CHAPTER THREE

Genetics and Behavior in the News

Dilemmas of a Rising Paradigm

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In the past two decades genetics has re-entered the public discourse in terms of explanations for a variety of behaviors and human problems (Nelkin and Lindee, 1995; Condit, 1999). I see genetics as a rising paradigm that is increasingly applied to a range of human behavior. One of the main vehicles for the dissemination of genetics findings and claims to the public is the news media.

This chapter examines some issues that stem from the way genetic research on behavior is presented in the news. The data come primarily from a study of how findings related to behavioral genetics (e.g., alcoholism, homosexuality, and depression and schizophrenia) have been reported in five major newspapers and three news magazines, along with other print media, over a

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thirty-year period (1965–95).* The print press can be seen as an exemplar of the news media; television and radio news tend to cover less science news in general and probably less about genetics as well. In addition to examining the actual news articles, I have interviewed fifteen science reporters about their work. Examples from this study are supplemented with data on press coverage of genes for breast cancer to illustrate some ways in which presentations in the news media can shape the public discourse on genetics.

Science in the News

The news media are a critical vehicle for disseminating new scientific findings into the culture. The popular press has increasingly reported on science and health, both as part of general reportage and in special science and health sections (Klaidman, 1991). While the news media report on only a very limited selection of the scientific findings published in professional journals (Houn et al., 1995; Conrad and Weinberg, 1996; Conrad, 1997), this coverage still accounts for a significant amount of information conveyed to the public through the media. Science and health have become important beats for newspapers. The *New York Times*, for example, employs more than a dozen science and health reporters and publishes health and science news every day, frequently more than one article. While there has been little specific research on how this dissemination of scientific knowledge is interpreted by readers, it is clear that the press is one of the most accessible means for transmitting new scientific and medical findings to the lay public.

Science and medical reporters tend to monitor a few of the most prestigious professional journals, which are then overwhelmingly represented in the news: *Science*, *Nature*, *New England Journal of Medicine*, *Journal of the American Medical Association (JAMA)*, *National Academy of Sciences*, *Nature Genetics*, and the *Lancet*. While other journals are occasionally reported, these prestigious journals dominate the medical and science news. For example, in a study of the press coverage of the association between alcohol and breast

*The newspaper sample includes the *New York Times*, the *Boston Globe*, the *Washington Post*, the *Wall Street Journal*, and the *Los Angeles Times*, which represent some of the largest and most influential newspapers in the United States. For homosexuality, the *San Francisco Chronicle* was added. The news magazines included *Time*, *Newsweek*, and *U.S. News and World Report*. Representatives of the gay press were largely drawn from the same cities as those of the mainstream press and included the *New York Native*, *Bay Windows*, the *Washington Blade*, the *San Francisco Sentinel*, and the *Bay Area Reporter* (see Conrad and Markens, 2001).

cancer over a thirteen-year period, 88 percent of the news stories came from studies reported in *JAMA* or the *New England Journal of Medicine* (Houn et al., 1995). Reporters believe this handful of journals usually publish the most consequential scientific research and presume the scientific findings are more likely to be of high quality because of the journals' reputation for rigorous peer review. While science reporters recognize that important research is also published elsewhere, it simply does not usually come across their radar screen. This narrow focus suggests that science reporting presents a very selective slice of biomedical research.

News science writing is not a straightforward process of "reporting" the facts of a study garnished with a few quotations from the researchers or other scientists. Science reporters, like other journalists, select, shape, and frame stories into news. Science and medical stories are difficult to present accurately and in an accessible manner. Journalists often need to convert complex and ambiguous scientific findings into nontechnical, compelling, and readable stories. This requires reporters to simplify findings and to present them in ways that are comprehensible to a lay readership. A few studies have examined how medical science has been presented in the media. Dorothy Nelkin (1987) argues that science reporting tends to be uncritical and engages largely in "selling science" to the public. Medical and science newswriting often frames stories as "a new breakthrough in medicine" or depicts the newsworthiness of a story as "scientists report for the first time." There is some evidence that only studies with positive findings become news and that there is a bias against negative studies (Koren and Klein, 1991). Negative studies and disconfirmations of previous findings are a crucial part of the development of scientific knowledge and important to obtaining accurate scientific understanding, but they are less likely to be reported in the news.

The New Genetics

Genetics is becoming an increasingly dominant explanation in medicine and science. People have long noted that some disorders ran in families, and scientists have proffered hereditary theories of various diseases, conditions, and behaviors. Eugenics was popular among scientists and lay people alike through the early part of this century (Kevles, 1985) but fell into disfavor owing to the paucity of valid scientific evidence and the horrors of Nazi genocide. With the study of DNA's structure, a new genetics has emerged in recent

decades. Along with producing some remarkable discoveries, the new genetics has also engendered fresh concerns about legal and ethical issues (Kevles and Hood, 1992; Kitcher, 1996)

In recent decades, molecular biology and medical genetics have moved to the cutting edge of science. Important new discoveries involving genes for cystic fibrosis, Huntington disease, fragile X syndrome, Duchenne muscular dystrophy, and types of breast and colon cancer, among others, have been reported. Many of these received widespread notice in the news media.

