

**MAKING SENSE OF RACE IN MEDICINE:
Genetic versus Social Explanations for Breast Cancer Disparities**

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This paper is dedicated to my mother, Dr. Megala Shanmuganathan Kilakkathi. I hope that my work will honor her memory and preserve her legacy.

INTRODUCTION: Ethnicity as Destiny?

If a picture is worth a thousand words, then the cover of *Time* magazine's October 15, 2007 issue speaks volumes about race and medicine. The image is of a naked white woman with a map of the world painted over her body. Above her blazes the headline "The Changing Face of Breast Cancer." This magazine cover illustrates (in the most literal sense of the word) that breast cancer has become racialized disease – in other words, it has become associated with certain racial groups. The editors deliberately chose to use a white woman to represent the original "face" of breast cancer. According to the accompanying article, this is because breast cancer was previously associated with white, affluent women in North America and Western Europe.¹ However, the author, Kathleen Kingsbury, writes that this might not be the case in the near future. She describes how breast cancer is rapidly becoming a worldwide problem: "Asia, Africa, Eastern Europe and Latin America have all seen their caseloads spike. By 2020, 70% of all breast-cancer cases worldwide will be in developing countries."² Based on these statistics, the "face" and race of breast cancer do, in fact, seem to be changing.

Several articles from the medical literature lend further support to this hypothesis. Five studies document two breast cancer disparities with regard to race: African-American women have a lower incidence of breast cancer than white women, but they also have a lower survival rate.³⁻⁷ Even more alarming, two of these five studies also report that although the breast cancer mortality rate for both white and black women is declining, this rate of decline is lower for black women.^{8,9} The results of these studies raise important questions about health disparities. Why do these trends exist? What differences between the groups account for the observed trends, and how do these differences produce different health outcomes?

The *Time* article proposed an answer to the questions raised above. It suggested that a possible explanation for the breast cancer disparities might be race-based biological differences between whites and non-whites. In a section ominously titled “Ethnicity as Destiny,” Kingsbury writes,

Of all the things that can determine a woman's chances of surviving breast cancer, perhaps one of the most powerful is the simple matter of race...black women in the U.S. and Africa...are at higher risk of developing a more aggressive form of breast cancer known as estrogen-receptor negative, or ER-negative...research findings released in June 2006 showed that 40% of premenopausal African-American breast-cancer patients have an even more dangerous form of ER-negative cancer called the basal-like subtype, resistant not only to estrogen but also to progesterone, another hormone that can be blocked by treatment.¹⁰

According to this article, certain races are more susceptible to certain types of breast cancer, and Kingsbury goes on to suggest that this susceptibility has a genetic component. Summarizing another study, she writes that Asians are more likely to be diagnosed with breast cancer at an early age. Based on the observation that hereditary cancers are also more likely to appear in younger women, Kingsbury echoes the study's conclusion that a large number of breast cancer cases in Asian women are attributable to certain mutations in genes predisposing one to breast cancer.¹¹ Thus, the article not only implies that breast cancer is a racialized disease, but it also suggests that these racial disease patterns have a genetic basis.

This idea is actually reflected in the medical literature. For example, one breast cancer paper describes in detail the biological properties of breast cancers found in black women and compares them with the corresponding data from their white counterparts. The study concludes that “intrinsic differences in tumor biology”^{12(p40)} between the groups could help account for the observed breast cancer disparities. The authors never explicitly define what they mean by “intrinsic differences,” but the phrase might be code for genetic difference. If this is, in fact, the intended usage, then according to the study, breast cancer is linked to genes through race.

Another article makes the proposed connection between race, genes, and breast cancer much clearer. The author, Peggy Porter, writes that “genetic determinants in African women may predispose them to high-risk tumors.”^{13(p215)} Thus, a linear relationship is set up: a woman’s race determines her genes, and her genes determine an eventual breast cancer outcome. In this way, a person’s race or ethnicity does appear to affect her destiny because it helps shape her health experience.

In my paper, I will argue that the latter statement might very well be true, but if it is true, it is not because of the former statement. Race, as we will see, has no meaningful genetic basis, and as such, it does not make sense to associate certain diseases with specific racial groups – that is, when racial groups are defined as genetically distinct subpopulations. For this reason, I will argue that breast cancer disparities are not a product of genetically-based biological differences between African-American and white women; instead, I will show that race is better understood as a complex social measure with strong biological implications. Black women are subject to a different set of social forces than their white counterparts and therefore occupy a different, and ultimately disadvantaged, position in society. This positioning scheme has distinct biological implications: as a result of various social inequalities, African-American women experience a different quality and pattern of health than white women.

I will argue that this fundamental difference in health experience is the source of breast cancer incidence and survival disparities between the two groups of women. In other words, in my paper, I will demonstrate that observable health disparities between racial groups are a reflection of *social* differences and inequalities – *not* biologically meaningful genetic differences. To argue convincingly that this is the case, I will show that: 1) biology is more than genetics, 2) biology is a social product, and 3) race is a social product.

The first chapter provides a historical and theoretical context for the debate surrounding race and breast cancer disparities. The 1980s and 1990s saw major health policy changes, as well as changes in scientific research priorities. I show how these two developments can help explain why some scientific studies present race as an inherent biological characteristic and depict disease as a primarily genetic outcome. These two actions in turn effectively portray breast cancer disparities as the product of race-based genetic differences between African-Americans and whites.

In the second chapter, I analyze genetic explanations for breast cancer disparities. Arguing that a one-to-one ratio between genetics and biology does not exist, I explain why the genetic arguments for the observed disparities do not represent an accurate understanding of disease causality.

In the third chapter, I propose an alternate model of disease causality, one that is based on the argument that race is a social measure. I examine how healthcare, socioeconomic, and environmental inequalities can act alone and in unison to produce poor health outcomes for black women. I also explain how these inequalities can shape health beliefs and behaviors and produce comorbid conditions that can further undermine the health of disadvantaged groups. By doing this, I demonstrate that because biology, in the form of health, is strongly influenced by social forces, it can be considered a social product.

In the final chapter, I show how race can also be understood as a social product by examining how the “African-American” racial category is constructed in a subset of the breast cancer literature. I argue that researchers function as active producers (versus passive discoverers) of scientific knowledge, and as such, they shape the knowledge that is produced. I analyze several articles written by Dr. Olufunmilayo I. Olopade, a Nigerian-born researcher

interested in African-American breast cancer patterns. As part of this analysis, I dissect Dr. Olopade's construction of racial categories to analyze the strength of each of her underlying arguments.

Before launching into my discussion, however, I would like to take a moment to explain my methods. My examination of race in breast cancer literature involved selecting different subsets of medical studies to provide primary source material to analyze in my discussions. To obtain material for the second and third chapters, I searched for relevant articles on PubMed (www.pubmed.org) using the following search terms: ("racial disparities" OR "racial disparity" OR "racial difference" OR "racial differences" OR "ethnic disparity" OR "ethnic disparities" OR "ethnic difference" OR "ethnic differences") AND "breast cancer" AND (("African American" OR "black") AND ("Caucasian" OR "white")). I also narrowed my search to only include studies published between 1995 and 2005. This search turned up sixty-seven articles.

After this initial search, I carefully read each of the sixty-seven articles to determine its relevance to my project. I identified thirty-six articles that specifically examined differences between African-American/black and white/Caucasian breast cancer patterns and proposed explanations for these disparities. Of these thirty-six articles, thirteen emphasized genetic explanations, and twenty-three emphasized non-genetic explanations. These two subsets of the breast cancer literature provided the primary source material for the second and third chapters, respectively. Finally, to obtain articles for the fourth chapter, I searched PubMed for breast cancer articles written by Dr. Olopade and found twelve that specifically addressed the observed disparities.

Additionally, I would like to clarify my usage of the terms "race," "ethnicity," "African-American," and "black" in this paper. If I offset the words "race," "ethnicity," and their

derivatives with quotation marks, it is to indicate that I am referring to a genetically- or biologically-based understanding of the terms. As I mentioned earlier, I will argue in this paper that race is a social measure with biological implications. Whenever I use this term without quotations, it is to indicate that I am referring to this particular definition of race. You will notice that I preferentially use the term “race” over “ethnicity” because race, with its heavy biological connotations, is the topic of discussion and debate in this paper. If I do use “ethnicity,” it is only to remain consistent with the language used in a particular book or study. As for the terms “African-American” and “black,” I will use them interchangeably in this paper. I do not want to use one term preferentially over the other because people belonging to these groups identify themselves with either or both labels, and I want to represent and respect this diversity of opinion.

These explanations and disclaimers aside, let us begin our examination of the use of race in breast cancer literature.

CHAPTER 1: Genes, Race, and Biology – Deconstructing the Scientific Vacuum

Introduction

Before we evaluate the genetic and social explanations for breast cancer disparities, we must first understand the historical and theoretical context of race in medicine. During the 1990s and 2000s, racial health disparities – such as those observed in breast cancer – were increasingly attributed to race-based genetic differences between the groups. I will argue that this trend was the product of key policy and scientific developments initiated during the 1980s and 1990s. On the policy end, different groups pressured Congress to pass the NIH Revitalization Act in 1993 to address the underrepresentation of women and racial and ethnic minorities in biomedical research. However, this legislation had the unintended effect of emphasizing ethnicity- and race-based differences between subsets of sample populations in scientific studies, as well as portraying race as an inherent, self-evident biological measure in the medical literature.

At around the same time of this policy shift, an equally important change was taking place in medical research: the move to focusing on genetic explanations for disease. The great promise of scientific endeavors such as the Human Genome Project, which was undertaken during this period, was that gene-based, individualized medicine would be the most effective way to treat diseases in the future. Later projects – for example, the proposed Human Genome Diversity Project – seemed to suggest that this individualization could be realized on the level of race. In fact, some companies have already begun capitalizing on this idea, as shown by the rise of pharmaceuticals marketed toward specific racial groups (i.e. BiDil).

The NIH Revitalization Act of 1993

Steven Epstein's *Inclusion* provides a neat history and analysis of the major health policy shifts that occurred during the 1980s and 1990s. According to Epstein, during the 1980s, a coalition of reformers began pressuring state and federal governmental agencies "to call for increased attention by researchers to specific groups in society and to warn against extrapolating onto those groups medical findings derived from the study of others."^{14(p53)} This coalition was made up of various groups representing diverse interests: women's health, racial and ethnic minorities' health, the health of children and the elderly, as well as the health of different "disease constituencies,"^{15(p54)} among others.¹⁶ Despite their varying interests and agendas, Epstein writes that they were all united by their concerns regarding "research underrepresentation and the biomedical scrutiny of differences."^{17(p55)}

What exactly does Epstein mean when he talks about "underrepresentation" and "the scrutiny of differences?" In other words, to what specific research practices did the health policy reformers object? According to Epstein, the groups demanding changes believed that health disparities were caused by the underrepresentation of various minority populations in biomedical research due to: 1) researchers' desire to protect "vulnerable" populations from potentially harmful experimentation (which Epstein calls "misguided protectionism") and 2) researchers' tendency to adopt the experience of the dominant social group as representative of other groups' experiences (which he dubs "false universalism").¹⁸ The reformers' arguments about underrepresentation were premised on the belief that significant biological differences separated the various minority groups (i.e. women, children, the elderly, racial and ethnic minorities) from the dominant population being studied (i.e. white men).¹⁹ We will examine the validity of this premise later in the paper.

After identifying key interest groups and dissecting their arguments for changing the existing health policies, Epstein describes the events that lead to the observed shift in medical research policy. According to his narrative, during the 1980s and 1990s, female members of the United States House of Representatives began pushing for greater inclusion of women in biomedical research. Their actions were partially the product of successful lobbying by women's health advocacy groups, such as the Society for the Advancement of Women's Health Research (SAWHR). The timing of the congresswomen's political move was crucial: since the budget for the National Institutes of Health (NIH) was due for reauthorization at the time, these female representatives (most notably Rep. Olympia Snowe and Rep. Patricia Schroeder, the cochairs of the Congressional Caucus for Women's Issues) took advantage of the bill's timing to call attention to women's health issues.²⁰ As a result of their efforts, a new section was added to the NIH Revitalization Act requiring researchers to include women as subjects in any projects funded by the NIH.

At the same time the Women's Caucus was pushing the legislation to include women in medical research, African-American representatives began calling for the inclusion of racial and ethnic minorities in NIH-funded studies. Epstein writes that "[o]nce members of the [Congressional] Black Caucus expressed interest in the new NIH initiative, members of the Congressional Caucus for Women's Issues were receptive...Consequently, the phrase 'and minorities' was added to the wording about inclusion of women."^{21(p79)} In this way, the Black Caucus was able to specifically address the underrepresentation of racial and ethnic minority groups in biomedical research.

Passage of the NIH Revitalization Act had significant consequences for the agency, as well as for researchers. First, it formally established two NIH offices dedicated specifically to the

health issues of women and minority groups: the Office of Research on Women's Health and the Office of Research on Minority Health. Second, the bill mandated the inclusion of women and racial and ethnic minority groups as research subjects in NIH-funded projects beginning in 1995. There were, of course, a few exceptions; inclusion of these groups could be waived in cases deemed "inappropriate" with regard to the subjects' health, the project's purposes, or other circumstances determined by the Director of the NIH.

In practice, however, the Act went beyond merely calling for the inclusion of formerly underrepresented groups in medical research – it effectively instructed researchers to design their studies to report the existence of any sex/gender or racial/ethnic differences in their sample populations. Quoting the Act, Epstein writes that it "required that NIH-funded trials be 'designed and carried out in a manner sufficient to provide for a valid analysis of whether the variables being studied in the trial affect women or members of minority groups, as the case may be, differently than other subjects in the trial.'"^{22(p82)} We can see how such ambiguous language might cause confusion. As Epstein writes, "[T]he legislation left it up to the NIH to develop formal guidelines specifying when inclusion might be inappropriate, how outreach programs should be designed to recruit women and minorities as subjects, and exactly how clinical trials should be structured in order to 'provide for a valid analysis' of differences."^{23(p83)}

Based on the above statement, we might predict that the policy ambiguities regarding the treatment of race in biomedical literature were eventually translated into methodological ambiguities. Unfortunately, this did happen. In a 2005 paper, Epstein explains, "In its implementation of the NIH Revitalization Act's directive concerning 'minorities,' the NIH could not entirely evade...definitional dilemmas [regarding the use of racial categories in medicine]."^{24(p196)} In other words, the agency did not (or could not) define racial or ethnic

groups, much less offer researchers a uniform, indisputable set of guidelines for identifying members of each group. As a result, researchers were free to formulate and employ their own definitions of racial or ethnic group identity in their studies.

One method of group classification available to researchers came from “Statistical Policy Directive No. 15” of the Office of Management and the Budget. This directive lists the five racial (“American Indian or Alaska Native,” “Asian,” “Black or African American,” “Native Hawaiian or other Pacific Islander,” and “White”) and two ethnic groups (“Hispanic or Latino” and “Not Hispanic or Latino”) used in the U.S. census. However, researchers using the groups presented in the census run into serious theoretical problems. Census categorization is far from objective; as Epstein writes, “[C]ensus categories are determined in response to a particular set of political needs and pressures, and they have changed with regular frequency since the initiation of the U.S. census in 1790.”^{25(p196)} We will unpack the meaning and implications of this statement in the next section.

The U.S. Census and the Construction of Race

Melissa Nobles’ *Shades of Citizenship* presents a detailed and thoughtful comparison and analysis of the United States and Brazilian censuses. Nobles argues that censuses are not objective; instead, she shows that both census-taking and racial category construction are dynamic, politically motivated actions with significant social implications. She begins by attacking the notion that race is self-evident: “Race is a complex and often internally contradictory set of ideas about human similarity and difference. Racial membership and racial boundaries are actively created and recreated through language, thought, social interactions, and institutional processes.”^{26(pxi)} Thus, the creation of census categories is complicated not only by the fundamental “definitional dilemmas” posed by the task of identifying racial groups, but also

by time: even when the census categories are made, they can be unmade or remade as the social and political climate dictates.

For an illustration of these ideas, let us consider a former racial category reported by the U.S. census: the “mulatto” classification. Nobles argues that the creation of this category was politically motivated. In a 2000 paper, she explains that the mulatto category was added in the 1850 census “not because of demographic shifts, but because of lobbying efforts of race scientists and the willingness of certain senators to do their bidding.”^{27(p1739)} She goes on to explain that, at the time, American race scientists were eager to prove that blacks and whites constituted two separate human species. To do this, they would have to show that mulattos, who represented the product of the two groups’ “interbreeding,” had shorter life expectancies and were less fertile than “pure-bred” individuals. In this context, the census represented a powerful tool; if it were made available to race scientists, it would enable them to collect the information they needed to prove their theories about race. The race scientists recognized this, and they successfully lobbied for the inclusion of the “mulatto” category in the 1850 U.S. census, where it remained until 1930. Nobles asserts that the adoption of this racial category also had important social implications: “The 1850 census introduced a pattern, especially in regard to the mulatto category, that lasted until 1930: the census was deliberately used to advance race science.”^{28(p1740)}

The application of the “mulatto” category becomes complicated if we consider how the group’s membership criteria changed over time. For example, in the 1850 census, colored people were identified as either “black” or “mulatto,” with “mulatto” signifying anyone of mixed black and white ancestry. By the 1890 census, however, census-takers were instructed to identify “quadroons” and “octroons” in addition to mulattos and blacks. Quoting the 1890 census

instructions, Nobles explains the differences between these four classifications: “The word ‘black’ should be used to describe those persons who have three-fourths or more black blood; ‘mulatto,’ those persons who have from three-eighths to five-eighths black blood; ‘quadroon,’ those persons who have one-fourth black blood, and ‘octroons,’ those persons who have one-eighth or any trace of black blood.”^{29(p1740)} Thus, individuals classified as mulattos in America in 1850 would *not necessarily* be classified as such in 1890 – the definition of the category changed over time. This example highlights an important point about racial groups: they are created, not discovered. This idea will be explored in greater detail in the final chapter.

