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Recently, two prescription drugs (tamoxifen and raloxifene) have become salient to breast cancer prevention. With the advent of these drugs, referred to as "chemoprevention," a mandate has emerged to classify certain women as high risk for breast cancer to determine a group of legitimate users of the drugs. This article examines the development and standardization of the model used to create such a group of high-risk women. The author argues that while the model remains uncertain and controversial, it has become the standard tool for the many jobs associated with legitimizing chemoprevention use in the United States. It has become the assumed standard—shaping practices, identities, and definitions—through its organizational embeddedness in the multiple practices and public images of chemoprevention despite its uncertainty and widespread critique.

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Recently, two prescription drugs, tamoxifen and raloxifene, have become salient to breast cancer prevention. With the advent of these drugs, referred to as "chemoprevention," a mandate has emerged to classify certain women as high risk for breast cancer to determine a group of legitimate users of the drugs. This article examines the emergence of a group of women designated high risk and the tools and devices that enable such classification. In particular, I explore the current standard tool for breast cancer risk assessment in the contexts of chemoprevention in the United States: the "Gail model,"

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describing its development and analyzing how and under what circumstances it became the "right tool for the job" (Clarke and Fujimura 1992). In this article, I argue that while it was initially used for the convenience of facilitating recruitment into a clinical trial, it has subsequently become the standard tool for the many jobs associated with legitimizing chemoprevention use in the United States. It has become the assumed standard—shaping practices, identities, and definitions—through its organizational embeddedness in the multiple practices and public images of chemoprevention despite its uncertainty and widespread critique.

This article draws from a social worlds analysis (Becker 1982; Casper and Clarke 1998; Clarke 1990, 1991, 1998; Clarke and Montini 1993; Strauss 1978, 1991) of the Study of Tamoxifen and Raloxifene (STAR), the largest breast cancer prevention clinical trial to date. My research employs a multisited ethnographic approach to explore the many social worlds that congeal to make the STAR trial possible. Multisited ethnography emphasizes local knowledge and multiple voices (Jasanoff et al. 1995), making it an ideal method for social worlds analyses and enabling the tracking of a particular biomedical phenomena—a chemoprevention clinical trial—across local and global domains and as it manifests in the lived experiences of those I interview (Rapp 1999).

My data sources include (1) qualitative, open-ended interviews with multiple participants on the STAR trial (women participating as research subjects, clinical trial coordinators, principal investigators, biostatisticians, health care providers, pharmaceutical representatives, and others); (2) qualitative, open-ended interviews with key informants important in the development of chemoprevention for breast cancer and risk assessment tools; (3) participant observation at professional meetings, informational sessions, and other sites of discussion about the STAR trial, risk assessment, and risk reduction; and (4) textual materials such as archival data, pharmaceutical company materials, clinical and popular literatures, and FDA proceedings. Data derived from each of these sources were entered into QSR NUD*IST software and analyzed using grounded theory methodology.

Background

Despite decades of research and billions of dollars spent on the "war against cancer," there remain few options for primary breast cancer prevention. Knowledge about what causes breast cancer is scientifically and politically problematic to ascertain. Given what is known, or more important, what is unknown about breast cancer etiology, there are few options for primary prevention. Primary prevention is further confounded by the fact that identi-

fying and eliminating a factor associated with increased breast cancer risk does not necessarily translate into disease prevention: great numbers of people must change their behaviors for even a few cases of the disease to be prevented. Chemoprevention has emerged within the past decade as one of the few options for breast cancer prevention and has not proven safe enough to be applicable to great numbers of people. Therefore, the use of chemoprevention drugs requires risk calculations that can accurately pinpoint a group of women for whom the intervention is likely to make a significant difference.

Currently, the only FDA-approved chemoprevention for breast cancer is tamoxifen, a drug produced and marketed by AstraZeneca under the brand name Nolvadex. Tamoxifen selectively interferes with estrogen actions: in some organs, notably the breasts, tamoxifen occupies the estrogen-receptor site and thus blocks endogenous estrogens that would otherwise bind to the site (Overmoyer 1999). Because estrogen is believed to encourage the growth of breast cancer, by blocking estrogen from the breasts or from a breast tumor, tamoxifen discourages breast cancer growth. In other sites, however, tamoxifen mimics estrogen. Thus, paradoxically, tamoxifen can cause cancer in the lining of the uterine wall because at that site, it produces estrogen-like effects. The way in which tamoxifen selectively interferes with or mimics estrogen in different parts of the body makes it part of a class of drugs called selective estrogen receptor modulators (SERMs).

In September 1998, the final results of the clinical trial that led to the Food and Drug Administration (FDA) approval of tamoxifen for risk reduction (Breast Cancer Prevention Trial [BCPT]) were published (Fisher et al. 1998). The results indicated that 124 women developed breast cancer (invasive and noninvasive) in the tamoxifen group and 244 women developed breast cancer in the placebo group. However, tamoxifen was also found to increase the risk of endometrial cancer (cancer in the lining of the uterus): 36 women in the tamoxifen group and 15 in the control group developed uterine cancer (37 percent of women in each group had undergone hysterectomies prior to enrollment). Tamoxifen also increased the incidence of pulmonary embolism (blood clot in the lung: 18 cases in the tamoxifen group, 6 in the control group) and deep vein thrombosis (blood clots in major veins: 35 in the tamoxifen group and 22 in the control group). The large numbers of these other life-threatening illnesses has led some to call tamoxifen "disease substitution" (Sherman 1998).

