**Race, Ethnicity, and Health**

can genetics explain disparities?

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**ABSTRACT** Over the past decade, numerous studies have documented profound racial and ethnic disparities in disease in the United States. This essay examines how popular and scientific concepts of race and ethnicity converge with dominant understandings of genetics to inform the design and interpretation of research, public health policy, and medical practice. Although there is some acknowledgment in the biomedical community that racial and ethnic categories are social and not genetic, ideas about race and ethnicity that circulate in biomedicine are contradictory. Thus, in practice genetic explanations for observed differences are common both in the scientific literature and in popular media accounts of biomedical research. Such explanations naturalize racial and ethnic difference and create a conceptual barrier to developing a research program that explores the complex ways in which social inequality and experiences of racial discrimination interact with human biology to influence patterns of disease. Importantly, genetically based ideas lead to disease prevention policies that are bound to be ineffective.

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Since the creation of the Office of Research on Minority Health in 1990 by the Director of the National Institutes of Health (NIH), a series of initiatives to include minorities in clinical research and to increase training of minority health professionals has focused attention on racial and ethnic disparities in health in the United States (ORMH History 2000). In 1998, President Clinton set the goal of eliminating disparities by the year 2010 (Eliminating Racial and Ethnic Disparities 2000). As a result, reports documenting racial and ethnic differences in incidence, mortality, and severity of disease appear on a regular basis in the biomedical literature. This research emphasis is important, but insufficient attention has been paid to the meaning attached to reported differences in patterns of disease and, importantly, to how these differences are interpreted and transformed into public health policy. Observed differences are generally explained through two dominant frames: one emphasizes genetic susceptibility to disease, while the other focuses on cultural practices. In both cases, it is minority communities—their inheritance or their culture and behaviors—that are seen as problematic. Rarely is the relationship among social conditions, power relations, and health the focus of study.

In this essay, I examine how social and scientific meanings of race and ethnicity have been constructed historically and how genetic explanations shape the design and interpretation of research on the health of racial and ethnic minorities. I argue that the current research emphasis on genetic explanations for disease disparities is problematic, since race and ethnicity are social, not genetic, categories comprised of individuals whose ancestry is highly diverse. Therefore, social explanations, not genetic ones, for disparities are most informative. Moreover, I argue that the knowledge of the history of human populations and how their group identities and affiliations change over time is too crude to correlate with gene frequencies and therefore is not helpful for understanding disparities. Finally, I argue that the current emphasis on genetic explanations reifies racial and ethnic classifications by reinforcing the notion of biological difference rooted in genetics. This reification leads to stigmatization of racial and ethnic minorities and to research strategies that divert attention from confronting the multidimensional ways in which racism, not race, influences patterns of disease.

**Historical Perspectives on Race and Science**

The concept of race as representing natural “types” which could be distinguished on the basis of visible attributes such as skin color, hair texture, and facial features was first elaborated in the 17th century (Schiebinger 1993). It was not until the 18th and 19th centuries, however, that the full force of scientific authority was marshaled to fix the boundaries of racial classification. Notably, the scientific work done during this period provided the intellectual underpinnings for the broad-based eugenics movement for improvement of the human “race,” which
was centered in the United States and Europe (Duster 1990; Kevles 1995; Pernick 1997). The appeal of eugenics extended to many sectors of privileged society, including social reformers, public health leaders, physicians, and, significantly for the codification of eugenic ideas into sterilization laws, legislative and judicial officials. Indeed, the history of public health and eugenics is a complex one, marked by many common goals and shared values about the meaning of race, class, and ethnicity, and the application of these meanings to disease prevention policies (Pernick 1997).

