probably owing to improvements in food handling and the increased consumption of fresh foods made possible when refrigeration replaced more toxic methods of food preservation, such as smoking, salting, and pickling. Pap smears have been credited with bringing down the incidence of cervical cancer because precancerous lesions can be detected and cut out before they are transformed into invasive tumors.

However, these modest gains are swamped by the cancers that show both increasing incidence and increasing mortality: cancers of the brain, liver, breast, kidney, prostate, esophagus, skin (melanoma), bone marrow (multiple myeloma), and lymph (non-Hodgkin’s lymphoma) have all escalated over the past twenty years and show long-term increases that can be traced back at least forty years. In recent years, breast cancer mortality among white women has begun to slow down, declining 6.8 percent from 1989 to 1993. However, the death rate is still higher than it was when Rachel Carson died of the disease in 1964, and it is still rising for black women. Moreover, breast cancer incidence rates are still rising for localized disease even as they are falling for more advanced-stage diagnoses (a shift probably indicating that breast cancer is being detected and treated earlier); the proportion of women developing the disease at all remains at the highest level ever recorded.

“Explanations for these increases do not exist,” according to Philip Landrigan, a pediatrician and leading public health researcher. Medical literature is accustomed to summations more temperate and indirect, but this one has been echoed again and again in recent research papers on trends in cancer rates. In a 1995 assessment of the situation, a research team at the National Cancer Institute similarly concluded, “Some trends remain unexplained . . . and may reflect changing exposures to carcinogens yet to be identified and clarified.”

Clarification about carcinogens, Landrigan believes, requires an environmental line of inquiry:

The possible contribution to recent cancer trends of the substantial worldwide increases in chemical production that have occurred since World War II (and the resulting increases in human exposure to toxic chemicals in the environment) has not been adequately assessed. It needs to be systematically evaluated.

I have read the preceding two sentences many times. Most of my life spans the time between Carson’s call for a systematic evaluation of the contribution of toxic chemicals to increased human cancers and Landrigan’s repetition of this call. Both give one pause.

I am struck also by the symmetry between Landrigan’s recommended course of action and an observation made thirty years earlier by two senior scientists at the National Cancer Institute, Wilhelm Hueper and W. C. Conway. “Cancers of all types and all causes display even under already existing conditions, all the characteristics of an epidemic in slow motion.” This unfolding crisis, they asserted, was being fueled by “increasing contamination of the human environment with chemical and physical carcinogens and with chemicals supporting and potentiating their action.” And yet the possible relationship between cancer and what Hueper and Conway called “the growing chemicalization of the human economy” has not been pursued in any systematic, exhaustive way.

The environment, it seems, keeps falling off the cancer screen. The circumstances surrounding the birth of the Illinois State Cancer Registry is a case in point. The registry came into being when the Illinois Health and Hazardous Substances Registry Act was signed by the governor in September 1984. As implied by its name, this state law was intended to “monitor the health effects among the citizens of Illinois related to exposures to hazardous substances in the work place and in the environment.” Accordingly, the registry system was to collect information not only on the incidence of cancer among the Illinois populace but also on their “exposure to hazardous substances, including hazardous nuclear material,” thus prompting public health studies that would relate “measurable health outcomes to environmental data to help identify contributing factors in the occurrence of disease.”

The cancer registry was funded. The hazardous substances registry was not.

Like a thriving child with a stillborn twin, the Illinois State Cancer Registry dutifully acquires information on health outcomes, but
About the rest, we heard various dire predictions from adults: perhaps they would all be felled by police truncheons or end up crazed and deafened from rock and roll. But I never heard anyone's grandmother predict that those born in the 1940s would surely undergo chemotherapy regimens in record numbers or that a cancer diagnosis would become as significant a generational marker as patchouli oil.

Nothing slows time down as much as waiting for lab reports. This time I am the patient. In the interior waiting room, dressed in a wrap-around smock identical to the ones worn by other human beings who have entered this room, I try to conjure Jeannie out of thin air. Of the ample supply of magazines provided us here, she would choose Vanity Fair. Of this, I feel certain. During these moments of waiting, which celebrity interview would she, in her unflagging attempt to bring me up to snuff on popular culture, read aloud to me? And when I drifted into anxious thinking, what clever thing would she say to keep me from floating off too far?

