ALSO BY DOROTHY ROBERTS

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Shattered Bonds: The Color of Child Welfare

FATAL INVENTION

How Science, Politics, and Big Business Re-create Race in the Twenty-first Century

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The Allure of Race in Biomedical Research

By the mid-1980s, researchers had again turned to the idea that racial identity was a useful way to distinguish human bodies for medical purposes. Ironically, as sociologist Steven Epstein has chronicled in his book *Inclusion: The Politics of Difference in Medical Research*, this campaign was led by minority and women health advocates in a push to diversify the populations studied in medical research. At the time, most clinical research subjects were middle-aged white men, whose bodies had been defined as the scientific norm. Critics demanded instead that medical research be made more inclusive to attend to the particular health needs of women, children, and racial minorities. Claims about justice in scientific research had shifted from protecting socially disadvantaged subjects from unethical practices toward promoting access to clinical trials and biomedical products.[1]

Inclusion of groups that were previously underrepresented in clinical research meant measuring biological differences across these groups—what Epstein calls the "inclusion-and-difference paradigm." In response to these demands, the federal agencies "ratified a new consensus that biomedical research—now a $94 billion industry in the United States—must become routinely sensitive to human differences, especially sex and gender, race and ethnicity, and age," writes Epstein.[2] Starting in 1986, a series of federal laws, policies, and bureaucratic offices institutionalized the scientific use of racial categories to ensure greater participation of minorities in clinical research and to address health inequities. Any federally supported university scientist performing biomedical research involving human beings or any company

seeking approval to market pharmaceuticals is required to include racial and ethnic minorities as research subjects and to analyze their findings by race.

The campaign for inclusion resuscitated federal interest in minority health, but one that focused on biological rather than systemic inequities. Within the space of a decade, the federal research bureaucracy incorporated a new focus on minority health at every level. A task force commissioned by Margaret Heckler, secretary of the Department of Health and Human Services (HHS) from 1983 to 1985, to examine the state of minority health released its pathbreaking report in 1985, documenting "a continuing disparity in the burden of death and illness experienced by blacks and other minority Americans as compared with our nation's population as a whole."[3] In the space of five years, HHS founded an Office of Minority Health, the Centers for Disease Control created the Office of the Associate Director for Minority Health, and the NIH established the Office for Research on Minority Health. Congress also began to require that biomedical researchers receiving government money pay attention to race. The NIH Revitalization Act of 1993 mandates that federally funded clinical studies enroll women and minorities as subjects to "elicit information about individuals" in these groups and, in the case of trials evaluating interventions, "examine differential effects on such groups,"[4] Many researchers interpret this policy as a requirement to break down research findings into racial categories. The act also specifies that researchers must use the racial categories provided in OMB Directive No. 15 for all federal reporting.

There were dissenters to the race-conscious approach to inclusion in biomedical research. Some conservatives argued that the NIH rules imposed unlawful gender and racial quotas on researchers, likening them to affirmative action policies. Some minority doctors dedicated to racial equality in medicine were also worried. Otis Brawley, an African American oncologist then at the NIH National Cancer Institute and now chief medical officer of the American Cancer Society, warned that the NIH Revitalization Act "may eventually do more harm than good for the minority populations that it hopes to benefit. The legislation's emphasis on potential racial differences fosters the racism that its creators want to abrogate by establishing government sponsored research on the basis of the belief that there are significant biological differences among the races."[5]

Race consciousness in federal funding guidelines creates a perplexing paradox. While designed to correct historic neglect of people of color in

biomedical research, requiring that biomedical researchers use race as a variable risks reinforcing the very biological definitions of race that have historically supported racial discrimination. Paying attention to racial disparities in health is crucial to eliminating them, but attention to race in biomedical research can also make these disparities seem to be grounded in biological difference rather than social inequality.[6]

The way out of this paradox is to focus on how race is used and defined by the research at hand. Some federal research initiatives that investigate the reasons for race-based disparities and develop programs to address them properly treat race as a social grouping that has consequences for people's health. The NIH guidelines fall into trouble when they import these social categories into research that reaches biological conclusions—as if race were a biological category. As I showed in chapter 3, forcing genetic findings from population and biomedical research into social categories of race threatens to make these categories seem genetically determined. The NIH requirements can easily be interpreted to treat races as biologically distinct populations whose health status and responses to therapies vary for genetic reasons inherent to each group.