Medical interest in the health of racial and ethnic minorities historically has reflected concerns about the social order and, at the same time, helped to construct popular understandings of race (Wailoo 1997, 2001). In the antebellum period, for example, detailed anatomical studies conducted by scientists, many of whom were physicians, played a central role in representing blacks as inferior, inherently diseased, and, as polygenists such as Samuel Morton argued, members of a different species (Gould 1981). With the emancipation of slaves after the Civil War, Southern physicians set about collecting more evidence to demonstrate scientifically the innate inferiority of blacks and their lack of fitness for freedom and citizenship. Frederick Hoffman, the chief statistician for Prudential Insurance Company of America, was a particularly vocal spokesperson for this view (Hoffman 1896). As Haller (1970) notes in his study of medical notions of race in the late 19th century, physicians in the post–Civil War south drew on Darwinian ideas of evolution and natural selection to argue that inherited susceptibility to diseases as varied as rickets, pneumonia, and tuberculosis would ultimately lead to the elimination of the black race, thereby solving the “race problem.” Continuing in this vein, laboratory researchers in the early 20th century studying the biology of blood and blood diseases expanded on racialized theories of disease causality based on inherited predisposition to disease (Wailoo 1991). In some cases, such as malaria, blacks were considered to be resistant to disease, thereby justifying their ability to work in malaria-infested regions. Nonetheless, by locating disease in physiologic difference—be it susceptibility or resistance—medicine served to mark blacks as deserving of their inferior social status in society.

In the early 20th century, the U.S. Public Health Service (USPHS) played a central role in the inspection and certification of newly arrived immigrants for entry into the United States, classifying national and ethnic groups as biologically and culturally distinct races—Celts, Slavs, Hebrews, Mediterraneans, Alpines, Nordics, Asians, Negroes, and so on, with the Anglo-Saxon “race” at the top of the evolutionary scale (Jacobson 1998). Race thus became a key predictive marker for disease status and social utility, and the centrality of the disease experience of undesirable or unfit “races” was a dominant feature of popular discourse that culminated in the Immigration Exclusion Act of 1924 (Kraut 1994). Likewise, the infamous and, despite some arguments to the contrary, generally
discredited Tuskegee experiment that followed the natural history of untreated syphilis in black men in Alabama, was conducted by the USPHS from 1932 to 1972 (Benedek and Erlen 1999; J. H. Jones 1981; Reverby 2000). The motivations for the Tuskegee experiment were complex, but one of the underlying assumptions that informed the study was the idea that blacks and whites were biologically distinct.

Following World War II, Nazi atrocities led to a period in which social theories prevailed over hereditary explanations of disease inequities (Duster 1990). Nonetheless, the stage for the current resurgence of interest in genetic explanations for racial differences in patterns of disease was set with Linus Pauling’s description of sickle-cell anemia as a molecular disease (Pauling et al. 1949). Because sickle-cell anemia had been understood as a race-specific hereditary disease since the 1920s, the publication of this article brought socially and politically charged concepts of race and disease together with the emerging field of molecular genetics. In ways that were invisible to researchers at the time, “Negro blood” would become “black genes” (Wailoo 1997).

The Office of Minority Health was established at a particular historical moment when vast resources were being dedicated to the project of sequencing the human genome. At the same time, health and many other social programs for the poor that were the legacy of the New Deal and the 1960s War on Poverty were being dismantled with stunning speed and lack of sensitivity for the people affected. In this context, social understandings of health were increasingly subsumed by genetic explanations. Thus, as was true earlier in the century, questions about the meaning of race, and increasingly ethnicity, are again explicitly linked with ideas of biological difference between racial and ethnic groups in both scientific and popular thought.

**Race, Genetics, and Defining Populations**

The understandings of race and racial classification systems that inform biomedical research draw on decades of scholarship, marked by contentious debates, in the humanities and social sciences as well as the natural sciences. While some continue to make extreme arguments for a “gene-based evolutionary theory” of race (Rushton 1995), the consensus among social scientists is that race is a socially produced, not a genetically determined concept (Smedley 1993). This view is captured in a recent statement issued by the American Anthropological Association: “Racial beliefs constitute myths about the diversity in the human species. The myths fused behavior and physical features together in the public mind, impeding our comprehension of both biological variations and cultural behavior, implying that both are genetically determined” (AAA 1999).

Research within the biological sciences has also provided strong evidence that human genetic diversity cannot be partitioned into genetically determined racial
categories. For example, using internal proteins—blood group proteins, serum proteins, and red blood cell enzymes—as markers of genetic variability, the population geneticist Richard Lewontin (1972) demonstrated that over 85 percent of the observed genetic variation occurred within racial groups, only 6.3 percent of variability occurred between racial groups, and approximately 8.3 percent of variability between population groups within a race. In other words, there is more genetic variation within than between different racial and population groups.