The advent of the Human Genome Project in 1989, the largest biological project in history (Kevles, 1992), has further fueled genetic research. The genome project is a fifteen-year international research initiative with the aim of sequencing all 3 billion base pairs of the human genetic structure. The project is proceeding ahead of schedule, and a "rough draft" of the genome sequencing was presented in 2000. This information will be used to find the chemical or genetic basis for the two thousand or so genetic diseases that affect humans, with the ultimate hope of producing new preventions and cures. The genome map will allow genetic research to progress more rapidly; this undoubtedly means that new claims about genetic associations and linkages with diseases, conditions, and behaviors will be increasingly forthcoming over the next decade.

The mapping of the human genome has been the object of several provocative metaphors, including a search for the "holy grail" (Gilbert, 1992), investigating the "the essence of human life," and decoding "the book of life." The genome project itself has been called biology's equivalent to the World War II Manhattan Project. Others have suggested that the gene is becoming a cultural icon in American society (Nelkin and Lindee, 1995), invested with almost mystical powers. Critics contend that the "geneticization" of human problems has expanded beyond scientific knowledge (Lippman, 1992) and that a kind of "genetic fatalism"—an assumption that a genetic association is deterministic and means a trait or behavior is unchangeable—underlies much public discourse about genetics (Alper and Beckwith, 1995). What is clear is that genetic research is relevant to an increasing number of diseases, conditions, and behaviors and that the genetic "frame" is commonly employed for explaining a wider range of human problems.

Simply put, an increasing amount of genetic research is being reported in the news (Nelkin and Lindee, 1995; Conrad, 1997; Van Dijck, 1998; Condit, 1999). In 1996, for example, news articles reported research associating genetics

with breast and colon cancer, diabetes, Alzheimer disease, homosexuality, "novelty-seeking" personality traits, bed-wetting, and obesity, among numerous other characteristics and conditions. Often these stories are highlighted on the front page of newspapers (most of those mentioned above were) because genetics seems to make good news. Stories about new findings are legion, so much so that it sometimes appears as if we're seeing announcements of "gene of the week" discoveries.

While ethicists and others have raised issues concerning the potential harm of genetic screening and its impact on decision making, the privacy and confidentiality of genetic information, the prospect of genetic discrimination, the dangers of coercion, and the revival of eugenics (e.g., Bartels et al., 1993), less attention has been paid to the effect of the public's understanding of genetics and its implications.

In this chapter, I will briefly examine two science reporting issues that have social and bioethical implications, what I call "finding and losing genes" and "the OGOD assumption" (one gene, one disease) (see Conrad, 1999). For each issue I will present two cases of genetic news presentations in some detail, to illustrate the specifics of reporting and different facets of each problem, followed by a discussion of some implications of these presentations for the public understanding of genetics.

Finding and Losing Genes

Articles reporting significant new genetic research are typically reported in prominent places in newspapers or magazines. Yet if subsequent research does not replicate the findings or disconfirms the first study's results, how does the news media cover the dissenting studies? The way the media reports on such subsequent studies contributes to an information flow problem in public discourse.

The Old Order Amish and a Gene for Manic Depression

In February 1987 major newspapers featured a front-page story reporting the discovery of a marker linked to a gene for manic-depressive illness. Janice Egeland and her colleagues (Kelsoe et al., 1989) had identified the genetic marker based on research involving extended families with a high incidence of the disorder among the Old Order Amish of Pennsylvania, a group with a stable and closed gene pool who keep good genealogical records. A genetic

marker is a specific genetic pattern (or difference) found in individuals with a certain disorder; researchers then assume the problematic gene is in the general vicinity of the marker. While the actual gene linked to manic-depressive disorder had not been located, the research indicated it was a dominant gene (i.e., it could be inherited from either parent) and pointed to its location (on the tip of the short arm of chromosome 11). The *New York Times*' headline was "Defective Gene Tied to Form of Manic Depressive Illness." The news story announced a breakthrough finding: "Scientists have discovered the first proof that some cases of manic-depressive illness are linked to a specific gene defect" (*New York Times*, February 26, 1987, p. 1).

A month later newspapers reported on an Israeli study of three large Jewish families that linked another genetic marker (this time on the X chromosome) with the development of manic-depressive illness. The *New York Times* headline announced, "Second Genetic Defect Linked to Illness: Manic-Depressive Disorders Traced to Faulty Gene on X Chromosome" (*New York Times*, March 19, 1987, p. A20).

Estimates suggest that 2 million Americans have manic-depressive illness. It has long been observed that the disorder has a propensity to run in families, and thus it was thought to be at least partly hereditary. The news articles sometimes offered new hope to people with the disorder and their families. "Once the faulty gene is identified," Dr. Risch, one of the study's authors added, "physicians should be able to help guide high-risk people in developing a strategy for a satisfactory way of life. It might also help in devising a treatment that would minimize the risk of manic-depressive attacks, he said" (ibid.).