Researchers often mechanically break down their findings by race to comply with the NIH guidelines. Even if they are careful to identify study participants in narrower geographic, ethnic, or indigenous terms, they are compelled by NIH rules to "aggregate" their findings into the approved racial categories.[7] They then proceed to report race-based conclusions at the end of the study—even when racial subsamples are too tiny for statistically sound results and even when race was not related to the purpose of the study in the first place. When I asked a scientist who studied genetic contributions to hypertensive heart disease why she reported her findings according to the race of research subjects, she responded simply, "I had to report by race to get NIH funding!" In his interviews with biomedical researchers studying racial differences in health, Steven Epstein "found that researchers consistently were unable to articulate clear definitions of 'race.'"[8] Good science requires a cogent definition of racial variables used in research as well as an intelligent hypothesis of why they are relevant. "I think that requiring these racial categories traps us into continuing to use them without breaking past them and trying to find other ways of actually directly measuring the things that we are really interested in," Stanford bioethicist Mildred Cho told me.[9]

It appears that Congress had not thought through these problems when it passed the NIH Revitalization Act in 1993. Most of the advocacy for diversifying biomedical research came from women's organizations, and the debate centered on the biological differences between the sexes. In fact, the original mandate in the NIH reauthorization bill, drafted by congressional staff in 1990, referred only to inclusion of women as research subjects. It was only after the Congressional Black Caucus belatedly intervened that the phrase "and minorities" was added to the language requiring inclusion of women in NIH-funded research.[10] Congress did not seriously weigh what it meant to treat race, along with sex, as a biologically distinctive category. The law lumps together women and minorities as "second-class citizens" who had been wrongfully excluded from clinical trials without recognizing that race and sex are not parallel kinds of identity. The biological distinctions between men and women (though far more fluid than commonly held) are not mirrored in biological distinctions among races.

For most of U.S. history, people of color were exploited in medical experiments that injured or stigmatized them while they were excluded from clinical trials designed to improve health. Congress was right to correct this injustice but went about it in the wrong way. The purpose of diversifying biomedical research should not be to find innate differences among racial groups. It should be, first, to give patients equal access to the benefits that can accrue simply by participating in a clinical trial and, second, to give scientists a richer resource to investigate the mysteries of human biology—what makes cancer tumors grow in human tissue, why a therapy is effective for some patients and not for others, and how to stop the progression of Alzheimer's, for example.[11] Adding minority patients to the research pool provides a more accurate reflection of human diversity.

Third, diversifying clinical research can aid in investigating how racism harms people's health. Scientists need a political, not a biological, definition of race to accomplish this. If race is treated accurately as a social category, there is nothing wrong with recruiting members of a particular racial group to investigate the causes of illness in the group and the best ways to eliminate them. Through its Racial and Ethnic Approaches to Community Health (REACH) program, the CDC Office of Minority Health sponsors scientific studies that "target" diseases within particular racial and ethnic communities. As chair of the board of directors of the Black Women's Health Imperative, I support the organization's research projects and educational programs

(some funded by the CDC) that have addressed black women's greater burden of diabetes, obesity, breast cancer, heart disease, and HIV/AIDS because these efforts tackle the preventable social reasons for these disparities.

In the decade since the federal inclusion policy was launched, little progress has been made, however. A 2009 analysis of fifteen health status indicators found that disparities widened significantly for a third of them: mortality rates for heart disease, breast cancer, diabetes, and suicide, as well as cases of tuberculosis. The disparity in infant deaths remained virtually unchanged. In short, "there was no significant trend toward overall improvement."[12] The 2007 National Healthcare Disparities Report, issued by HHS, had reached an equally dismal conclusion: "Overall, disparities in health care quality and access are not getting smaller."[13] The paradigm that includes minorities in biomedical research to discover their intrinsic biological differences has done nothing to close the racial chasm in health. Yet biomedical researchers are increasingly turning to genetic explanations for racial disparities in health and disease.

Searching for the Gene

Asthma, an inflammatory disorder of the airways, is the most common chronic disease striking children and is the illness that most frequently puts children in the hospital. Asthma symptoms are triggered by allergens or irritants in the environment that cause the airways to narrow. People with asthma have a hard time breathing and are subject to bouts of coughing and wheezing. "I think that asthma's worse for children, though, because play is part of childhood and children cannot play with real abandon when they feel so bad," writes Jonathan Kozol in *Ordinary Resurrections* about children living in the South Bronx, where asthma rates are sky-high. "Even mild asthma weighs their spirits down and makes it hard to smile easily, or to read a book with eagerness, or to jump into conversation with entire spontaneity."[14]

It has been estimated that one third of children living in public housing have allergic asthma.[15] Puerto Rican and African American children have especially high rates of the disease: while 8 percent of white children have asthma, 19 percent of Puerto Rican and 13 percent of black children do. Asthma prevalence and death rates have been increasing in recent decades, especially in inner-city communities, further widening the racial gap.[16]

