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Environmental Causation of Breast Cancer and Challenges to the Dominant Epidemiological Paradigm

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There are challenges to the dominant research paradigm in breast cancer science. In the United States, science and social activism create paradigmatic shifts. Using interviews, ethnographic observations, and an extensive review of the literature, we create a three-dimensional model to situate changes in scientific controversy concerning environmental causes of breast cancer. We

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identify three paradigm challenges posed by activists and some scientists: (1) to move debates about causation upstream to address causes; (2) to shift emphasis from individual to modifiable societal-level factors beyond an individual's control; and (3) to allow direct lay involvement in research, which may raise new questions and change how questions are approached, the methods used, and the standards of proof. We use our model to examine controversies about doing scientific research, interpreting scientific results, and acting on science. Ultimately, we aim to understand what impedes construction of new methodologies and knowledge about environmental factors in human disease.

Keywords: *breast cancer; environmental health; environmental causation; public participation; activism*

In 1962, Rachel Carson published her groundbreaking book *Silent Spring*, which drew attention to the potential detrimental effects of pesticides on wildlife and human health. Politicians, academics, and lay activists alike credit the book with ushering in the modern environmental movement, and with it, a series of policies that affect the lives of every American. *Silent Spring* helped catalyze a shift in social and scientific policy that would address the consequences of industrial activities on the environment and human health (Steingraber 1997). Carson's eloquent connection between chemicals in the environment and cancer, or what she dubbed the human price of industrialization, ended an era of public ignorance and created collective motivation for advancing environmental health research.[1] This type of scientific inquiry particularly emphasizes the association between environmental toxins and breast cancer, the disease from which Carson died.

This type of inquiry also highlights the structure and normative underpinnings of current scientific research—what we term the dominant epidemiological paradigm (DEP; Brown et al. 2001). Each disease has its own DEP, an embedded set of institutional structures, beliefs, and actions of academia, government, industry, nonprofit organizations, health voluntaries (e.g., American Cancer Society), and the media. Through the DEP, these social actors identify and define disease as well as determine their etiology, proper treatment, and acceptable health outcomes. The DEP for breast cancer is characterized by an outlook on disease that emphasizes individual behavioral factors rather than environmental and social factors in disease causation and health promotion. Such an individualistic focus is common since it seems more straightforward to change individual behaviors than to reorganize social

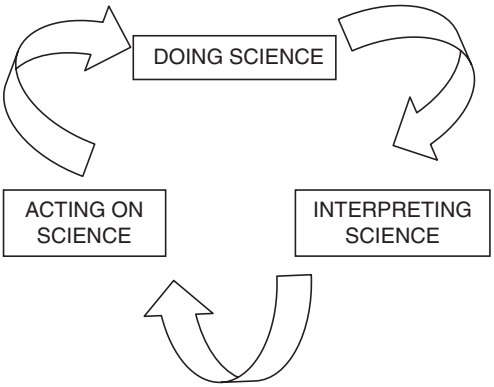
institutions and/or promote fundamental changes in industrial production and government regulation. Such individualist approaches also carry a moralistic undercurrent that holds individuals responsible for their health status despite population data that demonstrate the importance of social structural factors in determining health and disease in populations. The approaches frequently are termed *lifestyle approaches* since they deal with factors that are apparent choices, such as smoking, diet, alcohol use, and late first parity. But a lifestyle approach fails to see that personal behaviors are shaped largely by social structures.

We propose that controversies about scientific research in breast cancer challenge the DEP through three arenas that constitute the processes of fact making and knowledge production: doing scientific research, interpreting science, and acting on science, as shown in Figure 1. This knowledge-production structure is not linear but rather cyclical, in which the process of acting on science leads to further doing of science. Through working in these arenas, researchers pursuing environmental causation hypotheses pose a three-dimensional challenge to the larger scientific community. These three processes—doing, interpreting, and acting on science—set the stage for understanding how struggles to shape breast cancer science play out. In breast cancer research, we observe a persistent and widespread struggle against the dominant paradigm of breast cancer etiology. This is a three-dimensional struggle and may signify the beginning of a Kuhnian-type revolution in the field (Kuhn 1962). This paradigm struggle in breast cancer epidemiology plays out along three axes: (1) upstream versus downstream approaches to prevention (upstream approaches try to prevent the occurrence of the disease by preventing exposure), (2) individual versus environmental risk factors in disease causation, and (3) the degree of community involvement in research and data collection. These three interconnected paradigm struggles involve scientists, environmental-breast-cancer activists, the media, and government representatives, all of whom play shifting and fluid roles in these conflicts. We view these actors as the fundamental players in what we conceptualize as the DEP that shapes the direction of breast cancer research.

The Dominant Epidemiological Paradigm (DEP)

The DEP models how the three primary sectors—government, civil society, and science—contribute to public understanding, scientific knowledge, and social policy. In delineating the DEP in terms of breast cancer and

Figure 1
The DIA Model: Doing-Interpreting-Acting



the environment, we observe that the National Institutes of Health (NIH), Centers for Disease Control and Prevention (CDC), Environmental Protection Agency (EPA), Congress, the Department of Defense (DOD), and state departments of public health are major players in the governmental sector. When we examine civil society, we see that health voluntaries and media sources shape the dialogue pertaining to cancer causation, corporations push for leniency in regulation of chemicals and produce massive amounts of research to support that action, and social-movement groups attempt to stimulate public awareness about environmental causes. Environmental causation is contested and debated in scientific circles through journals, professional organizations, educational institutions, and internal scientific norms. The challenges to the DEP of which we speak are found primarily in the United States because of the strength of the breast cancer movement and especially its environmental wing (McCormick, Brown, and Zavestoski 2003). Hence, our analysis reflects a uniquely United States phenomenon.

Public challenges, coupled with internal dissent within scientific and governmental institutions, can create opportunities for major shifts in how public-health research is conducted. These challenges are made by activists through popular epidemiology, an approach that uses lay observations as a central component of examining environmentally induced diseases (Brown 1992). Challenges also are made by professionals through what we term

critical epidemiology, an approach that emphasizes population-level disease focus rather than individual-level; analyzes the importance of race, class, and gender stratification; and examines conflicts of interest that especially include the role and power of pharmaceutical firms. These challenges to the DEP are a fundamental issue in modern science, particularly the way that activists and affected people work to shape research methods and influence the direction of scientific inquiry.

The debates and controversies that characterize breast cancer epidemiology are a backdrop to changes in broader research traditions. In many illnesses, large numbers of research dollars have been devoted to analyzing individual and behavioral risk factors, but this line of inquiry has yielded equivocal answers (Brody and Rudel 2003). Therefore, researchers increasingly are exploring whether and how environmental factors play a role. These paradigm struggles often are precipitated by external political movements that can be either incremental developments, major milestone events, or the converging of political movements in new ways, such as between the environmental and women's health movements. These struggles also are precipitated by dissent within the scientific community, led by a critical mass of researchers who have become dissatisfied with the status quo, who feel that the dominant line of inquiry is not explaining disease etiology effectively, and who want to open new lines of scientific inquiry.

Many scientists puzzle about what causes breast cancer and how to prevent and treat it. Epidemiologists, toxicologists, geneticists, immunologists, and molecular biologists contribute to the large body of literature and a generally accepted approach, that is, the DEP. We consider the totality of these scientific fields to constitute a well-recognized area of breast cancer science; our concept of the DEP incorporates all of this.

These scientists also include some who pose alternatives to the mainstream models. To examine such disputes, we make several assumptions. We conceptualize as traditional those scientific approaches that focus on individual-level characteristics and factors, such as a woman's age at first birth and her diet. While it appears that individuals have control over such factors, those factors in fact are structurally shaped. Traditional approaches to science (and to consequent policy) are characterized further by a focus on individual versus population-level factors (a population-level approach asks why entire populations have differing morbidity and mortality, such as with the increase in breast cancer incidence in Japanese women who migrate to the United States) and by research on individual chemicals rather than synergistic effects of multiple chemicals.

