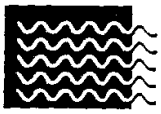
one



trace amounts

On a clear night after the harvest, central Illinois becomes a vast and splendid planetarium. This transformation amazed me as a child. In one of my earliest memories, I wake up in the back seat of the car on just such a night. When I look out the window, the black sky is so inseparable from the plowed, black earth—which dots are stars and which are farmhouse lights?—that it seems I am floating in a great, dark, glittering bowl.

Rural central Illinois still amazes me. Buried under the initial appearance of ordinariness are great mysteries. At least, I attempt to convince newcomers of that.

Were you to visit this countryside for the first time, its apparent flatness is probably what would impress you first—and indeed, for almost half the year, the landscape seems to consist of a simple plain of bare earth overlain by sky. But Illinois is not flat at all, I would insist, as I unfold geological survey maps that make visible the surprisingly contoured lay of the land. Parallel arcs of scalloped moraines slant across the state, each ridge representing the retreating edge of a glacier as it melted back into Lake Michigan and surrendered the tons of granulated rock and sand it had churned into itself.

Better than maps is a ground fog on a summer night when I drive you across these moraines and basins. Now you see how the shrouded bottomlands are distinguished from the uplands, the floodplains from the ridges, how the daytime perception of flatness belies a great depth. Out of

the car and walking, I encourage you to feel, as we traverse land that appears to be utterly level, the slight tautness in the thighs that comes with ascending a long grade versus the looseness in our feet that indicates descent.

Then there is the issue of water. Consider your own body, how the blood does not pulse through your tissues in great tidal surges—as was presumed before the English physician William Harvey discovered circulation in 1628—but instead flows within a diffuse net of permeable vessels. So too in Illinois, a capillary bed of creeks, streams, forks, and tributaries lies over the land. Your newly found skill of walking downhill will help you locate it.

And this is only the water that is visible. Under your feet lie pools of groundwater held in shallow aquifers—interbedded lenses of sand and gravel—and in the bedrock valleys of ancient rivers that lie below. One of these is the Mahomet, part of a river system that once ran west across Ohio, Indiana, and Illinois. Thousands of tons of debris, let loose by melting glaciers, completely buried the Mahomet River at the end of the last ice age. It now flows underground. In Mason County you can stand over a place where the Mahomet once joined the Illinois River. Here, in an area called the Havana Lowlands, the groundwater lies just below the earth's surface. In times of heavy rain, lakes brim up from under the earth and reclaim whole fields and neighborhoods.

In the eastern half of my county, Tazewell, the ancestral Mississippi River cut a valley 3 miles wide and 450 feet deep before glaciers exiled it to the western border of Illinois, its current channel. Buried by soil, clay, silt, and stones, the old Mississippi River valley is still down there, connected to the same ancient tributaries, its fractures and pores full of water. Islands still rise from the bedrock channel. If you could see through dirt, imagine the dramatic view you would have.

Of course, what you do see are corn and soybean fields. About 87 percent of Illinois is cropland, meaning that if you fell to earth in Illinois, chances are good you would land in a farm field. Illinois grows more soybeans and corn than any other state but Iowa. Read any supermarket la-

bel. Corn syrup, corn gluten, cornstarch, dextrose, soy oil, and soy proteins are found in almost every processed food from soft drinks to sliced bread to salad dressing. These are also the ingredients of the food we feed to the animals we eat. Thus, you could say that we are standing at the beginning of a human food chain. The molecules of water, earth, and air that rearrange themselves to form these beans and kernels are the molecules that eventually become the tissues of our own bodies. You have eaten food that was grown here. You *are* the food that is grown here. You are walking on familiar ground.

Illinois is called the Prairie State, but, to find prairie, you must really know where to look. Most of it vanished after John Deere invented the self-scouring steel plow in 1836. To be exact, 99.99 percent went under the plow. The .01 percent that escaped occupies odd and neglected places: along railroad tracks, encircling gravestones in old pioneer cemeteries, on hillsides too awkward to plow. Of the original 281,900 acres of tallgrass prairie in my home county, an official 4.7 fragmented acres remain (.0017 percent). I have never found them. Illinois conceals not only its topography but its ecological past as well, and even though I went on to become a plant ecologist, I have no real relationship to the native plants of my native state.