Last summer she waited with me for hours at the ultrasound clinic.

"They had a hard time seeing what they wanted to see," I reported back to her as we finally walked out the door. "And then one of the technicians looked at the image in the monitor and whistled."

She laughed. "You know that ranks right up there with 'Hey, nice tits!'."

My name is called and I follow the doctor down the corridor to her office. Like a defendant studying the faces of the jurors as they file back into the courtroom, I try to read her expression.

It seems my situation today is mostly good, but a little bit ambiguous. The specialists have conferred and would like to recommend I undergo a new type of test, which the doctor explains in clear detail.

"I know this isn't what you wanted to hear," she says, with genuine compassion. "But you don't need me to be your best friend right now."

Time lurches forward again. Where is she?

The rise in cancer incidence over calendar time is one line of evidence that implicates environmental factors. The increase in cancer incidence among successive generations is another. A third line of evidence comes from a close consideration of the cancers that exhibit particularly rapid rates of increase. If we restrict our view to these cancers, what patterns emerge? Who gets these cancers and what do we know about their possible causes?

After lung cancer in women, the three cancers ascending most swiftly in the United States are melanoma of the skin, non-Hodgkin's lymphoma, and multiple myeloma. These are not the most prevalent cancers—breast cancer remains the most frequently diagnosed cancer in women, for example—but these are the ones galloping forward at the fastest rate.

Melanoma accounts for only 5 percent of all skin cancers, but it is the most dangerous kind, accounting for 75 percent of skin cancer deaths. The U.S. incidence of melanoma rose nearly 350 percent between 1950 and 1991, and mortality rose by 157 percent. Between 1982 and 1989 alone, melanoma incidence jumped 83 percent. Each year, about 4 percent more people contract melanoma than the year before, and the average age at diagnosis is going down. The more common basal cell and squamous cell skin cancers are also on the rise. But because they rarely spread to other parts of the body and are seldom life-threatening, these skin cancers are not even included in cancer registry data. Only melanoma diagnoses are recorded in U.S. registries.

A melanoma is a cancer that begins in a melanocyte, a cell type that surely serves as the excuse for more wars, social strife, injustice, and oppression than any other human tissue. Melanocytes are the pigment-producing cells of the skin. Those who ponder the origins of racism would do well to consider the humble biology of the melanocyte. Comprising only about 8 percent of all skin cells, the melanocytes appear in microscopic cross section as dark, delicate shrubbery. They are surrounded by Langerhans cells, which migrate up from the bone marrow and play a role in immunity, and by keratinocytes, layers of flat stepping stones that comprise 90 percent of our epidermis and produce a waterproofing protein. The
melanocytes' slender branches extend between and around the keratinocytes and deliver to them the molecules of melanin they cannot produce on their own. Once inside the keratinocytes, the blackish-brown granules float to the surface and form a sunlight-absorbing cloak that lies over the fragile chromosomes inside the nuclei. Exposure to ultraviolet radiation—that high-energy wavelength lying just below violet in the spectrum of visible light—stimulates the melanocytes to make more melanin and in darker shades. More grains of melanin are dispatched to the keratinocytes. Therefore, we tan.

Everyone, regardless of race, has approximately the same number of melanocytes. Differences in skin color represent differences in the amount of melanin produced. Not everyone, however, has an equal chance of contracting melanoma. Incidence among whites is ten times higher than among blacks. Among white men, the disease more often originates in a melanocyte located somewhere on the trunk of the body; among white women, on the lower leg. When a melanocyte becomes cancerous—multiplying out of control and, if undetected, seeding itself in deeper and more distant parts of the body—its pigment-producing activities do not stop. The dark interior spaces of the body, where rays of sunlight never penetrate, thus become filled with black tumors that go on crazily producing molecules of light-shielding melanin with no companion cells to receive them.