Two years later the Amish study was disconfirmed. In November 1989, scientists, including some from the original research team, published a paper reporting that continuing research among the Amish led them to conclude that the genetic marker probably was not significant in identifying manic-depressive illness. In one particularly important change, two subjects from the original study who didn't have the chromosome 11 pattern and had shown no signs of mental disorder subsequently developed the illness. With the small sample size of these family pedigree studies, this shift invalidated the statistical significance of the study (Kelsoe et al., 1989).

To its credit, the *New York Times* reported this disconfirmation in the medical science section with the headline "Scientists Now Doubt They Found Faulty Gene Linked to Mental Illness" (November 7, 1989, p. C20). Several of

the other newspapers did not report the new disconfirming evidence at all or only mentioned it years later as part of another article. The *Times* story outlined how the new study "cast serious doubt on the conclusions of [the first] study that linked a faulty gene to manic-depressive illness." It discussed the difficulty of assigning specific causes to complex and variable illnesses. The news story ended by noting that the Israeli study linking a suspected gene on the X-chromosome to manic-depressive illness "still seems to be unchallenged." In 1993, however, a research paper reported that after more exhaustive analysis of the Israeli data the X-chromosome link could not be confirmed. The *Times* reported this in the health and medicine section with the headline "Scientists Now Say They Can't Find a Gene for Manic-Depressive Illness," while three of the four other newspapers in our sample never reported the disconfirmation. The *Times* story described how, in the wake of this disconfirmation and an additional one involving a gene on chromosome 5 thought linked to schizophrenia, scientists now believed that there was no single gene for mental disorders. Rather, it was likely that the interplay of a number of genes, in interaction with an individual's environment, might produce a disorder. Again, the article ended on an upbeat note for genetics by quoting one of the study's authors, "Nevertheless, Dr. Baron remains optimistic that scientists will tease out the genes that go afoul in mental disease, if only they are meticulous in their hunt" (January 13, 1993, p. D12).

Alcoholism and the D2 Dopamine Receptor

A similar situation occurred with alcoholism and the dopamine D2 receptor gene. In April 1990 the *Journal of the American Medical Association* (*JAMA*) published an article by Kenneth Blum, Ernest P. Noble, and associates that reported, for the first time, an allelic (specific-gene) association with alcoholism. Based on cadaver brain research with samples from alcoholics and nonalcoholics, the authors found that "the presence of the A1 allele of the dopamine D2 receptor (DRD2) gene correctly classified 77% of alcoholics, and its absence classified 72% of nonalcoholics." The authors concluded they had found a marker for a specific gene in a specific location (q22-q23 region of chromosome 11) that "confers susceptibility to at least one form of alcoholism" (Blum et al., 1990: 2055).

This study was widely reported in the news. All five major newspapers in my study sample reported the Blum-Noble findings on April 18, and all three news magazines reported it in the April 30 issue. The *New York Times* and the *Boston Globe* printed stories on page one, and the other papers ran them in prominent locations like page three. Headlines announced "Alcoholism Is Linked to a Gene" (*Wall Street Journal*) and "Scientists Link Alcoholism to Gene Defect" (*Boston Globe*). The newspapers reported it as a major breakthrough. The *New York Times*, for example, wrote: "A gene that puts people at risk for becoming alcoholics has been identified for the first time." The newspaper stories were optimistic, stating that this discovery strengthens the growing conviction that heredity plays a key role in alcoholism and that new drugs for alcoholism treatment could eventually emerge. The news magazines all reported the study but were somewhat more temperate with their optimism. While the news media did note some reservations about the study (e.g., its sample size and the unclear role of environment), the tenor of the reporting presented the discovery of a gene linked to alcoholism as a major breakthrough for medicine.

Eight months after publishing the Blum-Noble finding, *JAMA* published a study that essentially found no significant genetic differences between alcoholics and controls (Bolos et al., 1990): the DRD2 gene for alcoholism was not confirmed. This inability to replicate the findings received some attention from the press but much less than the Blum-Noble study. While four of the five newspapers in our study carried some report of the nonreplication, none of the news magazines did. The dissenting reports were also considerably shorter than those for the original study and were located in less prominent places (e.g., p. 20, p. 59). Typical headlines were "Researchers Cannot Confirm Link to Alcoholism" (*New York Times*) and "A Disputed Study of Alcoholics Finds No Genetic Pattern" (*Boston Globe*). While there was some reporting of the disconfirming study, often the stories were written in a manner that still affirmed a genetic basis for alcoholism. Both the *Times* and the *Boston Globe*, for example, quoted Blum as saying the gene was still relevant and that other studies supported their claim. An overall reading of the news coverage of these and related studies suggests the news media continued to emphasize a genetic link for alcoholism in their reporting, despite a prominent failure to replicate. The news magazines did not report the dissenting studies at all. (See Conrad and Weinberg, 1996.)