As we just saw, the formulation of the “mulatto” category had political motivations and social implications. Nobles suggests that the same is true for all racial categories: “[R]acial enumeration itself creates and advances concepts of race, bringing into being the racial reality that census officials presume is already there, waiting to be counted. Ideas of race, in turn, shape public policies.”^{30(pxi)} Let us return to Epstein with this idea. Employing census racial categories in biomedical research does little to resolve questions and problems regarding the use of race in medicine. Given that these categories are so politicized and have such ambiguous, time-dependent boundaries, their use in scientific studies adds an additional layer of complexity to experimental design and analysis. Epstein writes that if researchers fail to acknowledge and address these issues, they may end up presenting racial categories as “trustworthy markers of difference that yield reliable medical maxims.”^{31(p197)} As we will see in later chapters, many researchers do not discuss issues of racial classification and do, in fact, fall into this trap.

Another problem we will encounter in the biomedical literature about breast cancer disparities is the conflation of genes and biology. Before tackling the question of how this conflation occurs, it is important to understand the history of genetics in biology. As I stated in

the introduction to this chapter, an important change was taking place within science during the 1980s and 1990s, at around the same time the health policy shift discussed above was occurring. In this period, several important scientific projects showcased the power of genetics. These high-profile genetic projects had varying agendas, but almost all of them promised that their results would have direct health implications. I will argue in the following sections that the enormous amount of resources invested in these projects, as well as the incredible interest and publicity they generated, effectively shifted attention from the social causes of disease to genetic explanations and linked these genetic explanations to biologically meaningful “racial” differences.

Linking Genes and Medicine: The Human Genome Project and its Legacy

The Human Genome Project (HGP) was an international research endeavor that began in 1990. The goal of this project was to sequence the entire human genome in the hopes that this information might someday have important research and therapeutic applications. Based on the observation that any two people in the world share 99.99 percent of their DNA, the scientists in charge of the HGP concluded that anyone’s genome could be used to generate the sequence and so created their map of the genome from DNA supplied by a small sample of anonymous donors. While the project was under way, and possibly because the HGP received a great deal of attention, a gradual but observable shift took place during the 1990s in which biomedical research began to privilege molecular genetics. This shift had important public health implications, since non-genetic explanations for disease outcomes were less likely to receive attention and funding than genetic explanations.

Troy Duster discusses this idea at length in *Backdoor to Eugenics*. Using the example of cancer research, he writes,

[I]f the apparatus of research institutions is set up to look primarily for the molecular genetics of cancer, nutritional studies which might illuminate how different diets affect cancer rates are less likely to get funded by the peer review process... The Nobel Prize is more likely to lie in the higher technology adventure of the latter, not the arena of prosaic controlled experimental studies of nutritional intervention. In the zero-sum of scientific energy and resources, other [non-genetic] approaches [to treating cancer] get less attention, less status, and ultimately a weakened capacity to do empirical battle with the dominant paradigm.^{32(p24)}

Duster argues that poor health outcomes are often multifactorial; diseases can be produced by any combination of genetic, biological, environmental, social, cultural, and economic factors. Thus, understanding and treating multifactorial diseases requires examining *all* of the contributing factors. Based on this idea, if medical institutions privilege genetic explanations for disease and preferentially fund research investigating this one factor, they may actually be precluding a complete understanding of disease causality because they fail to consider the effects of other non-genetic factors.

Duster is also concerned that the privileging of genetic explanations for disease may be a self-propagating, self-fulfilling phenomenon. If research institutions are, in fact, more likely to fund projects focusing on the role of molecular genetics in producing disease outcomes, then the body of published literature will reflect this bias. The resulting inflation of the number of studies reporting genetic explanations for disease may make it appear as if genetic factors are the primary cause. This in turn could prompt more research into gene-based disease investigations, thereby sustaining the cycle. This action is exactly what Duster warns against when he writes, “[O]nce the research [into genetic causes for a disease] is under way, both the scientific and conventional wisdom begin to be overwhelmed by the use of the nomenclature of genetic disorder.”^{33(p57)}

James Griesemer raises another concern regarding genetic technology in a paper about advances in genetic engineering. He writes,

It is often observed that technical advance frequently outpaces our ability to comprehend its consequences...The reason given is usually the failure of old scientific, moral, political, legal, medical, and social concepts to encompass the new developments. But this is only a partial truth. An important additional cause is a gap that arises when the representational repertoire of technologists is not sufficiently developed alongside the techniques, skills, and practices that contribute to new technology. In other words, the lagging means to *express* concepts may cause comprehension (and dissemination) to lag behind innovation [italics in original].^{34(p76-77)}

Basically, Griesemer's point is this: while we may have the technological tools to produce advanced scientific knowledge, we cannot anticipate what the social, political, and moral implications of this new knowledge might be. Although he is specifically referring to the technology applied in genetic engineering, the general concept can be extended to the HGP. In 2000, a working draft of the human genome was released by the scientists leading the project. This draft received a great deal of press and praise, but it is still not clear what this information *means*. In other words, while scientists have a complete sequence of the human genome, they are not yet able to say where each gene is located and what its function might be (but they are working on it). Because we do not yet know what the human genome map can and will tell us, we are unable to project – much less process – the consequences of mapping it.

Duster posits that one possible, unintended consequence of the HGP is that it has renewed interest in genetic differences between groups. He writes,

...there has been a sharp U-turn in the strategic orientation of the Human Genome Project. Rather than [place] an emphasis upon our 'sameness,' the field of molecular genetics has now begun to emphasize the importance of looking for 'differences at the level of DNA...While there are approximately three billion base pairs of complete overlapping similarity...that recurring figure of 99.99 percent also means that, with only a .01 difference, there are still approximately *thirty million points of difference* in the DNA between any two people...Suddenly, the realization of a considerable amount of differentiation between individuals is the new perspective—and lurking in the corridors of computer-generated correlations and patterns, there will be an inevitable shift to a concern for differences between population groups [italics in original].^{35(p146-147)}

This newfound, gene-based “concern for differences” echoes the arguments made by interest groups advocating health policy changes in the 1980s and 1990s. According to Epstein, these

interest groups justified the inclusion of racial and ethnic minorities as medical research subjects by arguing that significant biological differences existed between their groups and the dominant white social group. Because the health policy shift and the scientific research shift occurred almost simultaneously, were these assumed biological differences eventually understood to be the product of race-based genetic differences? And were these differences subsequently reinforced by medical studies designed to investigate biological differences between sample populations?

Peter Wade seems to think so. In *Race, Nature and Culture*, he writes, “[F]rom about 1990, there has been an increasing use of deterministic genetic imagery to sustain ideas of racial...difference among humans.”^{36(p103)} It is worth noting that the date provided by Wade, 1990, coincides with the initiation of the Human Genome Project. Given the major health policy changes taking place during this time, the concurrent shift to privileging genetics in science may have influenced the way researchers in this period understood and applied racial categories in their studies. Unfortunately, Wade does not discuss this idea in detail, leaving his readers wondering if there is, in fact, a relationship between the two events.

An interesting study conducted by Simon Outram and George Ellison supports the theory that race has become “geneticized” as a result of changes in policy and research trends, and their paper presents a stronger, clearer case for this relationship than Wade’s text does. In their study, Outram and Ellison argue that

despite inherent methodological limitations in pregenomic racial science, the notion of ‘race-as-subspecies’...was operationalized [in science] and became associated with social notions of group identity based on a conflation of geographical origin, culture visible phenotype, and genetic ancestry...In the process...phenotypic difference[s] became *racialized* and were thought to be related to extensive genetic variation [italics in original].^{37(p160)}

To test their theory, Outram and Ellison interviewed twenty-two geneticists about the use of race and ethnicity in medicine. These geneticists were members of the editorial boards of the nineteen most highly cited journals that publish genetic studies using racial and ethnic groupings.³⁸ The interview questions were designed to examine four areas: 1) how race and ethnicity are operationalized in genetic research through nomenclature, classification, and measurements; 2) the interviewees' thoughts about the validity and reliability of using race and ethnicity as variables; 3) the relationship between racial and ethnic phenotype and genotype; and 4) possible methods to strengthen the use of race and ethnicity as variables in genetic research.³⁹

Outram and Ellison's work generated some fascinating results. They found that only a few of the geneticists they interviewed "seemed to recognize that race and ethnicity are fluid and contingent concepts and are, therefore, inherently unreliable as scientific classifications of fixed categories with discrete boundaries."^{40(p173)} Despite the lack of classificatory precision, many of the interviewees believed that racial and ethnic categories were valid scientific measures based on the observed distribution patterns of certain genetic traits within a few populations. Furthermore, with regard to nomenclature, Outram and Ellison found that most of the geneticists they interviewed preferred using the term "race" to "ethnicity" because the former had, they felt, stronger biological connotations. However, the interviewees also felt that the term "ethnicity" was more socially acceptable to use than "race," and they were also prepared to substitute "ethnicity" for "race." Finally, Outram and Ellison found that while the geneticists were aware of possible controversies surrounding the use of either race or ethnicity in medicine, "few were prepared to forego the use of racial or ethnic categories in genetic research – at least not until there was more conclusive evidence of their questionable utility."^{41(p174)}

Additionally, Outram and Ellison's work suggests that there is a relationship between the health policy shift and the change in scientific research priorities that both occurred in the 1980s and 1990s. As mentioned above, they found that a number of geneticists believed that racial categories were, to some degree, genetically meaningful. This indicates that, in these geneticists' minds, a concrete scientific link exists between genetics and race. Furthermore, Outram and Ellison found that most of the interviewees were hesitant to stop using racial or ethnic categories in their work. Although they do not offer explanations for the geneticists' hesitation, it is likely due, at least in part, to the NIH inclusionary mandates. Since many scientists depend on grants from this agency to conduct their research, forgoing the tracking of racial and ethnic minority data could easily mean losing funding. Thus, another chain materializes, this time linking race and medical research. Together, these connections between genetics, race, and medicine suggest that the health policy changes and scientific research shifts of the 1980s and 1990s might be interrelated.

The results of Outram and Ellison's study have several important implications for scientific research, specifically genetic research. The most important consequence is that race and ethnicity are becoming what Outram and Ellison term "essentialized." In other words, these categories are depicted in the medical literature as integral, inherent characteristics of the populations they are applied to. Additionally, Outram and Ellison's study suggests that geneticists are distancing, perhaps even divorcing their work from the social context in which it is produced. Because they are keen to avoid political controversy and scientific complications regarding the use of racial and ethnic categories in their research, geneticists may avoid explaining the social context of race and ethnicity. These actions effectively separate the scientific and social spheres and establish geneticists as the authorities on race and ethnicity

within their sphere of influence. As we will see in the next section, which talks about the Human Genome Diversity Project, neglecting the social context of race and taking scientific authority for granted can have dire consequences. In fact, it was these two oversights that eventually doomed this proposed genetic project.

The Human Genome Diversity Project

The Human Genome Diversity Project (HGDP) was a proposed follow-up project to the HGP. It arose out of a concern for respecting and preserving genetic diversity in the overall human population and was based on the assumption that different subpopulations are genetically distinct – in other words, they have different genes and gene distribution patterns. Because of diverse, vocal opposition from several groups – including some groups that the project had intended to serve – the HGDP never materialized, despite the fact that it started out with the support of several prominent scientists and geneticists (for example, Luca Cavalli-Sforza). Jenny Reardon's *Race to the Finish* chronicles the rise and fall of the HGDP. In her book, Reardon explains the noble motivations behind the project and details the nature of the objections that eventually led to the HGDP's collapse.

According to Reardon, the HGDP grew out of a desire “to study the origin and evolution of the human species”^{42(p72)} by examining gene frequencies in socially- and geographically-isolated populations. The reasoning behind this sampling choice was that such populations would not have undergone significant genetic mixing with other groups – in other words, they represented genetically “pure” human populations. Obviously, these assumptions raise a number of questions: What populations would qualify as “isolated?” What would be the criteria for sufficient genetic isolation? Who had the expertise or authority make such decisions or conduct

the sampling? In a sense, however, all of these questions are merely variations of a larger question: how does science – specifically genetics – define a race/ethnicity/population?

The central arguments mounted against the HGDP responded to the final question posed in the preceding paragraph. Various groups representing the interests of the “isolated populations” that the project intended to sample argued that the HGDP would produce racist, colonialist science. Related to this fear were concerns over informed consent. How would consent be obtained from members of sample populations? Who would be authorized to give and receive this consent? Would individuals be compensated for their participation, and if so, how would they be compensated? Opponents of the HGDP did not believe that the project adequately answered these questions, and their vocal criticism eventually (and permanently) stalled this project.

The example of the HGDP illustrates that there is a great deal of ambiguity and controversy regarding the relationship between genetics and the construction of categories such as “race” and “population.” But despite this contention, the notion of biologically meaningful difference is still appealing to some groups. In the next section, I will explain how postgenomics – which moves beyond sequencing DNA toward understanding gene function and expression – is currently being used to argue that race-based “individualized” medicine is (or, at least, should be) the therapy of the future.

Beyond the HGP and HGDP: Postgenomics and the Promise of Personalized Medicine

In a 2007 paper, Nadia Abu El-Haj argues that “[p]ostgenomics has become increasingly focused on genetic diversity identified along the classificatory lines of race.”^{43(p284)} She begins by summarizing the ongoing scientific debate regarding the use of race in medicine: do racial categories designate inherent biological differences, or do they represent social identities that

have biological implications? Although she does not delve into the details of each side's argument, Abu El-Haj asserts that both sides share a common belief: individualized therapy may someday be a method for treating disease.

The problem is how to approach the task of individualizing medicine. Sequencing every person's genome would be extremely expensive and time-consuming, and given our limited knowledge of gene expression and function, the sequences produced would not necessarily yield enough information to justify these costs. This is where race comes in. Abu El-Haj writes that “[a]rguments for or against the use of race in biomedical research involve disagreements about whether race is a good or bad ‘surrogate’...for sequencing every individual's genome.”^{44(p285)} These arguments will be examined in detail in the next two chapters. For now, however, let us consider the question of winners and losers.

Who stands to win if the first definition of race (i.e. that race is an inherent biological characteristic) is accepted as valid, and what do they stand to gain? Kaushik Sunder Rajan's *Biocapital* offers possible answers to these questions. Sunder Rajan examines the relationship between postgenomics and economics, arguing that “the life sciences represent a new face, and a new phase, of capitalism, and consequently...biotechnology is a form of enterprise inextricable from contemporary capitalism.”^{45(p3)} He compares the parallel universes of the Indian and American biotechnology industries in his book to demonstrate how each country's economic and cultural factors influence its attitudes toward biotechnology.

In his discussion of the American biotechnology industry,⁴⁶ Sunder Rajan describes how the society's veneration of capitalism and its intense interest in personalized medicine have given rise to enormous investments in pharmacogenomics, “which involves tailoring prescriptions and drug regimens to individuals based on their likely, genetically determined response to these

drugs.”^{47(p151)} Thus, one of the groups that would stand to gain from a race-based approach to individualized medicine is pharmaceutical companies investing in race-based pharmacogenomics. An example of such a company is NitroMed, which began marketing the first race-based drug, BiDil, in 2005.

