Refashioning Race: DNA and the Politics of Health Care

"For Sale: A DNA Test to Measure Racial Mix"
—Wade F4

"Shouldn't a Pill be Colorblind?"
—Stolberg 1

"I Am a Racially Profiling Doctor"
—Satel 56

"Does Race Exist?"
—Bamshad and Olson 78

Something is happening to race. Historically, in discussions of race and science, science has either been on the side of the devil or of God.[1] But science is a socially contingent knowledge-seeking activity. It can serve the interests of the State, for better or for worse; more commonly, it serves several social masters and produces mixed messages. After World War II, liberal ideologists, primarily through the UNESCO statement on race, rejected the typology of fixed racial categories in favor of the abstraction "universal man." Donna Haraway documents this brilliantly in a number of works. To back up the new ideology, liberals called on the social sciences and especially the biological sciences for documentation. As Haraway puts it, for phylogenies and types, new accounts of race substituted "gene flow, migration, isolation, mutation, and selection [as] the privileged scientific objects of knowledge" (*Primate* 202).

By making modern biology the mainstay of this new narrative of universal man, liberal policy makers hoped to banish racism and racial categories from our social systems. But this modernist moment, despite a flurry of efforts to beef it up in the 1990s, is in big trouble. As they focused on the institutional constructs of race, scholars emphasized that race is

not a natural category (see Graves and Vigilant); rather, it is forged from the discourses of politics, the law and history (see Greenberg; Jacobson; Nobles; and Omi and Winant). Despite public declarations of the end of biological race, however, the concept refuses to die. Biologists continue to argue over the evolutionary and genetic meanings of the category (see Couzin; Edwards; Proctor; Reardon; and Rosenberg et al.), while medical researchers persist under their own steam and by government mandate in using racial categories to evaluate the health and medical well-being of U.S. populations (see Bamshad et al.; Braun; Cooper and Freeman; Goodman; Gura; Hardy; Singleton, and Gwinn-Hardy; Holden; Lewis; Schwartz; and Stolberg).

In this article I explore two major themes. First, as I explicate contemporary scientific discussions of genes, medicine, and race I show how social, political, and scientific institutions and interests become mutually constitutive of twenty-first-century views on race. Second, as I have begun to do in other venues, writing specifically about gender (see "Bare Bones," *Sexing*, "The Problem"), I here explore how the idea of a "gene-environment system" applies to the production of racialized bodies. Understanding such systems can clarify our analyses of the uses and abuses of genetic research applied to the study of race and disease. To accomplish these tasks I begin with a brief history of biological ideas of race; I then turn to the efforts of biologists to modernize biological categories of race and the entangling of that project with efforts to explain and address health disparities between various racially and ethnically separated communities. I argue, in the end, that the scientific projects of geneticists aimed at understanding aspects of human history ought to be disentangled from current public health questions. These latter problems can often be addressed immediately and effectively using means already at hand.

Revisiting Race

During the past several years, a set of contradictions, animated, on the one hand, by well-intentioned liberal politics and, on the other, by technological advances in our ability to sequence and analyze DNA in combination with our intellectual devotion to the gene as an explanatory precept, has produced a fierce debate about both the scientific accuracy and the social utility of the race concept in medical research (see Aldhous;

Risch et al.; Satel; Schwartz; Wade, "Race"; Wilson et al.; and Wood). "It is no accident," writes historian Nancy Stepan, "that 'race' and 'sex,' in their primarily naturalized or biological meaning emerged in the eighteenth century, when the new political concept of the individual self and the individual bearer of rights was being articulated" (30). In the current controversy, the individual right in question is the right to equal health status. In 1993, faced with significant health disparities between groups falling into the census-based racial and ethnic categories (see Nobles; Stepan), the U.S. Congress included in the National Institutes of Health Revitalization Act the requirement that NIH-sponsored clinical trials include enough women and minorities to make it possible, using statistical analyses, to tell if a proposed medical treatment worked differently for men compared to women and for members of different racial groups. Behind this mandate lay the unexamined presumption that variables such as race, ethnicity, and gender "exert their effects through innate or genetically determined biologic mechanisms" (American Academy of Pediatrics Committee on Pediatric Research 1349).

Only rather slowly has the medical community realized that what appears at first to be an inclusive move—mandating participation by racially and sexually distinguishable groups in drug and other trials—might have a scorpion's sting, diverting attention from socioeconomic explanations of (and remedies for) health disparities (Brawley and Freeman). Sociologist Troy Duster offers a solution to the dilemma:

> *"Purging science of race"—where race and ethnic classifications are embedded in the routine collection and analysis of data (from oncology to epidemiology, from hematology to social anthropology, from genetics to sociology)—is neither practicable, possible, or even desirable. Rather, our task should be to recognize, engage and clarify the complexity of the interaction between any taxonomies of race and biological, neurophysiological, social and health outcomes. Whether race is a legitimate concept for scientific inquiry depends on the criteria for defining race, and will in turn be related to the analytic purposes for which the concept is deployed. (258–59)*

To carry out Duster's imperatives, we must first better understand some of the complexities of racial narratives in contemporary biology and medicine.

Race Talk in Contemporary Biomedical Science

The conflicting desires—on the one hand, to eliminate racial and social health disparities, while holding fast to the claim that visible variations among humans ought not, as they have in the memorable past, prevent claims to full citizenship, and, on the other hand, to value scientific investigation as an important method for producing reliable knowledge about how bodies stay healthy or get sick—have created one of the most confused and confusing debates in modern biology and medical science. While some feel that racial categories should be eliminated from medical research and practice, others argue that to do so will harm socially defined racial groups. Furthermore, while few medical researchers believe any longer in the eighteenth- and nineteenth-century Platonic notions of races as ideal types, some parties in the current debate argue that visual cues of human variability, such as skin color, eye shape, and hair texture, are reasonable proxies for a more complex underlying genetic variability among peoples of different geographic origins.

In her wonderful table of racial meanings transformed through the decades, Haraway suggests that race as an object of knowledge in the first third of the twentieth century morphed into the study of populations, and then, in the last decade of the twentieth century, into an analysis of the genome. While these newer categories dominated the studies of particular decades, the race term always lurks in the wings (see *Modest-Witness*). In the current literature, a variety of words, singly and in combination, both technical and popular, pepper scientific discussions of human variation. Examples include: race, diversity, ethnic, group, population, community, descent, ancestry, geographic origin, minority, gene frequency, haplotype, language communities, stocks, single nucleotide polymorphisms (SNPs, pronounced "snips"). In an insightful analysis of the difficulties genome scientists experience in talking about race while trying not to name it as such, Brady Dunklee argues that the personal discomfort, euphemisms, and plain confusion in current debates suggest that the wrangling biomedical scientists are in the process of producing a new racial biology. He writes:

> *Euphemism and implicit transferal of meanings between the "social" and the "biological" terms with many valences help negotiate complex tensions. The result is the production of a*

> *certain way of knowing race. Race cannot be bounded strictly, but gene frequency can differ among races. Races don't have proper edges, races don't have genes all to themselves, but the amount of some genes in races can differ, playing a role in medical disparities. (37)*

Race talk in the biomedical literature helps to produce race by supplying transfer points between different sorts of knowledge claims "by euphemizing categories including 'race' itself." Such "terminological instability" is a central component of the technology of racial difference (37).

During the first years of the twenty-first century the biomedical race literature exploded, with a bewildering array of positions taken on the proper use of terminology and categories both for practicing physicians and for genetic and public health researchers. Cancer researcher and health scholar Lundy Braun provides a road map for this debate. She divides publications in the period from 2000 to 2004 into five distinct perspectives ("Knowledge"). One group of researchers suggests that, since race is essentially a social category, the term should be dropped and substituted for by the concept of ethnicity. A second position, held by some epidemiologists and medical sociologists, is that although race and ethnicity are socially produced categories, belonging to such categories has profound biological effects. Such effects cannot be explained merely by reference to genetic variation (see Karlson and Nazroo; Krieger and Sidney, "Theories"; and Williams, "Race"). A third group questions the validity of any genetic studies of racial and ethnic disparities in disease, suggesting instead that studying the effects of racism deserves priority over the study of race. A fourth approach, espoused by the editors of the journal *Nature Genetics*, exemplifies the scientist's discomfort with race talk (Dunklee). Why not avoid the problem of race and ethnicity by using a "race neutral" approach ("Census")? Let individuals identify their own race or ethnicity, they suggest, and then look at their DNA sequences. When one takes such an approach, clusters of variable gene sequences bear a rough correspondence to group identification. As Braun writes, "[W]hether a fixed category or a more fluid concept defined statistically, 'race' simply [will] not disappear" (5).