Data and Methods

Our research is based on (1) a review of scientific literature on breast cancer and the environment; (2) thirty-seven interviews with scientists and activists involved in environmental-breast-cancer activism and research in the Boston and Cape Cod areas of Massachusetts, on Long Island in New York, and in the San Francisco Bay Area; (3) extensive ethnographic observation of Silent Spring Institute (SSI), the country's only research institute dedicated to examining environmental factors in breast cancer; and (4) ethnographic observation of public breast cancer activist events.

Our analysis of the scientific literature is not intended to be exhaustive. We focus on those studies that our literature reviews and respondents indicated were the most significant. Thorough scientific literature reviews that cover hundreds of studies can be found elsewhere (Snedeker 2001; Evans 2002; Brody and Rudel 2003), and these reviews tap the same literature that we have determined to be significant, providing us with validation for our choice.

Our respondents include prominent epidemiologists and activists in the field who are concerned with the state of the evidence about breast cancer etiology. We identified activists and researchers who collaborated on environmental-breast-cancer research as well as some scientists who had done major studies without activist involvement. Semistructured interviews were tape-recorded and transcribed; there were separate questionnaires for scientists and activists, though with some overlap of items. People were asked about their assessment of the current knowledge base for environmental causation of breast cancer, the most significant studies, the impact of key studies with negative findings regarding environmental links, obstacles to greater belief in environmental causation, the significance of the environmental endocrine hypothesis, and the role of government agencies, foundations, private organizations, and breast cancer activists in examining environmental causation. Questions also explored lay involvement in research.

We also conducted extensive ethnographic observations of SSI, including its daily work routines, meetings between activists and researchers, presentations by SSI staff, public meetings in which the researchers presented their work, scientific review panel meetings, and science-activist conferences. Additional ethnographic observations were made of other breast-cancer-activist events. Printed materials from organizations were collected to better understand the organizations' scientific approach, political stance, and public activities.

The Arenas of Knowledge Production: Doing, Interpreting, and Acting on Science (DIA)

As noted above, we identify three arenas of scientific knowledge production, which are interrelated and occur simultaneously: doing scientific research, interpreting science, and acting on science (i.e., policy recommendations). Paradigm struggles take place in all three. It is useful to break down the process of scientific research into these three arenas (portrayed in Figure 1) to examine paradigm struggles about dominant theoretical and methodological approaches to studying environmental links to breast cancer. These realms of scientific knowledge production set the stage for understanding movement along the axes that are discussed in this article.

Doing Scientific Research

Doing scientific research involves how scientists choose particular topics and questions, how they proceed with their investigations, and how they interact with funding, research, and support organizations. Doing research also includes how organizations shape research protocols and allocate funding.

Scientific research is circumscribed by trends in theoretical approaches, in which disciplines historically are limited by strict disciplinary boundaries and "scientific bandwagons" (Fujimura 1995). In breast cancer research, genetics and lifestyle bandwagons predominantly shape the research agenda (Davis 2002). This focus can be explained in part by an interest-groups–conflict-analysis perspective: scientists who follow the dominant approach have a greater ability to obtain research funding, scientific prestige, and career advancement. On their own, scientists might not be able to adopt alternative scientific approaches, but activist pressure on funding and research institutions enables them to do so. For example, the breast cancer movement pressured the DOD to include activists on review panels, an idea that was very novel at the time but is now more common. Breast cancer organizations participated in workshops at National Institute of Environmental Health Sciences (NIEHS) in 2002 that resulted in a program to fund research centers on breast cancer and the environment. These centers, first established in 2003, will use innovative methods (e.g., use of geographic information systems [GIS], examination of historical land-use patterns, and inclusion of a larger range of potential environmental carcinogens), provide community outreach, and have a translational core for making scientific information into usable public knowledge.

The DEP validates science that is constructed by experts. Mainstream research does not include affected persons in the scientific process, and therefore, misses an embodied account, or what Haraway (1988) calls "situated knowledges." This idea resonates with Hartsock's (1998) "standpoint" theory, which states that accurate knowledge must derive from affected communities. For Hartsock, knowledge from affected groups is not merely another voice in a relativistic world. Instead, knowledge is a counterhegemonic force introduced by those usually excluded from science. According to Hubbard (1990), science is not routinely subject to public accountability. Consequently, the pretense of objectivity masks how science structures social relations and distributes power (Hubbard 1990). Such an approach to science implies different methodologies, such as lay involvement in research, that many breast cancer activists advocate (Dickersin and Schnaper 1996). Through what we term *citizen science alliances*, environmental-breast-cancer activists have recruited and/or collaborated with scientists willing to investigate environmental factors and where lay perspectives can be legitimized in research. Activists also have made explicit the role of politics in research. This form of activism is facilitated by the tradition of public participation in science as well as the strength of the breast cancer movement (McCormick, Brown, and Zavestoski 2003).

Breast cancer research tends to focus on lifestyle (diet, alcohol use, age of first parity) and genetics (mutations on key genes). Few federal research dollars go to research on environmental factors. The NIEHS has supported research on environmental factors strongly, but it is one of the smallest and least influential institutes at the NIH. Conversely, the well-endowed and most powerful institute, the National Cancer Institute (NCI), invests relatively little on research that examines environmental links to cancer. As evidenced in its documents, NIH leaders often consider lifestyle factors to be environmental, and hence they claim to be conducting more environmental research than they actually are; activists are especially critical of this.

In the absence of significant funding for research on environmental factors, the breast cancer movement has had to press state and federal legislatures for special bills (McCormick, Brown, and Zavestoski 2003). The Long Island Breast Cancer Study Project was funded by an earmarked federal bill, and the SSI's Cape Cod Study was supported by a state bill pushed by the Massachusetts Breast Cancer Coalition and other breast cancer activists. Activists also were able to work with federal legislators to put considerable breast cancer funding in the Congressionally Directed Medical Research Program, which is funded by the DOD.

Interpreting Science

Interpreting science involves how scientists make sense of data. There are two normative standards that are critical to the interpretation of science: standards of proof and weight of evidence. Standards of proof are a major element of interpretive flexibility because they are the norms with which research results are interpreted. Collins' (1983) notion of "interpretive flexibility" informs us that different conclusions can be drawn from the same data. Multiple scientific truths can coexist or lead to disputes over what constitutes sound methodology and proof of causation. There are also disputes about what studies are included in deliberations about the weight of evidence regarding the carcinogenicity of certain exposures and risk factors. Results can be interpreted in isolation or in combination with experimental evidence from other fields such as toxicology. In terms of breast cancer, a discipline-specific body of evidence might suggest that certain environmental exposures contribute to increased rates, and novel hypotheses might require the consideration of multiple types of evidence. For example, Colborn, Dumanoski, and Myers (1997) synthesized research findings from human, environmental, in vivo, and in vitro toxicology studies to argue that endocrine-mimicking chemicals have effects on multiple species in multiple contexts. Additionally, occupational studies more frequently support a correlation between breast cancer risk and chemical exposure but often are not considered in tandem with general population studies.