One could argue that the proteins selected by Lewontin for analysis were not representative of the biological diversity of the human species. Had Lewontin studied the distribution of proteins involved in melanin production, for example, the allelic frequencies might have shown a different pattern. However, Lewontin’s conclusion that human races cannot be defined genetically has been confirmed by more recent work of Barbujani et al. (1997) who used 109 polymorphic DNA sequences (30 microsatellite loci and 79 polymorphic restriction sites) in 16 populations as markers of human genetic diversity. In agreement with Lewontin, these investigators found that 84.4 percent of genetic diversity occurred within populations, noting that even small populations were highly diverse. On the basis of their results, they conclude: “If loci showing a discontinuous distribution across continents exist, they have not been observed in this study.” They go on to state that “this study shows that previous findings of large individual diversity within populations were not due to the particular nature of the markers chosen, normally frequencies of protein variants at biallelic loci” (p. 4518). Others researchers have reached similar conclusions using computer modeling (Templeton 1999). Taken together, this work leads to the conclusion that racial groups do not represent reproductively isolated gene pools that can be identified on the basis of the frequency distributions of genetic markers.

Drawing on this and other similar scholarship, both the President’s Cancer Panel and the Institute of Medicine (IOM) have entered the debate, concluding that racial classification systems represent historical/political/social categories based on perceived differences (Freeman 1998; IOM 1999). A review in Science supporting this view concludes with the statement that “the myth of major genetic differences across ‘races’ is nonetheless worth dismissing with genetic evidence” (Owen and King 1999, p. 453). Leaders of the Human Genome Project, such as J. Craig Venter of Celera Corporation, have publicly stated that “Race is a social concept, not a scientific one,” and Eric S. Lander argues that: “There’s no scientific evidence to support substantial differences between groups . . . and the tremendous burden of proof goes to anyone who wants to assert those differences” (Angier 2000). Editors of leading scientific and public health journals have contributed in important ways to this debate by featuring discussions about the meaning of race.

Nonetheless, despite the acknowledgement by some in the biomedical community that race is not a genetically meaningful concept, the belief that the
genetic variability of humans can be partitioned into categories based on frequency distributions of polymorphic genes is a powerful notion that remains deeply entrenched in the thinking of biomedical researchers. This is evident in recent comments by Lander who, while affirming that concepts of race are simplistic, nonetheless argues for recording identifiers [for ethnicity] "to be sure to serve those populations medically" (Wade 2001). In her analysis of the heated controversy over the Human Genome Diversity Project, Jennifer Reardon (2001) argues that such contradictory perspectives on the meaning of racial and ethnic classification represent a continuation of mid-20th-century controversies between classical Mendelian geneticists and evolutionary biologists that centered on questions regarding the level—individual or group—at which evolution operates to produce human genetic diversity. Central to the debate at that time was the question of whether races are reproductively isolated groups that share a common gene pool.

In the United States, the debate in biomedicine over the appropriate categories of analysis has, to a certain extent, shifted from race to ethnicity. The IOM (1999) has proposed explicitly that ethnic categories be substituted for racial categories in health research. Defining the boundaries of ethnic groups is presented as unproblematic—or at least less problematic than defining the boundaries of racial groups. But that the substitution of ethnicity for race does not resolve the problem of whether ethnic populations are genetically similar is revealed when researchers attempt to define ethnicity. According to one researcher, ethnicity is seen as: "a broad concept that encompasses both genetics and culture . . . Ethnicity is about phenotype and genotype, and, if you define the terms of your study, it allows you to look at differences between groups in a valid way" (Angier 2000). A genetic basis for ethnicity—although not an exclusively genetic one—is clearly embedded in this definition.