Braun identifies one final position, which appeared in an online journal, *Genome Biology* (Risch et al.). In calling for the legitimacy of five geographic races, this position resonated with science writers for the *New York Times* (Wade, "Race," "Gene Study," and "Unusual") and elsewhere.

The ensuing popular attention as well as the considerable stature of the main author, Neil Risch, in the field of human genetics, has overwhelmed some of the other points of view, at least for those who are not central actors in this new race drama. In just two years, the ideas and arguments presented in the *Genome Biology* paper have become central to the biomedical rewriting of race.

Using Genes to Divide the World

Human geneticists, evolutionary biologists, and anthropologists are all interested in human evolutionary history. Where and when did the first humans arise? How did they disperse around the world? How and why did they come to look different? And are the visible differences in appearance important for understanding human health and disease? These are reasonable questions, and at certain levels, there is surprising unanimity about their answers. When unanimity breaks down, it is in part because of the complexity of the science involved and in part because of the weight of past scientific and social thinking about race. These components intertwine in ways that baffle attempts to unwind them. At the same time, the intersection of scientific argument with political and social goals produces new claims about and names for race, while also holding out the possibility that our old, familiar notions of race might be of some use after all. In this section, my goal is to analyze the science, the language, and the claimed medical implications of several recent scientific publications authored by top scientists in the field of human genetics.

Although eighteenth-century writers constructed racial categories that many took to be Platonic types, even then there was no clear agreement on how to define the categories. Blumenbach's 1776 group of five (Caucasian, Mongolian, Ethiopian, American, and Malay), which reappears eerily in the modern genetic literature, differs from Kant's 1775 group of four (European, Asian, American, and African) (Kittles and Weiss). In the centuries since these categories (which Kittles and Weiss call "the Big Few") were proposed, scientists have used many and varied classifications based on traits ranging from superficial appearance, language, culture, and geography to genes and proteins. Early-twentieth-century geneticists used the discovery of blood groups and types, which derive from protein differences that cause mixed blood of certain kinds to coagulate. But such genetic characters were relatively few in number, and the big push to use genes to examine (or create) racial categories began only in the 1950s,

when a technological breakthrough called electrophoresis made it possible to detect relatively small differences between similar proteins.[2]

The new DNA technology extends this ability to detect small DNA differences almost immeasurably. There are now lists, available on-line, of thousands of proteins and DNA regions that have been examined in populations worldwide.[3] Even before the publication of the human genome sequence, scientists had begun delineating single base pair variations at defined locations on the DNA of particular chromosomes. Although the exact number is unknown, there may be as many as thirty million of these so-called single nucleotide polymorphisms, or SNPs, in the collective human genomes (National Human Genome Research Institute). The possibility of measuring such a large number of possible differences in the DNA of two individuals has generated vast mapping projects (National Center for Biotechnology Information; The SNP Consortium Ltd.). It is likely, however, that only very few SNPs will turn out to be associated with genes that affect health and disease (Weiss and Terwilliger).

When SNPs were first discovered, there was initial concern that it was too much of a good thing, that with so many variants, mere chance would lead to statistical differences that had little biological import. (This would be analogous to studying IQ in huge populations and finding a statistical difference of two IQ points between two groups or two individuals.) Furthermore, the cost of identifying millions of SNPs would be staggering. But it rapidly became clear that haplotypes, groupings of genes containing variant SNPs, could be used to sort through the forest and find a few trees.

A SNP occurs when there is a mutation on a particular location on a chromosome. Usually the mutation is "neutral," that is, it does not affect human reproduction or longevity either positively or negatively.[4] Such new mutations appear in the midst of already-existing sequences of DNA. These already existing sequences with their new mutation are called haplotypes because they come from one of the two parentally donated chromosomes.[5] In the course of human history, new haplotypes form either by new mutation or by crossing over (recombination) between two chromosomes during the process of sex cell formation (meiosis). Because individual SNPs can be found within the same haplotype, and because within a particular stretch of chromosome only a few haplotypes account for most of the variation among people in a particular population, scientists can assess human variation by selecting SNPs that vary within known haplotypes. This means that instead of mapping ten to thirty

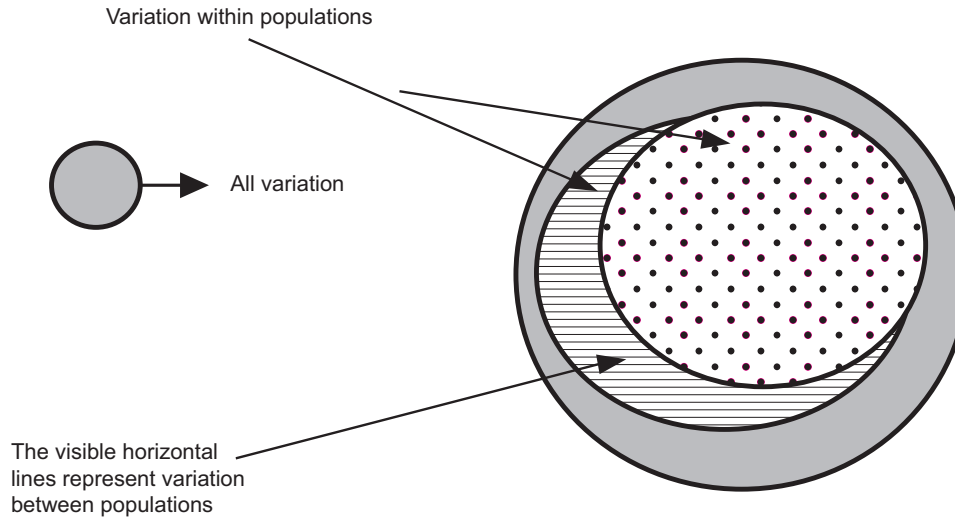

Figure 1
Genetic variation between and within populations: areas of overlap contain the same gene sets.

million SNPs, they can look at most of human genetic variation by selectively examining 200,000 to a million well-chosen SNPs across the genome (The International HapMap Consortium).

Snipping the genome has confirmed much of what scientists deduced about human variation when they surveyed protein differences around the world (Lewontin). About eighty-five percent of human variation is found in all populations and about fifteen percent between populations (see fig. 1). It is this uniformity and difference that led biologists and social scientists in the 1970s and 1980s to announce the end of race as a biological concept. Would that matters were so simple.

It is not surprising that groups with a common ancestry also have genetic similarities. Indeed, researchers find genetic similarities in groups that have a common cultural trait—ancestral language, for example—or a common geographic history. But one does not always imply the other. Shared language can result from invasion or colonization rather than common descent, while shared biological traits, such as skin color, can result from convergent evolution—a similar adaptation found in peoples of different geographic ancestry. In such cases, the idea that race is visible to the naked eye fails. As Kittles and Weiss put it, "Race is space only when carefully defined" (40). If we want to test the idea that humans can be successfully divided into genetic subgroups, how do we decide which people to sample and compare?

One of the first attempts to correlate haplotype patterns with diverse human groupings used SNPs from 275 people divided among four

population groups—90 from "Nigeria (Yoruba)," 93 from "12 multigenerational pedigrees of European ancestry, 42 unrelated individuals of Japanese and Chinese origin, and 50 unrelated African Americans" (Gabriel et al. 2226). Gabriel and colleagues use these categories without analysis or expressing concern that they accurately represent coherent or comparable groups of people. Dunklee points out, for example, that the term "Yoruban" does not define a group with an undisputed common history and that "Japanese" and "Chinese" refer to nations with diverse peoples and languages. The Yoruban group is unlikely to number more than several tens of millions, while the "Japanese and Chinese" derive from a population that is now roughly 1.4 billion.