Standards of proof are representative of the dominant conceptions of knowledge that claim science is neutral and value free because of accepted methodology, is a universal reflection of reality, and is controlled by a scientific community that can delineate its own work from personal interests (Harding 1998). These standards of proof include strength of association, significance level, study design (prospective vs. case control), temporality, biological plausibility, and timing of exposure in biological developmental cycles. These standards of proof have important implications. Mainstream science has a tendency to err on the side of uncertainty by not drawing conclusions until there is almost absolute certainty. Mainstream scientists worry that relaxing any component of scientific rigor will reduce the credibility of all science, and therefore, they believe that scientific standards should remain strict, regardless of circumstances. Reliance on these standards of proof affects whether peer review allows researchers to get government and/or private funding for further scientific study and for publication of results. Reliance on these standards of proof also affects whether government takes action in creating new regulations or strengthening existing ones.

Controversies about standards of proof often forestall scientific closure, despite overwhelming evidence (Adams and Brock 1999). In breast cancer research, the dominant interpretation of existing data is that there is not sufficient evidence to confirm the role of environmental factors. Data can lead scientists to different conclusions since debates about the meaning and implications of scientific data are not absolute. Disputes about the weight of evidence involve determining how much evidence is enough to warrant conclusions and whether the body of research literature shows reliable patterns across many studies. Deliberations on the weight of evidence also involve questions about whether the research findings come from appropriate channels—typically, peer-reviewed articles in major journals. Interpretive flexibility also comes up in discussions on weight of evidence, especially in the debate about whether unrefereed "gray literature" should be considered valid research. For example, as a National Academy of Science conference pointed out, rarely is research conducted by public health departments published, because of government reticence, outright censorship, or lack of interest and time to publish in academic forums (National Research Council 1991). The National Research Council (1997) followed that up with another volume that detailed methods of using the gray literature, including unpublished reports from the EPA, National Toxicology Program, and manufacturers' test data. For many chemicals, these reports are the only scientific studies on the chemical and its cancer risk. This source is important given the absence of exposure data on many chemicals that has been emphasized by environmental-breast-cancer researchers. Much of the gray literature published by government agencies is peer-reviewed, and hence, conforms to scientific canons of valid knowledge, even if it never gets published in journals. Indeed, the United States Department of Energy (DOE; 2003) organized a 2000 conference of government agencies to set up an Internet-based gray literature network, "GrayLIT Network," to facilitate use of such government reports from DOE, DOD, EPA, and NASA.

Acting on Science

Once research is conducted and interpreted, it must be acted on in political regulations. This involves scientists' calculating the weight of evidence, deciding how to approach scientific uncertainty about the subject at hand, and then creating policy that takes these factors into account. Latour's (1987) actor-network theory helps explain how scientists and advocates join together

to spread their hypotheses, approaches, and findings and to push action in policy directives. Actor-network theory holds that the acceptance of scientific discovery is not a linear progression of pure scientific deliberation. Rather, it is accomplished largely through scientists' recruitment of support from other scientists, media, government officials, and the general public. In this path, advocacy scientists (Krimsky 2000) or dissident scientists (Gaventa 2002) play a key role in mobilizing support by going outside traditional science into the realm of public advocacy. In addition, social-movement activists press for greater research on environmental factors by recruiting researchers sympathetic to their research agenda, as we show later.

A crucial part of acting on science is for scientists and regulators to recognize the extent of our knowledge gaps. Research on breast cancer and the environment is impeded by what activists call toxic ignorance (Roe et al. 1997). More than eighty-five thousand chemicals are registered for commercial use in the United States, but only a small portion of them, fewer than one thousand, have been tested for carcinogenicity, and even fewer have been fully and comprehensively tested for noncancer outcomes. Many toxins, such as dioxin, are formed as accidental by-products of production or incineration, and in many cases, scientists and regulators have not even identified them (Thorton, McCally, and Houlihan, 2002). Activists and scientists have called for increased human and environmental monitoring to generate crucial information about the origins and potential long-term effects of chemicals. Indeed, Scandinavian countries' ability to implement the precautionary principle, a perspective that holds that "When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically. In this context the proponent of an activity, rather than the public, should bear the burden of proof" (Raffensperger and Tickner 1999, p.8) on many policy decisions, is largely because they have high-quality monitoring databases with information on problems such as breast-milk contaminants. Activists and scientists also have argued for the adoption of pollution-prevention strategies that would decrease environmental emissions and population exposures.

Acting on science involves both whether to act and how to act. These decisions are made in large part by state and federal legislatures and agencies. But scientists also make these decisions, such as when they consider whether to press for regulatory policy. Decisions on whether to act are not based primarily on the weight of the evidence but involve political and economic

considerations; for example, they might consider the economic cost of substituting one chemical for another. Even when a decision to act is made, there is leeway in how to intervene. In terms of breast cancer, this involves decisions about whether to pursue an individual-responsibility approach (lifestyle and childbearing choices) or a social-responsibility approach (restricting or banning chemicals).

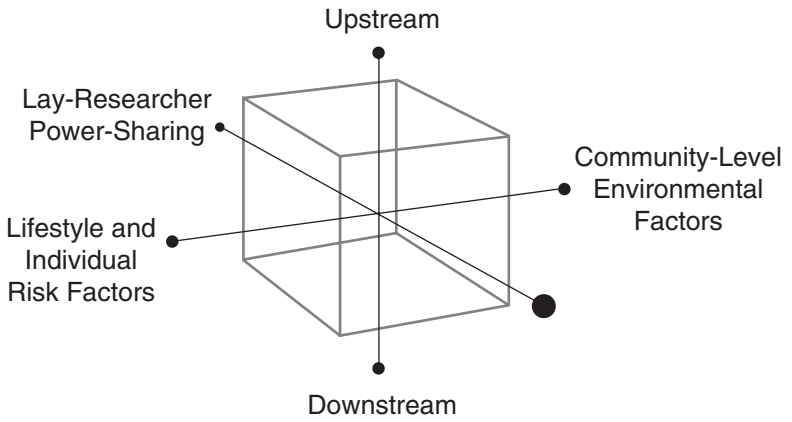
Some scientists have a growing dissatisfaction with the ability of mainstream science to answer difficult questions and with the resulting lack of action. They pose questions about scientists' social and moral responsibilities to affected people, communities, and the larger society. They also address the effect of funding sources on research, especially corporate funding. They question the veil of objectivity that separates science from social policy rather than recognizing the societal pressures that shape science. Some scientists raise questions about whether it is their responsibility to point out elevated disease rates and whether prepublication release of research is acceptable if it benefits public health. These challenges increasingly emphasize the precautionary principle as a guide to such approaches.

Three Axes of Challenges to Breast Cancer Research

Revolutions in science occur when a critical mass of researchers begins to question the validity of a dominant paradigm that no longer adequately explains empirical observations. A Kuhnian paradigm refers to an established worldview that shapes what problems scientists are encouraged to study and how to study them, while excluding those theories, hypotheses, and observations that do not match this worldview. We extend the Kuhnian notion of paradigm shifts, once limited to the internal functioning of the scientific world, to include external debates about environmental factors in disease. These debates take place within social movements, media, and congressional hearings, and they overflow from external debates into internal scientific circles.

We develop a three-dimensional model to situate and apply our findings from interviews, fieldwork, and analysis of the literature. This model offers three axes in which struggles for paradigm shifts occur. These axes (shown in Figure 2) represent continua involving various combinations of research, prevention, policy, and activism. Axis 1 (upstream-downstream) reflects the level of prevention; this is based on the familiar notion of downstream, or tertiary, approaches' being after the fact and upstream, or primary, approaches'

Figure 2
Dimensional Model of Disputes about Breast Cancer Research



being the most preventive (e.g., research on treatment efficacy vs. the relationship between dioxin exposure and breast cancer). Axis 2 represents a continuum of research foci from individual risk factors to community-level hazards. Research informed by the DEP emphasizes individual risk factors, such as age at first birth, though many of these factors are partially or even largely out of people's control. In contrast, environmental-breast-cancer and other activists pursue community-level environmental factors (such as proximity to pesticide spraying). Finally, Axis 3 reflects the degree of public participation in research, ranging from no lay involvement to research efforts that are mostly controlled by lay groups.