Truthfully, the closest I have felt to the prairie is when looking at plain, unadorned dirt. There are plenty of opportunities to do this in central Illinois—although the fields look less naked between October and April than they did when I was a child, thanks to low-till and no-till farming. These practices have largely replaced the habit of turning the field completely over after the harvest. The newer techniques leave on the surface a certain fraction of stalks, leaves, and stems to serve as a thin blanket against the wind. It's a tricky business: Too much residue leaves the soil compressed, without air, and unable to warm up in time for spring planting; water puddles on the surface. Too little residue, and the soil refuses to clump up at all, is prone to blow away or run with meltwater into the nearest creek bed.

Thus, each September at the Farm Progress Show, farm equipment representatives demonstrate all the latest technology for striking the perfect balance between these two states. Popular among farmers is the disc and chisel plow combination: parallel rows of slicing silver plates, like large pizza cutters, alternating with rows of beveled metal claws. These grids of discs and chisels are pulled, one by one, through an exhibition field as an announcer extols the virtues of each particular model. Observers, including me and my uncle, stand on either side of the tractor as it cuts a wide swath through corn stubble. We then step into the black wake and bend down to take a look. To assess a depth of penetration, we are encouraged to poke yardsticks into the chiseled furrows. We heft clumps of dirt in our hands to check diameter and ease of crumbliness. We then walk 10 yards over and form two lines on either side of the next tractor in the queue of tractors to cut a path through this field of stubble. We step in, bend down, heft clumps, stand up, walk over. And so on. It's a peculiar kind of country line dance. Each plowed strip is subtly different from the others.

There is no reason I should participate in this ritual except that my mother's family still farms the Illinois prairie and watching the earth being tilled offers me a connection to the past. Even though I live out of state now, it's important to me to maintain a relationship with both Illinoises—the present and familiar one as well as the Illinois that has vanished and is barely discernible. What remains of the 22 million acres of tallgrass prairie that once covered this state is the deep black soil that those grasses produced from layers of sterile rock, clay, and silt dumped here by wind and glaciers. The molecules of earth contained in each plowed clod are the same molecules that once formed roots and runners of countless species unfamiliar to me now. They died and became soil. This most obvious of realizations occurs to me every September as though for the first time. When I am touching Illinois soil I am touching prairie grass.

Illinois soil holds darker secrets as well. To the 87 percent of Illinois that is farmland, an estimated 54 million pounds of synthetic pesticides are

applied each year. Introduced into Illinois at the end of World War II, these chemical poisons quietly familiarized themselves with the landscape. In 1950, less than 10 percent of cornfields were chemically treated. Fifty-five years later, 98 percent were sprayed with pesticides. The most abundantly used is the weedkiller atrazine, which in 2005 was applied to 81 percent of Illinois cornfields—nearly 10 million acres of soil. With so much acreage now planted in field corn, fungus, which breeds on corn stubble, has emerged as a significant pest. The use of fungicides is now sharply up, refamiliarizing rural folk with an icon of the past: the low-flying crop duster droning above the fields in midsummer.

Pesticides do not always stay on the fields where they are sprayed. They evaporate and drift in air. They dissolve in water and flow downhill into streams and creeks. They bind to soil particles and rise into the air as dust. They migrate into glacial aquifers and thereby enter groundwater. They fall in the rain. They are found in snowflakes. And fog. And wind. And clouds. And backyard swimming pools. Little is known about how much goes where. By 1993, 91 percent of Illinois's rivers and streams showed pesticide contamination. Ten years later, the streams and rivers within my childhood watershed contained 31 different pesticides, and atrazine was in all samples. These chemicals travel in pulses: pesticide levels in surface water during the months of spring planting—April through June—are sevenfold those during winter and often contain levels of atrazine that exceed legal limits for drinking water. Even less is known about pesticides in groundwater. About 18 percent of all samples of groundwater surveyed in Illinois in 2006 contained atrazine byproducts, while a 1992 study found that one-quarter of private wells tested in central Illinois contained agricultural chemicals of some type. Drinking water wells in the Havana Lowlands region of Mason County showed some of the most severe contamination. A 2009 report identified two public drinking water systems in Illinois with running annual averages for atrazine in tap water that exceeded legal limits. In the same year, the wind blowing across my home county was so full of weedkiller that the air itself withered grape vines in a local vineyard.