If the populations to be compared have such different sizes and likely subdivisions—linguistic and otherwise—what makes them a plausible choice? The pictorial answer can be found in an illustration in a *Science* commentary by Jennifer Couzin. The uncaptioned color photos accompanying her piece show three faces—the smiling visage of a boy of apparent Asian ancestry, the dark-skinned face of a man that the reader is likely to presume to be of African ancestry, and a long-haired, blonde, blue-eyed white woman—I'm betting she's Swedish. What do these faces represent if not three of Blumenbach's five races? The word "race" appears in neither Couzin's nor Gabriel et al.'s articles, but it stares out at us nonetheless.

What, then, do Gabriel et al. have to tell us about SNPs and haplotypes? When they compared their groups two populations at a time, they found that up to ninety-five percent of the SNP pairs were the same. When they did differ, the differences were usually due to an incident of recombination in the Yoruban and African American populations but not in the European and Asian samples. When they looked, instead, at the similarity in haplotypes, they found near identity in the European and Asian populations. Of the twenty-eight percent of haplotypes occurring in only one population, however, ninety percent were found in the Yoruban sample. In other words, "most variation is found in Africa and what is seen outside of Africa is a subset of the variation found within Africa" (Pääbo 410). Note how the term "African" has been substituted here for the word "Yoruban." (On the genetic diversity of East, West, sub-Saharan, and supra-Saharan Africa, see Kittles and Weiss.)

Many current research papers on the genetic structure of human populations dance carefully—albeit unsuccessfully—around the word "race." Not so the work of geneticist Neil Risch and his colleagues.

As if pushed to the brink by a perceived "political correctness" in the genomic literature on human difference, Risch et al. throw down the race gauntlet, defining "racial groups on the basis of the primary continent of origin" (3). They use five geographic groups (which look suspiciously like Blumenbach's eighteenth-century categories) derived from five "continental ancestries"—African, European and Middle Eastern, Asian, Pacific Islander (includes Australia, New Guinea, and Melanesia), and Native American. Notably, these researchers reintroduce the word "Caucasian"—a notorious term throughout more than two centuries of race debate—as a synonym for "Europe and the Middle East."

Using a variety of rhetorical strategies, Risch et al. weave recent scientific work on human difference into a proposal to use racial self-identity in medical research and treatment. First, they maintain that "facts" can exist separately from "politics", and second, they imply that the facts of the matter (which represent truth) must trump politics (which represent social decisions). According to Risch and colleagues, those scientists who argue that race is biologically meaningless and that medical educators should emphasize the dangers of race-based medicine (e.g., Schwartz) do not rely on an "objective scientific perspective" (1). As a corrective, Risch and his colleagues propose to show that from "an objective and scientific [. . .] perspective there is great validity in racial/ethnic self-categorizations, both from the research and public policy points of view" (1).

Risch et al. motivate their position with a discussion of risk factors in disease, knowledge of which, they argue, can provide starting points for further investigation. Risks can be endogenous (for example, the difference in rates of breast cancer between men and women can reasonably be thought to have a large biological component) or behavioral (for example, rates of lung cancer related to different smoking habits). They presume that endogenous factors are not easily modified, while behavioral ones can be changed. In their view, when we know very precisely what causal factors are involved, risk estimates are precise and can form the basis of rational preventative action. When we know little, on the other hand, "the use of cruder surrogate factors can still provide valuable input for prevention and treatment decisions" (2). This last claim justifies their discussion of race and human genetic variation; they validate this motive on the grounds that "few genes underlying susceptibility to common diseases or influencing drug response have been identified" (2).

As I have already suggested, they review the evidence, based on comparing DNA and/or protein similarities, that humans fall roughly into

five groups. These "recapitulate[d] the classical definition of races based on continental ancestry" (3).[6] Populations outside of Africa descended from groups that left Africa within the last 100,000 years. As one might expect, these newer populations are less genetically diverse than the original African populations. Risch et al. propose that these five groups represent human races and that people whose ancestors originated from one of these groups belong to it, racially speaking, even if they now reside in some distant place. Ethnicity, they suggest, is self-defined and can be based on factors such as geography, religion, social ties, and cultural practices.

But, some would object, America is a melting pot. Over the generations we must, surely, have become genetically intermixed. Risch and colleagues use two arguments from rather different epistemological registers to suggest that such is not the case. First, they argue that self-categorization, as reflected in the 2000 U.S. Census, indicates that there has been less ancestral group intermixing than many would believe. After all, proferred six racial categories by the Census Bureau, 97.6 percent of the respondents claimed membership in only one race. The figures, they argue, "highlight the strong deviation from random mating in the U.S." (5). To confirm the impression derived from the Census, Risch turns to estimates of genetic admixture of Americans of European and/or African descent.

Risch's second response to the assertion of intermixing uses what geneticists call "admixture" studies, which rely on those five to fifteen percent of DNA sequences known to differ between populations. An admixture study uses genes that have very different frequencies in so-called ancestral populations. For example, in one investigation designed to study African Americans living in South Carolina, researchers chose SNPs that they could show had similar allele frequencies in several African populations (Sierra Leone, Nigeria, and the Central African Republic) but differed markedly in frequency from European samples (Germany, Ireland, and England) (see E. J. Parra et al.). If individuals from these populations migrated (by means forcible or otherwise) into the same geographic region and lived there for a suitable number of generations, the differences in allele frequency would have diminished if there had been significant intergroup mating. The degree of change in allele frequency (e.g., the increase in frequency in African Americans of genes originally found in high frequency only in the ancestral European populations) suggests how much intergroup reproduction has been going on.

The number obtained from such statistical studies is referred to as the "percent admixture." Note that this statistic is restricted to the

specific genes chosen for the study, which are, in turn, a small subset of at most fifteen percent of the human genome. Hence, admixture is *not* a measure of overall biological difference or similarity. The number is of clear medical importance only if it is derived from genes known to be involved in particular medical conditions *and* if those genes are known to differ in groups to which one wants to attach racial labels.

One such study suggests that the proportion of European admixture in populations who self-identify as African American ranges from twelve to twenty-three percent, with the highest level found in New Orleans and the lowest in Charleston, South Carolina (E. Parra et al.).[7] Populations from Jamaica have only 6.8 percent European admixture, information that would be important both practically and for studies of racial difference if, for example, the "black" population studied was of recent Afro-Caribbean origin, rather than, say, from New Orleans. Whether this average seventeen percent admixture is a lot or a little is a matter of perspective, and Risch makes this clear when he writes: "Thus, despite the admixture, African Americans remain a largely African group, reflecting primarily their African origins from a genetic perspective" (5).

The admixture studies suggest that Asians, Pacific Islanders, and Native Americans in the United States also have relatively low levels of European admixture. Genetic correlations with Census categories break down, however, for the Hispanic/Latino group. This is no surprise, since "Hispanic/Latino" may refer to people of African, European, and/or Native American ancestry. A study of 4000 SNPs from 313 genes also used the U.S. Census categories to look at genetic difference and similarity within the U.S. population. Beyond the underlying shared genotypes, these researchers found a larger percentage of alleles unique to the African American populations than to either those of Asian or European ancestry. But they also found that the Hispanic/Latino Census group could not be easily categorized (Stephens et al.). Although Risch et al. freely point to the fact that the Hispanic/Latino category is of less diagnostic value, they argue that the category could be useful to medical researchers who want to differentiate "genetic versus environmental sources for racial/ethnic differences" (6).