Axis 1: Upstream-Downstream

Axis 1 addresses whether the research focuses on treatment, intervention, or prevention. These are referred to metaphorically as upstream or downstream approaches. Sandra Steingraber's book *Living Downstream* (1997) recounts the familiar tale of villagers who notice helpless people floating downstream. The villagers develop increasingly sophisticated ways to rescue these people, yet few venture upstream to find out why people are

falling into the river in the first place. This upstream-downstream analogy describes the continuum of research and intervention strategies aimed at prevention of public health problems.

Primary prevention (upstream) emphasizes disease prevention in populations. In an environmental health context, this includes strategies aimed at preventing human exposure to toxics through pollution prevention and toxics-use reduction. Secondary prevention aims to provide screening, early detection of disease, and prompt intervention for people at risk for disease. Tertiary prevention (downstream) minimizes the effect of disease in people who are already quite sick. For breast cancer, a walk upstream implies a radical shift in research and intervention away from tertiary approaches such as treatment efficacy (surgery, radiation, and pharmacological treatments) and secondary prevention such as screening and early detection (through mammography,[2] breast self-exam, and biopsy) and toward minimizing exposures to risk factors and toxic substances that may be linked to disease. When intervention and research strategies emphasize community and population exposures to toxics, they raise formidable methodological challenges inherent to environmental epidemiology. Specific methodological barriers to studying links between chemical exposures and breast cancer include adequately measuring chronic low-level chemical exposures, long latency periods for diseases such as cancer, and controlling for other covariates such as diet.

These scientific challenges are coupled with the political and economic controversies associated with reorienting research and intervention toward a focus on the effect of chemical exposures on human health. The 2001 Bill Moyer PBS special "Trade Secrets" exposed how chemical manufacturers concealed employees' polyvinyl chloride contamination and demonstrated how chemicals have far more rights than people because of the economic and political clout of industry and its overwhelming influence on the regulatory process (Public Broadcasting System 2001). Similarly, describing the history of childhood lead poisoning, Rosner and Markowitz (2000) argue that health and safety standards sometimes affect industrial policy, but only as a response to public outcry and national concern. Indeed, government reluctance to regulate the industry's production decisions proactively, combined with the skepticism of many research scientists regarding environmental links to cancer, make agencies such as the NCI reluctant to fund environmental causation research. In the absence of solid funding, professional support, and other resources, environmental-breast-cancer researchers see themselves as breaking traditional scientific norms in their pursuit of alternative methods and advancing new theories of disease

causation. As we will discuss in the section on Axis 2 (individual lifestyle and environmental factors), environmental-breast-cancer researchers are challenging the DEP held by many parties in science, government, and the public—a paradigm that views diet, late childbearing, low parity, and genetics as the primary risk factors for breast cancer.

The Endocrine-Disrupter Hypothesis

The endocrine-disrupter hypothesis has played perhaps the most significant role in pushing research interests upstream. The hypothesis "asserts that a diverse group of industrial and agricultural chemicals in contact with humans and wildlife have the capacity to mimic or obstruct hormone function—not simply disrupting the endocrine system like foreign matter in a watchworks, but fooling it into accepting new instructions that distort the normal development of the organism" (Krimsky 2000, p. 2). Although the link between endocrine disrupters and breast cancer remains tentative, many researchers and activists looking at environmental causes of breast cancer feel that the hypothesis supports the direction of their own work. Based on this increasing knowledge about the health effects of many endocrine-mimicking chemicals, a growing number of scientists has moved to an upstream approach. As one scientist stated:

> We need to understand exposure to chemicals that are of interest for research on breast cancer and endocrine-related health outcomes. So this was the first step in learning more about exposure and that these chemicals are chemicals that need to be studied further and considered for regulatory [policy].

One scientist who studies environmental factors in breast cancer began to develop new scientific methods to capture the effects of endocrine disruptors:

> There are some significant methods developed in the first phase of the research that I consider to be central to our scientific contribution. One is the creation of . . . environmental sampling tools. We tested wastewater, groundwater, and drinking water for endocrine disrupters. There weren't any chemical methods just on the shelf that you could use, so we had to adapt them. We also used E-screen, which was developed by Tufts medical school before the study started, but the extraction method that allows for the bioassay to be used for environmental samples was developed in the Cape study . . . . So we became the first team to identify endocrine disrupters in ground water; they had been previously found in surface water.

These new methods are based on the idea that despite the lack of clear evidence that endocrine disrupters cause breast cancer, there is a widespread consensus that the endocrine-disrupter hypothesis warrants more investigation. Endocrine disrupters are working their way into people's bodies, though it is often unclear how. There is a high body burden, which is increasingly the subject of study, and there is evidence of hormonally related disease. Research and regulatory policy have flowed from this awareness, including the EPA's formation of the Endocrine Disruptor Screening and Testing Advisory Committee in 1996 (EPA 1998, 2001), the European Union's policy to require toxicity information on all major chemicals in commerce and industry (Tickner 2000), and a growing number of specialized research conferences by NIEHS and universities, as well as by increasing numbers of publications in scientific journals.

Even if the endocrine-disrupter hypothesis is not confirmed with regard to breast cancer, it has led to widely accepted knowledge on developmental and reproductive disorders (Colborn, Dumanoski, and Myers 1997). The endocrine-disrupter debate has made a huge impact on toxicology and epidemiology and has cast its stamp on public policy making (Krimsky 2000). We view this as a critical contribution of such intellectual debates—they are mechanisms for advancing science in general, even if they do not confirm all or most assumptions that presently are being studied. Indeed, the tradition of scientific curiosity shows countless such examples in which novel ideas catalyze new approaches.

Issues of Methods and Measurement

Scientists' approaches are affected by methodological challenges. As previously mentioned, environmental research faces the same obstacles as traditional epidemiology: difficulty with measurement, limited information about exposure, the multiplicity of variables that must be considered, and the newly emerging set of methods that must be adjusted to account for the causes being analyzed (Rudel et al. 2001). While these factors may discourage researchers from focusing on environmental causation, they also provide an opportunity to reshape scientific norms and practices to fit emerging knowledge about environmental factors.

Past studies of environmental causes of breast cancer have focused only on certain chemicals: DDT, DDE, PCBs, and other organochlorines. There is little reason for this focus other than that these chemicals are easily detectable and found in blood or tissue of women (Snedeker 2001). Although the prevalence of these chemicals offers a large enough study population to create

statistically significant findings, some researchers explain that this supposed benefit of studying organochlorines is actually a drawback:

> We are dealing with compounds where exposure is ubiquitous, some of them cannot be measured ten, twenty, thirty years later, and our abilities to find differences between populations is really marred by that.

Since there is such omnipresent exposure in women, specifically in their breast milk, it is impossible to find a control group against which a population can be studied (Snedeker 2001). Additionally, little is known about which specific organochlorines may be related to incidence. Therefore, there is complexity within this one group of chemicals that is unaccounted for across the scientific literature. The source from which the chemicals are collected (i.e., blood serum or tissue) also may be a factor that influences findings; some chemicals may produce carcinogenic mutations but disappear from the assayed tissues. However, the emergence of these studies provides the scientific language necessary for further study.

Studies of DDT, DDE, PCBs, and other organochlorines actually represent a small proportion of research on breast cancer. The vast majority of research emphasizes tertiary prevention. Breast cancer research has been a prominent part of NIH spending. While breast cancer activists applaud the importance of this research, they also question whether the money is being invested wisely. As one scientist remarked:

> Almost from the beginning when epidemiologists in an organized and systematic way started looking at the epidemiology of breast cancer, they focused on a very narrow range of factors. Almost all of them were related to women's reproductive systems—age at menarche, menopause, parity, lactation—then it branched out into body mass, host factors, family history, and genetic factors. Breast cancer and lung cancer, between the two of them, are probably the most widely studied, and I would say that maybe of the thousands of studies that have been done up to the early 1990s, almost none of them looked at environmental factors.