Some of the pesticides inscribed into the Illinois landscape have been linked to cancer. One of these is DDT. Banned for use decades ago, DDT is

so chemically stable that it remains the most common pesticide in fish in North American rivers and streams. A 2009 national survey of pesticide residues in homes across the United States found traces of DDT on 42 percent of kitchen floors. Like islands in preglacial river valleys, its presence endures. DDT has been variously linked, in human studies, to low sperm count, premature birth, diabetes, brain damage, pancreatic cancer, impaired breastfeeding, and breast cancer. Some of the pesticides inscribed into the Illinois landscape are hormonally active—even at vanishingly small concentrations. One of these is atrazine, which has been variously linked, in animal studies, to increased estrogen production, birth defects, sexual ambiguity, disrupted ovulation, and altered breast development.

A lot goes on in the 13 percent of Illinois that is not farmland. In 2007, 1,102 different industries released more than 114 million pounds of toxic chemicals into air, water, and soil, making Illinois the nation's thirteenth biggest polluter. In the same year, 763 chemical spills occurred—more than two a day—making Illinois ninth among states in number of reported toxic accidents.

Like pesticides, industrial chemicals have filtered into the groundwater and surface waters of streams and rivers. Metal degreasers and dry-cleaning fluids are among the most common contaminants of glacial aquifers. Both have been linked to cancer in humans. At last count, 415 dry cleaners throughout Illinois have poisoned soil, and at least 30 represent a threat to groundwater. An assessment of the Illinois environment concluded that chemical contamination "has become increasingly dispersed and dilute," leaving residues that are "increasingly chemically exotic and whose health effects are not yet clearly understood."

I was born in 1959 and so share a birthdate with atrazine, which was first registered for market that year. In the same year DDT—dichloro diphenyl trichloroethane—reached its peak usage in the United States. The 1950s

were also banner years for the manufacture of PCBs—polychlorinated biphenyls—the oily fluids used in electrical transformers, pesticides, carbonless copy paper, and small electronic parts. DDT was outlawed the year I turned thirteen and PCBs a few years later. Both have been linked to cancer.

I am compelled to learn what I can about the chemicals that presided over the industrial and agricultural transformations into which I was born. Certainly, all of these substances have an ongoing biological presence in my life. Atrazine remains the most frequently detected pesticide in water throughout the United States, found in three of every four American streams and rivers and 40 percent of all groundwater samples. PCBs still lace the sediments of the river I grew up next to as well as the flesh of the fish that inhabit it. PCBs are why I'm unfamiliar with the taste of smallmouth bass and channel catfish. In fact, I have never eaten fish from my own river. State fish advisories warn women and children against doing so. DDT also continues to separate people from fish. The coastal waters of Palos Verdes, California became unfishable after 100 tons of DDT were drizzled into the sea between 1947 and 1971. The nine-mile stretch of ocean floor where the poison lies is considered one of the most hazardous places in the nation. The current plan for remediation is to cover it over with 18 inches of silt. Work is expected to begin in 2011, nearly four decades after DDT was banned.

I honestly have no memories of DDT. Instead, my images come from archival photographs and old film clips. In one shot, children splash in a swimming pool while DDT is sprayed above the water. In another, a picnicking family eats sandwiches, their heads engulfed in clouds of DDT fog. Old magazine ads are even more surreal: an aproned housewife in stiletto heels and a pith helmet aims a spray gun at two giant cockroaches standing on her kitchen counter. They raise their front legs in surrender. The caption reads, "Super Ammunition for the Continued Battle on the Home Front." DDT is a ruthless assassin. In another ad, the aproned woman appears in a chorus line of dancing farm animals who sing, "DDT is good for me!" DDT is a harmless pal.

During the 1940s and 1950s, this chemical of multiple personalities found its way into all kinds of civic campaigns and household products. One Illinois town not far from where I grew up conducted aerial fumigations of DDT in an attempt to control polio, mistakenly thought to be spread by flies. Meanwhile, a paint company advertised a formulation that could be brushed onto porches, window screens, and baseboards. When dry, DDT crystals would rise to the surface, forming "a lethal film." Perfect for summer cottages and trailers. Perhaps I spent childhood vacations in some of them. And perhaps, while there, I slept soundly between pesticide-impregnated blankets. In 1952, researchers proudly announced that woolens could now be mothproofed by adding DDT to the dry-cleaning process.

