LIVING DOWNSTREAM

an ecologist
looks at cancer
and the environment

SANDRA STEINGRABER

ronmental Health Sciences; the Agency for Toxic Substances and Disease Registry; the National Program of Cancer Registries; the National Center for Health Statistics; the Illinois Geological, Water, and Natural History Surveys; the Illinois Department of Conservation; the Illinois and Massachusetts Departments of Public Health; and the Mason County Health Department. Once again, responsibility for the text is solely mine. The conclusions and recommendations expressed in this book may not be shared by those who assisted me in the research process nor by the agencies they represent.

All of us are indebted to those working, on local as well as national levels, for cancer prevention and environmental protection. I owe specific thanks to members of the Women's Community Cancer Project in Cambridge, Massachusetts; Nancy Evans of Breast Cancer Action; Cathie Ragovin, M.D., of the Massachusetts Breast Cancer Coalition; Barbara Balaban, Geri Barish, and Joan Swirsky of Long Island; Sandra Marquardt of Mothers and Others for a Liveable Planet; the staffs of Pesticide Action Network and the Northwest Coalition for Alternatives to Pesticides; Mary Taylor of Friends of the Earth, UK; Dr. Paul Johnston at the University of Exeter; the staff of the Women's Environmental Network, London; and the citizens and scientists who serve with me in Washington on the National Action Plan on Breast Cancer.

Closer to home, I received assistance from Kevin Caveny, district supervisor of my hometown water utility; Elaine Hopkins of the *Peoria Journal Star*; state representative Ricca Slone; and Dr. Earl and Marge Melchers of Pekin, Illinois. My parents, Wilbur and Kathryn Steingraber, and sister, Julie Skocaj, read drafts, checked facts, and freely offered the authority of their memories and experience.

No writer could ask for a more steadfast pair of advocates than I have found in my literary agent, Charlotte Sheedy, and my editor, Merloyd Lawrence. Both have been unflagging in their enthusiasm and support—even in the face of tribulations and unforeseen delays on my part. Dropping a finished chapter through Merloyd's Beacon Hill mail slot was the most satisfying of gestures. For her patience, caring, and sharp editorial judgment, I am forever grateful.

Finally, I express my abiding appreciation to Bernice Bammann, [illegible] writers Valerie Cornell and Karen Lee Osborne

PROLOGUE TO THE BRITISH EDITION

The seeds of this book were sown ten years ago in east Africa. It was here, while researching the ecological consequences of war and famine, that I first began to think about environmental destruction as an issue of human rights. For several months in 1986 and again in 1987, I interviewed refugees as they fled the civil war in Ethiopia and streamed across the Sudanese border. One afternoon, I spoke at length with a refugee farmer who had witnessed an entire hillside wash into a river after the army built a military road along its banks. At the end of the interview, my respondent, weary of my detailed questions, turned the tables on me. "Now tell me," he demanded, "about the rivers of your homeland."

I was happy enough to oblige. My father built our house on the east bluff of the Illinois River in Tazewell County. Looking out over that river valley while riding my tricycle is a beloved childhood memory. Indeed, it remains my most beloved landscape. All this I told him.

The farmer nodded and pursued me further. "Tell me now about the fish in your American river. How do they taste to you?"

This question I could not answer. For all the years I lived by the Illinois River—and I was born in 1959—its fish were contaminated with cancer-causing pesticides and industrial chemicals to the extent

that government advisories warned us against eating the most common species of sport fish. These warnings were (and remain) especially strict for children and women of reproductive age. I turned my thoughts to other rivers I have lived beside—the Huron, the Chicago, and now the Charles River in Boston—and realized that they, too, carry fish advisories. I, who have lived my whole life as a child or as a woman of reproductive age, had never tasted fish from the rivers I have lived by and loved.

The response of my refugee friend was one of bewildered distress. Not allowed to eat the fish from my own river? A clear violation of human rights! "We have already organized against the men who are poisoning our river. You must go home at once and drive away the ones who are poisoning your river!"

I did not tell him that I myself had become a cancer patient at the age of 20.

I did eventually go home, and I discovered that federal right-to-know laws in the United States now make information about the contamination of my environment publicly available. This knowledge allowed me, in ways not even possible a decade ago, to explore the extent to which toxic chemicals, including cancer-causing agents, have trespassed in the air, food, water, and soil of my hometown community. This newly acknowledged right to know offered me a chance to ask what connections might exist between these encroachments and the health problems of Tazewell County, Illinois. At the same time, cancer registries, also a recent phenomenon in the United States, provided me with a view of cancer's trajectory through time and a map of its distribution across space. I attempt here to bring these two categories of information together—data on environmental contamination and data on cancer incidence—to see what patterns might exist, to identify questions for further inquiry, and to urge precautionary action, even in the face of incomplete answers. Thus, I turned my attention from famine to cancer.

What has emerged for me personally from all this study—and this book does include the deeply personal—is an appreciation for just how ordinary my Illinois home is. As in many other commu-

quences. In this, the story of central Illinois is utterly unexceptional. It receives my scientific attention not because its history is so unusual but because it is so typical. It receives my devotional attention because central Illinois is the source of my ecological roots, and my search for these roots is part of the story.

Various published studies, gathered from far-flung corners of the biological literature, offer other glimpses of the connection between cancer and the environment. Woven into my discussion throughout this book, these range from reports on pesticides, river sediments, and trash incinerators to surveys of farmers, sport anglers, and nursing mothers. They include investigations of animals (wildlife, pets, livestock, and laboratory rats), as well as examinations of human tissues and cellular machinery (breasts, blood, hormone receptors, liver enzymes, and carcinogen-metabolizing genes). Few long-term, comprehensive studies on the environmental links to human cancers have been conducted—and I leave it to readers to judge the reasons for this neglect. However, the many small-scale, underfunded, and sometimes preliminary investigations that *do* exist create a startling picture when viewed together.

In this careful marshalling of evidence, I discovered, the studies from Britain sometimes far exceed those conducted in the United States—and sometimes lag behind.

The chapter called "Silence" describes my friendship with the young American cancer activist Jeannie Marshall. In the pile of papers Jeannie bequeathed to me at the time of her death were articles about the children of Seascale in northwest England. Jeannie had a particular interest in the now famous childhood leukemia cluster documented there because the town in which she grew up, Scituate, Massachusetts, is also part of a leukemia cluster. This fact corroborates evidence of a plausible link between radioactive emissions and cancer rates in the village of Seascale. Like Seascale, Scituate is a coastal town located near a nuclear facility. Like Seascale, this facility released pulses of radioactivity in a series of accidents years before. As with Seascale, scientists have not yet identified the exact routes of exposure (breathing? eating seafood? drinking water? playing on beaches?

Indeed, the children of Seascale are among the most intensely studied cancer patients in the world. The story began in 1983 when a Yorkshire television journalist investigated a rumor that high numbers of children were succumbing to leukemia in an obscure west Cumbrian village located a few kilometers from the Sellafield nuclear site. Beginning its life in the Cold War, Sellafield first served as a plutonium production plant in 1947. It later stored and reprocessed spent fuel from domestic nuclear power plants. It is known to have routinely spilled radioactive liquids into the Irish Sea. The cluster of childhood leukemias near Sellafield that was first documented on television was subsequently confirmed by scientists. An official public health inquiry showed that Seascale's children were ten times more likely to develop leukemia than children living elsewhere in England and three times more likely to develop other kinds of cancers.

This startling discovery unleashed an avalanche of further research and debate. Later studies identified substantial undocumented releases from Sellafield during the 1950s and uncovered childhood cancer clusters around other nuclear sites throughout the United Kingdom, including Scotland. Across the Atlantic, the situation in Seascale sparked renewed interest in investigating the adult cancers clustering around several small towns near the Pilgrim nuclear plant in southeastern Massachusetts. And when one of the natives of this community—who happened to be my dearest friend—lay dying of her disease, the message of Seascale's children became part of our last conversations together and thereby entwined into my life.

The chapter called "Time" further describes these events as well as exploring the evidence now emerging of cancer's rising incidence rates throughout the nations of the industrialized world. This evidence comes from cancer registries, and, here again, Britain leads the United States in knowledge. Relying on a patchwork of state-based registries, the U.S. has no comprehensive national cancer registry and instead extrapolates cancer rates from pooled data collected from less than 20 percent of the total population. This complicated effort was only begun in 1973, giving Americans a 20-year estimated picture of cancer time trends. Thus, when the *New York Times* recently announced an apparent rise in cancer incidence among American children that may be linked to rising rates of

chemical exposure, its analysis was hampered by a lack of complete cancer reporting. This realization has led to calls for a national registry of children's cancer—a project that will, if initiated, take years to bear fruit. England and Wales, on the other hand, established a national registry in 1945. By 1950, 72 centers in the country contributed data, and by 1962, full geographic coverage was achieved. This allows Britons (as well as New Zealanders, whose cancer registration has similar origins) a 30-year view of cancer's casualties. Moreover, cancer mortality data—gleaned from death certificates—have been collected and analyzed for more than a century in England and Wales. These trends show disquieting increases in deaths from cancers of the lung, pancreas, ovary, and breast among British citizens—even after adjusting the data for aging. Between 1968 and 1988 alone, brain cancers among all elderly Britons doubled—and tripled among elderly men. Deaths from multiple myeloma, a particularly painful cancer of the bones, also increased strikingly during this same time period—and older British males have been particularly hard hit.

In the chapter called "Space," I look at the tendency of cancer to concentrate in certain geographic regions. Once again, some of the most well-documented studies come from England, which has led the way in cancer mapping techniques in small areas. A 1997 study by the esteemed British cancer researcher E.G. Knox provides the most comprehensive picture yet of the close association between childhood leukemias and local environmental hazards. Dr. Knox mapped the home residencies of all 22,458 children dying from leukemias and other cancers in England, Wales, and Scotland between 1953 and 1980. Using atlases and business directories, he and his colleague, E.A. Gilman, also mapped the locations of all potentially hazardous sites—ranging from power plants to neighborhood auto body repair shops. Their findings reveal that children face an increased risk of a cancer diagnosis if they live within a few kilometers of certain kinds of industries—especially those involving large-scale use of petroleum or chemical solvents at high temperatures. These include oil refineries, air fields, paint makers, and foundries. The danger is greatest within a few hundred meters and tapers off with distance. Among children who had moved within their lifetimes, the relationship was stronger for their birth address than it

was for their address at the time of their death. This result, as Knox and Gilman point out, strongly suggests that very early—probably prenatal—exposures to environmental carcinogens create the threat of cancer in children.

Meanwhile, back in Massachusetts, renewed attention is being paid to the soon-to-be-famous cluster of childhood cancers in the manufacturing town of Woburn. Rivaling anything in Dickens' *Bleak House*, the legal intrigue enveloping these twenty-two child cancer victims, their parents, and their lawyers has even become the subject of both a non-fiction thriller and forthcoming Hollywood movie. Outside of all the courtroom drama, a new epidemiologic study has traced the cancer rate of Woburn's children back to solvent-contaminated drinking water consumed by their mothers *while they were pregnant*: the more contaminated the water coming into the home during pregnancy, the higher the subsequent risk of cancer in the child. Taken together, the British and the American studies both point to the exquisite vulnerability of the fetus to cancer-causing chemicals. And yet, our respective governments have set standards of exposure to environmental carcinogens with adults, not embryos, in mind.

Infant or adult, documenting our exposure to such chemicals requires a comprehensive inventory of toxic chemical releases, both routine and accidental. On this matter—which is taken up in close detail in the chapter called "War"—Great Britain trails behind the United States. Indeed, Knox and Gilman were hampered in their investigation of British childhood cancers because they lacked true measures of child exposures. While toxic release reports from industry do not provide such measures, they do provide estimates and grounds to launch further inquiry. In the United States, the Toxics Release Inventory, born in 1987, is the centerpiece of our community right-to-know laws. By contrast, its analogue in England and Wales, the Chemical Release Inventory, exists outside a human rights framework. Initiated in 1992, the British CRI is tied to the system that monitors compliance with operating permits. No fixed list of substances is reported; one incinerator may be required to disclose releases of 40 different substances, for example, while another must only report on four. These discrepancies make comparisons difficult work at best. Moreover, CRI does not identify industries or even

many chemicals by name nor does it always detail the nature of the release (rivers and sewers are both considered "water" releases, for example). These limitations frustrate the work of science.

In the latter chapters of the book I take a close look at our ecological surroundings, element by element. "Earth" investigates pesticides in food and agriculture. In the United Kingdom, pesticides are regulated under the Food and Environment Protection Act of 1985. As in the United States, these statutes are swamped by the problem of re-registering hundreds of old pesticides that received their legal clearance before any advance testing for their cancer-causing potential was required. In both countries, the pesticide re-registration process, underfunded and understaffed, grinds slowly on. In the meantime, untested substances are essentially free on bail, innocent until proven guilty.

In "Air," I consider not only the contaminants we inspire into our bodies through breathing but also the role the wind plays in conducting cancer-causing substances from industrial sites and farm fields, to, say, a lake bottom thousands of miles away. One of the most compelling examples of long-distance transport of carcinogens comes from Esthwaite Waters, a lake in rural, northern England whose sediments are contaminated with PCBs. Chemical dating reveals a broad range of PCB deposition dating back to 1929—long before the United Kingdom began manufacturing these now banned electrical fluids. The presence of PCBs in seventy-year-old sediments of Esthwaite Waters indicates airborne transport from either the European continent or the United States, where manufacture was already well underway by the 1920s.

In "Water," I trace the pathways of such lakes, rivers, and streams, as well as the hidden journeys of the groundwater that feeds them and fills the wells from which we drink.

In "Fire," I examine the misbegotten birth of one very potent and elusive carcinogen now believed to inhabit the tissues of every living person: dioxin. Much of its creation can be traced back to the stacks of garbage and medical waste incinerators where molecules of carbon and chlorine rearrange themselves to form this most wicked of organic chemicals. However, new data released in 1997 by the UK Environment Agency reveals that pesticide production is also a major

contributor to dioxin releases in England. And thus we move from fire back to earth again.

There are individuals who claim, as a form of dismissal, that links between cancer and environmental contamination are unproven and unprovable. There are others who believe that placing people in harm's way is wrong—whether the exact mechanisms by which this harm is inflicted can be precisely deciphered or not. At the very least, they argue, we are obliged to investigate, however imperfect our scientific tools.

Happily, the latter perspective is gaining esteem as many leading cancer researchers acknowledge the need for an "upstream" focus. As explained at an international conference at Leeds University in England, this image comes from a fable about a village along a river. The residents who live here, according to parable, began noticing increasing numbers of drowning people caught in the river's swift current and so went to work inventing ever more elaborate technologies to resuscitate them. So preoccupied were these heroic villagers with rescue and treatment that they never thought to look upstream to see who was pushing the victims in.

This book is a walk up that river.

Sandra Steingraber, 1997

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 three 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 89 percent of Illinois is cropland, meaning that if you fell to earth in Illinois, nine times out of ten you would land in a farm field. Illinois grows more soybeans than any other state in North America, and it produces more corn than any state but Iowa. Read any supermarket label. 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 you must really know where to look to find prairie. 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 (equals .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 the switch 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 is 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 in recent years has been 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 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 ten 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 is 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 in New England now, it is 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 twenty-two million acres of tallgrass prairie that once covered this state is the deep black dirt 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 the 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 89 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 sprayed with pesticides. In 1993, 99 percent were chemically treated.

Pesticides do not always stay on the fields where they are sprayed. They evaporate and drift in the jetstream. 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 buried river valleys and thereby enter groundwater. They fall in the rain. They are detectable in fog. Little is known about how much goes where. In 1993, 91 percent of Illinois's rivers and streams showed pesticide contamination. These chemicals travel in pulses: pesticide levels in surface water during the months of spring planting—April through June—are sevenfold those during winter, although detections never fall to zero. Even less is known about pesticides in groundwater. A recent pilot study found that one-quarter of private wells tested in central Illinois contained agricultural chemicals. Those sampled in the Havana Lowlands region of Mason County showed some of the most severe contamination.

Some of the pesticides inscribed into the Illinois landscape promote cancer in laboratory animals. Some, including one of the most commonly used pesticides, atrazine, are suspected of causing breast and ovarian cancer in humans. Other probable carcinogens, such as DDT and chlordane, were banned for use years ago, but like the islands in preglacial river valleys, their presence endures.

A lot goes on in the 11 percent of Illinois that is not farmland. Approximately fifteen hundred hazardous waste sites are in need of remediation—a list that does not include several thousand pits, ponds, and lagoons containing liquid industrial waste. And each year Illinois injects some 250 million gallons of industrial waste—which, until recently, included pesticides—through five deep wells that penetrate into bedrock caverns. These geological formations are overlain by aquifers and farmland. Illinois exports hazardous waste but also im-

ports it—almost 400,000 tons in 1992—from every state except Hawaii and Nevada. In this same year, Illinois industries legally released more than 100 million pounds of toxic chemicals into the environment.

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. A recent assessment of the Illinois environment concluded that chemical contamination "has become increasingly dispersed and dilute (and thus less visible)," 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 among the most common contaminants of midwestern drinking water, and all of us in the United States carry detectable levels of DDT and PCBs in our tissues. PCBs lace the sediments of the river I grew up next to as well as the flesh of the fish that inhabit it. DDT can remain in soil for several decades.

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 '50s, 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*?

However banished, DDT has an ongoing presence in our lives through several routes. Its persistence in soil means that some food crops continue to bear DDT residues. Migratory songbirds carry DDT molecules in their flesh, as do many freshwater fish. DDT is a common ingredient of hazardous waste sites. It has been detected in carpet dust. Global air currents carry DDT into the North American continent from countries where its use is still permitted. And DDT periodically wells up from the deep basins of the Great Lakes.

Moreover, even after its ban, DDT has continued to be shipped abroad. Laws banning the use of particular pesticides in this country do not prohibit their export. U.S. Customs records from 1992 reveal that several million pounds of unregistered, canceled, or suspended pesticides were loaded on ships and exported from the United States that year. As of 1994, nine tons per day of domestically banned pesticides left U.S. shores for foreign lands.

Lindane, chlordane, dieldrin, aldrin, heptachlor. These names, unfamiliar to us now, are a roll call of the other pesticides Rachel Carson featured in *Silent Spring*. All are now classified as known, probable, or possible carcinogens. All are now prohibited or heavily restricted for domestic use. Many are still manufactured and exported. A chemical company in my hometown, for example, 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 now 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 long computer scroll that documents industry's emissions, dumpings, and transfers of toxic chemicals. Lindane was banned for most uses in 1983, although it is still allowed in lice shampoos for humans and flea dips for dogs. Clearly, I have a more intimate relationship with lindane than I realized.

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, '50s, and '60s, the time between the widespread dissemination of these pesticides and their subsequent prohibition represents 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.

Banned pesticides, like fugitives from justice, have not entirely disappeared. We have forgotten about them, but they are still among us. They frequent foreign ports. They languish underground. But they are beginning to surface again in the tissues of women with breast cancer, sometimes under different names—DDT is metabolized in the human body into other chemicals, including one called DDE—and sometimes along with banned industrial chemicals belonging to the same chemical clan.

Four years after DDT was banned, researchers reported that women with breast cancer had significantly higher levels of DDE and PCBs in their tumors than in the surrounding healthy tissues of their breasts. Similar but weaker trends held for lindane, heptachlor, and dieldrin. The study was small—involving only fourteen women—but the findings provocative, because DDT and PCBs were already linked to breast cancer in rodents.

Other small studies followed. Some showed an association between breast cancer and residues of pesticides or PCBs; some did not. In 1990, Finnish researchers reported that women with breast cancer had higher concentrations of a lindanelike residue in their breasts than women without breast cancer. Indeed, women whose breasts sequestered the highest levels were ten times more likely to have breast

cancer than women with lower levels. Moreover, the pooled blood from women with breast cancer contained 50 percent more of this pesticide residue than the blood from women without breast cancer. Similarly, in 1992, a study of forty Connecticut women revealed that levels of PCB, DDE, and DDT in the breasts of women with breast cancer were 50 to 60 percent higher than in women who did not have breast cancer.

In 1993—seventeen years after the first pilot 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. Within six months, fifty-eight of these women were diagnosed with breast cancer. Wolff matched each of these fifty-eight women to control subjects—women without cancer but of the same age, same menstrual status, and so on—who had also visited the clinic. The blood samples of the women with breast cancer were then compared to their cancer-free counterparts.

On average, the blood of breast cancer patients contained 35 percent more DDE than that of healthy women. (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."

On the heels of the Wolff study came another by the Canadian researcher Éric Dewailly and his colleagues in Québec. Dewailly obtained breast tissue from women who had undergone biopsies for breast lumps. He chose twenty women whose lumps turned out to be cancerous and seventeen women whose lumps were benign. The removed lumps were then analyzed for chemical residues. Consistent with the findings of previous studies, the concentrations of several pesticides and industrial chemicals were moderately higher in the tissues of women with cancer than women without. When Dewailly restricted his comparison to estrogen-receptor positive tumors (that is, tumors sensitive to the presence of estrogen), the difference became more striking: DDE levels were substantially higher in women with

estrogen-receptor positive cancers than in the women of the control group.

Following Wolff's and Dewailly's work came the Krieger study, which yielded a more complicated picture. The Harvard epidemiologist Nancy Krieger, then at the Kaiser Foundation in Oakland, California, examined DDE and PCB levels in blood drawn from women in the 1960s and then frozen and stored for nearly thirty years. She compared the blood from 150 women who went on to get breast cancer sometime during those intervening three decades to blood from 150 women who remained cancer free. The central question: Can exposure to DDT and PCBs many years ago predict whether a woman will contract breast cancer? Previous studies looked at DDE and PCB levels at the time of diagnosis. Hers would be the first study to take into account the lag time between exposure and onset of disease. Three racial/ethnic groups were represented—African Americans, Asian Americans, and whites. When the three groups were combined, no significant differences were found. However, when each racial group was considered separately, the results changed. Whites and especially African American women with breast cancer had significantly higher levels of DDE than women without breast cancer, even as Asian American women continued to reflect the overall pattern of no difference. More mysteriously, while African American women with breast cancer showed more past exposure to PCBs than their counterparts without breast cancer, 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 interpretation of these results—which are not inconsistent with earlier studies but which do not actually confirm them either—has sparked considerable debate. Do DDE and PCB levels in blood serum accurately mirror their levels in women's breasts? (Evidence from other studies indicates they do.) Do we know whether DDE and PCB molecules remain stable when stored for thirty years? (Persistence is certainly a well-known trait of both chemicals.) What about the red rubber tops that capped the test tubes? Could chemical contaminants have migrated into the blood and marred the chemical analysis? (A speculative concern.)

