrapped cheese soaked in phthalates, when I paddle my kayak through the pesticide-laden waters of the nearby wildlife preserve, when I walk through my ordinary days, I wonder what strange world we’ve created for ourselves.

When I was writing this chapter, I spent a lot of time on the Sugar River, a little muddy river that runs through wildlife refuges and farmlands. For southern Wisconsin, this is a wild place. Herons rise up, hawks cry overhead, geese and cranes and mallards and wood ducks fly, raccoons and woodchucks and chipmunks and squirrels and coyotes hang out in the surrounding forest. Yet for all its wildness, the water is saturated with poisons. When I dip the paddle blade too deeply into the river, water runs over the shaft and down my wrists, and I can’t help but wonder if I really want that water anywhere near my skin. I watch the herons and hawks and cranes around me and wonder how many pesticide residues end up in their body fat. Hunters motor their boats into the shallow backwaters, and solvents from their fuel tanks are added to the chemical soup. Can any of this hurt us? Can any of it hurt the birds? Nobody really knows.

What we do know is that we’re all in this together: the atrazine that gets sprayed on my neighbor’s cornfield ends up in the river water, then in the fish, then in the herons and the raccoons that eat the fish—and it also ends up in my breasts, my belly, and my blood. What’s out there in wildlife and wild places is also in our bodies. Just as Colborn connected studies of wildlife with studies of human bodies, endocrine disruptors connect environmental histories of the body with environmental histories of wild places and wild animals.
One of our culture’s fondest illusions is that we can control our separation from nature: we can visit nature when we feel like it and live in a human world the rest of the time. According to a commentator on National Public Radio, most Americans spend only fifteen minutes outside each day. The rest of the time we drive in our cars, stare into our computer screens, watch our TVs, sit in our offices or schools, eat our phthalate-saturated suppers, and think the rest of the world is outside, staying where it belongs. But that’s not true. What is outside has come inside, making itself at home in our testes, in our wombs, in our most private reproductive dysfunctions. The environment includes wild places and wild things, but it also includes hair dye, golf courses, and ice cream—all full of endocrine disruptors. Our most intimate reproductive environments, the places that make us most female or most male, the places we are most vulnerable and most natural, may have been hijacked by the residues of our industrial world. This is a disturbing thought.

NOTES


8. Chris Metc. ment” (abstract, C gsa.conference.com/gsa/ “Excreted Drugs.”
control our separation, we adopt it and live in a culture that regularly releases minute quantities of these compounds into our homes, offices, streets, and bodies. Phthalates and other endocrine disruptors are used in myriad products, from our personal care products to our drinking water.


16. This particular report was from the BBC, September 2000; cited at Our Stolen Future’s Web site, written and maintained by John Peter Myers. The Web site maintains abstracts on recent research and links to scientific journals; see http://www.ourstolenfuture.org.


22. For a review of conflicting evidence on changes in the timing of puberty,
see D. Zuckerman, "When Little Girls Become Women: Early Onset of Puberty in Girls," The Ribbon 6 (2001); this is the newsletter for the Cornell University Program on Breast Cancer and Environmental Risk Factors in New York State; online at http://www.ccf.cornell.edu/cecry/Newsletter/geral/061/little_girls.cfm.


28. Ibid.


30. Ibid., 48.

31. Raloff writes: "Gender—both its physical expression and its characteristic behavior—traces more to the relative concentrations of various sex hormones circulating in the body than to the mere existence of certain dominant ones. For example, women produce some androgens, or male hormones. Indeed, a woman's body synthesizes estrogens from androgens such as testosterone. Similarly, though estradiol is the animal kingdom's primary estrogen, or feminizing hormone, it plays important roles in both men and women. At no time does an imbalance of sex hormones produce more obvious results than during fetal development. Too much estrogen at the wrong moment can turn an organism with male genes into what to all outward appearances is a female. Similarly, an overabundance of androgens can produce the sex organs of a male in a fetus with the genetic make-up of a female child."

32. Cadbury, Alte

33. Cadbury, Alter

34. Cadbury, Alter

35. Although it is not a scientific study, a current hunter-gatherer society in India (the Gonds) have found that among women in their 30s and 40s, the age of 38.9 years is often the most common age of menopause. For a review of the latest research, see J. W. B. Strassmann, "Menstrual and Menopausal Changes in American Women," Journal of the National Medical Association 66 (1974): 770-79.

36. Cadbury, Alte

37. Berkson, Hormo

38. For a review of the latest research, see K. Iwamoto, and E. E. H

39. Berkson, Hormo

40. Krimsky, Hormo

41. Krimsky, Hormo

42. Berkson, Hormo

43. Berkson, Hormo

44. Cadbury, Alterin

in a fetus with the genes to be female" ("Are Men Suffering from Prenatal or Childhood Exposures?").

32. Cadbury, Altering Eden, 36; Berkson, Hormone Deception, 43.

33. Cadbury, Altering Eden, 38.

34. Myers, "Contamination Threatens a Basic Reproductive Right." For example, see R. M. Sharpe, "Hormones and Testis Development and the Possible Adverse Effects of Environmental Chemicals," Toxicology Letters 120 (2001): 221–32.