Many research studies have identified the environmental allergens that

trigger asthma. For example, a team of scientists from Boston University School of Medicine traced the cause by exposing mice to dust particles collected from inner-city homes and studying the effects on their lungs. The culprit turned out to be exoskeletons and droppings from cockroaches.[17] From 2002 to 2005, New York University researchers attached air pollution monitors to the backpacks of children with asthma in the South Bronx. They found that the children, who were twice as likely to attend a school near a highway as children in other parts of the city, were exposed to fine-particle pollution from diesel exhaust (a known asthma trigger) that exceeded EPA standards.[18]

It is clear that exposure to pests and air pollution increases the risk of asthma, but why does the risk vary according to race? Why is the rate and severity so high in Puerto Rican and black children in particular? Esteban Gonzalez Burchard, the biopharmaceutical researcher at University of California at San Francisco we met in chapter 3, thinks it has to do with their genes. Burchard calls himself a "physician scientist" who studies the influence of race on the genetic causes of disease. He established the Genetics of Asthma Laboratory in a quest for the unique genetic signature that predisposes children of certain races to get sick with asthma. He also wanted to know why Puerto Rican children respond poorly to albuterol, the top asthma drug. To assist the effort, Burchard pulled together a multidisciplinary team of experts in genetic epidemiology, biostatistics, genomics, clinical asthma, and pulmonary molecular and cell biology. He has collected thousands of genetic samples, stored by race in the university's DNA Bank, to create a database his lab team can scan for genetic clues as to what distinguishes rates of asthma in different racial and ethnic groups. The distinctive genetic variant in Puerto Ricans, he hypothesizes, is related to their recent African ancestry and also explains asthma severity in African Americans.

I sat down with Burchard in June 2008 at his office in the Genetics of Asthma Laboratory to ask him why he was so sure that disparities in asthma had a genetic root. "I'm fascinated as to why disease rates and severity vary across populations and how racial or ethnic background influences that. I'm also impressed by how race modifies risk factors, whether they would be genetic or environmental," Burchard tells me. But he quickly leaves the environmental part to boast about his lab's recent discovery: "We just published a paper that came out two weeks ago in *Human Molecular Genetics* in which we identified an African-specific mutation. Meaning that we screened

Caucasians, Asians, different Hispanic subgroups, and Africans, and this mutation was specific to African origin." Burchard explains that the mutation is involved in regulating a protein that affects smooth muscle tone in the airways, saying, "Long story short, it causes more severe asthma."[19]

"When you say it's African specific, do you mean that this mutation is only found in people of African descent?" I asked. I was surprised because the scientific literature usually speaks of differing allele frequencies, not racially exclusive mutations. Yet Burchard told me the mutation was confined to people with African ancestry. I learned from a later reading of the article Burchard mentioned that this conclusion was based on his lab's screening of research subjects, including twenty-four African Americans, for the polymorphism C818T. "We did not find a single subject heterozygous for the C818T SNP in screening 96 Puerto Rican, 96 Mexican, 86 Caucasian, and 7 Asian asthmatics, implying this SNP is specific to populations of African origin," his research team concluded.[20] Yet, given the research on human genetic diversity, the uncertainties inherent in racial self-identification, and Burchard's small sample, I needed better evidence of this "implied" African gene. It makes no sense for African Americans but not Puerto Ricans to have this supposedly race-specific allele when people in both groups have recent African ancestry.

"Some people would say that by focusing on minority health, you're reinforcing the idea that minorities are biologically different. How you do respond to that?" I asked Burchard.

"I think populations *are* biologically different," he replied. "I mean, whether you are minority or non-minority, I think populations are biologically different, just like males and females are biologically different."[21] He continued, "So, for example, cystic fibrosis, that is a Caucasian mutation, only in Caucasians. We are finding it now in African Americans and Puerto Ricans. And that's because of the intermixing of populations."

But as we talked, Burchard's views of race and genetics became complicated by his understanding of race as a social category. "I identify as being Mexican or Hispanic, knowing that I am like Obama. I had a white father, my mom looks black—she's darker than you. She identifies as being Mexican, and when I told her she was twenty percent African, at first she said no, that's not correct. But in her infinite wisdom, she later said, fine, who cares." I pushed Burchard on the contradiction between his uses of social and biological race. He seemed to separate the race-based genetic research he was conducting from race-based social effects on health.

"That's why we need more research in this area. It's fascinating to think about the interaction between your biologic background and your genetic background and the social forces that are operating on it. Now, we know if we talk socioeconomic status, somehow your social position in life gets internalized. The physiological outcomes of stress translate into high blood pressure, which is translated into kidney disease and the heart disease, which is translated into premature death. We also know that the social discrimination, particularly in United States, somehow gets internalized in African American males in particular." By the end of our conversation, Burchard was emphasizing genetics less. "Personally, I think I have been pretty good at saying that it's not all genetic. People try to paint me as a pure geneticist, but I know that there is an interaction between social and environmental and genetic factors."

