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The Concept of Race in Medicine

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Abstract and Keywords

This article focuses on ethical issues of biomedical research. The ethical concerns raised maintain that potential benefits outweigh potential harms. The benefit is that collecting and reporting race data will help pharmacologists gain a better understanding of health, disease, and response to drug treatment. This, in turn, may help to eliminate many racial disparities in health outcomes. While there are some legitimate concerns associated with the use of race in medicine, these problems can be overcome. Answers to questions about the origins of racial variation in health outcomes are likely to vary from disease to disease and is likely to involve interactions among multiple environmental and social factors. The question of whether genetic factors are likely to play an important role in explaining race—associated health differences is largely unanswered—and can be answered only by future research.

Keywords: ethical issues, biomedical, benefits, health, disease, medicine, race, variation, research

1. Introduction

Recent data suggest significant health disparities among races in the United States today. For example, the rates of hypertension, renal disease, certain cancers, stroke, and heart disease have all been said to vary with respect to race (NCHS 1998; Food and Drug Administration 2003). When looking for tissue matches for a kidney or bone marrow transplant, doctors sometimes have more difficulty finding a match for African Americans than for Caucasian Americans. It has been suggested that this is due in part to differences in the possible antigen combinations on their cell surfaces. Moreover, pharmacological data suggest racial differences in drug metabolism, efficacy, and toxicity.

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Some studies report, for instance, that the effectiveness of certain heart medications (e.g., Enalapril and BiDil) vary with respect to race (Exner, et al. 2001; Food and Drug Administration 2003; Taylor, et al. 2004). Other studies suggest that Caucasian Americans are more likely than African or Asian Americans to have low levels of an enzyme that metabolizes certain psychotropic medications (Xie, et al. 2001; Food and Drug Administration 2003). Some of these differences may be due to socioeconomic status or some other environmental variable associated with race; nonetheless, race-associated differences in health outcomes are often said to remain even after controlling for such factors.

The practice of collecting and reporting data on racial differences in health outcomes is widespread in the United States and has been for some time (Jones, et al. 1991; Williams 1997; Schwartz 2001). However in the 1990s, this practice became more formalized—due, in part, to a law requiring that biomedical research funded by the National Institutes of Health use race as a research variable. This law, in conjunction with the completion of the Human Genome Project (and subsequent promise of a better understanding of the genetic basis of disease), has rekindled an ongoing debate over the scientific value of "race" as biomedical research variable.

Critics typically defend a position that I will call "eliminativism." As the name suggests, eliminativism is the idea that we ought to eliminate race as a variable in biomedical research.[1] This position is motivated by a host of ethical, epistemological, and metaphysical concerns. Can such research be carried out in a nonbiased way? Might research on the genetic basis of disease—and, thus, on possible genetic differences among races—prompt a new eugenics movement? Might such research misleadingly reify race as a biologically meaningful category? How is race to be conceptualized and measured for the purpose of biomedical research? Are data suggesting racial variation in health outcomes unreliable? Might the use of race as a biomedical research variable obscure the search for the true causes of health, disease, and differing responses to drug treatment?

Proponents typically defend a view that I will call "conservationism." Defenders of this view agree that the ethical concerns raised by the practice are to be taken seriously, but ultimately maintain that the potential benefits outweigh the potential harms. The main claimed benefit is that collecting and reporting race data will help epidemiologists and pharmacologists gain a better understanding of the etiology of health, disease, and response to drug treatment. This, in turn, may help to eliminate many racial disparities in health outcomes.

Conservationism comes in at least two varieties. I will call the first "strictly social conservationism." Strictly social conservationism is the idea that racial health disparities are due primarily to social and environmental factors, and race is an ineliminable part of the story. It is ineliminable, on this view, because the variable race is a measure of exposure to racism. Defenders of this view typically maintain that race is a social construct. Roughly stated, race constructionism[2] is the idea that our conventional racial

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categories are wrongly supposed to be biologically real when, in fact, they are a political product of human social practices.[3] Strictly social conservationists, therefore, doubt that genetic factors will play an important role in explaining differential health outcomes.

I call the second position “biosocial conservationism.” Biosocial conservationism is the idea that race is useful for exploring hypotheses about the social, environmental, and genetic causes of health, disease, and response to drug treatment. It differs from strictly social conservationism, in part, by endorsing a different conception of “race.” On this view, “race” is defined in terms of a complex interplay between social factors and geographic ancestry. It also differs from strictly social conservationism by allowing that genetic factors might play an important role in explaining some race-associated health differences.

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It is important to note that my use of the terms eliminativism and conservationism deviates somewhat from the way these terms are often used in the race and gender literature. According to my usage, eliminativism and conservationism are *local* theses about the scientific and practical value of race as a biomedical research variable. These positions are not to be confused with the more-global positions claiming that, in order to end racism, society as a whole ought to retain or eliminate racial classification schemes.[4] It is also important to note that there is an ambiguity over whether race ought to be conserved (or eliminated) in the short run or the long run. Eliminativists tend to maintain that it ought to be eliminated as soon as possible. Strictly social and biosocial conservationists, on the other hand, tend to maintain that race ought to be retained as long as there remain race-associated differences in health outcomes.

My aim in this chapter is to address the following question: To what extent, if any, is race a scientifically valuable biomedical research variable? This question is an epistemological one; it asks whether race helps or hinders empirical research on the etiology of health, disease, and response to drug treatment. This question should be distinguished from normative questions about the use of race in biomedical research. Given the various ethical problems that might be raised by this practice—such as racial discrimination in health care, racially biased research on health and disease, and the potential for a new eugenics movement—should researchers continue to use race as a biomedical research variable?[5] While I believe that the ethical issues and arguments are important, my focus will be on the scientific value, or lack thereof, of “race.” I conclude that the use of race in biomedical research is neither simple nor straightforward. Nonetheless, there are good reasons to retain race as a research variable.

2. Eliminativism

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Eliminativists cite a number of factors that, on their view, call into question the scientific value of race as a research variable. Some maintain, for example, that race is such a confused concept that data on differential health outcomes are, and always will be, unreliable. Others maintain that such research lacks scientific value because it is based on a false premise; it mistakenly assumes inherent biological differences among the races. Still others argue that using race as a research variable obscures the search for the true causes of health, disease, and response to drug treatment. In this section, I critically examine these objections. Although I consider these arguments separately, they are often advanced together. I conclude that, while there are some legitimate concerns associated with the use of race in medicine, these problems are not insurmountable.

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2a. Are race data statistically reliable?