Whether or not visual categories of race are likely to be affirmed by genetic admixture studies depends on time and place. For example, a recent study entitled "Color and Genomic Ancestry in Brazilians" (F. C. Parra et al.) examined admixture levels in Brazilians of light, intermediate, and dark skin color. Admixture ranged from thirty-two percent in

light-skinned Brazilians to fifty percent in "blacks"; in other words, there has been much greater intermixing in Brazil than in the United States, a fact which is, of course, widely known to historians. Furthermore, in Brazil, racial self-categorization can change with one's social status. The self-identification of a Brazilian is far less informative to the interested medical geneticist than that of a U.S. African American of slave descent. In more recent work, Risch and colleagues acknowledge this point, writing that "primary categories that are relevant for the current U.S. population might not be optimal for a globally derived sample" (Burchard et al. 1174). Of course, in a doctor's office in the United States it is unlikely that these fine genetic distinctions will be drawn. A "white" Brazilian immigrant will be treated as a "genetically pure" European, and a dark-skinned Brazilian will be presumed to be no different, genetically, than the descendent of a West African slave brought to the United States in 1800.

Risch and colleagues argue that race is a stand-in, a black box for something yet to be learned about the biology of disease. If we ignore it, we lose a tool that can be used to disentangle the multiple causes and treatments of disease, but once we use it, we need to be clear about what it can and cannot tell us. While he and his colleagues offer some sensible ideas, they nevertheless founder, albeit in a manner more sophisticated than most, on some old dichotomies. The first is the distinction between genes and environment. Consider, for example, claims that African Americans are less prone to osteoporosis than Asian or European Americans. Assuming the accuracy of the fact, for the moment, how might such a difference arise? One imaginable scenario is that whatever genetic differences exist between African Americans and European Americans, they do not include genes involved with bone metabolism. That would mean that the two groups both have the same frequencies of relevant genes, yet these genes direct different degrees (density differences) and types of bone development. The same genes have different effects in different environments.

Or, imagine that one or more genes relevant for bone development differ in frequency in these two populations. Consider also that individual bone cells—osteoblasts that build bone and osteoclasts that destroy bone—are jolted into activity in response to mechanical strains put on bones by weight-bearing activity (Mosley). Let's further hypothesize that a gene or genes that code for a highly sensitive mechano-receptor on the surface of osteoblasts is present in fairly high frequencies in African American populations. This hypothetical receptor responds to even very

low levels of mechanical stimulation by stimulating bone production. Let's further imagine that ancestral European populations carry a variant of this gene that is a little less sensitive to mechanical stimulation. If this were true (and nothing else affected bone development), and children from both populations grew up with identical levels of exercise and bone-stimulating activity, African Americans would end up with denser bones. On the other hand, if all European American kids spent their childhoods in intensive gymnastics training, while African American kids led less active lives, the bones of European ancestry children might be just as dense, or even denser, than the bones of children of African descent, despite the latter having genes that favor denser bone development. In both this and the previous example, difference and sameness result from a *gene-environment system* in which genes only assume importance when they respond to a particular environment and a particular environment changes the body by activating sets of genes.

Risch and his colleagues end their paper by relying on a second dichotomy, one between science and politics. Because racial and ethnic groups differ both genetically *and* culturally, Risch et al. find that such "groups should not be assumed to be equivalent, either in terms of disease risk or drug response" (11). A race-neutral or color-blind approach to biomedical research would be unable to eliminate health disparities. Note that they have built in an assumption that scientific research rather than social equality will illuminate the road to equal health status. They argue that identifying genetic differences among racial and ethnic groups is "scientifically appropriate," but that a "value system" attached to such findings is not scientific. They write: "The notion of superiority is not scientific, only political, and can only be used for political purposes" (11).

Leaning on a supposed science/politics dualism to avoid possible pernicious uses of allegedly "pure" scientific data is an old, well-meaning, but nevertheless toothless tactic. First, a great weight of historical evidence demonstrates that racial ideology has driven scientific belief for centuries (in this century see Gould; Haraway, *Modest-Witness* and *Primate*; and Kevles). It is naïve to imagine that our own attempts at pure science are any less tainted. While scientists will understandably want to keep their attention focused on the laboratory, they can (and some do) acknowledge that political questions of race drive our interest and shape our approach to the solution of certain biomedical questions. At the same time, we—scientists and social analysts alike—need to understand that in a racially divided world the knowledge we produce emerges from and

can, potentially, change the terms of the political argument. There is no doorway separating the Room of Science from the Chamber of Politics, no threshold where we can stand, neutrally protected from the pushes and pulls of either world. Science and politics are mutually constitutive and form a single system, and we do both elements of the system a disservice when we continue to pretend otherwise.

Racial Back-Talk

Both the popular press and the medical and genetics community have responded to Risch and his colleagues. Recent work has looked for a correlation between self-identity categories—most commonly the U.S. Census—and SNPs and haplotypes that vary by type and frequency in different parts of the world (Stephens et al.; Gabriel et al.). But do these results represent some artifact of the structure of the U.S. population that is "not to be taken as evidence that such differences represent any true subdivision of the human gene pool on a world-wide scale" (Pääbo)? A different approach to this problem entails looking at genetic variation in a wide variety of peoples sampled throughout the world without preconceptions as to the number of subdivisions one might expect to find.

Recently a group of geneticists published a large scale study using 377 genes from 1,056 people sampled from fifty-two populations found around the world (Rosenberg et al.; see also Bamshad et al.). Instead of SNPs, they examined stretches of DNA called microsatellites. This category of DNA varies a great deal and becomes a useful resource for comparing individual genetic structure. Instead of relying on information about sampling locations, these authors used a computer program designed to recognize subgroups with distinct allele frequencies. They asked the program to separate their data into any number of clusters from two upward. After the computer program produced the called-for number of clusters, Rosenberg et al. compared the groups to the known geographic origins of the individuals whose genes were examined. I have arranged some of their results in Table 1. Creating two clusters, which turn out to correspond to individuals from Africa and the Americas, suggests ancient human origins in Africa followed by rapid expansion through Eurasia and migration to the Americas (King and Motulsky). Creating five clusters reproduces the oft-cited subdivisions into groups that have lived for long periods in one of five major geographic divisions of the world. But move from five to six clusters, and the comforting correlations founder.

Table 1
Molecular variance worldwide.

From Rosenberg et al.

Sample	Number of Divisions	Cluster
Worldwide	2	Africa and America (North and South)
Worldwide	5	Africa, Eurasia, east Asia, Oceania, America
Worldwide	6	Africa, Eurasia, east Asia, Oceania, America, and Kalash language group
Africa	4	Mbuti Pygmies, Biaka Pygmies, San peoples and speakers of Niger-Kordofanian languages (Bantu, Yoruba, Mandeka populations)
Europe	No consistent subdivisions	
America	5	Karitiana, Sururi, Colombian, Maya, Pima
Oceania	2	Melanesian, Papuan
Middle East	5	Mozabite, Bedouin, Druze, Palestinian
East Asian	No consistent subdivisions	
Eurasia	No consistent subdivisions	
Central/South Asia	No consistent subdivisions	

Looking for genetic subclusters within continents, instead of dividing matters up worldwide, further complicates matters. Some geographic regions readily divided into genetic subclusters, as shown in Table 1. Other regions, even though they contain visually distinguishable individuals, are too genetically similar to subdivide (although the wonder of statistics is such that if much larger samples were used, some subdivisions would probably appear). Thus, for certain large geographic regions, what we know of a person's ancestry may tell us something about his or her genes. But genes and ancestors do not necessarily correlate with skin color, facial features, and hair textures, the visual signals of socially defined race.

Both Rosenberg et al. and commentators on this work used its publication as a chance to respond to Risch's nearly simultaneous opinion piece. Without ever introducing the word "race" into their publication, Rosenberg et al. comment that "as a result of variation in frequencies of both genetic and non-genetic risk factors, rates of disease [. . .] vary across populations [. . .] information about a patient's population of origin

might provide health-care practitioners with information about risk when direct causes of disease are unknown" (2384). Risch and others, they note, argue that self-reported population ancestry is preferable to genetically inferred ancestry as a way of discovering possible risk. Rosenberg and his colleagues on the whole agree, noting that "sizeable variation in ancestry within predefined populations was detected only rarely, such as among geographically proximate Middle Eastern groups" (2384).