Melanomas are clearly associated with exposure to ultraviolet radiation (albeit in a complicated way that is a matter of some debate), and here is where individual behavior and changes in the global environment come together. Basal and squamous cell cancers, which arise from keratinocytes, appear to increase in proportion to one's cumulative lifetime exposure to sunlight. Melanomas, by contrast, are thought to be initiated by acute exposures, such as a bad sunburn in childhood. In essence, the cells designed to protect us from the chromosome-breaking effects of the sun are themselves damaged by an overdose of the very element they strive to shield us from. Decades later, another insult of some kind causes wild cell divisions within the damaged melanocyte to commence. A melanoma forms. A borderline is crossed. This second event may be more sunlight, but it may also include exposure to certain chemicals. Excess rates of melanoma are found in rubber and plastics workers, as well as in those employed in electronics and metal industries.

The accelerating incidence of melanoma means exposure to ultraviolet radiation is probably increasing. This could be happening for two reasons. First, more people are spending more time in the sun. Second, the sunlight to which we are exposed contains more ultraviolet rays. Since the 1974 discovery that earth's ultraviolet-shielding ozone layer is thinning, a growing group of physicians and climatologists have come to believe both forces are at work, especially in raising the risk for future melanomas. The U.S. Environmental Protection Agency (EPA) projects that tens of thousands of additional fatal skin cancers will result from the 5 percent loss of ozone that has already occurred in the stratosphere above North America. Individual behavior also plays a role. Melanomas have been on the rise for many decades—since Coco Chanel first popularized the suntan in the 1930s, according to some researchers. However, the worldwide increase in melanoma incidence points to a role for ecological factors. A recent study published in the *Journal of the American Academy of Dermatology* observes:

> Because of the worldwide increase in melanoma incidence, global factors need to be considered as potentially involved. Stratospheric ozone depletion, allowing more intense UV light to reach the earth's surface, may, in part, be responsible.

Ultraviolet—or UV—light is a strange energy. It is responsible for creating the very layer of ozone that defends us from it. As UV rays stream into the stratosphere, they cleave oxygen molecules in half, creating free atoms of oxygen, which then react with intact oxygen pairs to form little triangles called ozone. Ozone in turn can absorb UV rays, preventing their further passage down miles of deep air to the earth's surface. The ozone layer intercepts some, but not all, of the UV rays beamed at us from the sun.

Chemicals responsible for destroying the layer of ozone that resides twelve to thirty miles above us include the now notorious chlorofluorocarbons (CFCs). They are unlikely culprits. Not carcinogenic or even toxic, CFCs belong to a big family of synthetic, organic, chlorinated chemicals whose other members—DDT and
PCBs, for example—certainly are. But however harmless at ground level, a CFC molecule behaves quite differently when wind currents sweep it into the ozone layer. Ultraviolet rays split the CFC molecule apart, releasing a chlorine atom that quickly reacts with a molecule of ozone. The triangle of oxygen falls apart as the chlorine temporarily binds with one member of the triad. An unstable union, the chlorine atom soon shakes free of its oxygen partner and goes on to react with and destroy other ozone molecules. Before the chlorine is finally enveloped by a raindrop and redeposited on the earth's surface, it may break apart some 100,000 ozone molecules.

Less ozone allows more UV rays to beam their way through the atmosphere and down to us. Some will be halted by the veil of melanin spread across our skin for that purpose. But if its absorptive capacity is exceeded—which happens easily to the fair-skinned among us—some rays will penetrate further inside the skin cells until they are absorbed by the DNA strands themselves. If this occurs inside a melanocyte, the resulting genetic damage can place this cell on the pathway toward melanoma. In this way, noncarcinogenic CFCs contribute to rising cancer incidence by intensifying incoming sunlight, thereby making it more carcinogenic.

Non-Hodgkin's lymphoma strikes at another tissue designed to protect us from harmful invasions: the knobby lymph nodes clustered in our throats, armpits, groins, and elsewhere. Our tonsils, the most accessible example, represent a constellation of lymph nodes wrapped in a mucous membrane.