35. Although it is impossible to know reproductive patterns from human evolutionary history, we can compare average numbers of menstrual cycles in current hunter-gatherer societies with current industrial societies. Researchers have found that American women experience approximately three times as many menstrual periods as women in foraging societies. For example, women in hunter-gatherer societies are about 16 years old at menarche, 19.5 years old at first birth, nurse for 3 to 4 years, average 5.9 live births, and have an average age at menopause of 47 years, with an average of 160 ovulations in their lifetime. In contrast, American women average 12.5 years old at menarche, 24 years old at first birth, nurse for an average of 3 months, average 1.8 live births, and average 50.5 years old at menopause, with an average of approximately 450 ovulations within their lifetime; data from S. B. Eaton, M. C. Pike, R. V. Short, N. C. Lee, J. Trussell, R. A. Hatcher, J. W. Wood, C. M. Worthman, N. G. Blumenthal, M. J. Konner, K. R. Hill, R. Bailey, and A. M. Hurtado, "Women's Reproductive Cancers in Evolutionary Context," Quarterly Review of Biology 69 (1994): 535–67. See also B. Strassmann, "Menstrual Cycling and Breast Cancer: An Evolutionary Perspective," Journal of Women's Health 8 (1999): 193–202; and S. B. Eaton and S. B. Eaton III, "Breast Cancer in Evolutionary Context," in Evolutionary Medicine, ed. W. R. Trevathan, E. O. Smith, and J. J. McKenna (New York: Oxford University Press, 1999), 429–42. For a review of this controversial subject, see Rachel Bayer, "The Impact of Increased Menstruation Rates on Women's Health and Reproductive Cancers," (2001); online at http://webpub.alleg.edu/employee/3rmswme/ES101/ResearchPapers/RachelBayer.html.

36. Cadbury, Altering Eden, 82–83; Berkson, Hormone Deception, 55.


39. Berkson, Hormone Deception, 63.

40. Krimskey, Hormonal Chaos, 5, 9; Berkson, Hormone Deception, 65.

41. Krimskey, Hormonal Chaos, 9; Berkson, Hormone Deception, 63.

42. Berkson, Hormone Deception, 62–63.


44. Cadbury, Altering Eden, 48.


46. Editorial staff, "An Environment for Development," Environmental Health Perspectives 107 (September 1999). For recent research on second- and third-


51. Ibid., 15.
58. Personal communication, anonymous reviewer.

66. Myers, "Contamination threatens a basic reproductive right."

67. Editorial staff, "Forum."


70. Myers, \url{http://ourstolenfuture.org}. Frederick von Saal recently found that bisphenol-A had measurable effects in laboratory experiments at levels thousands of times lower than previously thought. These low-dose effects challenge the adequacy of countless toxicity tests undertaken to establish standards. For years, the acceptable daily dose for bisphenol-A had been set at a no-effect level of fifty milligrams/kg, or fifty parts per million, based on data reported by the Society of Plastics Industry. Vom Saal, however, found effects at lower levels: at two parts per billion, twenty-five thousand times lower. When he fed pregnant mice bisphenol-A at two parts per billion (two micrograms per kg/day), their male sons had enlarged and hypersensitized prostates when they reached adulthood. An overview of this work can be found at \url{http://ourstolenfuture.org/NewScience}. The original research is S. C. Nagel et al., "Relative Binding Affinity--Serum Modified Access (RBA-SMA) Assay Predicts the Relative In Vivo Bioactivity," 70-76.


71. Cadbury, Altering Eden, 144, 155.

72. \url{http://abcnews.go.com/sections/living/DailyNews/toxicpolish_dbp001128.html}.


77. This work was cited in Raloff, “Common pollutants undermine masculinity,” who reported on a series of papers given at the March 1999 meeting of the Society of Toxicology in New Orleans, including E. Mylchreest, and P. M. D. Foster, “Dose-Response for Altered Male Reproductive Development and Function Induced by D(n-butyl) Phthalate,” and M. Sar, E. Mylchreest, and P. M. D. Foster, “D(n-butyl) Phthalate Induces Changes in Morphology and Androgen Receptor Levels in the Fetal Testis.”
79. Ibid.
86. For fuller discussions of this point, see Deborah Blum, Sex on the Brain: The Biological Differences between Men and Women (New York: Viking, 1998), and Anne Fausto-Sterling, Myths of Gender: Biological Theories about Women and Men (New York: Basic Books, 1985).
89. Editorial staff, “Working for Women’s Health,” Environmental Health Perspectives 108 (January 2000); online at http://ehpnet1.niehs.nih.gov/docs/2000/108-1/niehshnews.html, citing Barbara J. Davis, head of the Female Reproductive Pathology Group and now acting chief of the newly created Laboratory of Women’s Health at the National Institute of Environmental Health Sciences (NIEHS).
90. Fisher, “Gender Matters.”
91. Berksen, Hormone Deception, 146.
95. Cited in Editorial staff, “Working for Women’s Health.”
96. Berksen, Hormone Deception, 148.
102. D. S. Ht. J. S. Bergerson, an ands on Uterine I for Uterine Fibroids 829–34. For the regarding U. P. Thorg ministration of E Clinical Oncology 1 citogenicity of Di clinical Oncology 126


106. Myers, Our Stolen Future: new research online at http://www. ourstolenfuture.org/New/newstuff.htm#. For a comparison between exposure through breast-feeding and long-term exposure through food, see S. Patandin, P. C. Dagnelie, P. G. H. Mulder, E. Op de Coul, J. E. van der Veen, N. Weisglas-Kuperus, and P. J. J. Bauer, "Dietary Exposure to Polychlorinated Biphenyls and
110. Ibid., 232.
112. Ibid., 15. This section of the NRC report is a fascinating exploration of how different groups and individuals construct knowledge.
114. Schettler, "Endocrine Disruptors."