A Case Study for the Marketing of Race-Based Medicine: BiDil

In their paper about BiDil, Pamela Sankar and Johnathan Kahn chronicle its transformation from a race-neutral to a race-specific drug, arguing that BiDil does not produce race-specific therapeutic effects so much as it exploits race for commercial profit. BiDil was originally developed by Medco and started off as a combination of two generic drugs designed to treat congestive heart failure. In 1997, the Food and Drug Administration (FDA) rejected the company’s new drug application for BiDil – not because the drug was not clinically efficacious but because “Medco’s statistics were in too much of a muddle to meet the FDA’s criteria for new drug approval.”⁴⁸

What exactly was so “muddled” about the BiDil statistics? For an answer to this question, we can return to Steven Epstein. In the 1980s and 1990s, the FDA began making major health policy changes that paralleled the changes being made in the NIH during the same period. Epstein explains that during this time, the agency began asking pharmaceutical companies to provide specific information by “subset” – in other words, by age, race, and sex – for any differences in drug response that were observed during clinical trials.⁴⁹ Sankar and Kahn suggest that BiDil’s initial rejection stemmed from the FDA policy changes: “What changed...from the FDA’s 1996 rejection of BiDil and its 2001 encouragement was the emergence of a strong demand to respond to health disparities coupled with a growing acceptance [of] racial differences.”⁵⁰

After the initial FDA rejection, Medco surrendered the intellectual property rights to BiDil to Jay Cohn, who had been the lead cardiologist in the study. After analyzing the drug study results by race, Cohn and collaborating cardiologist Peter Carlson published a paper reporting BiDil's race-specific effects. Their paper generated the interest of biotechnology company NitroMed, which then obtained the rights to BiDil and subsequently received FDA approval. The drug turned out to be a financial boon for the privately held corporation; following the drug's approval by the FDA, the company raised over \$30 million from venture capitalists and later secured enough funds to go public. Summarizing this story, Sankar and Kahn remark, "Race...became relevant only when it offered a means to revive the commercial prospects of BiDil."⁵¹

The case of BiDil offers a fascinating example of how postgenomics, capitalism, and health policy can intersect with health disparities. It is widely known that African-Americans are more likely to suffer from hypertension – a major risk factor for congestive heart failure – than whites. A 2003 study investigating the prevalence of hypertension in the United States found that 33.5 percent of non-Hispanic blacks suffered from the disease, compared to only 28.9 percent of non-Hispanic whites.⁵² How can we account for this difference? If we are to believe the marketers of BiDil, the explanation lies in race-based genetic differences. They would say that there is some biologically meaningful genetic difference between African-Americans and whites that makes the former group more susceptible to hypertension than the latter – just as there is some meaningful biological difference that makes blacks more likely to respond to their drug than whites. In the closing of their paper, Sankar and Kahn explain the danger of such reasoning. They write that BiDil "not only...biologizes race but also...uses race as biology to create the impression that the best way to address health disparities is through commercial drug

development. By exploiting race in the service of product promotion, it distorts public understanding of health disparities and of efforts to address them.”⁵³

Conclusion: Science Is Not Produced in a Vacuum

I have tried to show in this chapter that scientific knowledge is strongly influenced by the time and place in which it is produced. I have described two major social changes that took place during the 1980s and 1990s: health policies were modified, and scientific research priorities shifted. Because both of these changes affected the process of producing of scientific knowledge, they also affected the knowledge that was eventually produced. In other words, science is not produced in a vacuum, but rather is grounded in and shaped by the social context in which it is produced. This is an important idea to keep in mind as we examine genetic explanations for breast cancer disparities.

CHAPTER 2: Genotype ≠ Phenotype – Genetic Explanations for Breast Cancer Disparities

Introduction

This chapter begins by examining the limitations of using genetic risk factors to explain breast cancer outcomes and the problems associated with understanding race as a purely genetic grouping. Building on this discussion, I will evaluate the validity of using race-based genetic differences to explain breast cancer disparities. To do this, I will analyze a sample of thirteen articles published between 1995 and 2005 that explain the observed breast cancer trends in terms of genetic differences between different racial groups. In my analysis of these articles, I will show how the language of science – and, specifically, the language of genetics – is used to argue for and ultimately construct racial categories. As we will see, this subset of the breast cancer literature presents a range of problems related to the treatment of race in medicine.

Breast Cancer as a Genetic Outcome

Genetic differences, in the form of gene mutations, can be used to argue for variations in breast cancer patterns. Mutations in three important genes – *BRCA1*, *BRCA2*, and *p53* – have been associated with increased breast cancer risk. *BRCA1* germline mutations have been found to confer a sixty to eight-five percent risk for breast cancer, and it is suspected that these mutations are responsible for twenty percent of hereditary breast cancer cases.⁵⁴ *BRCA2* mutations are believed to be responsible for an additional twenty percent of inherited breast cancers, and they are thought to confer a thirty to forty percent lifetime risk for the disease.⁵⁵ Finally, germline mutations of the *p53* gene, as observed in Li-Fraumeni syndrome, account for one percent of inherited breast cancers. Although these *p53* mutations are rare, they are believed to confer a ninety percent risk for the disease.⁵⁶

It is important to recognize that while these three genetic mutations represent significant risk factors for breast cancer, heritable breast cancers only account for about five percent of all cases of the disease.⁵⁷ In the remaining cases, breast cancer outcomes cannot be directly attributed to genetic factors. However, some of these cancers may be attributed to other, non-genetic risk factors, such as hormonal status, radiation, and the presence of previous breast cancers.⁵⁸ In the case of hormonal status, “[e]arly menarche, late menopause, and older age at first-term pregnancy all increase the risk of breast cancer.”^{59(p848)} Exposure to high levels of radiation – for example, undergoing treatment for Hodgkin’s disease – can also increase a woman’s risk of developing breast cancer. Lastly, women who have already had breast cancer “have at least a 10-fold increased risk of developing a second primary breast cancer.”^{60(p848)}

The variety of genetic and non-genetic risk factors serves to illustrate the complexity of breast cancer causality. Because only five percent of breast cancers can be directly attributed to genetic mutations, and because non-genetic risk factors can play an important role in producing breast cancer outcomes, focusing only on the contribution of genetic factors produces an oversimplified model of disease causality, which in turn precludes a complete understanding of breast cancer disparities. However, as we will see later in the chapter, many studies attributing gene mutations to breast cancer disparities overemphasize the importance of genetic factors while simultaneously ignoring the role of non-genetic factors.

Race as a Genetic Grouping

In 2002, a research group headed by Neil Risch published a controversial response to an editorial about race that was printed in the *New England Journal of Medicine*. The editorial in question asserted that race is not a biologically valid measure and advocated the abandonment of race-based medicine.⁶¹ Risch and his colleagues challenged these views, arguing that race does

have biological significance. In the introduction to their paper, they write, “[W]e demonstrate...that from both an objective and scientific (genetic and epidemiologic) perspective, there is great validity in racial/ethnic self-categorizations.”⁶² This passage highlights a rhetorical strategy we will see repeated in the breast cancer literature: invoking the authority of genetics. As I showed in the previous chapter, research priorities shifted during the 1980s and 1990s to privilege genetic knowledge, which in turn gave a great deal of scientific power and authority to the field of genetics. For this reason, when Risch asserts that genetics embodies scientific objectivity, he effectively dismisses potential counterarguments made by people who lack this authority.

Drawing on postgenomic arguments for individualized medicine, Risch and his colleagues claim that the most sensible and feasible way of identifying an individual’s genetic risk for a disease is to consider his or her race. Risch goes on to argue that racial groups can be genetically distinguished based on their continental origins. He identifies five such “continental groups”⁶³: Africans, Caucasians (people from Europe and the Middle East), Asians, Pacific Islanders, and Native Americans.⁶⁴ This choice of grouping might sound familiar. In the late eighteenth century, Johann Friedrich Blumenbach published his famous book *On the Natural Varieties of Mankind*, in which he described five distinct, continentally-based “races”: the Negroid (black) race, the Caucasian (white) race, the Mongolian (yellow) race, the Malayan (brown) race, and the American (red) race. To construct these groups, Blumenbach used craniometrical data taken from various skulls belonging to individuals from these different “races.”⁶⁵ Although Blumenbach’s methods and results have since been dismissed by scientists, it is hard not to hear echoes of his five phenotypically-based “continental races” in Risch’s five genetically determined “continental groups.”

This association might suggest that there is a relationship between genotype, phenotype, and race. In fact, Risch argues in his paper that such a relationship does exist in the context of disease. He writes that “the ultimate goal of genetic research is to identify those specific genes and gene variants that influence the risk of disease.”⁶⁶ Thus, a clear link is set up between disease genotype and disease phenotype. Based on this line of reasoning, it would follow that individuals with different disease genotypes would also have different disease phenotypes. Risch asserts that the most sensible way of grouping genetic differences is by geographic “race”: “[G]enetic differentiation is greatest when defined on a continental basis.”⁶⁷ By this logic, race (as given by continental origin) determines disease genotype, which in turn determines disease phenotype.

Three years after Risch’s paper was published, a group of researchers headed by Noah Rosenberg published another article arguing for a genetically-based understanding of race. In their methods section, the researchers explain that their study “applied a model-based clustering algorithm that, loosely speaking, identifies subgroups that have distinctive allele frequencies.”^{68(p2382)} We can see in this quote that, like Risch, Rosenberg et al. call on the authority of science to strengthen their position. They effectively remove themselves from their work and instead present their results as being computer-generated, which lends an air of scientific objectivity to their argument. If we read the article closely, however, we will see that this supposedly computer-generated product is actually generated by the researchers themselves. Rosenberg et al. gloss over the fact that the process of identifying genetically distinct populations starts when they input the desired number of groups into the algorithm.

In addition to borrowing Risch’s rhetorical strategy, the results of the Rosenberg et al. study also echo one of Risch’s central arguments. The researchers identified six major genetic clusters, and five of these groups were associated with “major geographic regions”^{69(p2381)}.

Africa, Eurasia, East Asia, Oceania, and America. These labels should sound familiar – Rosenberg’s five “populations” easily map onto Risch’s five “continental groups.” Thus, like Risch, Rosenberg argues for the existence of genetically distinct human subpopulations that roughly correspond with geographic regions. Interestingly, while Risch unabashedly uses “race” to describe these genetic subpopulations, Rosenberg’s group shies away from this term. In fact, they do not mention the word “race” once in the body of their six-page article – instead, they use the more neutral terms “population” and “group” to describe their population subsets. But because they adopt Risch’s arguments and rhetorical strategy, we can understand Rosenberg et al.’s “populations” to be equivalent to “races.”

There are several problems with understanding race purely as a product of genetics. The first major problem is the enormous genetic similarity across the human species. Ruth Hubbard writes that

because of the extent of interbreeding that has happened among human populations over time, our genetic diversity is pretty evenly distributed over the entire species. An occasional, relatively recent mutation may still be somewhat localized within a geographic area, but about 90 percent of the variations known to occur among humans as a whole occur also among individuals of any one national or racial group.^{70(p201)}

Thus, emphasizing small genetic differences obscures the overall similarity between different groups of people and presents an overly simplified picture of human population genetics.

The second issue, which comes from the “interbreeding” Hubbard describes, is the problem posed by people of mixed ancestry. Jonathan Marks explains this apparent paradox in *Human Biodiversity*: “[H]ow [can] an organism with half its genes derived from one stock be declared a member of another?”^{71(p111)} Furthermore, he writes that “where two groups co-exist with significant differentials in power and status, a great deal of weight is placed on relatively small amounts of heredity.”^{72(p111)} To illustrate this idea, Troy Duster writes that at certain

moments in American history, a person “could be anywhere from 1/32 to 32/32 black”^{73(p104)} and still be classified as black.

Thus, genetically speaking, racial categories are problematic to use because 1) they do not capture any underlying genetic homogeneity and 2) they are difficult, if not impossible, to define. Additionally, the process of group definition is not objective. Specific individuals or groups do the defining, and their motives for doing so are embedded in the eventual definitions they produce. We saw an example of this with the creation of the “mulatto” category in the 1850 census.

Identifying Race in Medical Literature: Issues of Transparency

When we examine the breast cancer literature, we see that many studies grapple with questions of how to treat and define race. Echoing Risch and Rosenberg, thirteen of the thirty-six breast cancer studies attributed the disparities to gene-based racial differences. Of these thirteen studies, four implied that race has a biological basis in genetics, while nine directly attributed racial differences to genetic variation between the groups (see Table 1). However, before taking a closer look at these articles and examining the various problems associated with genetic arguments for breast cancer causality, I would like to discuss issues surrounding racial group identification.

Of the thirteen studies that explained breast cancer disparities in terms of genetics, all but five did not include any explanation of how race was identified in their sample populations (see Table 1). This observation implies that race is an inherent, self-evident quality that does not demand any explanation. Furthermore, there are significant ambiguities in the methods of racial classification presented in the remaining studies. Consider third-party identification. One study⁷⁴ explained that subjects were classified based on patient demographics obtained from the

Surveillance, Epidemiology, and End Results (SEER) program. This, however, is not much of an explanation; the study does not provide information about how the SEER demographics were determined, nor does it address potential limitations of the SEER database itself. The same principles apply to a study that used race information that was "recorded in the hospital's clinical information system"^{75(p1545)} to classify its research subjects. The study did not explain how the hospital registry determined race, nor did it address potential ambiguities in the hospital's classification methods.

Table 1: Summary of Studies Attributing Racial Breast Cancer Disparities to Genetic Differences.

| Authors | Year | Directly or Indirectly Attributed to Genetic Differences | Group Description: Do they use "race" or "ethnicity" to define groups? | Method of Racial Classification |
|--|-----------------------------|--|--|---------------------------------|
| Shiao Y, Chen V, Scheer D, Wu X, Correa P | 1995 | Direct | Race only | Method not stated |
| Simon M, Severson R | 1996 | Indirect | Race only | Method not stated |
| Shiao Y, Chen V, Wu X, et al. | 1996 | Direct | Mostly race; one mention of ethnic groups | Method not stated |
| Shiao Y, Chen V, Lehmann H, Wu X, Correa P | 1997 | Indirect | Race only | Method not stated |
| Flaws J, Lim C, Luo J, Bush T | 2000 | Direct | Race only | Method not stated |
| El-Bastawissi A, White E, Mandelson M, Taplin S | 2001 | Indirect | Race or race/ethnicity | Self-identification |
| Haiman C, Pike M, Jaque S, et al. | 2002 | Direct | Mostly ethnicity, one mention of "racial/ethnic differences" | Self-identification |
| Chaudru V, Laing A, Dunston G, et al. | 2002 | Direct | Ethnicity only | Method not stated |
| Jones D, Cho J, Salamon E, Stefano G | 2003 | Direct | Race only | Method not stated |
| Hershman D, Weinberg M, Rosner Z, et al. | 2003 | Direct | Mostly race, one mention of ethnic groups | Third-party identification |
| Chen Z, Wu A, Gauderman W, et al. | 2004 | Indirect | Ethnicity only | Method not stated |
| Haffty B, Silber A, Matloff E, Chung J, Lannin D | 2006 (originally published) | Direct | Both | Self-identification |

| | | | | |
|--|--------------------|--------|---|-------------------------------|
| | online in 2005) | | | |
| Hance K, Anderson W, Devesa S, Young H, Levine P | 2005 | Direct | Mostly race, but also use "race/ethnicity" | Third-party identification |

Three other studies,⁷⁶⁻⁷⁸ on the other hand, specified self-identification as their method of racial categorization. But this method, too, provides limited information. In two of these studies,^{79,80} the researchers did not explain whether this self-identification consisted of choosing a label from a group of available categories or whether the participant could write in whatever group he or she identified with. The former method clearly indicates that the racial groups were predetermined by the researchers. Even in the latter scenario, a participant's response might still be informed by commonly-used social groupings, such as census racial categories.

The problems surrounding racial classification represent just one issue present in the thirteen breast cancer articles discussed above. These studies also demonstrate a variety of other problems related to their genetic arguments for disease outcomes. I have organized these various confusions into four general themes, which are presented in Table 2, along with a summary of why each is problematic. The rest of this chapter is devoted to an in-depth discussion of each of these themes.

Table 2: Themes in Studies Attributing Racial Breast Cancer Disparities to Genetic Differences.

| Theme | Problem |
|--|--|
| Principles of molecular genetics can always be applied to population genetics. | You cannot always use molecular genetics to understand population genetics. |
| The role of genes in explaining disease outcomes is overemphasized. | The role of gene regulatory mechanisms is de-emphasized. |
| Genetic explanations for breast cancer are overemphasized. | Non-genetic explanations for breast cancer are de-emphasized. |
| The relationship between genes and biology is overemphasized. | A one-to-one ratio between genes and biology does not exist, and to argue otherwise obscures a complete understanding of breast cancer causes. |

Confusions of Molecular and Population Genetics

James Griesemer explains that there is a relationship between molecular and population genetics. He writes,

While the abstract heredity relation may be aptly described in molecular terms as a flow of genetic information, reproduction—the process that causes inheritance—is not a flow of information but, rather, a flow of information-bearing matter. This flow has to do with both populational change in the distribution of genes and developmental control of gene expression.^{81(p82)}

I will argue in this section that this relationship is not always made clear in the medical literature. Breast cancer articles often downplay the importance of population genetics in explaining gene distribution patterns. As a result, there is an observable conflation or confusion of population and molecular genetics.

Jonathan Marks presents an example of how this confusion might appear. He explains that changes in single nucleotide polymorphisms (SNPs) – which are used to compare regions of genomes between individuals or groups of interest – often “have nothing to do with observable phenotypic differences manifested by people or populations...[because] most genetic variations are found in most populations, though in varying proportions.”^{82(p165)} In other words, small changes in molecular genetics do not necessarily translate into meaningful changes in population genetics. This reasoning seems to challenge the existence of race- or population-specific disease genes.

Troy Duster expands upon this idea. Although sickle cell anemia and sickle cell trait are often associated with black Americans, Duster comments that the disease is also present in Arabs, Sicilians, and other groups with Mediterranean origins. In fact, he notes that in one Greek population, sickle hemoglobin is found at roughly twice the rate in which it is found in African-Americans.⁸³ This example supports the idea that molecular genetics characteristics – in this

case, the mutant allele that causes sickle cell trait – do not always map neatly onto individual, discrete populations.