The watery fluid that fills the microscopic spaces between all of our cells is, for all intents, lymph. It does not receive that name, however, until it flows from those spaces, like rainwater from a field, into the creekbeds called lymphatic vessels. The origin of all this fluid is the bloodstream, and when held within that system, it is known as plasma. Each day, about three quarts of blood plasma leak out of the capillaries, swirl around freely, and then drain into the lymph vessels. Eventually, lymph becomes plasma again when it is poured back into blood just at the point where the jugular vein joins the subclavian in their return to the heart. Several tasks are accomplished during the ceaseless transformation of lymph to plasma and plasma to lymph.

The identification and destruction of foreign substances is one of them. Lymph nodes, scattered along the lymph vessels at various intervals, are honeycombed with a diverse array of cell types specialized for immune response. As the fluid is channeled through the nodes' intricate meshwork, alien life forms are trapped and killed. Lymph nodes can also send immune-responsive cells forth to circulate in other territories of the body.

Because the lymph system also serves as a highway for runaway cancer cells of all kinds, lymph nodes are a significant feature in the cancer landscape. Breast cancers very often spread to nearby lymph nodes, for example. Breast cancer patients are quickly categorized as node positive or node negative, a distinction that depends on whether breast cancer cells, shed from the original tumor, have lodged themselves in the lymph nodes beaded between the arm and the trunk of the body. Their presence there indicates the disease has likely dispersed to other, more distant locations.

As a way of measuring the extent of this cancer diaspora, node-positive women are further classified by the number of nodes containing breast cancer cells: 1 to 4 is one kind of identity; 11 to 17 is quite another. "How many nodes?" is very often the first question women in breast cancer support groups ask each other.

But a lymphoma is a different condition. In this case, the tumors derive from lymph tissue itself, not from immigrant cells that have floated in from someplace else. Lymphoma can arise inside a node, or, because lymph tissue is diffused throughout the body, it can originate almost anywhere elsewhere—in the spleen, for example, or even in the skin. Non-Hodgkin's lymphoma is therefore a collection of diseases, in contrast to the very specific and highly curable lymphoma called Hodgkin's disease.

While the incidence of Hodgkin's disease has declined modestly over the past two decades, non-Hodgkin's lymphoma has shot up—approximately tripling in incidence since 1950. This increase is evident in both sexes and within all age groups except the very young. Non-Hodgkin's lymphoma is also far less curable than Hodgkin's disease. Jackie Kennedy Onassis was killed by one of its most malignant incarnations.

AIDS has contributed to some, but not all, of the increase in
non-Hodgkin's lymphoma. A small but significant percentage of AIDS patients are diagnosed with lymphoma, which for many causes death. However, the steady upward momentum of non-Hodgkin's lymphoma incidence in the United States was already under way decades before the AIDS epidemic sank its teeth in.

Lymphomas do seem to be consistently associated with exposure to synthetic chemicals, especially a class of pesticides known as phenoxy herbicides. These synthetic chemicals were born in 1942 as part of a never-implented plan by the U.S. military to destroy rice fields in Japan. The most famous phenoxy is a mixture of two chemicals, 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) and 2,4-dichlorophenoxyacetic acid (2,4-D). This combination is called Agent Orange, and it was used between 1962 and 1970 by U.S. troops to clear brush, destroy crops, and defoliate rainforests in Vietnam. The military career of phenoxy herbicides was thus revived.

Linked to miscarriages and contaminated with dioxin, 2,4,5-T was eventually outlawed. By contrast, 2,4-D went on to become one of the most popular weed killers in lawns, gardens, and golf courses, as well as in farm fields and timber stands. It has been marketed under a schizophrenic collection of trade names: Ded-Weed, Lawn-Keep, Weedone, Plantgard, Miracle, Demise.

Evidence for an association between phenoxy herbicides and non-Hodgkin's lymphoma comes from several corners. Vietnam veterans have high rates of non-Hodgkin's lymphoma. So do farmers in Canada, Kansas, and Nebraska who use 2,4-D. Studies show that the risk of lymphoma to farmers rises with the number of days per year of use, the number of acres sprayed, and the length of time they wear their “application garments” before changing clothes. In Sweden, exposure to phenoxy herbicides was shown to raise one’s risk of contracting lymphomas six-fold. In a comprehensive review of the topic, the National Cancer Institute scientists Sheila Hoar Zahn and Aaron Blair concluded:

Incidence of NHL has increased, which could explain at least part of the rising incidence.