For thirty years, three hundred stoppered test tubes stood at attention in the back of a freezer, waiting to be rediscovered. Red blood. Red caps. Some of the women whose blood lay frozen in those tubes are dead of breast cancer, others have died from other causes, and others are no doubt still alive. Probably no one now living remembers those particular blood draws, but three decades later, our understanding about breast cancer and environmental contamination has become linked to the contents of these red-capped tubes.

Perhaps the image would seem less urgent if women born in the United States between 1947 and 1958 did not now have almost three times the rates of breast cancer than their great-grandmothers did when they were the same age. Or if pesticide use in the United States had not doubled since Rachel Carson wrote *Silent Spring*. But we do. And it has.

Ten thousand years of tallgrass prairie have left a fainter trace on the place I call home than twenty-seven years of DDT spraying. 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—more than three decades 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 DDT, 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.

TWO

silence

The very modern Beinecke Library at Yale University is the resting place for Rachel Carson's papers. The cool, gray archival boxes that contain her correspondence, lecture notes, and personal writings must be requested one at a time from the librarian's assistant. The special room for viewing them is hushed and spacious. A wall of windows looks out over a green collegiate lawn. One enters after a ritual of giving over all personal possessions to the librarian. No ink is allowed in the viewing room—only pencils or laptop computers.

Alone in this room with the first box, I sift slowly through the pages it holds as though I were sorting botanical specimens. It is an automatic reflex, although I have not worked in a botanical herbarium for years. Herbarium sheets, onto which the delicate skeletons of dried plants are pressed, must never be flipped over like pages in a

book but rather are to be laid gently in reverse order to the left of the stack one is looking through. When finished, the examiner places the sheaves, one at a time, on top of the stack to the right, and they thus assume their original position. At least, this is the method I was taught. Something about the ceremony of my current task has triggered this old behavior. I can only hope it approximates correct archival technique.

The sight of Rachel Carson's handwriting is exhilarating. I uncover a note to Carson from Jacqueline Kennedy. Deep in another file is a letter of complaint Carson sent to a music company after receiving an erroneous bill and an inferior record album. The extraordinary and the mundane lie together here.

I have come to eavesdrop, looking for no specific document but with a desire to listen to the voices behind *Silent Spring*. And while I do overhear some things, what I end up thinking about is silence.

In a nation where guarantees of free speech are carved into the heart of our legal system, we are very often baffled by those who claim they have been silenced. I myself have never feared my mail would arrive with passages blacked out by a censor's invisible hand. I have never wondered if the police would stop me on the way to class to announce that the content of my lecture was unacceptable. And yet perhaps we have all witnessed certain subtle codes of silence in operation—an unspoken agreement in the workplace or a family secret that everyone knows but does not discuss.

Rachel Carson was interested in three forms of silence. As a government scientist—she rose through the ranks of the U.S. Fish and Wildlife Service—Carson became concerned that the noise of important ecological debates carried on within federal agencies seldom reached the public. The long-running quarrel over the claim that pesticides were harmless was one she followed most closely. By virtue of her position, she had access to field reports clearly indicating that attempts to eradicate insect pests through massive chemical spraying programs had many unintended consequences for people and wildlife alike. This view, although denied vociferously by some in the government, was shared by many of Carson's colleagues. Yet the citizenry heard little of this debate. The problem was not so much that those

questioning the wisdom of eradication programs were spirited away in the middle of the night but that much of their data remained soundproofed in internal documents and technical journals, that follow-up research was sorely underfunded, and that government officials turned a deaf ear to bearers of bad news.

By 1952, Carson had become a best-selling author of nature books and was able to retire from government service. However, she continued to follow the pesticide debate as it clamored through the halls of the U.S. Department of Agriculture and the National Academy of Science. Meanwhile, evidence of harm was becoming visible to many average citizens—even in the absence of public discussion. In 1958, a writer friend in Massachussets sent Carson a letter full of painful details about a mosquito control campaign that had resulted in a mass death of songbirds near her home. Those that lay scattered around her DDT-contaminated birdbath had perished in a posture of grotesque convulsion: legs drawn up to their breasts, beaks gaping open.

This letter prompted Carson to begin a comprehensive investigation of pesticides. In letters to friends about this project, she referred often to her need to speak out in defense of the natural world: "Knowing what I do, there would be no future peace for me if I kept silent." Having documented a cavalcade of problems attributable to pesticides—from blindness in fish to blood disorders in humans—she could find no magazine or periodical willing to publish her work. Carson decided to write a book.

Its title, *Silent Spring*, refers to an eerier kind of silence: the absence of bird song in a world poisoned by chemicals. Indeed, Carson argued, pesticidal warfare, waged with reckless disregard, threatens to extinguish a chorus of of living voices—those of birds, bees, frogs, crickets, coyotes, and ultimately us. On this level, *Silent Spring* can be read as an exploration of how one kind of silence breeds another, how the secrecies of government beget a weirdly quiet and lifeless world.

Through this process of silencing, the interconnectedness of all life forms is revealed. Carson studied the failed attempt to prevent the Japanese beetle from invading Iroquois County, Illinois, a rural farming community located due east of my home county. After intense and repeated pesticide bombardments by air during the mid-1950s, many insect species, sickened by the spraying, became easy prey for insect-eating birds and mammals. These creatures became poisoned in turn and, in ever-widening circles of death, went on to sicken and kill those who fed on their flesh, leaving a landscape devoid of animal life—from pheasants to barnyard cats.

Meanwhile, the targeted beetle species continued its westward advance. The protracted war against this enemy had accomplished nothing, but the residues of dieldrin remaining in the water and soil—like landmines left behind by a retreating army—guaranteed further casualties for decades to come. All for the dream of a beetleless world. The ecological tragedy of Iroquois County, said Carson, is narrated by the mute testimony of its dead ground squirrels: found with their mouths full of dirt, they had gnashed at the ground as they died.

The third kind of silence that fascinated Carson was the hushed complicity of many individual scientists who were aware of—if not directly involved in documenting—the hazards created by chemical assaults on the natural world. While dutifully publishing their research, most were reluctant about speaking out publicly, and some refused Carson's requests for more information. Writing in *Silent Spring*, Carson acknowledged the constant threat of defunding that hushed many government scientists. But she made clear in her private correspondence that she had little respect for those who knew but did not speak, a combination she saw as cowardice:

> The other day I saw a wonderful quote from [Abraham] Lincoln. . . . I told you once that if I kept silent I could never again listen to a veery's song without overwhelming self-reproach. . . . The quote is "To sin by silence when they should protest makes cowards out of men."

After *Silent Spring* was published, Carson turned her attention to the political and economic reasons behind the fearful silence of her colleagues in science. In a speech to the Women's National Press Club, she questioned the cozy relations between scientific societies and for-profit enterprises, such as chemical companies. When a scientific society acknowledges a trade organization as a "sustaining associate," Carson asked, whose voice do we hear when that society speaks—that of science or of industry?

Carson was just beginning to develop her ideas on the interlocking economic structures that bound the direction of medicine and science to the interests of industry when she herself was silenced. Leaving behind an adopted son, plans for summer fieldwork, and sketches for two more books, Rachel Carson died of breast cancer on April 14, 1964.

Sheltered from wind and waves, the Rachel Carson National Wildlife Refuge in southern Maine is essentially a salt marsh. It bears little resemblance to the rest of the Maine coastline, where the intense drama of ocean meeting rock prohibits marsh grasses from taking root. It is, therefore, a very different place from the craggy tidal pools and moonlit coves of Rachel Carson's beloved summer home farther north.

Walking along the paths of the refuge that bears her name, I realize I feel less close to Rachel Carson here than in the climate-controlled sanctum of the Beinecke Library. At the dedication site, a large plaque dutifully lists the titles of her books and then credits her for inspiring millions to greater environmental consciousness. Its brief, abstract sentences remind me how remote a figure Carson became after her death. Like Rosa Parks, Carson is a symbol, a muse, a spark that ignited a social movement, a name to be invoked before a speech. In this, she seems unknowable and unhuman.

Still, my Illinois nerve endings are stirred by the softness of the landscape here. The lay of the land feels familiar, although most of the plant species are not. Salt meadow grass knits together the higher grounds, while the lower sweeps are bound by the taller and stiffer saltwater cordgrass. The sinuous borders between them represent the reach of the tide. The trail guide boasts that these two grasses together can produce as much plant matter per acre per year as a prime midwestern cornfield. I smile. No way.

It is November 1993. I have driven here from Boston with my friend Jeannie Marshall, who patiently endures my lecture on corn productivity and then turns my attention to the weather. "Doesn't it feel like a different season?" Jeannie asks. On the dry uplands, a rich

summery light pours through the oak trees that hang willfully onto their curled leaves. Like a flame, my dog streaks through the understory in pursuit of unseen life forms. Old oak leaves are a distinct shade of brown, which I am accustomed to viewing in hues of light more pale and dilute. We agree it is oddly beautiful to see them cast in such radiance.

The tidal creeks that worm their way through the stands of cordgrass confuse and delight me. I depend on surface water to reveal slope and direction, but poised here at the margin of the sea, these two concepts are subordinated to a larger force. At low tide, the creeks flow into the ocean. At high tide, the ocean flows into the creeks. The streambeds here pulse back and forth, flooding and draining, in a continual exchange of water and salt. There is no clear direction.

Which is exactly how I feel standing next to my friend: poised without direction in an uncertain but beautiful season. Hopeful yet unnerved.

Just diagnosed for a second time with a rare cancer of the spinal cord, Jeannie is in between surgery and radiation treatments. She is recovering quickly—getting well in preparation for becoming sick in an attempt to get well. She moves so nimbly along the paths looping through the refuge that I scarcely need to modify my own movements. If not for her cane, we could be mistaken for any two young day-trippers escaping from the city. But we are on an escape of another kind, and I feel protective and scan the path ahead for rocks, roots, and sinkholes.

Although our friendship is a recent one, the many parallels in our lives promote intense conversations whenever we are together. Both of us are writers in our thirties. Both of us became cancer patients in our twenties. Both of us grew up in communities with documented environmental contamination, high cancer rates, and suspicions that these two factors are related to each other. Both of us grew up in families constructed through adoption (I was adopted; Jeannie's mother was adopted), and we each have a keen curiosity about the interplay between heredity and environment in our lives.

And we have spoken at length about all of these topics. We have talked about what it means to have cancer as young women and about

the relative significance of genealogy and ecology in that context. We have discussed our relationship with our doctors, our families, our hometowns, our writing, our bodies.

The depth and easiness of our talking carry us along today—through the luminous oak groves, out along the boardwalks that float over salt meadow grass, up onto the observation deck that overlooks the confluence of the Mariland River and Branch Brook, whose waters throb back and forth. It seems to me in these moments that Jeannie and I have words for everything. We have rejected the cultural taboos of the past that wrapped the topic of cancer in shrouds of silence, but we have also turned away from the happy cancer chatter that regularly arrives in our mailboxes in the form of brochures and magazines dedicated to the concepts of coping, accommodating, and adjusting to this disease. In its place, we have created a language between us that is compassionate, smart, fearless, open.

What my friend and I do not choose to talk about this afternoon are the dark days that lie ahead for her. Days of lying under the crosshairs of a proton-beam cyclotron. Fatigue, vomiting, blood tests. Continuously handing one's body over to technicians and doctors in a process that we call becoming medicalized. But between us, we have years of experience with cancer. I have no doubt that when those days arrive we will find a vocabulary for every experience.

We pause to examine some small ponded areas near the brook. These are salt pannes—low spots that hold water when the tide ebbs. Evaporation concentrates the salt to such extraordinary levels that only a few inconspicuous plants can survive. Glassworts. Sea-blite. Life thriving among bitterness.

"I like this place," I finally admit.

"I do too. It's nice to be here."

❧

On average, breast cancer robs the woman it kills of twenty years of life. This means that in the United States, nearly one million years of women's lives are lost each year. In 1964, Rachel Carson died at age fifty-six—twenty years short of the average life expectancy for U.S. women at that time. Despite all the ways she was extraordinary, as a victim of breast cancer Carson was utterly typical.

Carson was diagnosed in 1960, in the thick of researching and writing *Silent Spring*. Her tumor spread to her lymph nodes and to her bones, eventually including her spine, pelvis, and shoulder. She continued writing, even though surgery left her exhausted and radiation treatments, nauseated. Other ailments—joint and heart problems that were exacerbated, if not caused, by the radiation—brought crippling and immobility. The tumors in her cervical vertebrae caused her writing hand to go numb.

Carson lived for eighteen months after finishing *Silent Spring*—long enough to smoke out a hornet's nest of ridicule and invective from the chemical industry, as well as to receive every imaginable award from the world of arts, letters, and science. Privately, Carson expressed relief and satisfaction at having lived to see *Silent Spring* complete—a reaction many of Carson's commentators and colleagues have repeatedly underscored.

But there is another story embedded in the remaining fragments of Carson's private writings. Far from viewing *Silent Spring* as her crowning achievement, Carson ached to go on to new projects as well as to seize the opportunities that her success now afforded. She did not go gently or gratefully into any good night. As her letters reveal, she died hoping for another remission, another field season, more time. And in this desire, Carson appears before us again as a typical woman with breast cancer.

From a letter to her dearest friend, Dorothy Freeman, in November 1963:

> There is still so much I want to *do*, and it is hard to accept that in all probability, I must leave most of it undone. And just when I have attained the power to achieve so much I feel is important! Strange, isn't it?

And a few months later:

> But in spite of the blow yesterday, darling, [presumably, news of more cancer] I am able to feel that another reprieve can perhaps be won. . . . Now it really seems possible there might be another summer.

There was not.

The winter of 1994 let go of Boston during the second week of March. Over a hundred inches of snow had fallen since December, and most of it lay in towering mounds over every inch of grass and concrete that was not a passage for car traffic or an entrance to a building. Now the ice piles were finally melting, and everything that had been lost or abandoned began to surface: mittens, shovels, coat hangers, trash cans, lumber, laundry baskets, entire automobiles. Stratified layers of sand, cat litter, and gravel, which had been trapped at various depths, redeposited themselves in swirling alluvial fans along the sidewalks as rivulets of meltwater streamed toward the storm sewers.

Jeannie and I move through this landscape on our way from the Massachussetts General Hospital to her apartment in the North End. Neither of us speaks. The sound of our boots on the gravelly outwash seems deafening. Jeannie is not using a cane today, and we are walking even faster than we did four months ago in the salt marsh. In my mind's eye, I am tossing all obstacles out of our way—chunks of ice, orange traffic cones, parked cars, cement barricades. I am aiming a wrecking ball at every building.

Neither of us can believe what we have just heard. After eight miserable weeks of radiation treatments to the tumor in her lower back, the original tumor in her neck—successfully removed and treated six years ago—has returned. "Massive recurrence," to quote the neurologist who had just received the scans from the radiologist.

In fact, he said these words to us as soon as we walked into his office and closed the door. We were still standing in our winter coats and had not yet found our chairs. "Massive recurrence." I struggled with my buttons, my scarf, the zipper to my book bag. My hands refused to work correctly. It had become my job in these settings to serve as the scribe and, as such, to provide complete documentation of conversations between patient and doctor.

This ritual could not withstand the current assault. I am a crack note-taker, but my hands did not want to write the words being spoken. All my attention was trained on overriding my desire to lay down the pen. The doctor spoke quickly and relentlessly as he described the tissues that were being "destroyed" or "strangled" by the chordoma's advance. He was clearly upset but seemed unable to blend his despair with a demonstration of compassion or hope.

Jeannie remained calm. She asked him to conduct a neurological exam; her symptoms, after all, were improving. Her body seemed to be telling a different story. He refused. What would be the point? The scans told the whole story. He asked her to look at them. She refused. They each accused the other of not listening. I focused on writing faster. It was a battle of narrative. Which told the true story? the radiologist's report? or Jeannie's body? Finally, the meeting ended.

"Don't shoot the messenger," he said flatly as we were once again standing and struggling with our coats.

Now we are back in Jeannie's apartment. A garbage truck backing down the street sets off a car alarm. I imagine setting fire to them both. Jeannie lies on the bed, saying nothing. I make tea.

Say something, I order myself. The words I have just transcribed in the doctor's office are the same ones I have dreaded since my own diagnosis. Now I have heard them spoken—by a doctor who was looking into the eyes of the person sitting next to me. Not mine. Not me.

Say something.

On the day of my diagnosis, I was hospitalized and friends from college came to visit. They politely stepped into the hallway when the doctor came in. He gently told me the results of the pathology reports and the treatment plan he had in mind. We sat together for a while. After he left, my friends gingerly reentered the room. They were trying to be appropriate.

"I have cancer."

There was silence—and then some kind of awkward talking, but no one really acknowledged what I had said, including myself. Later, I was furious with all of us.

Say something.

But what? I sit down at Jeannie's kitchen table and begin to review the notes I have taken to make sure they are legible and complete. Were these the words that were really said? Can their meanings be trusted? Perhaps we had simply entered an unfamiliar culture

where the phrase "massive recurrence" actually means "hello, have a seat," and "don't shoot the messenger" is a way of saying "so long, take care."

You are not saying anything.

I think back to the sunlit oak grove and the salt pannes where language was so easy. How sure I was then that I could be depended on to push any situation, no matter how dire, into the bright daylight of human speech. I think back to Rachel Carson. Tumors in her cervical vertebrae caused loss of functioning in her right hand, the writing hand. Jeannie is also right-handed. It is her left hand that is becoming weak.

In the four years Rachel Carson struggled with breast cancer, she worked to break silence in the public arena. Yet in her private life, she created at least two kinds of silence. One was permeable; one, absolute.

The former kind was a sort of drapery Rachel periodically pulled between herself and her confidante, Dorothy Freeman. In some of her letters to Dorothy, Rachel described the progress of her disease in detailed medical terms. But in others she spoke only in code, referring elliptically to "menacing shadows." Rachel often refrained from divulging bad news, downplayed the miseries of treatment, and stated her belief that the expression of fearful thoughts would only make them loom larger.

Reading again the collected letters between these two friends, I see an elaborate dance of silence. At times, Dorothy seemed relieved at the abstentions and forebearances, even seeming to encourage Rachel to keep her own counsel. Dorothy did not share her correspondent's taste for writing about cancer in detached, medical tones. She refers not to Rachel's radical mastectomy but to her "hurt side."

And yet at other times, Dorothy seemed to feel shut out by Rachel's silences. Both correspondents entreated the other not to censor her thoughts or feelings. Both correspondents also admitted they were not fully disclosing their own secret fears, out of a need to protect the other. Rachel sometimes pulled back the curtain and confided a darker story—one that admitted to pain and despair. Sometimes she followed these communications with retractions and apologies. And sometimes the letters containing these dark confessions were, upon request, destroyed.

Confessing and recanting. Withholding and divulging. This mesh of conflicting impulses is part of a familiar script that is enacted again and again between cancer patients and those who love them. And in this familiarity, Carson emerges once more, poignantly, as an ordinary woman.

The second kind of silence was a fortress of secrecy Rachel constructed around her own diagnosis, a secrecy she expected Dorothy to collude with her in maintaining. Rachel strictly forbade any discussion, public or private, about her illness. This decision was intended to retain the appearance of scientific objectivity as she was documenting the human cost of environmental contamination. She also wished to yield her enemies in industry no further ground from which to launch their personal attacks.

Accordingly, Rachel instructed Dorothy to say nothing of her condition to their mutual friends and aquaintances, lest rumors take root. If need be, Dorothy was to lie. "Say you heard from me recently and I said I was fine," she told Dorothy to tell her neighbors in Maine. "Say . . . *that you never saw me look better.* Please say that."

What personal price each of these women paid for upholding this code of silence is impossible to know. Being sworn to secrecy can be a terrible burden. Anticipating the unintentional slip of the tongue that could ruin one's career must have been equally crushing. Against this backdrop of agreed-upon silence is the fact that Carson's state of health should have been obvious to anyone who cared to look at her. But not seeing is another form of silence.

As soon as *Silent Spring* was published, Carson was thrust into the national spotlight. She spoke in front of Congress, at the National Press Club, and on national television. In the photographs and old film clips documenting these occasions, she looks for all intents and purposes like a woman in treatment for cancer. She wears an unfortunate black wig. Her face and neck exhibit the distorting puffiness characteristic of radiation. She holds herself in the ginger, upright

manner of one who has undergone surgery. The alteration in her appearance that followed her cancer diagnosis is dramatic.

The newspaper clippings in the Beinecke Library that trace her various public appearances in the waning days of her life are full of elaborate descriptions of what type of elegant suit Miss Carson chose to wear and how delightfully she comported herself. The accompanying pictures tell a different story. But it is a story read in silence by a woman from a future generation who knows how it will end.

Thanksgiving morning is sunny and mild. Jeannie and I decide to walk to Waterfront Park overlooking Boston Harbor. It is now more than a year since our buoyant walk through the wildlife refuge. Jeannie has just finished another round of radiation treatment, and because her balance has been affected, our pace is much slower. Orange tail swishing, my dog circles patiently, herding us toward the water. Somehow, Jeannie has managed to finish writing two articles, one about the search for cancer genes and another on breast cancer prevention for a British medical text. Feeling triumphant, she is in the mood to talk about cancer—but not her own.

"You remind me of Rachel Carson," I laugh.

We talk all the way to the ocean and back.

Silent Spring is remembered for the birds. When I ask people to name words, phrases, or images that Rachel Carson's book evokes for them, "thin eggshells" is among the most frequent responses. Yet this consequence of pesticide exposure—bird eggs so fragile they crush under the airy weight of their own brooding parents—is scarcely mentioned in *Silent Spring*. Perhaps we like to equate Carson with eggshell thinning because it is a problem that largely fixed itself after DDT and a handful of other pesticides were finally restricted for domestic use. In this way, Carson's predictions of disaster can be simultaneously viewed as both prophetic and successfully averted. A comfortable reckoning.

Of course, the fate of birds and other innocents caught in the chemical crossfire certainly was a central concern of *Silent Spring*. As proof of harm, their deaths were starkly visible. Who can deny the ground squirrels' cold little mouths packed with dirt? Or shrug off the pitiful sight of songbirds writhing in the grass? But *Silent Spring* makes clear that this kind of evidence, however immediate and tangible, is only one part of a much larger assemblage that also includes human cancer. Even while hiding the image of herself as a cancer patient, Carson provided many others: from farmers with bone marrow degeneration to spray-gun-toting housewives stricken with leukemia.