The process of "defining the terms of study" is an attempt to delimit the boundaries of ethnic groups, or more ambiguously "populations," by making direct correlations between molecular findings and existing knowledge of the history, languages, migration patterns, and reproductive practices of human populations. This interdisciplinary research presumes that the boundaries of ethnic groups can be stabilized on the basis of meaningful and agreed-upon criteria derived from expert knowledge. A leading proponent of this view is Luigi Luca Cavalli-Sforza, who has written several popular texts as well as the ambitious treatise on genes and populations The History and Geography of Human Genes (Cavalli-Sforza, Menozzi, and Piazza 1994). In his critique of The History and Geography of Human Genes, the archeologist Scott MacEachern (2000) has shown just how crude is the conceptual scheme that Cavalli-Sforza has applied to human populations. In particular, MacEachern argues that the sweeping generalizations made by Cavalli-Sforza and colleagues are predicated on the problematic assumption that language, ethnicity, and gene frequencies have evolved in parallel. In particular, MacEachern points out that in the context of Africa, where
colonialism played such a central role in shaping ethnic identities (and in generating ethnic rivalries), "it is abundantly clear that many of the 'tribes' so beloved of (even modern) Western commentators are not entities preserved unchanged from ancient times but rather the relatively recent products of intense participation in regional networks of political, social, and economic interaction" (p. 362). In other words, the historical, anthropological, and linguistic definitions of "populations" with which genetic findings are correlated represent superficial understandings of the dynamic history of present-day ethnic populations and how these populations were formed.

Another way to look at this problem of defining the boundaries of population groups is to consider the variability in methods of racial classification in different countries and how these classification systems change over time. In South Africa, for example, the Apartheid government legally mandated that people be grouped into categories referred to as "coloured," "native" (variably referred to as "African," "Bantu," or "black"), "white" or "European," and "Asian/Indian" (Populations Registration Act 1950). However, individuals identified as "coloured" in South Africa may, in the United States, self-identify or be identified as "black." In her comparison of racial categorization in the U.S. and Brazilian censuses during the 19th and 20th centuries, Melissa Nobles (2000) has shown that census categories for race and color shift in relation to changing political and social circumstances. In other words, the differing historical contexts in the United States, South Africa, and Brazil shape what we see, how we classify, and—importantly—what uses we make of those classifications. What is common to all contexts, however, is that the extraordinary diversity of people in each of these categories is rendered invisible by social, cultural, and political assumptions.

Thus the social power of perceived differences has made it difficult to gain clarity on what is meant by race and how racial categories are used. Multiple, frequently conflicting, and generally implicit understandings of the concepts of race and ethnicity circulate in biomedical circles, with some researchers proposing that race has no genetic meaning, others arguing that the estimated 5 to 6 percent genetic difference is sufficiently meaningful biologically to justify an intensive research program, and still others arguing that the whole controversy can be circumvented by substituting ethnicity for race. As pointed out by the editor of Nature Genetics: "At present, the messages are mixed: on the one hand, the public is told that there is no scientific basis for race and that there is more variation within populations than between populations. On the other hand, scientists use racial terms when describing research results, such as increased risk for breast cancer in Jews or for prostate cancer in Blacks, and frequently emphasize population-specific markers, alleles and disease susceptibility" (Editorial 2000, p. 98). We are thus left with a wide gap between theoretical understandings of the meaning of race and ethnicity and the use of these understandings by biomedical researchers.
The term *genetics* also has multiple meanings in biology, and these multiple meanings have contributed to the problem of interpreting racial and ethnic inequities in health (for a theoretical discussion of genetics, see Smith 1992). Broadly stated, *genetics* refers to the study of genes and genetic variation at the level of populations, organisms, cells, or DNA sequences. Mutations in genes can occur in somatic cells due to exposure to carcinogens—as is the case with most human cancers, for example—or in germ cells, in which case the mutation can be transmitted from parent to offspring. When invoked to explain racial disparities in disease, however, the term *genetics* generally implies inherited predisposition to disease.

Genetic explanations for racial and ethnic differences in incidence, mortality, and severity of a wide range of diseases are consistently invoked in the biomedical literature, in leading textbooks used in medical training (Cotran, Kumar, and Collins 1999), and in the popular press. These diseases include systemic lupus erythematosus, diabetes mellitus, prostate cancer, breast cancer, asthma, and cystic fibrosis, to name but a few (Gene Factor 1996; Hamosh et al. 1998; Hsu, Glaser, and West 1997; Lara et al. 1999; Mijovic et al. 1991; Morton 1994; Moser et al. 1998; Taioli et al. 1995). In some cases, inherited genetic differences between races are mentioned explicitly. Other times, as in a study that explains differential responses of Asian and white men to hormonal contraceptives as due to “inherent ethnic difference” (Johnson et al. 1998), the language used to explain difference is more indirect. Most commonly, genetic explanations are either included as one of a long list of possible explanations or researchers fail to state their hypotheses, leaving readers to simply make up their own (Osborne and Feit 1992).