Similarly, an 812-page study of herbicide-exposed Vietnam veterans conducted by the Institute of Medicine offered the following terse opinion:

Evidence is sufficient to conclude that there is positive association. That is, a positive association has been observed between herbicides and the outcome [non-Hodgkin's lymphoma] in studies in which chance, bias, and confounding could be ruled out with reasonable confidence.

Dogs also acquire lymphoma. One recent study showed that pet dogs living in households whose lawns were treated with 2,4-D were significantly more likely to be diagnosed with canine lymphoma than dogs whose owners did not use weed killers. Risk rose with number of applications: the incidence of lymphoma doubled among pet dogs whose owners applied lawn chemicals at least four times per year.

From jungle warfare to suburban dandelions: Our ongoing war against plants is now waged on a domestic grid of tiny battlefields. One in ten single-family American households now uses commercial lawn care services, and one in five applies the chemicals themselves. The evidence linking phenoxy compounds to non-Hodgkin's lymphoma is preliminary. No one knows exactly how traces of weed killer find their way into our extracellular fluid as it is funneled back and forth between blood and lymph. Absorption through the skin is considered the most likely route of exposure. No one has explicated the exact mechanism by which these chemicals might alter the cells inside the far-flung network of nodes, canals, and lymph tissues—otherwise and thereby set the stage for a lymphoma. No one knows whether phenoxy requires interactions with other agents to work their damage nor what proportion of the current rise in lymphoma might be attributed to phenoxy exposure.

Most of us are probably far less exposed to phenoxy herbicides than soldiers, farmers, or even our own beloved dogs who use our lawns for their bedrooms. Nevertheless, the presence of disease in these specific groups is a clue to which we, as readers of a complicated
mystery, need to pay attention when trying to determine why non-Hodgkin's lymphoma casts an ever-longer shadow among us all.

Bone marrow is the mother of lymphocytes, the immune cells that inhabit the lymph nodes. Multiple myeloma is cancer of the cells inside the bone marrow that give rise to a particular type of lymphocyte called plasma cells. Its main symptom is horrible pain. As the tumors grow, blood, lymph, and bone marrow are filled with an excess of abnormal plasma cells, which then churn out an excess of abnormal antibodies. The bones themselves, riddled with lesions, begin to fracture. Calcium spills into the bloodstream. Although multiple myeloma is thought to begin with a single mutation in a single cell, tumors created by aggregations of plasma cells are usually diffusely present throughout the bone marrow by the time of diagnosis. The skull is often severely affected.

As with non-Hodgkin's lymphoma, the incidence of multiple myeloma in the United States has approximately tripled since 1950, and the mortality rate is not far behind. As a sign of its rise from obscurity, some cancer newsletters now run announcements for multiple myeloma support groups. (In San Francisco, the third Saturday of each month at the Women's Cancer Resource Center is multiple myeloma day.)

There is much less to say about myeloma than about the other contenders for Most Swiftly Moving Cancer. Much less is known. It tends to stalk the elderly, and blacks are at higher risks than whites—but for unclear reasons. Because the presence of abnormal antibodies in the blood and urine provides a definitive diagnosis, the registration of multiple myeloma is considered to be particularly accurate.

Exposure to ionizing radiation is recognized as one probable cause. U.S. radiologists exhibit excess rates of multiple myeloma, as do survivors of the 1945 atomic bomb blasts in Japan. Some evidence suggests that workers in the nuclear industry also have increased risks for myeloma.

In contrast to solar radiation, such as UV light, ionizing radiation has energy sufficient to penetrate the skin's surface, stream through the soft tissues, and in some cases, enter the bones themselves. Released when atoms are split, ionizing radiation is so called because it alters the molecules through which it passes, knocking away their electrons and creating electrically charged particles, or ions. Because of this property, ionizing radiation is classified as a known human carcinogen at any exposure level. When the atomic modifications induced by radiation involve molecules of DNA, as they are into the nucleus of every cell, cancer-inducing mutations can result. Alternatively, radiation can create ions of surrounding atoms, which then bind with DNA to create mutations. In either case, our chromosomes are equipped with DNA repair mechanisms designed to detect and correct such problems, but it is a system that can be overwhelmed and overpowered. Multiple myeloma is one of the most recent cancers to be linked with exposure to radiation; the lag time between exposure and diagnosis is much longer than that for other cancers caused by irradiation of the bone marrow.

