RACE, GENES AND INTELLIGENCE

Pilar N. Ossorio, Ph.D., JD

Over the past two centuries biomedical science has, at times, provided justification for white privilege. Science has been used to support the proposition that differences in achievement reflect innate differences in ability among racial groups. Broadly speaking, the view that differences in academic achievement, IQ scores, employment status or wealth primarily reflect innate differences is called "biological determinism." [1] As the late Stephen J. Gould pointed out, at its core, biological determinism is "a theory of limits. It takes the current status of groups as a measure of where they should and must be (even while it allows some rare individuals to rise as a consequence of their fortunate biology)." [2, p. 28]

Biological determinism lost most of its scientific credibility by the mid-20th century, and lost much of its social and political power after World War II; however, it never entirely disappeared. Today, some people believe that persistent racial gaps in, for instance, school achievement, family income, and wealth must reflect innate differences in ability. One human trait that is postulated to play a role in many kinds of achievement is intelligence, and some commentators postulate that racial differences in average levels of intelligence explain achievement gaps.

At the same time, the new molecular genetics has captured the public imagination and has provided tools for conducting large-scale genetic comparisons between individuals and between human groups. Some people will look to modern genetics to provide scientific justifications for racial inequalities. Genetics is particularly appealing in this role because of its apparent precision, authority, and high-tech chic. Many people reason that if groups vary with respect to innate cognitive abilities, then the differences between groups must be attributable to differing racial patterns of genetic variation. To disentangle claims about race, genetics and intelligence, we must examine beliefs about race and intelligence, and understand what role genes reasonably could or could not play with regard to the intersection of these two concepts.

RACE AND GENETICS

Race is a concept that people use to make sense of the amazing panoply of human biological, social, cultural, and political variation. Since the 17th century, scientists have attempted to categorize and differentiate human populations. Perhaps the single most influential taxonomy of human races was that constructed by the Swedish naturalist Carolus Linnaeus, in his famous *Systema naturae*. The 1758 edition of this work initiated the modern science of systematically categorizing all living organisms. Linnaeus included humans in the *Systema* as an ordinary component of the natural world, subject to categorization as is any other organism. He identified four geographically-based human races, which he designated as subspecies. These four groupings are described in contemporary terms as Native Americans, Europeans, Asians and Africans.

Linnaeus's taxonomy described both the physical appearance and personality type that he believed characterized each race. *Homo sapiens americanus* (Native Americans) were red in color, had erect posture, straight and thick black hair, wide nostrils, scanty beards and "harsh" faces. They were, purportedly, liberty-loving, ill-tempered, and obstinate, and their social and political relations were governed by custom rather than formal law. *Homo sapiens europeaeus* (Europeans) had white skin, long flowing hair, and blue eyes. Their character was serious, sanguine, and smart, and their social and political relations were governed by formal law. *Homo sapiens asiaticus* (Asians) had yellow skin, black hair and dark eyes. According to Linnaeus, they were melancholy, greedy and governed by "opinion" rather than formal law. *Homo sapiens afer* (Africans) were black, with "frizzled" black hair, silky skin, flat noses and tumid lips. He described their character as crafty, impassive, lazy, careless and "ruled by caprice." [3]

It should be obvious that Linnaeus was not simply cataloging physical features or biological structures, he was also using non-biological traits (real or imagined), such as dress and specific forms of social organization, to construct his categories. He commingled socio-cultural traits and biological ones as though all had the same cause. Linnaeus may have believed that he was simply performing "objective science," analyzing human beings the same way that he would analyze snakes or ant eaters, but he was not. It should also be obvious that Linnaeus was constructing a social hierarchy in which whites (the group to which he belonged) were at the pinnacle and blacks were at the bottom. White's possessed the qualities he viewed as most admirable and valuable, blacks possessed the least of those qualities, and other races were somewhere in between.

Linnaeus's four geographic categories influenced nineteenth and early twentieth century anthropologists, and they are still evident in contemporary, "folk notions" of race. The link between continent of ancestry, a person's appearance, and her race is still central in most people's belief systems.

From Linneaus's day until the 20th century, the overarching goal of the study of human variation was to establish a small number of basic categories into which all human variation could be fit. Anthropologists collected large quantities of data—body measurements of people from around the world—while attempting to discover discrete, natural categories of people. But the more people the anthropologists measured, the more categories they found.

