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INTELLIGENCE, RACE, AND GENETICS

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A number of scholars claim to have studied relationships between intelligence, race, and genetics.¹ The thesis of this chapter is that many of these studies are not grounded in scientifically derived constructs but rather, in large part, in folk beliefs about them. There is a big difference between studying relationships between constructs and studying relationships between folk beliefs regarding those constructs. The bigger problem, however, is when one studies the latter but believes one is studying the former.

In the first part of this chapter, we review the constructs of intelligence and of race. In the second part, we discuss conceptual and methodological problems associated with studies that have attempted to examine the relationship between race, genes, and intelligence.

INTELLIGENCE

To study the interrelationships among intelligence, race, and genetics, we need to know what intelligence is. We do not know. Hence, any conclusions about its relationships to other constructs will be, at best, tentative.

Explicit Theories of Intelligence

One way to figure out what intelligence is has been to ask experts. Two major symposia have done so.² Each of the roughly two dozen definitions in each symposium was different. There were some common threads, such as the importance of adaptation to the environment and of the ability to learn. But these constructs themselves are not well specified. And very few tests of intelligence directly measure either one. Tests do not offer adaptive tasks one is likely to face in everyday life. Nor do any tests directly measure ability to learn, except dynamic tests that require learning at the time of test.³ Rather, traditional tests much more measure past learning, which can have resulted from differences in many things, including motivation and available opportunities to learn.

Some theories of intelligence extend this definition by suggesting that there is a general factor of intelligence, often labeled *g*, which underlies all adaptive behavior.⁴ In many theories, including the theories most widely accepted today,⁵ other mental abilities are hierarchically nested under this general factor at successively greater levels of specificity. For example, Carroll has suggested that three levels can nicely capture the hierarchy of abilities, whereas Cattell and Vernon suggested that two levels were especially important.⁶ In the case of Cattell, nested under general ability are fluid abilities of the kind needed to solve abstract reasoning problems such as figural matrices or series completions and crystallized abilities of the kind needed to solve problems of vocabulary and general information. In the case of Vernon, the two levels corresponded to verbal-educational and practical-mechanical abilities. What we know about group differences is largely about so-called *g* and major group factors, such as verbal and spatial skills. More modern theories extend intelligence much further, such as to creative and practical as well as analytical abilities⁷ or to eight distinct multiple intelligences.⁸

Implicit Theories of Intelligence

Lay conceptions of intelligence are quite a bit broader than the conceptions of psychologists⁹ who believe in general ability, or *g*.¹⁰ For example, in a study of people's conceptions of intelligence Sternberg and his

colleagues found that laypersons had a three-factor view of intelligence as comprising verbal, practical problem solving, and social-competence abilities.¹¹ Only the first of these abilities is measured by conventional tests. Experts in different occupations in the United States have somewhat different conceptions of intelligence, with their views of the relevant attributes tending to match the requirements of their occupations.¹² And conceptions of intelligence around the world vary even more than they do in the United States.¹³

The way intelligence is usually defined in studies of the alleged relationships between intelligence, race, and genetics is in terms of Boring's operational definition of intelligence as whatever it is that IQ tests measure.¹⁴ This definition is unsatisfactory for at least three different reasons. First, the definition is circular, defining the construct in terms of the operation and the operation in terms of the construct. Second, so-called IQ tests do not all measure the same thing.¹⁵ Third, as we have seen, theorists of intelligence do not themselves agree as to what intelligence is.

For convenience, we can follow Boring and operationally define intelligence in terms of IQ as measured by conventional tests. But it is not clear that tests of IQ measure the same construct among all the people to whom the tests are applied.¹⁶ The more culturally distinct the people, the greater are the differences in what the items measure.¹⁷ In part this is because IQ-test items are, largely, measures of achievement at various levels of competency.¹⁸ Items requiring knowledge of the fundamentals of vocabulary, information, comprehension, and arithmetic problem solving—so-called measures of crystallized abilities¹⁹—are clearly measures of achievement. Items requiring fluid abilities²⁰ involving abstract reasoning, once thought to be culture-fair,²¹ have proven even more susceptible to the effects of cultural and other environments than tests of crystallized abilities,²² suggesting they are in no way "culture-fair." Western-style schooling even more extensively inculcates these ways of abstract or fluid thinking than it does those measured by tests of crystallized abilities.

In sum, it is probably more accurate to say that these existing studies refer to the relation between "IQ" or "psychometric *g*" and what is labeled as "race," rather than to "intelligence" and these other constructs. Does the language we use matter? Yes. We need to acknowledge that we are using convenient, partial operationalizations of the construct of intelligence, and nothing more. As professionals, some of us may understand that

there is a large gap between the conceptualization and operationalization of intelligence. Others of us may act as though IQ tests somehow provide the kind of measurement of intelligence that a tape measure provides of height. When we are dealing with the lay audiences who learn about our work, it is especially important that we acknowledge that we have nothing even vaguely close to a "tape measure" of intelligence.

RACE

Just as there are different ideas about how to define and measure intelligence, there are several different ideas about how to define and measure race. Most scientists who study such matters believe that the original modern humans, of whom all living humans are descendants, lived in Africa.²³ They first appeared roughly two hundred thousand years ago. For whatever reasons—to find food, to satisfy wanderlust, to find better protection against predators, to find more land—small numbers of unrepresentative people started to migrate out of Africa about one hundred thousand years ago.²⁴

The "Out-of-Africa" hypothesis places the first immigrants from Africa in southwest Asia. Over the course of tens of thousands of years, that initial non-African population expanded until there are now at least some people to be found on all continents and in most regions of those continents, except for Antarctica, which, in general, is too cold to be hospitable, at least for modern humans. As people migrated, they adapted to better fit their environments. Much of that adaptation was cultural—different clothing, different foods, for example—but some of the adaptation was genetic (e.g., a genetic response to the increased prevalence of malaria that occurred as a result of people's creation of agricultural fields and their irrigation). However, it is difficult to prove that traits seen to differ are truly the result of different selective pressures, that is, of genetic adaptations. A major reason for the difficulty is that at the genetic level we see quantitative differences in the frequencies of genetic variants, not qualitative genetic differences, among populations. When multiple forms of a DNA sequence, either a coding sequence or a noncoding sequence, are present, the sequence is referred to as polymorphic and the forms as alleles at the polymorphism. Among populations of various kinds, allele

frequency differences at polymorphisms are the rule because of the chance effects known as "random genetic drift." In other words, as a result of both natural and social events, only some genotypes are transmitted through generations, whereas the others are lost; the lack of predictability in who will have children and who will not introduces powerful random noise into allele frequencies between generations. Thus, observing different allele frequencies does not in and of itself imply that local selection has operated. Even in the extreme cases of an allele absent in one part of the world and the only allele in another, we usually see a gradual difference (a cline) in allele frequencies in the populations along the geographic region between. One example is an allele at *EDAR*²⁵ that results in thicker hair.²⁶ The bottom line is that all the recent large-scale studies of human populations have concluded that genetic variation is clinal (i.e., gradual) around the world, with general loss of genetic variation in populations correlated with distance from Africa, much of this pattern a reflection of the way humans expanded throughout Eurasia, the Pacific, and the Americas in the last sixty to one hundred thousand years.²⁷

MECHANISMS OF GENETIC INFLUENCE

Four mechanisms have influenced the genetic evolution of populations.²⁸ Consider each in turn. The first is *mutation*, by which DNA changes in random ways. Mutation results in the rise of both functional (i.e., coding) and nonfunctional (i.e., noncoding) polymorphisms as well as other structural variants.