Rosenberg and colleagues negotiate an odd switchback in the three paragraphs that address the use of self-reported population ancestry as a proxy for genetic ancestry. In their closing paragraph, they stress that the challenge is to use "the small amount of genetic differentiation among populations to infer the history of human migrations" (2384). The goal of their study, clearly, is to make inferences about "historical models of migration" and "human genetic history." This goal differs from the intention to use population groups in diagnosis and medical treatment. In this case, the authors note that a patient's population of origin is relevant "to both genetic and non-genetic risk factors," that is, the relationship between genes, culture, and medical risk is not generally untangled by either genetic or self-assignment to an historical population group. In a comment on Rosenberg et al.'s article, two other geneticists emphasize this point, writing that "the link between historical genetic demography and medically important risk is complex. Disease susceptibility may be genetic but not geographically clustered, or geographically clustered but not genetic, or neither, or both" (King and Motulsky 2343).

Thus, as we negotiate this thicket of papers about genes, human history, and disease, we begin to glimpse where and why we stumble and trip. On the one hand, we uncover fascinating information about human history—that populations originated in Africa and migrated out into the rest of the world in a fairly short period of time. We can trace the paths taken by varied cultural and linguistic groups as they moved to their current locations. But that information keeps getting tangled up with a wish to classify the world into the Big Few racial groups and to use that classification as a tool in medical practice. This is where we trip.

In a direct attack on Risch et al., a different group of medical scientists note that "categorizing people on the basis of differences in allele frequencies is [. . .] not the same as apportioning the whole of human diversity into medically relevant groups" (see Cooper, Kaufman, and Ward 1167). There are, of course, well-known genetic diseases such as Tay-Sachs

or hemochromatosis (a disease found in 7.5 percent of all Swedes) that are found only in certain populations, although the populations in question are not currently considered to constitute races. But these conditions are rare compared to the health disparities observed for widespread and appallingly complex diseases such as hypertension, cardiovascular disease, or diabetes. Worse yet, argue Cooper and company, there is no convincing evidence linking genetic differences to the differences in morbidity and mortality in European versus African Americans caused by any of a wide variety of chronic diseases.

Risch and his colleagues' reprise, a direct response to the Burchard et al. article, deftly sidesteps the question of how best to change the differential morbidity and mortality rates found between self-identifying members of the various Census categories. Instead, shifting registers once again, they maintain that "racial and ethnic categories are useful for generating and exploring hypotheses about environmental and genetic risk factors" and that barriers to collecting information about race and ethnicity will "retard progress in biomedical research," thereby impairing clinical treatments (1171). Here, the argument seems to be that the best way to eliminate health disparities is by the successful wedding of genomics to epidemiology. That it might *already* be possible to diminish health disparities, with little in the way of new research, never comes up for discussion.

The Risch Reprise acknowledges the difficult history of the race concept, admitting that it is tempting to purge it from our modes of thought, especially if we think that continuing to focus on racial difference may actually cause discrepancies in health status and disease. But as they close, Risch and colleagues offer an overly simplified gloss on a politically complex matter by referring to a California State ballot initiative called Proposition 54 (The Racial Privacy Initiative). These smart, thoughtful scientists seem politically tone deaf to the issues raised by Proposition 54. Tellingly, their brief mention of this political initiative in a paper in the *New England Journal of Medicine* forces us directly into the politics of race, a location supposedly well-insulated from the scientific arena.

The Ironies of Race-Based Medicine

When Risch et al. referred to Proposition 54, they seemed to suggest that the same people who opposed using racial categories in medical practice were linked in some fashion to a right-wing political movement to end affirmative action. Proposition 54, defeated in the California special election that also brought Arnold Schwarzenegger into the Governor's office, was one political tactic employed by this movement. The proposed amendment to the California State Constitution would have prohibited state and local government from "using race, ethnic, color or national origin to classify current or prospective students, contractors or employees in public education." Prop 54 was concocted and supported by a coalition of organizations and individuals opposed to affirmative action. The conservative support for the amendment is clear after only a few minutes spent surfing the links on the coalition's website (http://www.racialprivacy.org). Although Prop 54 specifically permits classification for medical research, Risch and colleagues imply that such a ban could be next if biomedical scientists deny the importance of racial difference for the analysis of disease formation. Risch thus unfairly connects scientific opponents of his position on the use of racial categories to a right-wing antiaffirmative action agenda supported by organizations such as the Sacramento Chapter of the California Federated Republican Women or the Florissant, Missouri Township Republicans (to name some of the more benign groups that supported Prop 54).

Many possibilities flow from the use of racial categories in the Census (see fig. 2). Most obviously, we need these categories to study health disparities. Such studies tell us that blacks die more often than whites, Hispanics, Asians, or Native Americans of heart disease, stroke, breast cancer, lung cancer, and HIV/AIDS, while they are tied with Native Americans for the highest death rates due to diabetes (Helmuth). But how to improve the health of minorities? The answer in 1990 was to create an Office of Research on Minority Health at the NIH and in 1993 to mandate that enough minorities (and women) be included in clinical trials to allow separate statistical analysis of results. Sociologist Steven Epstein suggests that these policies, which govern government-funded or approved production of biomedical knowledge, "emerge out of what might be called 'multi-representational politics.'" Such politics combines concerns about numerical inclusion with ones about political voice and the symbolism of racial inclusion (Epstein 175).

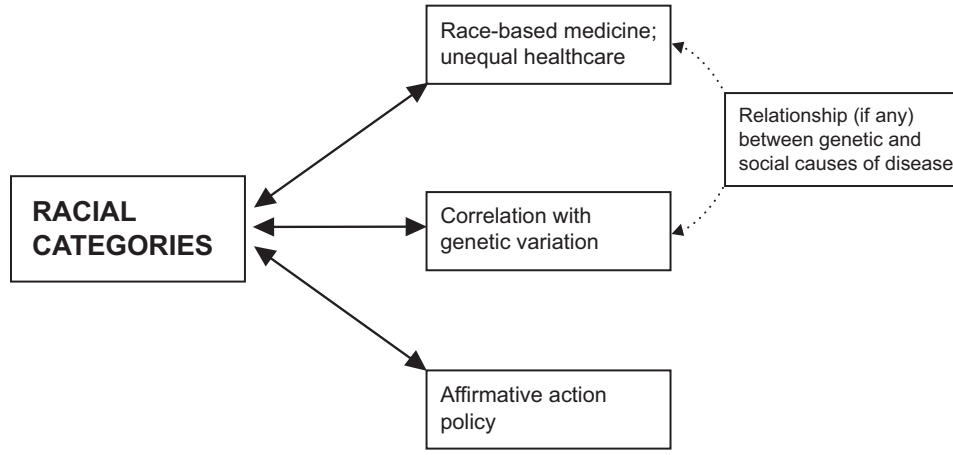

Figure 2
Policy vectors in the discussion of race and medicine.

Two related examples demonstrate how representational politics can play out in biomedicine. The first, a research letter to the journal *Nature Genetics*, examines the progress to date in mapping the more than five million SNPs with variations present (somewhere around the world) in a frequency of greater than ten percent. The authors of this letter assess the existing database containing the 2.7 million SNPs already recorded in order to determine whether it fairly represents the genetic variation found in European American and African American populations. They calculate that while the database contains almost eighty percent of SNPs common to European Americans, it has only fifty percent of those common to African Americans. In other words, the genetic variation in the giant SNP database *underrepresents* the variation found in African Americans. Until this underrepresentation is remedied, the authors argue, it will not be possible to use SNPs to detect possible genetic correlations with disease risk among African Americans (Carlson et al.). Here we see the claim that underrepresentation of genetic variation impedes the possibility of understanding the causes of disease formation and, presumably, our ability to remedy current health disparities. (Whether this form of genetic association is the best way to remedy health disparities goes unquestioned in this venue.) Similar assessments of the representativeness of the SNP database for populations outside of the United States are also starting to appear (Reich, Gabriel, and Altshuler).