Let us now turn our attention to the breast cancer literature and examine how the studies treat molecular and population genetics. One example of how molecular genetics might be confused with population genetics can be found in a 2002 breast cancer article. The study hypothesized that the observed racial disparities in breast cancer survival might be at least partially explained by differences in levels of endogenous steroid hormones. The researchers conducting the study concluded that black women may have greater exposure to these hormones than white women, and they attributed this result to “genetic variation”^{84(p370)} between the groups, in addition to other non-genetic factors. However, in their conclusion, the researchers did not mention ways of remedying the nutritional or weight differences between the groups and instead recommended that future studies be “aimed at identifying the relevant genes involved in regulating...hormone production and investigating whether functional polymorphisms in these genes lead to variation in ovarian function.”^{85(p370)}

This study serves as an example of the conflation of molecular and population genetics. First, the researchers assume that deleterious genes exist to explain higher levels of endogenous hormones – in other words, they assume that a particular disease genotype will produce an observed disease phenotype. Additionally, their assumption that “genetic variation” exists between blacks and whites suggests that race has a genetic basis. Based on these two statements, the article’s overall conclusion that black women have higher levels of circulating hormones than white women might suggest that African-Americans are more likely to possess the mutant gene(s) purported to exist by the researchers conducting the study. As such, it is possible that this study is conflating molecular and population genetics.

A second study provides an even clearer example of how molecular and population genetics might be confused in the literature. The 2003 paper examined the role of estrogen in producing different breast cancer outcomes in black women as compared to white women. The abstract states that “recently there has been a shift to understanding the racial differences in genotype...related to tumor growth.”^{86(pRA131)} This sentence leaves no question that the researchers conducting the study believe that race has a genetic basis.

In one section of their paper, the researchers consider what effects mutations in the UGT enzyme, which is involved in drug detoxification, might have in producing breast cancer. They write,

*UGT1A1*1* is the wil[d]type allele that maintains the appropriate amount of UGT levels in the blood serum. However, compared to wil[d]type, *UGT1A1*28*, *33*, and *34* had a decrease in transcription by 30%, 20%, and 50% respectively. Note, the wil[d]type and *UGT1A1*28* alleles were observed in the Caucasian population, and the *UGT1A1*33* and *34* were observed exclusively in the African American population...These mutated alleles were positively related to abnormal breast epithelial cell proliferation. Moreover, these polymorphisms are predominant in ER- breast tumors and over weight [sic] women which focuses primarily on the African American population.^{87(pRA131)}

The above excerpt links breast cancer to mutations in the UGT gene. It also clearly states that certain mutations – including two deleterious mutations – are “observed exclusively” in blacks, while others are predominantly found in whites. Thus, this paper uses observations in molecular genetics to argue for corresponding trends in population genetics.

On a literal level, the study’s invocation of “wild-type alleles” versus “mutant alleles” is extremely problematic. To imply that the wild-type variant of an allele is exclusively associated with one group is to imply that the other group is, by default, genetically mutant. When these groups are different races, as is the case in the article discussed above, such a suggestion could easily be interpreted as scientific racism. Additionally, describing breast cancer disparities in terms of “wild-type” and “mutant” alleles represents an inappropriate application of principles of

molecular genetics to population genetics. A mutant allele represents a variant of a gene that differs from the “wild-type” in its DNA sequence. This DNA mutation may produce a deleterious disease phenotype, but it might also be what is called a “silent mutation” – one that does not produce a phenotypic change. For this reason, it does not make sense to associate different alleles – which may or may not have observable phenotypes – with the purely phenotypic measure of race.

The example of silent mutations shows that there is not always a one-to-one ratio between phenotype and genotype. In the next two sections, I will explain two additional reasons why a direct correspondence is not always observed: gene regulatory mechanisms and environmental factors.

Why Genotype ≠ Phenotype Part I: The Importance of Gene Regulatory Mechanisms

James Watson, co-discoverer of the structure of DNA, once said, “We used to think that our fate was in our stars. Now we know, in large part, that our fate is in our genes.” As we saw earlier in the discussion of breast cancer risk factors, genes can and do play a role in producing biological outcomes. However, to say that “our fate is in our genes” completely ignores the importance of gene regulatory mechanisms, which are required for gene expression.

Gene expression is a complex process that involves a number of different players. Anne Fausto-Sterling writes in *Sexing the Body* that

genetic function can be understood only in the context of that developmental system we call the cell...Naked DNA cannot make a protein. It needs many other molecules—special RNAs to carry the amino acid to the ribosome and secure it, like a vise, so that other proteins can link it to its neighbor. Proteins also help transport the DNA’s message out of the nucleus and into the cytoplasm, help the DNA unwind so that other molecules can interpret its message in the first place and cut and splice the RNA template.^{88(p236-237)}

According to Fausto-Sterling, an individual gene is embedded in and therefore subject to the influences of an entire “developmental system,” represented by the surrounding cellular

environment. In this model, it is easy to imagine that if any one of these components malfunctioned, it could produce a devastating biological outcome, regardless of the functionality of the “naked” gene. Thus, presenting genes as the most important or the only important part of this system produces an incomplete model of disease causality.

Tom Shakespeare discusses the importance of understanding and accurately representing the full complexity of the entire “developmental system.” He writes,

The revelation that there are perhaps fewer than 30,000 genes in the human genome has sometimes been taken as evidence that genes cannot explain the complexity of human behavior and action. However, the real significance is to highlight the way that gene regulation and gene expression are central to understanding genetics. The switching on and off of genes...explains important biological differences.^{89(p125)}

Unfortunately, when we examine the breast cancer literature about racial disparities, we see that the role of gene regulatory mechanisms is often de-emphasized, even though, as Shakespeare argues, gene regulation is what ultimately determines gene expression. This in turn produces an incomplete picture of disease causality.

Carl Cranor describes how such a picture might be generated if one ignores the importance of gene regulation. He explains, “On the lookout for ‘suspect’ factors in the causal process, scientists may tend to ‘blame’ (in a metaphorical sense) a gene for the condition in question. They may quickly fix attention on genes simply because...[they] may make some causal contribution, however small, to the condition.”^{90(p133-134)} For example, if several articles were published that placed excessive emphasis on the role of a particular gene while ignoring that gene’s regulation, this action might suggest that the “naked gene” was more important than its regulatory mechanisms in producing a disease outcome.

In fact, we can see examples of this in the breast cancer literature. Two separate studies written by the same team of researchers^{91,92} investigate the action of the *p53* gene alone and cast its function in racialized terms. The earlier study states, “The differences in types of *p53* gene

alterations between blacks and whites suggests that genetic susceptibility and/or environment exposure could be different between blacks and whites...[suggesting] that *p53* gene alterations are an independent survival predictor for blacks but not for whites."^{93(p1489)} The second study links these genetic differences to breast cancer disparities: "[M]utations of the *p53* gene which result in loss of protein expression or abnormal protein with short half-life are associated with breast cancer death in blacks."^{94(p171)} As discussed previously, mutations in the *p53* gene have been associated with increased breast cancer risk. However, the *p53* gene is subject to regulation by a number of other genes, and its misregulation could produce similar disease outcomes. However, neither study mentions these regulatory mechanisms or the effect of gene misregulation. Consequently, *p53* gene mutations are presented as being the most important cause of breast cancer.

Like *p53*, mutations in the *BRCA1* and *BRCA2* genes have also been associated with an increased risk of breast cancer. A study published in 2005 attempted to explain breast cancer disparities in terms of *BRCA1* and *BRCA2* mutations. The researchers' goal was "to identify the molecular and biological factors associated with the more aggressive behaviour of breast cancer in African American women...[by studying]...the frequency and distribution of *BRCA1* and *BRCA2* mutations in young African American women with breast cancer."^{95(p133)} As in the case of the *p53* papers, this study was only interested in identifying mutations in these two genes and observing the distribution patterns of these mutations across racial groups. There is no mention in the statement of intent of examining gene regulatory processes, and, in fact, there is no discussion of these processes anywhere in the article. Once again, researchers overemphasize the importance of genes in producing disease outcomes, while at the same time ignoring the contributions of gene regulatory mechanisms.

Why Genotype ≠ Phenotype Part II: The Importance of External Environment

As we saw in the previous section, the medical literature can obscure the importance of the internal, cellular environment in gene expression. Another equally important factor involved in the gene regulation process is the external environment, and unfortunately, the literature also ignores or de-emphasizes its role. An individual's external environment is shaped by all the social, economic, and cultural forces he or she is subject to. These factors can in turn affect gene expression, thereby influencing disease outcomes. The importance of these various factors will be discussed in greater detail in the following chapter, but in this section, I will briefly outline why the external environment is an important explanation for breast cancer and explain why ignoring its role is detrimental to understanding cancer causality.

Evelyn Fox Keller explains the impact an individual's external environment can have on disease outcomes: "Genetics is a powerful tool for identifying particular components of the causal sequence that leads to particular diseases. But only for very exceptional diseases can these genetic components be considered apart from the environment...For the vast majority of diseases, the impairment of function requires as well the right (or wrong) environmental conditions."^{96(p96)} An individual's social environment, then, can affect the expression of different genes, which in turn can affect disease outcomes.

Troy Duster explains why ignoring non-genetic factors, such as the external environment, can be detrimental to understanding disease causality. He explains that focusing only on genetic factors represents a kind of medical reductionism: one disease, one cause.⁹⁷ Reducing multifactorial diseases to pure genetics also poses a significant classificatory problem. He writes,

[U]nder what conditions is a disorder characterized and treated as if it were more genetically than environmentally induced? If there were some kind of scientific precision in our measurement, then one could say for example that when a disorder is 51 percent or more 'genetic,' it will be called a 'genetic disorder,' and when a disorder is 51 percent or more 'environmental,' it will be called an 'environmental disorder.'^{98(p56)}

Obviously, no such scientific methodology exists. Duster's point is that it is almost impossible to determine whether a disease is a purely "genetic disorder" because the genes and the environment interact to produce biological outcomes. As we saw in the discussion of breast cancer risk factors, both genetic *and* non-genetic factors can influence disease outcomes. For this reason, scientists should not discount the role that non-genetic, environmental factors play in producing disease.

How does the breast cancer literature portray the relationship between an individual's genes and his or her environment? Unfortunately, this relationship is not discussed in a number of studies, and even when it is, the genetic factors often overshadow the non-genetic ones. For example, in the conclusion of a 2006 article, the researchers quote an editorial written by Lisa A. Newman, the Director of the University of Michigan Breast Care Center. Newman stated that "the door to the improved understanding of ethnicity related variation in breast carcinoma risk and outcome has now been wedged open by the powerful tools of molecular oncology."^{99(p135)} This quote effectively ignores the importance of the external environment, and the researchers' choice to quote Newman reinforces her implication that genes play the more important role in producing a breast cancer outcome.

Other articles also omit a discussion of environmental factors and focus on the genetic aspects of breast cancer. One article published in 1997 states in a section about the study's limitations that "some fairly large effects cannot be ruled out,"^{100(p591)} which could be understood to mean socioeconomic or environmental factors. Despite this potentially large oversight, the researchers conducting the study were still confident enough to conclude that "breast cancers...follow a different tumorigenic pathway by race."^{101(p591)} Similarly, an article published in 2003 states that the observed lower baseline white blood cell (WBC) counts in

African-Americans – which the paper suggests may explain chemotherapeutic dose reduction and lower survival – is caused by “genetic differences”^{102(p1545)} between blacks and whites. This study completely ignores the possibility that an individual’s environment can also lower his or her WBC count.

One 1996 study by Michael Simon and Richard Severson did mention that environmental factors play a role in breast cancer, but the importance of these factors was overshadowed by the emphasis placed on genetic factors. Their paper cited “differences in tumor biology,”^{103(p309)} as well as differences in “biological characteristics”^{104(p312)} between blacks and whites as possible explanations for the observed breast cancer disparities. It is unclear from these statements whether “biological characteristics” is code for genes. If this is the case, then the researchers’ claim that “differences in survival are most likely...due to differences in biological characteristics, and/or issues related to access to medical care”^{105(p312)} would place genetic factors above environmental ones. This is because of the sentence’s construction: while the “biological differences” are presented first and are therefore presented as a given, the “and/or” notation suggests that differences in access to care represent less certain explanations for the disparities.

Conclusion: Biology Cannot Be Reduced to Genetics

I chose to conclude the previous section with the paper by Simon and Severson because it neatly illustrates the overall problem in the breast cancer articles attempting to explain racial disparities in terms of genetics: the ambiguity surrounding the relationship between race, genotype, and phenotype. Their study is representative of this subset of the breast cancer literature, which often leaves questions about biology and genetics unanswered. When scientists

invoke “biological differences” between racial groups, what do they *really* mean? In other words, how are race, genetics, and biology related?

In this chapter, I have argued that understanding race as a pure genetic product presents significant definitional problems. Additionally, I have shown that disease, specifically breast cancer, is also not a pure genetic product by demonstrating that a one-to-one ratio between genotype and phenotype does not exist. Gene function alone does not produce a biological outcome; in reality, gene expression is influenced by the surrounding cellular environment, as well as by the external environment. In the next chapter, I will examine the role of this external environment by analyzing how social factors can shape breast cancer outcomes.

CHAPTER 3: Arguments for Intersectionality – Social Explanations for Breast Cancer Disparities

Introduction

In this chapter, I will present arguments for a second definition of race, one that views race as a social product with biological implications for health. This understanding of race is the basis for an alternate model for disease causality that is very different from the gene-based model previously presented. I will argue that biological outcomes can be socially produced by explaining how various social factors can influence health. By this line of reasoning, health disparities can be explained in terms of fundamental social inequalities.

Using another subset of articles about breast cancer disparities, I will argue that black women are more likely to experience socioeconomic, environmental, and healthcare inequalities than their white counterparts. Furthermore, beyond directly influencing health outcomes, these social inequalities can also explain “cultural differences” (i.e. differences in patterns of health beliefs and behaviors) between African-American and white women, as well as comorbid conditions. After making the case that social inequities help produce health disparities, I will consider how the different inequalities might intersect and compound each other to place black women in a uniquely disadvantaged social position.

A Brief Note on Definitions and Usages

Before launching into a discussion of various social inequities, I would like to define what I mean by the terms “environment” and “culture.” In the previous chapter, I explained how internal and external environments can influence disease outcomes. When I talk about “environment” in this chapter, however, I am referring specifically to an individual’s external

environment – the setting(s) in which she lives and works. We will see how the health of an individual’s home and work environment can affect that individual’s health.

The other term I would like to try to define is “culture.” There is a danger in attributing certain health beliefs or behaviors observed in African-Americans to *implicit* cultural values or patterns. Doing so amounts to the substitution of “an essentialized notion of culture for race.”^{106(p4)} I will argue that unequal social conditions shape and generate the observed “cultural differences” between African-American and white women. Thus, “culture” – as I use it in this chapter – is not an innate, independent entity but rather is produced by other social factors.

The definitions I have provided above merely represent a loose set of guidelines for breaking down potential causes of health disparities. In reality, these factors are co-produced within given social conditions and so are interrelated. This notion of intersectionality will be explored in greater depth later in the chapter.

Race as a Social Product

Whereas the previous chapter considered the argument that race is a genetically-based quality, this section will explain how race can be better understood as a product of social forces. In contrast to Risch and Rosenberg, Immanuel Wallerstein argues that racial groups are subjective, socially-constructed categories. He writes,

It may be obvious that there are a large series of genetic traits that vary, and vary considerably, among different persons. It is not at all obvious that these have to be coded as falling into three, five, or fifteen reified groupings we call ‘races.’ The number of categories, indeed the fact of any categorization, is a social decision.^{107(p382-382)}

In other words, individuals or groups create racial categories – there is no intuitive system of racial classification. We saw an example of how this “social decision” can play out in science in the Rosenberg study. The researchers determined what they believed to be valid standards of human classification and then grouped people according to the rules of their system.

In fact, whenever scientists or geneticists use the terms “race” or “ethnicity” in their research, they are participating in the creation and validation of such groupings. Lundy Braun explains how scientists using racial categories in their research can produce new understandings of these terms: because they actively produce knowledge, the researchers’ biases and agendas shape their final product.¹⁰⁸ Additionally, although racial groupings are actively (re)created by various actors, the authority wielded by scientists supplies additional validity to such categories. We already saw an example of this with the creation and subsequent tabulation of the “mulatto” category in the nineteenth century.

Both Wallerstein and Braun suggest that race has an important social component. But what social forces contribute to the creation of this racial identity? The work of Oliver Cox presents one possible answer. Cox wrote extensively about the relationship between race and class during the first half of the twentieth century and argued that

racial exploitation is merely one aspect of the problem of the proletarianization of labor, regardless of the color of the laborer. Hence, racial antagonism is essentially a political-class conflict. The capitalist exploiter, being opportunistic and practical, will utilize any convenience to keep his labor and other resources freely exploitable. He will devise and employ race prejudice when that becomes convenient.^{109(p208)}

According to Cox, then, the social construction of race has an economic component, suggesting that a relationship exists between race and class. But what exactly is this relationship?

To begin to answer this question, let us revisit Peter Wade. Building on Cox’s ideas, Wade writes that while economic conflicts can (re)produce race conflicts, “racialised ideas and practices can [also] affect economic structures.”^{110(p17)} According to Wade, there is a complex *interrelationship* between race and class: the two cannot be divorced from each other, nor can they be reduced to each other. For these reasons, it is misleading to present race as a purely biological product, as the studies discussed in Chapter 2 do. Instead of explaining health disparities in terms of proposed gene-based racial differences, I will show how race, when

defined as a social measure, can have significant implications for health. Before tackling the biological implications of racial identity, however, we need to understand how race is identified in the breast cancer literature attributing the observed disparities to social forces.