The second is *random genetic drift*, by which alleles in finite populations may change in frequency over time as a result of the accumulation of random sampling error in the passing on of alleles from generation to generation. When a number of individuals migrates and starts a new population, the sampling error (random genetic drift) is inversely proportional to the number of founding individuals, and allele frequencies may be very different in the new population from those in the parent population. As the new population grows over a few generations, the magnitude of the sampling error per generation decreases and the new population will continue to have different frequencies from the parent population. The extreme form of random genetic drift is referred to as a

"founder effect" because the population expanded from very few founders with a relatively restricted gene pool. For example, available evidence suggests that a small group of individuals left Africa, thereby changing the allele frequencies from those in the African populations left behind. On a smaller scale, the expansion of that population across Eurasia can be modeled as a series of smaller founder events resulting in gradual changes in frequencies along the paths of expansion.

The third mechanism is *gene flow* or *genetic exchange*, by which interbreeding among certain groups of individuals potentially results in those populations becoming increasingly similar to each other. Two populations that start off quite different genetically, if they mate, can produce offspring that represent the genes in both of the original populations. At a more local level, exchange between adjacent populations will, over time, smooth the geographic pattern into a smoother clinal gradient in frequencies.

The fourth mechanism is *natural selection*, by which organisms with gene patterns that are adaptive to a given environment become more prevalent over time. For example, organisms that can adapt to changing climatic patterns are at an advantage over those that adapt only with great difficulty.

MIGRATION AND ADAPTATION

Although all of these mechanisms are of importance, here we will illustrate only that of natural selection. Consider the following example. During the Industrial Revolution in late-nineteenth-century England, a particular dark-colored moth became more prevalent than a related light-colored moth. Why? It is believed industrial pollution had blackened the forests and improved the darker moth's camouflage against predators such as birds. The light-colored moth was too visible to survive. More recently, however, with restrictions on air pollution, the light moth has made a comeback.²⁹ The point, of course, is that natural selection is a constantly shifting process. It is influenced not only by an organism's biology, but also by the interaction of that biology with environmental conditions.³⁰

Is it better from the standpoint of adaptation to the physical environment to be a black moth or a light-colored moth? It depends on the

interaction between the organism's attributes, including color, and the particular environment. Is it better from the same adaptive standpoint to be a black person or a light-colored person? The answer is the same, of course. In zones with more intense exposure to sunlight, darker skin puts individuals at an adaptive advantage. The melanin that acts as pigmentation to produce darker skin better protects individuals against the damage that large amounts of ultraviolet radiation can cause to the skin. Left unchecked, this radiation increases susceptibility to skin cancer, especially melanoma, a form of skin cancer that easily can become fatal. In zones with weaker exposure to sunlight, lighter skin is an advantage.

One explanation of lighter coloration pertains to vitamin absorption. People rely on sunlight to produce active vitamin D₃ in the capillaries. The active form does not occur in great quantities in the food most people eat. Indeed, today milk is often supplemented with vitamin D₃ to prevent deficiencies. Lighter skin allows greater bodily production of vitamin D₃. Deficiencies in vitamin D₃ can cause rickets in children and osteoporosis in adults.³¹

A second explanation is of a different kind. There is as yet no conclusive evidence for positive selection for light coloration. Instead, evidence to date may favor as much or more the simple relaxation in northern climes of the strong selection for dark pigment found in equatorial regions as an explanation for light coloration in zones distant from the equator.³² Individual moths or other animals do not radically change in color in the course of their lifetimes. Rather, over time, those descendants that are better adapted are more likely to survive and reproduce, so that distributions of traits change. Human populations adapt over many generations. But not all organisms do. Some adapt very rapidly. Generations of bacteria, for example, adapt rapidly because of their extremely rapid rates of reproduction. It is for this reason that the same medication, Amoxicillin, which was effective in treating ear infections in the children of twenty years ago, is so much less effective in treating ear infections in the children of today. Bacteria have adapted, in the same way that malaria parasites have adapted to many quinine-based treatments and in the same way that the HIV virus is adapting to medications being used to treat it. All biological populations adapt, whether bacterial, human, or anything else.

There is another key fact in this story. Aside from the explanations of skin color, there are not a lot of scientifically supportable selective explanations

for the differences we see in people from different parts of the world. It is probable that much of the variation that we see among groups of humans indirectly resulted from the pattern of expansion and migrations accompanied by random genetic drift. Over the years, frequencies of specific alleles at various single-nucleotide polymorphic sites (i.e., Single Nucleotide Polymorphisms, SNPs) changed only slightly in terms of nucleotide composition, but enough to make differences, many of which we still do not fully understand. The changes are numerous. Less than 1 percent of the three billion nucleotide positions in the human genome varies globally, as SNPs and other types of variation; but that percent creates a large number of potential differences between any two people. Some of the individual polymorphisms have different frequencies around the world; others have similar frequencies everywhere. In addition, structural variation, so-called Copy-Number Variation (CNV), can also be found; this type of variation has been discovered recently and has attracted much attention.³³ At this point, little is known about geographic differences in CNV but certain regions of the genome do seem prone to generate these duplications of genes or deletions of genes. The human genetic material, the genome, shows considerable variation among individuals when examined at the DNA level and the variants have different frequencies in different parts of the world. How that DNA variation affects variation in individual common traits is as yet poorly understood. Yet, we see larger proportions of blond hair and blue eyes in people born in European countries than those born in Asian ones. We will see shorter people, on average, among those born in Asia than among those born in Europe. We see wider noses in West Africa, on average, than in East Africa. Nevertheless, even within groups, there is tremendous variation.

RACE AS A SOCIAL CONSTRUCTION

Where does race fit into the genetic pattern? Actually, it fits nowhere. *Race is a socially constructed concept, not a biological one.* It derives from people's desire to classify. People seem to be natural classifiers. Perhaps this tendency reflects, in part, what Gardner has referred to as "naturalistic intelligence."³⁴ Or perhaps it merely reflects a need to discern order in or even to impose it on nature. Any set of observations can be

categorized in multiple ways. People impose categorization and classification schemes that make sense to them and, in some cases, that favor their particular goals.

If one looks at geographic patterns, one will find many attributes that correlate with geography—nearby populations tend to be similar and distant populations dissimilar. This pattern is similar to common ideas of socially defined races but is more complex.³⁵ People in different places came to demonstrate different characteristics by adaptations to different environments, such as heterozygosity for sickle-cell hemoglobin as a partial protection against malaria, as well as by accumulation of random genetic drift. But as is so often the case, the same trait that may be adaptive in one circumstance may be maladaptive in another. For example, there is no advantage to sickle-cell hemoglobin in the absence of malaria and the anemia that results in homozygotic individuals poses a serious disadvantage.

Other adaptations are equally fickle. Today, our population is paying the price of tens of thousands of years in which people became genetically programmed to enjoy fats, sugars, and salt and to eat as much of them as they could when they have the opportunity. In the contemporary environment, the result is large numbers of overweight and obese individuals. Some people have more of a genetic predisposition to gain weight than others. Social stratification—classifying people into categories of higher and lower status in a society—has already ensued on the basis of weight.³⁶ Whether, ultimately, people with a genetic predisposition toward fatness will be classified as being of a separate race remains to be seen. The point is that an adaptation that is positive at one time or place may be indifferent at another and negative at still another.

One could pick any of a number of traits correlated with geographic patterns and find correlations with other related traits. It would be foolhardy, however, to view any one of these traits as causative of the others. That is what people have done who have viewed differences in so-called races as somehow causative of differences in IQ. It also would be foolhardy to group fairly arbitrary sets of traits and constructs that one then reifies as being natural, somehow God-given categories. One will find a distribution of traits in any of these groups, with only slightly more differentiation when comparing individuals from different groups rather than individuals within any one group.³⁷ Why would people do this, then? One reason is to justify existing social stratifications or to create new ones.

