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Reclaiming the Environmental Debate

The Politics of Health in a Toxic Culture

edited by Richard Hofrichter

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The Social Production of Cancer: A Walk Upstream

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In 1979, in between my sophomore and junior year in college, I was diagnosed with bladder cancer. Four years later, while a doctoral student in biology, I took the train from Ann Arbor, Michigan, to central Illinois for an appointment with my original urologist. This particular appointment was destined to turn out fine: there were no recurrences. 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 slowly moving away from each other. Lines become more sharply drawn. These changes always make me restless and when driving, I 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.[1] Through this process, they located the segment of DNA responsible for the transformation. By

comparing this segment with 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 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.[2] Besides the oncogene just described, two tumor suppressor 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.[3] 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. 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.[4] These genetically susceptible 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.[5] 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. Sadly, all of our knowledge about genetic mutations, inherited risk factors, and enzymatic mechanisms has not been 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.[6] The question thus 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 continuing 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.[7] 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,* published by the U.S. Department of Health and Human Services, 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."[8] A 1996 study investigated a sixfold excess of bladder cancer among workers exposed years earlier to *o*-toluidine and aniline in the rubber chemicals department of a manufacturing plant in upstate New York.[9] 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.[10]

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. Trihalomethanes—common contaminants of chlorinated tapwater—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? However improved the record of effort to regulate them, why have they all not been replaced by safer substitutes? To my knowledge, these questions remain largely unaddressed by the cancer research community.

Biased Focus

Genes

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.[11] 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.

Hereditary cancers, however, are the rare exception. Collectively, fewer than 10 percent of all malignancies are thought to involve inherited mutations.[12] 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.[13] The remaining 85 percent of colon cancers are officially classified as "sporadic," which essentially means that we don't know what causes them.[14] Breast cancer also shows little connection to heredity (probably between 5 and 10 percent).[15] Finding "cancer genes" is not going to prevent the great majority of cancers that develop.

Moreover, even when inherited mutations do 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.[16] 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.

Lifestyle

Risks of lifestyle are also not independent of environmental risks. 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 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.[17]

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.[18]

(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 identifies 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 identified as a carcinogen to which workers making polyvinyl chloride (PVC) 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 present the cause of the disease as a problem of *behavior* rather than one 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 continuing 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?

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.[19] Lists of cholera prevention tips were posted publicly. Their advice ranged from avoiding drafts and raw vegetables to abstaining from alcohol. Maintaining "regular" habits was also said to be protective. Decades later, improvements in public sanitation 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. Scores of cheerful pamphlets exhort women 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.)[20]

By itself, a lifestyle approach to preventing breast cancer is inadequate.[21] 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.

Finally, the adage that high-fat Western diets are the cause of breast cancer has not yet been supported by data.[22] Dietary fat has long been a centerpiece of study in the investigation of breast cancer risk. Yet, several

long-term studies have indicated that dietary fat is unlikely to play a major role by itself.[23] Rather than continuing to focus single-mindedly on the absolute quantity of fat consumed, several researchers have called for a more refined, ecological approach to diet.[24] 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.[25]

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 differentiates 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 principal 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.[26] Investigators have repeatedly confirmed that reproductive history contributes to breast cancer risk. We need to know now, Krieger argues, whether women with similar 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.[27] 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.[28] 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"—make 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 (programmed death of damaged cells), 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 death toll from environmental factors 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"[29] 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 environmental factors, perceived as making a small contribution to the cancer problem, is seen as inefficient.[30] Moreover, the rationale continues, not enough is known about environmental risks to make specific recommendations. (On the other hand, incomplete and inconsistent evidence about the role of dietary fat in contributing to breast cancer does not appear to be 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 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."[31] It never mentions or considers that Illinois is a leading producer of hazardous waste, a heavy user of pesticides, and home to an

above-average number of Superfund sites. Nor does this report correlate cancer statistics with Toxics Release Inventory data or attempt 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 the 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.

The Right to Know

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.[32] 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.[33]

The process of exploration that results from asserting our right to know about carcinogens in our environment is a different journey for every person. 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 at our past, then reassess our present situation, and finally summon the courage to imagine an alternative future.