Making visible the links between cancer and environmental contamination was challenging for Carson, and the task continues to be daunting. However agonizing their deaths, cancer patients do not collapse around the birdbath. Decades can transpire between the time of exposure to cancer-causing agents and the first outward symptoms of disease. When birds drop out of the sky in great numbers, we ask why. When someone we love is diagnosed with cancer, questions of cause are often of less immediate relevance than questions about treatment. Questions about the past are subordinated to questions about the suddenly uncertain future.

Based on all the data available to her in 1962, Carson laid out five lines of evidence linking cancer to environmental causes. While any one alone would be insufficient proof, when viewed all together, Carson asserted, a startling picture emerges that we ignore at our peril. First, although some cancer-producing substances—called carcinogens—are naturally occurring and have existed since life began, twentieth-century industrial activities have created countless such substances against which we have no naturally occurring means of protection.

Second, since the arrival of the atomic and chemical age that followed World War II, everyone—not just industrial workers—has been exposed to these carcinogens from the moment of conception until death. Industry manufactures carcinogens in such large quantities and in such diverse array that they are no longer confined to the workplace. They have seeped into the general environment, where we all come into intimate and daily contact with them.

Third, cancer is striking the general population with increasing

frequency. At the time of Carson's writing, the postwar chemical era was less than two decades old—less than the time required for many cancers to manifest themselves. Carson predicted that the full maturation of "whatever seeds of malignancy have been sown" by the new lethal agents of the chemical age would occur in the years to come. She also believed that the first signs of catastrophe were already visible. At the end of the 1950s, death certificates showed that a far greater proportion of people were dying of cancer than had been true at the turn of the century. Most ominously, children's cancers, once a medical rarity, were becoming commonplace—as revealed both by vital statistics and by doctors' observations.

Carson's fourth line of evidence came from animals. Experimental tests were beginning to reveal that low doses of many pesticidal chemicals in common use caused cancer in laboratory mice, rats, and dogs. Moreover, many animals inhabiting contaminated environments develop malignant tumors; *Silent Spring* not only documents acute poisonings of songbirds but also reports on cases of sheep with nasal tumors. These incidents supported the circumstantial evidence from human populations.

Finally, Carson argued, the unseen inner workings of the cell itself corroborate the story. At the time of *Silent Spring*'s publication, the mechanisms responsible for basic cellular processes such as energy production and regulation of cell division were just beginning to be elucidated. The role and structure of the twisting DNA molecule had been discovered only recently. From the glimmers she was able to gather from widely scattered studies, Carson spotlighted three properties that she believed would ultimately explain why these new chemicals were associated with cancer: they were able to damage chromosomes and thereby cause genetic mutations (a property shared with radiation, which had already been shown to cause cancer); they were able to mimic and disrupt sex hormones (high estrogen levels were already being correlated with high cancer rates); and they were able to alter the enzyme-directed processes of metabolism (by which we break apart molecules to generate energy and synthesize new substances). Carson predicted that future studies on the mysterious transformation of healthy cells into malignant ones would reveal that the roads leading to the formation of cancer are the same pathways that pesticides and other related chemical contaminants operate along once they enter the interior spaces of the human body.

Like the assembling of a prehistoric animal's skeleton, this careful piecing together of evidence can never furnish final or absolute answers. There will always be a few missing parts, first because experimenting on human beings is not, thankfully, considered ethically acceptable. Human carcinogens must, therefore, be identified through inference. One set of clues is provided by observations of people who have been inadvertently exposed to substances suspected of having cancer-causing tendencies. But often these people have been exposed to unknown quantities over unknown periods of time. Observations of laboratory animals exposed to known quantities of possible carcinogens supply a second set of clues. But different animals can vary in their vulnerability to certain kinds of cancers and in their sensitivity to certain kinds of chemicals. Which species should serve as our surrogates in these studies? Rats? Mice? Fish? Dogs? Which species' lymph nodes, bone marrow, brain tissue, prostate glands, bladders, breasts, livers, and spinal cords behave most like those in humans when exposed to particular substances?

Another reason for scientific uncertainty is that the widespread introduction of suspected chemical carcinogens into the human environment is itself a kind of uncontrolled experiment. There remains no unexposed control population to whom the cancer rates of exposed people can be compared. Moreover, the exposures themselves are uncontrolled and multiple. Each of us is exposed repeatedly to minute amounts of many different carcinogens and to any one carcinogen through many different routes. From a scientific point of view, such combinations are especially dangerous because they have the capacity to do great harm while yielding meaningless data. Science loves order, simplicity, the manipulation of a single variable against a background of constancy. The tools of science do not work well when everything is changing all at once.

It is March 1995. Winter and spring have hung together in the air for weeks, neither yielding to the other. On the phone, Jeannie is trying

to describe to me a new sensation she feels across the skin of her chest. It is vague and formless. There are no real words for it. I am attempting to understand how this symptom fits together with a few other recent problems she has reported. Morning vertigo. A funny feeling when she swallows. What picture is emerging here? What does her doctor say? She turns back my questions.

"Let's talk about the chapter you're writing now. What is it called?"

"Silence."

"Let's talk about that."

THREE

time

Like a jury's verdict or an adoption decree, a cancer diagnosis is an authoritative pronouncement, one with the power to change your identity. It sends you into an unfamiliar country where all the rules of human conduct are alien. In this new territory, you disrobe in front of strangers who are allowed to touch you. You submit to bodily invasions. You agree to the removal of body parts. You agree to be poisoned. You have become a cancer patient.

Most of the traits and skills you bring with you from your native life are irrelevant, while strange new attributes suddenly matter. Beautiful hair is irrelevant. Prominent veins along the soft skin at the fold of your arm are highly prized. The ability to cook a delicious meal in thirty minutes is irrelevant. The ability to lie completely motionless on a hard platform for half an hour while your bones are scanned for signs of tumor is, conversely, quite useful.

On November 9, 1994, the results of the incinerator referendum in Forrest showed 466 against and 406 for. Some members of the Forrest Development Corporation vowed to proceed anyway, but Kirby demurred. "We're apprehensive about committing to the project if that support for it is soft. We don't want to have to fight a battle every time we want a sewer extension."

The following September, an appellate court in Springfield, Illinois, upheld unanimously the decision of the Illinois Pollution Control Board regarding the unfair siting approval of the incinerator in Havana. The judges cited both a Massachusetts trip paid for by Kirby's corporation and the improper influence of that corporation on the hearing officer.

On January 11, 1996, the Illinois General Assembly repealed the Retail Rate Law. According to the governor, "Most communities do not want the incinerators. And it is time we stopped asking our taxpayers to subsidize them."

On January 25, 1996, John Kirby died of malignant mesothelioma—a form of lung cancer—in a Springfield, Illinois, hospice.

ELEVEN

our bodies, inscribed

Among forest trees, size and age can be remarkably dissociated. Seedlings germinating in deep shade are often swiftly overtaken by those sprouting up in light-filled spaces nearby. Saplings browsed by a passing deer lose vertical height relative to neighbors less palatable. By these and other means, senior members of a forest community sometimes grow old beneath a canopy of younger trees.

Field ecologists, therefore, rely on tree-ring analysis to reconstruct the history of forests. I once spent a summer in Minnesota engaged in this kind of work, which begins with pressing the bit end of a hand borer against the bark of a tree at chest height, leaning against it with all one's weight, and slowly turning the handle until the steel threads have chewed into the flesh beneath and have wound themselves straight into the tree's exact center. A slender wand of cool,

damp wood is then extracted with the narrowest of spatulas, sealed in an envelope, and, along with an assortment of other tree cores, taken back to the laboratory to be read.

These cores are banded with colored rings, each representing a season of growth. An experienced dendrochronologist (which I am not) can identify in the subtler patterns of these circles not only age but also periods of changing light levels, insect plagues, drought, flood, or fire. An individual tree carries within its own body an ecological chronicle of the entire community.

In this, people are not so different. Our bodies, too, are living scrolls of sorts. What is written there—inside the fibers of our cells and chromosomes—is a record of our exposure to environmental contaminants. Like the rings of trees, our tissues are historical documents that can be read by those who know how to decipher the code.

Body burden refers to the sum total of these exposures and encompasses all routes of entry (inhalation, ingestion, and skin absorption) and all sources (food, air, water, workplace, home, and so forth). In the case of fat-soluble, persistent chemicals, body burdens provide a measure of cumulative exposure. For example, 177 different organochlorine residues can be detected in the body of an average middle-aged American man. Some of these exposures occurred in infancy, others in adolescence, and still others in adulthood. In the case of chemicals quickly metabolized and excreted, the body burden is an index more akin to a press release than a biography. It reports on the status of immediate and ongoing exposures to particular contaminants at single points in time.

The problem with body burdens is that they require sampling each and every fluid and compartment of tissue. This task can be accomplished during an autopsy, but for living people, total exposures are more often derived from measurements taken from a specific source. Blood, urine, breast milk, exhaled air, fat, semen, hair, tears, sweat, and fingernails have all been used for this purpose.

Different tissues work more or less well for different contaminants. The blood inside umbilical cords, for example, may identify compounds that pass through the placenta and enter the bodies of developing fetuses. Their presence provides clues to the causes of childhood cancers. So far, these include PCBs and an array of pesticides. Urine, on the other hand, is a good medium for looking at water-soluble contaminants, such as organophosphate and carbamate pesticides. Sampling urine, researchers have estimated that the bodies of most members of the U.S. population contain detectable levels of the insecticide chlorpyrifos, a common ingredient in pet flea collars, lawn and garden pest control products, indoor foggers, and roach, ant, and wasp poisons.

PCB levels in blood have been demonstrated to correlate roughly with their overall body burden, once differences in fat content have been accounted for. Hence, a simple blood draw can provide an estimate of lifetime PCB exposure. (Blood contains a certain fraction of fat.) Nevertheless, complications arise even here. Different organs seem to sequester differing proportions of each of the 209 chemical varieties. If PCB molecules were all created equal, this partitioning process would matter less. However, members of the PCB family differ in their persistence, potency, and carcinogenic potential. Furthermore, PCBs are broken down into different metabolic products that distribute differently though human tissues. If greater amounts of the more toxic varieties differentially settle into the lung, kidney, and uterus rather than the liver, breast, and adrenal glands, for instance, then a simple measure of total PCB concentration in blood plasma may not tell the whole story.

A sponge for oil-soluble chemicals, body fat is considered an especially sensitive indicator of exposure to persistent environmental contaminants. In Japan, researchers examined a variety of industrial contaminants in preserved fat collected from men who had died between 1928 and 1985. The highest concentrations of DDT, PCBs, and chlordane were found in samples collected during their respective periods of maximum production, import, and use. In a 1996 study conducted in Mexico, researchers found that levels of DDT in living human tissues varied predictably across geographic space: residue levels in both abdominal fat and breast fat were highest in areas of intense agriculture and in tropical regions where DDT was used for malaria control.

Breast milk has a lexicon all its own. About 3 percent fat, it con-

tains high concentrations of fat-soluble contaminants. These pollutants are carried by the blood into the breast from fat reserves scattered throughout the body and probably including the breast fat itself. Since 1951, surveys of human milk in the United States have consistently shown contamination by an array of persistent, chlorinated chemicals. The issue of insecticides in breast milk received close attention from Rachel Carson in 1962. A dozen years later, 99 percent of breast milk sampled in the United States was also shown to contain PCBs. About one of every four of these samples contained PCB concentrations exceeding the legal limit (2.5 parts per million), above which level commercial formula is pulled from the shelves. Or, to express this another way: by 1976, roughly 25 percent of all U.S. breast milk was too contaminated to be bottled and sold as a food commodity.

The cancer risks assumed by these mothers and their nursing infants—now adults, some with children of their own—remain to be seen. The possible relationship between carcinogens in breast milk and breast cancer (or cancer in offspring) has not been systematically investigated.

A study of more than eight hundred nursing mothers in North Carolina has uncovered three patterns that make this question an urgent one. Researchers found that the concentration of organochlorine chemicals in breast milk increased with the age of the mother, increased with the amount of sport fish consumed, and decreased dramatically over the course of lactation and with the number of children nursed. The first trend indicates that our bodies are still amassing fat-soluble contaminants faster than we can eliminate them. The second attests to the ongoing contamination of our rivers, streams, and lakes.

The third fact is the most ominous one. Organochlorine contaminants are not easily expunged from our tissues. Their sharp decline in concentration over the course of breast-feeding, therefore, represents the movement of accumulated toxins from mother to child. It signifies that during the intimate act of nursing, a burden of public poisons—insect killers, electrical insulating fluids, industrial solvents, and incinerator residues—is shifted from one generation into the tiny bodies of the next.

Happily, concentrations of a few of the most pernicious contaminants of breast milk are stabilizing or even beginning to drop. Long-term monitoring of human milk in Germany, for example, showed slight declines during the early 1990s in levels of dioxins, furans, organochlorine pesticides, and PCBs. Similarly, pooled samples of human milk archived in the Mothers' Milk Centre in Stockholm, Sweden, show declines in many PCB and DDT metabolites from 1972 to 1992. These trends indicate that efforts to shut down known sources of these chemicals are finally beginning to have an effect on their respective body burdens.

The human body is an endless construction site where demolition and renovation occur simultaneously and continuously. Different tissues carry on this work at different rates; the lining of the stomach is entirely overhauled every few days, while a complete restoration of the bones' internal scaffolding requires years. All tissues replace themselves through the orderly process of cell division—mitosis—in which one cell splits in half and becomes two. Damaged and aged cells slated for removal undergo a programmed form of death known as apoptosis. All this activity is coordinated through an elaborate system of communication that cell biologists are just beginning to understand.

A certain amount of supervision is provided by a cell's own DNA, which sends out from the nucleus periodic messages instructing the cell to begin (or cease) dividing. We know also that chemical signals from neighboring cells can alter the pace of this process. And we know that marching orders sometimes arrive from distant headquarters. These often take the form of hormones, as when estrogen from a woman's ovaries causes the cells in her breasts to begin dividing.

However scant our knowledge about its regulation, the actual feat of mitosis, its procession of precise, elegant steps, is becoming increasingly clear. Mitosis begins inside a circle within a circle: the nucleus of the cell where the DNA is quartered.

The first step is the doubling of each of the strands of DNA, the chromosomes. Their duplication will enable both daughter cells to

receive a complete set. For this task, a crew of enzymes creates an exact replica of each original chromosome (which is split in half lengthwise and used as a template for its own duplication). Lying side by side, the two identical strands are then cuffed together and come to resemble a gangly letter *H* or sometimes a stout *V*.

Humans possess forty-six individual chromosomes, each consisting of a curly DNA ladder and each bearing many thousand genes. Once all forty-six gene-studded chromosomes have been so copied, a dance begins. The nuclear membrane disintegrates. The chromosomal couples move to the center of the cell and form a vertical line. Fine threads called spindle fibers extend horizontally from opposing ends of the cell and attach to each member of a pair. The fibers contract. Simultaneously, the twinned chromosomes pull apart, their midpoint connections giving way as the left and right halves of the *H*s and the *V*s are towed through the watery protoplasm to opposite poles. Just as the cell begins to pinch in half, a membranous curtain closes around each new grouping of single-stranded chromosomes, and they are once again cloistered within a nucleus. They will remain there, directing the synthesis of proteins, until the mitotic cycle begins anew and once again releases them.

Cancer is mitosis run amuck. Instead of reproducing in careful, methodical fashion, cancer cells carry on replication and division despite a myriad of directives designed to restrain such activity. Cancer cells are dancers deaf to the choreographer. They are builders in flagrant disregard of zoning ordinances and architectural blueprints. They are defiant, disobedient, and in the view of many cancer biologists, almost purposeful in the ways they disrupt cellular biochemistry.

Besides a propensity for unrelenting growth, a cancer cell is known for two other traits: invasiveness and primitivism. The ability to invade other tissues distinguishes cancer from other freakish outgrowths, such as warts. This facility operates at both a local level—cancer also ignores property lines—and a distant one, as when cancer cells are shed from the primary tumor and seeded throughout the body as metastases. Destroying healthy tissue and clogging vital passageways, both habits make cancer life-threatening.

By *primitive*, biologists mean that the tissues created by cancer

appear to have reverted back to some earlier, cruder, unformed stage of development. They no longer bear much resemblance to the differentiated structures of which they were originally a part. Typically, the hard lump in the breast that turns out to be a malignancy is a direct descendent of one of the smooth, flat cells that wallpaper the interior surfaces of the slender mammary ducts. But, microscopically, the tumor's mass of cells no longer looks anything like the benevolent sheets of breast epithelial tissue it came from. In general, the less a tissue resembles its previous, respectable, specialized self, the more virulent the cancer. Along with runaway growth and the propensity to spread, this tendency to devolve into an immature, unrecognizable state is the result of a long accumulation of genetic injuries.

A cancer cell, then, is made, not born. Cancer arises through a series of incremental changes to chromosomal DNA. Some of these DNA alterations can be inherited, but the vast majority are acquired during the lifetime of an individual when genes perfectly healthy at the time of conception become damaged. This process can happen through numerous pathways. Routine errors made during DNA replication are one. Sabotage by carcinogens is another. About 100,000 different genes are strung along our chromosomes. To contribute to cancer, at least some of these encounters between carcinogens and genes must involve the handful that help govern cell division.

These growth-regulating genes come in two basic varieties. The first group are called oncogenes. In their normal state, these bits of DNA convey messages that encourage cell division. When mutated, however, oncogenes become hyperactive and ratchet up the rate of growth. Working on exactly the opposite principle are the tumor suppressor genes. Normally, they dampen the rate of cell division. In some circumstances—as when signs of DNA damage are about—they actually halt mitosis altogether and thereby nip in the bud the possible genesis of cancerous growth. Loss or inactivation of tumor suppressor genes may contribute to the birth of a tumor. If a mutant oncogene is a stuck accelerator pedal, then damaged tumor suppressor genes are faulty brakes. Either problem can result in runaway cell growth.

Different kinds of cancers are associated with different kinds of

mutations. The cells of most colon tumors, for example, turn out to contain both hyperactive oncogenes and nonfunctional tumor suppressor genes. One specific tumor suppressor gene located on chromosome 17 has been fingered in several big-ticket malignancies, including cancers of the lung, breast, colon, esophagus, bladder, brain, and bone. Indeed, alterations of this gene, named p53, may be involved in as many as half of all human cancers. Much as a gunshot wound indicates what kind of firearm was used in an assault, the particular nature of the p53 mutation often suggests the type of carcinogen responsible for the damage. Cigarette smoke leaves one kind of lesion, ultraviolet radiation another, and exposure to vinyl chloride a third. The mutational spectrum of this gene is so broad that the lung tumors from uranium miners can sometimes be distinguished from the lung tumors of smokers simply by looking at the specific location of the mutation. Breast tumors frequently display p53 mutations in a spectrum resembling that seen in lung tumors and varying across geographic regions.

Harm can befall growth regulator genes through a whole variety of pathways. Benzo[a]pyrene can adhere to a section of chromosome and, in so doing, create a DNA adduct. Like bits of chewing gum stuck to a strand of hair, adducts can cause mistakes to be made during the next cycle of DNA replication. Other carcinogens disable the spindle fiber apparatus, causing chromosomes to pull apart improperly. By these and other means, daughter cells can end up receiving mutated oncogenes and/or missing or impaired tumor suppressor genes. Alterations in other kinds of genes can abet the process. For example, DNA repair genes normally function to fix chromosomes vandalized by mutating agents or damaged accidentally during the normal course of mitosis. An injury to a repair gene is, therefore, a treacherous event, as it can lead to the accumulation of genetic lesions of all kinds. Fortunately, the carcinogenic process is lengthy and complicated, often requiring decades to unfold. It is also capable of being arrested at many points along the way.

In the language of cancer biology, the making of a cancer cell involves three overlapping stages: initiation, promotion, and progression. To become a full-blown malignancy, a cancer cell must pass through them all.

The first rite of passage, initiation, is characterized by small structural alterations to the cell's DNA strands. Arising spontaneously or resulting from an encounter with a carcinogen, these modifications—like tiny tattoos—are swift, permanent, and subtle. A small hole here. An inconspicuous inversion there. Cells so affected remain, to the human eye, indistinguishable in shape and appearance from their undamaged counterparts. Nevertheless, many initiated cells meet an early demise through the winnowing action of apoptosis. Any agent, then, that interferes with cell death can contribute to cancer by permitting damaged cells to continue along the pathway to tumor formation.

The immune system also plays a role in the selective destruction of incipient cancer cells, which presumably reveal their hand by exhibiting biochemical traits recognizable as abnormal. At what specific stage immune cells begin to mount a reaction is not entirely clear. It is known that certain environmental contaminants, including dioxin, suppress human immunity and that immune suppression is associated with several kinds of cancers, most notably leukemias and lymphomas. Recent studies from the former Soviet Union have shown clear relationships between exposure to certain pesticides and depression of the immune system's T cells.

Initiated cancer cells that escape detection advance to the next stage, promotion, which requires additional exposures to cancer-stimulating substances. Unlike initiation, promotion unfolds over a long period and may involve no actual mutations. In general, cancer promoters encourage cells to divide not by altering the physical structure of genes but by altering the expression of their chemical messages. Genes that are normally quiescent, for example, may become activated. Estrogen, in some cases, acts as a cancer promoter. As demonstrated in lab animals, so do many organochlorine compounds. The good news is that these effects wane when such agents are removed from the body.

Quite often, cancer promoters perturb an intricate communications pathway known as signal transduction. This system consists of a team of proteins relaying messages back and forth between the perimeter of the cell and the heartwood of the nucleus. By mechanisms barely elucidated, signal transduction proteins play a key role in the timing and coordination of cell division. Promoting agents can af-

fect the production and behavior of these courier molecules without permanently damaging the genes that code for their manufacture. The result is an expanded cluster of abnormal cells.

Like initiation but unlike promotion, the progression stage involves exposures that inflict physical injury to the DNA molecule. Mutations pile up. Chromosomes fall into disrepair and become increasingly unstable. Ironically, substances that act at this stage bestow on the cells they cripple some of cancer's most fearsome abilities: the capacity to spread and invade, enhanced sensitivity to hormones, and a knack for attracting blood vessels to the growing mass of tumor cells. Some researchers believe that arsenic, asbestos, and benzene can each function as cancer progressors, under certain conditions.

Agents that contribute to cancer do not all fall neatly into the categories of initiator, promoter, and progressor. Some, like radiation, are complete carcinogens that can play all three roles by themselves. Others, such as dioxin, appear to behave as promoters at low doses and complete carcinogens at higher levels, and they may also interfere with apoptosis. Still others initiate at low doses and promote and progress when their concentration in the body rises.