These two cases indicate that science reporting on genetics maintains a bias toward positive reports, those suggesting genetic associations with behaviors or diseases. This is not surprising, given that others have reported that the media has a bias against negative studies (Koren and Klein, 1991). In addition, genetic discoveries may pass for good news among all the bad news like crimes

and routine news like business and politics. But while the discoveries are trumpeted to the public with front-page stories, the dissenting or disconfirming studies are noted (if at all) in smaller back-page items. This kind of reporting is likely to convey the impression that this or that gene has been found, but is unlikely to correct that view once they are lost.

This of course is the logic of news: finding something new is news, but not finding it may not be. Nevertheless, science is always provisional. Disconfirmations and modifications are to be expected as part of the scientific process, even if such complexities don't always make good news. Moreover, news writing often has to simplify to the extent that many of the scientific caveats or qualifications may be lost. And, it seems, the press considers discoveries important news, while disconfirmations or failures to replicate are treated as inherently less important or uninteresting (or perhaps the media simply believe the public takes that view). This parallels crime reporting: Arrests or indictments for a crime may be front-page news, but dismissal of charges or an acquittal get far less attention and my be reported in back pages, if at all. Putative discoveries are news; their negation rarely is.

The OGOD Assumption

News media use catch phrases to signify complex phenomena, from *Whitewater* to *Generation X* to the *tobacco wars*. This is especially evident in headlines, but it permeates reporting as well. Genetic influence is complex, multifaceted, and indirect, yet it is often presented in the news as if a single gene is responsible for a behavior or condition, be it a gay gene, a breast cancer gene, or a fat gene (Conrad, 1999). We might call this the OGOD model—for "one gene, one disease." The assumption here, of course, is that a single gene determines the disease or trait.

BRCA1 as the Breast Cancer Gene

Breast cancer is the most common cancer for women. It is usually estimated that one in eight women (12%) will contract it, and that forty-six thousand women in the United States die from it each year. In late 1990 Mary-Claire King of the University of California, Berkeley, announced that her lab had identified a marker for some forms of breast cancer somewhere on the lower end of chromosome 17. This discovery was based on research with families that had

multiple cases of breast (and ovarian) cancer (Hull et al., 1990). The genetic susceptibility for women in families prone to breast cancer is estimated to be strikingly high—perhaps ten to twenty times higher than for women without the susceptibility.

With this genetic signpost, a number of labs worldwide raced to identify the actual gene, now dubbed BRCA1. In the fall of 1994 Mark Skolnick and his colleagues at the University of Utah reported in *Science* that they had isolated BRCA1 (Miki et al., 1994). In a very unusual course of events, an announcement of the discovery was released three weeks before the publication date of the scientific journal article, in large part due to an *NBC News* report and persistent rumors of the impending discovery. On September 15, all major newspapers carried the story, most of them on the front page. Headlines announced: "Gene for Inherited Form of Breast Cancer Is Located" (*Washington Post*); "Scientists Identify a Mutant Gene Tied to Hereditary Breast Cancer" (*New York Times*); and "Scientists Say They've Found Gene That Causes Breast Cancer" (*Wall Street Journal*). The news stories were quite detailed, describing the BRCA1 gene as a tumor-suppressing gene and circumscribing indications that this gene was implicated in only about half of all hereditary incidence and perhaps in only 5 percent of the total incidence of breast cancer. All women have the BRCA1 gene, but only a small percentage seem to have the mutated version that makes them more susceptible to breast cancer. For the women with the BRCA1 mutation, researchers claimed there was an 80 percent likelihood of getting breast cancer by age 70 and a 50 percent chance of developing it by age 50. Natalie Angier of the *New York Times* pointed out that roughly six hundred thousand women carry the genetic defect.

This was unquestionably a major discovery, depicted by some scientists and reporters as a window to understanding all breast cancer. Early reports following the BRCA1 discovery implicated the gene only with inherited forms of breast cancer in highly susceptible families, that is, a defective BRCA1 gene was deemed to cause only a small proportion of all breast cancer. Yet if one examines the press coverage of this and subsequent findings, BRCA1 is frequently referred to as "the breast cancer gene." Taking headlines as an indicator, we see many designations of it as such, some beginning even before its actual discovery; for example: "The Breast Cancer Gene: A Women's Dilemma" (*Time*, Janyary 17, 1994, p.1); "Vexing Pursuit of Breast Cancer Gene" (*New York Times*,

July 12, 1994, p. C1); "Breast Cancer Gene's Impact Limited" (*Washington Post*, September 20, 1994, p. 7); and "Return of the Breast Cancer Gene" (*Newsweek*, November 13, 1995, p. 72).