Another SERM drug, raloxifene, is currently being tested against tamoxifen as a chemopreventive agent that may or may not have as many life-threatening side effects. Raloxifene (Evista) is produced by Eli Lilly and was approved by the FDA in 1997 for prevention of osteoporosis in postmeno-

pausal women. In the Multiple Outcomes of Raloxifene Evaluation trial, designed to investigate the benefits of raloxifene on the incidence of bone fractures, a subset analysis found it also decreased the incidence of breast cancer without the accompanying rise in endometrial cancer. The results were considered promising enough to warrant further clinical investigation and, together with the results of the BCPT, helped to prompt the initiation of STAR. STAR is a randomized, double-blind clinical trial that compares tamoxifen treatment with raloxifene for the reduction of risk of breast cancer. There is no placebo arm of the study, indicating that tamoxifen has already come to be considered the standard of care for chemoprevention against which new drugs will be compared.

For a prevention strategy such as chemoprevention to be marketable in the face of high costs such as endometrial cancer and thromboembolic events, a category of women is necessary for whom the benefits of taking chemopreventive drugs are perceived to outweigh the risks associated with them. For chemoprevention to be a viable idea, "high risk" as a concept needs to be constructed, agreed on, and mapped onto individual women's bodies so that individual women, health care providers, and others can decide whether the benefits of taking a drug such as tamoxifen outweigh the risks, given an individual woman's particular level of risk for breast cancer.

Assessing a woman's breast cancer risk to decide whether to biomedically intervene is part of a larger recent phenomenon of "diagnosing risk" as a disease state in and of itself. This creates a category of people who Margaret Lock (1998, 9) calls "the pre-symptomatic ill." Within this model, we are all becoming ill because we are all, to varying degrees, at risk for something. The consequences of categorizing people as high risk are rarely neutral or unproblematic. Being diagnosed "at risk" has numerous potential negative implications—from discrimination faced at work or with insurance coverage to an increased sense of unease and distrust with one's own body. On the other hand, such categorization may provide access to life-saving biomedical interventions.

This shift to diagnosing risk as an illness category in and of itself is reflective of the recent phenomenon of biomedicalization (Clarke et al. 2000). Within biomedicalization, technoscientific tools and innovations help to create new kinds of at-risk subjectivities by providing access to knowledge about the body that was previously inaccessible. Historically, breast cancer was diagnosed when women presented with ulcerating tumors, had breast pain, or had palpable lumps. As detection technologies advanced, breast cancers were detected earlier and earlier. Today, mammography and biopsy technologies enable diagnosis of "precancers" (ductal carcinoma in situ

[DCIS] and lobular carcinoma in situ [LCIS]), controversial and uncertain designations indicating cancer to some and risk to others (see, e.g., Lerner 1998).[1] Now, with the availability of drugs for prevention, a mandate has emerged to diagnose cancer even earlier, when it is still merely a risk for and not an actual entity.

Risk discourses can be thought of as attempts to tame uncertainty, to control undesirable outcomes by identifying pathologies hidden deep within the body (Lupton 1995). In contemporary society, most events are assumed to be governed by natural laws, predictable and thus controllable (Lock 1998; Porter 1995). Statistics and prediction emerged as scientific tools for managing uncertainty. By making accurate predictions about the world, probability theory provided what were considered to be rational solutions to acting in uncertainty (Porter 1995). Constructing predictive models of risk creates groups to whom undesirable outcomes are considered most likely to occur, thus attempting to manage the inherent uncertainty of risk.

What Lock (1998) and others (e.g., Gifford 1986) point out, however, is that risk is far from self-evident. The processes through which statistical probabilities are translated into meaningful, individualized information are problematic. Such assessments involve negotiation and often conflict among perspectives. This is because particular interpretations of statistical probabilities come to dominate. Lupton (1995) asserted that the precise mathematical calculations used to assess risk obscure social processes and controversies. Here, I attempt to make visible the social and sometimes controversial processes underlying mathematical calculations for breast cancer risk.

Diagnosing Breast Cancer Risk

The Gail model has become the standard tool for the classification of high risk for breast cancer for the purposes of chemoprevention in the United States. The Gail model, named after Mitchell H. Gail, the principal investigator of the research on which the model is based, was used to calculate "high risk," a designation tool that would determine a woman's eligibility for participation in the BCPT. This trial resulted in FDA approval of tamoxifen for the reduction of breast cancer incidence in 1998. Through this approval process, the Gail model became the legally accepted way to calculate who is a candidate for this drug in clinical practice. The Gail model has since become increasingly embedded in much wider practices, discourses, and representations of breast cancer risk and chemoprevention. But what is the Gail model? What does it calculate, and how does it construct these seemingly

clear boundaries around "normal" and "high" risk in a knowledge arena that is steeped in controversy, conflict, and uncertainty?

The Gail Model: What Is It?

The Gail model was developed using data on breast cancer risk from research conducted with participants of the Breast Cancer Detection and Demonstration Project (BCDDP). Between 1973 and 1975, the BCDDP, conducted by the American Cancer Society and funded by the National Cancer Institute (NCI), recruited approximately 280,000 women aged thirty-eight to seventy-four years from twenty-eight centers in the United States to be screened for breast cancer annually for five years (Gail et al. 1989, p. 1880). It was subsequently realized that the women participating in the study and the data collected about them were an incredibly valuable cohort. The cohort became a resource for various other research projects, including the research on which the Gail model is based.