As Epstein suggests, the question of who will speak for particular groups is fused with that of underrepresentation. This can be seen in the announcement that Howard University will develop and become home to a new DNA and health database derived from people of African descent. Founders of this so-called biobank, dubbed GRAD (for Genomic Research on the African Diaspora), hope, by 2008, to have collected DNA samples from 25,000 volunteers. GRAD will be the first biobank to look specifically at African Americans and will complement biobanks already planned or started in Europe and the United States. Here, the questions of representation and leadership merge, since this biobank will be directed by Dr. Georgia Dunston, an African American human geneticist at a historically black university. Dunston argues that this undertaking will help "disentangle genetics from socioeconomic and other environmental factors in understanding disease among African Americans" (Kaiser 1485). In forming GRAD African American health professionals take the lead in improving health care in "their" communities. On the one hand this step is more than appropriate, since it seems likely that these scientists will have better knowledge of health issues in African American communities, better insight into how to decrease racial disparities in health, and better access to the people under study. On the other, the establishment of the databank, at least symbolically, supports the idea of substantial biological difference among racial groups.

Once we are inside the genetic discourse, key assumptions about genes and disease formation become invisible. Focusing on the problems of representation and inclusion prohibits us from asking (1) whether knowing more about genes is the best or most efficient way to address health disparities; (2) whether we might reasonably expect *different* biological processes to underlie disease formation in different races; and (3) why the normal white male is not a good model for other groups. Given centuries of contrasting white male normality with female and racial deviance, contradicting this third assumption is more than a little appealing. But it is actually a position that requires dissection. And so, in the concluding portion of this paper, I will take scalpel and probe to these three often invisible suppositions that lie just beneath the surface of much biomedical research on race.

Genes and Health

There are, of course, genes, such as those for Tay-Sachs or Huntington's Disease, that directly and inexorably cause illness and death. Furthermore, genes such as these are relatively impervious to environmental modulation, and for this reason, it makes sense to "go after the gene" in order to figure out how to prevent or treat the disease. But single gene disorders do not account for the poor health of minority U.S. populations. For example, sickle-cell anemia, found more often (but by no means exclusively) in African Americans, explains only 0.3 percent of the greater number of deaths among African Americans (Williams, "Race"). The conditions of concern—cancer, hypertension, diabetes, among others—are of complex origin. Genetic variation may well be one component of some or all of these disease states, but does that mean that looking for "predisposing genes" is the best way to improve the health of large groups of people? Although there may be other legitimate justifications for such projects, spending lots of money on admittedly impressive and scientifically fascinating ventures such as the International Haplotype Mapping Project is not the most efficient or cost-effective way to improve the health of large numbers of people (Gura).[8]

Geneticists Kenneth Weiss and Joseph Terwilliger argue that the hunt for SNP-disease connections will be difficult, expensive, and not particularly fruitful. "No one can deny," they write, "that disease pathways have been identified by genetic epidemiology studies, making contributions to our biological knowledge. So far, however, it is lifestyle changes that have made the most impact on reduced (or increased!) incidence of chronic diseases" (Weiss and Terwilliger 155).

Which of the major diseases contribute the most to the death rate differentials? In one recent study, researchers adjusted for age, sex, and race in order to compare mortality rates between people who had finished high school and those who did not. These researchers found that people with less education lost three and a half times as many potential life years compared with those with more. The contributors to this life loss difference, in decreasing order of impact, were ischemic heart disease, lung cancer, stroke, congestive heart failure, pneumonia, and lung disease. Notably, these are all smoking-related diseases, suggesting that SNP mapping is the long, roundabout road to reducing mortality. Providing the means and motivation to stop smoking might, indeed, be a more efficient use of our health dollars. Hypertension was only a minor contributor,

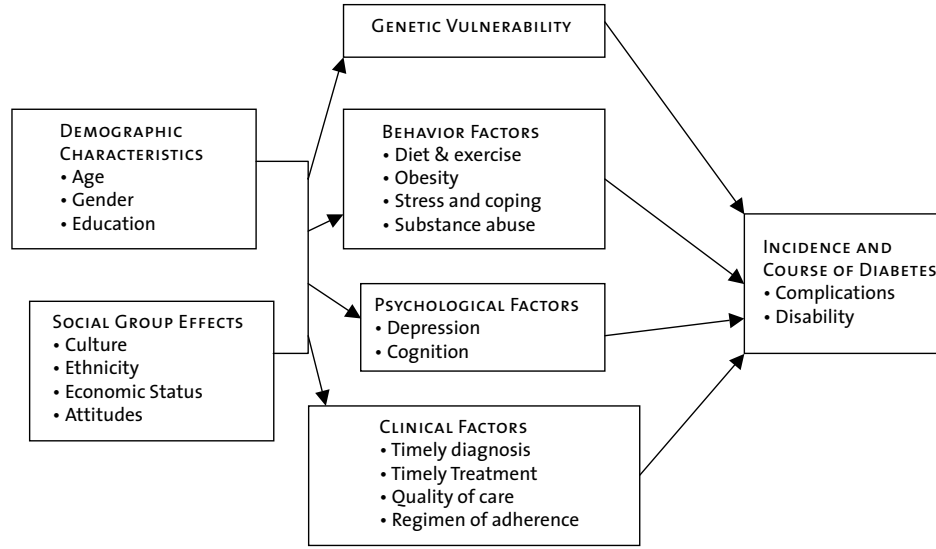

Figure 3
Conceptual framework of risk factors for the development of diabetes.

Adapted from Black.

and there was no difference in these two groups in life years lost due to HIV/AIDS (Wong et al.).

When these same researchers adjusted for age, sex, and education, they found that blacks lost thirty-five percent more potential life years than whites. In contrast to the results for education level, death from hypertension contributed the most to the disparity, followed by HIV disease, diabetes, and homicide. As the authors point out, dozens of studies examine racial differences in the treatment of heart disease, but "ischemic heart disease contributes only 5.5% to the total racial disparity [. . .] HIV disease and hypertension each contribute two to three times as much" (1590). Although I will return to hypertension in a moment, let me point here to the fact that HIV is a preventable and now treatable disease; again, we don't need to map more SNPs to address this significant contributor to mortality differences between African Americans and those of European descent.

Wong et al. have given us an idea of which diseases cause some people to die sooner than others, but they do not try to tell us how education level and race contribute to increased disease rates. However, frameworks to help answer such questions exist. In a recent analysis of factors contributing to diabetes, epidemiologist Sandra Black drew the diagram recreated in Figure 3 (Black).[9] Clearly, the social aspects of race, working via their effects on education, income, access to medical care,

and racism-related stress, contribute significantly to the production of diabetes. Furthermore, genetic vulnerability can only be understood in the context of all other risk factors. Critics—myself included—of the "all genes all the time" approach to eliminating health disparities caused by complex diseases argue that changing social contexts and relationships and improving income and education are at least as important for preventing and mitigating the effects of Type II diabetes as the pursuit of high-tech gene mapping strategies. Black, for example, suggests four basic public health measures that would, if well executed, have an immediate effect on racial differentials in diabetes morbidity and mortality (see also Williams, "Racial").

Epidemiologists and public health scholars also have an ongoing debate about use of the race word. This debate, however, is couched in an understanding of the social production of disease, rather than aimed at modernizing biological categories. Public health researchers consider the utility of social categories for the study of disease formation. Often the argument goes one step further, asking how belonging to a socially defined racial (or ethnic) category has *biological* effects that result in disease production. Participants in this debate distinguish between the use of socially defined racial categories for surveillance, that is, in the collection of statistics used to assess the health status of different population groups, and the use of race as a variable in public health research (Fullilove, "Fullilove"; Buehler).

One exchange of ideas held in the pages of the *American Journal of Public Health* in 1998 and 1999 offers instruction. Epidemiologist Raj Bhopal and Public Health physician Liam Donaldson (both of the University of Newcastle) note that only rarely has debate about racial labeling focused on the majority group (Bhopal and Donaldson). In the North American and British public health literature, synonyms for "white" include Caucasian, Anglo, European, and Western. Bhopal and Donaldson consider racial labels for white people "in the context of naming the population against which the health of racial and ethnic minority groups is compared" (1304). They point out that a U.S. Office of Management and Budget directive dating from 1977 defines a person as white if he or she comes from Europe, North Africa, or the Middle East, while in Britain Middle Easterners and North Africans are not counted as white. Given the wide range of ethnic and geographic backgrounds found among bureaucratically defined white people, Bhopal and Donaldson suggest that the label is of little value. "It encourages the division of society by skin color,

reinforcing racial stereotyping and hides a remarkable heterogeneity of cultures" (1304).