It is important to note that while BRCA1 and the subsequently discovered BRCA2 (on chromosome 13) are genes linked to breast cancer, they account for only a small percentage of all cases. The BRCA1 defect is present in roughly 5 percent of breast cancer cases, hardly sufficient to be deemed "the breast cancer gene." Research subsequent to the original BRCA1 report has suggested that a BRCA1 mutation may be more prevalent among Ashkenazi Jews (as high as 1 in 100 compared to perhaps 1 in 800 among the general population) (Struewing et al., 1995), and BRCA1 mutations have been found in some noninherited tumors. Yet even with these additional studies it is probably most appropriate to call the mutated BRCA1 a gene that indicates a specific, very high-risk breast cancer among families with a history of breast and ovarian cancer. It is important to keep in mind that an overwhelming proportion of breast cancer, at least at this point, appears to be noninherited. This is not to say that future scientific findings could not implicate BRCA1 or other mutations in a wider range of breast cancer causation, but current evidence does not warrant designating it the breast cancer gene.

Why should this apparent journalistic shorthand matter? In the most general sense, calling BRCA1 the breast cancer gene (quotes are never used here) suggests that inherited genes are the main cause of breast cancer, which is not the case. While the environment as a factor in breast cancer is discussed in both the scientific and news media articles, focus on the breast cancer gene turns our attention away from environmental sources to germ-line DNA.

Focusing on breast cancer genes raises a number of issues. First, calling a disease agent the breast cancer gene may lead women, understandably anxious about the disease, to request tests for the gene from their physicians. It is not clear what, if any, relevance BRCA1 mutations at this time has for women without family histories of breast cancer. Thus far the gene's explanatory power is limited to high-risk families. Yet many other women will want to know whether they are free of the gene defect. Would a negative test lull women into a false sense of security about their risk for breast cancer, perhaps leading them to neglect breast self-exams or appropriate mammograms? Second, testing for BRCA1 is an imperfect predictor. Some women in high-risk families who test positive for the gene defect may choose to undergo "prophylactic bilateral mastectomies," a radical operation removing both breasts,

which, while reducing risk, still does not guarantee protection from breast cancer. Likewise, those who test negatively, while justifiably relieved, still carry at least an average risk for breast cancer, a one in eight lifetime prevalence. Third, even for high-risk families, locating the gene does not necessarily allow prediction of the course of the disease or lead directly to treatment. The gene for Huntington's disease, which was discovered several years ago, has had little clinical impact on that disorder so far.

In the near term, given the potential genetic testing market, we are beginning to see that commercial products allowing for the easy testing for BRCA genes will become readily available (see Kolata, 1996). While virtually all the news articles noted that the discovery of the gene would have no immediate impact on treatment or even on prevention and detection, some were more optimistic about the implications. One reporter, for example, suggested that the discovery "sets the stage for a blood test, probably in a year or two, that would identify women who would benefit from intensive monitoring and preventive treatments" (Saltus, 1994, p. 1). Another science writer was much more cautious, however. "Even when a test goes on the market, only a fraction of the women who believe they are members of families high for breast or ovarian cancer should consider it" (Brownlee and Watson, 1994, p. 78). This writer suggests that only women with two or more first-degree relatives (mother, daughter, sister) with breast or ovarian cancer, or those with multiple family members stricken with breast cancer before age 40, should be tested when such a test becomes available.

More than a year after the discovery, Francis Collins, director of the Center for Human Genome Research and himself a genetic researcher studying breast cancer, suggested that for a variety of scientific, ethical, and social reasons, BRCA1 testing should only be done in the context of a research setting (Collins, 1996). If the *breast cancer gene* becomes part of the common parlance, then many more people beyond those in high-risk families may want to be tested. Even putting aside the potential cost of unnecessary testing, what impact will a negative BRCA test have on women in a society in which breast cancer risk is still so high?

It may someday turn out that BRCA1, BRCA2, and other genes yet to be discovered are parts of a collection of gene mutations that cause breast cancer. It is also possible that scientists will discover that genes and the environment interact in subtle ways to produce breast cancer or that some breast cancers are fundamentally environmental. As important as the discovery of the BRCA1

gene is, journalists and scientists should avoid simplistic designations that misinform and potentially misguide people about the risk of breast cancer.

"Gay Genes"

Scientists and physicians have offered theories about a hereditary predisposition toward, or a congenital nature of, homosexuality for more than a century (Greenberg, 1988). In July 1993 Dean Hamer, a neurogeneticist at the National Institutes of Health, published an article in *Science* reporting the discovery of a genetic marker associated with homosexuality. Hamer and his colleagues traced the family pedigrees of seventy-six homosexual men and found that 13.5 percent of brothers, 7.3 percent of maternal uncles, and 7.7 percent of maternal cousins were homosexual. They then conducted a DNA linkage analysis on forty pairs of gay brothers and found that thirty-three pairs shared genetic markers on the Xq28 region of the X chromosome. Hamer and his colleagues concluded that "at least one subtype of male sexual orientation is genetically influenced" and inherited through the maternal line (Hamer et al., 1993, p. 321).

Our research examined how five studies linking biology and homosexuality, published between 1990 and 1993, were reported in the mainstream and gay press (see Conrad and Markens, 2001). Most of these studies received substantial media attention, although the discussion here will be limited to Hamer's research because it is the only study to claim to have located a specific genetic marker.