What is race? How is it to be conceptualized and measured for the purposes of biomedical research? These questions are important, in part, because race is an imprecise concept that has taken on a number of different, sometimes inconsistent, meanings. For example, according to one (historically) prominent use, races are groups of people who share a number of overt physical similarities, and psychological and behavioral traits as well, due to a shared biological essence (roughly defined as an intrinsic and explanatory property possessed by all and only or most of the members of a race). Another prominent conception defines races as phenotypically and genetically distinct populations that inhabit their own geographic range and differ significantly from other such populations. This conception is similar to the previous one in that both assume that the members of a race have a number of features in common due to some intrinsic biological factor(s). Yet the latter does not assume the existence of a race-specific essence, nor does it typically focus on racial differences in psychological and behavioral traits.[6] Race has also been used to refer to groups of people who share certain overt characteristics—skin color, hair type, eye shape, etc.—without assuming significant genetic, psychological, or behavioral similarities. In addition, the term is sometimes used to refer to groups of people who share a common ancestry. These are just a few of the ways that race has been conceptualized. Further variation in meaning can be found by addressing questions such as the following: Which overt physical features are the racial ones? How are we to understand the notion of shared ancestry? Are races to be defined in terms of ancestry alone, similarity alone, or some combination of the two? If the latter is the correct view to take, how are we to weigh the relative importance of each factor?

In addition to the intensional differences described above, there is (and has been) considerable uncertainty over the extension of “race.” How many races are there, and which groups should we recognize as races? Let us consider the U.S. Census for illustration. The 2000 Census recognized five major racial groups (American Indian or Alaska native, Asian, black or African American, native Hawaiian or other Pacific Islander, and white), whereas the 1990 Census recognized four (American Indian or Alaska native,

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Asian and native Hawaiian or other Pacific Islander, black or African American, and white). Looking back a full century earlier, the 1890 Census recognized eight racial categories (white, black, mulatto, American Indian, Chinese, Japanese, quadroon, octoroon), and the 1860 Census recognized three (white, black, mulatto). Numerous other examples of disagreement over the extension of the term can be found (Banton and Harwood 1975; Smedley 1993). What is important for our purposes is that different conceptions sometimes result in cross-classifying classification schemes and, thus, give inconsistent answers as to which racial group an individual, or set of individuals, belongs.

Variation in the meaning of “race” poses a potentially serious problem for its use in biomedical research. It raises the question of whether biomedical data (p. 482) based on race are statistically reliable. Data are statistically reliable when they are consistent and repeatable—meaning that one would get the same results were the data collected again and again using the same method. For race to be a scientifically useful variable, one would want reliability not only within a single study, but also across different studies, so as to increase the scope of generalizations about the causes of certain diseases (or responses to certain drug treatments) within appropriately specified contexts.

Concerns about reliability have led some critics to argue that race lacks scientific value as a research variable and, thus, that it ought to be eliminated. According to defenders of this view, part of the problem is that, until recently, no standards were in place for conceptualizing race. Not only would the concept frequently go undefined, when it was defined, different studies (often on the same subject) would sometimes rely on different, inconsistent, conceptions of race (Sankar and Cho 2002). Nor were there well-codified criteria in place for assigning individuals to racial categories—and, again, the method of assignment was frequently unstated. Racial self-identification and observer assessment are two of the most widely used methods of assignment. Due in part to confusion over the meaning of “race,” these methods do not always give consistent results (Williams 1997).

In the 1990s, however, a number of federal health organizations responded to concerns about reliability by issuing guidelines for the collection and reporting of race data.[7] The standard that has been most widely adopted is the 1997 version of the Office of Management and Budget's (OMB) Statistical Policy Directive No. 15. This directive recommends that respondents self-report their race based on five categories: American Indian or Alaska native, Asian, black or African American, native Hawaiian or other Pacific Islander, white. In an effort to recognize mixed-race individuals, respondents are offered the option of selecting more than one race. The directive also specifies that races are not biologically justified categories, but are a product of human social practices. The original purpose of this directive was to standardize the collection and reporting of race data for the enforcement of civil rights laws. The adoption of this standard on the part of federal health organizations has, therefore, been defended on the grounds that it will help ensure consistency across studies and data sets in biomedical as well as economic and sociopolitical contexts. Because these guidelines reflect the current standard for

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conceptualizing and measuring race, this is the conception that I will use throughout the rest of the chapter (unless otherwise indicated).

Critics, nonetheless, maintain that reliability problems persist and that the OMB guidelines leave too much room for inconsistency (Schwartz 2001; Haga and Venter 2003). Part of the problem is that the guidelines don't specify criteria for racial self-identification. They allow different individuals to rely on different, possibly inconsistent, conceptions when assigning themselves to racial categories. Suppose, for example, that one person conceptualizes race in terms of overt characteristics alone. Although this person may have Asian ancestors, she might not self-identify as Asian because she does not “look” Asian. Another person, (p. 483) similar in appearance and ancestry, might conceptualize race primarily in terms of ancestry. Thus, she might classify herself (at least in part) as Asian. In addition, inconsistencies can arise even when different individuals use the same (or similar) conception(s) of race. Suppose, for instance, that two individuals—who look very similar with respect to skin color, hair type, bone structure, etc.—conceptualize race in terms of overt characteristics alone. Some eliminativists argue that because racial traits vary independently, if these two individuals rely on different characteristics when assigning themselves to racial categories, they might end up in different racial groups. Examples such as these have led some eliminativists to express skepticism about the possibility of ever coming up with an adequate standard for conceptualizing and measuring race (Bagley 1995; Schwartz 2001; Haga and Venter 2003). The idea at work, here, is that race is such a fuzzy concept that there will always be ambiguity in its meaning. Consequently, according to defenders of this view, biomedical data are likely to be unreliable, no matter how race is conceptualized and measured.

Concerns about reliability have merit. Proponents and critics alike acknowledge that reliability problems exist (Bagley 1995; Schwartz 2001; Haga and Venter 2003; Williams 1997; Root 2001). Yet proponents often maintain that many of these problems can be resolved. Some argue, for example, that there are statistical methods to correct for some measurement errors (Root 2001). One might also argue that, in spite of variation in meaning, competing conceptions are not as divergent as critics suggest. Take the OMB's recommendation, for example. Although there are some differences in the categories recognized by the 1977 and 1997 versions of Statistical Policy Directive No. 15, there is also significant overlap. Both versions recognize the following three racial categories: American Indian or Alaska native, black or African American, and white. The main difference is that the 1977 version treats “Asian and native Hawaiian or other Pacific Islander” as a single race, whereas the 1997 version divides this category into two distinct races (“Asian”; “native Hawaiian or other Pacific Islander”). Furthermore, more than one standard can be allowed—so long as each is well specified and researchers compare apples with apples.

What conclusions should we draw about the scientific value of race and, thus, about whether it ought to be eliminated from biomedical research? Although the critic is right to point out that the race concept is difficult to operationalize, it is not clear that such

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difficulties are enough to ground eliminativism. Numerous concepts in science are difficult to conceptualize and measure—including important terms like “niche” and “environment.” Yet there is little doubt that these concepts are scientifically valuable. In order to provide a strong argument for eliminativism, critics must demonstrate that reliability problems are likely to arise *no matter how race is conceptualized and measured*. Yet little support has been offered for this claim. What has been demonstrated is that “race” is multiply ambiguous and that past and current efforts at operationalizing this concept have been unsuccessful. There is no reason to suppose, however, that race is such a confused concept that it cannot be successfully operationalized. Consider, for example, (p. 484) the 1997 version of Directive 15. As noted earlier, the main problem with this directive is that it does not provide adequate criteria for racial self-identification. It needs to specify the characteristics that people should use when assigning themselves to racial categories. It should also provide a recommendation for how to weigh the relative importance of each characteristic. Remember, reliability is simply a matter of consistency. As long as the concept can be spelled out in enough detail to allow for reasonable consistency, the data will be reasonably reliable.