Fellow baby boomers just a few years older do not rely on old magazine ads to recall DDT. From memory, they can describe the fogging trucks that rolled through their suburban neighborhoods as part of mosquito, Dutch elm disease, or gypsy moth control programs. Some can even describe childhood games that involved chasing these trucks. "Whoever could stay in the fog the longest was the winner," remembers one friend. "You had to drop back when you got too dizzy. I was good at it. I was almost always the winner." Says another, "When the pesticide trucks used to come through our neighborhood, the guys would haul their hoses into our backyard and spray our apple trees. Mostly we kids would throw the apples at each other. Sometimes we would eat them."

Hazards that are universally common or repetitive assume "the harmless aspect of the familiar," observed the wildlife biologist Rachel Carson in her book *Silent Spring*, published when I was three years old. "It is not my contention that chemical insecticides never be used," Carson emphasized. "I do contend we have put poisonous and biologically potent chemicals indiscriminately into the hands of persons wholly ignorant of their potentials for harm. We have subjected enormous numbers of people to contact with these poisons, without their consent and often without their knowledge." She went on to predict that future generations would not condone this lack of prudent concern.

Reading *Silent Spring* as a member of this generation, across a distance of more than three decades, I gain another view of DDT. What impresses me most is just how much was known about the harmful aspects of this familiar and seemingly harmless substance. As Carson made clear, the scientific case against DDT—even by the late 1950s—was damning. It was not objective science, nor was it blissful ignorance, that created the impression that DDT was somehow both our most lethal weapon against undesirable life forms ("killer of killers," "the atomic bomb of the insect world") and a completely benign helpmate. In fact, scientific study after scientific study showed that DDT was failing at both roles. It triggered population explosions in insect pests who evolved resistance and whose natural enemies were killed by the spray. It poisoned birds and fish. It disrupted sex hormones in laboratory and domestic animals. It showed signs of contributing to cancer. By 1951, it had become a contaminant of human breast milk and was known to pass from mother to child.

Nevertheless, people continued using DDT until Carson's preliminary damning evidence was supplemented with more and more corroborating damning evidence, producing a great accumulation of damning evidence, and its registration was finally revoked in 1972. I find this phenomenon boundlessly fascinating. Across my desk are spread forty years of toxicological profiles, congressional testimonies, laboratory studies, field reports, and public health investigations of toxic chemicals both officially outlawed and officially permitted. Like crossing and recrossing the same field, I move back and forth between *Silent Spring* and the scientific literature that preceded it, between *Silent Spring* and the scientific literature published in the decades since. At what point does preliminary evidence of harm become definitive evidence of harm? When someone says, "We were not aware of the dangers of these chemicals back then," whom do they mean by *we*?

DDT, lindane, aldrin, dieldrin, chlordane, heptachlor. These names, unfamiliar to us now, are a roll call of the pesticides Rachel Carson featured in *Silent Spring*. All have links to cancer in at least some studies. All are now

prohibited or heavily restricted for domestic use. Lindane was banned for most uses in 1983 and banned entirely in 2006, although a controversial exemption allows its ongoing use as a treatment for lice and scabies. And yet a chemical company in my hometown released several pounds of lindane into the air in 1992 and dumped several more pounds into the sewer system. I know this because federal right-to-know laws make such events public information. Thus, lindane appears in the 1992 federal government's Toxics Release Inventory for Tazewell County. I was stunned to discover it there as I scanned the electronic list that documents emissions, dumpings, and transfers of toxic chemicals. Lindane has been associated in several studies with cancers of the lymph system.

Aldrin and dieldrin were banned in 1975, although aldrin was allowed as a termite poison until 1987. Aldrin converts to dieldrin in soil and inside our tissues. Dieldrin suppresses the immune system and produces abnormal brain waves in mammals. As late as 1986, dieldrin was still turning up in milk supplies because the soils of hayfields sprayed more than a decade earlier remained contaminated. Most agricultural uses of chlordane in the United States were ended in 1980 and heptachlor in 1983. Both have been linked to leukemia and certain childhood cancers.