These shifting biological possibilities bring with them many social implications. First, they explain why no safe dose of a carcinogen exists. They also explain why similar exposures can pose very different degrees of danger to different people. The trace presence of a cancer-promoting pesticide in drinking water, for example, may represent absolute hazard to those whose breast, prostate, colon, or bladder tissue has already been initiated by some prior event (perhaps during childhood or because of occupation) or to those rare few born with a mutated gene that predisposes them to cancer. Individuals whose genetic material has suffered less previous damage may more successfully ward off the effects of promoting agents—as would those lucky persons who happen to possess a set of metabolism genes that allows for especially efficient detoxification and excretion of promoting substances.

The implications become even broader when we consider the dozens of known and suspected carcinogens to which we are routinely exposed and which may work alone, in concert, or cumulatively anywhere along the cancer continuum. In rats, for example, DDT

acts to accelerate tumors induced by an agent called 2-acetamidophenanthrene, even though neither one alone is capable of causing tumors to progress to a detectable level.

In the words of the veteran cancer biologist Ross Hume Hall, "Too often cancer research has focused on finding the last straw. It's time we looked at all the straws."

They have been compared to footprints, fingerprints, graffiti, and stigmata. They have also been hailed as the jewel in the crown of molecular epidemiology and described as decoding tools by which to read the body. They are biological markers, and, defined most plainly, they are indicators of physical damage caused by the interplay between human genes and environmental carcinogens. As such, biological markers serve as both signals of past exposure and predictors of future cancers.

Adducts, formed by mutation-inducing chemicals that adhere to DNA, are one type of marker. As discussed in Chapter Six, the tissues of beluga whales living in contaminated stretches of the St. Lawrence River display high concentrations of benzo[a]pyrene adducts. Similarly, in laboratory animals, researchers consistently find tight correlations between exposure to chemicals known to cause cancer and the concentration of adducts in the DNA of certain tissues. In humans, the relationship between adduct levels and cancer risk has not been worked out as definitively. However, some compelling evidence is now emerging from one of the most polluted regions on earth: Silesia, Poland.

Hard up against Poland's southern border, Silesia is blanketed with chemical plants, foundries, smelters, steel mills, coal mines, and cokeries (the great ovens that distill coal into coke for steelmaking). The cancer death rate is also impressively high here, persuading the molecular epidemiologist Frederica Perera of Columbia University to examine Silesian DNA closely. Her pioneering work has uncovered consistent associations between toxic exposures and adduct formation, on the one hand, and adduct formation and cancer risk, on the other.

Perera and her coworkers focused on polycyclic aromatic hydrocarbons, such as benzo[a]pyrene, which are released into Silesia's air in great abundance, mostly as by-products of coal and coke burning. Simply measuring their airborne concentration turns out not to be a reliable indicator of individual human exposure because polycyclic aromatic hydrocarbons are not only available for inhalation but also stick to skin (and are absorbed) and insinuate themselves into food (and are ingested). Moreover, these carcinogenic contaminants are handled differently by different people, depending on genetic and other factors that affect metabolism and detoxification.

The proof is in the cells' pudding. Perera found that the DNA of Silesian coke workers and Silesian city dwellers bore similar loads of polycyclic aromatic hydrocarbon adducts. These levels were two to three times higher than among rural folk. Perera also discovered a pronounced seasonal effect: the number of adducts rose during the winter months, when coal burned for domestic heating adds to the burden of aromatic hydrocarbons contributed by industry. Moreover, the level of adducts was correlated with the presence of chromosomal mutations thought to be affiliated with lung cancer. Together with studies showing that people with lung cancer carry higher burdens of polycyclic aromatic hydrocarbon adducts on their DNA than people without the disease, Perera's findings "strongly suggested that severe air pollution could indeed help induce lung cancer."

As Perera observed, DNA adducts provide us with a molecular link between environmental exposure and genetic injuries relevant to cancer. But they are not the only biological marker to do so. Alterations in certain proteins can also signal that villainy is afoot. For example, as a result of rearranging the genetic code, the carcinogen vinyl chloride triggers the production of a defective signal transduction protein. The presence of this protein in blood serum is therefore an unmistakable marker of vinyl chloride exposure. Alterations in DNA repair enzymes indicate other kinds of foul play, as do elevated levels of enzymes used for metabolizing foreign substances. The premier example here is cytochrome P450 enzymes, levels of which, as we have seen in Chapter Ten, rise rapidly in response to the presence of dioxinlike molecules.

Mutations themselves have a story to tell. For example, abnormally high levels of chromosomal breakages and genetic rearrangements have been identified in Minnesota fumigant and pesticide applicators. Some of these alterations consistently affect certain areas of chromosomes 14 and 18, and these mutations are of particular interest to researchers because they are the ones most commonly observed in non-Hodgkins lymphoma patients.

Certain mutational patterns are indicators of free radical exposure. A free radical, not part of any one classifiable chemical group, is any atom or molecule with just one electron in its outermost orbital. Electrons prefer to circle in pairs. When one is missing, the particle to which they belong becomes reactive—quick to surrender or absorb an electron from nearby molecules. If these molecules are chromosomes, mutations may result.

As part of the normal process of breaking apart food and hormone molecules, the cells of our bodies are constantly generating free radicals (and these undoubtedly contribute to our load of acquired DNA mutations). Fortunately, we possess several means of protecting our chromosomes from the resulting electron scramble—including the use of dietary vitamins to soak up free radicals as they are produced. Research by the molecular epidemiologist Donald Malins indicates that certain environmental contaminants generate free radicals when the body attempts to detoxify and metabolize them. Malins and his colleagues are currently attempting to determine whether specific patterns of free radical damage in the DNA of the human breast could provide a means of predicting breast cancer risk. Breasts may be particularly susceptible to free radical damage, even in the absence of toxic exposures. The process of metabolizing estrogen is itself a free radical–generating operation. Foreign chemicals that add to this burden—or that compromise DNA repair systems designed to counteract the ravages of routine free radical damage—may amplify the risk of breast cancer. In other words, while free radical generation is a normal but unfortunate consequence of fueling ourselves with chemical energy, preliminary evidence—from both animal and human tissue studies—suggests that chronic exposure to certain toxic substances can, in some circumstances, overwhelm the body's multilayered defense system against free radical stress and thereby acceler-

ate the rate at which we accumulate genetic injury. More research along this line of inquiry is essential.

The first clue that estrogen might play a role in breast cancer came in 1896 when a British surgeon reported that removal of the ovaries sometimes caused breast tumors to shrink. Many exhaustive studies conducted over the years since then have clearly indicated that a woman's chances of developing breast cancer are related in some way to her lifetime exposure to estrogen. Early first menstruation, late menopause, and late or no childbirths all raise a woman's lifetime exposure to estrogen and all are considered established risk factors for breast cancer—as is having a mother or a sister with the disease. Even so, taken together, such factors still account for only a minority of breast cancer cases.

Because the origin of most breast cancers remains unexplained and because there exists an apparent connection between breast cancer and naturally occurring estrogen, scientific attention has begun to turn to the possible role of xenoestrogens—chemicals foreign to the human body that, directly or indirectly, act like estrogens. We have already examined the evidence on xenoestrogens from epidemiological studies, animal data, and human cell cultures in Chapters Five and Six. I focus here on the specific pathways by which these hormone mimics leave their signatures within the cell.

But first, a bit of background on estrogen itself. Manufactured from cholesterol by a woman's ovaries each month, estrogen circulates in the blood, passes freely in and out of all organs and tissues, is eventually metabolized by specific enzymes, and, with the help of the liver, is eliminated from the body through the gut. Most cells are completely unaffected by all this activity. The cells of certain tissues, however, contain receptors that latch on to estrogen molecules as they float through. The estrogen-receptor complex then goes to work inside the nucleus. Some genes are activated, while others are switched off. Different messages are sent out from the nucleus and, hence, different proteins manufactured. For tissues possessing estrogen receptors, the net effect of these various alterations is an increase in cell proliferation. The cells of the vagina, the uterus, and the breast all contain large numbers of estrogen receptors. In the presence of estrogen, they divide. Ovulation, breast development, menstruation, and pregnancy are all made possible by estrogen's actions.

Estrogen comes in several chemical configurations, each with its own name. By far, the most potent one is estradiol. Its particular structure allows it easy passage from blood into surrounding cells. To regulate this movement, estradiol is not permitted to travel about unescorted. Instead, most estradiol molecules are attached to serum proteins that slow down their entry into target tissues and thereby blunt their dramatic effects.

Like estradiol, xenoestrogens slip from blood serum into the interior of cells, attach themselves to estrogen receptors, and, by tinkering with particular genes, elicit growth-promoting changes within target tissues. The ability of certain synthetic chemicals to mimic estrogen in these regards has been known for some time, but until recently, many researchers had assumed that any breast cancer risk created by this sort of mischief paled in comparison to the sovereign power of a woman's own hormones. This assumption was based on several observations. First, few synthetic chemicals closely resemble the ornately designed estrogen molecule, and estrogen is the key that must fit into the receptor's lock in order to ignite the whole process. Second, assays show that foreign estrogens are much less potent than naturally occurring estradiol. Indeed, most are thousands, even millions, of times weaker. Third, xenoestrogens exist in much lower concentrations in the body than naturally occurring estrogens, which surge to impressive levels during the first half of a woman's menstrual cycle. Also, many of the plants we eat, such as soy, contain naturally occurring plant estrogens, which are far more commonly encountered by our cells than their synthetic counterparts, such as pesticide residues. In short, xenoestrogens have been presumed rare, ineffective, and dilute.

Several recent findings have cast doubt on such reassuring suppositions. It turns out, for example, that close physical resemblance is not required for successful estrogen impersonation. As a lock, the estrogen receptor accepts many keys, some widely divergent in shape and size. Organic compounds that look nothing like estradiol—from pesticides to plastics to surfactants—can possess estrogenic properties. Xenoestrogens are far more common than anyone had imagined.

Furthermore, many artificial estrogens compensate for their low numbers through longevity and enhanced availability. As we have seen, synthetic xenoestrogens are not easily metabolized and excreted. They linger, sometimes for decades. Recent studies have also shown that some xenoestrogens, including DDT, are not as tightly bound to blood proteins as estradiol. They can, therefore, enter target cells more quickly and at lower concentrations; they are more available.

Xenoestrogens not only mimic natural estrogens directly but also can indirectly enhance their effects. Some, for example, appear to stimulate the manufacture of more estrogen receptors. More receptors means an amplified response to estradiol. Still others influence how estradiol is metabolized and eliminated from the body. This second effect has been the subject of several recent studies led by the biochemical endocrinologist Leon Bradlow at the Strang Cornell Cancer Research Center in New York and his collaborator Devra Davis.

As explained by Bradlow, estradiol molecules can be broken apart by metabolic enzymes in one of two ways. The first one alters carbon atom number 2. The second alters carbon atom number 16. Which of these two pathways estradiol takes turns out to be critical. The 16-metabolite is still estrogenic; it is easily reabsorbed across the gut and is capable of binding to estrogen receptors just like its parent, estradiol. More menacingly, the 16-metabolite can directly damage DNA. It is believed capable of both initiating and promoting breast cancer. Indeed, many researchers consider the level of this metabolite a potential marker for breast cancer risk. In contrast, the 2-metabolite is minimally estrogenic and nontoxic to DNA, and it may even protect the breast against cancerous changes. According to Bradlow and his colleagues, a low 16-to-2 ratio is desirable.

Unfortunately, many contaminants push the ratio in the other direction. In cultured cells, the pesticides DDT, atrazine, and endosulfan—as well as benzene and certain PCBs—all skew the balance away from 2 and toward the 16 pathway. In essence, these environmental contaminants turn the natural hormone estrogen into a weapon that is aimed at the breasts it caused to grow in the first place.

I had bladder cancer as a young adult. If I tell people this fact, they usually shake their heads. If I go on to mention that cancer runs in my family, they usually start to nod. *She is from one of those cancer families*, I can almost hear them thinking. Sometimes, I just leave it at that. But, if I am up for blank stares, I add that I am adopted and go on to describe a study of cancer among adoptees that found correlations within their adoptive families but not within their biological ones. ("Deaths of adoptive parents from cancer before the age of 50 increased the rate of mortality from cancer fivefold among the adoptees. . . . Deaths of biological parents from cancer had no detectable effect on the rate of mortality from cancer among the adoptees.") At this point, most people become very quiet.

These silences remind me how unfamiliar many of us are with the notion that families share environments as well as chromosomes or with the concept that our genes work in communion with substances streaming in from the larger, ecological world. What runs in families does not necessarily run in blood. And our genes are less an inherited set of teacups enclosed in a cellular china cabinet than they are plates used in a busy diner. Cracks, chips, and scrapes accumulate. Accidents happen.

My Aunt Jean died of bladder cancer. Raymond and Violet both died of colon cancer. LeRoy is currently under treatment. These are my father's relatives. About Uncle Ray I remember very little, except that he, along with my dad, was one of the less loud of the concrete-pouring, brick-laying Steingraber brothers. Aunt Jean laughed a lot and once asked me to draw a pig so she could tape it to her refrigerator door. Red-haired Aunt Vi cooked magnificent dinners, was partial to wearing pink, and was married to a man truly untempted by silence. Together, she once remarked, the two of them sure knew how to enjoy themselves. Her widowed husband, my Uncle Ed, is now being treated aggressively for prostate cancer. Nonetheless, at last report, he was busy building a shrine to his wife out in the backyard. When it comes to expressions of grief, my father's side of the family tends toward large-scale construction projects.

The man who was to be my brother-in-law was stricken with intestinal cancer at the age of twenty-one. He cleaned out chemical

drums for a living. Three years before Jeff's diagnosis, I was diagnosed with bladder cancer, and three years before my diagnosis, my mother learned she had metastatic breast cancer. That she is still alive today is a topic of considerable wonder among her doctors. Mom is matter-of-fact about this, although she will, if prompted, shyly point out that she has outlived her oncologist and three of her other doctors, two of whom died of cancer.

My mother was first diagnosed in 1974, a year that is considered an anomaly in the annals of breast cancer. Graphs displaying U.S. breast cancer incidence rates across the decades show a gently rising line that suddenly zooms skyward, falls back, then continues its slow ascent. The story behind the blip of '74 has been deemed a textbook lesson in statistical artifacts.

In this year, First Lady Betty Ford and Second Lady Happy Rockefeller both underwent mastectomies. The words *breast cancer* entered public conversation. Women who might otherwise have delayed routine checkups or who were hesitant to seek medical opinion about a lump were propelled into doctors' offices. The result was that a lot of women were diagnosed with breast cancer within a short period of time, my mother among them.

When I, at age fifteen, inquired why my mother was in the hospital, the answer was "Because she has what Mrs. Ford has." When my mother, at age forty-four, questioned whether a radical mastectomy was necessary, she was told, "If it's good enough for Happy, it's good enough for you."

Back at home, a new fixture appeared on the dresser in my parents' bedroom: a bald Styrofoam head. It had come with the wig—which it dutifully wore when my mother wasn't—and it remains in my mind as the most vivid image of her illness. Its features were peculiar. It lacked ears. Its closed eyes and too-small nose were half formed, as though worn smooth by water. It wore the serene, expressionless face of someone drowned or unborn.

Not that the rest of us were any more demonstrative. My father vanished into his workshop. I became the heroine of homework and long walks. My twelve-year-old sister wrote protracted, angry manifestos—and then tore them up into small fragments. These were secretly reassembled and read by our mother, who steadfastly believed that an atmosphere of normalcy was health promoting.

Some twenty years later, Mom and I sit out on my Boston balcony, drinking iced tea. I describe some medical decisions that I am facing. She provides calm, thoughtful advice—as I knew she would. Finally, I ask her about all those years of chemotherapy, surgeries, and bad news. Did she feel supported during that time?

She looks away. "Too much sympathy would have weakened me." It isn't exactly an answer to my question, and I want to ask what she means. But I don't.

My sister and I sit out in her backyard, drinking beer and watching her boys chase fireflies. I realize—as though for the first time—that she had seen her mother, sister, and fiancé all in treatment for cancer by the time she was old enough for college. I ask her about this.

"It just kept happening." Julie says, ticking off the chronology of diagnoses we both have memorized. "You and I quit talking for a while. Dad and Mom quit talking. We all got very quiet."

"That's how I remember it, too. Everybody lost their vocabulary." I want to ask her about Jeff's death and about the Styrofoam head. But I don't.

TWELVE

ecological roots

In 1983, I took the train home to Illinois for the holidays—and an appointment at the hospital.

The scheduling of cancer checkups is always an elaborate decision. The calendar date must sound auspicious. Monday or Tuesday appointments are best; otherwise, one risks waiting through the weekend for the results of a laggard lab test or delayed radiology report. It's also best if these appointments fall within a hectic, deadline-filled month so that frenetic activity can preclude fretfulness. During the years I was a graduate student, this meant the ends of semesters, which explains why some half dozen Christmas carols now remind me of outpatient waiting rooms. This particular appointment was destined to turn out fine. What I remember most clearly is my journey there by train.

Something about the landscape changes abruptly between

northern and central Illinois. I am not sure what it is exactly, but it happens right around the little towns of Wilmington and Dwight. The horizon recedes, and the sky becomes larger. Distances increase, as though all objects are moving slowly away from each other. Lines become more sharply drawn. These changes always make me restless and, when driving, drive faster. But since I am in a train, I close the book I am reading and begin impatiently straightening the pages of a newspaper strewn over the adjacent seat.

That is when my eye catches the headline of a back-page article: SCIENTISTS IDENTIFY GENE RESPONSIBLE FOR HUMAN BLADDER CANCER. Pulling the newspaper onto my lap, I stare out the window and become very still. It is only early evening, but the fields are already dark, a patchwork of lights quilted over and across them. They have always soothed me. I look for signs of snow. There are none. Finally, I read the article.

Researchers at the Massachusetts Institute of Technology, it seems, had extracted DNA from the cells of a human bladder tumor and used it to transform normal mouse cells into cancerous ones. Through this process, they located the segment of DNA responsible for the transformation. And by comparing this segment to its unmutated form in noncancerous human cells, they were able to pinpoint the exact alteration that had caused a respectable gene to go bad.

In this case, the mutation turned out to be a substitution of one unit of genetic material for another in a single rung of the DNA ladder. Namely, at some point during DNA replication, a double-ringed base called guanine was swapped for the single-ringed thymine. Like a typographical error in which one letter replaces another—*snow* instead of *show*, *block* instead of *black*—the message sent out by this gene was utterly changed. Instead of instructing the cell to manufacture the amino acid glycine, the altered gene now specified for valine. (Nine years later, other researchers would determine that this substitution alters the structure of proteins involved in signal transduction—the crucial line of communication between the cell membrane and the nucleus that helps coordinate cell division.)

Guanine instead of thymine. Valine instead of glycine. I look away again—this time at my face superimposed over the landscape by the window's mirror. If, in fact, this mutation was involved in my

cancer, when did it happen? Where was I? Why had it escaped repair? I had been betrayed. But by what?

Thirteen years later, I possess a bulging file of scientific articles documenting an array of genetic changes involved in bladder cancer. Besides the oncogene just described, two tumor supressor genes, p15 and p16, have also been discovered to play a role. Their deletion is a common event in transitional cell carcinoma, the kind of cancer I had. Mutations of the famous p53 tumor suppressor gene, with guest-star appearances in so many different cancers, have been detected in more than half of invasive bladder tumors. Also associated with transitional cell carcinomas are surplus numbers of growth factor receptors. Their overexpression has been linked to the kinds of gross genetic injuries that appear near the end of the malignant process.

The nature of the transaction between these various genes and certain bladder carcinogens has likewise been worked out in the years since a newspaper article introduced me to the then new concept of oncogenes. Consider, for example, that redoubtable class of bladder carcinogens called aromatic amines—present as contaminants in cigarette smoke; added to rubber during vulcanization; formulated as dyes for cloth, leather, and paper; used in printing and color photography; and featured in the manufacture of certain pharmaceuticals and pesticides. Aniline, benzidine, naphthylamine, and *o*-toluidine are all members of this group. The first reports of excessive bladder cancers among workers in the aniline dye industry were published in 1895. (Recall also Wilhelm Hueper's dogs, described in Chapter Six.) More than a century later, we now know that anilines and other aromatic amines ply their wickedness by forming DNA adducts in the cells of the tissues lining the bladder, where they arrive as contaminants of urine.

We also now know that aromatic amines are gradually detoxified by the body through a process called acetylation. Like all such processes, it is carried out by a special group of detoxifying enzymes whose actions are controlled and modified by a number of genes. People who are slow acetylators have low levels of these enzymes and are at greater risk of bladder cancer from exposure to aromatic amines. Members of this population can be readily identified because

they bear significantly higher burdens of adducts than fast acetylators at the same exposure levels. These genetically suspectible individuals hardly constitute a tiny minority: more than half of Americans and Europeans are estimated to be slow acetylators.

Very likely, I am one. You may be one, too.

We know a lot about bladder cancer. Bladder carcinogens were among the earliest human carcinogens ever identified, and one of the first human oncogenes ever decoded was isolated from some unlucky fellow's bladder tumor. More than most malignancies, bladder cancer has provided researchers with a picture of the sequential genetic changes that unfold from initiation through promotion to progression, from precursor lesions to increasingly more aggressive tumors.

Sadly, all this knowledge about genetic mutations, inherited risk factors, and enzymatic mechanisms has not translated into an effective campaign to prevent the disease. The fact remains that the overall incidence rate of bladder cancer increased 10 percent between 1973 and 1991. Increases are especially dramatic among African Americans: among black men, bladder cancer incidence has risen 28 percent since 1973, and among black women, 34 percent.

Somewhat less than half of all bladder cancers among men and one-third of all cases among women are thought to be attributable to cigarette smoking, which is the single largest known risk factor for this disease. As we saw in Chapter Three, the lung cancer rate among white men in the United States is now falling, reflecting—at long last—the significant decline in smoking among members of this demographic group. If a parallel decline in bladder cancer incidence among white men should follow, we would have reason to finger tobacco as one possible explanation for the 1973–1991 increase. So far, it has not, but perhaps bladder cancer simply has a longer lag time than lung cancer. In the meantime, the question still remains: What is causing bladder cancer in the rest of us, the majority of bladder cancer patients for whom tobacco is not a factor?