We could of course refer to moths as being of different "races" (black and white), in the same way we sometimes refer to humans as being of different "races." We do not typically use the term for moths, presumably because we are less interested in creating social stratifications for moths than for people, and race is one way to help create these stratifications. Of course, we recognize that our chapter may have the opposite effect from that intended: Some believers in biological race may realize that moths (and perhaps dogs, cats, and other animals that come in multiple colors) have been sorely neglected in the literature on racial differences, and that there is still time to remedy this situation. To the extent we define race as simply different sets of physical features, we could say, of course, that the moths are of different races. But the term, used in this way, becomes simply a word for saying the moths look different! And the surplus meaning associated with the word, at least as it is used in human descriptions, vanishes.

Over time, peoples who migrated changed both by chance and by adaptation to their environments in various ways. What is "good" depends on the adaptations that need to be made, and these adaptations change from time to time and place to place. For example, our ancestors in Africa were almost certainly dark-skinned because dark skin provided better protection against the particular challenges of the environment, most notably, ultraviolet and other harmful forms of radiation. Other traits, such as straight or curly hair, have no evident adaptive value and population differences probably reflect chance differences. Curiously, then, socially constructed judgments as to how socially to classify people are made on bases that have no relation to the original reasons that people came to look one way or another.

There is nothing special about skin color that serves as a basis for differentiating humans into so-called races. Any two groups of people that differ in one way are likely to differ in a cluster of ways. For example, as noted by Marks, geneticists have found that 54 percent of people who have designated themselves as Hebrew priests, many of whom have the surname Cohen, have a certain pattern of two genes on the Y chromosome.³⁸ In contrast, only 33 percent of Jews who do not view themselves as priests have this pattern. What conclusion is to be drawn? Well, the correct conclusion is that different groups of people will differ in various respects. The authors of the study concluded that one could infer a genetic Jewish priestly line dating back to the Biblical Aaron.³⁹ Other

bases for differentiation could be chosen as well, including the aforementioned one of girth. The point is that people will often draw conclusions that go well beyond the data, as when they take a correlation to imply causation or when they take genetic variation to have implications for a Jewish priestly line. There may be a causal link, but the evidence is insufficient to conclude as such.

As another example, Fish has pointed out that people who have lived over many generations in cold climates, such as Eskimos, have tended to develop rounded bodies to maintain heat and thus stay warmer.⁴⁰ Some populations in very hot climates, such as the Masai, have tended instead to develop lanky bodies. The hypothesis is that the high ratio of surface area to volume results in their radiating a lot of heat and thus staying cooler. While reasonable, both adaptation hypotheses lack rigorous scientific proof. Possibly, they could be just coincidences. Scientists do not know for sure.

In the American folk taxonomy of race, as Fish argued, lanky and rounded people can be, respectively, two kinds of blacks and whites.⁴¹ But one could as easily decide that a more "basic" taxonomy of races would be in terms of lanky and rounded bodies, in which case there would be black and white members of the lanky and rounded races. One would find a number of genetic patterns that, on average, correspond to lankiness and roundedness, in the same way one would find genetic correlate patterns corresponding to darker or lighter skin, or Cohens versus non-Cohens, or basketball players versus wrestlers.

It has been argued that the challenges faced by those who migrated to Northern climates were greater than those faced by people in Southern climates, and that this difference might have led to higher intelligence of those who went northward.⁴² However, anyone who has spent any significant time in Africa might well dispute this claim. One of the greatest challenges of tropical climates is fighting tropical diseases to survive, and the challenges of fighting diseases are greater in the tropics than they are further north. Indeed, children acquire from an early age specialized knowledge, not acquired further north, regarding natural herbal medicines that can be used to combat tropical illnesses.⁴³ To the extent that warmer climates encourage greater aggression,⁴⁴ learning how to compete successfully so as to survive in such environments also might promote intellectual development. We are not arguing that people

in warmer climates did indeed develop higher intelligence, but rather, that one could create speculative arguments supporting greater intellectual growth in such climates, as has been done to support the notion that there was greater intellectual growth as a result of challenges up north. Indeed, post hoc evolutionary arguments made in the absence of fossils at times can have the character of ad hoc "just so" stories designed to support in retrospect whatever point the author wishes to make about present-day people.

Differences in socially constructed races stem largely from geographic dispersions that happened long past, starting about one hundred thousand years ago but continuing until about three thousand years ago in some areas. Today we see the physical correlates left by the dispersions. Much of that variation is continuous across distances but with different traits showing different rates and patterns of change. What "race" does is to reify these differences as deriving from some imagined natural grouping of people that does not, in fact, exist, except in our heads.

What we see in terms of skin color correlates very well with our developed folk taxonomies, but only weakly with genetic differentiations. For example, the amount of genetic variation in Africa is enormous and is much greater than that in the rest of the world.⁴⁵ In contrast, in terms of the amount of phenotypic variation, or differences in appearances, Africa is at least comparable to the rest of the world. The phenotypic differences are nevertheless notable. For example, in Africa, one can find very tall Masai, and very short Pygmies who probably gained an adaptive advantage by virtue of their shortness for locomotion through forest vegetation.⁴⁶ Yet, some may lump together all these Africans as the same, though genetically they differ more from each other, in many cases, than they do from those who perceive themselves to be of higher social, or even biological, value.

Humans have devised various metaphors for understanding why some people are more successful, according to whatever standards society invents, than others. Usually, the comparisons are drawn by those who consider themselves successful for the benefit of others who consider themselves successful, or on the road to success.⁴⁷ A curiosity of history is that people come to believe in the reality of their own metaphors. For example, some have believed, and some still believe, in "royal blood." Educated people probably realize that the expression is metaphorical; others

probably believe that the blood of royals differs in some key respect from the blood of others.

For readers of this chapter, a biological concept of "royal blood" probably seems silly. But at the same time, we know that there are distinguishing blood groups. For example, most of us are familiar with the ABO and Rh blood-typing systems. According to Lewontin, there are roughly thirty-five blood group systems, with fifteen serving at least somewhat effectively to distinguish different human populations.⁴⁸ Royal blood, at least within families, may well show distinguishing blood groups, just as nonroyal families would. So in this trivial sense, royal blood can be said to exist, but differently in different royal families. In this same trivial sense, there can be differences in distributions of blood groups across religious groups, people with different body shapes, or people with different skin colors.

How mixtures are labeled is a function of social status. In the United States, blacks generally have lower social status than whites, so supposed admixtures of blood determine degrees of "blackness." Having any blackness makes one socially black in some degree. So one can be light black, or medium-skinned, or dark black, but one is still socially black. Even if one of mixed parentage inherited none of the physical features of blackness, one would still be classified socially as black, although one might pass for white.⁴⁹ Where blacks are of higher social status, degrees of whiteness may all be seen as departures from true blackness.

The concept of race serves a social, not a biological, purpose. Different types of parentage have, at various times and places, given rise to racial labeling, as, for example, in the "Aryan race," the "German race," the "Jewish race," and so forth. In Apartheid South Africa, the races were Bantu (Black African), colored (including people of perceived mixed descent), Indian/Asian, and white. In contemporary North American society, we mix together the black and colored "races," somehow believing, as noted above, that if someone has any degree of nonwhiteness, it puts that individual into the black category. Hitler designated as a member of the Jewish race anyone who had supposed Jewish blood, which could date back to one's great-grandparents.

In Brazil, the supposed races are different again.⁵⁰ A *loura* has straight blond hair, blue or green eyes, light skin color, and a narrow nose and thin lips. A *branca* has light skin color, eyes and hair of any color, a nose that is not broad, and nonthick lips. In Brazil, Fish points out, a *branca* is white.