"Then why establish a lab devoted to finding the genetic roots of racial differences in asthma?" I asked. "Right now I don't think we know enough about the potential outcomes of genetic testing in specific racial groups. I think in the case of the mutation that we identified, if it did pan out, it could be a novel drug target that we would say, gee, if you are an African American male, here is the drug that you should take. . . . That would be an example of how our sort of work could be directly translated into clinical applications."

The Genetics of Asthma lab is one of countless research projects at universities and biotech firms around the country hunting for the genes that are responsible for health disparities in America. They are supplementing a large body of published studies that claim to show that racial gaps in disease prevalence or mortality are caused by genetic differences. In addition to asthma, disparities in infant mortality, diabetes, cancer, and hypertension have all been attributed in the scientific literature to genetic vulnerability that varies according to race. Most of these studies never even examined the genotypes of research subjects, as Burchard's lab does; they just infer a genetic source of racial differences when they fail to find another explanation. As interest in health disparities converges with the genomic science of race, a new brand of racial stereotyping is gaining hold in biomedical research.

Consider an effort to explain the enduring black–white gap in premature births and low birth weight. A team of obstetric researchers examined all

births in Missouri between 1989 and 1997 to test the hypothesis that "black race independent of other factors increases the risk of extreme preterm birth and its frequency of recurrence." The researchers used statistical methods to calculate the independent influence of race, socioeconomic status (whether the mother was a recipient of Medicaid, food stamps, or the WIC program), and maternal medical risk factors such as lack of prenatal care and cigarette smoking. An article published in 2007 in the *American Journal of Obstetrics and Gynecology* reported that black women were not only more likely to deliver preterm babies but also to have preterm births in subsequent pregnancies. Because this overrepresentation occurred even when they controlled for the medical and socioeconomic factors, the researchers concluded that their findings "suggest a probable genetic component that may underlie the public health problem presented by the racial disparity in preterm birth." Although conceding that they may have overlooked "hidden variables" that also contribute, they nevertheless speculated about an unproven genetic mechanism operating in "the black race":

> We postulate that although preterm birth is a detrimental outcome in pregnancy, it may be a result of a selective advantage, conferring inflammatory protection against other disease processes. This selective advantage phenomenon has been well described for diseases afflicting the black race, particularly sickle cell disease, glucose-6-phosphate dehydrogenase deficiency, and nitrous oxide synthase polymorphisms and their effects on the incidence of malaria.

The article ended by downplaying "disparate access to medical care or other environmental factors," arguing that "our data suggest that the proposed genetic component to preterm birth may be a greater etiological contributor than previously recognized"—despite presenting no genetic data whatsoever![22]

Despite its weaknesses, the Missouri birth study was dignified with a published roundtable discussion in which commentators granted that "the genetic link is very strong" and that the disparity "may best be explained by a genetic etiology."[23] The research also led to the headline "Study Points to Genetics in Disparities in Preterm Births" in the *New York Times*, which repeated the totally unsubstantiated conjecture that premature births may provide some evolutionary advantage to black women. Neil Risch, the colleague of Esteban Burchard who conducts genetic research on health disparities, criticized the study's inference of a genetic cause without ever examining genes. "They're inferring something is genetic by elimination of other factors," he told the *New York Times*. "But geneticists believe that to implicate something as genetic requires direct evidence, as opposed to evidence of absence."[24]

In this study and others like it, guesswork about a peculiar black predisposition toward unhealthy births imports an old notion about sickle cell disease "afflicting the black race."[25] Whenever I give a talk on this topic, there is inevitably someone in the audience who invokes the mantra that sickle cell anemia is a black genetic disease and therefore proves that race is a genetic category. This misconception was first popularized in the early twentieth century by hematology experts who believed the capacity to develop sickled cells was uniquely inherent in "Negro blood."[26] Stereotypes about black resistance to malaria and susceptibility to sickle cell justified sending black workers to malaria-infested regions in the first part of the century and later led to discriminatory government, employer, and insurance-testing programs in the 1970s.[27]

The error is easily exposed by looking at two world maps, one highlighting the regions around the globe where malaria is prevalent, the other highlighting areas where sickle cell disease is present. The maps mirror each other perfectly. By comparing them, it is plain to see that malaria and sickle cell aren't restricted to Africa and that much of Africa is unaffected. High frequencies of the trait also occur in parts of Europe, Oceania, India, and the Middle East, all places where there is malaria. In fact, people in the town of Orchomenos in central Greece have double the rate of sickle cell disease reported among African Americans.[28] If frequency of the sickle cell gene determined racial boundaries, it certainly would not prove there is a black race. Instead, as Jared Diamond pointed out in the November 1994 issue of *Discover*, if we grouped together people by the presence or absence of the sickle cell gene, "we'd place Yemenites, Greeks, New Guineans, Thai, and Dinkas in one 'race,' Norwegians and several black African peoples in another."[29] It would be more accurate to call the groups with the sickle cell gene the "anti-mosquito race." Of course, that would be a silly way of grouping people, except for studying the sickle cell gene. But "black race" is an equally silly way of grouping people for identifying genetic contributions to disease.