For those of us born in the 1940s, 1950s, and 1960s, the time between the widespread dissemination of these pesticides and their subsequent prohibition represent our prenatal periods, infancies, childhoods, and teenage years. We were certainly the first generation to eat synthetic pesticides in our pureed vegetables. By 1950, residue-free produce was so scarce that the Beech-Nut Packing Company began allowing detectable levels of residue in baby food.

At what point does preliminary evidence of harm become definitive evidence of harm? When someone says, "We were not aware of the dangers of these chemicals back then," who is the *we*?

With a focus on breast cancer, let's look at the evidence of harm for three chemicals: DDT, PCBs, and atrazine.

In 1976, four years after DDT was banned, researchers reported that women with breast cancer had significantly higher levels of DDE (dichloro diphenyl dichloroethylene) and PCBs in their tumors than in the surrounding healthy tissues of their breasts. (DDT is metabolized in the human body into DDE, a chemical that acts like estrogen.) The study was small but the finding provocative because DDT and PCBs were already linked to breast cancer in rodents.

Other studies followed. Some showed an association between breast cancer and residues of pesticides or PCBs. Some did not. In 1993—seventeen years after the first study—the biochemist Mary Wolff and her colleagues conducted the first carefully designed, major study on this issue. They analyzed DDE and PCB levels in the stored blood specimens of 14,290 New York City women who had attended a mammography screening clinic. On average, they reported, the blood of breast cancer patients contained 35 percent more DDE than that of healthy women, but PCB levels were only slightly higher. The most stunning discovery was that the women with the highest DDE levels in their blood were four times more likely to have breast cancer than the women with the lowest levels. The authors concluded that residues of DDE "are strongly associated with breast cancer risk."

By now, breast cancer activists were paying attention. Throughout the 1990s, as breast cancer rates continued rising, they urged scientists to direct more research dollars down lines of inquiry that would reveal, once and for all, whether exposure to pesticides and industrial chemicals was contributing to breast cancer. They pointed out that pesticide use in the United States had doubled since Rachel Carson wrote *Silent Spring* and that women born in the United States between 1947 and 1958 had almost three times the rate of breast cancer that their great-grandmothers had when they were the same age. Women cancer activists marched in the streets carrying signs proclaiming, "Rachel Carson was right!" Taking a

page from the playbook of AIDS activists, these women demanded a seat at the table where research proposals were reviewed and funding decisions made.

Studies were funded and papers published. Yet the results were maddeningly inconsistent. For every finding of a positive association, another showed no association or yielded a complicated picture. One study found that African American women with breast cancer showed more past exposure to PCBs than their counterparts without breast cancer. Mysteriously, however, the trend for white women went in the opposite direction: the highest levels of blood PCBs tended to occur in women *without* the disease. The largest and best-designed investigation within this suite of studies, published in the *New England Journal of Medicine* in 1997, found no association at all between risk of breast cancer and blood levels of PCBs and DDT. Interpretation of these contradictory results sparked considerable debate, but the majority opinion within the scientific community was that women with breast cancer, as a group, do not appear to have higher body burdens of DDE and PCBs than women without breast cancer.

Some researchers found these results reassuring. Others worried that these studies had not considered the underlying genetic differences among women nor taken into account the timing of exposure. What if, they asked, some genetic subgroups were more susceptible to environmentally induced breast cancers than others? Furthermore, most studies had measured levels of DDE or PCBs in adult women—after much of their residues had been eliminated from the body and the chemicals themselves long banned. What if contemporary measures do not accurately reflect historical exposures? What if the important variable is DDT exposure during childhood or adolescence—when the developing breast is most vulnerable? Animal studies clearly demonstrate the importance of toxic exposures that occur in early life when the breast is most sensitive to damage.

The ideal study would be designed like this: go back in time to a year of peak DDT usage—say, 1963—gather blood from U.S. girls, and then

follow them through their adult lives to see if those exposed to the highest levels of DDT as children went on to suffer higher rates of breast cancer as adults.