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2b. The biological reality, or lack thereof, of race

A second argument against the use of race in medicine rests on the claim that race is a biological fiction (Schwartz 2001). Defenders of this view maintain that the practice of collecting and reporting race data presupposes the scientific validity of the biological race concept. While there is no single biological conception of race, defenders of this view often assume that there are certain core features common to all or most biological conceptions of race.[8] In particular, biological conceptions of race are often said to require reliable covariation among the overt physical characteristics typically used to individuate races and inherent biological (genetic) differences among these groups.[9] Yet, the argument continues, these assumptions run counter to what contemporary science teaches about race. Anthropometric data on the distribution of certain phenotypic traits within and among human populations from around the world indicate that the phenotypic traits typically used to individuate "races" vary independently (American Anthropological Association 1998; Diamond 1994). These data are said to show that the assumption of reliable covariation is unfounded. Likewise, genetic data on the distribution of protein and DNA polymorphisms (within and among human populations from around the world) indicate significant genetic variation within, and little genetic variation among, the major "racial" groups (Lewontin 1972). These data are said to call into question the assumption that there are inherent biological differences among the races. The conclusion that is typically drawn is that race ought to be eliminated as a biomedical research variable because the biological race concept lacks scientific validity.

There are at least two problems with this type of argument for eliminativism. First, the genetic and anthropometric data cited against biological conceptions of race are not as conclusive as people often suppose. While these data do undermine any conception of race that assumes significant phenotypic and genetic continuity within—and significant phenotypic and genetic differences among—the races, they do not show that race is a biological fiction. Because racism often rests on such assumptions—as well as the assumption that there are significant psychological and behavioral differences among the races—this is good news. The problem for the eliminativist is that not all biological conceptions of race require that races be defined by appeal to discrete clusters of phenotypic and genetic properties. Robin Andreasen (1998, 2005) and Philip Kitcher ([1999]2003), for example, have independently (p. 485) defended somewhat different phylogenetic conceptions of race. Though there are important differences between their views, both maintain that races, if they exist, ought to be understood as lineages of human breeding populations. Because phylogenetic conceptions of race define race taxa as lineages, they do not require discrete phenotypic and genetic boundaries among them. Nor do they require significant phenotypic and genetic continuity within the races. They merely require reasonable reproductive isolation among human populations. Consequently, phylogenetic conceptions of race are compatible with the genetic and anthropometric data cited above. Likewise, Massimo Pigliucci and Jonathan Kaplan (2003) defend an ecological race concept.[10] They maintain that races are best understood as subspecies that have become genotypically, and often phenotypically, differentiated as

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a result of differential selection pressures from different local environments. Pigliucci and Kaplan also hold that ecological races can be named on the basis of very few characteristics. It follows that, like phylogenetic conceptions of race, the ecological race concept is compatible with data typically cited against biological conceptions of race.

Second, this argument for eliminativism wrongly assumes that the scientific value of race in biomedical research depends upon the scientific validity of the biological race concept. To see this assumption at work, consider the following quotation (Schwartz 2001, 1393):

> Beyond the bedside, race-based medical research is widespread. The pseudoscience of race is well-represented in clinical investigations. In March 2001, under the search term “Negroid race,” Medline contained 13,592 citations, of which 1301 appeared in 1999 or 2000. Among these studies are race-based investigations of lipid metabolism, renal dysfunction, responses to vasodilators, sexual maturation, drug metabolism, neurodegenerative diseases, and even Dupuytren's contracture. Such research mistakenly assumes an inherent biological difference between black-skinned and white-skinned people. It falls into error by attributing a complex physiological or clinical phenomenon to arbitrary aspects of external appearance. It is implausible that the few genes that account for such outward characteristics could be meaningfully linked to multigenic diseases such as diabetes mellitus or to the intricacies of the therapeutic effect of a drug.

This type of reasoning is also at work in the following quotation:

> In its draft guidance, FDA states that the collection of data per the OMB categories will “enhance early identification of differences in physiological response among racial and ethnic subgroups.” Although this may appear superficially true, we believe this reasoning lacks scientific merit. Several studies have confirmed that greater genetic variation exists within groups than among them. (Haga and Venter 2003, 466)

In other words, FDA's claims of scientific value are said to be spurious because race is biologically unreal.

There is no reason to suppose, however, that race must be biologically real in order to be a scientifically valuable research variable. Indeed, the standard recommended by the OMB suggests that race is a social construct. Likewise, defenders of **(p. 486)** strictly social conservationism deny the biological reality of race, but nonetheless maintain that it is a useful biomedical research variable. On their view, race plays an important role in explaining the origins of certain social and environmental disparities that might, in turn, bring about differential health outcomes. They conclude that race ought to be retained in order to study the effects of racism and racial injustice on health and disease. I will have more to say about this hypothesis below. Right now, let us examine one more argument for eliminativism.

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2c. Race as a surrogate variable

A common goal shared by epidemiology and pharmacology is the identification of factors reliably associated, either directly or indirectly, with risk of disease or response to drug treatment. Because the direct causal variables are often unknown, and most likely interact with one another in complex ways, epidemiologists and pharmacologists often rely on surrogate variables. Roughly defined, a “surrogate variable” is one that is used as a substitute (or proxy) for the variable(s) of interest. Examples of surrogate variables used in biomedical research include gender, occupation, marital status, geographic location, race, and so forth. When categories such as these are used as proxy variables, it is often assumed that they are correlated with more-direct causal variables. For instance, when geographic location is used as a proxy, the idea is not that geographic location per se causes differential health outcomes. Rather, such outcomes are assumed to be the result of other factors, such as exposure to certain environmental toxins, that are associated with geographic location. Likewise, race is often used in biomedical research as an indirect way of getting at a number of different factors (environmental, social, cultural, economic, and possibly genetic) that play a more-direct causal role in health and disease.

Some eliminativists argue that, as a surrogate variable, race is devoid of scientific value. Francis Collins (2004), for example, holds that, while there is no doubt that racial health disparities exist, the relationship between race and disease is complex and poorly understood. His argument begins with the claim that race is an imperfect surrogate for numerous social and environmental risk factors (such as education, access to health care, culture, diet, environmental exposure, economic status, social marginalization, discrimination, stress, and so forth). Let us consider the relationship between race and poverty for an illustration of Collins' objection. In the United States today, a larger proportion of the African American population lives below the poverty line when compared with the percentage of Caucasian Americans who live below the poverty line. Yet the association between race and poverty is imperfect. Not all African Americans, for example, are poor—and in terms of raw numbers, there are more poor whites than poor African Americans. A similar story can be told about the relationship between race and each of the other social and environmental variables listed above. Collins adds that, at the genetic level, race is an imperfect surrogate for ancestral geographic origin. Likewise, ancestral geographic origin is a rough surrogate for genetic risk factors. He adds (p. 487) that all of these factors—genetic and environmental—interact with one another in complex ways. Collins concludes that etiological research ought to move beyond the use of weak and imperfect proxy variables such as race. It should focus, instead, on the more proximate genetic and environmental factors that influence health and disease.