Another favorite playground for genetic speculation is hypertension. Until

recently, virtually every study of hypertension among African Americans accepted the premise that blacks have higher rates of the disease than whites because of inherited susceptibility.[30] In volumes 27 through 30 of the scientific journal *Hypertension*, published in 1996 and 1997, thirty articles hypothesized the existence of innate physiological differences among racial groups.[31] Since then, theories about the precise genetic mechanism behind the hypertension gap are legion. Authors of one study published in the *Journal of Hypertension* in 2000, for example, "postulate that the genetic factor increasing the propensity of black people of sub-Saharan African descent to develop high blood pressure is the relatively high activity of creatine kinase, predominantly in vascular and cardiac muscle tissue."[32]

Even Oprah was familiar with one of the genetic theories for the hypertension gap. On an "Ask Dr. Oz" segment of *Oprah* in 2007, an audience member asked, "Why do I sweat so much?" After citing overactive thyroid, body toxins, and high blood pressure as possible causes, Dr. Mehmet Oz turned to Oprah. "Do you know why African Americans have high blood pressure?" Oprah replied with confidence, "The reason why African Americans have higher blood pressure, Dr. Oz, is because during the Middle Passage, the African Americans who survived were those who could hold more salt into their body."

"I'm off the show, you don't need me anymore—that's perfect!" Dr. Oz cheered.[33]

One of the most popular yarns about black genetic difference is the "slavery hypothesis" for hypertension. Originally spun by Thomas Wilson and Clarence Grim in the 1980s, the theory holds that blacks in America today suffer from higher rates of hypertension because their ancestors survived the brutal transatlantic voyage from Africa by overcoming water deprivation and dehydrating illnesses owing to their genetic predisposition to retain sodium. This hereditary trait, proponents claim, came to dominate the gene pool of enslaved Africans and was passed down to a disproportionate share of present-day African Americans. Their genetically impaired ability to excrete salt expands water volume in the blood vessels, leading to higher rates of hypertension. But many experts—including slavery historian Philip Curtin, on whose work Wilson and Grim had relied; biological anthropologist Fatimah Jackson; and epidemiologist Jay Kaufman—have refuted this conjecture on methodological, evidentiary, and theoretical grounds, while others have provided more plausible social explanations for African American hypertension rates.[34] For one thing, blacks in former slave societies like the West Indies do not have the high hypertension rates of blacks living in the United States.

A landmark study led by Richard Cooper contested the conventional wisdom that blacks have an inherent predisposition to hypertension.[35] Comparing hypertension rates around the world, Cooper analyzed three surveys of blacks from Africa, the Caribbean, and the United States and eight surveys of whites from the United States, Canada, and Europe. Collectively, the studies enrolled 85,000 participants. If African Americans' higher hypertension risk were genetic, we would expect that people of African descent are more likely to have high blood pressure than people of European descent. Instead, after pooling the global data, Cooper found just the opposite. White populations on average have a substantially higher burden of hypertension. Germans have the highest. Nigerians have the lowest. U.S. whites come close to black Nigerians and Jamaicans, while U.S. blacks come close to whites from England and Spain.

Although the slavery hypothesis has been thoroughly debunked, it still holds sway in the popular imagination and even in professional circles. The theory is described in numerous hypertension textbooks without mention of the refutations and "frequently invoked in the medical literature to justify the more general proposition of innate biologic difference in cardiovascular disease risk and treatment efficacy," writes Jay Kaufman.[36] The myth received a shot in the arm when the Harvard economist Roland G. Fryer Jr., an African American, embraced it in his larger research project to "figure out where blacks went wrong."[37] Fryer co-authored a 2005 paper with two white colleagues in the Harvard economics department, Edward Glaeser and David Cutler, attributing the six-year disparity in life expectancy between blacks and whites to blacks' inherited tendency to retain salt.[38] Fryer rehashed the discredited theory on the CNN series *Black in America*. A 2005 *New York Times Magazine* article about Fryer by Stephen Dubner, coauthor with Steven Levitt of *Freakonomics*, states that Fryer "came across a period illustration that seemed to show a slave trader in Africa licking the face of a prospective slave," presumably to "try to select, with a lick to the cheek, the 'saltier' Africans." Dubner writes that Glaeser and Cutler appreciated having a black collaborator to circulate the theory: "There's an insulation effect," Glaeser said. "There's no question that working with Roland is somewhat liberating." According to Dubner, Fryer is able to "raise questions

that most white scholars wouldn't dare."[39] It is not the case, however, that white scholars are fearful of attributing racial disparities in health to genetic difference; most of the hundreds of articles making precisely these claims are authored by whites and published in prestigious scientific journals. Rather, having a black co-author on a dubious theory about black genetic difference confers seeming legitimacy.