Hamer's research was widely reported. It was on the front page of five of the six newspapers we studied (we added the *San Francisco Chronicle* to our sample for reporting on homosexuality; the *Wall Street Journal* ran it on page B1). Three of the papers ran two stories on the study; typically, one reporting the findings and a second focusing on reactions or potential implications. All three news magazines covered the story, including *Newsweek*, which featured a nine-page cover story.

The lead in the *Washington Post* (July 16, 1993, p. 1) story was typical: "Scientists at the National Institutes of Health have discovered evidence that some gay men have inherited one or more genes that predisposed them to being homosexual." This was usually followed by a more detailed description of the study and its findings. But for the first time, several articles began to use the term *gay gene* to describe the findings (even though Hamer explicitly noted that it is unlikely a single gene is responsible for homosexuality). For example,

in three of the headlines, the term *gay gene* was used: "Research Points to Gay Gene" (*Wall Street Journal*); "New Evidence of 'Gay Gene' in Some Men" (*San Francisco Chronicle*); and "Coming to Grips with Finding the Gay Gene" (*San Francisco Chronicle*). In 1993 the *San Francisco Chronicle* was the only paper to use the term in both the headline and the article.

The gay press, at least as represented by the six gay newspapers we examined, also reported Hamer's study. They gave this study more coverage than they had the previous studies but relatively less attention than the mainstream press had given it. Only one editorial used the term *gay gene*, although other headlines implicitly referred to gay genes: "It's in the Genes" or "Gays with Designer Genes."

But within two years the term *gay gene* had become more common. Hamer and a journalist coauthored a book about the research leading to the discovery of the Xq28 association with homosexuality, the subtitle of which was "The Search for the Gay Gene and the Biology of Behavior," and the term *gay gene* was used frequently in the book (Hamer and Copeland, 1995). When Hamer published a second study in 1995 (Hu, et al., 1995), refining and replicating his research with a different sample, the news media again reported it. By then the term *gay gene* was common currency; headlines included "Search for a Gay Gene" (*Time*, June 12, 1995, p. 60); "New Evidence of a 'Gay Gene'" (*Time*, November 13, 1995); "Is There a 'Gay Gene'?" (*U.S. News and World Report*, November 13, 1995); "In Search of the Gay Gene" (*Advocate*, December 26, 1995, cover); and "Study Provides New Evidence of 'Gay Gene'" (*Washington Post*, October 31, 1995, p. 1). Even a news item in *Science* had the headline "NIH's 'Gay Gene' Study Questioned" (June 30, 1995, p. 1841). The writers also used the term *gay gene* much more frequently in the articles. David Miller (1995) notes it is regularly used by the press in the United Kingdom as well.

There are some stylistic issues here worth noting. On the one hand, it simply may be a journalistic shortcut to use the term *gay gene* instead of saying *marker for a gene for homosexuality*—the former certainly has a more catchy sound to it. At the same time, the use of the term *gay gene* may indicate an increasing acceptance of the existence of genetic causes of homosexuality. Recall that both of Hamer's studies located only a section of the X chromosome (Xq28) that has a marker associated with homosexuality. At best, the researchers have discovered the approximate location where a "gay gene" might reside. It also makes a difference whether the term *gay gene* is set off in quotes. Placing it in quotation marks flags the point that it is merely a term

that is in use (like saying "so-called gay gene"). Without quotes, use of the term suggests that there is such an entity—even though no such gene has yet been isolated.

It is also important to note that some writers designate it *a* gay gene, while others call it *the* gay gene. While neither is scientifically accurate, the latter is more problematic. Hamer's studies are based on researching a very specialized sample of homosexual men: gay men who have brothers who are also gay, both of whom are willing to go public about it. Neither Hamer nor any other researcher claims that this or any other gene is likely to be *the* cause of homosexuality, yet calling it *the* gay gene suggests that it is the cause. Not all gay men have the marker on the Xq28 region (most probably don't), so clearly there are other causes, be they environmental, psychological, or genetic (caused by other genes). And there is no evidence that whatever gene resides on Xq28 is deterministic; is it still "the gay gene" if there are individuals who have it and are not homosexual? At the very least, it is misleading to call this marker the gay gene. The more the news media uses such terms, the wider the dissemination of the image of homosexuality as a genetically driven phenomenon, with all this implies.

One other point should be mentioned here. Scientists are not neutral figures in relation to the media. Some scientists have become very media savvy and know how to promote their research and ideas in the news. Dean Hamer is both a scientist and an entrepreneur for the idea that homosexuality has a genetic basis. He has appeared on radio, on television, and in documentaries advocating this idea and has written two books promoting a genetic view of human behavior (Hamer and Copeland, 1995, 1998). While Hamer's popular writing may be an extreme example of a scientist attempting to promote a viewpoint, scientists frequently use the media to infuse their ideas or views into the public sphere. The optimism about genetics in the news is not always a creation of the media; it is also a reflection of the scientists. (To cite another example, Ernest Noble has been a steadfast supporter of the genetic approach to alcoholism, even in the wake of critical and disconfirming research.)