In the United States, a *branca* individual from Brazil would more likely be classified as "Hispanic." Then there is a *morena*, who has brown or black hair that is wavy or curly but not tight curly, tan skin, a nose that is not narrow, and lips that are not thin. *Morenas* in the United States are classified as black or Hispanic. There are a number of other Brazilian terms used to describe socially constructed racial categories, such as *mulata* and *preta*, and to the Brazilians, these terms are every bit as real as the black, white, and Asian categories are in the United States. They *are* real. But as in the United States, they are folk, not biological, taxonomies, which are used to socially stratify people, often in the name of science. At best, the effects are innocuous. At worst, they become the bases of genocides.

People generally use skin color to distinguish races, but not always. During the genocide in Rwanda, the Hutus used other physical attributes, such as height, to distinguish Tutsis. Because there had been so much intermarriage between Hutus and Tutsis, the distinctions were generally weak, and many people were killed simply because they seemed closer to the imagined Tutsi prototype than the Hutu one, regardless of their origins.

The history of the concept of race is inextricably intertwined with attempts by the winners to explain or justify why they perceive themselves to be winners. Consider, for example, the term "Caucasian." It is an odd term, in some ways, because although it is used to refer to "whites," in Russia, people from the Caucasus are considered dark relative to many other Russians. Especially because of political difficulties in Chechnya and surrounding areas, these dark Caucasians today are viewed with suspicion and distrust in much of Russia. So the term that is accepted as "scientifically" identifying white people in the United States, often in preference to the term "white" to give more of a feeling of scientific classification, is used in a way that is largely opposite in contemporary Russia. Where did the term come from then? It was invented in 1795 by Johann Friedrich Blumenbach,⁵¹ who chose the name because he believed that the Georgians, from the Mount Caucasus region, were the most beautiful race of men (his words). The term stuck. So people in English-speaking countries with white skin have the honor of having a name they imagine to be the formal name for their race representing what one naturalist in 1795 believed was the most attractive "race" and what today largely is believed to be rather dark, not white, skin according to Russian standards. Thus, the term is scientifically unsupportable and part of an old racist

typology. The term is just as racist as Negroid and Mongoloid, terms the politically sensitive American will not use.

ORIGINS OF THE CONCEPT OF RACE

Whence emerged the concept of "race"? The concept of race as a classification scheme representing allegedly natural "types" distinguishable on the basis of clear visual attributes such as skin or eye color, hair texture, and certain facial and bodily features was initially introduced in the seventeenth century.⁵² However, it took these ideas almost a century to attract the attention of scientific "authorities." According to Gould, Linnaeus (in 1758) first proposed four races: *Americanus*, *Europaeus*, *Asiaticus*, and *Afer* (or African).⁵³ He also alluded to two other categories that did not prove as useful for social purposes as the other categories: wild boys (feral children) discovered in the forests, and monsters, hairy men with long tails, who emerged from tales of travelers. Blumenbach (in 1775), building on the work of Linnaeus, first proposed a grouping of "races," namely, Caucasians, Mongolians, Ethiopians, and Malays. This early history was no more scientific than the later history was to be. That is, race started out as a not so subtle way of socially classifying and, ultimately, stratifying people hierarchically—as better or worse. For example, Linnaeus viewed the white as sanguine and muscular, and the black as phlegmatic and relaxed.

Historically, the formation of the concepts of race and ethnicity was influenced by two main perspectives.⁵⁴ One perspective was formed in the context of the eugenics movement and was used to refer to presumed biological differences between socially defined populations.⁵⁵ The other perspective was formed in the context of physical anthropology and the social sciences and rejected the idea of the biological significance of racial classifications. It argued that race and ethnicity are primarily cultural and historical products of human history.⁵⁶ Today, whereas some still defend the basis for the "gene-based evolutionary theory" of race,⁵⁷ the majority of cultural anthropologists are in agreement that race is a socially constructed, not an evolutionary determined or biologically supported, concept.⁵⁸ Of course, science does not find truth by majority rule. *The problem with the concept of biological race is not that it is supported by only a*

minority of anthropologists, but that it has no scientific basis. Moreover, attempts to link intelligence, race, and genetics have also lacked adequate scientific foundation.

INTELLIGENCE, RACE, AND GENETICS

Despite the inadequacies that have been pointed out with regard to the definitions of both race and intelligence, several studies have attempted to examine the relationship between race and intelligence using proxies that are intended to intimate a biological basis for each construct. One set of studies attempts to give biological credence to the concept of race by equating it with skin color. Another set of studies attempts to justify the reification of race using the argument that race is an important factor in customizing medical diagnoses and treatments. A third set of studies uses correlational twin study data to make causal assertions about the relationship between intelligence, genes, and race. A fourth set of studies relies on misguided interpretations of heritability studies for making cross-racial comparisons. Each of these arguments suffers from a number of serious flaws.

Skin Color Is Not Tantamount to Race

Many studies that purport to investigate race as a biologically based construct use self-reported skin color as a proxy for some sort of presumed innate biological marker of race.⁵⁹ There are several problems with this approach. First, the operational definition of race based on skin color varies substantially over time and space. What one group sees as one race based on a certain color (e.g., "black" in the United States), another group may see as another race based on the same color (e.g., "colored" in South Africa).

Second, even if self-reported skin color could be reliably measured over time and space, there is no genetic evidence to support the idea that individuals with a shared skin color share other types of genes in common more frequently than individuals of different skin colors. A simple thought experiment will illustrate this point. Suppose we put one hundred randomly selected people in a room together behind a curtain. We then have a competition in which the object of our game is to choose

the individuals with the closest genetic match. Contestant A and Contestant B each only get one variable on which to evaluate those one hundred individuals. Contestant A chooses skin color as his variable. Thus, the forearms of one hundred individuals are stuck through the curtain and individuals are categorized into groups according to the degree to which their skin colors are similar. Contestant B chooses to listen to each of the one hundred individuals say a sentence in English. The individuals who sound the most alike (indicating that they are from the same part of the world, most likely, even if they differ drastically in their skin color) are then categorized into groups. Chances are that Contestant B will win the game much more often than Contestant A if we run an analysis of all of the alleles these two individuals in both groups share. The individuals who sound more alike are more likely to hail from the same parts of the globe and will therefore be more genetically similar to each other. But certainly similarity in English speech patterns does not form the basis for a racial classification.

The point is that although skin color is genetically determined, it does not imply that people with the same skin color share many other genes in common. In fact, there is a tremendous amount of variability in the extent to which two individuals with the same skin color share their remaining genes.⁶⁰ By contrast, the genetic evidence does suggest that individuals from a similar part of the world tend to share more genes in common than people from parts that are remote from one another. The more geographically distant individuals are from each other, the fewer genes they seem to have in common, on average.

Third, the data presented by Templer and Arikawa⁶¹ and by Lynn⁶² showing correlations between "national skin color" and "national average IQ" suffer from many conceptual and methodological problems that have been addressed in detail by others in the literature.⁶³ One of the more blatant problems with these data is that the samples used are not random selections from the population, but rather samples of convenience. Perhaps the most basic lesson of survey methodology is that sample size is no substitute for sample representativeness. Although a truly representative sample of approximately three thousand individuals can reasonably accurately represent three hundred million individuals, even a sample size of over two million individuals, when not representatively sampled, can lead to gross errors in statistical inference due to the infiltration

of uncontrolled third variables. This finding was perhaps most notoriously illustrated by the 1936 US election in which *Literary Digest*, the top pollster in the United States at the turn of the century, conducted a poll of over two million individuals and predicted that Alf Landon would defeat Franklin Roosevelt in a landslide. The result, of course, was just the opposite. FDR defeated Landon in a landslide. The problem was that the pollsters did not recognize the confounding variables that crept into the study when they drew their sample from lists of car owners and telephone owners in 1936 and they relied on voluntary response (just over 20 percent of individuals who were mailed surveys responded). Thus, any data from nonrepresentative samples that are used to make inferences to a larger population are suspect.