The slavery hypothesis may be particularly egregious, but in fact the whole body of genetic explanations for health disparities is questionable. To begin with, most of these studies suffer from the methodological sloppiness I discussed in chapter 3. They group research subjects into conventional racial categories, fail to explain the relationship between these social categories and genetic traits, and then reach conclusions about genetic difference among the subjects. A survey published in the *Journal of Medical Ethics* in 2006 examined 268 published reports of genetic research that used race as an independent variable.[40] The research team found that 72 percent of the studies failed to explain their methods for assigning race to research subjects. Despite this glaring flaw, 67 percent of the same studies drew conclusions associating genetics, health outcomes, and race.

But there is a far more fundamental defect. Genetic explanations for health disparities are basically implausible. Remember, the issue is not whether genes affect health—of course they do—but whether *genetic difference* explains *racial disparities* in health. If you approached health disparities with a completely open mind, with no preconceived assumption that racial differences must be genetic, it would make perfect sense that social groups that have been systematically deprived for centuries have worse health than social groups that have been systematically privileged. The logical cause is the social distance between them and all the ways societal advantage and disadvantage affect people's experiences, environments, and access to resources, including health care.

Likewise, it would seem strange for a large group of people as genetically diverse as African Americans to have such a concentrated genetic susceptibility to so many common complex diseases. When he came to the United States from his native Nigeria, the geneticist Charles Rotimi was struck by the gulf between white and black health. "It seemed highly unusual to see these disparities," he told me. "I will call it a privileged perspective—I came from a different environment and to see that a group of people in this society were so heavily at risk for multiple conditions was a curious thing. I

thought, this cannot be genetics—why would a group of people inherit so many bad things?"[41] A more plausible hypothesis, given the persistence of unequal health outcomes along the social matrix of race, is that they are caused by social factors.

In order to conclude that the cause of health disparities is genetic, scientists must first rule out more logical social explanations. That is a near-impossible task because of the nature of gene–environment interactions: it is very hard to separate a genetic cause from environmental influences. Definitively showing a genetic cause for a racial disparity in disease prevalence or outcome would require the kind of experiment researchers perform on laboratory mice. The standard scientific test required to prove that a phenotypic difference (such as different disease rates) results from a genetic difference is a controlled breeding experiment with rigorously regulated environments that spans at least two generations. Applied to human beings, the study would last sixty years and examine the offspring of men from different races who mated with at least four carefully selected women, so the offspring could be compared. The researcher would also have to dictate "what those children could eat, where they could live, and what exercise regime they could have maintained," points out evolutionary biologist Joseph Graves Jr.[42]

Perhaps it is unfair to expect such a high degree of scientific precision. But studies that conclude health disparities are caused by genetic difference do not even come close. These studies typically control for the socioeconomic status (SES) of the research subjects in an attempt to compare subjects of different races who have the same SES. If there remains a difference in the prevalence or outcome of a disease, the researchers typically attribute the unexplained variation to genetic distinctions between racial groups. But this conclusion suffers from a basic methodological error. The researchers failed to account for many other unmeasured factors, such as the experience of racial discrimination or differences in wealth, not just income, that are related to health outcomes and differ by race. Any one of these unmeasured factors—and not genes—might explain why health outcomes vary by race. Statisticians call this the problem of residual confounding: falsely concluding that there is a causal relationship between two variables (here, genetics and disparate health outcomes) because other variables are not measured.[43]

An important aspect of this problem is that SES measures used in genetic studies are woefully inadequate. The typical measures—occupation, income,

and education—do not capture fully the social and economic factors that determine social status. Whites and blacks with the same income and educational levels occupy different rungs on the social ladder and are not interchangeable. Because of their racial privilege, whites earning the exact same salary as blacks tend to have greater wealth—money in the bank, property and investments, and anticipated inheritance. A large federal survey showed that, even after adjusting for SES and household characteristics, blacks were more likely than whites to have experienced economic hardships during the previous year.[44] Blacks who appear to be as poor as poor whites when income alone is measured are at greater risk of being unable to pay the rent, having their utilities shut off, and being evicted. Black poor people experience a more intense poverty than white poor people.[45] Even when black individuals reach the middle class, chances are they have close relatives who are poor, so they bring family financial needs with them into a higher bracket. Their neighborhood conditions also tend to be drastically different. Blacks are more likely to live in all-black neighborhoods with fewer services, more pollution and crime, and higher overall poverty rates. College-educated African Americans applying for jobs routinely "whiten" their résumés, deleting clues to employers that they are black because they fear their race will hurt their chances of getting an interview.[46] Because of a multitude of individual and institutional biases against blacks, the typical measures fail to control adequately for true SES.