By the early 20th century, many scientists were beginning to believe that nature had not produced discrete, biological categories of humans. Scientists had difficulty allocating the world's people into categories based on biological measurements, in part because the different physical traits that scientists measured—pigmentation, hair texture, facial features, leg lengths, etc.—do not vary in concert with each other. Consider two features that many people use as bases for racial categorization—skin color and nose shape. Dark skin color and wide nose shape are features associated with people from sub-Saharan Africa, and with a race designated as black. Light skin color and narrow, longer noses are features associated with people from Europe, a race variously designated as white, Caucasian, or Anglo. But some individuals have dark skin color and narrow,

long noses. This is true of many people from the middle east and northern Africa. Skin color and nose shape are not always correlated, they can vary independently of each other, and so using these two features can lead to more than two ways of categorizing people.

Eventually, the accumulated anthropological data showed that patterns of human biological variation are complex, and that biological variation between human groups is generally a matter of gradual change rather than abrupt discontinuities. On average, people tend to be more physically similar to others who are born and live near them, and one group gradually gives way to another. Charles Darwin argued that the weightiest of all arguments against the notion of distinct human races was that "they graduate into each other… [the naturalist] of a cautious disposition, . . . will say to himself that he has no right to give names to objects he cannot define." [4] Because human biological variation is complex and continuous, allocating people to categories requires us to "draw lines" where none exist in nature.

Differences in features, and as it turns out, in DNA sequences, are greatest between groups of people who are geographically distant from each other. The pattern in which some measurable feature varies gradually, and the variation correlates with geographic distance, is called "clinal" variation. [5] Many human traits and many human genetic differences exhibit a clinal pattern. [6]

Modern molecular genetics has largely confirmed what anthropology discovered in the early twentieth century. As a general rule, all people possess the same genes, in the same order, on their chromosomes. Genomes (the complete complement of DNA in a cell) of people from all points on the globe are remarkably similar. This is why it was possible and reasonable to undertake the Human Genome Project (HGP) to create a composite reference DNA sequence that was pieced together from bits of several people's genomes. [7-9]

Although humans are quite genetically similar, no two people are genetically identical unless they are monozygotic (identical) twins. There are various methods of conducting genetic comparisons between individuals or groups, and the different methods do not always give exactly the same results, so claims about genetic similarity and difference should be made cautiously, and numerical claims should be viewed as a estimates or approximations. Given this caveat, it is widely accepted among contemporary geneticists that any two unrelated humans are about 99.8 percent or 99.9 percent genetically identical. [10-12] But because the human genome contains approximately 3 billion nucleotides (DNA building blocks), a 0.1 percent or 0.2 percent difference translates into millions of sites at which two people will have a different nucleotide. Some genetic variation involves differences in the number of copies of a particular DNA sequence a person possesses (copy number variation). Copy number variation has been linked to human differences in drug response, resistance to HIV infection, risk of developing autism or schizophrenia, and other human traits. [13-16]

Nearly all of the genetic variation among humans is found within any human group. If one assesses the genetic variation in a group of Yoruba people from Nigeria, and in a group of Swedish people from the town of Malmo, somewhere between 85 percent and 95 percent of the genetic variants will be found in both groups, although some variants will be found at a higher frequency in one group than the other. [17] Furthermore, if one examines a single gene or region of the genome, then an individual whose recent ancestors are from Lagos, Nigeria, may be more similar to somebody from Malmo, Sweden, than to most other people from Nigeria. [12] Depending on how one measures, the component of genetic variation that occurs between human groups from different continents could be as low as 2.8 percent, whereas the component of genetic variation between human groups from the same continent could be 2.5 percent. [18] If one associates races with particular continents, then all but 2.8 percent of the human genetic variation is found within any race.

When the frequency of a gene variant does differ between one human group and another, one typically sees a pattern where, for instance, gene variant A is found in 15 percent of people from Group 1 and found in 23 percent of people from Group 2. Typically, the difference in frequency is relatively small and most people will have the same variant, regardless of the group to which they belong. There are, however, a few gene variants that differ quite significantly from one human group to another.

The gene with highest frequency of between-group variation is one that influences skin pigmentation in humans and other organisms—the SLC24A5 gene. [19] One version of this gene has been found in 98 to 100 percent of people in European population samples studied, but a different version of this gene has been found in 93 to 100 percent of people sampled from parts of Africa, East Asia and the Americas. Interestingly, over half of the African-Americans studied have either one or two copies of the gene variant commonly found in European populations. [19,20]

To the extent that genes underlie pigmentation, perhaps the most visible of human differences, we should expect that some of those genes will have quite different frequencies among people of different skin color. What may be surprising is that pigmentation is genetically very complex. At least five different genes strongly influence skin pigmentation, and there are hundreds of other genes that may play a minor or occasional role. [19] Because of this complexity, scientists cannot use variants of pigmentation genes as a means of racial classification. There are no pigmentation gene variants that are found only and always in “white people” but not in people of other racial groups.