The National Breast Cancer Coalition has urged Congress and the administration to consider changes in the grant mechanisms and the research structure at the NCI. Activists are calling for external monitoring of the NIH and believe the public should design and participate in an oversight process that will track how the money is being spent and whether it is being allocated wisely (National Breast Cancer Coalition 2001).

In summary, current and traditional approaches to prevention tend to fall along the downstream end of Axis 1. The endocrine-disrupter hypothesis challenges the DEP and pushes the research focus upstream, though findings based on the endocrine-disrupter hypothesis are often inconclusive and heavily criticized (more will be said on this in the next section). Support for the endocrine-disrupter hypothesis has led to significant amounts of primary prevention of toxic exposures in Europe, for example, by banning phthalates from children's toys and pacifiers. In the United States, however, various methodological challenges still make the upstream approach much more difficult to pursue because of problems with measurement, exposure timing and recording, and lack of a control group. Partially because of these difficulties, there is far less funding for research on environmental factors than on lifestyle and genetics. Despite these problems, we observe an increase in research based on the endocrine-disrupter hypothesis, one of the main scientific hypotheses that are moving scientific conduct upstream. Struggles about interpreting science are very intense, with a general reluctance among scientists to accept the evidence from animal studies, a disregard for gray literature and human reproductive health effects as applicable to human carcinogenesis, and a belief that the weight of evidence favors a lack of connection to environmental causation. Possibly the greatest obstacle to moving science more upstream is that if science finds that chemicals need to be better regulated, industry may face major financial difficulties. Therefore, it is in the best interest of industry to take a more downstream approach that focuses on curing diseases rather than preventing them. Acting on science involves the choice of focusing upstream on prevention or downstream on curative approaches. Acting is only rudimentary for this area of breast cancer and the environment; yet, there has been some upstream regulatory action, largely in Europe.

Axis 2: Individual Risk Factor/Community-Level Environmental Hazards

While Axis 1 has implications for intervention, Axis 2 examines the focus of research—individual level (i.e., characteristics of individuals) or community level (i.e., aggregate characteristics of populations or geographical areas). In Axis 1, we discussed the emphasis on endocrine disrupters as part of the upstream approach, since the identification of chemical causes leads to a primary preventive approach of reducing or eliminating exposure. Here, in Axis 2, we revisit chemical factors, this time looking at individual versus collective units of analysis. Additionally, while Axis 1

represents the general trends of epidemiological research, Axis 2 relates these general struggles to the specific case of breast cancer research. Axis 1 is concerned with how a number of social sectors (e.g. industry, government regulators) can prevent exposure in the first place, hence reducing disease. Axis 2 and Axis 3 are concerned with the internal methods and data collection of clinical research, including the degree to which lay involvement plays a role.

The traditional and dominant approach to disease research focuses on individual risk factors, while environmental-breast-cancer researchers and activists pursue population- or community-level factors. Underlying science and policy are what Sylvia Nobel Tesh (1988) calls hidden arguments, political ideology about legitimate sources of knowledge and moral arguments about human nature and what constitutes a good society. These hidden arguments, or conceptual frameworks, inform which questions get asked, which do not, and how researchers go about investigating them. Furthermore, these frameworks theorize about the causes of disease (Tesh 1988), and therefore, they influence how researchers conceptualize and operationalize the determinants of health (Zierler and Krieger 1997). Finally, theoretical frameworks also inform how researchers prioritize among possible models for disease prevention.

The biomedical model is the dominant framework that informs how most health researchers are trained to conduct research in the health sciences, although as Krieger and Zierler (1995) argue, the tenets of the biomedical model rarely are specified. The central question of research informed by the biomedical model is How do humans become ill? Disease, according to this conceptual framework, is purely a biologic phenomenon that can be understood through positivist, value-free research. Further, this model assumes that diseases are addressed best through treatment and by mitigating against individual-level risk factors for disease. However, the other defining assumption of the biomedical model is that it does not address societal or structural determinants of health, and therefore, cannot explain why the distributions of health and illness tend to correspond to society's economic and social structure (Krieger and Zierler 1995). Biomedical models emphasize biological and physiological phenomena, while lifestyle models emphasize people's behaviors. These two approaches show up together in research studies because they both focus on the characteristics of individuals. The primary distinction is that prevention or intervention is achieved through individual behavior change rather than administrated by or dictated from a medical professional.

In contrast to frameworks that conceptualize disease causality and prevention at the individual level, political economy of health or social production of

disease models ask how economic and political relations affect health. This includes an emphasis on the role of environmental factors in disease. Under the assumptions of this framework, disease prevention is achieved not through medical treatment or through individual behavior change but through changes in industrial production practices. Political economy or social production of disease theories focus on the dynamic relationship between macrolevel structures and individual bodies: the individual always is seen in relationship with the political economy and social world (Krieger and Zierler 1995). According to Geoffrey Rose's (1985) distinction between studying sick individuals versus sick populations, an individual risk-factor approach seeks to answer the question, Why do some individuals have breast cancer? Conversely, a population-based line of inquiry asks, Why do some groups of women have breast cancer, while in other populations, it is rare? While the former approach may help us understand why there is variation in disease among individuals, it misses the fundamental public health and epidemiological challenge of elucidating those determinants that explain population differences in disease incidence. In terms of intervention, the population-based approach is more radical because it implies the need for mass environmental control methods or the alteration of socioeconomic norms that give rise to widespread hazardous exposures and collective behaviors that enhance the vulnerability of certain communities to disease (Rose 1985).

Traditional Approach to Individual Risk Factors

The dominant paradigm used in studying breast cancer has been circumscribed by the biomedical model. In a period of growing genetic determinism, genetic makeup has been a major research focus, and knowledge claims based on genetic explanations of disease are considered especially credible (Conrad 1999). The discovery of the BRCA-1 and BRCA-2 mutations led to much attention to genetic causes, even though it since has been recognized that genetic causes account only for some 5 to 10 percent of all cases (Davis and Bradlow 1995). A recent study of forty thousand sets of twins found that genetics play only a minor role in causing breast cancer, while environmental causes play the primary role (Lichtenstein et al. 2000).

Activists and some scientists have criticized the way that genetic approaches focus only on individual risk factors. While genetics may account for a small amount of individual susceptibility, it cannot explain why breast cancer rates have increased in a period that is too short for genetic changes in the population (Davis and Webster 2002). If genetics

research focuses on individual risk factors, it likely will result in campaigns for people to alter their individual behavior. Such research is not unimportant, but nonmainstream scientists think it should not be the sole focus. For example, one researcher pointed to other areas of study that reveal promising leads:

> We have evidence that synthetic hormones affect breast cancer risk. I also think that the wildlife studies are very compelling as a whole. That the effects of endocrine disrupters on wildlife, I think, are a very important body of support for further investigation of environmental causes of breast cancer. And then there are laboratory studies that have identified mammary carcinogens in animals. Cell studies have shown synthetic chemicals make human breast cancer cells grow in a lab. That's a signal to me that it should be a priority to study them in humans.