Nor do typical SES variables measure a research participant's socioeconomic position across time. The "snapshot" model of SES data collected by biomedical researchers ignores subjects' entire life experiences.[47] Poverty and deprivation early in life may affect a tumor or heart condition or diabetes later in life. As I discuss in the next chapter, a pregnant woman's living conditions shape fetal development in ways that have lasting effects on a child's health into adulthood. This omission is compounded by the distinctive nature of black child poverty: black children are not only three times more likely to be poor than white children, but they are also more likely to be poor for their entire childhoods. So equating a black bank teller with a high school diploma earning $25,000 a year and a white bank teller with a high school diploma earning $25,000 a year may overlook extremely different life circumstances, such as childhood years in poverty, current family wealth, or neighborhood segregation, that can have a huge influence on their health.

Even with better measures of socioeconomic status, there would remain a

fatal flaw. Studies testing a genetic hypothesis fail to account for the impact of racism on health at both the individual and societal levels. Genetic studies do not even attempt to measure the health effects of experiencing racism or of inequitable social systems.[48] There is growing evidence that living in a society that devalues your intelligence, character, and beauty, where you encounter discrimination on a daily basis, and in which entire institutions systematically disadvantage the group you belong to, exacts a toll on health that scientists are only beginning to fathom. Researchers cannot resort to genetic causes when they have omitted this crucial variable. "The biology is a fall-back black box that many researchers use when they find racial differences," says Harvard sociologist David Williams, a leading expert on health disparities. "It is a knee-jerk reaction. It is not based on science, but on a deeply held, cultural belief about race that the medical field has a hard time giving up."[49] Leaping to genetic conclusions after failing to account for the impact of racism on health is fundamentally unscientific.

All these methodological problems lead back to a more basic question about research testing the hypothesis that health disparities are caused by genetic differences. Perhaps it is so easy to leap to genetic conclusions, but so hard to prove them scientifically, because the hypothesis itself is faulty.[50] It is founded on a misunderstanding of race as a naturally created biological division instead of a politically invented social division. The belief in natural races despite the evidence obscures the circular logic of studies of race and genetics. Scientists observe racial disparities in health and hypothesize they are caused by biological difference based on an ideological premise that race is a biological category. After collecting data on health disparities, they conclude that unexplained differences between racial groups must be genetic, which they claim proves that races are biologically different.

But this type of research has not *proven* that health disparities stem from innate biological difference. It has simply restated the original observation of health disparities in genetic terms based on an unsubstantiated assumption of biological distinctions among races. Witness the tautological explanation appearing in the 1995 text *Biologic Variation in Health and Illness*: "Human beings are similar; they are of the same species, but belong to several different races; hence, they may differ in several important ways: in growth and development rates, in enzyme systems, in disease susceptibility, and in response to environmental stresses."[51] Because it is assumed that races differ biologically, the differences between them appear to be biological.

There is another fatal flaw in the hypothesis that health disparities are caused by genetic difference. It is not just difficult to isolate genetic causes without the type of experiment I described earlier that carefully breeds the research subjects and raises them in controlled environmental conditions. It is actually *impossible* to separate genetic from environmental contributions to health. We usually talk about genes plus environment, as if one is added to the other and each part can be independently measured and quantified. But any genetic scientist worth her degree knows that DNA's contribution to disease *always* interacts with environment in a dynamic and ongoing process. Genes are not the original foundation for health that is acted upon by the environment. From the moment a pre-embryo is created, its traits are determined *both* by genes and environment, and this interaction continues during every moment of its existence. This is why genetically identical organisms, including human beings, raised under different environmental conditions differ in physiology.[52]

When experts claim that "genes are responsible for fifty percent" of a disease or behavior, it gives the impression not only that genes are more important than they are, but that it is possible to separate genetic and environmental contributions.[53] My eight-year-old son understood this when he realized that if he cloned himself, he could not guarantee his clone would be exactly like him. "He would turn out different if you treated him differently," he observed wisely. We do not need clones to know that identical twins, with the same complement of genes, are different from each other even before they are born because of their positioning within the womb or chemical changes that happened in their cells while they were still gestating. Once they get out into the world, they develop into two distinct individuals, even when raised in the same home, because their environments and experiences are different. Similarities in twins raised apart do not obviate the myriad differences that still exist between them.