A Familiar Problem: “Definitional Dilemmas” of Race

In contrast to the articles described in the previous chapter, several studies examined in this chapter provide thoughtful commentary about the “definitional dilemmas” posed by racial identification. One of these papers states,

Too often in research, race is viewed as being directly related to a health outcome when, in fact, it is a proxy variable for, or an influence of, many aspects of socioeconomic and healthcare experience that subsequently affect health outcome. Thus, race is representative of patient characteristics such as biological factors pertaining to heredity, health status as measured by comorbidities, and health concern that determines care-seeking behaviors. But, race may also be a covariable for the degree of access to care and the quality of medical care received from the healthcare system, when discerning measures such as insurance status, the source of care, and the doctor-patient relationship might be more appropriate variables to study.^{111(p228)}

Echoing Cox and Wade, this study asserts that race is a complex social measure that is, in some sense, related to class. Additionally, the researchers argue that race has biological implications (although they hold a place for heredity). Two other breast cancer studies echo these arguments: one article stated that race could be understood as an index of “cultural differences in health beliefs and attitudes,”^{112(p2306)} while the other asserted that the “external, environmental components of race,”^{113(p145)} such as socioeconomic status, could help explain the observed breast cancer disparities.

While the three studies discussed above provide insightful commentary about the treatment of race in medicine, they are exceptions to the rule. Of the twenty-three articles analyzed in this section, the remaining twenty did not explain their definitions of “race” and “ethnicity.” Without explicitly stating what they mean by these terms, researchers are also unable to clarify their method of racial identification. Of the twenty-three studies analyzed in this

section, nine did not explicitly provide information about their methods of racial classification (see Table 3). Of the remaining articles, nine reported identification by a third party, such as an interviewer or cancer registry, while five cited self-identification (see Table 3). However, these two methods also present their own sets of problems. Third-party identification is subject to personal biases and beliefs, and, as explained in the previous chapter, even self-identification is fraught with ambiguities.

One study, however, did comment on the problems associated with racial classification, paying particular attention to the problem of third-party classification. The researchers used information from the North American Association of Central Cancer Registries (NAACCR) to track race and ethnicity. Commenting on the limitations of these registries, they stated, “The quality of data on race and ethnicity is limited by the quality of information available in records. Misclassification bias may be significant...The 'race/ethnicity' question appears on most admission forms, but if not completed by the patient, the question may not be asked by admission staff.”^{114(p103)} In spite of this serious limitation, the researchers conducting the study did not discuss potential solutions to remedy this misclassification problem, and, in fact, few studies devote any discussion to the ambiguities surrounding racial classification.

Table 3: Summary of Studies Attributing Racial Breast Cancer Disparities to Social Forces.

| Authors | Year | Explanation for Disparity | Group Description: Do they use "race" or "ethnicity" to define groups? | Method of Racial Classification |
|---|------|--|--|---------------------------------|
| Glanz K, Resch, N | 1996 | Healthcare inequalities (access to care), cultural differences (education) | Mostly race | No method |
| Chang S, Kerlikowske K, Glanz K, Resch, Nápoles-Springer A, Posner S, Sickles E, Pérez-Stable E | 1996 | Healthcare inequalities (access to care), socioeconomic inequalities, cultural differences (education) | Mostly race | Third-party identification |
| Miller A, Champion V | 1997 | Socioeconomic inequalities, cultural differences (education) | Race only | Third-party identification |

| | | | | |
|--|------|--|----------------|----------------------------|
| O'Malley M, Earp J, Harris R | 1997 | Healthcare inequalities (access to care), cultural differences (education) | Race only | Self-identified |
| Simon M, Severson R | 1997 | Socioeconomic inequalities | Race only | Third-party identification |
| Jones B, Kasl S, Curnen M, Owens P, Dubrow R | 1997 | Comorbid conditions | Race only | Third-party identification |
| Howard D, Penchansky R, Brown M | 1998 | Healthcare inequalities (access to care), cultural differences (education) | Race only | No method |
| Lannin D, Mathews H, Mitchell J, Swanson M, Swanson F, Edwards M | 1998 | Socioeconomic inequalities, cultural differences (education) | Race only | No method |
| Velanovich V, Yood M, Bawle U, et al. | 1999 | Healthcare inequalities (access to care) | Race only | No method |
| Moorman P, Jones B, Millikan R, Hall I, Newman B | 2001 | Comorbid conditions | Race only | No method |
| Miller B, Hankey B, Thomas T | 2002 | Socioeconomic inequalities | Ethnicity only | No method |
| Johnson E | 2002 | Healthcare inequalities (access to care) | Race only | Third-party identification |
| Cui Y, Whiteman M, Langenberg P, et al. | 2002 | Comorbid conditions | Race only | Self-identified |
| Harris D, Miller J, Davis D | 2003 | Healthcare inequalities (access to care and quality of care), socioeconomic inequalities, cultural differences (education) | Race only | No method |
| Griggs J, Sorbero M, Stark A, Heininger S, Dick A | 2003 | Healthcare inequalities (quality of care), comorbid conditions | Mostly race | Self-identified |
| Magai C, Consedine N, Conway F, Neugut A, Culver C | 2004 | Cultural differences | Ethnicity only | Third-party identification |
| Polednak A | 2004 | Comorbid conditions | Both | Self-identified |
| Elmore J, Nakano C, Linden H, Reisch L, Ayanian J, Larson E | 2005 | Healthcare inequalities (quality of care) | Mostly race | Third-party identification |
| Li C | 2005 | Healthcare inequalities (access to care and quality of care), socioeconomic inequalities | Both | Third-party identification |
| Joslyn S, Foote M, Nasser K, Coughlin S, Howe H | 2005 | Healthcare inequalities | Both | No method |
| Smith K, Wray L, Klein-Cabral M, et al. | 2005 | Healthcare inequalities | Ethnicity only | No method |
| Hershman D, McBride R, Jacobson J, et al. | 2005 | Healthcare inequalities | Race only | Third-party identification |
| Tammemagi C, Nerenz D, Neslund-Dudas C, Feldkamp C, Nathanson D | 2005 | Comorbid conditions | Mostly race | Self-identified |

Institutional Racism and Medicine

To understand race's biological implications, we must first examine the systems that produce and perpetuate social inequalities. Ruth Hubbard argues that social forces can help explain health disparities. She writes,

To come up with rational explanations [for health disparities], we need to take account of the fact that the median income of African Americans since 1940 has been less than two-thirds that of Americans of European descent. Disproportionate numbers of African Americans live in polluted and run-down neighborhoods, work in more polluted and stressful workplaces, and have fewer escape routes out of these living and work situations than Euro-Americans.^{115(p200-201)}

Hubbard later identifies racial discrimination as an explanation for these social inequalities. She states that the everyday racial prejudice blacks experience can help explain the difference in their quality of health as compared to that of white Americans.¹¹⁶ She does not, however, explicitly identify who or what is the source of this discrimination.

In *Racism, Health, and Post-Industrialism*, Clovis Semmes argues that American medical institutions represent a source of discrimination and connects them to health disparities between different racial groups. Semmes writes that “racial inequity resides in historically based institutional arrangements.”^{117(p105)} Building on this point, he argues that the “overt and covert exclusion of African Americans from the medical system”^{118(p105)} is a form of institutional racism that directly and negatively impacts the health of black Americans.

Patricia King expands on Semmes' theory. Extending the institutional racism explanation beyond medical institutions, she writes,

Persistent social and economic inequalities in the society provide the context for large disparities in health outcomes and in healthcare that exist between blacks and whites in the United States...Social, physical, and economic environments are viewed as major contributors to these disparities. The impact of these factors in turn is linked to the norms and values of the broader society that shape the way critical resources and opportunities are distributed.^{119(p68)}

King suggests that medical institutions play a role in producing health disparities, but they are not the only institutions to blame. She implicates the various social, political, and economic institutions that produce an environment in which the health of one group is privileged over the health of another.

Semmes and King argue that institutional racism exists in theory, but they do not offer examples of what it looks like in practice. For examples of its visible effects, we will turn to Lorna Scott McBarnette. McBarnette cites two primary indicators (although, arguably, there are many others) supporting the existence of institutional racism: 1) the hours of operation of many health institutions and 2) the dearth of black health professionals.¹²⁰ She explains that the conventional hours of operation are inconvenient for many poor women, who often have to negotiate inflexible work schedules, children who need care, and limited travel accommodations.¹²¹ As for the second indicator, McBarnette writes that “although African Americans make up approximately 12% of the U.S. population, less than 3% of U.S. physicians, less than 3% of dentists, and less than 2% of biomedical scientists are of African American descent.”^{122(p64)} These statistics suggest that institutional racism exists in the American healthcare system. In the next section, we will examine how this racism can negatively impact the health of black women.

Healthcare Inequalities

One possible explanation for health disparities involves differences in healthcare. For example, the structure and policies of the United States’ healthcare system limits certain groups’ access to care. McBarnette writes that

gaps in insurance coverage restrict access to services for growing numbers of the population, and African American women are especially vulnerable because of numerous demographic factors...specifically, unemployment and marginal employment...Another problem is that people who are in poor health [such as African-American women] or have

preexisting conditions are prevented, by virtue of their health status, from qualifying for coverage under most plans.^{123(p61-62)}

Clearly, this insurance inequity is a serious problem. Not only are a vast number of people slipping through the cracks of the healthcare system, but, according to McBarnette, the people that this system fails may also be those that need healthcare the most. Furthermore, these insurance barriers are more likely to affect African-Americans: a 2000 study found that in 1996, 19.2 percent of black Americans had no usual source of healthcare (i.e. were uninsured), compared to 15.1 percent of white Americans.¹²⁴ Thus, insurance differences could help explain the observed breast cancer disparities by accounting for differences in access to care.

This argument is reflected to some degree in the breast cancer literature. Of the twenty-three articles citing non-genetic explanations for the observed racial disparities, thirteen studies cited healthcare differences as a contributing factor, nine of which specifically addressed the unequal access to care (see Table 3). However, seven¹²⁵⁻¹³¹ of these nine studies suggested that disparities in access to care only functioned as a “minimal”^{132(p232)} or “partial”^{133(p379)} explanation for differences in breast cancer trends, essentially de-emphasizing the impact of this social inequality.

Two articles, however, took a much stronger stance, arguing that future breast cancer disparity studies need to pay greater attention to insurance inequalities, which can compromise individuals’ access to healthcare. A 2002 paper stated that the general lack of health insurance in its African-American sample population was an issue that demanded attention.¹³⁴ Another article published a year later went even further. The study argued that “[f]uture studies should ideally include a measure of insurance status to address this important limitation, because insurance has been shown to affect care-seeking behavior and provider attitudes and medical decision-making.”^{135(p699)} Because insurance inequities can influence “provider attitudes and medical

decision-making,” then perhaps limiting black women’s access to healthcare also affects the quality of care they receive.

But do black women experience a different quality of healthcare than white women? To answer this question, we will return to McBarnette. Echoing the arguments about institutional racism presented earlier, she writes,

Black Americans receive fewer medical services than their white counterparts. During the past 30 years, persistent barriers to preventative and primary care services influenced not only the quality of life but also the patterns of illness observed among African Americans. Despite the gains associated with the Civil Rights Act of 1964; Medicaid and Medicare legislation of 1965; and the Title VI of the Civil Right Act, which prohibited racial discrimination in any institution receiving federal funds, African Americans continue to have disproportionately large numbers of premature and excess deaths, compared to whites.^{136(p63)}

Thus, McBarnette suggests that institutional racism creates and perpetuates “barriers” to care, and these barriers in turn affect the quality of healthcare available to African-Americans. Furthermore, McBarnette asserts that the quality of care a group experiences influences the “patterns of illness” within that group. Based on this argument, health disparities could be explained, at least in part, by differences in healthcare quality.

Interestingly, the breast cancer literature does not emphasize differences in care between black and white women. In fact, of the twenty-three articles analyzed in this section, only four mention this important health difference.¹³⁷⁻¹⁴⁰ However, each of these four articles forged a strong causal link between the lower quality of healthcare experienced by black women and racial disparities in breast cancer. I already indicated that insurance inequity can affect the quality of healthcare.¹⁴¹ One study took an even stronger position on the issue, arguing that “African-American women are experiencing a different process of care than white women”^{142(p28)} in the current healthcare system.

The researchers' findings in the previous study were also supported by a paper published in 2005. This article enumerated various pathways by which African-American women might experience a different quality of care than white women with regard to breast cancer treatment: "disparities exist in the receipt of definitive primary therapy, conservative therapy, and adjuvant therapy by race, and...these differences result in more frequent recurrences, shorter disease-free survival times, and higher mortality among racial/ethnic minorities."^{143(pS2-7)} For this reason, a third article published the same year argued that future studies should focus specifically on different groups' abilities to obtain adequate healthcare.¹⁴⁴

From the above discussions of healthcare disparities, we see that there are a greater number of articles citing differences in access to care than there are articles identifying differences in the quality of care. What is the reason for this apparent trend? One possible explanation is that access inequities are faceless problems – because they are the product of institutional and policy biases, there is no individual person to blame. In the case of healthcare quality, however, there *is* an individual who could be blamed for the problem: the physician. When researchers cite social inequalities in screening or treatment practices as a possible explanation for healthcare disparities, there is an implicit burden of guilt placed upon the physicians responsible for administering and overseeing these procedures. Perhaps researchers investigating health disparities – many of whom are physicians themselves – are hesitant to criticize the practices of their colleagues, so they underemphasize or underreport differences in the quality of healthcare.

One article, however, did definitively link physicians to quality of care. The study found that doctors were often reluctant to recommend mammography to patients who could not afford the procedure.¹⁴⁵ The researchers conducting the study observed a "strong association between

physician recommendation and mammography use”^{146(p785)} and cited this finding as a potential explanation for the breast cancer disparities. In the above example, there is an observable difference in healthcare quality, and the study directly attributes this difference to the physicians’ biases.

Socioeconomic Inequalities

In the next two sections, we will examine other underlying social inequalities that can also account for the observed breast cancer trends, one of which is socioeconomic inequity. The work of Cox and Wade presented earlier suggests that race and class are interrelated social measures. McBarnette explains how this relationship can affect the health of black Americans:

The construct of socioeconomic status is used frequently and convincingly to identify and describe the position of social groups within the larger society. This construct is critically important in any discussion of the health status of African American women because it is derived from the notion of a stratification of people from lower to higher in terms of access to power, prestige, and property.^{147(p43)}

McBarnette suggests that different groups have varying abilities to access a given set of resources – “power, prestige, and property” – based on their social position. The unequal distribution of resources within a society could in turn create a situation in which different social groups have different health experiences. In other words, there is a complicated relationship between race, socioeconomic status, and health.

Ichiro Kawachi and Bruce Kennedy examine this relationship by addressing the deleterious health effects of having an unequal distribution of economic resources within a society. The researchers state that “the distribution of income in society – in addition to the absolute standard of living – matters for population health.”^{148(p18-19)} They go on to elucidate three mechanisms by which income inequality might produce poor health outcomes.¹⁴⁹ First, they link this inequality with underinvestment in educational and social programs to address health disparities. Second, they present evidence that income inequality undermines social

cohesion by increasing levels of civic mistrust. Finally, they write that this inequality invites social comparisons, which in turn increases individuals' feelings of frustration and psychological distress.

Kawachi and Kennedy's study demonstrates that socioeconomic status matters with regard to health. The question is: How much? Pamela Jackson and David Williams argue that it matters a great deal. They assert that "SES [socioeconomic status] is one of the strongest known determinants of variations in health."^{150(p136)} To support this point, they present data revealing large differences in respondents' health quality by income and by race/ethnicity. Not surprisingly, the lowest income brackets reported the lowest quality of health, and within each income bracket, blacks and Hispanics reported a lower quality of health than whites. What *is* surprising, however, is that the differences in reported health quality were greater between income categories *within* each racial/ethnic group than the differences observed *between* the various groups.¹⁵¹ Although race cannot be reduced to class, this observation suggests that racial differences in health – for example, the observed breast cancer disparities – are partially the product of socioeconomic inequalities.

If we examine breast cancer studies, however, we do not see this message reflected in the literature. Of the twenty-three articles attributing breast cancer disparities to non-genetic factors, only seven studies¹⁵²⁻¹⁵⁸ identified socioeconomic differences as a contributing factor. Mary Anglin offers a possible explanation for this apparent deemphasis. According to her, the studies investigating causes of breast cancer disparities are limited by the information available to them: "Given the failure of cancer registries in the United States to routinely collect socioeconomic data, analyses of the relationship between social class and breast cancer incidence (or mortality) are, at best speculative."^{159(p318)} By failing to collect or include patients' socioeconomic

information, the cancer databases effectively de-emphasize the role socioeconomic inequities play in explaining breast cancer disparities and make it impossible to study the effects of such inequalities.