One problem with this argument is that race can be a useful research variable, even if it is an imperfect surrogate for the more proximate causes of health and disease (when taken individually). This is so for two related reasons. First, while Collins is right that biomedical research can be made more precise through direct examination of proximate

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causes, as he himself notes, these factors most likely interact in complex ways that are poorly understood. Thus, rather than providing an argument in favor of eliminativism, Collins has provided a reason for retaining race as a variable. Proxy variables can provide a useful *starting point* for understanding complex causal networks. When the causal picture becomes more developed, it may be possible to move beyond imperfect surrogates in favor of direct causal variables. In the meantime, however, it seems that race ought to be retained. Second, it is widely agreed, even by Collins himself, that race is a reasonably good predictor of certain health outcomes. But if race is such a flawed surrogate, why is it a good predictor of health outcomes? One possibility is that race is a poor surrogate for each *factor taken individually*, but a reasonably good surrogate for certain *collections* of interacting causal factors; it may even be causally related to some of these factors. Indeed, as we will see below, some conservationists defend the scientific value of race on the grounds that race acts as a measure of the effects of racism on health and disease (Williams 1997; Jones 2001; Root 2001). The idea at work, here, is that because we live in a racially stratified society, there is (and was) an unequal distribution of power and privilege among the races in the United States today (and in the past). These inequalities are said to carry important health consequences. For example, because African Americans are underrepresented in many high-paying, high-status professions, they are more likely to be exposed to certain occupational health hazards, environmental toxins, inadequate health care, social stress, and so forth. Thus, on this view, they are at higher risk, when compared with Caucasian Americans, for example, for certain diseases, such as depression, heart disease, or some types of cancer. I will take a closer look at these ideas in the next section. For now, it is important to note that this position provides a response to Collins' argument for eliminativism. By explaining how race could be an imperfect surrogate for individual proximate causes of health and disease, but a reasonably good predictor of health outcomes (due to interrelations among multiple factors), it provides a reason for retaining race as a variable in biomedical research.

A further argument for eliminativism can be found in Cooper et al. (2003).[11] Cooper and his colleagues maintain that, whereas the causes of health and disease are local, heterogeneous, and vary independently, races are broad categories and are too coarse-grained to be biomedically useful. In support of this claim, they cite data suggesting that there is more genetic variation within than among the major **(p. 488)** racial groups. They take these data to show that genetic variation within *Homo sapiens* fails to correspond to the broad categories that we call races. These data are, thus, said to cast doubt on the usefulness of race as a surrogate for genetic risk factors. Cooper and his colleagues share an equally pessimistic view of the use of race as a surrogate for nongenetic risk factors. They argue that, just as there is significant genetic variation within major racial groups, there is also significant variation within the races in the patterns of *social* and *environmental* risk factors. They add, “[W]ithout the context provided by such variables as the level of education, occupation, type of diet, and place of residence, race as a social category is not a useful predictor of health outcomes” (2003, 1169). Indeed, they conclude that the use of race in medicine hinders biomedical research by leaving one

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blind to individual context as well as to the proximate causes that give rise to health and disease.

This argument for eliminativism is no more successful than Collins' argument. One reason is that it assumes that certain contextual variables—such as level of education, type of diet, access to health care, etc.—are ignored when race is used as a surrogate variable. Although this may be true in some cases, it is (most likely) not universally true. More important, however, this is not how race *ought* to be used. Indeed, because it is likely that numerous factors interact in the production of health and disease—and because researchers frequently aim to control for confounding variables—a proper use of race in medicine recognizes the importance of context and takes such variables into consideration. One should not eliminate race as a research variable simply because some scientists might do sloppy science.

A second problem is that Cooper and his colleagues fail to take population thinking seriously.[12] They treat variation as a distraction—as something that gets in the way of the search for the true causes of health and disease. They are correct when they maintain that racial categories are coarse-grained and that the causes of health and disease are local, heterogeneous, and vary independently. They are also correct when they maintain that there is significant genetic variation within the races (as well as significant variation in nongenetic risk factors). While it may follow that it is sometimes difficult to infer something about a particular *individual's* risk profile based on her race, it does not follow that race hinders biomedical research. Part of the reason is that the individual is not the unit of interest in this context; the unit of interest is the population. By focusing on variation *within* the races, Cooper et al. downplay the existence of *differences* among races with respect to genetic and nongenetic risk factors. Genetic differences, for example, do exist among the races, but they are differences *on average.* Of course, as Cooper et al. note, it is an open empirical question whether (and to what extent) these differences explain race-associated differences in health outcomes. Yet, as I will argue in section 3b, rather than providing support for eliminativism, this provides a reason to retain “race.” Many researchers agree that racial health disparities exist. We do not want to foreclose the question of *why* such disparities exist by prematurely eliminating race as a biomedical research variable.

(p. 489) 2d. Concluding remarks

Eliminativists have provided several reasons that one ought to proceed with caution when using race as a biomedical research variable. Yet, their objections are not strong enough to ground eliminativism. Reliability problems, for example, do exist—but there is no reason to suppose that it is impossible to operationalize “race.” Likewise, race can be a social construct and still be a useful research variable. It can also be a useful research variable even if it is an imperfect surrogate for the more proximate causes of health and

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disease. Let us now examine some of the arguments in favor of retaining race as a biomedical research variable.

3. Strictly Social versus Biosocial Conservationism

Conservationism is motivated by data suggesting that race is a good predictor of a wide variety of health outcomes. As we saw earlier, not only do data suggest that the rates of numerous diseases vary with respect to race, data also suggest racial variation in response to multiple types of drug treatment. Race-associated differences are often said to remain after controlling for income, education, and other social factors. Defenders of strictly social and biosocial conservationism maintain that such differences demand an explanation—and, thus, that we ought to retain race as a biomedical research variable.

Two types of hypotheses have been proposed. First, there is strictly social conservationism. Defenders of this view do not deny that health and disease are biological processes; I call this position "strictly social" conservationism because it embraces the idea that racial variation in health outcomes is explained primarily (or solely) by variation in social and environmental factors. Defenders of this view endorse both a positive and a negative thesis. The negative thesis denies that genetic factors are likely to play an important role in explaining most health disparities. The positive thesis states that race is ineliminable for explaining race-associated health differences. It is ineliminable, on this view, because part of what researchers measure with the variable "race" are the effects of racism on health and disease. Biosocial conservationism, on the other hand, takes a mixed view. Defenders of this view agree that social and environmental factors, including differential exposure to racism, are likely to be an important part of the story. The main difference is that biosocial conservationism allows that genetic factors might also play an important role in explanations of racial health disparities.