In the 1980s, Mitchell H. Gail, a biostatistician at the NCI, and his colleagues used data from the BCDDP and a subsequent case-control study conducted with participants of BCDDP to develop a model that could calculate a woman's absolute risk of developing breast cancer, the probability that a particular woman with a particular set of risk factors will develop breast cancer over a given time period. According to Gail, realization of the need for such a model arose from conversations with a doctor who was treating patients coming to the NCI for breast cancer counseling. This doctor wanted to be able to give the women he counseled particular, individualized risk information, but at the time, there were only methods of predicting relative risk, the ratio of the incidence rate of age-specific breast cancer among women with a particular set of risk factors and the incidence rate among women without known risk factors. As Gail (interview by J. Fosket, March 1, 2001) stated, "many studies have produced relative risk so you can compare the risk of a woman with particular risk factors to the risk of a woman without any risk factors. But to actually make a clinical decision you need to know what the actual chance is over a particular time period." No such individualized model existed.

According to Gail, the development of such a model would ideally emerge from a large-scale cohort study in which information on potential risk factors and follow-up information were collected from all participants. The BCDDP closely approximated this ideal scenario except that information on potential risk factors was not collected on all women. However, it was collected on a subset of 2,852 women with breast cancer and 3,146 controls (Gail et al. 1989, 1880) for a separate case-control study using participants from BCDDP. Here, demographic information, family and medical history infor-

mation, and health behavior information were collected to determine what factors contributed to breast cancer incidence. The results demonstrated that age, family history of breast cancer in a first-degree relative, late age at first childbirth, early menarche, and multiple previous benign breast biopsies were most significantly related to increased breast cancer risk (Gail et al. 1989).

Using these data from the case-control study together with the data from the larger BCDDP study, Gail and colleagues were able to proceed as if they had known the risk factor information for every woman in the cohort. For the entire cohort of 280,000 women, age-specific absolute risk was known because their ages were known, and follow-up data enabled researchers to see what percentage of women in what age group went on to develop breast cancer. The case-control study yielded relative risk information, and by combining this with the absolute age-specific risk information gleaned from the cohort study, Gail and colleagues were able to produce risk-factor-specific absolute risk.

National Surgical Adjuvant Breast and Bowel Project (NSABP) statisticians Carol Redmond and Stewart J. Anderson subsequently modified the Gail model for use in the BCPT. The modification predicts only invasive breast cancer rates and replaces the incidence rates derived from BCDDP with age-specific invasive breast cancer incidence rates in the NCI's Surveillance Epidemiology and End Results database. These data are then combined with the relative risk information derived from the case-control study and calculated via the same formula as the original Gail model to produce absolute risk estimations. They also provided separate estimations for black women and white women. This modification is now the standard Gail model. It was legitimated through its statistical validation on the BCPT, where the expected and observed numbers of breast cancer cases were closely matched[2] (Costantino et al. 1999).

Defining "High Risk"

The Gail model assesses a woman's risk of developing breast cancer and articulates the information as a percentage—a number that signifies the probability that she will develop breast cancer over the next five years. The ability of the model to abstract the complexity and uncertainty of a woman's multiple risk factors into a simple number contributes to its success. Numbers are eminently mobile, decontextualized, and factlike. They can travel widely, regardless of individual interpretations of them. However, assessing what the number produced by the Gail model means is also an interpretation, not an inherent fact. For example, the Gail model might tell you that you have a 2.3

percent increased absolute risk of developing breast cancer, but whether that is an average, excessive, or minimal risk is a decision that requires interpretation and negotiation. It is such processes of interpretation that classifies a particular risk number produced by the Gail model as "high."

Currently, "high risk" is classified as 1.7 (rounded up from 1.66). That is, high risk is defined as anything including and above a 1.7 percent estimated risk of developing invasive breast cancer over the next five years. Put another way, high-risk women include women whose estimated risk of not developing breast cancer over the next five years is 98.3 percent or lower. Clearly, in some contexts, having a 98.3 percent estimated risk of not developing breast cancer over the next five years might be deemed considerably good odds. So, how did this number come to define high risk for breast cancer?

The current standard definition of 1.7 (rounded up from 1.66) as equaling high risk resulted from decisions surrounding who would be considered eligible for the BCPT when the trial was being designed in the early 1990s. As the first major clinical trial testing an intervention for the prevention of first-time breast cancer incidence, there were no standards to use to decide which women should be considered high risk enough to qualify for participation in the study. Instead, this trial was setting the stage for future definitions of high risk. In addition to the ethical dimensions of including in the study only women at high risk for breast cancer, as with other clinical trials, researchers wanted to recruit women at high risk to maximize the effect demonstrated. Dr. Costantino (interview by J. Fosket, November 20, 2000), the coordinating statistician of the BCPT (as well as STAR), described these decisions to me:

> We needed some way to assess risk. When you're designing a trial you want to design it to address a population who are at high risk for the disease. Now one way that you can do that is just to use age and other demographic factors. . . . If you look at the rates of disease by age for breast cancer you can see that by age 60 the rates of breast cancer are pretty high. So, one way, we said okay, well we'll just do this study among women who are age 60 or older and on average they should be at high risk. But, we then said, we can't just leave out all of the women who are less than sixty because the drug's going to be used in all women and we need to be able to have a way to include women who are under sixty, but they have to be at high risk. So, we said, well how can we determine what their risk is? Now, there were a couple of models at that time that were out there for predicting breast cancer risk. . . . But, the other ones were really specifically designed to assess women's genetic risk for breast cancer not the overall general risk in a general population. And I should say, Gail model was the only model out there that had been validated that was a general model. So, we took it for that reason, since it was the only validated model that would be good to predict in the general population since only about less than five percent of

> the women have a genetic disease, so, you want a model that's going to be applicable to at least 95% of the women. And that's the one we chose.