Environmental Inequalities

In addition to socioeconomic and healthcare inequalities, African-Americans are also more likely to experience environmental inequalities than their white counterparts. This is especially true for black women. According to the *Women of Color Health Data Book*, black women are more likely than white women to have occupations that expose them to environmental hazards and are more likely to live in areas located near toxic waste landfills.¹⁶⁰ For these reasons, African-American women are more likely to be exposed to environmental pollutants and toxins,¹⁶¹ some of which could be carcinogens.

There is evidence that exposure to such compounds could help explain breast cancer disparities. Nancy Krieger explains that certain carcinogens preferentially accumulate in breast tissue: “several investigations have documented biologically significant levels of pesticide metabolites...and other halogenated hydrocarbons...in breast milk.”^{162(p212)} To support this view, she also states that high levels of carcinogens are found in the breast tissue of breast cancer patients, and lower levels of the same substances are observed in the blood.¹⁶³ Krieger also notes that higher concentrations of these substances “occurred not only among poor as compared to affluent women [but also] among black as compared to white women.”^{164(p212)} These findings suggest that social inequities – namely, racial and socioeconomic inequalities – can explain differences in exposure to environmental toxins. This in turn can help account for the observed breast cancer disparities.

Interestingly, none of the twenty-three breast cancer studies analyzed in this section mentioned environmental inequities. This may seem like a surprising result, but given that environmental conditions are difficult to determine – let alone control for – it makes sense that researchers might choose to ignore this factor altogether or incorporate its effects into another, more measurable variable, such as socioeconomic status. However, since the researchers did not always explain their measures of socioeconomic status, it is impossible to know whether or not they considered or accounted for potential environmental differences between the sample groups in their studies.

Cultural Differences

As I mentioned earlier, I am not using the term “cultural differences” to signify an inherent divergence between different groups’ health beliefs and behaviors. Instead, I would argue that a particular group’s beliefs and behaviors are shaped by that group’s social position. For example, the *Women of Color Health Data Book* reports that black women are more likely than white women to report underusing mammography, the primary screening method for breast cancer.¹⁶⁵ But given the limited access to care and the lower quality of care available to African-Americans, this observation is not particularly surprising.

The underutilization of health services could help explain the observed differences in breast cancer mortality, and there is some support for this idea in the breast cancer literature. A 2005 paper found that “black women were more likely than white women to terminate treatment prematurely, and that they were twice as likely to die as white women,”^{166(p6642)} and the study cited this finding as a possible explanation for the observed breast cancer patterns. Furthermore, another article published the same year urged subsequent studies to investigate “disparities in healthcare screening and treatment”^{167(p104)} in the hopes of addressing breast cancer disparities.

Eight of the twenty-three articles examined in this chapter cited cultural differences as a possible explanation for the observed breast cancer trends (see Table 3). One study states in its conclusion that “beliefs [and] attitudes...contribute significant and unique variance to screening behaviors, even when other variables are controlled.”^{168(p2305)} Based on this reasoning, differences in health beliefs could lead to differences in health behaviors, which might help explain the breast cancer disparities.

One potential source of the difference in health beliefs is educational differences. Because white women are likely to be better educated than African-American women, they are more likely to be aware of certain health risks and can take steps to avoid them.^{169,170} The *Women of Color Health Data Book* builds on this point. It verifies the link between education and health beliefs, but goes beyond this connection to suggest that a relationship also exists between health beliefs and eventual health outcomes. The text states, “Blacks generally are less educated about the danger signs and more pessimistic about treatment for cancer than are whites. Both of these facts also contribute to making cancer the terminal disease many blacks conceive it to be.”^{171(p15)} Thus, differences in education, which produce differences in beliefs about cancer, could help explain the observed disparities in breast cancer.

Several articles in the medical literature support this idea. Seven studies found that African-American women were less educated than their white counterparts and cited this difference as an explanation for the observed breast cancer trends (see Table 3). Two articles discussed black women’s decreased knowledge of breast cancer symptoms and risks, citing the population’s limited exposure to cancer information as an explanation for this knowledge deficit.^{172,173} Additionally, one study found that lower education was associated with certain

health beliefs, such as fear of mammography,¹⁷⁴ while another associated lower education with certain health behaviors, namely missing appointments with care providers.¹⁷⁵

Comorbid Conditions

Based on the above discussions, black women are more likely to experience poor health than white women. In other words, because of numerous social disadvantages, black women may start off at a lower baseline of health than their white counterparts if and when they are diagnosed with breast cancer. For this reason, the existence of such comorbid conditions might help explain the variation in breast cancer patterns between the groups. McBarnette writes that African-American women have poorer diets than white women and are more likely to be obese.¹⁷⁶ She then links this dietary difference to cancer disparities: citing the results of a 1989 study¹⁷⁷ linking nutrition to cancer outcomes, McBarnette states that “35% of all cancers may be associated with nutritional causes.”^{178(p52)}

Years of poor nutrition could easily produce comorbid conditions such as obesity, as well as obesity-related diseases such as hypertension and diabetes. The presence of such conditions in breast cancer patients could compromise their health and survival, and in fact, the medical literature supports this hypothesis. Six of the twenty-three articles analyzed in this section cited obesity and its related conditions as possible factors contributing to the observed breast cancer disparities (see Table 3). Four of these articles examined the relationship between obesity and breast cancer, and all of them concluded that the higher prevalence of obesity in African-American women might help explain the differences in cancer patterns between the groups.¹⁷⁹⁻¹⁸²

Two of these four studies also proposed mechanisms by which obesity might affect cancer outcomes. One study examined how obesity might affect survival by promoting cancer growth. It explained that

[a]dipose tissue is an important source of estrogen...Obese women have higher local levels of estrogen in the breast, as well as lower levels of sex hormone-binding globulin (SHBG). The high levels of estrogen from the adipose tissue and low levels of SHBG are thought to result in high levels of active estrogen in obese women, which may promote the growth of tumor cells.^{183(p532-533)}

In contrast, another study examined how obesity could affect treatment administration to negatively impact survival. This paper investigated the possibility that overweight and obese patients experienced chemotherapeutic dose reduction. It concluded that since black women were more likely than white women to be obese, they were more likely to experience this obesity-related dose reduction, which might in turn impact their chances of survival.¹⁸⁴

Two other studies considered the effects of obesity-related comorbid conditions. The first article found that obese black women were more likely than their white counterparts to have hypertension, coronary heart disease, cerebrovascular disease, and diabetes and linked the presence of these four additional comorbidities to lower breast cancer survival rates.¹⁸⁵ Similarly, the second article explored the link between hypertension and diabetes and breast cancer and concluded that “comorbidity was an important predictor of survival and explained important amounts of survival disparity.”^{186(p1771)}

Intersections

Up until this point, I have been discussing each inequality and its consequences separately, but this is not an accurate way of understanding these problems or gauging their effects. In reality, there is a great deal of interaction between the different social inequities, which complicates their effects on health. These interrelationships are especially important when we consider breast cancer disparities. Mary Anglin writes that “[p]roblematic health outcomes and high rates of mortality from breast cancer can be viewed as the end results of social processes operating through the intersections of gender, class, race, ethnicity, and

community.”^{187(p321)} This section will explore what these intersections might look like and how they can affect the health of African-American women.

The first intersection we will consider is the relationship between socioeconomic status and access to care. As discussed earlier, McBarnette identifies the standard hours of operation for health facilities as an indicator of institutional racism in the American healthcare system. This claim is based on the fact that these hours are inconvenient for many women living in low-income communities. Poor women can have a difficult time finding and obtaining transportation to and from the facilities, and they may find it difficult to take off from work to make their health appointments. In this way, socioeconomic inequalities can compound existing healthcare inequities to negatively impact health. There is evidence supporting this relationship in the breast cancer literature. One study found that “patients who reported that they had put off seeing a doctor because of money or transportation problems were also significantly more likely to present with late-stage disease.”^{188(p1803)} Thus, a breast cancer patient’s (in)ability to find transportation to and from treatment facilities can impact her chances of survival.

A person’s socioeconomic status can also have a strong influence on his or her external environment. In the case of African-American women, for example, their limited access to education affects their ability to obtain highly paid, highly skilled jobs. As a result of this occupational disadvantage, many black women are forced to take less skilled jobs (for example, janitorial positions) that can increase their exposure to various toxins (such as industrial cleaning solutions), which can in turn negatively impact their health. McBarnette lists “solvents, dyes, heavy metals, pesticides and herbicides”^{189(p52)} as examples of toxins that low-income black women may come in contact with during the workday. Additionally, low-paying jobs affect an individual’s ability to find housing in pollution- and toxin-free areas. As mentioned previously,

low-income areas are more likely to have high levels of pollution and to be located near toxic waste landfills. For this reason, the low-income housing that many poor black women occupy often has elevated levels of environmental toxins, and these substances can increase their risk of developing breast cancer.

A relationship also exists between health inequalities and “cultural differences” (i.e. health beliefs and behaviors). Given the enormous health barriers African-American women encounter, we might expect these women to be more hesitant to use the healthcare system. As a result of this hesitation, black women might not have as much knowledge about breast cancer risks or available health services, and for this reason, they might also express fear or anxiety when they use these services. In fact, the findings of one breast cancer paper seem to support this hypothesis. The study cited African-American women’s “poorer knowledge...lower estimations of personal risk, embarrassment, and stress”^{190(p2301)} as potential explanations for lower breast cancer screening rates and linked this observation to the observed breast cancer disparities.

A New Model of Disease

In *Backdoor to Eugenics*, Troy Duster discounts the view that all diseases have a strong, clear genetic basis and argues instead that most diseases are multifactorial – in other words, they have multiple causes. I have argued that breast cancer is not only a disease of multiple causes, but also a disease in which these various causes are interrelated. Genetics can certainly play an important role, but disease outcomes are also influenced and shaped by a variety of complex, intersecting social forces. How, then, can we reconcile both the genetic and social models of disease?

For an answer to this question, we will return to Anne Fausto-Sterling’s developmental systems model. We saw in the previous chapter that understanding gene expression requires an

understanding of the complex cellular environment in which a gene is located. Proper gene expression requires the function and coordination of multiple actors and processes. Additionally, we saw in this chapter how an individual's social environment can also influence biological outcomes. Thus, gene expression is subject to the influence of multiple environments. Fausto-Sterling explains that the "activities [of genes] are sandwiched somewhere in the middle of chains and networks of events that integrate organisms with their environment."^{191(p26)} In this way, she writes, "[C]ulture...leaves material imprints on the body."^{192(p26)}

By applying this model, we can understand health disparities in terms of social disadvantage. Racial differences in disease patterns cannot be explained by genes alone because both race and disease are not pure genetic products. To understand health disparities, we must consider the substantial influence social inequities have on health. Different racial groups experience different sets of social forces, and these social forces act on genes and gene systems to shape eventual disease outcomes. In this way, health disparities are social inequalities made visible in human bodies.

Why, then, is there such emphasis placed on race-based genetics in discussions regarding health disparities? Echoing the postgenomic arguments for race-based medicine, Fausto-Sterling writes that "the genetic refashioning of race is in large part a rhetorical move, designed to justify a research enterprise that is fascinating in its own right, but which, were it not for the claims of health relevance, might not receive the massive levels of funding currently available."^{193(p30)} Fausto-Sterling also warns that this "new biology of race diverts our attention from solving problems using solutions we already have at hand."^{194(p30)} Thus, funding race-based genetic research effectively perpetuates health disparities because these projects "divert our attention" from the social processes causing these disparities.

Conclusion: Biology as a Social Product

In this chapter, I have demonstrated how social forces – such as healthcare, socioeconomic, and environmental inequalities – can negatively impact the health of African-American women. These social inequities can also interact with each other to produce patterns of health beliefs and behaviors and comorbid conditions that further compromise the health of black women. Based on these arguments, I outlined a new model of disease causality in which social forces act on genes and gene systems to produce biological outcomes. Thus, health disparities can be understood in terms of social inequalities: different sets of social forces will produce different health outcomes.

In these first three chapters, I have shown that race and biology are both social products. The last chapter will focus on the actual process of producing scientific knowledge, paying close attention to the actions of scientific researchers. I will examine how modern scientists create racial categories, and I will analyze their underlying arguments for these groupings. The process of knowledge production also raises fascinating questions about power and authority. How do researchers use the authority of genetics to argue for genetically defined racial groups? What are the implications of their work? I will attempt to answer these questions in the next chapter.

CHAPTER 4: Constructing the “African-American” Racial Category – A Case Study

Introduction

This chapter will examine several breast cancer studies conducted by Dr. Olufunmilayo I. Olopade, a Nigerian-born breast cancer researcher affiliated with the University of Chicago. Given that Dr. Olopade is an African woman investigating racial health disparities in America, her studies present a unique opportunity to explore how the “African-American” racial category is constructed within a particular subset of breast cancer literature. Dr. Olopade’s website states that she “has a special interest in women of African descent”¹⁹⁵ because they have a greater risk of developing aggressive or early-onset breast cancers. In fact, this idea of “African descent” is repeatedly invoked in her work. In several of the articles discussed in this chapter, Dr. Olopade extrapolates African breast cancer trends onto black women in America, based on the assumption that black Americans have genetic roots in Africa. I will examine her breast cancer studies to investigate how she creates an “African-American” biological identity, and I will evaluate the validity of her arguments. My analysis of this literature draws on theories of group identity from the works of Michel Foucault, Mary Douglas, Bruno Latour, and Michel-Rolph Trouillot.

A Brief Note Regarding Intent

Before launching into my analysis, I would like to clarify my intent in this chapter. My goal is not to attack Dr. Olopade but to examine how the African-American category is constructed in a subset of breast cancer literature. I believe that Dr. Olopade and her colleagues are conducting important, worthwhile research, and I have no question that their efforts are well intentioned. However, any argument that divorces race from its social, political, and

historical context to present the view that it is an inherent, genetically-based quality has racist implications, and it is these implications that I would like to identify and address.

Biopower: The Coproduction of Dual Identities

In *Society Must Be Defended*, Michel Foucault argues that a major shift in the nature of political power took place during the nineteenth century in Europe. According to Foucault, sovereign power was previously manifested as the right to “take life or let live,”^{196(p241)} but this power later became complimented by another right, the right to “make life and let die.”^{197(p241)} This shift embodied a fundamental change in the way political powers conceived of and controlled individuals. While techniques of power in the seventeenth and eighteenth centuries were focused on controlling individual bodies, in the nineteenth century, the focus shifted to controlling people *en masse*. Thus, in the mind of the modern State, a person possesses two simultaneous identities: 1) an individual identity and 2) a group identity, as belonging to an entire species.¹⁹⁸ Foucault terms the power derived from manipulating this dual identity “biopower.”

Foucault’s notion of biopower can be applied to medical research to understand racial classification. As noted earlier, after the policy changes of the 1980s and 1990s, the NIH mandated that researchers design their projects to investigate potential differences by race/ethnicity, sex/gender, age, etc. As a result of these requirements, participants in medical studies came to be identified by the racial or ethnic groupings that were chosen by researchers. In categorizing their subjects in this manner, medical researchers effectively projected a specific group identity onto each individual research subject.

We can also use Foucault to understand why such a classification system might be problematic. He writes that “the first function of racism...is to fragment, to create caesuras

within the biological continuum.”^{199(p255)} Foucault’s statement captures the danger of race-based genetics. Classifying people into discrete genetic groups ignores the overall distribution of genetic variance across populations that constitutes the “biological continuum.” Furthermore, fracturing this “biological continuum” undermines the staggering amount of genetic commonality between groups.

Foucault goes on to suggest that scientists derive power from dividing populations into different groups. He writes, “Medicine is a power-knowledge that can be applied to both the body and the population, both organism and biological processes, and it will therefore have both disciplinary effects and regulatory effects.”^{200(p252)} The phrase “power-knowledge” is worth exploring. Perhaps Foucault is suggesting that medicine is both the product of knowledge, as well as a medium for producing it. If we consider race, for example, race-based medical knowledge draws on social and historical conceptions of race, which would make it a product of social and historical processes. Additionally, race-based medicine can reinforce principles of racial group membership. It can do this by either manipulating individual identity to classify subjects into racial groups, or by manipulating group identity to distinguish trends within and between racial groups.

The “regulatory effects” of medicine are also worth discussing. According to Foucault, during the nineteenth century, governments began trying to control people as a group instead of trying to control each person individually. He explains that this shift required a change in control techniques: whereas individual control required disciplinary mechanisms, group control requires regulatory mechanisms. Foucault states that “regulatory mechanisms must be established to establish an equilibrium, maintain an average, establish a sort of homeostasis, and compensate for variations within [a] general population.”^{201(p246)} This idea can be directly applied to group

identities in medical literature. The creation of a biological group identity is based on two assumptions: 1) there are enough significant genetic differences between groups to warrant different group designations and 2) variations within the same group are unimportant or of lesser importance than the differences between groups. The latter assumption basically restates Foucault's point in the passage above: without establishing genetic "homeostasis" within a certain population, a researcher could not classify that population as a uniform racial group.

Bounded Bodies and Racial Purity

Like Foucault, Mary Douglas emphasizes the importance of internal consistency in group construction. Although her book *Purity and Danger* is an anthropological study of "primitive" religions, it presents several concepts that we can apply to ideas about racial group construction in biomedical literature. Douglas also discusses the creation of group identities, although she frames her discussion in terms of purity versus pollution, rather than homeostasis versus variation. She writes, "Where there is no differentiation there is no defilement."^{202(p161)} Applying Douglas' theory to race, we can conclude that a racially "pure" group will not display observable differences between its members. This theory is consistent with Foucault's ideas presented earlier.