In making the argument that racial and ethnic disparities in disease are rooted in genetic differences between races, scientists frequently cite the prevalence of sickle-cell anemia in American and West African blacks. For several reasons, however, using sickle-cell disease as a model for understanding racial disparities in disease is problematic. First, it confuses the idea of group identity with group ancestry. The group identity captured by the category “African American” does not reflect a single path of ancestry. African Americans have many different ancestries, as do Africans, as do whites, as do Asians, and so on. For example, I self-identify as Irish American for social and cultural reasons, including the tall tales of an Irish American grandmother. This self-identification has little to do with what is a much more complex tapestry of ancestry. If individual identity is complex, group identity is infinitely more complex.

By identifying sickle-cell anemia with African Americans and black West Africans, its prevalence in a wider range of peoples, such as Latinos, inhabitants of northwestern India, and inhabitants of areas around the Mediterranean, was rendered invisible. This invisibility was further reinforced by the convention of plac-
ing this hemoglobinopathy in a category separate from the thalassemias, a heterogeneous group of red blood cell disorders, some of which are also caused by mutations in the $\beta$ chain of the hemoglobin molecule and which produce anemia of variable severity. Consequently, many in medicine are unaware today that the town of Orchomenos in central Greece has a rate of sickle-cell anemia that is twice that of African Americans, whereas black South Africans do not carry the sickle-cell trait (Kevles 1995). Like the $\alpha$- and $\beta$-thalassemias, hemoglobin S correlates with the geographic distribution of malaria, not with race. Sickle-cell anemia is not a "race specific" disease (Editorial Comment 1947) that reflects the underlying genetic make-up of West Africans and African Americans. The higher prevalence of sickle-cell disease in African Americans is in a very important sense an artifact of classification. In Brazil, for example, where many African Americans would be considered white the prevalence of a disease such as sickle cell might be very similar between people considered black and people considered white. By virtue of ancestry, not all African Americans are at risk of sickle-cell disease, whereas some white populations and Latinos have a risk which is not well documented. The frequency of alleles coding for hemoglobin S or particular Duffy blood group antigens (Hamblin and Di Rienzo 2000) or other polymorphic alleles thus cannot be used to construct a category termed "African American" or "black."

Finally, sickle-cell disease itself is a poor model for most diseases that affect humans. Like most human traits, the vast majority of human diseases, such as Alzheimer's, cancer, and heart disease are etiologically and clinically more complex than the so-called monogenic disorders, although the molecular basis of even monogenic disorders is turning out to be surprisingly complex. In the classic Mendelian disorder cystic fibrosis, mutations in the transmembrane conductance gene are often not predictive of pulmonary disease, pointing to the existence of modifier genes and environmental factors in the pathogenesis of this disease (Genetic testing 1997). Similarly, in the case of the phenylketonuria phenotype, the mutant genotype is not a reliable predictor of phenotype at the enzymatic, metabolic, or cognitive levels (Scriver and Waters 1999).

While genetic predisposition may explain some of the variability in the presence and severity of disease in individuals, the question that has to be addressed is whether genetic predisposition is a useful explanation for racial and ethnic disparities in disease. If racial and ethnic groups (except, perhaps, very isolated populations) do not represent distinct gene pools, then genetic explanations for health inequities are weak, if at all, informative.

**Biological Expression of Life Experience**

If the evidence indicates that there is no genetic essence to the conventional categories of race and ethnicity, a critical question to pose is: can racial and ethnic categories nonetheless still be useful in studying the health of populations? If we
think of race and ethnicity as social instead of genetic constructs, what light do these constructs throw on matters of health and disease? Although a full discussion of the construction of racial and ethnic identities is beyond the scope of this essay, the idea that race and ethnicity are socially produced—or “folk-theoretical” (Appiah 1996) systems of classification—holds that these categories reflect distinctions among peoples, the languages they speak, the identities they craft, and the institutions and cultures they create which are dynamic, shifting throughout history and across cultures. Racial and ethnic categories, therefore, cannot be fully understood outside of the ways in which the process of categorization is materialized in the everyday life of people and the political uses the categories serve.