The interpretation of genetic factors could affect medical practices. Knowledge of genetic factors could be used to focus on a variety of practices, ranging from therapeutic intervention to prevention. Differing approaches to genetic risks also could lead some scientists to ask why certain populations are more vulnerable than they were previously. The increasing ability to discover more vulnerable populations could be a tool for making the case for tighter regulation, rather than such drastic practices as preventive tamoxifen (a drug used to treat estrogen-positive breast cancers) or prophylactic mastectomy. Alternative approaches could examine the interaction of genetic risk and environmental exposure. Another promising direction is toxicogenetics, in which researchers study biomarkers of toxic exposure and effect.

Other scientific work has focused on the potential increase in risk because of late first parity, alcohol consumption, diet, and exercise, though such factors account for a small proportion of cases (Kant et al. 2000; Thompson 1992). Despite this small proportion, individual risk-factor approaches continue to dominate for several reasons. First, they fit well with traditional biomedical conceptions of disease causation, which emphasize individual-level variables; second, leading cancer researchers, institutes, and agencies have placed most of their efforts on this perspective, largely because research on individual risk factors is easier to design and test than environmental factors, and hence, easier to get funded; third, there is only weak evidence for environmental causation; and fourth, research into environmental causation places responsibility on the business sector and on the government agencies that fail to regulate that sector adequately.

Based on interviews with researchers and our review of the literature, we conceptualize the existence of two main approaches to research on environmental factors. Traditional research focuses mainly on individual risk factors and occasionally adds environmental variables. Innovative approaches put primary emphasis on environmental factors, which otherwise have been largely ignored. Proponents of an innovative approach argue that more environmental research should be conducted because traditional approaches have led to little knowledge of causation. Innovators point to weak findings from genetic studies and strong findings from immigration studies.

Mixed Results and Conflict in the Search for Environmental Factors

Proponents of an innovative approach argue that genetic explanation counts for too little and that it cannot account for the jump in a woman's lifetime risk of developing breast cancer from one in twenty (by age eighty) in 1950 to current estimates of one in seven. Another area of environmental study is immigration-related data that show the breast cancer risk for Asian immigrant women has increased 80 percent in the first generation with the rates for their daughters approaching those of United States–born women (Stellman and Wang 1994).

The body of environmental-breast-cancer research, which has been equivocal, is far smaller than the literature on genetics and individual risk-factor epidemiology. Some studies have shown a correlation between environmental toxins and breast cancer incidence, while others have not supported this conclusion. However, many researchers emphasize that lack of evidence does not disprove an argument for environmental causation. Rather, that direction was slowed by the constant critique of methods from mainstream sources. One scientist remarked:

> A lot of people have a basic distrust of [environmental] epidemiology as a more squishy kind of science that they don't believe works anyway. Then there is the basic concept of trying to look at gene-environment interactions. Well, the genes always win because the science is much more rigorous and it's reproducible. The environmental stuff you can just get tied up in forever, in muddy and disputed methodology. We've also always had this cooptation of environmental factors by more lifestyle factors. It's much more acceptable to talk about people's diet and smoking than to try to pin things on industrial chemicals.

Wolff et al. (1993) was one of the earliest to show a direct correlation between increased risk of breast cancer and DDE, the chemical breakdown product of DDT, a pesticide banned in the United States but still used worldwide. Since then, an increasing number of studies have focused on

this group of organochlorine pesticides used mainly since World War II. Wolff et al.'s (1993) landmark study was the first large-scale study to draw major scientific attention because of its conclusion that there is a relationship between DDE and breast cancer incidence. But later work by Hunter and colleagues (1997), as part of the Nurses' Health Study, cast doubt on Wolff et al.'s original conclusions. Because of the size, quality, and reputation of the Nurses' Health Study, these results posed a major obstacle to proponents of environmental causation.

Other research has focused on chemicals more generally. Some studies are retrospective using health data and an ecological design rather than individual body burden. Examples of such research are Griffith et al.'s (1989) and Najem et al.'s (1985) studies of increased breast cancer rates in the vicinity of hazardous-waste sites. Most of the significant data come from body-burden studies. Dorgan et al. (1999) tested blood-serum levels of PCBs for effects on breast cancer risk but were unable to compare different groups of PCBs, as had been done in previous studies, with positive results. They found no support for organochlorine pesticides' and PCBs' playing a role in breast cancer etiology. Hoyer, Jorgensen, Brock, et al. (2000) also provided new evidence showing the adverse affects of some organochlorines on breast cancer risk. Guttes et al. (1998) found evidence for a correlation between breast cancer incidence and DDT, DDE, HCH (hexachlorocycloethane), and some PCBs. While Hoyer, Jorgensen, Grandjean, et al. (2000) did not find a connection to PCB levels, they supported a connection to the pesticide Dieldrin.

In evaluating research on pesticides and breast cancer risk, Snedeker (2001) notes that while there is a lack of support for a correlation in Western women of European descent, the most common population being studied, further studies of other populations should be conducted. Krieger (1990) found a related trend of differences in morbidity and mortality across racial groups as they age. Millikan et al. (2000) support a similar conclusion in their study of black and white women by concluding that strong support for a correlation between breast cancer risk and exposure to pesticides does not exist but noting correlations among subgroups.

One point of controversy in these studies is whether the recency of exposure affects risk. Studies in developing nations in which DDT still is used are a way to test this question, although here, the evidence is mixed as well. Research in Mexico and Colombia (Olaya-Contreras et al. 1998; Romieu et al. 2000) has shown a strong correlation between DDT serum levels and breast cancer risk, while evidence gathered from women in Vietnam, Brazil, and Mexico has shown negative results (Mendoca et al. 2000; Lopez-Carrillo et al. 1997; Schecter et al. 1997).

The mainstream scientific community has been very skeptical of environmental causation of breast cancer, and proponents have received criticism from their peers and from others outside of the scientific community. One environmental-breast-cancer researcher said:

> It is hard to get people's attention when you are from Silent Spring [Institute] and doing research about environmental causes of breast cancer. It's hard to break through and get people's attention, scientists' attention. You go through a kind of hazing where you have to establish talking about what you're doing, your credibility with each new member. You don't have on your name tag, Harvard University. You really have to go through a process of demonstrating your credibility.

There have been studies contradicting findings that link environmental factors and breast cancer and editorials condemning environmental causation hypotheses (MacMahon 1994; Hunter et al. 1997; Safe 1997). Additionally, there have been junk-science critiques of environmental-breast-cancer research on the part of more conservative scientists; proponents of the junk-science critique argue that environmentalists exaggerate or fabricate data that support the role of environmental factors in disease and that this is a politically motivated assault on modern science (Samet and Burke 2001; Ong and Glantz 2001). Hunter et al.'s (1997) study, considered by many to be the most solid evidence against environmental causation, was accompanied by a deprecatory critique written by Stephen Safe (1997), a breast cancer researcher funded by chemical firms who has been an outspoken critic of environmental-breast-cancer research. He began his editorial by using the term *chemophobia* to imply that those interested in chemicals were merely paranoid, and he referred to environmental-breast-cancer research as "paparazzi science." Because this was published in the influential *New England Journal of Medicine*, the article was relatively influential. That the journal would even publish such a diatribe indicates the extent to which established science and medicine will go to discredit the environmental hypothesis.

Similarly, the scientific and media response to the August 2002 publication of the results of the Long Island Breast Cancer Study Project (Gammon et al. 2002) was exceedingly sharp and appeared to be more of an attack than a scientific discussion. Critics point out that the negative results from the Long Island Study show both that environmental causation is not a useful research direction and that public participation is dangerous for science. Scientists from well-known institutions who opposed this backlash were unable to get op-eds or even letters to the editor published, including James

Huff, a leading scientist at the National Toxicology Program, and Lorenzo Tomatis, former head of the International Agency for Research on Cancer. The fact that one of the two studies found a positive, if nonlinear, relationship between polycyclic aromatic hydrocarbons (PAHs) and breast cancer was virtually overlooked.