While high risk was defined as 1.7 for the BCPT, in practice, the average risk calculated by the Gail model for participants in the trial was 3.2 percent (Chlebowski et al. 1999). This means that, on average, the women who participated in the trial were at higher risk than was minimally required for eligibility. However, because the eligibility for the trial began at 1.7 percent, this has been standardized as the high-risk threshold. It became the number that defined high risk in the FDA approval of tamoxifen and is the number disseminated in information and advertising of tamoxifen. Thus, the definition of high risk is at least 1.7. Social constructionist perspectives of technology emphasize that in looking at technologies, it is important to consider the interests and commitments embedded in the technologies themselves. In this case, given that the average risk number in the BCPT was 3.2, and yet the subsequent risk threshold that has come to define high risk is 1.7, one obvious benefit of this particular tool is to those whose interests lie in casting a wider net in the classification of high risk. In this case, 26 million U.S. women (as publicized by the NCI) are estimated to be eligible consumers of chemoprevention based on the Gail model–derived, high-risk threshold of 1.7. While the use of 1.7 initially arose to accommodate the requirements of a clinical trial, it has had far-flung consequences in constructing a category of women who are seen, and may see themselves, as high risk.

"The Right Tool for the Job"

The Gail model has emerged as the "right tool" for the "job" of breast cancer risk assessment for chemoprevention. Clarke and Fujimura (1992) articulated the theory that "tools," "jobs," and "rightness" are co-constructed through social processes of developing doable problems, crafting/tinkering, and making ad hoc arrangements to address those problems. Rightness is produced through (inter)actions and negotiations and is not a special property of a particular tool or a particular job. Following these assumptions, I explore the ways that the rightness of the Gail model emerges as a social process. Fundamentally, I argue that the Gail model has become the "right tool for the job" through its organizational embeddedness in the practices of chemoprevention. The model is embedded in health care practice, research, policy, and marketing and is thus resilient even in the face of critique and criticism.

Organizational Embeddedness

The rightness of the Gail model for the job of assessing breast cancer risk in the context of chemoprevention is very much an example of the crucial role of organizational embeddedness to the maintenance of a tool as "right for the job" (Casper and Clarke 1998; Clarke and Casper 1996; Griesemer 1992). The Gail model has become the right tool because it is so entrenched in the formulation and execution of the job of risk assessment for chemoprevention. Here, I describe the embeddedness that emerges as a result of the Gail model's use in research that served as a proof of principle of chemoprevention for breast cancer and its subsequent translation into policies associated with chemoprevention. Then I describe how the manufacturing and distribution of the model into simple devices for clinical risk assessment and the marketing of tamoxifen and breast cancer risk have further entrenched the model in the practices of chemoprevention.

Research and Policy

Through my interviews with researchers on the BCPT, I learned that when this trial was initiated in 1992, the Gail model was not taken for granted as the right tool for the job of calculating high risk for this trial. Instead, various options were discussed, with the Gail model being the one that was ultimately chosen. The use of the Gail model for this trial ended up having far-reaching consequences in policy, clinical practice, and research. Most significantly, the use of the Gail model in the BCPT led to its embeddedness in policy guidelines for tamoxifen. Once the tool was used in the BCPT, it became embedded in the subsequent FDA approval of tamoxifen for risk reduction, which was based on the results of the BCPT. The usefulness of tamoxifen for risk reduction had only been "proven" within the clinical trial, and thus the FDA used the same calculation for general tamoxifen eligibility (1.7) as was used in the trial. Dr. Costantino (interview by J. Fosket, November 20, 2000) stated, "Because it was used to help define eligibility for the trial when the FDA approved the drug tamoxifen for the use of prevention it said, only among those individuals who are shown to be eligible as was defined by the NSABP study [BCPT]. Which means you have to use the Gail model to assess eligibility and that more or less made it the standard."

Through the FDA approval process, use of the Gail model was extended beyond the parameters of the BCPT and into general biomedical practice. The connections between policy and scientific research means that the conceptualizations used in research are often mirrored in policy, and in this case,

the very definition of appropriate users of chemoprevention[3] relies on the Gail model. So, in practice, the Gail model defines the parameters of breast cancer risk. Tamoxifen for risk reduction can be prescribed only to women who are at high risk for breast cancer, and high risk is understood in this context as having a 1.7 percent increased risk of developing breast cancer as calculated vis-à-vis the Gail model.

Furthermore, the use of the Gail model to calculate risk in the BCPT resulted in its embeddedness in the research process itself. Part of the work that classification systems do is to render things equivalent so that they can be compared across sites (Bowker and Star 1999). Biomedical research places great faith in the ability to compare research to produce credible knowledge. The Gail model has categorized high risk in a way that can be compared across multiple prevention trials. To begin with a new model would be perceived as undermining the research process, and thus commitments to the Gail model run deep. In addition, most major research projects must be approved by internal and external human subject review boards and must also undergo assessment and evaluation to secure funding. Since the Gail model has already been statistically validated in a research context in which such validation means a great deal, it is unlikely that any new project attempting to use a different definition of risk would be accepted by any of these reviewing entities (unless it were explicitly comparing a new model to the Gail model).

Bowker and Star (1999) underscored the ubiquity of classifying and standardizing, such that any particular classification system will be itself embedded and shaped by a complex web of interdependent other systems. This is evident in breast cancer risk, in which a standardized classification of high risk is necessary because of multiple other classification systems—the regulatory classification of appropriate users of pharmaceuticals, human subject review boards, and other research guidelines classifying what counts as ethical clinical research. These and others embed the Gail model into the complex web of ongoing practices of chemoprevention.