I also possess another bulging file of scientific articles. These concern the ongoing presence of known and suspected bladder carcinogens in rivers, groundwater, dump sites, and indoor air. For example, industries reporting to the Toxics Release Inventory disclosed

environmental releases of the aromatic amine *o*-toluidine that totaled 14,625 pounds in 1992 alone. Detected also in effluent from refineries and other manufacturing plants, *o*-toluidine exists as residues in the dyes of commercial textiles, which may, according to the *Seventh Annual Report on Carcinogens*, expose members of the general public who are consumers of these goods: "The presence of *o*-toluidine, even as a trace contaminant, would be a cause for concern." A 1996 study investigated a sixfold excess of bladder cancer among workers exposed years before to *o*-toluidine and aniline in the rubber chemicals department of a manufacturing plant in upstate New York. Levels of these contaminants are now well within their legal workplace limits, and yet blood and urine collected from current employees were found to contain substantial numbers of DNA adducts and detectable levels of *o*-toluidine and aniline. Another recent investigation revealed an eightfold excess of bladder cancer among workers employed in a Connecticut pharmaceuticals plant that manufactured a variety of aromatic amines. This study was reported as having national implications because the main suspect, dichlorobenzidine, has been widely used throughout the United States.

What my various file folders do *not* contain is a considered evaluation of all known and suspected bladder carcinogens—their sources, their possible interactions with each other, and our various routes of exposure to them. As we have seen, trihalomethanes—those unwanted by-products of water chlorination—have been linked to bladder cancer, as has the dry-cleaning solvent and sometime-contaminant of drinking-water pipes, tetrachloroethylene. I possess individual reports on each of these topics. What I do not have is a comprehensive description of how all these substances behave in combination. What are the risks of multiple trace exposures? What happens when we drink trihalomethanes, absorb aromatic amines, and inhale tetrachloroethylene? Furthermore, what is the ecological fate of these substances once they are released into the environment? What happens when dyed cloth, colored paper, and leather goods are laundered, landfilled, or incinerated? And why—almost a century after some of them were so identified—do powerful bladder carcinogens such as amine dyes continue to be manufactured, imported, used, and released into the environment in the first place? However

improved the record of effort to regulate them, why have safer substitutes not replaced them all? These questions remain, to my knowledge, largely unaddressed by the cancer research community.

Several obstacles, I believe, prevent us from addressing cancer's environmental roots. An obsession with genes and heredity is one.

Cancer research currently directs considerable attention to the study of inherited cancers. Most immediately, this approach facilitates the development of genetic testing, which attempts to predict an individual's risk of succumbing to cancer, based on the presence or absence of certain genetic alterations. These efforts may also reveal which genes are common targets of acquired mutation in the general population. (Hereditary mutations are present at the time of conception, and they are carried in the DNA of all body cells; acquired mutations, which accumulate over an individual's lifetime, are passed only to the direct descendents of the cells in which they arise.)

Hereditary cancers, however, are the rare exception. Collectively, fewer than 10 percent of all malignancies are thought to involve inherited mutations. Between 1 and 5 percent of colon cancers, for example, are of the hereditary variety, and only about 15 percent exhibit any sort of familial component. The remaining 85 percent of colon cancers are officially classified as "sporadic," which, confesses one prominent researcher, "is a fancy medical term for 'we don't know what the hell causes it.'" Breast cancer also shows little connection to heredity (probably between 5 and 10 percent). Finding "cancer genes" is not going to prevent the vast majority of cancers that develop.

Moreover, even when rare, inherited mutations play a role in the development of a particular cancer, environmental influences are inescapably involved as well. Genetic risks are not exclusive of environmental risks. Indeed, the direct consequence of some of these damaging mutations is that people become even more sensitive to environmental carcinogens. In the case of hereditary colon cancer, for example, what is passed down the generations is a faulty DNA repair gene. Its human heirs are thereby rendered less capable of coping

with environmental assaults on their genes or repairing the spontaneous mistakes that occur during normal cell division. These individuals thus become more likely to accumulate the series of *acquired* mutations needed for the formation of a colon tumor.

Cancer incidence rates are not rising because we are suddenly sprouting new cancer genes. Rare, heritable genes that predispose their hosts to cancer by creating special susceptibilities to the effects of carcinogens have undoubtedly been with us for a long time. The ill effects of some of these genes might well be diminished by lowering the burden of environmental carcinogens to which we are all exposed. In a world free of aromatic amines, for example, being born a slow acetylator would be a trivial issue, not a matter of grave consequence. The inheritance of a defective carcinogen-detoxifying gene would matter less in a culture that did not tolerate carcinogens in air, food, and water. By contrast, we cannot change our ancestors. Shining the spotlight on inheritance focuses us on the one piece of the puzzle we can do absolutely nothing about.

Risks of lifestyle are also not independent of environmental risks. And yet public education campaigns about cancer consistently accent the former and ignore the latter. I collect the colorful pamphlets on cancer that are made available in hospitals, clinics, and waiting rooms. When I was teaching introductory biology and also spending many hours in doctors' offices, I began to compare the descriptions of cancer in the tracts displayed in the skinny, silver racks above the magazines with the chapter on cancer provided in my students' textbook. Here are some of my findings.

On the topic of how many people get cancer, a pink and blue brochure published by the U.S. Department of Health and Human Services offers the following:

> Good News: Everyone does not get cancer. 2 out of 3 Americans never will get it.

Whereas, according to *Human Genetics: A Modern Synthesis:*

> One of three Americans will develop some form of cancer in his or her lifetime, and one in five will die from it.

(Since these materials were published, the proportion of Americans contracting cancer has risen from 30 to 40 percent.)

On the topic of what causes cancer, the brochure states:

> In the past few years, scientists have identified many causes of cancer. Today it is known that about 80% of cancer cases are tied to the way people live their lives.

Whereas the textbook contends:

> As much as 90 percent of all forms of cancer is attributable to specific environmental factors.

In regard to prevention, the brochure emphasizes individual choice and responsibility:

> You can control many of the factors that cause cancer. This means you can help protect yourself from the possibility of getting cancer. You can decide how you're going to live your life—which habits you will keep and which ones you will change.

The genetics book presents a somewhat different vision:

> Because exposure to these environmental factors can, in principle, be controlled, most cancers could be prevented. . . . Reducing or eliminating exposures to environmental carcinogens would dramatically reduce the prevalence of cancer in the United States.

The textbook goes on to identify some of these carcinogens, the routes of exposure, and the types of cancer that result. In contrast, the brochure emphasizes the importance of personal habits, such as sunbathing, that raise one's risk of contracting cancer. Thus, in my students' textbook, vinyl chloride is identifed as a carcinogen to which PVC manufacturers are exposed, whereas in the brochure, occupations that involve working with certain chemicals are called a risk factor. The textbook declares that "radiation is a carcinogen." The brochure advises us to "avoid unnecessary X-rays." Both emphasize the role of diet and tobacco.

In its ardent focus on lifestyle, the Good News brochure is typical of the educational pamphlets in my collection. By emphasizing personal habits rather than carcinogens, they frame the cause of the disease as a problem of *behavior* rather than as a problem of *exposure* to disease-causing agents. At its best, this perspective can offer us practical guidance and the reassurance that there are actions we as individuals can take to protect ourselves. (Not smoking, rightfully so, tops this list.) At its worst, the lifestyle approach to cancer is dismissive of hazards that lie beyond personal choice. A narrow focus on lifestyle—like a narrow focus on genetic mechanisms—obscures cancer's environmental roots. It presumes that the ongoing contamination of our air, food, and water is an immutable fact of the human condition to which we must accommodate ourselves. When we are urged to "avoid carcinogens in the environment and workplace," this advice begs the question. Why must there be known carcinogens in our environment and at our job sites?

The experience of the anthropologist Martha Balshem is revealing here. In the late 1980s, Balshem served as a health educator in an industrial, working-class community near Philadelphia where cancer rates were discovered to be unsually high. In response, the cancer control program of which she was part launched a public outreach campaign urging residents to adopt healthier lifestyles. The residents themselves suspected environmental causes and reported to the educational team that many neighborhood dogs were also afflicted with cancer: Did their pets have faulty personal habits as well? In her book *Cancer in the Community* Balshem recalls:

> As representatives of the cancer center, we sought to deflect this concern and stressed lifestyle changes to reduce cancer risk. Privately, we acknowledged our own feelings or suspicions that the profound pollution we observed in the community was somehow linked to the high cancer rates. We said to each other that this did not present us with a moral dilemma, because in any case, people were well advised to quit smoking, improve their diets, and get regular cancer tests.

In the end, Balshem came to believe the lesson she was transmitting—"accept authority and accept blame"—was the wrong one.

Cancer is certainly not the first disease to inspire this kind of message. In 1832, at the height of an epidemic, the New York City medical council announced that cholera's usual victims were those who were imprudent, intemperate, or prone to injury by the consumption of improper medicines. Lists of cholera prevention tips were posted publicly. Their advice ranged from avoiding drafts and crude vegetables to abstaining from alcohol. Maintaining "regular habits" was also said to be protective. Decades later, improvements in public sanitation (as mentioned in Chapter Eight) would bring cholera under control, and the pathogen responsible for the disease would finally be isolated by the bacteriologist Robert Koch in 1883. Of course, the behavioral changes urged by the 1832 handbills were not all without merit: uncooked produce, as it turned out, was an important route of exposure, but it was a fecal-borne bacteria—and not a salad-eating lifestyle—that was the cause.

The orthodoxy of lifestyle today finds its full expression in the public educational literature on breast cancer. In scores of cheerful pamphlets, women are exhorted to exercise, lower the fat in their diets, perform breast self-examinations, ponder their family history, and receive regular mammograms. "Delayed childbirth" (after age twenty) is frequently mentioned as a risk factor. (I have never seen "prompt childbirth" in the accompanying list of cancer prevention tips—undoubtedly because such advice would be tantamount to advocating teenage pregnancy.)

All by itself, a lifestyle approach to preventing breast cancer is inadequate. First, the majority of breast cancers cannot be explained by lifestyle factors, including reproductive history. We need to look elsewhere for the causes of these cancers. Second, mammography and breast self-examinations are tools of cancer detection, not acts of prevention. The popular refrain "Early detection is your best prevention!" is a non sequitur: Detecting cancer, no matter how early, negates the possibility of preventing cancer. At best, early detection may make cancer less fatal, allowing us, as the epidemiologist Robert Millikan puts it, "to live in a toxic soup without breasts or prostates, et cetera."

Finally, the adage that high-fat Western diets are the cause of breast cancer has not yet been supported by data. Dietary fat has long been a centerpiece of study in the investigation of breast cancer risk.

And yet, several long-term, heavily funded studies have indicated that dietary fat is unlikely to play a major role by itself. Rather than continuing to focus singlemindedly on the absolute quantity of fat consumed, several researchers have called for a more refined, ecological approach to diet. Two obvious starting points would be to assess the link between breast cancer and diets high in animal fat and to launch a definitive investigation into the extent to which various kinds of fats are contaminated by carcinogens. We already know with certainty that animal-based foods are our main route of exposure to organochlorine pesticides and dioxins. It's time to look at the whole picture.

Even reproductive choices have environmental implications. Breasts, for example, do not complete their development until the last months of a woman's first full-term pregnancy. During this time, the latticework of mammary ducts and lobules differentiate into fully functioning secretory cells. This process of specialization permanently slows the rate of mitosis, dampens the response to growth-promoting estrogens, and renders DNA less vulnerable to damage. According to the leading hypothesis, a full-term pregnancy early in life protects against breast cancer precisely because it reduces a woman's vulnerability to carcinogens and other cancer promoters, such as estrogens.

One of the principle proponents of this hypothesis, the Harvard epidemiologist Nancy Krieger, has urged its further testing. She has also urged a redirection of breast cancer research toward environmental questions. Investigators have repeatedly confirmed that reproductive history contributes to breast cancer risk. We need to know now, Krieger argues, whether women with similiar reproductive histories but divergent exposure to carcinogens have marked differences in breast cancer incidence. This need is made urgent by the results of animal studies showing that exposure to certain organochlorines hastens the onset of puberty. As we have noted, early first menstruation—along with late parenthood—is considered a risk factor for breast cancer in women.

Within the scientific community, grand arguments have ensued from the attempt to classify and quantify cancer deaths due to specific causes. Traditionally, the final result of this task takes the visual form of a great cancer pie sliced to depict the relative importance of different risk factors. "Smoking" is always a big wedge, monopolizing about 30 percent of the circle. "Diet" is also a sizable helping. Depending on who's doing the apportioning, an array of other lifestyle factors—"alcohol," "reproductive and sexual behavior," and "sedentary way of life"—divvy up the remainder, along with "occupation" and "pollution."

The quarreling begins immediately. How do we account for malignancies, such as certain liver cancers, to which both drinking and job hazards contribute? Or lung and bladder cancers where both job hazards and smoking conspire? Should the effects of pesticides be tallied under "pollution" or under "diet"? What about pollution's indirect effects—such as hormonal disruption, inhibition of apoptosis (cell death), and immune system suppression—that act to augment the dangers of risk factors across the board? What about formaldehyde, which seems to bind with DNA in such a way that it prevents repair of damage induced by ionizing radiation, possibly raising the cancer risk from medical X-rays?

Interactions between risk factors aside, how can the environment's death toll be calculated at all when the vast majority of industrial chemicals in commerce have never been tested for their ability to cause cancer?

The futility of what the cancer historian Robert Proctor calls "the percentages game" has not deterred public health agencies from using this kind of simplistic accounting to formulate cancer control policies and educational programs. Lifestyle is the bull's-eye of cancer prevention efforts, while targeting of environmental factors, perceived as a small contribution to the cancer problem, is seen as inefficient. Moreover—the rationale continues—not enough is known about environmental risks to make specific recommendations. (Incomplete and inconsistent evidence about the role of dietary fat in contributing to breast cancer is, on the other hand, not an obstacle to advising women to change their diets.)

In my own home state, a recent county-by-county cancer report reproduced an old cancer pie chart, published originally in 1981, that relegated environmental factors to a single, tiny slice and depicted tobacco and diet as major risk factors. The report concluded, "Many persons could reduce their chances of developing or dying from cancer by adopting healthier lifestyles and by visiting their physicians

regularly for cancer-related checkups." The fact that Illinois is a leading producer of hazardous waste, a heavy user of pesticides, and home to an above-average number of Superfund sites is neither mentioned nor considered. No attempt is made in this report to correlate cancer statistics with Toxics Release Inventory data. No attempt is made in this report to determine whether cancer might follow industrial river valleys, rise in areas of high pesticide use, or cluster around contaminated wells.

Lifestyle and the environment are *not* independent categories that can be untwisted from each other: to talk about one is to talk about the other. A discussion about dietary habits is necessarily also a discussion about the food chain. To converse about childbirth and breast cancer is also to converse about changing susceptibility to carcinogens in the breast. And to advise those of us at risk for bladder cancer to "void frequently" is to acknowledge the presence of carcinogens in the fluids passing through our bodies.

During the last year of her life, Rachel Carson discussed before a U.S. Senate subcommittee her emerging ideas about the relationship between environmental contamination and human rights. The problems addressed in *Silent Spring*, she asserted, were merely one piece of a larger story—namely, the threat to human health created by reckless pollution of the living world. Abetting this hidden menace was a failure to inform common citizens about the senseless and frightening dangers they were being asked, without their consent, to endure. In *Silent Spring*, Carson had predicted that full knowledge of this situation would lead us to reject the counsel of those who claim there is simply no choice but to go on filling the world with poisons. Now she urged recognition of an individual's right to know about poisons introduced into one's environment by others and the right to protection against them. These ideas are Carson's final legacy.

The process of exploration that results from asserting our right to know about carcinogens in our environment is a different journey for every person who undertakes it. For all of us, however, I believe it

necessarily entails a three-part inquiry. Like the Dickens character Ebenezer Scrooge, we must first look back into our past, then reassess our present situation, and finally summon the courage to imagine an alternative future.

We begin retrospectively for two reasons. First, we carry in our bodies many carcinogens that are no longer produced and used domesticially but which linger in the environment and in human tissue. Appreciating how, even today, we remain in contact with banned chemicals such as PCBs and DDT requires a historical understanding. Second, because cancer is a multicausal disease that unfolds over a period of decades, exposures during young adulthood, adolescence, childhood—and even prior to birth—are relevant to our present cancer risks. We need to find out what pesticides were sprayed in our neighborhoods and what sorts of household chemicals were stored under our parents' kitchen sink. Reminiscing with neighbors, family members, and elders in the community where one grew up can be an eye-opening first step.

This part of the journey is, in essence, a search for our ecological roots. Just as awareness of our genealogical roots offers us a sense of heritage and cultural identity, our ecological roots provide a particular appreciation of who we are biologically. It means asking questions about the physical environment we have grown up within and whose molecules are woven together with the strands of DNA inherited from our genetic ancestors. After all, except for the original blueprint of our chromosomes, all the material that is us—from bone to blood to breast tissue—has come to us from the environment.

Going in search of our ecological roots has both intimate and far-flung dimensions. It means learning about the sources of our drinking water (past and present), about the prevailing winds that blow through our communities, and about the agricultural system that provides us food. It involves visiting grainfields, as well as cattle lots, orchards, pastures, and dairy farms. It demands curiosity about how our apartment buildings are exterminated, clothing cleaned, and golf courses maintained. It means asserting our right to know about any and all toxic ingredients in products such as household cleaners, paints, and cosmetics. It requires a determination to find out where the underground storage tanks are located, how the land was used be-

fore the subdivision was built over it, what is being sprayed along the roadsides and rights-of-way, and what exactly goes on behind that barbed-wire fence at the end of the street.

Acquiring a copy of the Toxics Release Inventory for one's home county, as well as a list of local hazardous waste sites, is a simple place to begin (see the Afterword that follows). Such information is not available for the years prior to 1987 and so tells us less about our formative years than it does about the present decade. Nevertheless, these documents often contain clues to the past as well: the toxic chemicals loitering around an abandoned Superfund site, for example, can reveal what kinds of activities occurred there decades earlier.

In full possession of our ecological roots, we can begin to survey our present situation. This requires a human rights approach. Such an approach recognizes that the current system of regulating the use, release, and disposal of known and suspected carcinogens—rather than preventing their generation in the first place—is intolerable. So is the decision to allow untested chemicals free access to our bodies, until which time they are finally assessed for carcinogenic properties. Both practices show reckless disregard for human life.

A human rights approach would also recognize that we do not all bear equal risks when carcinogens are allowed to circulate within our environment. Workers who manufacture carcinogens are exposed to higher levels, as are those who live near the chemical graveyards that serve as their final resting place. Moreover, people are not uniformly vulnerable to effects of environmental carcinogens. Individuals with genetic predispositions, infants whose detoxifying mechanisms are not yet fully developed, and those with significant prior exposures may all be affected more profoundly. Cancer may be a lottery, but we do not each of us hold equal chances of "winning." When carcinogens are deliberately or accidentally introduced into the environment, some number of vulnerable persons are consigned to death. The impossibility of tabulating an exact body count does not alter this fact. A human rights approach to cancer strives, nonetheless, to make these deaths visible.

Suppose we assume for a moment that the most conservative estimate concerning the proportion of cancer deaths due to environmental causes is absolutely accurate. This estimate, put forth by those

who dismiss environmental carcinogens as negligible, is 2 percent. Though others have placed this number far higher, let's assume for the sake of argument that this lowest value is absolutely correct. Two percent means that 10,940 people in the United States die each year from environmentally caused cancers. This is more than the number of women who die each year from hereditary breast cancer—an issue that has launched multi-million-dollar research initiatives. This is more than the number of children and teenagers killed each year by firearms—an issue that is considered a matter of national shame. It is more than three times the number of nonsmokers estimated to die each year of lung cancer caused by exposure to secondhand smoke—a problem so serious it warranted sweeping changes in laws governing air quality in public spaces. It is the annual equivalent of wiping out a small city. It is thirty funerals every day.

None of these 10,940 Americans will die quick, painless deaths. They will be amputated, irradiated, and dosed with chemotherapy. They will expire privately in hospitals and hospices and be buried quietly. Photographs of their bodies will not appear in newspapers. We will not know who most of them are. Their anonymity, however, does not moderate this violence. These deaths are a form of homicide.

A human rights approach to cancer would also speak out against other deprivations besides gross loss of life. The dispossession of Chattanooga Creek is one example. In 1993, the U.S. Agency for Toxic Substances and Disease Registry dispatched a group of representatives to Chattanooga, Tennessee, expressly to teach schoolchildren to stay away from the local creek, which happens to be surrounded by no less than forty-two hazardous waste sites. In the agency's words: "Training workshops highlighted the dangers of fishing, swimming, and playing in the creek and of eating fish from the creek. . . . Children were encouraged to take the information home and share it with their parents."

No one can quantify what the loss of a creek means to a child in Tennessee or measure the grief of parents who must forbid their son or daughter from exploring along its banks. But I think we can say with assurance that the transformation of a popular swimming hole into a cancer hazard and child's play into a cancer risk factor is a terrible diminishment of our humanity. And we can say that the agency's

gesture of educational responsibility is indicative of a vast national *ir*-responsibility.

According to the most recent tally, forty possible carcinogens appear in drinking water, sixty are released by industry into ambient air, and sixty-six are routinely sprayed on food crops as pesticides. Whatever our past exposures, this is our current situation.

After having carefully appraised the risks and losses that we have endured by tolerating it, we can begin to imagine a future in which our right to an environment free of such substances is respected. It is unlikely that we will ever rid our environment of all chemical carcinogens. However, as Rachel Carson herself observed, the elimination of a great number of them would reduce the carcinogenic burden we all bear and thus would prevent considerable suffering and loss of human life. Three key principles can assist us in this effort.

One is the idea that public and private interests should act to prevent harm before it occurs. This is known as the *precautionary principle*, and it dictates that indication of harm, rather than proof of harm, should be the trigger for action—especially if delay may cause irreparable damage. Central to the precautionary principle is the recognition that we have an obligation to protect human life. Our current methods of regulation, by contrast, appear governed by what some frustrated policymakers have called the dead body approach: wait until damage is proven before action is taken. It is a system tantamount to running an uncontrolled experiment using human subjects.

Closely related to the precautionary principle is the *principle of reverse onus*. According to this edict, it is safety, rather than harm, that should necessitate demonstration. This reversal essentially shifts the burden of proof off the shoulders of the public and onto those who produce, import, or use the substance in question. The principle of reverse onus requires that those who seek to introduce chemicals into our environment first show that what they propose to do is almost certainly *not* going to hurt anyone. This is already the standard we uphold for pharmaceuticals, and yet for most industrial chemicals, no firm requirement for advance demonstration of safety exists. But chemicals are not citizens. They should not be presumed innocent unless proven guilty, especially when a verdict of guilt requires some of us to sicken and die in order to demonstrate the necessary evidence.