In what follows, I critically examine both positions. In section 3a, I temporarily bracket the question of the role that genetic factors might play. I focus, instead, on (p. 490) the positive thesis of strictly social conservationism. I argue that, while strictly social conservationists tell a plausible story about the relationships between race and certain social and environmental risk factors, more empirical work needs to be done. In section 3b, I return to the debate over the role (if any) played by genetic factors. Here, I defend biosocial conservationism. I argue that the question of whether genetic factors are likely to play an important role in explaining many race-associated health differences is largely unanswered—and can be answered only by future research.

3a. The role of racism

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When defenders of strictly social conservationism maintain that differential exposure to racism (overt and/or institutional) is an important factor for explaining racial health disparities in the United States today, they are not denying the importance of other social and environmental variables (such as level of income, education, area of residence, access to health care, etc.). Rather, they are suggesting that there is a causal connection between race and (some of) these other variables. On their view, race is a social status category that is causally linked to some of the more proximate causes of health and disease.

Arguments for the positive thesis often start with the claim that race is a social construct. Let us recall that race constructionism is the idea that races are wrongly supposed to be biologically real, when in fact they are a political product of human social practices. Next, defenders of this thesis note that, in the United States, race has played, and continues to play, an important role in structuring human social life. Not only has it played a role in structuring interpersonal relationships, it has also played a role in determining access to goods, resources, and opportunities. While these features of race have their origins in historical circumstance, defenders of this view stress that, in the United States today, race continues to reflect and reinforce differences in life chances. Finally, defenders of this view maintain that these differences are likely to have important consequences for health and disease. Some maintain, for example, that due in part to our history of legalized segregation, race remains a partial determinant of one's educational and employment opportunities and one's economic status (Williams 1997; Jones 2001; Root 2001). These variables, in turn, are said to have an important effect on environmental risk in occupational and residential contexts; they are also said to play a role in determining access to and quality of health care and health education. Some add that interpersonal factors—such as racial prejudice or discrimination—can play a role in determining health outcomes (Williams 1997; Jones 2001; Root 2001; Sankar et al. 2004). It has been suggested, for example, that the psychological stress brought on by perceived racism can bring about differential rates of depression and high blood pressure (Jones 2001; Sankar et al. 2004).

I will not spend much time critically discussing the positive thesis of strictly social conservationism. Part of the reason is that, as noted earlier, the heart of the (p. 491) debate within conservationism is over whether it is reasonable to suppose that genetic factors are relevant for explaining some racial health disparities. Defenders of biosocial conservationism acknowledge, for example, an association between race and socioeconomic status—as well as their collective potential for explaining some race-associated health differences (Risch et al. 2002; Burchard et al. 2003). That being said, the positive thesis is a hypothesis that requires further development and more empirical support.

First, there is the question of how much of a role racism is said to play. While it is reasonable to suppose that racism plays *some* role in explaining *some* health disparities, defenders of this thesis often make a stronger claim—namely, that overt and institutional discrimination are major contributors to racial variation in health outcomes (Kaufman et

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al. 1997; Root 2001; Sankar et al. 2004). Not only might this be an overstatement, this claim requires further clarification. When defenders of the positive thesis maintain that racism is a major contributing factor, do they mean that racism plays some role (though perhaps not a major role) in explaining most health disparities—or do they mean that racism is a major factor in explaining most health disparities? A third possibility is that racism plays a large role in explaining some health disparities but a small role in others, and perhaps in some cases it plays no role whatsoever. For the sake of discussion, let us suppose that the positive thesis is the idea that racism plays an important role in explaining numerous racial health disparities.

Once clarified, the positive thesis is a hypothesis that needs testing. This thesis cannot be vindicated by appealing to the fact that racism exists (has existed) and by telling a plausible story (or series of plausible stories) about the relationship between racism and differential health outcomes. Instead, one needs to provide empirical support for a series of causal hypotheses. The positive thesis of strictly social conservationism is not a single hypothesis, but a web of causal hypotheses—each of which aims to describe the precise role that some aspect of racism plays in explaining some well-specified health outcome. Data suggest, for example, that African Americans are less likely than Caucasian Americans to receive certain types of preventive health care. Proposed explanations include inadequate health care coverage, practitioner bias, and/or discriminatory practices in the health care system (Shavers et al. 2006). Data also suggest racial differences in housing conditions—and their collective impact on health and disease. The proposed explanation, in this case, is institutional racism (Williams 1997; Sankar et al. 2004). Numerous other hypotheses that aim to describe a causal pathway through which some specific aspect of racism can influence specific health outcomes could be proposed. If it turns out that racism plays an important role in many such explanations, the positive thesis will be vindicated. If, on the other hand, such explanations fail time and again, scientists may begin to suspect that the positive thesis is faulty.

Let us take a closer look at some of what will be involved in testing these types of more specific hypotheses. Consider, for example, a hypothesis claiming that racial bias and discriminatory practices in medicine play an important role in (p. 492) explaining why African Americans are referred less frequently than Caucasian Americans for cardiac catheterization. Like the positive thesis, this hypothesis is not a single hypothesis, but a collection of hypotheses—all of which require independent testing. First, it needs to be established that African Americans are, in fact, referred less frequently than Caucasian Americans for cardiac catheterization. Next, one needs to demonstrate the existence of racial bias and discriminatory practices in the health care system, while controlling for other factors, such as level of education, economic status, and occupation. One must also demonstrate that racial bias and discriminatory practices play an important causal role in differential rates of referral. While some empirical support has been offered for the first two steps, too often authors rely on plausibility alone as a reason for accepting the third and crucial step—namely, the one that aims to establish a causal connection between race

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and differential rates of referral. It is important to keep in mind that this type of mistake is not isolated to the example just discussed. This same type of objection could be advanced against numerous other hypotheses claiming a connection between racism and racial variation in certain health outcomes.

The suggestion that we need to retain race as a variable in biomedical research in order to research the effects of racism on differential health outcomes is worth taking seriously. There is no doubt that we live in a racially stratified society. Nor should there be much doubt that racial stratification can have an impact on a number of social and environmental variables that more directly affect health and disease. Nonetheless, as one can see from what I have said above, providing the empirical support for the positive thesis is no small task. While some evidence has been offered in its favor, defenders of strictly social conservationism frequently draw a fairly strong conclusion—racism is an important factor in explaining a reasonably large proportion of the racial variation in health outcomes—from somewhat slender evidence. Without more data, it is hard to know exactly how much of a role racism in fact plays.

3b. Race, genetics, health, and disease

In this section, I examine the debate within conservationism over the role that genetic factors might play in explanations of racial health disparities. As already noted, defenders of strictly social conservationism express doubts about the importance of genetic factors for understanding race-associated health differences. Based on such doubts, they maintain that we ought to retain race *solely* for the purpose of exploring hypotheses about the social and environmental causes of health disparities (Root 2001, 2003; Cooper 1993). Defenders of biosocial conservationism agree that there will be many cases in which the explanation is primarily social or environmental. Nonetheless, because they are more optimistic about the role that genetic factors might play, they maintain that race ought to be retained for investigating the social, environmental, and genetic origins of health disparities. After considering arguments for each side, I defend biosocial conservationism. (p. 493) I also argue, however, that one must not overstate the importance of genetics for understanding race-associated health differences.