Devices

The Gail model has become the standard practice for assessing breast cancer risk and, as such, it has been packaged into user-friendly devices to make it accessible to, and easily usable by, health care providers. The NCI and AstraZeneca currently manufacture these devices. The latter's devices are considered promotional materials for tamoxifen (which they manufacture under the name Nolvadex) and must therefore be FDA approved. Here, I describe the "risk disk" developed by the NCI and widely disseminated to

provide easy access to risk assessment after the results of the BCPT were announced. The assumption was that with the positive results of the trial, women would be asking for tamoxifen and doctors would be wanting to prescribe it but would not know how to assess risk to determine a woman's eligibility for the drug. This device was meant to bridge that gap in knowledge.

The disk loads easily onto a computer and takes the user through a step-by-step process visually resembling a slide show. The first slide reads "Breast Cancer Risk Assessment" in white lettering against a blue background. In the corner are the words "The National Cancer Institute," written in their distinct red and white font. The middle of the slide reads "An Interactive Tool to Measure a Woman's Risk of Invasive Breast Cancer." The next slide explains that the device was developed by scientists at the NCI and the NSABP to allow health professionals to estimate a woman's individualized risk of developing invasive breast cancer. The explanation notes that data for the device came from the Breast Cancer Detection and Demonstration Project, funded by the NCI. Curiously, it never mentions that the computer program is based on the Gail model. Indeed, there is no mention of the Gail model anywhere in the disk or its accompanying materials,[4] indicating the invisibility of the tool once standardized.

The next slide gives the user the option of learning more about breast cancer risk or calculating a patient's breast cancer risk. In choosing the first option, the user is provided a list of risk factors. Next, the user is told about tamoxifen. Information about tamoxifen—its risks, benefits, and future prospects—fills the rest of the slides. In total, there are seven slides devoted to tamoxifen, one to raloxifene and tamoxifen, only one slide devoted to breast cancer risk, and one slide that attempts to help the user (presumably a health care provider) explain the risk information to their patients. This disproportionate emphasis on tamoxifen highlights that while the risk disk is packaged and marketed as a device for assessing breast cancer risk, its commitments and interests in doing so are to market tamoxifen to consumers.

On taking the second path (calculating breast cancer risk) the risk assessment process begins by asking a series of questions, one per slide, that can be answered by clicking on "yes" or "no." The first question asks about previous diagnoses of LCIS or DCIS, factors that were not included in the Gail model. If the answer is "yes," the program stops at the next slide—a slide that provides an explanation that previous diagnoses of LCIS or DCIS increase breast cancer risk, that this device is not meant to accurately assess risk for women with such histories, but that evidence suggests that such women would benefit from tamoxifen therapy for reducing the risk of invasive breast cancer. Again, a clear goal of the device is to promote tamoxifen.

If the user answers "no" to the question about LCIS and DCIS, the next slide begins a series of questions about the presence or absence of each of the Gail model risk factors: age, age at first menstrual period, age at first live birth, number of first-degree relatives (mother, daughter, sister) diagnosed with invasive breast cancer, and whether the woman has ever had a biopsy. The user is also asked to classify the woman by race: white, black, or Asian. There is no "other" category. If these three choices do not fit, the only option is to leave the question blank all together, in which case, as the text informs the disk's users, "the program will use data for white females to estimate the predicted risk." Once each of these questions is answered, the user can calculate five-year risk and lifetime risk. The results are represented numerically as a percentage (i.e., 1.7 percent) and as a graph. In addition, the particular risk assessment is compared both numerically and graphically to another hypothetical woman of the same race and age but with no other risk factors.

In addition to the risk disk, there are numerous other user-friendly devices derived from the Gail model to assist in risk assessment processes in the clinic and research settings, including a handheld calculator and a cardboard booklet. The existence of such devices further entrenches the Gail model by increasing the ease and convenience of its use in clinical and research practices. Furthermore, these devices tightly couple the Gail model with breast cancer risk assessment by increasingly being the only tools available for such assessments. This tight coupling is also achieved through the marketing of tamoxifen, which discursively relies on the Gail model–derived 1.7 risk number.

Marketing Risk

Soon after receiving FDA approval for tamoxifen for the reduction of incidence of breast cancer, AstraZeneca embarked on a major direct-to-consumer advertising campaign of tamoxifen. In 1997, the FDA "clarified its rules" on pharmaceutical company advertising, resulting in a relaxation of its restrictions on the kinds of advertising permitted and enabling mass media advertising of prescription drugs to the lay public (Findlay 2000, 1). This was a major shift; previously, prescription drugs were predominately marketed to physicians since such drugs require a doctor's prescription, presumably following professional diagnosis and risk/benefit analysis. Since the relaxation of marketing restrictions, prescription drugs advertised directly to consumers represent the largest and fastest growing drug sales (Findlay 2000). With direct-to-consumer advertising, prescription drugs have entered into the public imagination to a much greater extent and are depicted as any other con-

sumer good, in that like soap, clothes, or a new car, they can be purchased freely in an effort to enhance one's life.