Accordingly, perceived differences can have real consequences. Rather than genetic groupings, racial and ethnic categories as currently constituted in the United States (and throughout the world) embody profound inequities in access to political power and wealth, experiences of racism, and systems of privilege, all of which affect the lives of racial and ethnic minorities—and which have profound effects on health. The epidemiologist Camara Jones (2001) has described three levels of racism—institutional or structural, personally mediated, and internalized—all of which influence health and all of which require systematic investigation. Thus, in this particular historical context, these unstable systems of classification, including the theoretically inadequate distinctions between racial and ethnic groups, together with measures of social class (Davey Smith 2000), represent significant though crude indicators of health status and remain important to understanding and correcting the injustice that leads to disparities in disease. The usefulness of racial and ethnic categories, though, depends on historical circumstances. In another context, such as that of South Africa, where the continued use of racial classification has the potential to reinforce the divisions that are the legacy of the brutality of the Apartheid era, a different approach to the use of these categories in health research might be considered.

As currently framed, much basic science research on health disparities reduces biology to molecular genetics. This reductionism ignores the dynamic nature of populations, of DNA, and of the complex relationship among genes, organisms, and environment (Lewontin 2000). Instead of a single-minded focus on identifying race- or ethnic-specific polymorphisms, or on studying cultural behaviors which fail to acknowledge the diversity and changing character of culture, an alternative framework of analysis would examine the potential biological mechanisms through which life experience can affect health. Such an analysis would explore the dynamic nature of the relationship between humans and their social and physical environment and the effects of this interaction on the expression of genes, rather than ascribing an exaggerated biological meaning to a string of nucleotides. Such an analysis has the potential to provide explanations for differences in prevalence of disease or disease-related mortality, as well as puzzling features of the natural history of disease, such as the earlier age of onset of breast...
cancer in African Americans. In other words, causal explanations of disease would look at the disease experience of individuals or socially constructed population categories in the context of, not separate from, the social and physical environment.

For example, it is not difficult to imagine that in many circumstances people of high social class are more likely to have the economic resources to maintain a nutritious lower-fat diet than people with lesser means. Low-fat diets or diets rich in a variety of nutrients might influence health (although undoubtedly in much more complex ways than generally appreciated), by modulating the levels of hormones or other biologically significant proteins which, in turn, could regulate gene expression (Rose et al. 1987). Thus, social class could affect human health by altering cellular physiology. Viewed from this perspective, one could also envision that a range of life experiences, including that of racial discrimination based on perceived differences, might also compromise health through physiological mechanisms that are not inherited. This approach would help explain how social inequality works to compromise health.

Intriguing evidence to this effect was provided by Krieger and Sidney (1996) in their study of hypertension in African Americans. These investigators developed measures for both the experience of and the response to racial discrimination and concluded that physiological effects of racism contribute to the high rate of hypertension in African Americans. The evidence that racial discrimination might have biological consequences not mediated through inherited difference in genetic material was heralded by scientists and the popular press as providing new insight into the pathogenesis of hypertension. However, the relationship between racism and hypertension remains understudied.

Unless we are clear about the meaning of race, interpretations of basic mechanistic studies of diseases such as hypertension can easily imply, if not explicitly state, genetic difference. For example, a recent study which was widely reported in the popular media proposed that racial differences in prevalence of hypertension could be due to an abnormal hemodynamic response in African Americans during stress mediated by endothelium-derived nitric oxide (NO) (Cardillo et al. 1998; Health Watch 1998). Through careful experimentation (although the sample size was quite small: 14 whites and 12 African Americans), the researchers explored the mechanism for reduced activity of NO in African Americans and concluded that the difference in NO-dependent vasodilator activity occurred at the level of sensitivity of smooth muscle cells to NO. On the basis of these findings, clinical trials in African Americans for a drug that restores levels of NO will soon begin (Rosenberg 2001).

Reduced NO activity in African Americans could be due to environmental factor or factors. However, the failure to explicitly propose an environmental interpretation leaves the distinct impression—an impression that is shaped by popular understandings of race—that either genetic or environmental interpretations are equally possible. While it is undoubtedly possible that individual dif-
ferences in smooth muscle cell responsiveness to NO are inherited, inheritance cannot be offered as an explanation of racial differences in hypertension if race is a socially formed category. Nonetheless, this study could provide important clues to the biological mechanisms by which discrimination might affect the health of a social group.