What, then, to call these pesky white people? Bhopal and Donaldson offer the idea that terms such as "reference, control, and comparison populations" might be of use. These, they suggest, carry no assumptions about race and ethnicity, reflect their use in statistical comparisons, and would require authors to offer some description of the origins of the varying groups of people under comparison. In a neighboring commentary, health researcher and Professor of Psychiatry Mindy Fullilove ups the ante, suggesting the complete abandonment of "race" as a variable in public health research ("Comment"). Much hullabaloo follows.

The traditional social science argument for racial classification is that it is a necessary prerequisite for studying the health effects of racism and racial discrimination. But, Fullilove argues, "[I]f racism is a principal factor organizing social life, why not study racism rather than race?" (1297). Why, she continues, use an unsound classification system for scientific research, and, finally, why accept a system that is not only biologically worthless, "but also full of evil social import" (1297)? Fullilove offers alternatives: study geography or "place" (see as examples studies of health effects of residential segregation: Gee; Krieger, Williams, and Zierler); study the specific behaviors, values and social networks associated with particular ethnic groups; and make equality a central research subject. Epidemiologists Nancy Krieger, David Williams, and Sally Zierler agree with the proposition that it is essential to study racism rather than race. But they dislike the idea of replacing the term "white" with the couplet "comparison group." This move, they suggest, consolidates "whites" as the norm, making other groups inherently something other than normal. Furthermore, they claim, such a move covers over a valuable research endeavor—studying variation in health within a particular group (e.g., comparing African Americans who do/do not smoke, or have/have not graduated from high school).

Similar discussions of how best to use racial or ethnic categories to study health disparity continue to dot the medical literature (see Bhopal; Bhopal and Donaldson; Kaplan and Bennett; Nazroo; and Williams, "Racial"). But only a few scientists have tried to understand the mechanisms by which the experience of racism translates into diverse disease states. The effects of racism will sometimes be indirect. In the case of diabetes, for example, housing segregation caused by red-lining or educational disparities that lead to lower income mean that individuals

may not have safe places to exercise and may not be able to buy healthy food. Many inner city areas lack supermarkets stocked with low carbohydrate, high nutrition foods at an affordable price. Limited access to health care exacerbates the problem. Diabetes is diagnosed late, making it that much harder to control and resulting morbidity that much more likely.

Diseases such as hypertension, however, may result directly from physiological responses to the daily insults of racism. In the first, and now classic, attempt to demonstrate such a direct relationship, Krieger and Sidney correlated elevated blood pressure with the experience and acceptance of or resistance to racial discrimination in black and white men and women aged twenty-five to thirty-seven. Black adults who reported challenging unfair treatment had lower blood pressures than those who accepted discrimination as a normal life event. Resistance to unfair treatment correlated with lower blood pressure in middle-class than in working-class blacks. A class difference also showed up in the comparisons of middle- and working-class whites (Krieger, "Racial"). The Krieger and Sidney study has the potential to open up a wide and fruitful arena for studying the mechanism of racism in chronic disease formation. Indeed, in the eight years since that publication, a number of additional studies have emerged, but, on the whole, these do not dig into the physiology of the matter in a particularly satisfying way (Harrell, Hall, and Taliaferro).

It is a mark of the hegemony of the gene's eye view of disease formation that even as I write about the direct and indirect effects of racism on human physiology, I feel compelled to reiterate that such studies do not suggest the complete irrelevance of individual genetic makeup. In fact, such indirect effects point to something quite interesting about genes: genes do not build organisms from the bottom up; rather, their activities are sandwiched somewhere in the middle of chains and networks of events that integrate organisms with their environment. Elsewhere, I begin to develop a systems theory approach to understanding how culture (broadly conceived) leaves material imprints on the body (Fausto-Sterling, "Bare Bones"). Here, I offer an increasingly accepted model of human physiology that suggests that so-called essential hypertension, the health scourge of African Americans, is in some sense a natural physiological response to the deprivations and stresses of being a person of color in America. In other words, it is not that *different* biological processes underlie disease formation in different races, but that different life experience activates physiological processes common to all, but less provoked in some.

For quite some time the idea that our bodies seek homeostasis, a stability regulated by feedback control, has dominated our accounts of physiology. The reigning metaphor is that of a thermostat: heat shuts off when room temperature rises above a set point and turns on again when the temperature falls below the set point. If blood pressure were controlled by homeostasis, then any time it went above or below a set point, homeostatic feedback mechanisms ought to bring it into normal range.

Constantly elevated hypertension not caused by something obvious such as a constriction in a major blood vessel is called essential hypertension, and as we have already seen, it has bad health effects. But it turns out that homeostasis is probably not a good model for how our bodies respond to daily events. In normal adults, for example, over a twenty-four-hour period blood pressure rises and falls rather predictably. Blood pressure does not seem to return to a single set point, but instead is regulated to match anticipated demand (Sterling). Returning to the thermostat metaphor, imagine that there were frequent changes in room temperature—the window opens, then shuts, a wood stove in the room belches out heat and then cools off; and imagine a "smart" thermostat that learns on the basis of past experience to anticipate such changes and raise or lower its temperature even before the expected event. This response, employing mechanisms that involve the brain and whole body and that change the controlled variable (be it temperature, blood pressure or insulin-controlled levels of blood sugar) by overriding local feedback controls to meet anticipated demand, has been called *allostasis*.

Although the concept has a longer history, the word, first coined in 1988 (see Sterling and Eyer; Schulkin) has gained in popularity among physiologists concerned with animals' physiological function in complex, often dangerous environments (see McEwen and Lasley; Schulkin). Allostasis depends on communication between so-called low level feedback systems that locally regulate physiological responses and higher order mechanisms in the brain. It is the brain that learns, often using the emotional responses of fear, joy, pleasure, and desire, to anticipate future events and to regulate (up or down) physiological responses in advance of such expectations.

The allostasis model does not ratchet up the hunt for a "broken" gene to explain essential hypertension. Instead, it proposes that hypertension is an orchestrated response to a predicted need to remain vigilant to a variety of insults and danger—be they racial hostility, enraging acts

of discrimination, or living in the shadow of violence. Over time, all of the components that regulate blood pressure adapt to life under stress. As neurophysiologist Peter Sterling writes, "although the endpoint may be tragic, every step along the path seems perfectly 'appropriate'" (17). The framework of allostasis, in which pathologically high blood pressure emerges as a step-wise, cumulative, and predictable response to life stress, provides a very different way to investigate the relationship between race and hypertension. It suggests developmental research, for example, starting with studies of the relationships between important childhood stressors and incremental hypertensive responses. But what does the allostasis model suggest about interventions?

Refashioning Race

The logic of searching for genes that contribute to complex diseases runs something like this: if we find contributing genes, these may help us develop drugs tailored to the disease. If some key genes can be found in higher frequencies in certain populations, then we can tailor drug formation to those groups. This is the so-called designer drug argument, which, I should mention in passing, has had little success to date (see Goldstein, Tate, and Sisodiya; Haga and Ventner; Heinrichs; Kahn; and Shields). Existing drugs, which are not group specific, aim at targets low down in the command and control net and, in accord with the homeostasis model, aim to restore blood pressure (or another item of concern, e.g., glucose level in the case of Type II diabetes) by reducing blood volume (diuretics), widening the diameter of blood vessels (vasodilators) or reducing the output of the heart (via cardiac antagonists). To understand this point further, consider the diagram in Figure 4. Drugs aimed at local feedback controls would affect blood pressure by addressing cardiac output, blood volume, or the width of relevant blood vessels. But these do not solve the problem if the higher control levels, the brain and the hormone systems it controls, continue in a hyper-alert state—sending signals out to the lower systems and eventually changing the blood pressure set point in much the same way our intelligent thermostat anticipates the upcoming temperature and overrides local control.