Finally, all activities with potential public health consequences should be guided by the *principle of the least toxic alternative*, which presumes that toxic substances will not be used as long as there is another way of accomplishing the task. This means choosing the least harmful way of solving problems—whether it be ridding fields of weeds, school cafeterias of cockroaches, dogs of fleas, woolens of stains, or drinking water of pathogens. Biologist Mary O'Brien advocates a system of alternatives assessment in which facilities regularly evaluate the availability of alternatives to the use and release of toxic chemicals. Any departure from zero should be preceded by a finding of necessity. These efforts, in turn, should be coordinated with active attempts to develop and make available affordable, nontoxic alternatives for currently toxic processes and with systems of support for those making the transition—whether farmer, corner dry-cleaner, hospital, or machine shop. Receiving the highest priority for transformation should be all processes that generate dioxin or require the use or release of any known human carcinogen such as benzene and vinyl chloride.

The principle of the least toxic alternative would move us away from protracted, unwinnable debates over how to quantify the cancer risks from each individual carcinogen released into the environment and where to set legal maximum limits for their presence in air, food, water, workplace, and consumer goods. As O'Brien observed, "Our society proceeds on the assumption that toxic substances *will* be used and the only question is how much. Under the current system, toxic chemicals are used, discharged, incinerated, and buried without ever requiring a finding that these activities are necessary." The principle of the least toxic alternative looks toward the day when the availability of safer choices makes the deliberate and routine release of chemical carcinogens into the environment as unthinkable as the practice of slavery.

Sitting at my desk in my Boston apartment, I am skimming through a journal article about hormone disruption in young female rats. The study is unusual because the animals were exposed not to a single chemical but to a real-life, low-level mixture of substances derived

from the dust, soil, and air from a dioxin-contaminated landfill site. After only two days, the test animals exhibited abnormal changes in their livers, reproductive organs, and thyroid glands. Even rats exposed only to air from the landfill experienced significant changes in their development. These results indicate, the authors concluded, that current methods used for calculating health risks from chemical mixtures may "underestimate certain biological effects."

Flipping back to the beginning of the report, my eye catches on a familiar word: *Illinois.* The contaminated dust, soil, and air mixtures used in this study were collected from an old, inoperative landfill in Illinois.

Dust. Soil. Air. The year after my cancer diagnosis, I signed up for a field ecology class and learned to identify plant species in the rarest of rare Illinois habitats: the black soil prairie. Its remnants are almost completely confined to a few old pioneer gravesites. Hunkered down between headstones, I cupped the unfamiliar plants in my hands and tried to will into existence thousands of acres of these grasses and herbs, the sound of animals running, wildfires, birdsong.

As I became ever more enchanted with the Illinois prairie, I found that I was, nevertheless, unable to banish from my heart its remaining enemies—the nonnative invading species. Queen Anne's lace, ox-eye daisy, chicory, foxtail, goat's beard, teasel: all European immigrants, these are the familiar weeds of roadsides and fallow fields. My mother taught me the names of most of them. I am especially fond of teasel. It represents a special threat to prairie plants because mourners brought bouquets of it into the old prairie cemeteries, where it set seed and spread. In the winter, its stiff wands stand in the snow like pinecones on the ends of antennas. I keep a few stalks near my desk to remind me of home. I keep a scientific monograph of prairie plants on the shelf for the same reason.

After finishing the article on the health hazards of trace chemical mixtures, I look at the brown, spiny flowers and then out the window at the city I live in. Dust. Soil. Air. What I see are the contours of home.

USEFUL ADDRESSES

Australia

Friends of the Earth
Box 222
Fitzroy 3065
Victoria
Australia
email: foefitzroy@peg.apc.org
tel: 61 3 9419 8700

Canada

Friends of the Earth/Les Amis de la Terre
Suite 306
47 Clarence Street
Ottawa
Ontario K1N 9K1
email: foe@intranet.ca
tel: 1 613 241 0085

England, Wales and Northern Ireland

Friends of the Earth
26–28 Underwood Street
London
N1 7JQ
email: info@foe.co.uk
tel: 44 171 490 1555

Greenpeace UK
Canonbury Villas
London
N1 2PN
email: info@uk.greenpeace.org
tel: 44 171 865 8100

The Women's Environmental Network
87 Worship Street
London
EC2 2BE
email: wentrust@hotnail.com
tel: 44 171 247 3327

Ireland

Earthwatch
20 Grove Road
Rathmines
Dublin 6
email: foeeire@iol.ie
tel: 353 49 73773

New Zealand

Environment and Conservation Organisations of New Zealand
PO Box 11057
126 Vivian Street
Wellington
Aotearoa/New Zealand

Scotland

Friends of the Earth
Bonnington Mill
72 Newhaven Road
Edinburgh
EH6 5QG
email: foescotland@gn.apc.org
tel: 44 131 554 9977

JTEH	*Journal of Toxicology and Environmental Health*
MDPH	Massachusetts Department of Public Health
NCI	National Cancer Institute
NEJM	*New England Journal of Medicine*
NIH	National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
NRC	National Research Council
NRDC	National Resources Defense Council
OSHA	Occupational Safety and Health Administration
PDT	*Pekin Daily Times*
PJS	*Peoria Journal Star*
SSJR	*Springfield State Journal Register*
USDA	U.S. Department of Agriculture
USDHHS	U.S. Department of Health and Human Services
WHO	World Health Organization

Note: Organized by page number, the citations provided below represent the primary sources I consulted and are not intended to serve as a comprehensive review of the scientific literature. Some of the articles, monographs, and texts cited here are difficult to obtain, and some are highly technical in nature. Whenever I was aware of them, I also provided references to articles appearing in popular publications (*Science News* and the *New York Times*, for example) that can be found in most public libraries and that, I hope, may be more accessible to lay readers.

PROLOGUE

1–2 research in east Africa: J. Clay, S. Steingraber, and P. Niggli, *The Spoils of Famine* (Cambridge, MA: Cultural Survival Press, 1988)

3–4 Seascale: V. Beral et al. (eds.), *Childhood Cancer and Nuclear Installations* (London: BMJ Publishing Group, 1993); M. J. Gardner, "Childhood Leukaemia around the Sellafield Nuclear Plant," in P. Elliot, et al. (eds.), *Geographical and Environmental Epidemiology: Methods for Small-Area Studies* (Oxford, England: Oxford University Press, 1992), pp. 291–309.

4 *New York Times*: J. H. Cushman, "U.S. Reshaping Cancer Strategy As Incidence in Children Rises," *New York Times*, 29 Sept. 1997, pp. A-1, A-14.

4 British cancer registry: A. J. Swerdlow, "Cancer Incidence Data for Adults," in Elliot et al.

4 trends in British cancer mortality: D. L. Davis and D. Hoel (eds.), *Trends in Cancer Mortality in Industrialized Countries* (New York: New York Academy of Sciences, 1990), pp. 9, 195, 206.

5 Britain has led the way in mapping disease clusters: see Elliot, et al.

5 Knox study: E. G. Knox and E. A. Gilman, "Hazard Proximities of Childhood Cancers in Great Britain from 1953–80," *Journal of Epidemiology and Community Health* 51 (1997): 151–59.

5 Woburn cancer cluster: The film is based on the best-selling book, J. Harr, *A Civil Action* (New York: Random House, 1996).

5 new epidemiological study: Bureau of Environmental Health Assessment, *Woburn Childhood Leukemia Follow-Up Study* (Boston: Massachusetts Department of Public Health, May 1996).

6 shortcomings of British CRI: Mary Taylor, "Insisting on our Right to Know: Stories from Europe," presentation to TRI Data Use Conference, Washington, D.C., 8–10 September 1997; Friends of the Earth UK, "Toxics in Your Backyard: Your Right to Know about Industrial Pollution—a Case Study at Avonmouth," March 1997.

6 pesticide regulation in the UK: Peter Beaumont, *Pesticides, Policies and People* (London: Pesticides Trust, 1993).

6–7 Esthwaite Waters: G. Sanders, et al., "Historical Inputs of Polychlorinated Biphenyls and Other Organochlorines to a Dated Lacustrine Sediment Core in Rural England," *Environmental Science and Technology* 26 (1992): 1815–21.

7 dioxin releases in the UK: UK Environment Agency, "Dioxin Releases to Land, 1993," Environment Agency. *A Review of Dioxin Releases to Land and Water in the UK* (Bristol: Environment Agency, 1997).

7 Leeds University statement: "Population Health Looking Upstream," (editorial), *Lancet* 343 (1994): 429–30.

ONE *trace amounts*

2 Mahomet River: J. P. Kempton and A. P. Visocky, *Regional Groundwater Resources in Western McLean and Eastern Tazewell Counties with an Emphasis on the Mahomet Bedrock Valley*, Cooperative Groundwater Report 13 (Champaign: ISGWS, 1992); J. P. Kempton et al., "Mahomet Bedrock Valley in East-Central Illinois: Topography, Glacial Drift Stratigraphy, and Hydrogeology," in N. Melhorn and J. P. Kempton (eds.), *Geology and Hydrology of the Teays-Mahomet Bedrock Valley System*, Special Report 258 (Boulder, Colo.: Geological Society of America, 1991); J. P. Gibb et al., *Groundwater Conditions and River-Aquifer Relationships along the Illinois Waterway* (Champaign: ISWS, 1979); M. M. Killey, "Do You Live above an Underground River?" Geogram 6 (Urbana: ISGS, 1975).

2–3 the ancestral Mississippi River valley: M. A. Marino and R. J. Schicht, *Groundwater Levels and Pumpage in the Peoria-Pekin Area, Illinois, 1890–1966* (Champaign: ISWS, 1969), 3; S. L. Burch and D. J. Kelly, *Peoria-Pekin Regional Groundwater Quality Assessment*, Research Report 124 (Champaign: ISWS, 1993), 6.

3 Illinois farm statistics: IFB, *Farm and Food Facts* (Bloomington, Ill.: IFB, 1994).

3 the disappearance of the Illinois prairie: IDENR, *The Changing Illinois Environment: Critical Trends*, summary report and vol. 3, ILENR/RE-EA-94/05 (Springfield, Ill.: IDENR, 1994); S. L. Post, "Surveying the Illinois Prairie," *The Nature of Illinois* (winter 1993): 1–8; R. C. Anderson, "Illinois Prairies: A Historical Perspective," in L. M. Page and M. R. Jeffords (eds.), *Our Living Heritage: The Biological Resources of Illinois* (Champaign: INHS, 1991).

5 current pesticide application in Illinois: L. P. Gianessi and J. E. Anderson, *Pesticide Use in Illinois Crop Production* (Washington, D.C.: National Center for Food and Agricultural Policy, 1995), table B-2. Fifty-four million represents pounds of active ingredient. This figure is an extrapolation derived from small-scale surveys. Other than California and New York, both of which maintain state pesticide registries, no state or federal agency keeps

track of pesticide use (unless the pesticide is classified as restricted). In Illinois, records are kept only on the number of acres sprayed, not amounts sprayed per acre. Moreover, many newer pesticides are more potent at low dosages. Decreases in pounds or ounces sprayed per acre do not necessarily indicate decreases in pesticide reliance or toxicity. See IDENR, *Changing Illinois*, summary report, 81.

5 percentage of corn treated with pesticides in 1950: IDENR, *Changing Illinois*, vol. 3., 78.

5 percentage of corn treated in 1993: IASS, *Agricultural Fertilizer and Chemical Use: Corn—1993* (Springfield, Ill.: IDA, 1994).

5 pesticide drift: C. M. Benbrook et al., *Pest Management at the Crossroads* (Yonkers, N.Y.: Consumers Union, 1996); C. A. Edwards, "The Impact of Pesticides on the Environment," in D. Pimentel et al. (eds.), *The Pesticide Question: Environment, Economics, and Ethics* (New York: Routledge, 1993), 13–46; D. E. Glotfelty et al., "Pesticides in Fog," *Nature* 325 (1987): 602–5.

5 pesticides in Illinois surface streams: A. G. Taylor and S. Cook, "Water Quality Update: The Results of Pesticide Monitoring in Illinois' Streams and Public Water Supplies" (paper presented at the 1995 Illinois Agricultural Pesticides Conference, Univ. of Illinois, Urbana, 4–5 Jan. 1995).

5 pesticides in Illinois groundwater: A. G. Taylor, "The Effects of Agricultural Use on Water Quality in Illinois" (paper presented at the 1993 American Chemical Society Agrochemicals Division Symposium, "Pesticide Management for the Protection of Ground and Surface Water Resources," Chicago, Ill., 25–26 Aug. 1993); S. C. Schock et al., *Pilot Study: Agricultural Chemicals in Rural, Private Wells in Illinois*, Cooperative Groundwater Report 14 (Champaign: ISGWS, 1992).

5 atrazine's link to cancer: EPA, *The Triazine Herbicides: Atrazine, Simazine, and Cyanazine*, Position Document 1, Initiation of Special Review, OPP-30000-60-4919-5 (Washington, D.C.: Office of Pesticide Programs, 1994); A. Pinter et al., "Long-term Carcinogenicity Bioassay of the Herbicide Atrazine in F344 Rats," *Neoplasma* 37 (1990): 533–44; A. Donna et al., "Triazine Herbicides and Ovarian Epithelial Neoplasms," *Scandinavian Journal of Work Environment and Health* 15 (1989): 47–53; A. Donna et al., "Ovarian Mesothelial Tumors and Herbicides: A Case-Control Study," *Carcinogenesis* 5 (1984): 941–42.

5–6 hazardous waste in Illinois: C. W. Forrest and R. Olshansky, *Groundwater Protection by Local Government* (Champaign: IDENR and IEPA, 1993); W. H. Allen, "Hazardous Waste: Past, Present, Future," *The Nature of Illinois* (winter 1992): 13–16. Updated estimates were obtained from the IEPA's Office of Chemical Safety in Jan. 1997.

5–6 number of waste sites in Illinois: IDENR, *Changing Illinois*, summary report, 29, 68; R. D. Brower and A. P. Visocky, *Evaluation of Underground Injection of Industrial Waste in Illinois*, Joint Report 2 (Champaign: Illinois Scientific Surveys, 1989). Updated estimates were obtained from the IEPA's Office of Chemical Safety in Jan. 1997.

5–6 import and export of hazardous waste: IEPA, *Summary of Annual Reports on Hazardous Waste in Illinois, 1991 and 1992: Generation, Treatment, Storage, Disposal, and Recovery* (Springfield, Ill.: IEPA, 1994), v; IEPA, *Illinois Non-hazardous Special Waste Annual Report for 1991* (Springfield, Ill.: IEPA, 1993). Updated estimates were obtained from the IEPA's Office of Chemical Safety in Jan. 1997.

6 legal releases of toxic chemicals: IEPA, *Sixth Annual Toxic Chemical Report*, IEPA/ENV/94-151 (Springfield, Ill.: IEPA, 1994), v.

6 metal degreasers and dry-cleaning fluids: IDPH, *Chlorinated Solvents in Drinking Water* (Springfield, Ill.: IDPH, Division of Environmental Health, n.d.).

6 quote from a recent state assessment: IDENR, *Changing Illinois*, summary report, 6.

6 universal detections of DDT and PCBs in human tissues: R. R. M. Sharpe, "Another DDT Connection," *Nature* 375 (1995): 538–39; W. J. Rogan et al., "Polychlorinated Biphenyls (PCBs) and Dichlorodiphenyl Dichloroethene (DDE) in Human Milk: Effects on Growth, Morbidity, and Duration of Lactation," *AJPH* 77 (1987): 1294–97.

6 DDT can remain in soil for several decades: J. B. Diamond and R. B. Owen, "Long-Term Residue of DDT Compounds in Forest Soils in Maine," *Environmental Pollution* 92 (1996): 227–30.

6–7 archival film clips appear in "Rachel Carson's *Silent Spring*," documentary film by Peace River Films, aired on PBS, *The American Experience*, 8 Feb. 1993.

7 old magazine ads for DDT are reprinted in E. P. Russell III, "'Speaking of Annihilation': Mobilizing for War against Human and Insect Enemies, 1914–1945," *Journal of American History* 82 (1996): 1505–29; and in J. Curtis et al., *After* Silent Spring: *The Unsolved Problem of Pesticide Use in the United States* (New York: NRDC, 1993), 2.

7 DDT for polio control: T R. Dunlap, *DDT Scientists, Citizens and Public Policy* (Princeton, NJ.: Princeton Univ. Press, 1981), 65.

7 DDT in paint: This ad, for Sherwin-Williams, appeared in 1946. See E. C. Helfrick as told to M. Riddle, "Mass Murder Introduces Sherwin-Williams' 'Pestroy,'" *Sales Management*, 15 Oct. 1946, pp. 60–64. See also E. P. Russell III, *The Nature of War: Pest Control, Chemical Warfare, and American Culture, 1914–1962* (in preparation).

7 DDT in blankets: DDT was also incorporated into starch finishes. See T. F. West and G. W. Campbell, *DDT and Newer Persistent Pesticides* (New York: Chemical Puhlishing Co., 1952), 163–74. In addressing the question of whether the routine use of DDT in textiles could pose threats to human health, these authors reached the following conclusion: "Extensive investigations have been carried out and it would appear that DDT is as safe as many chemicals at present in everyday use, and probably a good deal safer than many" (173).

7 quotes from fellow baby boomers: Jean Powers of Dover, Mass., and John Gephart of Ithaca, N.Y.

7 "the harmless aspect of the familiar": R. Carson, *Silent Spring* (Boston: Houghton Mifflin, 1962), 20.

7–8 "It is not my contention . . .": Ibid., 12.

8 Carson on future generations: Ibid., 13.

8 "killer of killers," "the atomic bomb of the insect world": J. Warton. *Before* Silent Spring: *Pesticides and Public Health in Pre-DDT America* (Princeton, NJ.: Princeton Univ. Press, 1974), 248–55.

8 failure of DDT: Carson, *Silent Spring*, 20–23, 58, 103, 107–9, 112, 113, 120–22, 125, 143–44, 206–7, 225, 267–73; T. R. Dunlap, *DDT: Scientists, Citizens and Public Policy* (Princeton, N.J.: Princeton Univ. Press, 1981), 63–97.

8 DDT in breast milk: E. P. Laug et al., "Occurrence of DDT in Human Fat and Milk," *AMA Archives of Industrial Hygiene arid Occupational Medicine* 3 (1951): 245–46.

9 EDT's ongoing presence: USDA, *Pesticide Data Program, Annual Summary Calendar Year 1994* (Washington, D.C: USDA, Agricultural Marketing Service, 1994), 13; R. G. Harper et a1., "Organochlorine Pesticide Contamination in Neotropical Migrant Passerines," *Archives qf Environmental Contamination and Toxicology* 31 (1996): 386–90; ATSDR, "DDT, DDE, and DDD" (fact sheet) (Atlanta: ATSDR, 1995); R. G. Lewis et a1., "Evaluation of Methods for Monitoring the Potential Exposure of Small Children to Pesticides in the Residential Environment," *Archives of Environmental Contamination and Toxicology* 26 (1996): 37–46; W. H. Smith et al., "Trace Organochlorine Contamination of the Forest Floor of the White Mountain National Forest, New Hampshire," *Environmental Science and Technology* 27 (1993): 2244–46; EPA, *Deposition of Air Pollutants to the Great Lakes: First Report to Congress*, EPA-453/R-93-055 (Washington, D.C.: EPA, 1994).

9 export of DDT and other banned pesticides: In 1992, 600,000 lbs. of DDT were shipped out of U.S. ports. Some analysts suspect this cargo may represent a transshipment—cargo imported and then exported again. Poor labeling of pesticide exports make careful tracking very difficult. J. Raloff, "The Pesticide Shuffle," *Science News* 149 (1996): 174–75; Foundation for the Advancement of Science and Education, *Exporting Risk: Pesticide Exports from U.S. Ports* (Los Angeles: Foundation for the Advancement of Science and Education, 1996); J. Wargo, *Our Children's Toxic Legacy: How Science and Law Fail to Protect Us from Pesticides* (New Haven, Conn.: Yale Univ. Press, 1996), 163–64; D. J. Hanson, "Administration Seeks Tighter Curbs on Exports of Unregistered Pesticides," *Chemical and Engineering News*, 14 Feb. 1994, 16–18; Monica Moore, Pesticide Action Network, personal communication.

9 uses of lindane: M. Moses, *Designer Poisons: How to Protect Your Health and Home from Toxic Pesticides* (San Francisco: Pesticide Education Center, 1995); EPA, *Suspended, Cancelled and Restricted Pesticides*, 20T-1002 (Washington, D.C.: EPA, 1990); Curtis, *After* Silent Spring.

9–10 aldrin and dieldrin: J. B. Barnett and K. E. Rodgers, "Pesticides," in J. H. Dean et al. (eds.), *Immunotoxicology and Immunopharmacology*, 2nd ed. (New York: Raven Press, 1994), 191–211; R. Spear, "Recognized and Possible Exposures to Pesticides," in W. J. Hayes and E. R. Laws Jr. (eds.), *Handbook of Pesticide Toxicology*, vol. 1. (New York: Academic Press, 1991), 245–46; EPA, 1990, *Suspended*; Carson, *Silent Spring*, 26.

10 chlordane and heptachlor: Spear, "Possible Exposures," 245; P. F. Infante et al., "Blood Dyscrasias and Childhood Tumors and Exposure to Chlordane and Heptachlor," *Scandinavian Journal of Work Environment and Health* 4 (1978): 137–50.

10 pesticides in baby food: Dunlap, *DDT*, 68.

10 women with breast cancer have higher levels of DDE and PCBs in their tumors: M. Wasserman, "Organochlorine Compounds in Neoplastic and Adjacent Apparently Normal Breast Tissue," *Bulletin of Environmental Contamination and Toxicology* 15 (1976): 478–84.

10–11 the Finnish study: H. Mussalo-Rauhamaa et al., "Occurrence of beta-Hexachlorocyclohexane in Breast Cancer Patients," *Cancer* 66 (1990): 2124–28. Lindane is the gamma isomer of hexachlorocyclohexane.

11 the Connecticut study: F Falck Jr. et al., "Pesticides and Polychlorinated Biphenyl Residues in Human Breast Lipids and Their Relation to Breast Cancer," *AEH* 47 (1992): 143–46.

11 the New York City study: M. S. Wolff et al., "Blood Levels of Organochlorine Residues and Risk of Breast Cancer," *JNCI* 85 (1993): 648–52; D. J. Hunter and K. T. Kelsey, "Pesticide Residues and Breast Cancer: The Harvest of a Silent Spring?" *JNCI* 85 (1993): 598–99; M. P. Longnecker and S.J. London, "Re: Blood Levels of Organochlorine Residues and Risk of Breast Cancer" (letter and response by M. S. Wolff), *JNCI* 85 (1993): 1696–97.