A second major problem with these data is that they concocted a measure of "national skin color" using the opinions of three graduate students. Not only does the concept of an "average" national skin color ignore important variability within each nation with regard to skin tone differences, but the fact that three independent students agreed on these classifications suggests only that they share the same implicit theories, prejudices, erroneous preconceptions, etc. Thus, what is being measured is not "national skin color" but rather social stereotypes about skin color.

In addition to those critiques, however, there is also a statistical-inference problem known as the ecological fallacy that plagues such data. The ecological fallacy occurs when relationships observed in groups are assumed to hold for individuals.⁶⁴ Cross-level inferences are not supportable without running multilevel statistical models.⁶⁵ Kreft and de Leeuw illustrate the problem of cross-level inferences when they note that:⁶⁶

Kreft et al. . . . ran a study in which data were collected on workers in 12 different industries. Individual-level variables are education level as the explanatory variable, and income as the response variable. The type of industry, as well as the distinction between public and private industries, are the second-level variables. An analysis with these data, executed at the level of individual workers, shows a positive relationship between education level and income: the higher the educational level, the higher the personal income. An analysis executed at the higher level, the industry level, with 12 industries as observations, shows a surprisingly opposite result. A negative relationship shows up between education and income.

The higher the average educational level of an industry, the lower is the average income of workers in that industry. Universities and colleges are a good example.

In a similar vein, investigations that occur at the group level (e.g., "group intelligence" and "national skin color"), which are then used to make individual-level inferences, cannot be supported without multilevel statistical models.

Racial Profiling in Medicine

One of the other major arguments found in the literature suggests that race must have a biological basis because the field of medicine is currently actively pursuing racial profiling for medical treatments. For example, some research exists to show that different ethnic groups in the United States exhibit substantial average differences in areas such as disease incidence, prevalence, severity, and response to treatment.⁶⁷ Furthermore, there is ample evidence to suggest that the health disparities observed between different ethnic groups in the United States arise mostly through the environmental effects of discrimination, poverty, restricted access to health care, stress, and other socially mediated forces.⁶⁸

It is worth noting that in February 2001 the editors of the medical journal *Archives of Pediatrics and Adolescent Medicine* asked authors no longer to use race as an explanatory variable and not to use obsolescent terms.⁶⁹ Some other high-impact peer-reviewed medical journals, such as the *New England Journal of Medicine* and the *American Journal of Public Health*, have made similar appeals.⁷⁰

In the end, perhaps the fundamental problem with racial profiling in medicine is that it ignores the importance and potential confounding of environmental influences. As Risch et al. point out, "The true complication is due to the fact that racial and ethnic groups differ from each other on a variety of social, cultural, behavioral, and environmental factors as well as gene frequencies, leading to confounding between genetic and environmental risk factors in an ethnically heterogeneous study."⁷¹ In other words, even if one could be confident about true genetic differences between groups and if one could create customized drugs for particular populations, one would need to take into account the effects

that systematic cultural differences make with regard to areas such as diet and exercise.

Twin Studies

Perhaps the most widely used approach historically to studying the link between intelligence, race, and genes has been through the use of twin studies. After all, the argument goes, identical twins share 100 percent of their genes in common, siblings share 50 percent of their genes in common, and strangers share 0 percent of their genes in common. Thus, by studying each combination reared in the same environment or reared apart, we should be able to firmly disentangle the influence of genes from the influence of environment on variables such as intelligence. Indeed, several studies have demonstrated that over time, the correlations between the IQ scores of identical twins become stronger whereas the correlations of IQ scores among siblings shrink to nearly zero.⁷² On the surface, these data appear to provide a powerful argument in favor of the influence of genes upon IQ. Unfortunately, it is not so simple to disentangle genetic influences from environmental influences this way.

For example, as Flynn has pointed out, for us to make a genetic attribution to the high correlation in IQ among identical twins, we must assume that their environments have no more in common than those of randomly selected individuals.⁷³ Yet, this assumption is likely to be untenable. Indeed, small genetic differences can interact with the environment to lead to what are called "multiplier effects." As an example, Flynn asks us to consider an analogy. Suppose a pair of identical twins is separated at birth. Both individuals may live in a social context that values a sport like basketball. And both twins may have a slight genetic advantage that makes them taller and quicker than average children. As a result of this slight advantage, both individuals will most likely be selected at an early age to play on the local basketball team. They may then receive more practice, coaching, and playing time, which then influence their basketball playing ability. The effects of these increased opportunities for further exposure to the game and development of skills lead both individuals to demonstrate strong basketball skills as they age. By contrast, consider a second set of identical twins separated at birth who are genetically shorter and chubbier than the average child. These children would

likely not be selected for the basketball team and would not be exposed to the increased hours of practice, etc. As a result, these twins would also score similarly poorly on a test of basketball ability when they age. Thus, "genetic advantages that may have been quite modest at birth have a huge effect on eventual basketball skills by getting matched with better environments—and genes thereby get credit for the potency of powerful environmental factors."⁷⁴ Thus, one way to explain the increased similarity is via multiplier effects. A second explanation for the recent data by Davis et al. is that as people age, they gain more direct control over their environment and are therefore better able to select environments that are aligned with their genetic predispositions.⁷⁵ For example, more athletic children may turn into adults who like to spend most of their time outside working whereas children who may not be as athletic may enjoy spending more time reading. Thus, as individuals age, they will tend to choose environments that enhance their strengths, and these environments may differ from individual to individual.

Race and Heritability Studies

The explosion of genetic research within the last ten to fifteen years has brought the concept of race back to the surface, with some researchers arguing that new molecular data have given the concept of race new significance in the context of medicine and public health.⁷⁶ One might think that, because the concept of race originated as a social proxy for the description of biological differences, at least the biologists studying race would agree on its definition. However, the reality is different. When variation in genetic markers or allelic variants is considered, opinions range widely. One view is that socially defined racial differentiation is most pronounced and even discontinuous when it is evaluated on the basis of continental residence.⁷⁷ A second view is that there is continuity in genetic variation across socially defined races and that various races are not distinct, but rather a single lineage with a shared evolutionary fate.⁷⁸ On this view, there is no biological value in the concept of race.⁷⁹ However, considering these positions, it is important to understand that, even within these extreme views, researchers agree that, although human populations might differ dramatically in terms of proportions or frequencies of alternative forms of genes, that is, allelic variants, they do not

differ in the kinds of genes they possess.⁸⁰ In fact, both extreme views may have some merit.⁸¹

A key argument of this article is that race is every bit as real as royal blood. It exists in some trivial sense as a correlate of various biological groupings stemming from migration and breeding patterns, and no more. However, just as royal families are usually interconnected and difficult to partition off fully, defining the boundaries between races is impossible. As *The American Heritage Dictionary of the English Language* notes on usage, "many cultural anthropologists now consider race to be more a social or mental construct than an objective biological fact."⁸²

Although attempts have been made to establish genes for intelligence, no single gene has been conclusively identified.⁸³ To date, there have been six genome-wide scans for genes contributing to intelligence and cognition.⁸⁴ The results of these scans vary, but there are interesting partial overlaps. Specifically, the findings coincide in regions on chromosomes 2q (for four out of six studies), 6p (for five out of six studies), and 14q (for three out of six studies). These overlapping regions have been tentatively interpreted as indicative of the presence of genes that could explain some of the variance in IQ. Further, research has shown that specific genes such as *APOE*, *COMT*, and *BDNF* may play a role in intelligence; however, an in-depth understanding of the role of these genes remains elusive.⁸⁵ The IQ QTL project—a project aimed at identifying quantitative trait loci (QTL) contributing to genetic variation in intelligence⁸⁶—has attempted to establish QTLs associated with intelligence, but to date, whatever positive findings have emerged have either failed to replicate,⁸⁷ or produced weak signals that have not yet been attempted to be replicated in independent samples.⁸⁸ Most recently, Deary et al. found that "there is still almost no replicated evidence concerning the individual genes, which have variants that contribute to intelligence differences."⁸⁹ Of course, the future may bring conclusive identifications: we just do not know yet.