Human beings are quite genetically similar because humans are a relatively young species, one that has not had much time to differentiate (as measured by number of generations). [12,21] No human group has been reproductively isolated from others for long enough to become a different species or subspecies.

The greatest amount of genetic variation between human individuals is found among people of Africa. For many regions of the human genome, there are more variants

found among people of Africa (and the recent African diaspora) than are found among people in the rest of the world. This is probably because humans have resided in Africa for much longer than we have resided any place else in the world, so our species has had time to accumulate genetic changes within the people in Africa. A relatively small group of people migrated out of Africa 30,000 to 100,000 years ago, and only a fraction of the human genetic variation went with them. [5,11,22] After migrating out of Africa, humans underwent a very rapid expansion to all parts of the globe. One implication of the high degree of genetic diversity among people of Africa is that it is incorrect and incoherent to think of black people as a genetically unitary group.

Scientists are intensely interested in using the 5 to 15 percent of genetic variation that occurs between populations to study the history of the human species. Understanding this variation also helps them to conduct studies on genetic causes of human disease. Recently, geneticists have begun measuring hundreds of thousands of sites of genetic variation in each of thousands of people's genomes, then statistically grouping people according to their overall patterns of genetic similarity and difference. Such analyses can group together people whose ancestors all came from the same continent, and differentiate them from people whose ancestors came from a different continent. [11, Jorde, 2004 #15,23] Some commentators have equated these statistical groupings with human races.

But the ability to statistically cluster members of the human species is not the same thing as "finding" a natural, biological category. [24] Using the same scientific approach, scientists can also statistically group people whose grandparents were born in Finland and separate them from people whose grandparents were born in Sweden. [25] Researchers can group people of Iceland according to the county or counties in which their ancestors (five generations back) were born. [26] This does not mean that the people of Finland and Sweden are of different races, or that Iceland's counties are populated by different races. Also, keep in mind that only about 0.1-0.2 percent of the human genome differs from one person to another, and only 5-15 percent of that variation can be used to distinguish between human groups, so statistical grouping of people by patterns of genetic variation is based on a minute fraction of our genomes.

Finally, it is crucial to reemphasize that the amount of genetic variation between groups is very small compared to the 85 to 95 percent of variation found within human groups. That one can genetically group together people whose four grandparents all came from Beijing, China, and differentiate them from people whose four grandparents all came from someplace else does not mean that the people whose grandparents came from Beijing are substantially genetically alike. People in any particular place are not genetically homogenous, and people certainly are not homogenous across entire continents. The vast majority of human genetic variation is between individuals, including individuals who can be assigned to the same racial, ethnic, or national group.

Because humans have high within-group genetic variation, genes are unlikely to explain average differences in IQ test scores of different racial groups. We do not know the extent to which genes underlie a person's ability to perform complex mental tasks,

but there is no reason to think that people whose relatively recent ancestors all came from one continent would have different variants of any relevant genes than do people whose ancestors came from another continent. If potential "cognition genes" are similar to other genes, then most variants will be found within all groups of people at similar frequencies. [24]

WHAT IS RACE?

> "Order is Heaven's first law: and, this confessed,
> Some are, and must be, greater than the rest…
> Without this just gradation, could they be
> Subjected, these to those or all to thee?"
> Alexander Pope, *Essay on Man* (1733) [2, p. 31]

If races are not distinct genetic groups of human beings, then what are they? Some scholars have argued that because humans are so genetically similar, and because there are no discrete genetic groups, human races do not exist or are not "biologically relevant." [27,28] However, the issue of whether something exists, and whether it is "biologically relevant," is completely separable from the question of whether it can be found in our genes.