AstraZeneca's tamoxifen advertising campaign focuses on the desirability of knowing one's risk status as the central message, featuring tamoxifen secondarily. Fundamentally, this campaign aims to sell risk and risk assessment, tapping into women's worries about breast cancer and then selling tamoxifen as the solution to the problem of risk. One widely distributed print ad depicts a young, thin, white woman in a black lacy bra and underwear. She is seated with her back to the camera on what appears to be a bed. The ad depicts just her torso, cutting off her head above her neck. The large print across the photo reads, "If you care about breast cancer, care more about being a 1.7 than a 36B." Smaller print under the photo reads, "Know your breast cancer risk assessment number," and below that, "Know that NOLVADEX® (tamoxifen citrate) could reduce your chances of getting breast cancer if you are at high risk." Below these words, smaller print describes the risk assessment process and informs readers that "a score of 1.7 or above is considered high risk. Most likely you won't be at high risk, but you owe it to yourself to find out. Knowing your number gives you power, and knowing about Nolvadex should give you hope." The risks associated with tamoxifen are briefly described, and women are urged to call their doctors and ask for a risk assessment test. A toll-free number (Zeneca's) is provided to receive a free video and more information. Finally, large print finishes off the ad at the bottom of the page reading, "NOLVADEX® Tamoxifen Citrate, There *is* something you can do."

The rhetoric embedded in this ad highlights risk assessment as a new tool of individual empowerment. Reference to "knowledge" is repeated several times, and risk assessment is aligned with knowing about one's body. Knowledge of one's risk number is explicitly stated to give one "power." The rhetoric of self-knowledge as empowerment mirrors messages originally deriving from the women's health movements, and its use here in an advertisement benefiting the pharmaceutical industry highlights the co-optation of the rhetoric of the women's health movement by dominant biomedicine (Clarke et al. 2000; Clarke and Olesen 1999; Fosket, Karran, and LaFia 2000; Whatley and Worcester 1989). Such rhetoric removed from the context of feminist empowerment practices is turned on its head and (re)scripted into a blame-the-victim message, mandating women to undergo the risk assessment process because they "owe it" to themselves. The burden for knowing such information is placed on women, with the implication that this can somehow assist them in not developing breast cancer. This rhetoric reinforces ideologies of individualism and the idea that health is an individual responsibility.

The number 1.7 is represented as an individualized risk number, promoting the alluring notion that one can use statistics and medical science to uncover customized information about just them. Once individual risk is "discovered," the individual woman is "empowered" to take individual action, to take responsibility for her risk through the tamoxifen "solution."

The ad attempts to make 1.7 a familiar number, as familiar as one's bra size (36B). Rhetorically, as Press and Burke (2000) pointed out in their critique of this advertisement, by aligning it with 36B, 1.7 is constructed as an inherently meaningful number, internal to each woman. As 36B measures the actual shape and size of an individual woman's body, by symbolically representing 1.7 as an equivalently meaningful measurement, this "risk" number appears to also measure something tangible and material, as opposed to a handful of factors that have been stabilized into a risk assessment model.

In addition to aligning 1.7 with bra size, the ad claims that women should care more about being a 1.7 than they do about being a 36B. By urging women to care more about their so-called risk number than they do about the size of their breasts, the discourse embedded in this advertisement again co-opts messages from women's health movements by implying that concerns over body image are distracting women from more important health issues, such as cancer. When originating in feminist-oriented women's health, such messages typically critique structural inequalities that maintain a focus on women's bodies as sources of sex and beauty while ignoring their health and well-being. In contrast, as is often the case when feminist projects are co-opted in attempts to sell products to women, all progressive social implications of such a message are stripped in this ad and women are depicted as shallow and mindless (literally headless) sitting around in their black lacy bras worrying about breast size when they really should be worrying about breast cancer. Relying as it does on stereotypically rendered images of women as sexualized objects, the discourse embedded in this ad paradoxically also functions to displace women's breasts as embodied and individually meaningful (i.e., as sources of sexual pleasure, nurturance, or pride) and reconstruct them as sites of danger—risk for cancer.

Fundamentally, this advertisement campaign and others like it are enormously successful in tightly coupling the Gail model–derived number 1.7 with high risk for breast cancer. This succeeds in further entrenching the model in the practices of risk assessment for breast cancer. As women and health care providers alike become increasingly familiar with the number 1.7 as a definition for high risk, inserting a new risk model that would result in new risk language becomes increasingly difficult. Through its packaging in user-friendly devices and the marketing of its risk number to sell pharma-

ceuticals, the Gail model has become ubiquitous in the realm of chemoprevention for breast cancer. In the next section, I interrogate how this rightness undergoes continual negotiation in everyday practice.

Contesting the Gail Model: Dissenting Voices

The "rightness" of a tool is situationally constructed in conjunction with the job itself, and different interests may conceive of the job differently. Casper and Clarke (1998) highlighted the importance of asking the question, For whom is a particular tool conceived of as right? "Rightness," "tools," and "jobs" must be explored together, as each will emerge in unique ways in specific situational and historical contexts (Clarke and Fujimura 1992). In addition, becoming the right tool does not necessarily imply stabilization, as the rightness of a tool may be constantly undergoing negotiation (Casper and Clarke 1998). This holds strongly in the case of the Gail model. Importantly, the Gail model is not considered the right tool for all breast cancer risk assessment jobs, and it remains controversial and contested at many sites.

Critiques of the Gail model emerge from multiple social worlds and by multiple social actors. They emerge from epidemiologists, breast cancer activists, and those who use the model and are in all other ways advocates of the model but who nevertheless raise particular limitations of the model. For instance, breast cancer activists have been outspoken critics of chemoprevention for breast cancer and describe breast cancer risk as inherently uncertain and political, and thus assessing such risk to prescribe pills for prevention is viewed as extremely problematic. The San Francisco–based group, Breast Cancer Action, has long asserted the need for greater focus on what is actually causing breast cancer, particularly on potential environmental causes. The group views the barriers to the production of such knowledge as political and economic and sees the current classification of high risk as another way to gloss over this major political problem by oversimplifying and obscuring the uncertainty of breast cancer risk, thus legitimizing a pharmaceutical solution to prevention as opposed to one that locates and eliminates fundamental causes of breast cancer. In addition, the risk threshold constructed by the Gail model is seen to maximize the number of eligible consumers of tamoxifen or other chemopreventive drugs in a way that benefits AstraZeneca and other large pharmaceutical companies and does not benefit women's health.