11 the Québec study: É. Dewailly et al., "High Organochiorine Body Burden in Women with Estrogen Receptor–Positive Breast Cancer," *JNCI* 86 (1994): 232–34. Increasing incidence of receptor-positive breast cancer is largely responsible for the increase in breast cancer rates that occurred between the mid-1970s and the mid-1980s. See A. G. Glassand and R. N. Hoover, "Rising Incidence of Breast Cancer: Relationship to State and Receptor Status," *JNCI* 82 (1990): 693–96.

12–13 the California study: N. Krieger et al., "Breast Cancer and Serum Organochlorines: A Prospective Study among White, Black and Asian Women," *JNCI* 86 (1994): 589–99; B. MacMahon, "Pesticide Residues and Breast Cancer?" *JNCI* 86 (1994): 572–73; S. S. Sternberg, "Re: DDT and Breast Cancer" (and responses by the authors), *JNCI* 86 (1994): 1094–96; J. E. Brody, "Strong Evidence in a Cancer Debate," *New York Times*, 20 Apr. 1994, p. C-11; D. A. Savitz, "Re: Breast Cancer and Serum Organochlorines: A Prospective Study among White, Black, and Asian Women," *JNCI* 86 (1994): 1255. Questions about the tubes' red caps have been raised by Dr. Devra Lee Davis.

13 breast cancer among women born between 1947 and 1958: D. L. Davis et al., "Decreasing Cardiovascular Disease and Increasing Cancer among Whites in the United States from 1973 through 1987: Good News and Bad News," *JAMA* 271 (l994): 431–37.

13 pesticide use since *Silent Spring*: Pesticide use doubled between 1964 and 1982, as measured by weight of active pesticidal ingredients. See Wargo, *Toxic Legacy*, 132.

13 failure to pursue research on cancer's environmental connections: See, for example, M. S. Wolff, "Pesticides—How Research Has Succeeded and Failed in Informing Policy: DDT and the Link with Breast Cancer," *EHP* 103, suppl. 6 (1995): 87–91.

TWO *silence*

15–16 Carson's concern about pesticide debates: L. J. Lear, "Rachel Carson's *Silent Spring*," *Environmental History Review* 17 (1993): 23–48. See also Lear's definitive biography, *Rachel Carson: Witness for Nature* (New York: Holt, 1997).

16 letter from Duxbury: T. T. Williams, "The Spirit of Rachel Carson," *Audubon* 94 (1992): 104–7; P. Brooks, *The House of Life: Rachel Carson at Work* (Boston: Houghton Mifflin, 1989), 229–35.
16 "Knowing what I do . . .": Carson's letter to Freeman, June 28, 1958, reprinted in M. Freeman (ed.), *Always, Rachel: The Letters of Rachel Carson and Dorothy Freeman* (Boston: Beacon, 1995), 259.
16–17 Iroquois County: Rachel Carson, *Silent Spring* (Boston: Houghton Mifflin, 1962), 91–100.
17 refusal of scientists to send Carson information: Dr. Linda Lear, personal communication.
17 threat of defunding: Carson, *Silent Spring*, 94–95.
17 "The other day . . .": Carson's lerter to Freeman, 27 June 1962, reprinted in Freeman, *Always, Rachel*, 408.
17 Carson's speech to the Press Club is quoted in Brooks, *House of Life*, 302–4.
20 20 years of life lost: Dr. Devra Lee Davis, personal communication.
21 Carson's cancer diagnosis and physical ailments: Carson's letters to Freeman, 1960–1964, in Freeman, *Always, Rachel*; Brooks, *House of Life*; Dr. Linda Lear, personal communication.
21 Carson's relief at finishing *Silent Spring*: Carson's letter to Freeman, 6 Jan. 1962, in Freeman, *Always, Rachel*, 391.
21 two quotes from letters to Freeman: 3 Nov. 1963, and 9 Jan. 1964, ibid., 490, 515. See also letters dated 6 Jan. 1962; 2 Mar. 1963; and 25 Apr. 1963.
24 Carson's letters to Freeman that speak openly: 3 Jan. 1961; 23 Mar. 1961; 25 Mar. 1961; and 18 Sept. 1963, ibid., 326, 364, 365–66, 469.
24 letters that speak elliptically: 17 Jan. 1961; 15 Feb. 1961; 25 Oct. 1962; 25 Dec. 1962; and 2 Jan. 1964, ibid., 331, 346, 414, 420, 508.
24 Freeman's reference to Carson's mastectomy: Freeman's letter to Carson, 30 Apr. 1960, ibid., 305.
24–25 their entreaties and admissions: See, for example, Freeman's letter to Carson, 6 Mar. 1963, ibid., 441.
25 the darker story: Freeman's letters to Carson, 4 and 17 Mar. 1961, ibid., 356, 363.
25 confessions and recantations: Carson's letters to Freeman, 23 Jan. 1962; 26 Mar. 1962; 10 Apr. 1962; 14 Feb. 1963; 18 Feb. 1963; 2 Mar. 1963; 14 Jan. 1964, ibid., 395, 399, 404, 434–37, 439–40, 516.
25 Carson's prohibition of discussions about her health: M. Spock, "Rachel Carson: A Portrait," *Rachel Carson Council News* 82 (1994): 1–4; Dr. Linda Lear, George Washington University, personal communication.
25 quotes instructing Dorothy: Carson's letters to Freeman, 1 Apr. and 20 May 1962, in Freeman, *Always, Rachel*, 401, 405.
25–26 photographs and old film clips: Beinecke Library archives, Yale University; "Rachel Carson's *Silent Spring*," documentary film by Peace River Films, aired on PBS, *The American Experience*, 8 Feb. 1993.
27 farmers and housewives with cancer: Carson, *Silent Spring*, 227–30.
27 first line of evidence: Ibid., 219–20.
27–28 second and third lines of evidence: Ibid., 221.
28 "whatever seeds of malignancy . . .": Ibid., 226.
28 death certificates and children's cancers: Ibid., 221–22.

28 animals with cancer: Ibid., 222–39.
28–29 cellular mechanisms of carcinogenesis: Ibid., 231–35.
28 effect on sex horniones: Ibid., 235–37.
28 effect on metabolism: Ibid., 231–32.
28–29 Carson's prediction: Ibid., 232–33.
29 interspecies differences in susceptibility: H. C. Pitot III and Y. P. Dragan, "Chemical Carcinogens," in D. Klaassen (ed.), *Casarett and Doull's Toxicology: The Basic Science of Poisons*, 5th ed. (New York: McGraw-Hill, 1996), 248–49; NRC, *Animals as Sentinels of Environmental Health Hazards* Washington, D.C.: National Academy Press, 1991).
29 uncontrolled human experiment: A lack of unexposed controls makes human studies difficult but not impossible. Theoretically, all that is required for such studies are measurable differences in exposure levels among segments of the human population. For example, all of us are believed to carry detectable levels of dioxin in our tissues. The question of whether dioxin contributes to human cancers can be addressed by studies that compare cancer incidence rates among those heavily, moderately, and lightly exposed. All other things being equal, a positive trend would indicate a dose-response relationship, which is considered strong evidence by cancer researchers. The wider the spread in exposure levels, the more likely the relationship—if indeed one exists—will reveal itself. As such, researchers interested in conducting human studies often look for "natural experiments" where an unfortunate event—such as a toxic spill of some sort—has exposed an identifiable sector of the population to a heavy dose of the substance in question. Disease rates among this group can then be compared to those of the general population whose exposures to this substance may be common and ongoing but are occurring at much lower levels.

THREE *time*

Unless otherwise stated in the following notes, all statistics cited in this chapter on U.S. cancer incidence and mortality rates come from the National Cancer Institute's SEER Program Registry: L. A. G. Reis et al. (eds.), *SEER Cancer Statistics Review 1973–1991: Tables and Graphs*, NIH Pub. 94-2989 (Bethesda, Md.: NCI, 1994). Statistics on cancer incidence and mortality in Illinois come from H. L. Howe and M. Lehnherr, *Incidence in Illinois by County, 1986–1990*, Epidemiological Report, ser. 92, no. 4 (Springfield, Ill.: IDPH, 1992). Statistics on cancer incidence and mortality in Massachusetts come from S. Gershman, *Cancer Incidence in Massachusetts, 1982–1990* (Boston: MDPH, Massachusetts Cancer Registry, 1993).

32 number of cancer diagnoses in 1995: ACS, *Cancer Facts and Figures—1995* (Atlanta: ACS, 1995).
35–36 uncertainties in data ascertainment: H. Menck and C. Smart (eds.), *Central Cancer Registries: Design, Management, and Use* (Chur, Switzerland: Harwood Academic Press, 1994); O. M. Jensen et al. (eds.), *Cancer Registration: principles and Methods*, IARC Scientific Publication 95 (Lyon, France: IARC, 1991).
35 *Cancer Registry News* is a publication of the Massachusetts Cancer Registry.
36 percentage of recent upsurge in breast cancer attributable to earlier detection:

226 letter to the editor in Havana: A. Robertson, *Mason City Banner Times*, 10 June 1992, p. 11.
226 letter to the editor in Forrest: C. Kaisner, "Suddenly in Forrest, Greed Has Become No. 1 Attitude," *Bloomington Daily Pantagraph*, 6 Aug. 1994.
227 letter about Kirby's smoking habits: R. Hankins, letter to the editor, *Mason County Democrat*, 3 June 1992, p. 2.
227 endorsement of risk: "Editorial," *Fairbury Blade*, 20 July 1994, p. 2.
227 condemnation of risk: "Dioxin Findings Raise New Fears" (editorial), *Jacksonville Journal-Courier*, 15 Sept. 1994, p. 10.
227–228 P450 enzymes and Ah receptors: Webster and Commoner, "Dioxin Debate"; G. Lucier et al., "Receptor Model and Dose-Response Model for the Effects of Dioxin," *EHP* 101 (1993): 36–44; T. R. Sutter et al., "Targets for Dioxin: Genes for Plasminogen Activator Inhibitor-2 and Interleukin-1B," *Science* 254 (1991): 415–18.
228 antiestrogenic qualities of dioxin: L. Birnbaum, "Endocrine Effects of Prenatal Exposure to PCBs, Dioxins, and Other Xenobiotics: Implications for Policy and Future Research," *EHP* 102 (1994): 676–79.
228 substances that bind with the Ah receptor: "The Problem with Tallying Dioxin," *Science News* 146 (1994): 206.
228–229 Ah receptors knocked out in mice: R. Stone, "Dioxin Receptor Knocked Out," *Science* 268 (1995): 638–39; P. Fernandez-Salguero et al., "Immune System Impairment and Hepatic Fibrosis in Mice Lacking the Dioxin-Binding Ah Receptor," *Science* 268 (1995): 722–26.
229 dioxin's other shadowy habits: T. Colborn et al., *Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival?—A Scientific Detective Story* (New York: Dutton, 1996), 110–21; A. P. van Birgelen et al., "Synergistic Effect of 2,2',4,4',5,5'-Hexachlorobiphenyl and 2,3,7,8-Tetra-Chlorodibenzo-*p*-Dioxin on Hepatic Porphyrin Levels in the Rat," *EHP* 104 (1996): 550–57; N. I. Kerkvliet, "Immunotoxicology of Dioxins and Related Compounds," in Schecter, *Dioxins and Health*, 199–225; S. P. Porterfield, "Vulnerability of the Developing Brain to Thyroid Abnormalities: Environmental Insults to the Thyroid System," *EHP* 102, suppl. 2 (1994): 125–30; S. Rier et al., "Endometriosis in Rhesus Monkeys (*Macaca mulatta*) following Chronic Exposure to 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin," *Fundamental and Applied Toxicology* 21 (1993): 433–41.
229–230 John Kirby's career: T. L. Aldous, "Kirby Sees Havana Opportunity, Opposition," *PDT*, 22 Oct. 1993, pp. A-1, A-12; A. Lindstrom, "Sherman Horse Track Sure Bet—Promoters," *SSJR*, 6 Jan. 1977; "John Kirby, Williamsville, Ponders Race for Senate," *SSJR*, 9 Oct. 1973; J. O'Dell, "Hens with Glasses a Barnyard Spectacle," *SSJR*, 27 Aug. 1973; K. Watson, "John Kirby Eyes Candidacy," *SSJR*, 8 Aug. 1968; K. Watson, "Page Names Kirby," *SSJR*, 7 Jan. 1963.
230 quotes by Kirby: Aldous, "Kirby sees Havana."
232 quote from Summit: B. M. Rubin, "Summit's Push for Incinerator Sparks Unusual Bunch of Foes," *Chicago Tribune*, 24 Oct. 1993, Southwest sec., p. 1.
233 number of U.S. incinerators: B. Paigen, "How to Be a Dioxin Detective," in Gibbs, *Dying from Dioxin*, 205–36.

233 fish of the Vermilion: IEPA, *Illinois Water Quality Report, 1992–93*, vol. 1, IEPA/WPC/94-160 (Springfield, Ill.: IEPA, 1994).
234 quote by John Kirby: J. Knauer, "Incinerator's Future Smoldering after 'No' Vote," *Fairbury Blade*, 16 Nov. 1994, pp. 1, 3.
234 appellate court decision: E. Hopkins, "Court Backs Pollution Board's Incinerator Ruling," *FJS*, 13 Sept. 1995, p. B-5.
234 repeal of retail rate law: R. B. Dold, "Clearing the Air," *Chicago Tribune*, 12 Jan. 1996, pp. 1–23.
234 Malignant mesothelioma is a cancer of the membranes surrounding the lungs. It is caused almost exclusively by exposure to asbestos fibers. Cigarette smoke interacts synergistically with asbestos in creating risk.

ELEVEN *our bodies, inscribed*

235–236 tree-ring analysis: R. Phipps and M. Bolin, "Tree Rings—Nature's Signposts to the Past," *Illinois Steward* (summer 1993): 18–21.
236 organochlorine residues: Anne Colston Wentz, testimony before the Subcommittee on Health and the Environment of the Committee on Energy and Commerce, U.S. House of Representatives, Health Effects of Estrogenic Pesticides Hearings, 21 Oct. 1993 (Washington, D.C.: GPO serial no. 103-87, 1994), 133.
236 contaminants found in a variety of human tissues: D. Holzman, "Banking on Tissues," *EHP* 104 (1996): 606–10; M. Moses et al., "Environmental Equity and Pesticide Exposure," *Toxicology and Industrial Health* 9 (1993): 913–59. (See especially pp. 922–26.)
236–237 organochlorines in umbilical cords: L. W. Kanja et al., "A Comparison of Organochlorine Pesticide Residues in Maternal Adipose Tissue, Maternal Blood, Cord Blood, and Human Milk from Mother/Infant Pairs," *Archives of Environmental Contamination and Toxicology* 22 (1994): 21–24; H. Autrup, "Transplacental Transfer of Genotoxins and Transplacental Carcinogenesis," *EHP* 101, suppl. 2 (1993): 33–38.
237 chlorpyrifos in urine: "Chlorpyrifos Metabolites in 82% of U.S. Population," *Pesticide and Toxic Chemical News*, 8 Nov. 1995, pp. 15–16.
237 PCBs in blood as a measure of body burden: Å. Gergman et al., "Selective Retention of Hydroxylated PCB Metabolites in Blood," *EHP* 102 (1994) 464–69; A. Schecter et al., "Polychlorinated Biphenyl Levels in the Tissues of Exposed and Nonexposed Humans," *EHP* 102, suppl. 1 (1994): 149–58. Mixtures of PCBs also vary by source and by geographic region. The mix of PCB types found in the tissues of someone exposed primarily through eating freshwater fish is likely very different from that found in someone exposed on the job, for example. These complications make determining the health effects of PCB exposure an arduous task. Adding to the difficulty is the fact that the 209 different PCB variants have differing physiological properties. Some appear to be estrogenic, others antiestrogenic. See H. A. Tilson et al., "Polychlorinated Biphenyls and the Developing Nervous System: Cross-Species Comparisons," *Neurotoxicology and Teratology* 12 (1990): 239–48.
237 body fat: L. Kohlmeier et al., "Adipose Tissue as a Medium for Epidemio-

logic Exposure Assessment," *EHP* 103, suppl. 3 (1995): 99–106; B. G. Loganathan et al., "Temporal Trends of Persistent Organochlorine Residues in Human Adipose Tissue from Japan, 1928–1985," *Environmental Pollution* 81 (1993): 31–39; L. López-Carillo et al., "Is DDT Use a Public Health Problem in Mexico?" *EHP* 104 (1996): 584–88.

237–238 breast milk: M. N. Bates et al., "Chlorinated Organic Contaminants in Breast Milk of New Zealand Women," *EHP* 102, suppl. 1 (1994): 211–17; P. Fürst et al., "Human Milk as a Bioindicator for Body Burden of PCDDs, PCDFs, Organochlorine Pesticides, and PCBs," *EHP* 102 (1994): 187–93; M. R. Sim and J. J. McNeil, "Monitoring Chemical Exposure Using Breast Milk: A Methodological Review," *AJE* 136 (1992): 1–11.

238 1951 discovery of breast milk contaminants: E. P. Laug et al., "Occurrence of DDT in Human Fat and Milk," *A.M.A. Archives of Industrial Hygiene and Occupational Medicine* 3 (1951): 245–46.

238 Carson on breast milk: R. Carson, *Silent Spring* (Boston: Houghton Mifflin, 1962), 23.

238 PCBs in breast milk: W. J. Rogan et al., "Polychlorinated Biphenyls (PCBs) and Dichlorodiphenyl Dichloroethene (DDE) in Human Milk: Effects of Maternal Factors and Previous Lactation," *AJPH* 76 (1986): 172–77.

238 one-quarter of breast milk illegal: Ibid.

238 relationship between cancer and carcinogens in breast milk: Several small-scale studies have indicated that levels of organochlorinated pesticides in breast fat are higher in breast cancer patients than in controls. Other studies do not find this pattern. See discussion in Chapter 1.

238 Prolonged breast-feeding reduces modestly the risk of breast cancer to the mother, although it is not clear whether this benefit is due to the excretion of breast carcinogens or to some other unidentifed factor. See L. A. Brinton, "Breastfeeding and Breast Cancer Risk," *Cancer Causes and Control* 6 (1995): 199–208; and P. A. Newcomb, "Lactation and a Reduced Risk of Premenopausal Breast Cancer," *NEJM* 330 (1994): 81–87. See also É. Dewailly et al., "Protective Effect of Breast Feeding on Breast Cancer and Body Burden of Carcinogenic Organochlorines" (letter), *JNCI* 86 (1994): 803.

238 North Carolina study: Rogan, "Polychlorinated Biphenyls (PCBs)."

238–239 some breast milk contaminants beginning to drop: K. Norén et al., "Methylsufonyl Metabolites of PCBs and DDE in Human Milk in Sweden, 1972–1992," *EHP* 104 (1996): 766–73; Fürst, "Human Milk as a Bioindicator"; A. Somogyi and H. Beck, "Nurturing and Breast-feeding: Exposure to Chemicals in Breast Milk," *EHP* 101 (1993): 45–52.

239 apoptosis: H. C. Pitot III and Y. P. Dragan, "Chemical Carcinogenesis," in C. D. Klaassen (ed.), *Casarett and Doull's Toxicology: The Basic Science of Poisons*, 5th ed. (New York: McGraw-Hill, 1996), 227.

239 local and distant control of mitosis: Dr. Thomas Webster, Boston University, personal communication.

240 process of carcinogenesis: J. Felton, "Mechanisms of Cancer Induction and Progression: Endogenous and Environmental Factors," in *Evaluating the National Cancer Program: An Ongoing Process*, Proceedings of the President's National Cancer Panel Meeting, 22 Sept. 1993 (Bethesda, Md.: NCI), 14–16.

240 purposefulness: Robert Millikan, personal communication. See also S. B. Nuland, *How We Die: Reflections on Life's Final Chapter* (New York: Random House, 1993), 202–21.

240–241 invasiveness and primitivism: R. A. Weinberg, "How Cancer Arises," *Scientific American*, Sept. 1996, 62–70; E. J. Mange and A. P. Mange, *Basic Human Genetics* (Sunderland, Mass.: Sinauer, 1994), 350.

241 Inheritance of flawed genes is thought to account for a small percentage of all U.S. cancers. See F. P. Perera, "Uncovering New Clues to Cancer Risk," *Scientific American*, May 1996, 54–62.

241 oncogenes and tumor suppressor genes: W. K. Cavenee and R. L. White, "The Genetic Basis of Cancer," *Scientific American*, Mar. 1995, 72–79; J. C. Barrett and M. D. Shelby, "Mechanisms of Human Carcinogens," in R. D'Amato et al. (eds.), *Relevance of Animal Studies for the Evaluation of Human Cancer Risk* (New York: Wiley-Liss, 1992), 415–34.

241 accelerator and brakes: J. P. Oliner, "The Role of p53 in Cancer Development," *Scientific American*, Sept.–Oct. 1994, pp. 16–25.

242 colon cancers involve both oncogene and tumor suppressor gene mutations: Cavanee and White, "Genetic Basis of Cancer"; B. Vogelstein et al., "Genetic Alterations during Colorectal-Tumor Development," *NEJM* 319 (1988): 525–32.

242 p53 involved in half of cancers: Oliner, "Role of p53."

242 nature of p53 damage indicates carcinogen responsible: Perera, "Uncovering New Clues."

242 p53 mutations in breast cancer: R. Millikan, "Studying Environmental Influences and Breast Cancer Risk: Suggestions for an Integrated Population-Based Approach," *Breast Cancer Research and Treatment* 35 (1995): 79–89; B. Newman et al., "The Carolina Breast Cancer Study: Integrating Population-Based Epidemiology and Molecular Biology," *Breast Cancer Research and Treatment* 35 (1995) 51–60; P. J. Biggs et al., "Does a Gentoxic Carcinogen Contribute to Human Breast Cancer? The Value of Mutational Spectra in Unravelling the Aetiology of Cancer," *Mutagenesis* 8 (1993): 275–83.

242 benzo[a]pyrene and DNA adducts: Perera, "Uncovering New Clues."

242 disruption of spindle fibers: Barrett and Shelby, "Mechanisms of Human Carcinogens."

242 significance of DNA repair genes: B. Proujan, "DNA Repair," *EHP* 104 (1996): 18–19.

242–243 three stages of carcinogenesis: Pitot and Dragan, "Chemical Carcinogenesis"; S. H. Yupsa and C. C. Harris, "Molecular and Cellular Basis of Chemical Carcinogenesis," in D. Schottenfeld and J. F. Fraumeni Jr., *Cancer Epidemiology and Prevention* (Philadelphia: Saunders, 1982), 23–43.

243 role of the immune system (dioxin, leukemias and lymphomas, Soviet Union and T cells): R. Repetto and S. S. Baliga, *Pesticides and the Immune System: The Public Health Risk* (Washington, D.C.: World Resources Institute, 1996).