But even apart from the sharp attacks, the scientific findings from Long Island do put a powerful constraint on supporters of environmental causation. Many people had hoped for positive results, even in the light of activist criticism that the study had examined only a small number of the many chemicals that activists believed should be included. One activist pointed to the methods she felt were problematic:

> Off the top of my head, epidemiology is not really a very good science for noninfectious disease, particularly a noninfectious disease that is clearly environmental in origin, because in order to arrive at some significant statistical understanding of the outbreak of a disease, you need a control group. How can you have a control group? So all you get when you use epidemiology is a statistically insignificant result. So that epidemiology becomes an apologetic when it's used with cancer, and that's exactly what happened in Long Island. No statistical significance was found. On top of which, they looked only at chemicals in the blood.

Combined with Hunter's (1997) findings in the Nurses' Health Study, the Long Island results are a major blow to the search for environmental factors.

Occupational studies more often have found support for environmental causation (Labreche and Goldberg 1997). Hansen (1999) found that for 7,802 relatively young women employed in industries using organic solvents (e.g., textiles, chemicals, paper and printing, metal products, and wood and furniture), increasing duration of employment was associated with greater risk of breast cancer. Among those employed ten years or more in a solvent-intense industry, the adjusted odds ratio for developing breast cancer was 2.0 (Hansen 1999), meaning, on average, the risk of cancer doubles after ten years of occupational exposure to solvents. A recent study showed a connection between agricultural and manufacturing employment in which women were exposed to chemicals and increased breast cancer risk (Thomas et al. 2001).

Occupational studies may capture information about chemicals that do not bioaccumulate or dissipate quickly, and hence, are missed by body-burden studies. While occupational studies were conducted earlier and have been more supportive of environmental factors, they have tended to receive less attention in the scientific community. The low level of interest accorded to occupational studies is an indication of the fragmentation in current breast cancer research

and must be viewed as a major obstacle, even though that fragmentation is caused partly by some supporters of the environmental hypothesis.

Epidemiologists and toxicologists who study environmental factors in breast cancer understand that some of the issues and obstacles mentioned above will affect their work directly. From our interviews, we see how some scientists who were unable to detect environmental causation were disappointed, saying things such as, "I wanted to find something." They had a plausible hypothesis, were motivated by a desire to serve the public health, and expected that they might find positive results. One scientist said:

> The more people who are interested in something, I think, the better chance is that you're going to find out something about it. Just sort of by definition, you have to go looking more likely to find something.

But, failing to do so, they have not given up on such efforts. They have turned their efforts to new approaches and measurements, as with SSI's research (discussed in a subsequent section). Some have sought to advance what we term a critical-epidemiology perspective that challenges canons of traditional epidemiology (also discussed later on). Others have understood the difficulties in environmental epidemiology to be so substantial that they invoke the precautionary principle as a defensive public-health approach to reduce or ban chemicals even in the absence of definitive knowledge of environmental causation.

In summary, aided by a broad social awareness of the disease, environmental-breast-cancer research has been used to construct a more political-economic framework (e.g., the role of the economy in the increase in breast cancer; the role of laypeople in research) and to highlight new areas of legitimate scientific discourse (especially the endocrine-disrupter hypothesis). Our analysis is driven by an attempt to understand the obstacles to researching environmental causes of illness, and by extension, broader challenges to constructing new knowledge. Research at the environmental end of this second axis, however, remains a weak part of the overall field.

Axis 3: Lay Involvement in Research

Axis 3 captures the extent to which research involves lay-activists. At one end of the continuum, scientists work independently of lay input, and laypersons may serve as participants or subjects without contributing to research-question formulation, methods, data analysis, or dissemination of data. The opposite end of the continuum is anchored by cases with active

lay involvement, in which laypersons direct research efforts in collaboration with scientists. Here, nonscientist and lay knowledge are equally important components to the research and knowledge-production processes. In some cases, expert-produced science has not explained changes in health status adequately or has contradicted lay experience. As a result, laypeople and activists have generated new science while erasing lay-expert boundaries and transporting science into public spheres for public use. They accomplished this by seeking out independent scientific advisors, conducting health surveys, creating what we call lay maps, or interjecting lay knowledge into traditional, scientific ways of generating explanations. Lay participation also can take on other forms that lie between these poles, depending on the unique constellation of actors, resources, sociopolitical factors, and underlying philosophies about science.

The inherent uncertainty in breast cancer epidemiology makes citizen involvement particularly important. Lay activists, especially those who focus on environmental factors, push science in new directions, forge novel collaborations, and generate new methodologies. They do not accept data that contradict their lived experience, and they repeatedly insist that researchers and funders acknowledge the significance of their contributions. They do not see science as a privileged domain but as a tool to achieve their goals. Despite institutional barriers that may bar them from certain types of participation, they continue to push for involvement in several ways. Breast cancer activists have challenged researchers to explore environmental hypotheses, secured funding, worked to redirect research questions, critiqued outcomes, shaped public acceptance of projects outcomes, and called for additional research with retooled hypotheses and methods (Platner et al. 2002). As one activist noted:

> Power isn't only knowledge; it isn't only that I know how to run GIS statistical analysis. That's not all power is. Power is perspective; it is sensibility, and I even think power is intuition, too, even though they would probably totally disagree with that . . . Maybe you need to use the scientific process but you need to broaden the approach, broaden what you would ask, think of a new angle. There needs to be a lot more flexibility around doing that than there has been. Part of it is bringing new ideas and ideas to the table that scientists may not think about.

The magnitude of lay involvement in breast cancer research signifies the broad societal importance of the disease itself and is representative of campaigns for public representation in other illnesses as well.

Many activists, with the technical assistance from supportive epidemiologists, have achieved proficiency with the scientific literature to effectively engage scientists, navigate scientific arenas, and gain entrée into formal settings in which policy decisions are made (Dickersin et al. 2001). The National Breast Cancer Coalition's Project LEAD is one example in which lay activists participated in expert-led workshops on many scientific issues in breast cancer to prepare them for service on state and federal review panels. This is much further reaching than traditional, unidirectional technical assistance, which can translate experiential knowledge into scientific jargon or repackage experiential knowledge with a technical façade to augment its currency with decision makers. So while technical assistance represents a practical solution to facilitating citizen involvement within a slow-to-change system, it is not a form of democratic power sharing and knowledge transfer. On the other hand, direct citizen participation in the research process is a form of such democratic science practice.

Forming citizen-science alliances generally has been an effective vehicle for activists to engage more fully in scientific endeavors. This is exemplified in the work of the Environmental Working Group (EWG), which works with scientists to conduct impact research in which results are leveraged using savvy media and communication strategies to promote changes in environmental regulation and policy making. The EWG launched a national media campaign to publicize these results in advance of the official release of the CDC's National Report on Human Exposure to Environmental Pollutants. In 2003, the EWG released online a study titled "Body Burden" that involved a scientific collaboration with the Mount Sinai School of Medicine in New York and Commonweal (Environmental Working Group 2003). Results also were published in a peer-reviewed public health journal (Thornton, McCally, and Houlihan, 2002). Researchers found an average of ninety-one industrial compounds, pollutants, and other chemicals in the blood and urine of nine high-profile volunteers, including television journalist Bill Moyers and Andrea Martin, recently deceased founder and director of the Breast Cancer Fund. A total of 167 chemicals were found in the group. None of the people tested work with chemicals on the job or live near industrial facilities. The EWG's decision to collaborate on a body-burden study was strategic from a scientific and political point of view; indeed, results from body-burden testing often have resulted in swift action by government and corporate leaders. Following a medical study showing high mercury levels in the blood of patients whose diets were high in mercury-contaminated fish, the state of California recently sued several grocery

chains, requiring them to label these products. When a chemical found in Scotchgard stain repellant was found in virtually all Americans, 3M Company was compelled to change the formula, despite the lack of data on the health effects of the substance (Environmental Working Group 2003).