We must begin retrospectively for two reasons. First, we carry in our bodies many carcinogens that are no longer produced and used domestically, but which linger in the environment and in human tissue. Appreciating how even today we remain in contact with banned chemicals such as polychlorinated biphenyls (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 discover what pesticides were sprayed in our

neighborhoods and what sorts of household chemicals our parents stored under the 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 in and the molecules of which 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 grain fields, as well as cattle lots, orchards, pastures, and dairy farms. It demands curiosity about how pests in our apartment buildings are exterminated, how our clothing is cleaned, and how golf courses are maintained. It means asserting our right to know about any and all toxic ingredients in such products as household cleaners, paints, and cosmetics. It requires a determination to discover the location of underground storage tanks, how the land was used before a 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.

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 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.[34] 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 the 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 each of us does not 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.[35] Though others have placed this number far higher,[36] 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.[37] 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.[38]

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.[39] Whatever our past exposures, this is our current situation.

Guiding Principles for Reducing Toxics

After having carefully appraised the risks and losses that we have endured by tolerating this situation, 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.[40] 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 might cause irreparable damage.[41] 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 taking action.[42] 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.*[43] According to this edict, it is safety, rather than harm, that should necessitate demonstration. This reversal essentially shifts the burden of proof from the public to 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. 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 provide 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.[44] 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 assessment of alternatives 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. The highest priority for transformation should be assigned to 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 carcinogen released into the environment and where to set legal maximum limits for their presence in air, food, water, the 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" (personal communication, M. O'Brien, 1997). 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 reprehensible as the practice of slavery.

Notes

1. R. A. Weinberg, "A Molecular Basis of Cancer," *Scientific American* (November 1983):126–42.

2. I. Orlow et al., "Deletion of the pl6 and pl5 Genes in Human Bladder Tumors," *Journal of the National Cancer Institute* 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.

3. 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," *Environmental Health Perspectives* 102 (Suppl. 6) (1994):11–16; P. L. Skipper and S. R. Tannenbaum, "Molecular Dosimetry of Aromatic Amines in Human Populations," *Environmental Health Perspectives* 102 (Suppl. 6) (1994):17–21; S. M. Cohen and L. B. Ellwein, *Environmental Health Perspectives* 101 (Suppl. 5) (1994):111–14.

4. P. Vineis and G. Ronco, "Interindividual Variation in Carcinogen Metabolism and Bladder Cancer Risk, "*Environmental Health Perspective* 98 (1992):95–99.

5. 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* [1994]:910–13).

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

7. Environmental Protection Agency, *1992 Toxic Chemicals Release Inventory: Public Data Release.* EPA 745-R-001 (Washington, D.C.: EPA, 1994, p. 79).

8. U.S. Department of Health and Human Services, *Seventh Annual Report on Carcinogens* (Research Triangle Park, N.C.: USDHHS, 1994, p. 389.

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

10. 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,* September 21, 1995, p. 42.

11. Francis Collins, Richard Klausner, and Kenneth Olden, statement on cancer, genetics, and the environment before the Senate Committee on Labor and Human Resources, March 6, 1996 (U.S. Department of Health and Human Services press release).

12. National Cancer Institute, *Understanding Gene Testing,* NIH Pub. 96-3905 (Bethesda, Md.: NCI, 1995).

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

14. Bert Vogelstein, "Heredity and Environment in a Common Human Cancer," lecture at Harvard Univ. Medical School, May 3, 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, p. 245).

15. 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," *Journal of the American Medical Association* 2 (70) (1993):338–43.

16. D. Holzman, "Mismatch Repair Genes Matched to Several New Roles in Cancer," *Journal of the National Cancer Institute* 88 (1996):950–51.

17. "Cancer Prevention" (pamphlet) (Bethesda, Md.: U.S. Department of Health and Human Services, n.d.).

18. G. Edlin, *Human Genetics. A Modern Synthesis,* 2d ed. (Boston: Jones & Bartlett, 1990). Quotations are from pages 184–204.

19. C. E. Rosenberg, *The Cholera Years: The United States in 1832, 1849, and 1866* (Chicago: Univ. of Chicago Press, 1962, pp. 1–60).

20. 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," *Journal of the National Cancer Institute* 87 (1995):1846–53.