There is a difference of opinion in the gay community about whether finding genetic links to homosexuality is good, bad, or irrelevant for gays. Some suggest that linking homosexuality to genes shows that homosexual orientation is "natural" and implies that gays shouldn't been blamed, stigmatized, or discriminated against for an orientation that arises from their genetic makeup. Individuals who take this view argue that if the public understood a gay orientation to be an innate characteristic, like skin color, then our society would realize that gays deserved legal protection against discrimination. The t-shirt that proclaims "Gay by Nature, Proud by Choice" represents this viewpoint. In contrast, there are people in the gay community who are less sanguine about the finding. They are concerned that finding a gene for homosexuality would lead to a remedicalization of homosexuality. Possible "treatments" for the "defect," such as testing fetuses and aborting those with the implicated genes, or possibly even "eugenic" interventions (e.g., as depicted in the play *Twilight of the Golds*) might follow. Others have suggested that it is not sensible to base rights on biology, and thus finding genes for homosexuality is irrelevant to justice (Nardi, 1992).

While Hamer replicated his own study with a different sample, George Ebers and his associates (Rice et al., 1999) were unable to replicate Hamer's findings and concluded that the Xq28 marker is not proven to be linked to male homosexuality. While it is not in our realm to adjudicate the science, current claims about "the gay gene" are best seen as contentious. At this moment, "the gay gene" is more a social construction than a biological reality. Nevertheless, its designation in the public sphere may be having an impact on how we think about homosexual orientation and how we treat people who are gay.

In the next section I outline some broader ramifications of the presentation of genetic findings in the news.

Implications

Embedded in the ways genetics is reported in the news are an array of implications for the public understanding of genetics. This chapter has only begun to articulate these, but it seems clear that the way problems are constructed in the culture and the common knowledge around them affect people's perceptions and what they do about those problems. We can identify a number of specific issues.

1. *Genetic optimism and raising public expectations.* Genetic associations with human behavior are regularly reported prominently in the press (often on page one). The tone of this reporting reflects a genetic optimism, which comprises beliefs that genes are causally related to human behavior, that scientists will discover these relationships, and that these discoveries will lead to treatments or the reduction of suffering (see also Conrad, 2000). Reporters

covering studies on genetic molecular biological associations try to be upbeat and leave the reader with the idea that the discovery of gene linkages is "good news." This stance is manifested in the ways the stories are written, and, specifically, in cases of disconfirmation or nonreplication, the way the last word emphasizes that better science or more research will eventually find the genes.

Genetic optimism reinforces the "magic bullet" conception of medicine—that, if we just identify the right gene for an ailment, science will come up with a specific biomedical treatment for it. Demands for genetic tests may increase, although their medical purpose remains questionable. As the discoveries of genetic linkages to Alzheimer or Huntington disease have shown, treatments don't necessarily follow from gene identification. This type of genetic optimism, even when tempered by discussions about social implications (e.g., stigma or the potential loss of insurance) or by the knowledge that genetic tests or treatments are not yet available, pervades the news reporting of genetics. Despite these caveats, the tone of the reporting is likely to raise the public's expectation of what will come from genetic discoveries.

2. *Disconfirmations and cultural residues.* Scientific findings are provisional and always subject to modification; failure to replicate results can lead to new understanding. The two examples of disconfirmation or nonreplication presented (and to these one could add the discovery and disconfirmation of a gene for schizophrenia on chromosome 5 and several others) raise several issues around science reporting. Discoveries of genetic associations for common human problems are deemed important enough to become front-page news and are presented in a positive light. Yet if subsequent research disconfirms the original study the press allocates far less space and attention to the contradicting findings, often completely ignoring them. Thus genetic discoveries receive wide play and are infused into the culture, but if they are disconfirmed fewer people know about it. This creates a progenetic bias in the news and ultimately becomes a vehicle for misinformation. The public is left with mistaken ideas about the relationship of genes to problems, and patients or families may become frustrated when the medical profession cannot deliver on the media's promise.

The press's neglect of disconfirmations can produce errant cultural residues, obsolete ideas that remain part of public knowledge. The news media are the major avenue by which information about genetics enters the culture. Without proper reporting of disconfirmations, outdated or false ideas can become fossilized in the culture. For example, in the late 1960s the news media widely reported the putative relationship between the XYY chromosome and criminal behavior (Green, 1985). This connection was disconfirmed in the 1970s. Yet when asked if they knew about XYY, some of my undergraduate students, who had not even been born at the time of this controversy, replied, "Isn't that the criminal gene?" Without clear and proper reporting of disconfirmations, genetic information (correct or incorrect) will linger and misinform those who encounter it. This may play a part in decision making for medical services as well as shaping how society (e.g., employers or insurance companies) respond to the particular disorders. Moreover, the fact that "successes" become news and "failures" do not reinforces the idea that genetics can explain more of the world.