As a result, virtually all attempts to study genes related to intelligence have been indirect, through studies of heritability. But heritability is itself a troubled concept. Are differences in intelligence between so-called races heritable? The question is difficult to answer in part because it is difficult even to say what can be concluded from the heritability statistic commonly used. Consider some facts about heritability.⁹⁰

What Heritability Tells Us

Heritability (also referred to as h^2) is the ratio of genetic variation to total variation in an attribute *within* a population. Thus, the coefficient of heritability tells us nothing about sources of between-population variation. Moreover, the coefficient of heritability does *not* tell us the proportion of a trait that is genetic in absolute terms, but rather, the proportion of variation in a trait that is attributable to genetic variation within a specific population.

Trait variation in a population is referred to as phenotypic variation, whereas genetic variation in a population is referred to as genotypic variation. Thus, heritability is a ratio of genotypic variation to phenotypic variation. Heritability has a complementary concept, that of environmentality. Environmentality is a ratio of environmental variation to phenotypic variation. Note that both heritability and environmentality apply to populations, not to individuals. There is no way of estimating heritability for an individual, nor is the concept meaningful for individuals. Consider a trait that has a heritability statistic equaling 70 percent; it is nonsense to say that the development of the trait in an individual is 70 percent genetic.

Heritability is typically expressed on a 0 to 1 scale, with a value of 0 indicating no heritability whatsoever (i.e., no genetic variation in the trait) and a value of 1 indicating complete heritability (i.e., only genetic variation in the trait). Heritability and environmentality add to unity (assuming that the error variance related to measurement of the trait is blended into the environmental component). Heritability tells us the proportion of individual-difference variation in an attribute that appears to be attributable to genetic differences (variation) within a population. Thus, if IQ has a heritability of .50 within a certain population, then 50 percent of the variation in scores on the attribute within that population is due (in theory) to genetic influences. This statement is completely different from the statement that 50 percent of the attribute is inherited.

An important implication of these facts is that heritability is *not* tantamount to genetic influence. An attribute could be highly genetically influenced and have little or no heritability. The reason is that heritability depends on the existence of individual differences. If there are no individual

differences, there is no heritability (because there is a 0 in the denominator of the ratio of genetic to total trait variation in a given population). For example, being born with two eyes is 100 percent under genetic control (except in the exceedingly rare case of severe dysmorphologies, with which we will not deal here). Regardless of the environment into which one is born, a human being will have two eyes. But it is not meaningful to speak of the heritability of having two eyes, because there are no individual differences. Heritability is not 1: it is meaningless (because there is a 0 in the denominator of the ratio) and cannot be sensibly calculated.

Consider a second complementary example, occupational status. It has a statistically significant heritability coefficient associated with it,⁹¹ but certainly it is not under direct genetic control. Clearly there is no gene or set of genes for occupational status. How could it be heritable, then? Heredity can affect certain factors that in turn lead people to occupations of higher or lower status. Thus, if things like intelligence, personality, and interpersonal attractiveness are under some degree of genetic control, then they may lead in turn to differences in occupational status. The effects of genes are at best indirect.⁹² Other attributes, such as divorce, may run in families, that is, show familiarity, but again, they are not under direct genetic control; in fact, the familiarity may be because they are culturally "inherited."

Heritability Can Vary Within a Given Population

Heritability is not a fixed value for a given attribute. Although we may read about "the heritability of IQ,"⁹³ there really is no single fixed value that represents any true, constant value for the heritability of IQ or anything else, as Herrnstein and Murray and most others in the field recognize.⁹⁴ Heritability depends on many factors, but the most important one is the range of environments. Because heritability represents a proportion of variation, its value will depend on the amount of variation. As Herrnstein pointed out, if there were no variation in environments, heritability would be perfect, because there would be no other source of variation.⁹⁵ If there is wide variation in environments, however, heritability is likely to decrease.

When one speaks of heritability, one needs to remember that genes always operate within environment contexts. All genetic effects occur

within a reaction range, so that, inevitably, environment will be able to have differential effects on the same genetic structure. The reaction range is the range of phenotypes (observable effects of genes) that a given genotype (latent structure of genes) for any particular attribute can produce, given the interaction of environment with that genotype. For example, genotype sets a reaction range for the possible heights a person can attain, but childhood nutrition, diseases, and many other factors affect the adult height realized. Moreover, if different genotypes react differently to the environmental variation, heritability will show differences depending on the mean and variance in relevant environments.⁹⁶ Thus, the statistic is not a fixed value. There are no pure genetic effects on behavior, as would be shown dramatically if a child were raised in a small closet with no stimulation. Genes express themselves through covariation and interaction with the environment, as discussed further later.

Heritability and Modifiability

Because the value of the heritability statistic is relevant only to existing circumstances, it does not and cannot address a trait's modifiability. A trait could have zero, moderate, or even total heritability and, in any of these conditions, be not at all, partially, or fully modifiable. The heritability statistic deals with correlations, whereas modifiability deals with mean effects. Correlations, however, are independent of score levels. For example, adding a constant to a set of scores will not affect the correlation of that set with another set of scores. Consider height as an example of the limitation of the heritability statistic in addressing modifiability. Height is highly heritable, with a heritability of over .90. Yet height also is highly modifiable, as shown by the fact that average heights have risen dramatically throughout the past several generations.

As an even more extreme example, consider phenylketonuria (PKU). PKU is a genetically determined, recessive condition that arises due to a mutation (or, rather, a number of various rare mutations resulting in similar functional damages to the coded protein, see below) in a single gene, the *PAH* gene, on chromosome 12 (with a heritability of 1), and yet its effects are highly modifiable. Feeding an infant with PKU a diet free of phenylalanine prevents the mental retardation that otherwise would become manifest. Note also that a type of intellectual disability that once

incorrectly was thought to be purely genetic is not. Rather, the intellectual disability associated with PKU is the result of the interaction with an environment (a "normal" diet) in which the infant ingests phenylalanine. Take away the phenylalanine and you reduce level of, or, in optimal cases, eliminate intellectual disability. Note that the genetic endowment does not change: the infant still has a mutant gene causing phenylketonuria. What changes is the manifestation of its associated symptoms in the environment. Similarly, with intelligence or any other trait, we cannot change (at least with our knowledge today) the genetic structure underlying manifestations of intelligence, but we can change those manifestations, or expressions of genes in the environment. Thus, knowing the heritability of a trait does not tell us anything about its modifiability.

Within-Population Effects Versus Between-Population Effects

One of the worst intellectual slips that have been made by investigators of heredity and environment (or rather, most often, by interpreters of findings on heredity and environment) is to generalize the effects of within-population studies between populations. For example, some investigators have made attributions about effects of racial or ethnic group differences on the basis of behavior-genetic studies, even while admitting that such conclusions are sometimes flawed.⁹⁷ All of the behavior-genetic designs in the studies noted above can ascertain effects of genetic variation only within populations. For example, they may tell us something about the extent to which individual differences in the measured intelligence of people in a particular group are associated with genetic factors. They say nothing about sources of between-population differences in levels of measured intelligence.