The additive model of nature versus nurture misrepresents human biology. Under a more accurate model of interactive effects on health, there is no separable genetic cause that researchers can identify through a process of elimination. As Cooper and Kaufman put it, the question of whether observed racial differences in blood pressure, low birth weight, or asthma are caused by genes "falls properly within the realm of nescience—the unknown and the unknowable."[54] It seems heretical to say that scientists are incapable of knowing everything. Scientists are supposed to speculate about possible

causes of observed realities. By putting forth creative hypotheses that can be tested, scientific imagination advances our understanding of human biology. Yet contemporary scientific publishing, which generally tends to ignore studies that *disprove* hypotheses, rarely reports when these hypotheses go wrong. Instead, researchers tend to "postulate" genetic mechanisms for racial disparities that are never proved outright.

Some health disparities research is now focusing on gene–environment interactions rather than trying to isolate genetic causes of disease. As Francis Collins put it in 2004, researchers investigating risk factors for disease "must be *equally* rigorous in their collection of genetic and environmental data. If only genetic factors are considered, only genetic factors will be discovered" (my emphasis).[55] Beyond paying lip service to vaguely defined "nongenetic" factors, however, most genomic scientists are not incorporating rigorous measures of environmental factors—especially social ones—into their health disparities research. What's more, the genetic associations they discover tend to attract more academic and media attention, usually eclipsing the social influences on health altogether. Genes are frequently described as "the cause" of disease, while environmental contributions are merely "triggers," and little attention is paid to how the environmental and the biological actually interact. This emphasis on genetic versus social contributions is reflected in federal research funding. For the years 1995 to 2004, a search of research awards in the National Institutes of Health database using the term *genetics* identified 21,956 new grants (including 181 cross-indexed by the term *race*), while only 44 new grants were indexed by the terms *racism* or *racial discrimination*.[56] When the NIH launched a new center to study population health, it was originally named the Center for Genomics and Health Disparities—the environmental component was completely missing from the title. It has since been reconfigured as the Center for Research on Genomics and Global Health to eliminate the implication that it is studying genomic causes of health disparities.

It is possible that Nigerians, Jamaicans, and African Americans are all genetically prone to high blood pressure, but there is something in the environment that causes elevated rates in this country and lowers rates elsewhere. Or perhaps there is a SNP more prevalent in people with African ancestry that makes them more susceptible to environmental triggers for asthma. But if our goal is eliminating the gap between white and black hypertension or asthma in the United States, our focus should be on the environmental

causes of the gap because these are factors that can and should be changed. Continuing to dwell on an unknown genetic component of health disparities only distracts scientists from the more relevant task of identifying and tackling the preventable causes of disease. Spotlighting genetic "causes" as more important than environmental "triggers" steers solutions to health disparities toward gene-targeted therapies rather than toward improving the environment for everyone. It can cause other kinds of trouble—especially when racial stereotypes come into play. A black man in San Diego who developed hypertension because of exposure to toxic chemicals lost half of his disability award after a doctor reported that blacks are genetically prone to hypertension.[57] The failure of racial science to stem the disaster of the racial gap in American health is not surprising, given the flawed ideological, theoretical, and methodological foundation that supports it.

Why, then, do scientists continue to hunt for genetic explanations for race-based health disparities? The faith in biological race is incredibly powerful. Every methodological error or theoretical infirmity is seen not as a reason to question the hypothesis, but as a challenge to look harder for the genetic difference that is presumed to exist. Dissenters are often marginalized, their scientific objections dismissed as "politically correct" or failing to grasp the importance of genetics. Nevertheless, another group of researchers has taken up a more promising line of investigation that demonstrates that racial injustice, and not genes, causes America's glaring inequities in health.

6

Embodying Race

White women in Chicago are slightly more likely than black women to get breast cancer, but black women are twice as likely to die from it. That is a startling statistic by itself. But what is equally as shocking is that in 1980 Chicago's black and white breast cancer mortality rates were identical: black and white women died at the same rate. Over the course of the next twenty-five years, the astounding gap emerged.[1] Consider this additional aspect: the disparity in breast cancer mortality in New York City is only 15 percent. In Chicago, the racial gap is ten times greater than in New York.

It is unlikely that genes explain these numbers. Did something change in white women's DNA between 1980 and 2005 that decreased their likelihood of dying from breast cancer? Is there something genetically distinct about black women in Chicago versus New York that makes breast cancer deadlier? A more logical explanation is that there is something about having breast cancer that changed and that affected black and white women in Chicago differently.

Life and Death in Chicago

In 2006, a group of Chicago breast cancer researchers released their study showing the alarming racial divergence in breast cancer deaths. An article in *Chicago* magazine featured a photo of co-author Steven Whitman, an accomplished epidemiologist with a PhD in biostatistics from Yale who directs the Sinai Urban Health Institute.[2] Whitman and the Institute have been at