When race is used in biomedical genetics, it is frequently assumed to provide an indirect measure of geographic ancestry. Geographic ancestry, in turn, is assumed to provide an indirect measure of genetic differences among human populations. Part of the justification for assuming these surrogate relationships comes from the history of human evolution. Historically, human populations were separated by large geographic barriers—such as tall mountains, large deserts, and large bodies of water. This geographic separation brought about a certain amount of reproductive isolation among human breeding populations.[13] Differences in religion, culture, and language, most likely, reinforced these endogamous mating patterns—and, thus, played a role in creating some

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of the genetic structure that currently exists among human populations (Risch et al. 2002; Edwards 2003; Burchard et al. 2003).

Because strictly social and biosocial conservationists agree, for the most part, about the importance of social and environmental factors in explanations of racial health disparities, arguments for biosocial conservationism often aim to show that genetic factors are likely to be important in explanations of some racial health disparities. At least two arguments have been offered in defense of this view. Defenders of the first argument often begin by noting that there are approximately 15 million DNA polymorphisms in humans. They add that some, yet to be identified, proportion of these variants will be relevant for understanding health and disease. The biomedical relevance of DNA polymorphisms can be appreciated by considering the fact that many well-known inherited diseases—such as Tay-Sachs disease, muscular dystrophy, sickle cell anemia, and cystic fibrosis—are caused by very small changes at the genetic level. Research also suggests that relatively small genetic differences can increase one's risk of complex diseases, such as Alzheimer's disease (Burchard et al. 2003). Next, it is argued that population genetics studies indicate that, in addition to significant genetic variation within the five racial groups identified by the OMB, there are also important genetic differences among these groups (Rosenberg et al. 2002; Risch et al. 2002; Burchard et al. 2003; Edwards 2003). This variation has been demonstrated using a number of different methods—including the reconstruction of ancestral tree diagrams, multilocus genotyping, and studies on the distribution of allele frequency differences at microsatellites and single nucleotide polymorphisms. Although these methods give somewhat different results, most studies agree that genetic differentiation is greatest when defined on a continental basis. In addition, it is argued that population genetics studies suggest that genetic variants in the lower frequency ranges (below 20%) are more likely to be race-specific (Risch et al. 2002; Burchard et al. 2003). Because disease-predisposing alleles also tend to be in the lower frequency ranges, defenders of this argument conclude that it is reasonable to suppose that some of these race-specific variants are relevant to health and disease.

There is some controversy, however, over the biomedical relevance of these data. Cooper and his colleagues (2003), for example, argue that the success of the (p. 494) population genetics studies cited in defense of biosocial conservationism depends, in part, on the effect of population-specific alleles and, in part, on the collective effect of differences in the frequency of common alleles. Yet, they claim, most population-specific alleles are likely to be nonfunctional. “Rather, like a last name, they merely help to verify the geographic origin of a person's ancestry” (1167). Cooper et al. add that differences in the frequency of common alleles will produce differences in disease risk only if the disease is caused primarily by interactions among multiple loci. They maintain, however, that this is “both mathematically and biologically implausible” (1167). While they don't explain what they mean, presumably the objection is that the relevant variant alleles must vary concordantly (with one another and with race) when, in fact, they are likely to vary independently (from one another and from race). Cooper et al. conclude that the more-relevant outcome—that “the sets of common functional polymorphisms are distributed in

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discrete racial categories"—has not been established. They also express skepticism that such an outcome will ever be established based on the fact that genetic data suggest that there is more genetic variation within than among the races.

This objection is problematic in a number of different ways. First, while it is true that population genetics studies frequently rely on nonfunctional genetic markers, more empirical research is needed before one can conclude that *most* population-specific alleles are nonfunctional. Second, the focus on common alleles in this objection is misleading. Part of the reason is that many of the alleles underlying disease susceptibility are likely to be rare (in the lower frequency ranges) (Risch et al. 2002). Because Cooper et al. (2003) have little to say about the role played by rare alleles, most of their objection misses its mark. Indeed, their demand that "the sets of common functional polymorphisms [be] distributed in discrete racial categories" is a throwback to essentialism. As noted in section 2c, it is biologically unrealistic to expect a (near) one-to-one correlation between certain genetic variants and race; it is also unreasonable to expect that races form discrete categories. Furthermore, as we will soon see, population geneticists have identified a number of race-associated genetic variants that are known to play an important role in health and disease (Burchard et al. 2003).

Now, let us examine a second argument for biosocial conservationism. Defenders of this argument often begin by citing examples of known genetic disorders associated with race. The most widely cited examples come from Mendelian disorders, such as Tay-Sachs disease, cystic fibrosis, and sickle cell anemia (Bamshad and Olson 2003). Cystic fibrosis, for example, is usually found in people of European ancestry. Likewise, sickle cell anemia is more common among people who have ancestral origins in Africa or the Mediterranean. In addition to Mendelian disorders, there are also some examples of race-associated complex genetic disorders (disorders that are due to multiple interacting genetic and environmental factors) (Risch et al. 2002; Burchard et al. 2003; Bamshad et al. 2003). Some of these disorders are a partial product of alleles that are present in one race but virtually absent in all others. For example, C282Y is a mutant allele that is associated with (p. 495) hemochromatosis; it is found in nearly all European populations and is nearly absent in nonwhite groups. Likewise, venous thromboembolic disease is associated with a genetic variant called factor V Leiden. This variant is present at reasonably high frequency in whites, but is rarely seen in East Asians and Africans. The CCR5-delta32 variant, which is protective against a certain strain of HIV, is found at an unusually high frequency in Northeastern Europeans and is mostly absent in other groups. Other race-associated genetic variants are present in all racial groups but vary in their frequency from race to race (Burchard et al. 2003). For example, a different variant of CCR5 is associated with different rates of HIV-related disease progression in European Americans and African Americans. Another example is APOEε;4, which appears to increase a person's risk of Alzheimer's disease and is present at different frequencies among blacks, whites, and Asians. Finally, preliminary research suggests race-associated differences in drug metabolism and response to drug treatment (Bamshad et al. 2003). After listing a number of different types of examples, defenders of this argument remind readers that research on the genetic basis of disease is in its infancy. They add that,

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based on what is already known, we should be optimistic that more examples of this sort will be discovered. They, therefore, conclude that it is reasonable to suppose that genetic factors will be important in explaining a non-negligable proportion of the racial variation in health outcomes.