An illustration of the impossibility of making between-population claims from within-population data has been given by Lewontin.⁹⁸ Specifically, in a study using a set of protein markers (blood groups, serum proteins, and red blood cell enzymes) as indicators of genetic differences between populations, Lewontin estimated that roughly 85 percent of the genetic variance occurs between any two individuals within any socially identified racial groups, roughly 9 percent occurs among different populations within a socially identified race, and only the remaining 6 to 7 percent occurs between socially identified races. Other researchers arrived at the

same conclusions using more powerful datasets obtained with more technologically advanced methodologies⁹⁹ or through simulation analyses.¹⁰⁰

Different populations—racial, ethnic, religious, or whatever—may encounter quite different environments, on average. Whatever the heritability of intelligence or other attributes within a given setting, no conclusions can be drawn about heritability as a source of differences across settings. The fact that IQs have increased so much over the years suggests that environments differ widely over time.¹⁰¹ They likely differ substantially as well for members of different groups at a given time.

Nisbett reviewed published studies investigating sources of differences in cognitive abilities between white and black individuals.¹⁰² These studies, using designs unlike the behavior-genetic studies described above, have directly sought to investigate genetic and environmental effects on intelligence. For example, one design (Scarr and Weinberg) has been to look at black children adopted by white parents. Of seven published studies, six supported primarily environmental interpretations of group differences, and only one study did not; the results of this one study are equivocal.¹⁰³ What the Scarr and Weinberg work study did show is that IQs of adopted children are more similar to those of their biological mothers than to those of their adopted mothers. Less clear are the “racial” implications of their findings.

Moreover, there is much published evidence indicating that heritability estimates vary across populations. For example, estimates of the heritability of IQ in Russian twin studies conducted in the Soviet era tended to be higher than comparable estimates in the United States.¹⁰⁴ This observation made sense: environmental variation in Russia under the Soviet regime was constrained; consequently, heritability estimates were higher. Most of the IQ heritability studies up to today have been carried out in various countries of the developed world. Relatively little information exists regarding the heritability of IQ in the developing world, although some studies suggest that heritability may be substantial, at least outside the Western countries that most often have been studied.¹⁰⁵

In sum, heritability estimates do not explain the genetic regulation of behavior and do not provide accurate estimates of the strength of the genetic regulation. Heritabilities are like snapshots of a dancer. Heritabilities will not tell us either what the dance is about or what is coming next in the dance. The true genetic nature of humans is far from being defined.

But what is absolutely clear is that genes do not act in a vacuum; they act in the environment, and their actions can be altered by the environment.

BIOLOGICAL AND GENETIC DATA AS RELATED TO THE CONCEPT OF RACE

One would hope that, because the concept of race was originally, if falsely, conceived as a concept to signify the degree of biological differences between groups of people, the strongest support for the concept of race would originate from biological and genetic data. Does it? Here we review some examples of the relevant research. First, it appears that the global distribution of genetic variation in humans is not easily sorted by so-called races. As reviewed recently, scientists have studied diverse populations for many polymorphisms.¹⁰⁶ These studies involve polymorphisms in the nuclear DNA, including variation in the non-recombining Y chromosome and autosomal (i.e., located on chromosomes other than Y and X) markers as well as polymorphisms in the mitochondrial DNA.¹⁰⁷ A clear consensus picture has emerged of the distribution of genetic variation around the world, at least in broad strokes. These data overwhelmingly support the following model for recent human evolution and diversification of populations.

Modern *Homo sapiens* evolved once in Africa about two hundred thousand years ago and then spread throughout the rest of the world and simultaneously diversified starting about fifty to one hundred thousand years ago. During that spreading out, modern humans supplanted now-archaic humanlike populations identifiable as having spread outside of Africa, such as Neanderthals. The evidence is that effectively only one population left Africa and settled in southwest Asia. That population was characterized by a large founder effect before it expanded into other regions. From that population, different pathways of expansion occurred into Europe and separately across Asia. At some point in Asia, not yet clearly identifiable, additional expansions occurred, one expansion into northeast Asia and then into the Americas, plus a separate expansion into Melanesia and Australia. Associated with all of these expansions is accumulating random genetic drift at all polymorphic sites of the genome. Thus, allele frequencies generally show gradual changes as one moves

around the world. Of course, recent migrations (over the last few thousand years) of established populations into already-occupied regions can result in some adjacent populations having very different allele frequencies, but that has been rare until historic times. Today in the United States, for example, we have populations from very different parts of that geographically continuous spectrum of allele frequencies. Those distinct allele frequencies do not mean that different "races" exist, only that different parts of a continuum have been sampled. An analogy is the distinction between the colors blue, yellow, and red as samples from a continuous spectrum of light. Those colors only have meaning because the spectral sensitivities of the photoreceptors in our eyes and the neurological circuits interpreting the signals interact with a label arbitrarily imposed on some narrow range of wavelengths from a continuous spectrum.

There is no question that populations, defined geographically, demonstrate dramatic variability in frequencies, not only for the several million normal polymorphisms not associated with causing genetic disorders but also for many disease-related genetic alleles (variants). The genetic alleles (variants) can be readily seen in ALFRED, the Allele Frequency Database.¹⁰⁸ The issue is not whether this variation is present or not; the issue is whether explaining this variation should occur at the levels of populations per se (e.g., Lapps, Chuvash, Nyanja, or Corsicans), continents (e.g., Europe or Africa), or alleged races. After our review of the literature, we think that variation that seems to be meaningful and transferable into helpful public health or educational policies is at the level of specific populations. Global socially constructed categories such as race do not appear to be useful proxies for genetic features.

Second, considering evidence for a biological basis for racial classification, it is important to appreciate comparatively the amount of genetic variation observed within and among specific racial categories. In this context, let us turn for an illustration to the research on genetic bases of complex diseases. From rapidly accumulating evidence, it seems that a number of geneticists have stated that most common complex diseases, such as diabetes, hypertension, cancer, and so forth, appear to be at least partially governed by genetic mechanisms, shared by most, if not all, populations around the world.¹⁰⁹ This statement has triggered a number of large-scale studies such as projects in Iceland and Estonia, where population-wide genome banks have been created in the hope of identifying

specific alleles associated with common diseases within populations so major pathways of genetic disorders can be discovered and later generalized to other populations.¹¹⁰ Although the effectiveness of this approach is yet to be determined, this approach has been encouraged by new evidence indicating that many uninterrupted or rarely interrupted chunks of DNA (referred as haplotypes) appear to be common across different populations socially classified as belonging to different races.¹¹¹

To appreciate the significance of this finding, consider the example of population variability in mutations in the phenylalanine hydroxylase (*PAH*) gene—the gene whose disrupted protein results in the manifestation of phenylketonuria (see above). It has been established that multiple mutations in this gene result in the disorder. The mutations differ in terms of their specific location within the gene, and the frequencies of individual mutations vary across populations. However, each of these mutations appears to arise on one of a limited number of haplotypes and continues to be associated with that haplotype. Most common haplotypes are seen in all populations and the greatest number of haplotypes is seen in African populations.¹¹²

Third, the essence of the race-intelligence-genetics discussion has been an assumption that if race is somehow a surrogate for unknown genetic mechanisms, then observed racial differences in intelligence and achievement can be explained by genetic differences. But can they be? Although we have gained significant understanding of monogenetic (i.e., single-gene) conditions, there are still enormous blank spots in our understanding of complex human traits (i.e., traits controlled by many genes, often in combination with many environments), such as blood pressure, autism, reading disability, or intelligence. To illustrate, consider the observation that the majority of rare single-gene disorders (e.g., Tay Sachs, sickle-cell anemia, thalassaemia) are caused by mutations in a gene that result in the production of changed and therefore often faulty proteins. In the literature, these deleterious mutations are typically referred to as “coding single nucleotide polymorphisms” (cSNPs). Consider two facts about cSNPs. First, they are rare; second, they are of recent origin, presumably dating to the post-African diaspora.¹¹³ Both assertions have implications for the discussion here.