Foucault suggests that homogeneity is a necessary condition for the construction of group identities, but he does not offer any ideas about how different groups might be differentiated from one another. In a way, Douglas picks up where he left off, as she presents several theories about the creation of boundaries. She writes,

It is part of our human condition to long for hard lines and clear concepts. When we have them we have to either face the fact that some realities elude them or else blind ourselves to the inadequacy of the concepts.

The final paradox of the search for purity is that it is an attempt to force experience into logical categories of non-contradiction. But experience is not amenable and those who make the attempt find themselves led into contradiction.^{203(p163)}

Here, Douglas suggests that boundaries – such as those between ethnic groups – do not exist in nature, waiting to be discovered, but rather are actively produced by people.

If we combine this idea of active racial group production (versus passive discovery) with Douglas' and Foucault's theories about group identity, we can generate a hypothesis about the creation and maintenance of racial groups. Researchers construct these groups by separating and classifying people based on proposed biological commonalities, and they maintain these group identities once they are established by erecting an impermeable boundary around each group. Defining this boundary involves excluding other individuals based on perceived biological differences.

Douglas discusses the implications of boundary transgression in her book. She writes, “Danger lies in transitional states, simply because transition is neither one state nor the next, it is indefinable. The person who [passes] from one to another is...in danger and emanates danger to others.”^{204(p97)} This idea can be applied to the construction of racial categories in biomedical literature. To researchers trying to classify their subjects into racial groups, people of mixed ancestry pose a significant classificatory problem. Because they do not neatly fall into any one category, they represent a dangerous, liminal group, one that threatens the existing system of classification. For example, we saw how the “mulatto” category challenged U.S. census-takers in the nineteenth century. The fundamental classificatory problem embodied by this group of people forced the census to constantly redefine the group's membership criteria.

Today, black Americans can also be considered a genetically liminal group. They may have genetic roots in Africa, but because of the limitations of currently available genetic tests, it is impossible to identify or emphasize any one genealogical line over the others with scientific certainty. One limitation of the available tests is their inability to provide a full genetic family

portrait. They can only identify a single paternal lineage (fathers' fathers) through the Y-chromosome and a single maternal lineage (mothers' mothers) through mitochondrial DNA. If we go back five generations, then, current genetic tests would only offer information about two of thirty-two possible ancestors.

Another equally significant limitation comes from the “control” ethnic populations sampled by these tests. It is misleading, for example, to identify a Zulu ancestor in an African-American’s genetic family tree. All populations change over time, so someone who identifies as Zulu now (the aforementioned “control” population) may not have been classified as Zulu in the past. Furthermore, the Zulu designation is problematic because it is a historical construction. Robert Ross argues that “political allegiance lay at the basis of ethnicity identity”^{205(p34)} in nineteenth-century South Africa, and these “polities to which South Africans owed allegiance were almost all of nineteenth-century creation.”^{206(p34)} For this reason, it does not make sense to argue for genetic ancestry based on historically-constructed group identities.

Emphasizing Similarity and Erasing Difference: Silencing vs. Amplification and Visibility vs. Invisibility

So far, we have established that 1) boundaries are erected around groups by excluding those with meaningful biological differences, 2) that the bounded groups themselves are united by meaningful biological similarities between their members, and 3) that any biological dissimilarities within a particular group must be insignificant compared to the similarities within the group and the differences between other groups. We have not yet discussed how unifying commonalities are determined to be significant, or how differences within groups might be erased. I am using the word “erase” here quite deliberately. I want to emphasize that differences between individuals in a group are not passively ignored but instead are actively erased. In other

words, there is a conscious process involved in creating group identities. We can understand this process in terms of silencing versus amplification and visibility versus invisibility.

Michel-Rolph Trouillot describes how the function of silencing is involved in the production of history. He argues that “any historical narrative is a particular bundle of silences.”^{207(p27)} We can apply this concept to understanding how racial categories are constructed in medical literature. The differences between individuals within a perceived racial group are silenced to preserve the “purity” (to borrow Mary Douglas’ idea) of the group. I would also argue that there is a corresponding amplification process in the construction of group identities. In the case of amplification, similarities between members of a particular racial group are overemphasized to argue that “homeostasis” (to borrow Foucault’s idea) is achieved within the group. In both cases, we observe the manipulation of both group and individual identities, invoking Foucault’s principles of biopower.

Bruno Latour’s work echoes Trouillot’s ideas about emphasis and de-emphasis and can also be applied to group construction. He discusses power in terms of visibility versus invisibility, and his appeal to the sense of vision seems especially appropriate when dealing with racial group identification. In his analysis of Pasteur’s laboratory, he writes,

[The scientists] are expert inside their own walls at setting up trials and instruments so that the invisible actors—which they call microbes—show their moves and development in pictures so clear even a child would see them. The invisible becomes visible and the “thing” becomes a written trace they can read at will as if it were a text... Then everything [people] have to talk about is not only visible but also readable, and can be easily pointed at by a few [scientists] who by doing this dominate.^{208(p270-271)}

Latour argues that power is enacted through the process of making something that was formerly invisible visible. He believes that this power is derived from the act of interpreting the new information – those who can “read” the now-visible elements are the ones who can “dominate” this information and shape the new knowledge.

This idea applies to racial categorization: the differences between different groups are made visible by researchers, while the differences between individuals within a group are downplayed or made invisible. Furthermore, by creating racial groups and then classifying their research subjects into these groups, medical researchers provide biological validation for social categories. This is because, as Latour argues, “sciences are one of the most convincing tools to persuade others of who they are.”^{209(p259)} In this way, biologically-based (mis)understandings of race can, over time, come to be seen as fact.

As we will see, Dr. Olopade’s articles illustrate the principles of racial group construction discussed in the previous sections. Although they never mention how racial groups are identified, her studies consistently treat black Americans as a discrete, uniform biological group. By making “unique” and “founder” mutations visible within the genetic makeup of the two groups, she amplifies genetic commonalities between African-Americans and Africans to argue that black Americans have genetic roots in Africa. At the same time, Dr. Olopade silences the non-African ancestry of black Americans by making genetic differences between blacks and whites visible and by downplaying the complexity of tracking racial ancestry. Finally, by emphasizing a gene-driven understanding of breast cancer disparities, she also renders the influence of social forces invisible.

Defining Racial Categories: Group Descriptions and Geographic Labels

Understanding racial group construction in Dr. Olopade’s articles requires analyzing the language used to describe various populations of interest. If we chronologically survey her twelve breast cancer articles, we observe significant changes in the way they describe the sample populations (see Table 4). With the exception of a 2003 article discussing “[r]acial and ethnic disparities”^{210(p47)} in breast cancer survival, there are only brief, scattered mentions of “race” and

“ethnicity” in the literature until 2005. However, in all four articles published in 2005, as well as in another article published in 2007, Dr. Olopade uses “race” and “ethnic group” both frequently and interchangeably, which suggests that there is no difference between the two terms.

Table 4: Summary of Dr. Olopade’s Breast Cancer Articles.

| Authors | Year | Major Explanation(s) for Breast Cancer Disparities | Group Description: Race or Ethnicity? | Labels Used to Describe White and Black Americans | Geographic Descent Invoked? | Method of Racial Classification |
|---|------|--|---------------------------------------|---|-----------------------------|---------------------------------|
| Gao Q, Neuhausen S, Cummings S, Luce M, Olopade O | 1997 | Genetic | None | “Caucasian,” “Black,” “African American” | Yes | Method not stated |
| Gao Q, Tomlinson G, Das S, et al. | 2000 | Genetic | Mentions ethnicity | “Caucasian,” “African American” | Yes | Method not stated |
| Gao Q, Adebamowo C, Fackenthal J, et al. | 2000 | Genetic | None | “White,” “Black,” “African American” | Yes | Method not stated |
| Olopade O, Fackenthal J, Dunston G, Tainsky M, Collins F, Whitefield-Broome C | 2003 | Genetic | Mentions ethnicity | “White,” “African American” | Yes | Method not stated |
| Ademuyiwa F, Olopade O | 2003 | Genetic | Mostly race, mentions ethnicity | “White,” “Caucasian,” “Black,” “African American” | Yes | Method not stated |
| Polite B, Olopade O | 2005 | Genetic and non-genetic | Both | “White,” “African American” | No | Method not stated |
| McClintock M, Conzen S, Gehlert S, Masi C, Olopade F | 2005 | Genetic and non-genetic | Both | “White,” “Black” | Yes | Method not stated |
| Masi C, Olopade O | 2005 | Genetic and non-genetic | Both | “White,” “African-American” | No | Method not stated |

| | | | | | | |
|--|------|---------|---------------|--|-----|-------------------|
| Nanda R, Schumm L, Fackenthal J, et al. | 2005 | Genetic | Both | “White,” “African American” | Yes | Self-identified |
| Olopade O, Schwatsman n G, Saijo N, Thomas C | 2006 | N/A | Mentions both | N/A | Yes | N/A |
| Bell D, Kim S, Godwin A, et al. | 2007 | Genetic | Ethnicity | “White,” “Caucasian,” “African-American” | Yes | Method not stated |
| Huo D, Kim H, Adebamowo C, et al. | 2007 | Genetic | Both | “Caucasian,” “African American” | Yes | Method not stated |

Another trend we can observe in Table 4 is the regular invocation of geographic descent. In all but two breast cancer studies, geographic ancestry is used to describe black and white populations. As mentioned in the introduction to this chapter, Dr. Olopade describes herself as being interested in “women of African descent.” This phrasing emphasizes the African roots of the black American population, while at the same time obscuring other non-African ancestries. This idea of “African descent” resurfaces in her breast cancer articles. In the earliest study published, Dr. Olopade describes African-Americans as “blacks of African descent.”^{211(p1236)} This African descent or “ancestry” is also invoked in another breast cancer study published in 2000,²¹² as well as in studies published in 2003²¹³ and 2005²¹⁴.

Dr. Olopade also uses geographic ancestry to subdivide white sample populations. For the most part, she considers the white/Caucasian group to be a homogenous group “of European ancestry,”²¹⁵⁻²¹⁸ but she occasionally divides this larger group into several smaller sub-populations based on their country of origin. In an article published in 2005,²¹⁹ as well as in another study published in 2007,²²⁰ she refers to subsets of the white population by their nationality, or, occasionally, their general geographic region (e.g. “Northern European”). This

choice has implications for group classification. Using geographic descriptors to define groups suggests that race and ethnicity have social and political components. In other words, geographically-based racial categories can be used to represent groups experiencing similar social and political forces. However, this is not the primary use of geographic descriptors in Dr. Olopade's work. Instead, she argues that geography can be used to identify and define genetically similar populations. We will explore this application of geography in the next section.

There is one particular subset of the white population that is consistently separated from the rest of the group by its religious affiliation, and that group is the Ashkenazi Jews. In three different studies,²²¹⁻²²³ Dr. Olopade defines subjects "of Ashkenazi Jewish descent" as distinct from the white population. This grouping choice is interesting because it is defined by socio-cultural characteristics, rather than by geographic origins. Even though this second classification choice also suggests that race has a social component, she argues that Ashkenazi Jews are genetically distinct from the general white population.

Classificatory Ambiguities Revisited

Before moving on, it is important to address the layers of ambiguity present in Dr. Olopade's methods of racial classification. As we have seen with the previously discussed breast cancer literature, her studies rarely address the question of how subjects are categorized. Of the twelve studies analyzed in this chapter, only one article explicitly mentioned its method of identification (see Table 4). Even in this study, which used self-identification to classify its population, it was not entirely clear how the subjects identified themselves: Were they supplied racial categories (the check-a-box method), or were they given the opportunity to write in a group? If the subjects were able to write in a group affiliation, were they influenced by census

categories or other common classificatory labels? And if and when subjects wrote in a category, did the researchers then regroup their varied responses into the five populations (“white, Ashkenazi Jewish, African American, Hispanic, and Asian”^{224(p1925)}) reported in the study? The article did not address any of these questions.

An additional layer of ambiguity becomes apparent when we consider the definitional inconsistencies in Dr. Olopade’s method of group categorization. For example, in the 2005 study discussed above, the “African American” and “Asian” groups seem to be constructed along geographical lines, while the “Ashkenazi Jewish” and “Hispanic” groups seem to be created along religious and linguistic lines, respectively. On the other hand, the “white” population is defined against the Ashkenazi Jewish and Hispanic groups: these subjects are described as “non-Hispanic, non-Jewish whites.”^{225(p1926)} Thus, members of the “white” group are not united or identified by strong commonalities, but rather are defined by their lack of shared features with other populations.

Interestingly, one article published in 2006 calls for more precise definition of individuals participating in cancer research studies. But despite its noble aims, this article, too, handles race questionably. Dr. Olopade writes that “certain identifiable patient groups may help molecular cancer epidemiologists elucidate the complex relationships...of treatment response.”^{226(p2135)} However, she does not explain what makes these patient groups “identifiable” – in other words, she does not offer examples of how to define or identify different racial groups.

Locating Origins and the Manipulation of Geography

As discussed in the previous section, one of the ways Dr. Olopade constructs group identity is through shared geography. She uses continents as stand-ins for racial groups – as in the case of “Asians,” “Europeans,” and “African Americans” – and she also subdivides the

“white” population into smaller groups based on her subjects’ nationalities. However, these systems of classification prove to be fundamentally flawed when they are used to represent genetically distinct groups. In the case of continental identities, there is so much variation *within* these groups that it does not make sense to define each one as a discrete population. Asia, for example, is a large continent populated by many groups of people, so the group descriptor “Asian” is not particularly informative in scientific studies. Additionally, as noted in the discussion regarding the work of Risch and Rosenberg, modern continental groupings recall nineteenth-century theories about the existence of five continentally-derived races. Thus, retaining these terms appears to lend modern scientific validity to past centuries’ race science.

Similarly, several arguments can be made against biological groupings based on nationality. Countries are often made up of subpopulations, which would contribute enough variation to the national gene pool to defy the creation of a nation-based genetic grouping. For example, the United States’ diverse ethnic and racial makeup would make it difficult to create a single biological group identity based on nationality. Moreover, countries pose an additional problem because they represent a political and social grouping. Nature does not draw borders around a country – people do. For this reason, it is difficult to argue that national identities will map neatly onto genetic ones.

In spite of these problems, Dr. Olopade’s work manipulates geographical groupings to create gene-based racial identities. One of the ways she accomplishes this is by emphasizing or making visible the African ancestry of her black American subjects. In the abstract of a paper published in 2000, she writes,

In populations in the African tropics, breast cancer has been considered to be a rare disease, predominantly afflicting young women. However, the International Agency of Research on Cancer Bulletins and surveys in seven African countries have shown that breast cancer incidence increased from 15.3 per 100,000 in 1976 to 33.6 per 100,000 in 1998...multiple studies in the United States (US) have also documented a higher breast

cancer incidence and death rate in pre-menopausal Black women than in non-Hispanic whites...it is likely that the shared genetic background of Africans and US Blacks contributes to the greater susceptibility to early onset breast cancer in both groups.^{227(p192)}

Thus, Dr. Olopade uses similar breast cancer patterns observed in Africans and black Americans to argue that there is a “shared genetic background” between these populations.

The populations in this study seem to be structured along geographic lines, as suggested by the phrases “populations in the African tropics” and “surveys in seven African countries.” However, as we saw earlier, using a continent to define a genetically distinct population is problematic. Like Asia, Africa is a large continent populated by many different groups of people, and as such, we would expect a considerable amount of genetic diversity within the continent. In fact, there is evidence that large genetic variation exists: as Sarah Tishkoff and Kenneth Kidd explain, “Most studies of genetic variation in autosomes, the X chromosome, and mtDNA [mitochondrial DNA], using many types of markers, show higher levels of genetic variation in African populations than in non-African populations.”^{228(pS22)}

In spite of this variation, Dr. Olopade uses the descriptors “Nigerian” and “African” interchangeably in the 2000 article. Furthermore, she uses Nigerian breast cancer trends to draw conclusions about African women. These choices suggest that Nigerians are representative of Africans as a group, which in turn implies that a “model” African body exists. In this way, Dr. Olopade echoes the continentally-based racial categories presented by Risch, Rosenberg, and, ultimately, Blumenbach.

In addition to extrapolating trends from a sample Nigerian population onto an imaginary, uniform “African” population, Dr. Olopade also draws conclusions about black American breast cancer trends based on the disease patterns of this imagined “African” population. In the 2000 article discussed above, she writes,

Studies of...women [in the African diaspora] may reveal ancient mutations of African origin analogous to ancient mutations in other populations. One such ancient mutation...has been identified in families of West African ancestry from Florida, Washington DC, South Carolina, Bahamas, and Ivory Coast...The collection of information about the spectrum of *BRCA1* and *BRCA2* mutations in African women could reveal genetic components of breast cancer common to all Blacks in the African diaspora.^{229(p192-193)}

Dr. Olopade suggests that certain gene mutations may be present in similar patterns in all groups descended from a single “ancient” African population. Based on this shared genetic ancestry, she argues that gene mutation patterns in present-day African women represent a source of genetic information about breast cancer in black Americans.