And then someone did just that. More or less. As described in a paper published in 2007, Barbara Cohn and her colleagues at the University of California unearthed medical records and banked blood samples of women who had visited a clinic between 1959 and 1967 to seek routine prenatal care. Knowing that DDT came on the market in 1945, Cohn was able to calculate how old each woman was when she was first exposed to DDT. And she was also able to trace these women and learn their current breast cancer status. The results were clarifying: Women exposed to DDT after age 14—those born in 1931 or before—showed no association between exposure to DDT and breast cancer. But among women exposed to DDT when they were younger than 14, a significant relationship existed: women with high DDT levels were five times more likely to be diagnosed with breast cancer by age 50 than those with the lowest levels. In other words, this study showed a fivefold increase in breast cancer risk among women who had experienced high exposures to DDT before puberty but not in women so exposed after their breasts had already developed. Thanks to hundreds of test tubes that stood silently in the back of a freezer in Oakland, California for a half century, and thanks to breast cancer activists who insisted that environment studies go forward, we now know that DDT exposure in childhood can significantly increase breast cancer risk in adulthood. And we gained this knowledge nearly forty years after DDT was banned.

Meanwhile, other researchers went to work categorizing women genetically. They looked closely at women who had inherited a variation of CYP1A1, a gene that is involved in metabolizing hormones and that is known to be influenced by PCB exposure. About 10–15 percent of white women in the United States are thought have the variant gene. The proportion of black women who have it is not yet known. When data on women with the variant gene were examined in isolation, a picture began to emerge: women who possessed both the genetic variation as well

as a high PCB body burden had an elevated rate of breast cancer. Indeed, their rate of breast cancer was two to three times higher than that of women with lower levels and without this genetic trait. The evidence to date now supports an association between breast cancer and PCB exposure for subpopulations of women who have inherited this particular genetic variation. And we gained this knowledge nearly thirty years after PCBs were banned.

The story of atrazine today is much like the story of DDT and PCBs as it was told decades ago. Worrisome findings followed by equivocal ones. Inconsistencies. Contradictions. Balls of confusion. The difference is that atrazine is not banned. It is the second most abundantly used pesticide in the United States, and its manufacturer plays an aggressive role in defending its product. A proven endocrine disruptor, atrazine causes breast cancer in one strain of rat. Some argue that it does so by a mechanism not relevant to humans. The human studies themselves are inconclusive and, while a few show possible associations, most do not report a link between adult exposure to atrazine and breast cancer. However, no human studies have looked at early-life exposures to atrazine, which is when atrazine exerts its strongest effects in lab animals. A 2009 study called for an investigation into how atrazine might be affecting the pace and tempo of sexual maturation in girls. (Early puberty is, by itself, a known risk factor for breast cancer.) Other human studies have found suggestive evidence for an association between atrazine exposures and several other cancers, including lymphoma and cancer of the prostate, ovary, testes, and brain. There is also suggestive evidence for unique toxicities arising from mixtures of atrazine with other farm chemicals. Laboratory studies report possible synergistic effects: among invertebrate animals, atrazine induces an enzyme that makes a second pesticide, chlorpyrifos, more toxic. In this way, exposure to one contaminant can turn another into a more powerful poison. Are these results applicable to humans? It's not yet clear.

By 1994, the evidence against atrazine was troubling enough that the U.S. Environmental Protection Agency (EPA) initiated a special review

of its registration. Nine years passed. Meanwhile, across the Atlantic, regulators in Europe announced the results of their own review: atrazine was banned throughout the European Union. Finally, in 2003, the EPA announced its decision: continued use of atrazine was approved. This was an intensely controversial decision. One researcher pointed out in disgust that DDT was abolished on the basis of less evidence than we now had for atrazine.

In October 2009, the EPA announced a plan—and a timetable—for a new evaluation of atrazine.

Ten thousand years of tallgrass prairie have left a fainter trace on the place I call home than twenty-seven years of DDT, forty-six years of PCBs, and fifty years of atrazine. Because it is my home, I am driven to pursue the question of the past and ongoing contamination of Illinois and its possible link to the increasing frequency of cancer there. I believe that all of us, wherever our roots, need to examine this relationship. And I think it reasonable to ask—nearly a half century after *Silent Spring* alerted us to a possible problem—why so much silence still surrounds questions about cancer's connection to the environment and why so much scientific inquiry into this issue is still considered "preliminary."

From dry-cleaning fluids to pesticides, harmful substances have trespassed into the landscape and have also woven themselves, in trace amounts, into the fibers of our bodies. This much we know with certainty. It is not only reasonable but essential that we should understand the lifetime effects of these incremental accumulations.