First, the rarity of cSNPs implies that they are unlikely candidates for controlling quantitative traits such as blood pressure, bone density, and

intelligence. The more likely candidates, due to their abundance, are so-called nontranscribed regulatory elements of the genome (i.e., a piece of DNA that does not contribute to the production of proteins, noncoding sequences). The amount of variation in these elements is remarkable. At this point, the significance of this variation, because it has no obvious impact on the proteins, is unclear. However, information from research in other than human organisms is of interest here. For example, in *Drosophila*, these noncoding alleles have been closely associated with quantitative traits.¹¹⁴ Second, the timing of the origin of cSNPs is linked to the observation that their frequency varies among populations.¹¹⁵ The reasoning is simple. Because cSNPs arose after the differentiation of the populations, their distribution is a consequence of ethnic differentiation, not a reason for it. It appears that common noncoding variants, some of which are assumed to contribute to or even to underlie susceptibility to common diseases and to variation in quantitative traits, are observed worldwide and can be referred to as "panethnic" alleles.¹¹⁶ In other words, to the best of our knowledge today, there are no explainable population differences in noncoding allele frequencies that can be meaningfully linked to variation in phenotypes. We simply do not see a clear pattern of ethnic differences in allele frequencies that can be associated with differences in specific phenotypes. Ethnic groups, of course, are socially defined. "Race" sounds like it is biologically defined. It is not. It, too, is socially defined.

SOCIAL VERSUS BIOLOGICAL DEFINITIONS OF RACE

When biological and behavioral markers of socially defined races are investigated, the studies primarily or even exclusively rely on participants' self-reporting of socially defined racial, ethnic, and cultural groups. Many studies use social labels such as Asian American or African American, Chinese, or Hispanic, implicitly ignoring the fact that these labels generalize across substantial amounts of cultural, linguistic, and biological diversity.¹¹⁷ For example, "Hispanic" includes diverse populations from areas such as Cuba, Puerto Rico, the Dominican Republic, Guatemala, Costa Rica, Argentina, and, of course, Spain. The ancestry of individuals in these groups varies from entirely African, entirely Native American, and entirely European to any possible mixture of these three. Even ignoring

the substantial variation within each of these large regions, there is no basis, except for certain social-cultural traits, for grouping these individuals. Even when a more specific populational reference such as Yoruba (i.e., a West-African population of over ten million people who are dispersed throughout different countries in Western Africa) is made, this reference subsumes a great amount of intra-Yoruba variability.¹¹⁸ Moreover, self-naming of social labels might change, depending on past and present social surroundings of the surveyed participants. For example, during the Soviet era, many immigrating Soviet Jews referred to themselves as Jewish by ethnicity, but upon their arrival to Israel or the United States they referred to themselves as Russians.¹¹⁹ In the United States, indeed, Judaism is not viewed as an ethnicity, but as a religion. Similarly, individuals who met the classifications of "colored" established by the apartheid government of South Africa would have, probably, self-identified themselves as black in the United States.¹²⁰ Thus, because most medical and psychological research on racial differences is based on self-defined racial or ethnic categories and there is substantial evidence questioning the accuracy of these self-classifications, the validity of racial and ethnic differences as commonly investigated is questionable.

People will probably always label themselves and others, regardless of what scientists find. The problem is not the use of social labeling per se, but rather the confusion of it with biological labeling. And it is especially problematical when scientists contribute to this confusion by using social labels in a way that suggests they are somehow biological.

The important message here is that the division lines between racial and ethnic groups "are highly fluid and that most genetic variation exists *within* all social groups—not *between* them."¹²¹ Studies based on hundreds of genetic polymorphisms confirm earlier studies such as that by Lewontin cited above¹²² and show that only 11 to 23 percent of observed genetic variation is due to differences among populations and that is mostly attributable to differences in allele frequencies, not all-or-nothing genetic differences.¹²³ In fact, most common genetic variants exist in almost all populations. The overwhelming majority of the variation occurs among individuals with different genotypes within each population. One study found even less variation among populations, but highly polymorphic multiallelic markers were studied and they may have been biased toward high heterozygosity (i.e., the two chromosomes of an individual

having different alleles) in many different populations, thereby minimizing the between-population variation.¹²⁴ Clearly, when common polymorphisms are studied, there is only a minority of the genetic variation that occurs among populations. Variants that are restricted to only a few populations in one part of the world are almost never common even in those populations.

Finally, let us regard if and how the concept of race matters in such areas of life as public health and education. Let us consider examples from public health (the data are from the US National Center for Health Statistics, 1998). When age-adjusted death rates of occurrence per 100,000 individuals are reviewed, the rates for white, black, Hispanic, and Asian are as follows: heart disease—121.9, 183.3, 84.2, and 67.4, respectively; cancer—121.0, 161.2, 76.1, and 74.8, respectively; liver disease/cirrhosis—7.1, 8.0, 11.7, and 2.4, respectively; and diabetes mellitus—12.0, 28.8, 18.4, and 8.7, respectively. Three points are important to mention here. First, there are clearly some group differences in these data. However, these differences are inconsistent: for example the incidence of heart disease was the highest among blacks, but the incidence of liver disease was the highest among people of Hispanic origin. Second, all of these conditions are considered to be in part genetic disorders because of the overwhelming amount of data in the field attesting to the importance of the genetic factors in the development and manifestation of these diseases. Third, all these diseases are considered to be complex; therefore, the genetic mechanisms of these conditions have not yet been decoded. Thus, we cannot argue that these observed differences in rates are genetic because we do not know what the genetic mechanisms are.¹²⁵

Similarly, there are some group-average differences in scores on tests of academic abilities and achievement among children socially labeled as white, black, Hispanic, and Asian. How large the differences are, and what groups they favor, depend on what, in particular, is tested. For example, Sternberg and the Rainbow Collaborators found that analytical tests of the kind traditionally used to measure so-called general abilities tend rather strongly to favor Americans of European and Asian origin, but tests of creative and practical thinking show quite different patterns.¹²⁶ We also know that there is a substantial genetic influence contributing to individual differences in the level of academic achievement.¹²⁷ Yet, we do not know a single gene that has been identified as contributing to either

academic achievement or IQ. So, the statement that racial differences in IQ or in academic achievement are of genetic origin is, when all is said and done, a leap of imagination. The literature on intelligence, race, and genetics constitutes, in large part, leaps of imagination to justify, post hoc, social stratifications. There is nothing wrong, in principle, with people expressing their views on social policy. But they need to recognize these views for what they are—social policy pronouncements, not science.

CONCLUSION

In conclusion, the meaning of intelligence is, at this time, ill-defined. Although many investigators study “IQ” or *g* as operational definitions of intelligence, these operationalizations are, at best, incomplete, even according to those who accept the constructs as useful.¹²⁸ Research suggests that properties of intelligence beyond *g* may be somewhat different from those of *g*.¹²⁹ Race is a social construction, not a biological construct. And studies currently indicating alleged genetic bases of racial differences in intelligence fail to make their point even for these socially defined groups. In general, we need to be careful, in psychological research, to distinguish our folk conceptions of constructs from the constructs themselves.

NOTES

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