Critiques of the Gail model also emerge from those who use the model and are in most ways advocates of its use. These critiques predominantly stem

from what are perceived to be limitations of the model based primarily on the appropriateness and adequacy of the data originally used to construct the model. Like other such models, the Gail model is based on the state of knowledge at the time it was constructed and is limited by the constraints of the particular research from which it emerged. First, critiques emerge from the fact that 91 percent of the case-control study population from which the Gail model was developed consisted of white women, creating uncertainty and the highly problematic assumption that what is true for white women can be taken to be true for all women. In response to this obvious absence, the model has recently been modified to incorporate some race-specific information, but this is not considered adequate by all users.

Another factor contributing to the perceived limitation of the model is the omission of risk factors today considered significant that were not included in the model. Gail describes biological risk factors that he thinks might be significant but are not included in the model. Certain factors that seemed to increase breast cancer risk such as alcohol consumption and estrogen replacement therapy were not included in the model because there were too few women exposed to these to say with confidence that they contributed to breast cancer incidence. Given recent research on both of these risk factors, it is possible that a model constructed today would include them. This is not to say that such inclusion would make it a better model but to emphasize that a model today would be just as much an artifact of its context and would surely leave out factors that, in the future, we will recognize as risks just as the Gail model did. This is an inevitable problematic of trying to model based on unstable and constantly shifting knowledge and in a particular historical moment in our understanding of risk in general and specific risks in particular.

Dr. Wickerham (2000), the protocol officer on the STAR trial, said of the model during a National Public Radio talk show, *Talk of the Nation/Science Friday*, "The models, like the Gail model, are better than we had, but they're relatively nonspecific for the individual. Far better than we've had in the past. There needs to be more work done. The problem is the information from the Gail model is an outgrowth of a large mammogram study from the seventies that involved a quarter of a million women. It's hard to come up with that much data to allow us to refine it further." Importantly, Dr. Wickerham described the problems involved in extricating ourselves from the entrenchment of the Gail model due to the unlikelihood of coming up with "better" data. Here, the model is constructed as "good enough" by virtue of the absence of new data with which to create something better.

In its own promotional materials of the model, the NCI also points out its limitations. They cite the following contexts in which the Gail model is not

accurate: "It is not accurate for women who are younger than age 20, who have already had a diagnosis of breast cancer or who are known to have genetic alterations in breast cancer susceptibility genes (BRCAI or BRCA2). There is also some doubt about whether women from other countries will have accurate results because the tool is based on U.S. women." Gail echoed this concern in an interview with me (March 1, 2000). In describing some of the limitations of the model, he articulated, "If a woman has just migrated from rural China she probably has a lower risk—a good deal lower than projected by the model. Because there is a well-known migration effect and we don't know what it is." This interests me for a couple of reasons. First, it implies that risk and disease are geographically distributed in unpredictable ways—that political and geographic national boundaries make a difference for health and illness. Indeed, many studies have borne out such theories with regard to the distribution of breast cancer (i.e., Eaton et al. 1994; Pike 1990; Strassmann 1999; Ziegler et al. 1993). Second, and implicated by this first point, claiming geographical disparity suggests that some kind of environmental or lifestyle (diet, patterns of exercise, reproduction) factors play a role in breast cancer incidence, thereby explaining why it varies from place to place. While this is also widely documented in cross-cultural studies of breast cancer incidence (Eaton et al. 1994; Pike 1990; Strassmann 1999; Ziegler et al. 1993), it is interesting that it would be invoked here since these are factors that are decidedly absent from the Gail model.

Critique of the model by users also focuses on the usefulness of the risk threshold 1.7 to unproblematically guide the practice of prescribing tamoxifen (or raloxifene in the context of research). As discussed earlier, the results of the BCPT indicated a benefit of tamoxifen treatment for all women under the age of 50 at a 1.7 percent risk or greater, but the results indicate that for older women, the risks of thromboembolic events and endometrial cancer may in some cases outweigh the benefit of tamoxifen. These findings led Gail and colleagues (1999, 1843) to conclude in their risk/benefit assessment of tamoxifen that "the use of tamoxifen should not be based on a single number, such as a projected 5-year risk of IBC [invasive breast cancer] of 1.66%, but rather should be based on a weighing of the various risks and benefits of tamoxifen. For older women at higher risk of endometrial cancer, stroke, and pulmonary embolism, higher levels of projected 5-year risk of IBC would be needed to justify the use of tamoxifen." In the article from which this quote is drawn, Gail and colleagues provided formulae for assessing the risks of endometrial cancer, stroke, pulmonary embolism, and other risks along with the Gail model breast cancer risk assessment, and they suggested methods for combining these into a risk/benefit profile for individual women considering tamoxifen therapy for breast cancer risk reduction. This explicitly acknowl-

edges the limitations of the Gail model by itself to guide the use of tamoxifen, asserting that, at least for older women, the use of tamoxifen should be guided by assessments of tamoxifen's risks as well. Yet no corresponding simple model has emerged to assess these risks, and thus no devices or advertising campaigns have widely disseminated the idea that it is necessary to take a woman's risk of endometrial cancer, stroke, or pulmonary embolism into consideration along with her Gail model–derived breast cancer risk number.