21. M. P. Madigan, "Proportion of Breast Cancer Cases in the United States Explained by Well-Established Risk Factors," *Journal of the National Cancer Institute* 87 (1995):1681–85.

22. D. J. Hunter et al., "Cohort Studies of Fat Intake and the Risk of Breast Cancer—A Pooled Analysis," *New England Journal of Medicine* **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," *American Journal of Epidemiology* **137** (1993):502–11. The role of dietary fat in 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.

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

24. 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, p. 83).

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

26. N. Krieger, "Exposure, Susceptibility, and Breast Cancer Risk," *Breast Cancer Research and Treatment* **13** (1989):205–23.

27. 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, November 20, 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.

28. 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: Oxford Univ. Press, 1981); and a rebuttal by S. S. Epstein and J. B. Swartz, "Fallacies of Lifestyle Cancer Theories," *Nature* 2(89) (1981):127–30.

29. Described in Proctor, *Cancer Wars,* pp. 54–74. See also J. M. Kaidor and K. A. L'Abbe, "Interaction between Human Carcinogens," in *Complex Mixtures and Cancer Risk,* H. Vainio et al., eds., IARC Scientific Pub. 104 (Lyon, France: International Agency for Research on Cancer, 1990, pp. 35–43).

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

31. Illinois Department of Public Health, *Cancer Incidence in Illinois by County, 1985–87,* Supplemental Report (Springfield, Ill.: IDPH, 1990, pp. 7–8).

32. 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 Cong., 1st sess., June 4, 1962.

33. Carson, *Silent Spring* (Boston, Mass.: Houghton Mifflin, 1962, pp. 277–78).

34. R. Perera, "Uncovering New Clues to Cancer Risk," *Scientific American* (May 1996): 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), *Environmental Health Perspectives* **103** (1995):10.

35. 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* (September 1996):80–87.

36. Proctor, *Cancer Wars.*

37. 10,940 is 2 percent of 547,000, the projected figure for total cancer deaths in 1995. See American Cancer Society, *Cancer Facts and Figures—1995,* rev. (Atlanta, Ga.: ACS, 1995).

38. 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, May 21, 1994).

39. M. Eubanks, "Biomarkers: The Clues to Genetic Susceptibility," *Environmental Health Perspectives* **102** (1994):50–56.

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

41. 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* (August/September 1991):65–72.] See also T. O'Riordan and J. Cameron (eds.), *Interpreting the Precautionary Principle* (London: Earthscan, 1994).

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

43. 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, pp. 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).

44. 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: A Journal of Environmental and Health Policy* **3** (Winter 1993): 39–42; and K. Geiser, "Protecting Reproductive Health and the Environment: Toxics Use Reduction," *Environmental Health Perspectives* **101** (Suppl. 2) (1993):221–25.

3

Deconstructing Standards, Reconstructing Worker Health

Charles Levenstein and John Wooding

An 18-year-old Vietnamese émigré to the United States, working for a cleaning and maintenance company, is crushed in a printing plant when a press that he has been cleaning malfunctions and he goes unwittingly to his death. A 35-year-old secretary—a white woman—is forced to undergo operations on both of her wrists because of repetitive strain injuries she has incurred at her computer workstation. A 60-year-old African American, a "retired" miner, sits in a rocker on his front porch carefully gasping for breath, hoping his heart holds out under the strain of oxygen deprivation.

Worker disease and injury flow directly out of the choices of technology made by employers. The use of labor; the intensity of the work; the machines, workstations, and chemicals that endanger worker health—the "working conditions"—are determined by managers, company planners, engineers, and sometimes corporate lawyers whose minds focus on one thing only—making profit.

Occupational disease and injury are "unintended consequences" of technological choices driven by financial imperatives: That is what we mean when we say that the social relations of production determine the health and well-being of workers. Identifying the problem, however, is easier than finding a solution to it. If workers had strong unions and exerted substantial political power in the country, they might be able to play a serious role in the evaluation of the technologies of production, and even prevent the use of dangerous processes and substances.

What, however, would they do? Demand better safety standards? Limit uncontrolled exposures to hazardous chemicals? Legislate reasonable hours of work? Perhaps worker organizations would assert control over