243–244 signal transduction: Weinberg, "How Cancer Arises"; Pitot and Dra-

gan, "Chemical Carcinogenesis"; Barrett and Shelby, "Mechanisms of Human Carcinogens."

244 cancer progressors: Barrett and Shelby, "Mechanisms of Human Carcinogens."

244 carcinogens do not fall into neat categories: Pitot and Dragan, "Chemical Carcinogenesis"; Barrett and Shelby, "Mechanisms of Human Carcinogens."

244 dioxin may interfere with apoptosis: J. M. Samet, "Dioxin and Cancer: The Never-Ending Story," *Cancer Causes and Control* 7 (1996): 302–4.

244 differing sensitivities and risks: Perera, "Uncovering New Clues."

244–245 DDT as a cancer accelerator: J. D. Scribner and N. K. Moffet, "DDT Acceleration of Mammary Gland Tumors Induced in the Male Sprague-Dawley Rat by 2-Acetamidophenanthrene," *Carcinogenesis* 2 (1981): 1235–39.

245 quote by Ross Hume Hall: Personal communication.

245 biological markers: Perera, "Uncovering New Clues"; S. Anderson et al., "Genetic and Molecular Ecotoxicology: A Research Framework," *EHP* 102, suppl. 12 (1994): 3–8; M. Eubanks, "Biological Markers: The Clues to Genetic Susceptibility," *EHP* 102 (1994): 50–56; S. Blakeslee, "Genes Tell Story Why Some Get Cancer While Others Don't," *New York Times*, 17 May 1994, p. C-3; M. A. Saleh et al. (eds.), *Biomarkers of Human Exposure to Pesticides* (Washington, D.C.: American Chemical Society, 1994); F. P. Perera, "DNA Adducts and Related Biomarkers in Populations Exposed to Environmental Carcinogens," *EHP* 98 (1992): 133–37.

245 correlations in lab animals: F. A. Béland and M. C. Poirier, "Significance of DNA Adduct Studies in Animal Models for Cancer Molecular Dosimetry and Risk Assessment," *EHP* 99 (1993): 5–10.

245–246 Polish studies: S. Øvrebø et al., "Biological Monitoring of Polycyclic Aromatic Hydrocarbon Exposure in a Highly Polluted Area of Poland," *EHP* 103 (1995): 838–43; F. P. Perera et al., "Molecular and Genetic Damage in Humans from Environmental Pollution in Poland," *Nature* 360 (1992): 256–58; K. Hemminki et al., "DNA Adducts in Humans Environmentally Exposed to Aromatic Compounds in an Industrial Area of Poland," *Carcinogenesis* 11 (1990): 1229–31.

246 quote by Frederica Perera: Perera, "Uncovering New Clues."

246 vinyl chloride and signal transduction proteins: P. W. Brandt-Rauf et al., "Mutant p21 Protein as BioMarker of Chemical Carcinogenesis in Humans," in M. Mendelsohn et al. (eds.), *Biomarkers and Occupational Health: Progress and Perspectives* (Washington, D.C.: Joseph Henry Press, 1995), 163–73.

246 alterations in enzymes: Eubanks, "Biological Markers."

247 Minnesota pesticide applicators: V. F. Garry et al., "Pesticide Applicators with Mixed Pesticide Exposure: G-banded Analysis and Possible Relationship to Non-Hodgkin's Lymphoma," *Cancer Epidemiology, Biomarkers, and Prevention* 5 (1996): 11–16. See also V. F. Garry, "Survey of Health and Use Characterization of Pesticide Appliers in Minnesota," *AEH* 49 (1994): 337–43; and S. Lipkowitz et al., "Interlocus V-J Recombination Measures

Genetic Instability in Agriculture Workers at Risk for Lymphoid Malignancies," *Proceedings of the National Academy of Science* 89 (1992): 5301–05.

247 free radicals: B. Halliwell and O. I. Aruoma, *DNA and Free Radicals* (New York: Ellis Horwood, 1993); D. C. Malins et al., "The Etiology of Breast Cancer: Characteristic Alterations in Hydroxyl Radical-Induced DNA Base Lesions during Oncogenesis with Potential for Evaluating Incidence Risk," *Cancer* 71 (1993): 3036–43; S. S. Thorgeirsson, "Endogenous DNA Damage and Breast Cancer," *Cancer* 71 (1993): 2897–99.

248 1896 discovery: H. Magdelenet and P. Pouillart, "Steroid Hormone Receptors in Breast Cancer," in P. J. Sheridan et al. (eds.), *Steroid Receptors and Disease: Cancer, Autoimmune, Bone, and Circulatory Disorders* (New York: Marcel Dekker, 1988), 436–65.

248 breast cancer risk and estrogen exposure: M. C. Pike et al., "Estrogens, Progestogens, Normal Breast Proliferation, and Breast Cancer Risk," *Epidemiologic Reviews* 15 (1993): 17–35.

248 focus on xenoestrogens: D. L. Davis and H. L. Bradlow, "Can Environmental Estrogens Cause Breast Cancer?" *Scientific American*, Oct. 1995, 166–72; D. L. Davis, "Medical Hypothesis: Xenoestrogens as Preventable Causes of Breast Cancer," *EHP* 101 (1993): 372–77.

248–249 life history of estrogen: Davis and Bradlow, "Can Environmental Estrogens"; P. Toniolo et al., "Reliability of Measurements of Total, Protein-Bound, and Unbound Estradiol in Serum," *Cancer Epidemiology, Biomarkers, and Prevention* 3 (1994): 47–50.

249 behavior of xenoestrogens: Davis and Bradlow, "Can Environmental Estrogens"; N. M. Brown and C. A. Lamartiniere, "Xenoestrogens Alter Mammary Gland Differentiation and Cell Proliferation in the Rat," *EHP* 103 (1995): 708–13.

249 researchers have assumed small role for xenoestrogens: D. L. Houghton and L. Ritter, "Organochlorine Residues and Risk of Breast Cancer," *Journal of the American College of Toxicology* 14 (1995): 71–89.

249 xenoestrogens are common: P. Common, "Environmental Estrogenic Agents Area of Concern," *JAMA* 271 (1994): 414–16.

250 xenoestrogens may be more bioavailable: S. F. Arnold et al., "A Yeast Estrogen Screen for Examining the Relative Exposure of Cells to Natural and Xenoestrogens," *EHP* 104 (1996): 544–48.

250 indirect effects of xenoestrogens: Davis and Bradlow, "Can Environmental Estrogens."

250–251 xenoestrogens alter estrogen metabolism: Davis and Bradlow, "Can Environmental Estrogens"; H. L. Bradlow et al., "Effects of Pesticides on the Ratio of 16 alpha/2-Hydroxyestrone: A Biologic Marker of Breast Cancer Risk," *EHP* 103, suppl. 7 (1995): 147–50; N. T. Telang et al., "Induction by Estrogen Metabolite 16 alpha-Hydroxyestrone of Genotoxic Damage and Aberrant Proliferation in Mouse Mammary Epithelial Cells," *JNCI* 84 (1992): 634–38.

251 cancer among adoptees: T. I. A. Sørensen et al., "Genetic and Environmental Influences on Premature Death in Adult Adoptees," *NEJM* 318 (1988): 727–32.

252 1974 breast cancer blip: ACS, *Breast Cancer Facts and Figures 1996* (Atlanta: ACS, 1995), fig. 2.

TWELVE *ecological roots*

255 bladder tumor experiment: R. A. Weinberg, "A Molecular Basis of Cancer," *Scientific American*, Nov. 1983, pp. 126–42.

256 genetic changes involved in bladder cancer: I. Orlow et al., "Deletion of the p16 and p15 Genes in Human Bladder Tumors," *JNCI* 87 (1995): 1524–29; S. H. Kroft and R. Oyasu, "Urinary Bladder Cancer: Mechanisms of Development and Progression," *Laboratory Investigation* 71 (1994): 158–74; P. Lipponen and M. Eskelinen, "Expression of Epidermal Growth Factor Receptor in Bladder Cancer as Related to Established Prognostic Factors, Oncoprotein Expression and Long-Term Prognosis," *British Journal of Cancer* 69 (1994): 1120–25.

256 aromatic amines and DNA adducts: D. Lin et al., "Analysis of 4-Aminobiphenyl-DNA Adducts in Human Urinary Bladder and Lung by Alkaline Hydrolysis and Negative Ion Gas Chromatography–Mass Spectrometry," *EHP* 102, suppl. 6 (1994): 11–16; P. L. Skipper and S. R. Tannenbaum, "Molecular Dosimetry of Aromatic Amines in Human Populations," *EHP* 102, suppl. 6 (1994): 17–21; S. M. Cohen and L. B. Ellwein, *EHP* 101, suppl. 5 (1994): 111–14.

256–257 slow and fast acetylators: P. Vineis and G. Ronco, "Interindividual Variation in Carcinogen Metabolism and Bladder Cancer Risk," *EHP* 98 (1992): 95–99.

256–257 One researcher offers the following reflection on the bladder cancer situation in England: "The continued use of known carcinogenic substances in British industry for many years after their identification, the wide range of industries with a known or suspected increased risk of bladder cancer, and our ignorance of the carcinogenic potential of many materials used in current manufacturing should be a cause for continuing concern" (R. R. Hall, "Superficial Bladder Cancer," *British Medical Journal* 308 [1994]: 910–13).

257 rise in bladder cancer incidence and its link to cigarette smoking: D. T. Silverman, "Urinary Bladder," in A. Harras (ed.), NIH Pub. 96-691 *Cancer Risks and Rates* (Bethesda, Md.: NCI, 1996), 197–99. Routine screening for bladder cancer is not done. Thus earlier detection or improved diagnostic techniques are unlikely explanations for recent increases in incidence rates. P. A. Schulte et al. (eds.), "Bladder Cancer Screening in High-Risk Groups," *Journal of Occupational Medicine* 32 (1990): 787–945.

257–258 *o*-toluidine: EPA, *1992 Toxics Release Inventory: Public Data Release*, EPA 745-R-001 (Washington, D.C.: EPA, 1994), 79.

258 quote from carcinogen report: USDHHS, *Seventh Annual Report on Carcinogens* (Research Triangle Park, N.C.: USDHHS, 1994), 389.

258 1996 study: E. M. Ward et al., "Monitoring of Aromatic Amine Exposure in Workers at a Chemical Plant with a Known Bladder Cancer Excess," *JNCI* 88 (1996): 1046–52.

258 another recent investigation: R. Ouellet-Hellstromt and J. D. Rench, "Bladder Cancer Incidence in Arylamine Workers," *Journal of Occupational and Environmental Medicine* 38 (1996): 1239–47; J. D. Rench et al., *Cancer Incidence Study of Workers Handling Mono- and Di-arylamines Including Dichlorobenzidine, Ortho-toluidine, and Ortho-dianisidine* (Falls Church, Va.: SRA Technologies, 1995); "Study Finds Bladder Cancer Threat among Conn. Plant Workers," *Boston Globe*, 21 Sept. 1995, p. 42.

259 focus of cancer research: Francis Collins, Richard Klausner, and Kenneth Olden, statement on cancer, genetics, and the environment before the Senate Committee on Labor and Human Resources, 6 Mar. 1996 (USDHHS press release).

259 fewer than 10 percent of all malignancies involved inherited mutations: NCI, *Understanding Gene Testing*, NIH Pub. 96-3905 (Bethesda, Md.: NCI, 1995).

259 hereditary colon cancer: G. Marra and C. R. Boland, "Hereditary Nonpolyposis Colorectal Cancer: The Syndrome, the Genes, and Historical Perspectives," *JNCI* 87 (1995): 1114–25; N. Papadopoulos et al., "Mutation of a *mutL* Homolog in Hereditary Colon Cancer," *Science* 263 (1994): 1625–29.

259 definition of *sporadic*: Bert Vogelstein, "Heredity and Environment in a Common Human Cancer" (lecture at Harvard Univ. Medical School, 3 May 1995). In exploring the use of the term *sporadic* by cancer researchers, historian Robert Proctor observed, "The presumption is apparently that heredity is orderly, while environmental causation is chaotic, perhaps even indecipherable. . . . Genetics offers hope for new forms of therapy, but also seems to imply resignation with regard to the possibility of prevention." See R. N. Proctor, *Cancer Wars: How Politics Shapes What We Know and Don't Know About Cancer* (New York: Basic Books, 1995), 245.

259 breast cancer's hereditary connection: Five to 10 percent is the estimate most often cited. A recent prospective cohort study of more than 100,000 women placed this figure even lower—at about 2.5 percent. See G. A. Colditz, "Family History, Age, and Risk of Breast Cancer: Prospective Data from the Nurses' Health Study," *JAMA* 270 (1993): 338–43.

259–260 DNA repair and hereditary colon cancer: D. Holzman, "Mismatch Repair Genes Matched to Several New Roles in Cancer," *JNCI* 88 (1996): 950–51.

260 pink and blue brochure: "Cancer Prevention" (pamphlet) (Bethesda, Md.: USDHHS, n.d.).

260–261 genetics textbook: G. Edlin, *Human Genetics: A Modern Synthesis*, 2nd ed. (Boston: Jones & Bartlett, 1990). Quotes are from pp. 184–204.

262 quote by Martha Balshem: M. Balshem, *Cancer in the Community: Class and Medical Authority* (Washington, D.C.: Smithsonian Institution Press, 1993), 3.

262–263 lifestyle factors and cholera: C. E. Rosenberg, *The Cholera Years: The United States in 1832, 1849, and 1866* (Chicago: Univ. of Chicago Press, 1962), 1–60.

263 Some researchers argue that "delayed childbirth" among white women explains much of the elevated incidence of breast cancer in the northeastern states. See S. R. Sturgeon, "Geographic Variation in Mortality

from Breast Cancer among White Women in the United States," *JNCI* 87 (1995): 1846–53.

263 majority of breast cancers unexplained by lifestyle: M. P. Madigan, "Proportion of Breast Cancer Cases in the United States Explained by Well-Established Risk Factors," *JNCI* 87 (1995): 1681–85.

263 quote by Robert Millikan: Personal communication.

263 total dietary fat and breast cancer: D. J. Hunter et al., "Cohort Studies of Fat Intake and the Risk of Breast Cancer—A Pooled Analysis," *NEJM* 334 (1996): 356–61; D. J. Hunter and W. C. Willett, "Diet, Body Size, and Breast Cancer," *Epidemiology Reviews* 15 (1993): 110–32; E. Giovannucci et al., "A Comparison of Prospective and Retrospective Assessments of Diet in the Study of Breast Cancer," *AJE* 137 (1993): 502–11. The role of dietary fat and creating breast cancer risk remains uncertain in part because the range of fat intake among the various groups of women studied has, so far, been relatively narrow.

263–264 as two leading researchers have observed, energy intake from fat has been declining as breast cancer has increased: Hunter and Willett, "Diet, Body."

264 Drs. Devra Lee Davis, Samuel Epstein, and Janette Sherman are among the researchers calling for a more ecological approach to diet. See S. S. Epstein, "Environmental and Occupational Pollutants Are Avoidable Causes of Breast Cancer," *International Journal of Health Services* 24 (1994): 145–50; and J. Sherman, *Chemical Exposure and Disease: Diagnostic and Investigative Techniques* (Princeton, N.J.: Princeton Scientific Publishing, 1994), 83.

264 Consumption of animal fat (or meat) is most strongly linked to colon and prostate cancers. See W. C. Willett, "Diet and Nutrition," in D. Schottenfeld and J. F. Fraumeni Jr. (eds.), *Cancer Epidemiology and Prevention*, 2nd ed. (Oxford, England: Oxford Univ. Press, 1996), 438–61.

264 reproduction, breast development, and the environment: N. Krieger, "Exposure, Susceptibility, and Breast Cancer Risk," *Breast Cancer Research and Treatment* 13 (1989): 205–23; S. G. Korenman, "Oestrogen Window Hypothesis of the Aetiology of Breast Cancer," *Lancet* 1980: 700–701.

264 organochlorine exposure and early puberty in rats: This topic is currently under exploration by Dr. Mary Wolff, who is interested in all factors—including childhood diet and level of physical activity—that contribute to the onset of puberty in girls. M. S. Wolff, "Organochlorines and Breast Cancers," presentation at the American Public Health Association, New York, 20 Nov. 1966. See L. M. Walters et al., "Purified Methoxychlor Stimulates the Reproductive Tract in Immature Female Mice," *Reproductive Toxicology* 7 (1993): 599–606; P. L. Whitten et al., "A Phytoestrogen Diet Induces the Premature Anovulatory Syndrome in Lactionally Exposed Female Rats," *Biology of Reproduction* 49 (1993): 1117–21; R. J. Gellert, "Uterotropic Activity of Polychlorinated Biphenyls and Induction of Precocious Reproductive Aging in Neonatally Treated Female Rats," *Environmental Research* 16 (1978): 123–30.

264 grand arguments over cancer causes: See, for example, R. Doll and R. Peto, *The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today* (Oxford, England: Oxford Univ. Press, 1981); and rebuttal by S. S. Epstein and J. B. Swartz, "Fallacies of Lifestyle Cancer Theories," *Nature* 289 (1981): 127–30.

264–265 the percentages game: Described in R. N. Proctor, *Cancer Wars: How Politics Shapes What We Know and Don't Know about Cancer* (New York: Basic Books, 1995), 54–74. See also J. M. Kaldor and K. A. L'Abbé, "Interaction between Human Carcinogens," in H. Vainio et al. (eds.), *Complex Mixtures and Cancer Risk*, IARC Scientific Pub. 104 (Lyon, France: IARC, 1990), 35–43.

265 cancer control policies embrace lifestyle and downplay environment: The ACS does not discuss environmental factors in its recent report on cancer prevention. See ACS, *Cancer Risk Report: Prevention and Control, 1995* (Atlanta: ACS, 1995). See also K. R. McLeroy, "An Ecological Perspective on Health Promotion Programs," *Health Education Quarterly* 15 (1988), 351–77.

265–266 quote from Illinois cancer report: IDPH, *Cancer Incidence in Illinois by County, 1985–87*, Supplemental Report (Springfield, Ill.: IDPH, 1990), 7–8.

266 Rachel Carson on environmental human rights: Senate testimony hearings before the Subcommittee on Reorganization and International Organizations of the Committee on Government Operations, "Interagency Coordination in Environmental Hazards (Pesticides)," U.S. Senate, 88th Congress, 1st session, 4 June 1962.

266 Carson's belief: Carson, *Silent Spring*, (Boston, Mass.: Houghton Mifflin, 1962), 277–78.

268 we do not all bear equal risks: F. Perera, "Uncovering New Clues to Cancer Risk," *Scientific American*, May 1996, pp. 54–62; S. Venitt, "Mechanisms of Carcinogenesis and Individual Susceptibility to Cancer, *Clinical Chemistry* 40 (1994): 1421–25; G. W. Lucier, "Not Your Average Joe" (editorial), *EHP* 103 (1995): 10.

268–269 2 percent estimate: Harvard Center for Cancer Prevention, "Harvard Report on Cancer Prevention," *Cancer Causes and Control* 7, suppl. 1 (1996): 3–59; D. Trichopoulos et al., "What Causes Cancer?" *Scientific American*, Sept. 1996, pp. 80–87.

269 other estimates: Proctor, *Cancer Wars*.

269 10,940 is 2 percent of 547,000, the projected figure for total cancer deaths in 1995. See ACS, *Cancer Facts and Figures—1995*, rev. (Atlanta: ACS, 1995).

269 anonymity and homicide: The environmental analysts Paul Merrell and Carol Van Strum have argued that the concept of acceptable risk is tolerated only because of the anonymity of its intended victims. See P. Merrell and C. Van Strum, "Negligible Risk: Premeditated Murder?" *Journal of Pesticide Reform* 10 (1990): 20–22. Likewise, the molecular biologist and physician John Gofman has argued, "If you pollute when you DO NOT KNOW if there is any safe dose (threshold), you are performing improper experimentation on people without their informed consent. . . . If you pollute when you DO KNOW that there is no safe dose with respect to causing extra cases of deadly cancers, then you are committing

premeditated random murder" (J. W. Gofman, memorandum to the U.S. Nuclear Regulatory Commission, 21 May 1994).

269 quote from the ATSDR: ATSDR, *FY 1993 Agency Profile and Annual Report* (Atlanta: ATSDR, 1993), 15.

270 number of carcinogens in the environment: M. Eubanks, "Biomarkers: The Clues to Genetic Susceptibility," *EHP* 102 (1994): 50–56.

270 Rachel Carson's observation: Carson, *Silent Spring*, 248. See also M. J. Kane, "Promoting Political Rights to Protect the Environment," *Yale Journal of International Law* 18 (1993): 389–411.

270 precautionary principle: This principle was endorsed in 1987 by European environmental ministers in a meeting about the deterioration of the North Sea. (K. Geiser, "The Greening of Industry: Making the Transition to a Sustainable Economy," *Technology Review*, Aug.–Sept. 1991, pp. 65–72.) See also T. O'Riordan and J. Cameron (eds.), *Interpreting the Precautionary Principle* (London: Earthscan, 1994).

270 dead body approach: Devra Lee Davis, quoted in "Is There Cause for 'Environmental Optimism'?" *Environmental Science and Technology* 29 (1995): 366–69.

270 principle of reverse onus: This principle has been embraced by the International Joint Commission in their Eighth Biennial Report on Great Lakes Water Quality (Washington, D.C., and Ottawa, Ontario: International Joint Commission, 1996), 15–17. See also discussions of proof in T. Colborn et al., *Our Stolen Future: Are We Threatening Our Fertility, Intelligence, and Survival?—A Scientific Detective Story* (New York: Dutton, 1996); and G. K. Durnil, *The Making of a Conservative Environmentalist: With Reflection on Government, Industry, Scientists, the Media, Education, Economic Growth, and the Sunsetting of Toxic Chemicals* (Bloomington: Indiana Univ. Press, 1995).

270 principle of the least toxic alternative: My ideas on this topic are inspired in part by those of biologist Mary O'Brien. See M. H. O'Brien, "Alternatives to Risk Assessment: The Example of Dioxin," *New Solutions* (winter 1993): 39–42; and K. Geiser, "Protecting Reproductive Health and the Environment: Toxics Use Reduction," *EHP* 101, suppl. 2 (1993): 221–25.

270 quote by Dr. Mary O'Brien: personal communication.

271–272 abnormal changes in juvenile rats: M-H. Li and L. G. Hansen, "Enzyme Induction and Acute Endocrine Effects in Prepubertal Female Rats Receiving Environmental PCB/PCDF/PCDD Mixtures," *EHP* 104 (1996): 712–22.

INDEX