Critique also emerges in the day-to-day practices of assessing risk via the Gail model for the purposes of enrolling women in the STAR trial. Many of the research coordinators with whom I spoke described concern with the risk threshold 1.7 to unproblematically guide enrollment of women into STAR. One coordinator expressed her opinion that women who join should be at higher risk: that for some women who might be deemed high risk, seeing a doctor and checking her breasts might be a wiser decision than joining STAR: "I think it's a better idea for women that are at higher risk. I think that for women that had a grandmother that had breast cancer that was found at age 75 maybe, you know, go to you doctor more often. Maybe do your own breast checks more often." Another coordinator described her local site as enrolling women who all had risks greater than 1.7: "We've got women that are 6-12 percent. Twelve percent is the highest we've seen. That's the women with LCIS. So we're not having the woman who comes in at 1.7, 1.66, or 1.6 or whatever it is. We don't have any 1.7. All of ours are higher." Some research specialists I spoke to from STAR reported considering the number 1.66 to be a starting point, the minimal requirement for diagnosing high risk, the determination of which will depend on many other factors. In descriptions of everyday practice, individual health care providers, researchers, or women concerned about risk interpret the Gail model's risk assessment in personally meaningful ways while adhering generally to the widely agreed-on classification of high risk as 1.7. Thus, while 1.7 was the threshold for participation in the BCPT, the average risk number of the participants was 3.2 (Chlebowski et al. 1999), and for many of the researchers I have interviewed, "being at least a 2.5" or "a 3.0" is considered the minimum risk number for which they would recommend a woman to participate in the trial. For others, 1.7 seems perfectly reasonable.

Conclusions

The critiques of a high-risk classification system and the Gail model as a tool for classification come from many different places, including those who

rely on the model for their everyday work practices. My informants who work with the Gail model as they enroll patients in STAR openly discussed some of the drawbacks of the tool and the high-risk category it attempts to construct. This demonstrates that while standardization may be occurring, conflict and uncertainty remain present. As Clarke and Casper (1996) argued, standardization may proceed along with interpretive flexibility, a theoretical point that captures the complexity of the standardization of high-risk vis-à-vis the Gail model and helps to explain both the concurrent embeddedness of this model to define high risk for chemoprevention as well as its contestations by various groups and variations in the actual practices of such classification.

Furthermore, my research with various types of users of the model highlights that few expect the Gail model to unproblematically provide a clear mandate for whether an individual woman will decide to take tamoxifen. Instead, there is widespread acknowledgment that the model is a tool to aid in what will be a complex decision-making process. Thus, individual risk reduction decisions are not the kinds of uncertainty that this model is meant to tame. Rather, the model manages the uncertainties associated with legitimacy and ethics of breast cancer risk and risk-reduction strategies. Thus, the job that the Gail model does is to a large extent symbolic: it does not mandate physicians to prescribe tamoxifen to women with a 1.7 risk or higher nor make their (or women's) decisions to do so easy or straightforward; rather, it makes these things legitimate options—a significant achievement for advocates of chemoprevention in an arena (breast cancer prevention) that is steeped in controversy.

The Gail model has traveled in time from its development to the current arena of breast cancer prevention via tamoxifen. The state of the knowledge has changed since its development, and many, including Mitchell Gail, argue that if a risk model were to be developed today, it would look different. But the Gail model has proved itself flexible enough to travel this distance and remains a legitimized tool for determining what counts as knowledge about breast cancer risk. Similarly, this model travels over geographical and bodily boundaries, standardizing women's risks and bodies in different contexts. Such stabilization of knowledge is necessary within the social arena of chemoprevention because it legitimates who is an eligible participant in chemoprevention trials and who can currently be prescribed tamoxifen in the clinical setting. It shapes who pharmaceutical companies can legally and ethically market their products to and what kinds of profits they can expect to make from chemoprevention drugs. Agreement about high risk for breast cancer does legitimation work in an arena of uncertainty and in the context of controversial and risky drugs, and the Gail model is constructed as right for

this job, in large part due to its organizational embeddedness in research, policy, clinical practice, and the marketing of chemoprevention.

Notes

1. For a sociological discussion of "when is cancer," see Clarke and Casper (1996) on classification of Pap smears.

2. By using the data from the placebo arm of the Breast Cancer Prevention Trial (BCPT), researchers were able to conduct a validation study of the model in which it was found to accurately predict risk overall (Costantino et al. 1999). However, because only 1.7 percent of the BCPT participants were women of color, the model has been validated only for white women. In addition, while this study validated the model overall, there were some instances in which it over- or underestimated risk. The model underestimated risk in women with a history of breast cancer, lobular carcinoma in situ, or ductal carcinoma in situ (and is explicitly not recommended for use to estimate risk in such women). It overpredicted risk in young, unscreened women and somewhat underestimated risk for women over the age of 59 (Gail et al. 1999, 1830).

3. In clinical practice, the Gail model is used to assess eligibility for tamoxifen for risk reduction because tamoxifen is currently the only chemoprevention approved by the Food and Drug Administration (FDA) for breast cancer. Raloxifene is used for risk reduction in the context of clinical trials that also use the Gail model–derived 1.7 to define high risk. Presumably, if raloxifene also becomes an FDA-approved breast cancer chemoprevention, it will also use the Gail model to assess eligibility for its use since this will again be based on the results of the clinical trial that used the model.

4. This connection can be found elsewhere in published materials about the development of the risk disk (Benichou 1993).

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