SSI is a classic example of a citizen-science alliance approach, as we found in our ethnographic observations. The SSI scientists are both similar to and different from many mainstream scientists. They work in an alternative institutional site that is not part of a university, think tank, or government agency. Their mission statement contains an affirmation of the citizen-science alliance as part of their work. Some of their scientists come from activist backgrounds and remain activists. However, while SSI scientists are appreciative of lay input, they are staunch in their unwillingness to alter their standards of proof. They believe that standards of proof are necessary for good science and for establishing their legitimacy in the scientific world. To do so, they believe, required "a lab of our own," as executive director Julia Brody said at a 2004 talk.

That sentiment of "a lab of our own" captures the desires of a whole movement of activists and scientists who want a society-wide lab of their own to examine environmental factors in breast cancer—not just physical settings for this kind of research but committed action on the part of the regulatory apparatus and research-funding network, a more inviting atmosphere from journal editors and reviewers, a firmer place in university curriculum, and a more balanced news media.

SSI sits in the middle of paradigm struggles about doing science in that SSI researchers support challenges that activists pose to traditional standards of proof, and yet, they consistently maintain academic rigor. In working toward these ends, SSI uses a wide variety of innovative methods to create a well-rounded understanding of the multiple possible factors that could be related to breast cancer incidence (Silent Spring Institute 1998). It includes GIS methods, survey methods, and novel epidemiologic and toxicologic methods. One innovation is exemplified by the development of a scientific shopping trip in which researchers collected samples of air and dust from various locations in stores and homes to be examined for endocrine disrupting and carcinogenic chemicals (Rudel et al. 2003). SSI initiated these new methodologies because of the need to collect information about exposures that women have in multiple locations and through time. This was also a change in focusing on the actual experience of women in their day-to-day lives rather than the general tendency of breast cancer research to examine chemicals in the lab. Therefore, SSI collected samples

from places women typically frequent, including sites not usually considered in epidemiological research, such as stores and dry cleaners. Through these techniques, SSI was able to conclude that hormonally active agents are common and important indoor exposures that require further research and to characterize chemicals previously unstudied (Rudel et al. 2001).

SSI's commitment maintains public channels of communication (including maintaining an office on Cape Cod), engages in outreach via educational sessions, and provides extensive scientific information to the general public. One example is the *Cape Cod Breast Cancer and Environment Study Atlas*,[3] which contains Cape-specific information on breast cancer incidence, historical pesticide use, drinking-water quality, census data, and land use such as the location of waste-disposal sites and the dramatic transition from forested land to residential housing. SSI believes that scientific data should be circulated widely so that people actually and potentially affected by breast cancer can have the fullest possible access to information.

Through citizen-science alliances, activists can alter scientific agendas by pressing scientists to think about the research questions they pose and the immediate relevance of their questions to community health and women's health in particular. Citizen-science alliances create new knowledge by using novel research designs and methodology (e.g., body-burden testing and GIS mapping) informed by the personal experiences of women with breast cancer. In doing so, these alliances have the potential to influence permanently the methods through which future studies are conducted, because of new ideas introduced by those usually excluded from the scientific realm (McCormick, Brown, and Zavestoski 2003). Citizen-science alliances also often are framed in ways in which study results can be leveraged effectively to expedite policy action. In this way, the shift toward increased lay-activist participation in scientific research has influenced and/or parallels shifts in breast cancer epidemiology previously mapped along Axes 1 and 2.

Ultimately, the significant directions in lay involvement in review panels and research participation have not been matched by the inclusion of laypeople in the policy apparatuses where regulatory decisions are made. So, activists and advocates have become part of two of our arenas—doing science and interpreting science—but have not been integrated into acting on science. Activists seek implementation of policies they believe are useful; for example, a coalition including the Breast Cancer Fund sued the EPA to speed up its endocrine-disrupter analysis. This demonstrates that activists need to take exceptional measures to become part of the policy practice.

Conclusion

We have examined the disputes about environmental factors in breast cancer research by identifying three paradigm challenges posed by activists and some scientists to the broader scientific community: (1) to move debates about causation upstream to address the cause; (2) to shift emphasis from individual to modifiable societal-level factors beyond an individual's control; and (3) to allow direct lay involvement in research, which may raise new questions and change how questions are approached, the methods used, and even the standards of proof. This three-dimensional model enabled us to examine controversies in different arenas of the scientific process: doing scientific research, interpreting science, and acting on science.

According to the activists and researchers we interviewed, individual risk factors do not explain breast cancer etiology satisfactorily; therefore, the search for alternative variables (including environmental factors) and alternative methods remains an important agenda point for many breast cancer researchers and activists. Key studies of breast cancer and the environment have yielded equivocal results, showing that this area of research is a work in progress. The lack of generally acceptable answers to the question of what causes breast cancer has prompted people to look at the question differently. In doing so, environmental-breast-cancer research has opened up pathways for more primary prevention research such as endocrine-disruptor research and a resultant focus on precautionary principle. It also has identified novel methodologies with which to explore breast cancer etiology further. This methodology orients research upstream to seek out preventable causes, includes lay input, and considers community and population-level factors. In addition, proponents of environmental causation theories have brought attention to interaction effects, both those involving two or more chemicals and those involving chemicals plus other factors (e.g., smoking, alcohol, viruses).

When environmental factors are the subject of inquiry, discussions of science are contested highly because they lead to potential challenges to the underlying production, distribution, disposal, and regulatory practices of our society. For example, for Axis 1—upstream prevention versus downstream action—corporations and government regulators will be unlikely to accept willingly the removal of many chemicals from circulation or to bear the exorbitant costs of extracting these chemicals from the environment. For Axis 2—individual- versus community-level research—corporate interests likely will oppose a research agenda that targets their products rather than

individual susceptibilities. For Axis 3—degree of lay participation—corporate and governmental actors likely will oppose strong lay participation since such involvement often stems from an approach that values community over corporate rights and that supports greater democratic participation in science, which in turn invites challenges on the other axes.

New methods of knowledge construction that account for lay perspectives are pivotal in creating new means to study and understand environmental causes of disease. While scientists involved with breast cancer advocates strongly uphold scientific rigor, the activist agenda also has facilitated new methodologies and proposed a new set of norms for standards of proof through the precautionary principle. By tracing the struggles about breast cancer research approaches, this analysis contributes to medical sociology and social studies of science and knowledge by theorizing about impediments to our comprehension of potential environmental causes of illness, and on a larger scale, our ability to construct new knowledge. Our discussion of impediments does not imply that there can be an obstacle-free science; rather, we argue that the DEP obstructs science from considering the role of environmental factors, which accounts for a component of the broader science.

The paradigm challenges to breast cancer research also occur around environmental health more broadly. Therefore, we aim to understand what impedes construction of a new body of knowledge about environmental factors in human illness. This will be helpful as science and the public turn attention to environmental factors in other diseases, including asthma, Parkinson's disease, and autism. Our conceptual framework of three axes can be applied to other areas of research on environmental health, and our approach to the scientific process (doing, interpreting, and acting on science) can be applied more generally to other scientific issues in which scientists and citizens advocate for "a lab of their own."

Notes

1. By environmental, we mean here the effects of toxic substances in people's immediate or proximate surroundings—primarily chemicals, air pollution, and radiation.

2. Epstein (1978) historically characterized mammography as a key tool of prevention. However, because of protests from many breast cancer activist groups, mammography now is billed more accurately as an early-detection technology.

3. See http://www.silentspring.org/newweb/atlas/index.html(accessed September 19, 2003).

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