Multiple myeloma is also associated with exposure to a variety of chemicals—metals, rubber, paint, industrial solvents, and petroleum. Farmers and agricultural workers exposed to pesticides and herbicides have higher rates of multiple myeloma than the general population. Multiple myeloma is on the rise in all major industrialized countries. But the parallel increase among both sexes argues against a purely occupational cause. According to one researcher who has examined multinational mortality trends, the patterns of multiple myeloma among generational cohorts suggest a general environmental exposure of some kind, common to all industrialized countries, which would have begun increasing at the turn of the twentieth century.

Other researchers urge an investigation of one very specific industrial chemical: benzene. Consisting of a simple ring of six carbon atoms, benzene is used as a solvent in which other petrochemicals are dissolved, as an additive to gasoline, and as a raw material for the creation of synthetic materials including certain foams, plastics, and pesticides. It is a ubiquitous pollutant of outdoor and indoor air and a common contaminant of drinking water. Benzene can pass through the waterproofed layer of our skin and thus seep into blood upon direct contact; it also evaporates quickly and can be easily inhaled.

Benzene is a suspect in myeloma because it is a known offender in a related crime, namely, leukemia. A proven bone marrow toxin, benzene alters the cells of the marrow that give rise to leukocytes, or white blood cells. Could the same toxin also preside over alterations in the marrow's production of plasma cells? According to the U.S.
Agency for Toxic Substances and Disease Registry, “Although this is plausible, no scientific proof of a causal relationship exists.” The question thus becomes, Is anyone looking?

Bone marrow. Lymph nodes. Skin. From the body’s dark tunnels to its sunlit surface, cancers of all kinds are presenting themselves with increasing frequency. Melanoma, lymphoma, and multiple myeloma are simply traveling at especially high velocities.

A month before her death, Jeannie initiated a massive housecleaning project. She reorganized all her files, returned books, gave away clothing. Waiting for me on her kitchen table one morning was a stack of medical papers, department of public health reports, press releases, and newspaper clippings. They were her collection of articles about the cluster of cancer cases in southeastern Massachusetts, where she grew up.

“I thought you might want them for your research.”
“You don’t want to keep these?”
“You take them.”

Eighteen months after Jeannie’s death, I finally read them—prompted by the release of a new study confirming the patterns documented by the previous ones. Jeannie’s cancer is not included in any of these studies, which concern sharply rising leukemia rates in five neighboring towns during the 1980s and their possible relationship to documented radioactive releases at the Pilgrim nuclear power plant—the result of a fuel rod problem—ten years earlier. While no firm cause-and-effect relationship has been established, meteorological data indicate that coastal winds may have trapped the airborne radioactive isotopes and recycled them within a five-town area.

“Individuals with the highest potential for exposure to Pilgrim emissions... had almost four times the risk of leukemia as compared with those having the lowest potential for exposure.”

Although one of the towns is her own, Jeannie’s cancer was far too rare for the case-control comparisons made here. Her cancer has no known cause, and cancer registries do not track its incidence. I will not find her here.

Pekin is the judicial seat of Tazewell County, Illinois. It is situated across the Illinois River and a few miles downstream from Peoria. Just outside of Pekin’s city limits, about two miles west of the house I grew up in, is the unincorporated subdivision of Normandale. The community was created in 1926 to provide housing for factory workers, and its streets are named for the original prewar products that the residents who slept here at night toiled by day to create: Karo Street (after the syrup), Quaker Street (after the paper mill’s round oatmeal boxes), Fleischmann Street (after the yeast).

Normandale is home to 480 people, a popular supper club, a beautiful brick church, and a root-beer stand where I hung out with my best friend in the summers after we learned to drive. Eating onion rings in her father’s car, Gail Williamson and I debated the merits of German versus Latin, big universities versus small colleges, sex versus