3. *The OGOD model and the overgeneticization of human problems.* When news reporters use terminological short cuts such as *gay gene, breast cancer gene, obesity gene,* or *thrill-seeking gene,* it oversimplifies complex issues. These simplified terms are especially common in headlines, but they appear frequently in the news stories as well. They overgeneticize the issues they describe: genes are depicted as if they were the most significant factor causing a phenomenon, when they may be only a piece. Such terms convey the notion that single genes can cause diseases, which may be appropriate for cystic fibrosis or Huntington disease (see also Chapter 1 in this volume; Conrad, 1999), but this single-gene model does not suit most behaviors or even diseases like cancer. Calling a discovery "the breast cancer gene" suggests it is at least involved with most breast cancer, when it actually is linked to only 5 percent of the incidence. In other cases, such as homosexuality or alcoholism, no "gene" was ever isolated; rather, researchers only identified genetic markers that suggest genes involved are probably located nearby. Strictly speaking, there is no "gay gene" at this time.

In short, by terming a finding "the" gene, news media give the impression that genetics is the primary cause of the phenomenon, which may not be the case. It privileges genetics in public discourse and reinforces ideas of "genetic essentialism" (Nelkin and Lindee, 1995), the idea that human beings are fundamentally products of their DNA. In its extreme, this can become a type of "genetic fatalism," whereby genetic associations to behaviors or conditions are pessimistically deemed to be deterministic and unchangeable (Alper and Beckwith, 1993), an unintended and unarticulated result of geneticization.

4. *Geneticization and the shift to individual responsibility.* When genes are

implicated in behavior, conditions, or diseases, we often see a shift in responsibility. Behaviors or traits can be attributed to the individual's DNA, with certain consequences. On the one hand, many in the gay community believe that if genetics are associated with sexual orientation, then they will not be "blamed" for their difference and laws will be passed to protect them from discrimination (see Chapter 9). Assuming this is a valid argument—and there are those who would question it—what might this same notion mean for individuals and families suffering from alcoholism? On the other hand, when we deem breast cancer a "genetic disease," we shift responsibility for cause and prevention. If genetics is seen as the first cause, then we minimize attention to environmental factors that are associated with breast cancer and invest fewer resources in identifying environmental contributions to the disease. If the idea of breast cancer as genetic disease gains common currency, it may affect whether women conduct self breast exams and seek out mammograms. And, as mentioned earlier, even in the cases where a BRCA1 mutation is identified, it is not clear what steps women with the gene can take to prevent the onset of the disease. Is it not premature and problematic to present breast cancer as if it were predominantly a genetic disease?

This shift in responsibility from society to the individual aligns with our current political climate, which increasingly blames individuals rather than social conditions for human problems. Thus genetics could become part of an ideological shift away from environmental and social analyses of problems, fostering the decline of public responsibility for human misfortune and misery. To the extent that news reporting privileges genetics in its coverage, the media contribute to this shift in the public eye.

Conclusion

The media's coverage of genetics—whatever excellence is achieved in terms of information, technical accuracy, and clarity of presentation—manifests journalistic conventions that ultimately misrepresent the role of genetics in human behavior. Not to imply that they necessarily treat genetics differently than they treat other scientific and medical topics—while that may very well be the case, I have no comparative evidence to substantiate such a claim. Rather, I do contend that because the media function the way they do, and because genetic information has particular potential impact, the implications of genetics news raise significant social concerns. News conventions, such as those

leading writers to ignore disconfirmations and depict discoveries of "the" gene, compromise the accuracy of science reporting. Perhaps inadvertently, the media reinforce simplistic notions of how genes affect behavior or contribute to diseases. These may affect public attitudes and engender new cultural myths about the role of genetics in explaining and addressing human problems.

In recent years we have observed at least one promising change in the reporting of genetics. Reporters, perhaps chastened by the prospect of disconfirmations and failures to replicate, now frequently mention that though a finding may be interesting and provocative, it still must be replicated by other studies. By the time Hamer's first study was published (1993), all the articles pointed out that these findings needed to be confirmed, and at least four specifically noted that scientific reports of genes for schizophrenia, manic-depression, and alcoholism were later discredited (*Los Angeles Times, Boston Globe, New York Times, Washington Post*). This small change at least informs the public about the provisional nature of scientific findings and the need to be alert to replications and disconfirmations. But it will also be important for the media to report on further research, as important claims that have been widely reported are modified.

All of these examples raise issues about how genetics has been "packaged" in the media and how it affects what people think about particular problems. Media presentations of genetics are the raw materials by which individuals and communities create their own understandings of the role of genes in disease and behavior. While communities select and interpret from various presentations of cultural information, news depictions are a key source for shaping public understanding. To the degree that this source utilizes conventions that misrepresent genetic reality, community perceptions will reflect a partial and inevitably biased perspective of the role of genes in life's problems.

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