Critics of the use of race in biomedical genetics, however, are unconvinced by this argument. They offer three related objections. First, there is a difference of opinion about whether race is the category of interest for understanding the distribution of some of the diseases cited above (Cooper et al. 2003; Root 2003). It has been argued, for example, that sickle cell anemia does not vary with respect to race because it is not limited to those with African ancestry. Critics also maintain that race is irrelevant for understanding Tay-Sachs disease because it is associated with persons of Jewish descent, not whites. Moreover, because the frequency of cystic fibrosis is said to vary widely within Europe, it is, once again, denied that race is the category of interest. Second, some critics maintain that, even if race is the variable of interest in some of these cases, Mendelian disorders are relatively rare. They are said to account for approximately 2% of all of the diseases that affect humans (Cooper et al. 2003). Third, critics typically argue that only a handful of genes underlying a propensity of common diseases or affecting drug response have been identified—and even fewer have been shown to correlate with race. All of these considerations taken together are said to provide good reasons for doubting that genetic factors will play an important role in explaining racial health disparities.

My view of the above argument—as well as the question of the importance of genetic factors for understanding race-associated health differences—is that the truth is likely to be somewhere in the middle. As discussed above, correlations between race and numerous disease-associated genetic variants do exist. These data, therefore, provide preliminary evidence in favor of the view that genetics might sometimes be important for understanding race-associated health differences. As we have just seen, however, critics maintain that, in many of these cases, **(p. 496)** race is not the category of interest. Yet it is not clear that they are right about this. Sickle cell anemia, for example, *is* most common among people of African descent. People whose ancestors come from the Mediterranean are also at risk, but less so than those of African ancestry. Likewise, people who have ancestral origins in Europe *are* at higher risk for cystic fibrosis than other groups, in spite of the fact that there is variation within the European population. Finally, while it is true that Tay-Sachs disease is associated with persons of Jewish descent, it is inaccurate to say that race is irrelevant, in part because this ethnic category is correlated with race. The critics' mistake in each of these examples is to, once again, make the biologically unrealistic demand that there be a (near) perfect correspondence between race and disease-associated genetic variants.

Yet as we saw earlier, critics have a ready-made two-part response: (a) Mendelian disorders are relatively rare, and (b) few genes underlying a propensity to common diseases or influencing drug response have been identified, and even fewer have been shown to correlate with race. While I agree with both of these statements, it does not follow that one ought to be skeptical about the importance of genetic factors for

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understanding race-associated health differences. This conclusion is premature. Researchers on both sides agree that very little is known about the genetic basis of most common diseases. It follows that it is an *open empirical question* whether genetic factors will play an important role in explaining some racial variation in health outcomes. Rather than providing support for strictly social conservationism, this tips the scales in favor of biosocial conservationism. Let us recall that strictly social conservationism is the idea that we ought to retain race *solely* for the purpose of exploring hypotheses about the social and environmental causes of health disparities. Indeed, some even argue that the use of race in biomedical research "should be limited to studies of the impact of racial discrimination on health" (Root 2001, 21). While biosocial conservationists may sometimes be too optimistic about the role that genetic variants are likely to play, the thesis is preferable to strictly social conservationism. Unanswered questions about the role that genes might play can only be answered by future research.

Yet, as we saw earlier, the critics maintain that there are good reasons to doubt the need for future research (Williams 1997; Root 2001; Cooper et al. 2003). Defenders of this view, once again, cite data suggesting more genetic variation within than among the races. Next, they argue that because there are (at best) very few genetic differences among the races, there is very little reason to suppose that researchers will someday discover race-associated genetic variants that are relevant to understanding health and disease.

One problem with this argument is that, as noted earlier, it is not strictly true that there are no genetic differences among the races. Studies indicate that, although there is significant variation within the races, genetic differences among the races also exist (Rosenberg et al. 2002; Risch et al. 2002; Burchard et al. 2003; Edwards 2003). A further reason is that estimates of inter- and intra-racial genetic variation are based primarily on common alleles. Disease-predisposing alleles, on the other hand, tend to be relatively rare (Risch et al. 2002; Burchard et al. 2003). In (p. 497) fact, for many Mendelian disorders, common alleles are frequently race-specific. In addition, recent genetic research suggests that the lower-frequency alleles are more likely to be race-specific than are common variants. Such estimates may, therefore, under represent racial variation in disease-predisposing alleles.

Now, I would like to make a somewhat different criticism of strictly social conservationism. Let us suppose, for the sake of argument, that there are good reasons to doubt the importance of genetic factors for understanding a reasonable proportion of racial health disparities. Does it follow that we ought to retain race solely for understanding the social and environmental causes of health and disease? The answer is no. Even if only a handful of health differences that can be explained by genetic differences among the races, we ought to retain race as a research variable. Biomedical genetics is not solely concerned with understanding common diseases; researchers also aim to understand the etiology of rare diseases. Furthermore, the use of race in biomedical genetics is not limited to exploring questions about racial variation in health outcomes. Even if genes play little to no role in explaining race-associated health differences, researchers might still learn something important by using race as a proxy

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variable. As noted in section 2c, surrogate variables (such as race) sometimes provide a useful starting point for understanding complex causal networks. As research progresses and the causal picture becomes more developed, it may be possible to move beyond imperfect surrogates in favor of direct causal variables.

I want to be careful, however, not to overstate my case. One reason is that the correspondence between race and disease-associated genetic variants is likely to be somewhat imperfect. Let us recall that, when race is used in biomedical genetics, it is often assumed to be a surrogate for evolutionary ancestry—and evolutionary ancestry is often assumed to be a surrogate for genetic differentiation among human populations. While there is a good deal of correspondence between the categories recognized by the OMB and the ancestral lineages identified in population genetics studies, this correspondence is not perfect (Rosenberg et al. 2002; Risch et al. 2002; Burchard et al. 2003; Bamshad et al. 2003). Likewise, due in part to convergent evolution and due in part to racial admixture, the correspondence between ancestral geographic origin and genetic differentiation is not perfect. Indeed, if one is thinking about the role of genetic factors in understanding race-associated health differences in the United States today, one should not expect anything more than a rough correspondence. Many population genetics studies on genetic differences among human populations rely on indigenous groups that show relatively little admixture (even today). Because admixture is comparatively more common among racial groups in the United States today, the genetic structure suggested by examining indigenous groups will only approximate the genetic structure among populations in the United States today.

That being said, I would like to reiterate that there is some preliminary evidence to support claims about the importance of genetics for understanding some—yet to be specified—proportion of racial variation in health outcomes. Not only have some disease-associated variants already been identified, genetic (p. 498) differences do exist among the races, and some of these might be relevant for understanding differential health outcomes. I would also like to reiterate that very little is currently known about the role that genetic factors might play in explaining race-associated health differences. This provides the best reason for retaining race in biomedical genetics research. It also provides the best reason for preferring biosocial conservationism to strictly social conservationism.

4. Eliminativism, Biosocial Conservationism, and the Metaphysics of Race

What is the relationship between the utility (or lack thereof) of race in biomedical research and the ontological status of racial classification schemes? In response to this question, it is generally presumed that eliminativism and strictly social conservationism require that race be a biological fiction. Biosocial conservationism, on the other hand, is

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typically thought to require the biological reality of race. In this section, I take a closer look at these assumptions. I argue that the claim that race is a biological fiction acts as a red herring in several arguments for eliminativism and strictly social conservationism.