Using drugs to address the local manifestation (e.g., constricted blood vessels or too much blood volume) of a higher-level problem is a bad idea. First, because of their location in the web or cascade of effects, they cannot act specifically, and they thus cause new medical problems as they

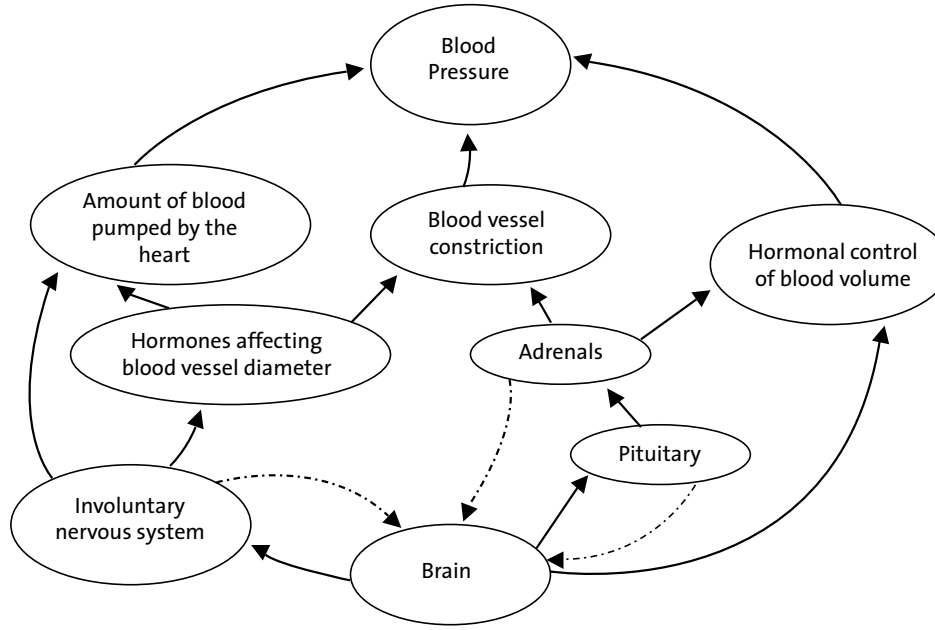

Figure 4
"The brain sets blood pressure via multiple, mutually reinforcing mechanisms" (Sterling 6).

This diagram is a simplified version of events depicted in Sterling's Figure 4.

attempt to solve old ones. Second, lower-level treatments make matters worse because the brain has learned to expect a need for hypervigilance, and once established, it orders the local system to maintain high blood pressure. Thus, each time a lower-level treatment decreases blood pressure, the brain tries all the harder to increase it. It is a smart thermostat gone wild. Third, damping down a lower-level physiological response messes up the local feedback system, making it insensitive to predicted need, even though such sensitivity is the point of physiological regulation in the first place. For these reasons, hypertension is controlled in fewer than twenty-six percent of hypertensive patients who use pharmacological therapies (Sterling).

In contrast, the allostasis model emphasizes higher-level interventions, adjusting entire systems of physiology and attending to the emotions and unmet needs that produce the negative physiological results of hypervigilance. Over the long term, a shift in the expectation for hypervigilance ought to reset the body's response systems, allowing function in a range less likely to produce excess morbidity and mortality. Current "lifestyle" recommendations include a substantive change in diet, weight loss, moderate exercise, and reduced alcohol intake, all of which are at least as effective as drug therapy for some levels of hypertension (Sterling).

Sterling notes that "the most successful interventions do not deny the sense of need. Rather they find ways to satisfy it by enlarging positive social interactions and revivifying the sense of connectedness" (28).

If the allostasis model is right, and if, as argued earlier in this paper, we already know how to solve a lot of the public health problems before us, including those that exact an excess toll from various minority communities, then why aren't we just doing it? And why are geneticists spending so much time on huge, multinational SNP and haplotype mapping projects, promising that their new definitions of race will help members of the newly defined racial groupings to improve their health? An analysis of our health care system's love of high-tech solutions for low-tech problems, of our national unwillingness to fund and carry out well designed public health measures, and of the force of the pharmaceutical industry as it drives for pill-based solutions to socially produced ills is much more than I can accomplish in this paper. Not here, not now, but . . .

The medical promises for genetic race talk are overblown; the genetic refashioning of race is in large measure 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. The rewriting of biological race has other supporters as well. If, indeed, there are to be great benefits from analyzing the human genome, then underserved minorities justifiably want a piece of the pie. In fact, they want to have a hand in baking the pie, hoping that doing so will ensure a baked good more to their taste. This is the import of plans such as the Howard University-based GRAD project. Finally, the pharmaceutical industry hopes to engineer enormous profits by producing new drugs. It won't matter that many of these drugs will do little to solve the large-scale health problems. Sales will still be brisk.

Some fear that the genetic study of difference will create newly stigmatized populations, lead to genetic discrimination in the workplace, or simply reinforce old prejudices, making it more difficult than ever to address the social problems of racial discrimination in all of its negative aspects. While there is probably merit to each of these fears, I don't think they are the main problem. It is not what the new biology of race will produce so much as it is that the new biology of race diverts our attention from solving problems using solutions we already have at hand. It does so in part by insisting that genes build bodies outside of culture and then deposit them on earth, where social systems tinker with them just a bit.

We need, instead, to develop the habit of thinking about genes as part of gene-environment systems, operating within networks that produce new physiologies in response to social conditions. In this view, bodies are not static slaves to their biology. Rather, it is our biological nature to generate physiological responses to our environment and experience. We use genes to produce such responses. This understanding of the relationship between the social and the biological gives us new epistemological set points that would drive us to seek social solutions to health disparities, using technology as an aid but not as the motor. This would be a welcome change indeed.

I would like to acknowledge the advice and comments of Lundy Braun, Molly Przeworski, and the editors of differences*, especially Ellen Rooney.*

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Notes

1 For a discussion of both God and the devil, see Gould.

2 Electrophoresis uses a sorting medium such as paper or a porous gel placed in an electrical field. Depending on their amino acid composition, proteins have a net electrical charge and will migrate toward or away from the positive or negative pole until they reach equilibrium with their charge. Hence similar proteins, which have had small changes in their amino acid sequences during the course of evolution, can be separated from one another. This allows scientists to study variation in many different groups of proteins, not just those associated with blood type.

3 See, for example: http://alfred.med.yale.edu/alfred/ethics.asp or http://www.ncbi.nlm.nih.gov/SNP/.

4 There are many reasons for such neutrality—the most common being that mutations occur in those vast stretches of DNA which never produce key proteins or RNAs and thus do not alter development.

5 Each parent produces a gamete which is haploid and when the gametes fuse they make a diploid embryo.

6 These are: sub-Saharan African, Caucasian (Europe, Middle East and North Africa), Asian, Pacific Islander (e.g. Australian, New Guinean and Melanesian), East Asians and Native Americans (including both North and South America). Risch categorizes Africans as those with primary ancestry in sub-Saharan Africa, including African American and Afro-Caribbean; Caucasians include those with ancestry in Europe, the Middle East, and West Asia, including the Indian subcontinent; Asians are from eastern Asia, including China, Indochina, Japan, the Philippines and Siberia; Pacific Islanders are

those indigenous to Australia, Papua New Guinea, Melanesia and Micronesia, as well as other Pacific Island groups further east, and Native Americans are indigenous populations of North and South America.

7 By contrast, the high frequency African genes are admixed in the European-descent gene pool in the United States at the rate of only about 1%. The study also found, not surprisingly, that European men contributed most of the European genes to the African American population.

8 I hasten to point out that many of the hap-mappers agree with me on this, but Risch's point of view has been more widely disseminated.

9 They defined life years lost as the difference between age at death and the maximal number of years a person could have lived (set to seventy-five years), that is, anyone living more than seventy-five years lost no life years.

10 It is of interest that this article appears in a special issue of the *American Journal of Public Health* devoted to the health of women of color.

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