Reducing biology to genetics is also apparent in a recent report on racial differences in the outcome of left ventricular dysfunction (Dries et al. 1999). These authors argue that the outcome for black patients was worse than for white patients, even after adjustment for differences in severity of clinical disease and other variables, including socioeconomic status. The unstated implication, as noted by respondents, was that genetic differences accounted for the “residual” effects (Stolley, Saha, and Hebrar 1999).

The key point here is that biology cannot be reduced to a sequence of DNA molecules. Genes are only one component of the complex biology of an organism, and the regulation and expression of genes are subject to extensive modulation—by other genes as well as by non-genetic internal and external factors. To fail to appreciate this view of biology is to miss important insights into the physiological functioning of the human organism and its relationship to the environment.

**Implications for Disease Prevention Policy**

That the health of African Americans and other minority groups in the United States is poor—and deteriorating relative to that of whites—is indisputable (Kilborn 1998; Pappas et al. 1993). As previously noted, there are many possible explanations for differences in the incidence, mortality, and severity of disease, including access to medical care and treatment, income level, and exposure to toxic chemicals. Even after attempting to control for access to medical care, racial bias in treatment, and social class, disparities in morbidity and mortality remain (Blendon 1989). The central question then is: do we explain this excess as due to inherited characteristics of a particular group, or do we acknowledge that factors such as nutrition, occupation, unemployment, substandard housing, and racial discrimination may have physiological effects that we have not yet figured out how to measure but are, nonetheless, quite real?

Disease prevention policies differ dramatically depending on whether genetic factors or environmental and social conditions are invoked to explain racial inequities in causes and the natural history of disease. If racial discrimination is an important factor in the etiology of hypertension, reduction of the burden of hypertension in the black community requires addressing racism and how racism works, rather than focusing so exclusively on lifestyle factors or perceived cultural differences, all of which are loaded with blame and direct responsibility for change to the individual rather than the society.

Scientific ideas of genetic distinctions between races and ethnic groups legit-
imize popular notions of racial difference and inferiority, serving to build a profound conceptual barrier to a deeper analysis of how the social environment influences human biology and the human experience with disease. Although molecular epidemiology may occasionally identify disease-related mutations that are found with higher frequency in some populations, the usefulness of this research strategy for understanding complex diseases is limited by the complexity of disease processes and by the historically contingent nature of the classification systems used to define populations.

Conclusions

In this essay, I address several questions relevant to research on the health of racial and ethnic minorities in the United States. What does it mean operationally to say that race and ethnicity are socially produced and socially defined categories? How does such a notion of race and ethnicity influence research strategies? Do social definitions of race and ethnicity allow for research on the genetics of these populations, or would more multidimensional research strategies that examine the effects of structural factors such as access to health care, the effects of poverty on health, and the biological expression of racial discrimination be more productive?

The categories for describing race and ethnicity recognized by the U.S. federal government clearly do not capture the complexity of life experience of racial and ethnic minorities, especially given the serious limitations of commonly used measures of social class (Krieger, Williams, and Moss 1997). I have argued that to retain racial and ethnic labels and to use them to explain disparities in disease requires clarity about the social nature of these labels. History is replete with narratives of diseases thought to be specific to particular racial groups for reasons of inherited predisposition. Many of these stories have been subsequently discredited as changed political, economic, and social conditions allowed a reinterpretation of the science, but some remain an integral part of scientific folklore and continue to inform biomedical research. There is rich variation, including genetic variation, in the human species. Skin color is a phenotypic trait regulated by multiple genes and environment (Sturm, Box, and Ramsay 1998). Although efforts to categorize human genetic diversity have historically relied on skin color, even skin color is not a marker for race. Increasingly, researchers are attempting to identify population-specific genetic variants. But to make sense of these variants, it is necessary to correlate gene frequencies with knowledge of human history, a history that is superficial and informed by Western assumptions about non-Western peoples. Identifying and interpreting frequency distributions of genes is both a scientific and a social process, guided by scientific and social assumptions.

The meanings attached to race and ethnicity shape in a fundamental way experimental design, interpretation of scientific findings, training of health practitioners and biomedical researchers, and development of public health policy.
Interpreting racial inequities in health through what Troy Duster (1990) has referred to as the “prism of heritability” is less threatening to the social structure than explanations that consider the physical and social environment. But such explanations fail to reveal ultimate causes, and in the final analysis they do little to ameliorate the burden of disease.

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