Let me begin by noting two assumptions shared by most participants in this debate. First, it is generally assumed that the biological reality of race implies reasonable genetic continuity within each race and a reasonable genetic distinctness among the races. Second, it is assumed that genetic data suggesting more variation within than among the races provide evidence against the biological reality of race. This type of argument against the biological reality of race originated with Richard Lewontin (1972) and has been repeated many times since its original formulation (American Anthropological Association 1998; Root 2001, 2003; Zack 2002; Keita et al. 2004). For convenience, I will call it “Lewontin's genetic argument.”

The influence of Lewontin's argument cannot be overstated. These two assumptions have led to a number of spurious arguments in the debate over the scientific value of race as a research variable. In section 2b, we saw that some eliminativists wrongly suppose that it is possible to show that race lacks value as a biomedical research variable on the grounds that it is a biological fiction. In section 2c, I showed that Cooper et al. mistakenly conclude that race is a poor surrogate variable, in part, because there is more variation within than among the races. In addition, as we saw in section 3b, some defenders of strictly social conservationism often erroneously assume that Lewontin's argument can be used to show that genetic factors are unimportant for explaining racial health disparities.

(p. 499)

Lewontin's genetic argument has also played a role in the mistaken belief that eliminativism and strictly social conservationism require the biological unreality of race. To see this assumption at work, one only needs to note that defenders of eliminativism often set the stage for their view by asserting that race is a social construct and by citing Lewontin's genetic argument in support of this claim. Likewise, defenders of strictly social conservationism take the idea that race is a biological fiction to be foundational for their positive thesis. This thesis is frequently developed by defending the idea that race is a biological fiction and by discussing the various ways in which race is a social status category. It is then argued that race, as a social status category, is causally linked to a number of social and environmental factors that play a more-direct role in health and disease.

I would like to suggest, however, that the idea that race is a biological fiction acts as a red herring in these contexts as well. Part of the reason is that there is an error in the way that people on all sides of the debate typically reason about the biological reality of race. It is often assumed that Lewontin's genetic argument is sufficient for showing that race is biologically unreal. However, this reasoning is mistaken. One reason has been provided by Anthony Edwards (2003). Edwards maintains that Lewontin's argument is based solely on a locus-by-locus analysis of his data. This is problematic, according to Edwards, because gene correlations are also important for understanding populations'

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genetic structure. He adds that, once gene correlations are taken into consideration, there is likely to be more genetic differentiation among human populations than is commonly supposed. Edwards concludes that Lewontin's argument is circular: By relying on gene frequencies alone, he ignores the structural aspects of his data and then concludes that these data possess no such structure. Like Edwards, many geneticists accept Lewontin's data, but nonetheless maintain that the allele-frequency differences that exist among human populations are highly structured and are useful for identifying distinct lineages (Prichard et al. 2000; Risch, et al. 2002; Rosenberg, et al. 2002; Bamshad et al. 2003). I should add that subsequent versions of Lewontin's argument also rely on a locus-by-locus analysis of newer genetic data. Consequently, these objections apply to those versions as well.

There is also a second (related) reason that the biological unreality of race is a red herring. As noted above, both the ecological race concept and phylogenetic conceptions of race allow for the possibility that human races are biologically real—despite data suggesting more genetic variation within than among the races. In addition, both conceptions are compatible with eliminativism and with strictly social conservationism. Phylogenetic conceptions of race are compatible with these views because such conceptions merely require reasonable reproductive isolation among human breeding populations. This condition can be met, even if genetic variation plays little to no role in explaining racial variation in health outcomes. Now, let us recall that the ecological race concept requires that there be adaptive differences among the races. This conception of race will be compatible with eliminativism and strictly social conservationism whenever the adaptive differences in question are irrelevant for explaining racial variation in health outcomes. **(p. 500)** Indeed, races could be defined in terms of genetic clusters and be compatible with eliminativism and strictly social conservationism. This will occur when the genes that make up the cluster are determined to lack biomedical relevance.

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5. Conclusion

In the debate over the scientific value of race as a research variable, I have defended biosocial conservationism, not because there is more empirical evidence in its favor, but because—relative to the alternatives—it leaves the maximal number of empirical questions open. Strictly social conservationism, by defending the idea that race ought to be retained *solely* for the purpose of exploring the social and environmental causes of race-associated health differences, defines out of existence the question of whether genetic factors might sometimes be important. Likewise, eliminativism forecloses *all* questions about the origins of racial variation in health outcomes.

Nevertheless, the use of race in biomedical research is neither simple nor straightforward. Not only is the question of how race ought to be conceptualized and measured complicated, questions about the etiology of health and disease are complicated. Answers to questions about the origins of racial variation in health outcomes are likely to vary from disease to disease. Moreover, the answer in each case is likely to be multifactorial and to involve interactions among multiple environmental and social factors, sometimes including racism and possibly also genetic factors. If we proceed with overly simplified beliefs about what race is, its ontological status, or about the role that race is likely to play in helping us to understand the origins of disease—we are likely to come to the wrong conclusions (or to the right conclusions for the wrong reasons). It is also important to keep in mind that the question of whether race is a scientifically valuable biomedical variable is not one that can be decided in a general way. This issue is not likely to be settled by appeal to a global argument (or set of such arguments) that supplies a general reason, or set of reasons, for the use or the abandonment of race in all biomedical contexts. Instead, what is needed is a close examination of specific hypotheses that aim to explain racial variation in the incidence of a specific health outcome.

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Notes:

(1.) There is a debate within eliminativism about whether “ethnicity” should replace race as a variable in biomedical research. See Williams (1997) and Sankar and Cho (2002) for critical discussions of this suggestion.

(2.) See Haslanger (2000), Root (2000), and Sundstrom (2002) for some defenses of race constructionism.

(3.) Biological race realism can be roughly defined as the idea that human racial categories exist independently of human classifying activities.

(4.) See Wasserstrom (2001), Young (2001), and Haslanger (2005) for discussions of global forms of eliminativism and conservationism.

(5.) There are several related questions about the value of race in the treatment of individual patients. Is it useful to take a patient's race into account when diagnosing and

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treating individual patients? Even if it is useful, is it advisable to do so—given the potential for harmful social consequences? These questions are beyond the scope of this chapter.

(6.) See Andreasen (1998, forthcoming) for discussion of some of the problems with these and other biological conceptions of race.

(7.) See Food and Drug Administration (2003), appendix 1, for a summary of these developments.

(8.) See Andreasen (forthcoming) for a discussion of this assumption.

(9.) To see this assumption at work, see Root (2001, 2003), Zack (2002), Keita and Kittles (1997).

(10.) John Dupré (2003) expresses sympathy for this view.

(11.) While Cooper et al. (2003) defend eliminativism, elsewhere Cooper appears to endorse strictly social conservationism (1993).

(12.) See Mayr ([1959]1994) and Sober (1980) for discussions of the distinction between typological and population thinking.

(13.) The extent of reproductive isolation among human populations in the distant past is still under debate in the evolutionary literature. Defenders of the out-of-Africa hypothesis, for example, tend to maintain that reproductive isolation was fairly extensive, whereas defenders of the trellis model tend to deny this claim.

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