However, when she invokes the common ancestry of black Americans and West Africans, there is a confusion of both time and location in her logic. In terms of location, for example, while the ancestors of African-Americans may have been shipped as slaves to America from the West African coast, the cultural groups they originally belonged to may not have been from that region. Additionally, another source of geographical confusion is the diverse family trees possessed by the American descendents of these African slaves. For example, we know from the discussion of the “mulatto” category that after African slaves arrived in America, there was a great deal of mixing between this group and the white group. But there was also intermixing between the different African cultural groups:

[H]istorians have cited statements by European and American observers at various times and places in Africa and the Americas that in order to discourage slave revolts, communication among new Africans was suppressed by separating and fractionalizing the various African ethnicities during their transport on Atlantic slave trade voyages as well as after they arrived in the Americas.^{230(p55)}

These observations, coupled with the aforementioned limitations of current genetic tests, would suggest that it does not make sense to emphasize black Americans’ West African roots while obscuring other possible ancestral lineages.

In terms of temporal confusion, Dr. Olopade's claim that African-American populations have definitive West African ancestry is based on the assumption that West African populations have been genetically static over the centuries. As I explained in the section about genetic ancestry tests, this assumption is invalid because all populations change over time: someone who might be considered West African now may not have been classified as such in the past. By extension, the extrapolation of data from a current West African population onto a modern black American population cannot be considered scientifically valid.

I mentioned before that Dr. Olopade uses geographic markers, such as country of origin, to subdivide the white populations in her studies. She also indirectly invokes geographic origins whenever she interchanges the "white" racial descriptor with the term "Caucasian," which she does in several of her breast cancer papers (see Table 4). The "Caucasian" racial category is a social and historical construction that was coined by Blumenbach in the late eighteenth century. Although the term is now casually used to describe white people, historically, it was originally used to designate people belonging to a specific geographic region. Blumenbach created the term to describe what he believed to be the most beautiful "race" of people, the Georgians, who lived in the Caucasus mountain region.²³¹ Clearly, the word "Caucasian" has deeply-embedded ethnocentric, racist connotations, and for this reason, Dr. Olopade's consistent use of the term is problematic.

Uniting Racial Groups Through Common Mutations: Invoking the Authority of Genetics

In her breast cancer articles, Dr. Olopade explicitly argues that data from African populations can be used to draw conclusions about black Americans. In this section, I will investigate how she constructs this argument by examining her supporting evidence, paying particular attention to the way the discourse of genetics is applied. To do this, I will first show

how the authority of genetics is used to lend credibility to the creation of biologically-based racial groups. I will then analyze how common genetic mutations – namely founder mutations and unique mutations – are used to construct a genetically-based African-American racial group.

As we have seen in the previous chapters, medical researchers often ignore or overlook their role as producers of racial categories. Similar to Risch and Rosenberg, Dr. Olopade uses the authority of genetics to argue that racial groups have a biological basis. For example, in a 2005 article, she writes,

While numerous theories have been proposed to explain [the difference in breast cancer incidence between African-American women and white women], including age at menarche, time of first delivery, parity, sociodemographic factors, body mass index, and underlying genetic difference, none are completely satisfactory and more research is needed in this area.^{232(pS167)}

Dr. Olopade suggests that genetic differences between African-Americans and whites exist, and furthermore, she implies that these differences represent a valid scientific “theory” for explaining the observed breast cancer disparities.

To construct a uniform, genetically-based African-American category, Dr. Olopade applies theories related to mutational commonality. In seven of the twelve breast cancer articles,²³³⁻²³⁹ Dr. Olopade repeatedly mentions “founder mutations.” These are mutations that appear in one or more “founding members” of a certain population and are subsequently propagated within this population through inbreeding. The term invokes a common ancestry. In these seven articles, Dr. Olopade suggests that certain mutations in *BRCA1* and *BRCA2*, which appear to be more common in African-American populations, are actually founder mutations that can be traced back to West African populations. For example, in one article, she states that “[i]nherited germline mutations in *BRCA1* may reflect a genetic founder effect and create disparities in breast cancer among young Black women in the United States and Nigeria.”^{240(p33)}

This claim, which is representative of the statements made about founder mutations in the other papers, suggests that *BRCA1* founder mutations can be used to explain breast cancer disparities.

Dr. Olopade's argument about founder mutations is problematic for several reasons. First, mutational commonalities cannot always be attributed to founder mutations. In fact, they can also arise through "independent mutational events,"^{241(p241)} which she admits could be a possibility in another paper. Second, the use of founder mutations is complicated by her geographic groupings. As I explained previously, these groupings can confuse both time and location, and they also ignore the limitations of current genealogical testing.

In addition to highlighting potential founder mutations, Dr. Olopade also emphasizes the importance of "unique" breast cancer mutations in her work. In five articles,²⁴²⁻²⁴⁶ she argues that certain genes are only present in populations "of African descent," and in three of these studies, she expresses certainty that the specific mutations she has isolated are unique to people "of African ancestry." For example, in one of these three articles, she writes, "[W]e found that three...mutations...were novel. The majority of the cases listed in the database are of northern European ancestry, and these mutations may not have been seen previously because they are exclusive to African Americans."^{247(p1234)} Similarly, in a second article, she writes, "[I]t is clear that African Americans exhibit a unique spectrum of deleterious *BRCA1* mutations and variations in comparison to other ethnic groups."^{248(p239)} In the third article, she even suggests that two variant alleles in the *UGT1A1* gene "can be used as markers for African ancestry,"²⁴⁹ based on their higher prevalence in indigenous African populations and black American populations. By claiming that certain genetic mutations are unique to black Americans, Dr. Olopade is able to construct a genetically uniform and genetically distinct African-American racial category.

However, as with the arguments about founder mutations, there are a number of problems associated with her logic. For one, large-scale studies have not yet been conducted to investigate the prevalence of breast cancer genes in non-white, non-black, non-American populations. Without analyzing a much larger and more diverse set of data, it would be difficult to prove conclusively that certain mutations are “unique” to any given population. It is also important to recognize that the different sample populations are predetermined – they are defined by the researchers conducting the study.

Silencing Social Explanations: The Invisibility of Socioeconomic and Environmental Factors

I argued in the previous section that Dr. Olopade’s articles draw on the authority of genetics to argue for gene-based racial group definition, and, consequently, genetic explanations for breast cancer disparities are emphasized in this subset of the literature. As it turns out, her articles also actively de-emphasize the importance of social explanations for the observed disease patterns. Of the twelve studies analyzed in this section, nine did not address the social forces that can influence breast cancer outcomes (see Table 4). Although three articles do carefully consider the influence of socioeconomic and environmental factors, even these papers place more emphasis on genetic components of disease causality. This section will examine the treatment of both genetic and non-genetic factors in each of the three studies.

The first article, titled “Breast Cancer and Race,” is roughly divided into two equal parts. The first considers various genetic causes of breast cancer, while the other examines the role of social factors. The placement of the genetic explanations before the social ones might suggest that genetic factors are more important in producing disease outcomes. This idea is also supported by a statement from the end of the genetics section: “[A]lthough no specific genetic abnormality has been found that preferentially predisposes African American women toward

more advanced, aggressive cancers...[this field of research] is an area that is still in its infancy but will likely yield clinically relevant results in the near future.”^{250(p172)} Thus, even though biologically meaningful genetic differences between these groups have not yet been identified, Dr. Olopade still asserts that they exist.

Similar themes are present in the second article, though they are less obvious. In “Mammary Cancer and Social Interactions,” Dr. Olopade contends that premenopausal black women’s disproportionately high mortality from breast cancer is partly caused by psychosocial predispose mechanisms. She writes that “[l]iving as a minority...can also entail a daily accumulation of stressors, loneliness, and social isolation”^{251(p34)} – conditions that she links to increased breast cancer risk. This paper highlights the importance of an individual’s social environment in breast cancer development and, perhaps, downplays the role of genetically-based race. As additional support for this idea, Dr. Olopade argues that “one cannot reduce the understanding of behavior or physical traits to genetic information.”^{252(p32)}

Later in the paper, however, she seems to imply that race does have a genetic component. She writes, “Indigenous tribes of the island of Taiwan are genetically homogenous within a tribe but diversified among them. Moreover, they have little genetic relationship to the Han of Mainland China.”^{253(p32)} Dr. Olopade’s assertion that the groups “have little genetic relationship” to each other obscures the fact that the indigenous Taiwanese people and the people in Mainland China share 99.99 percent of their DNA. Furthermore, immediately preceding this passage, she states that racial disparities in breast cancer likely arise “from the reciprocal interplay of culture and biology that defines ethnicity and race.”^{254(p32)} While it is not clear how “race” and “ethnicity” are being defined in this sentence, given the information immediately following it, it

is possible that Dr. Olopade is suggesting that culture produces ethnicity, while biology produces race.

The third article, titled “Racial and Ethnic Disparities in Breast Cancer,” builds on the themes presented in the previous article. The conjunction “and” in the title suggests that there is a meaningful difference between race and ethnicity, and the way each term is treated in the paper supports this idea. The article is divided into three sections: the first discusses social forces that can influence breast cancer outcomes, the second examines individual factors, and the last considers cellular mechanisms.²⁵⁵ Interestingly, the group descriptors change from section to section. Whereas in the first section, Dr. Olopade primarily uses the term “ethnicity” to describe the subject groupings, in the next section, she uses “race” more frequently. In the final section, “ethnicity” is only occasionally used, and “race” becomes the primary group descriptor. Given the content discussed in each section, these language choices suggest that race is had a genetic component, whereas ethnicity is the product of social forces.

Conclusion: Race as a Social Product

In this chapter, I have presented a case study for how racial categories are actively produced in medical literature. I drew from the works of Foucault, Douglas, Trouillot, and Latour to generate a model for racial group construction. In this model, the members of a group must be united by visible, meaningful commonalities, and any dissimilarity within the group must also be obscured. Furthermore, boundaries must be erected around these groups to exclude individuals who are determined to be significantly different. Of course, the meaning and significance of these differences and similarities are not discovered but rather defined. In the medical literature, these definitional decisions are made by researchers investigating questions of race in medicine.

We saw in Dr. Olopade's studies how the discourse of genetics can be applied to racial group construction. Drawing on the authority of genetics, she constructed a unified African-American racial group by identifying common genetic mutations. She reified this racial construction by emphasizing the group's African ancestry and highlighting genetic commonalities between black Americans and modern Africans. At the same time, Dr. Olopade effectively erased other, non-African lineages and erected a genetic boundary around the group by arguing that certain mutations were "exclusive" to the black American population. However, her arguments collapse when we consider the limitations of genetic ancestry testing, the problems posed by geographically-based genetic groupings, and the social and genetic complexity of African-American racial identity.

CONCLUSION: The Bigger Picture

I chose to divide up the thirty-six breast cancer studies I analyzed in the second and third chapters because I wanted to conduct a detailed textual analysis of each subset of the literature. However, isolating these two discussions obscured the fact that both sets of articles were produced simultaneously, which in turn made it difficult to discuss any overall trends. To address this omission, I would like to devote part of my conclusion to an examination of some of these trends.

Figure 1: Comparison of Breast Cancer Articles by Year.

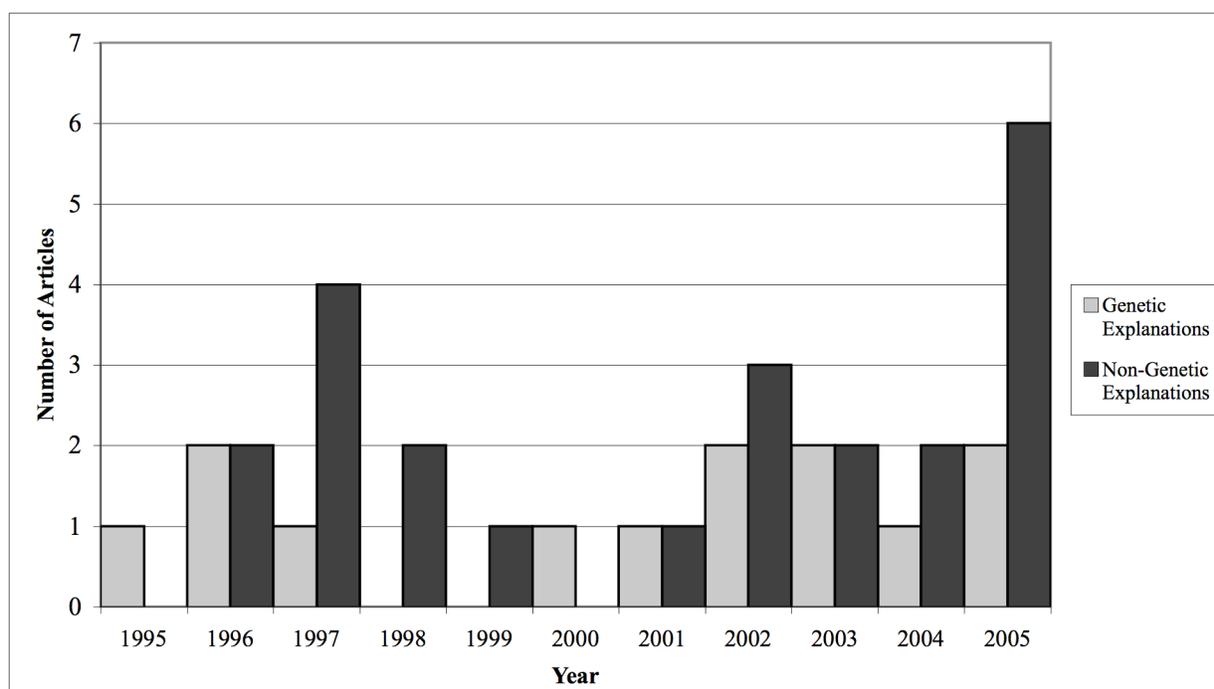


Figure 1 compares the two categories of breast cancer articles by year. We can see that the distribution of articles proposing genetic explanations for breast cancer disparities is skewed left, which suggests that interest in this area of research has increased over time. This trend is not

surprising, given the health policy changes of the 1980s and 1990s, as well as the scientific community's shift to more genetics-driven research during the same period.

In contrast, research interest in non-genetic explanations appears to have fluctuated in the same period, although there is a notable peak in 2005. This peak might be explained by the fact that questions regarding the use of race in medicine have been generating a great deal of attention in recent years, especially given the controversies surrounding race-based genetic projects, such as the HGDP, and race-based drug marketing, as in the case of BiDil. This peak could also indicate that research tides are turning. Perhaps gene-based models of disease and race are falling out of favor with medical researchers, and perhaps health disparities are increasingly being understood as the product of complex, intertwined social forces. Only time will tell if this is truly the case.

There is one other major trend I would like to highlight, and that is that there is a need for greater transparency and clarity with regard to the use of race in medicine. Researchers should always define their use of the terms "race," "ethnicity," and their derivatives in their studies, and this recommendation applies to both genetic and non-genetic investigations into health disparities. Both subsets of the literature should acknowledge and analyze the various social contexts of race, disease, and health. By this, I mean that researchers should recognize that it is impossible to divorce biological outcomes from the contexts in which they are produced. Additionally, because they are using race to classify subjects into groups, medical researchers should explain in detail how they determined their categories and how they grouped their subjects into these categories. The omission of this information presents race as a self-evident marker, which, as I have argued, is certainly not the case.

These recommendations are part of a larger call for change. All knowledge – including scientific knowledge – is produced rather than discovered, and as such, the knowledge produced will be shaped by the people involved in its production. For this reason, medical researchers should recognize that scientific knowledge cannot be separated from the society in which it is produced, and they should address the fact that they and their work are grounded in this social sphere. This acknowledgement is particularly important when dealing with the use of race in medicine. When researchers use racial categories in their work, these groupings are often based on loose, informal understandings of race. Since these popular definitions do not necessarily have a valid scientific basis, this action can be particularly problematic. Because science – particularly genetics – has a large and formidable sphere of influence, unclear scientific work could potentially reinforce flawed understandings of race. In other words, scientists should be aware of the incredible power and authority they wield, and they should take responsibility for the social implications of their work.

I want to take a moment to clarify that I do not think that the individual researchers conducting and publishing these studies are racists. In fact, I believe that these men and women are extremely well intentioned, and I applaud their efforts to address breast cancer disparities by attempting to identify its causes. On the other hand, I do think that their work could easily have racist implications because of the consistently ambiguous use of race. Future studies into breast cancer and other health disparities can avoid these implications by clarifying what they mean by “race,” “ethnicity,” and their derivatives.

Breast cancer is a political issue. Quality health should be a right – not a privilege. Sadly, existing social forces create a system in which black women experience a diminished quality of

health compared to their white counterparts. In this sense, the scientific studies focused on genetic explanations for health disparities do a great disservice to African-American women. Instead of discussing the social forces behind health disparities, these studies shift attention to the “naked gene.” In doing so, they divorce race from its social context, imply that race has a genetic basis, and insist that this genetic basis is responsible for observed disease patterns – in essence, they argue for an incomplete, inaccurate model of disease causality. To eliminate breast cancer disparities, we must address the various social forces that collectively disempower African-American women. More specifically, we will have to address the structural and institutional racism that, together, create a system that privileges the health of certain groups over others.

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