Many non-genetic features of our world and ourselves are real because we make them so, we bring them into existence through beliefs, customs, laws, physical arrangements of our environment, and numerous every-day acts. Marriages, schools, and subways are not encoded in any person's genes, but they are all real. Likewise, race is real because people believe in it and act on those beliefs. Race is deeply rooted in the consciousness of individuals and groups, and it structures our lives and our physical world in myriad ways. It is a strong predictor of where people live, [29,30] what schools they attend, [31] where and how their spirituality is practiced, what jobs they have, and the amount of income they will earn. [32] Race is real because human beings continually create and recreate it through the process of racialization. [33]

There is no unitary definition of race, no definition that applies in all places, at all times and for all purposes. Scholars who include race as a variable in their studies must operationalize the concept of race in a manner that meets the needs of their study, while acknowledging that such "working definitions" merely "fulfill the need for an analytical strategy, they do not reflect a fixed social or biological reality." [34, p. 4]

Scholars who study race, from numerous fields, generally agree on a few themes regarding how notions of race operate in society. One point of agreement is that race is a second order construct—a belief about beliefs, behaviors, and traits. A person's racial self-identification, and her ascription of race to other people, is based on her beliefs about skin color, head shape, hair texture, religion, ancestry, language spoken, nationality, dress, political philosophy and many other factors. Genes encode some of the traits on which people base racial attributions and identity, but genes do not encode all of them.

Because a person's race is not reducible to her genetically encoded traits, one could know everything about a person's genome and still not know her race. [35]

Another point of agreement among race scholars is that race is a very malleable concept. This claim developed from numerous observations of how the meaning of racial categories, and the categories themselves, change over time. [33,36,37] One well known example of this malleability is that every decennial census since the early twentieth century has defined race differently than the previous census. [38,39] Thus, a person who was white in one census might have been Mexican or black in another. Another example is that some people have a different race on their birth certificate and their death certificate.

The malleability of the concept of race means that one must be very careful in comparing different studies in which race is correlated with some other feature, such as income or IQ. Studies done at different times, or by different researchers, may not be measuring the same people or the same social reality when they refer to, for instance, Native American or white people. Furthermore, because race plays such a powerful role in shaping our lives it is correlated with many factors that likely influence cognitive ability. These correlated factors may confound the results of studies by making it appear that race explains or "causes" difference in IQ test results when some other racially-correlated factor, such as diet, parental income, neighborhood environment, etc., actually explains the difference.

Another point of agreement among race scholars is that race involves practices that create hierarchies of social privilege and deprivation. While individuals may move from one racial group to another, the practices and social institutions that create racial hierarchy are long standing and deeply entrenched. Some group is always at the pinnacle of privilege, and in the United States this has been white people. This feature of race has not changed since Linneaus's and Alexander Pope's time. The concept of race emerged in Western culture concomitant with European colonialism, it was one ideology that could be used to justify conquest. [40] In the Americas, the asserted racial superiority of white or European-descended people functioned to justify the enslavement of African-descended people, the near extermination of Native Americans, and the extremely oppressive labor policies directed towards Chinese and Japanese workers. Contemporary racial hierarchies are enforced by less explicit means, but they still operate to rank people on scales of social status and privilege.

In the contemporary world, beliefs about racial difference, and racial superiority or inferiority, may be articulated in the language of molecular genetics and genomics. Modern genetics has great authority, and beliefs about race that once relied on vague notions of innate difference can be made to sound more precise and credible by framing them as genetic explanations. Educational achievement, wealth, and other measures of status often run in families, a fact that may increase the intuitive credibility of genetic explanations. Societal institutions operate to entrench groups who wield power into self-perpetuating dynasties. From the Tudor monarchical dynasty in 16th century England to the Bush and Kennedy family dynasties in the 20th and 21st centuries in America, families

with access to power pass their positions of privilege on to succeeding generations in a process resembling the hereditary transmission of genetic traits. Furthermore, genes can be viewed as the substrate by which God or natural selection rendered some groups superior and others inferior.

RACE AND INTELLIGENCE

Just as there is no unitary definition of race, there is no agreed upon or single definition of intelligence. One aphorism holds that intelligence is what intelligence tests measure. Psychometricians argue that intelligence tests measure reasoning skills, although the tests also measure knowledge. [41] Some innovative scholars have developed theories of emotional intelligence and multiple intelligences—multiple types of cognitive function that are valuable and measurable, and that may manifest differently in different contexts. [24,42-44] The typical IQ test does not measure multiple intelligences; instead, the test produces a single intelligence quotient (IQ).

Some scholars argue that one's IQ indicates one's general cognitive ability, or *g*. [1,2] Many other scholars argue that the notion of a single, general quality that underlies performance on all cognitive tests is incoherent. [24,42-44] Stephen Jay Gould has provided a thorough explanation and critique of the concept of *g* in *The Mismeasure of Man*. [2] *g* has been a useful concept for commentators who seek to create social hierarchies based on intelligence, because "…ranking requires a criterion for assigning all individuals to their proper status in a single series." [2, p. 24]

Alfred Binet, the developer of the first intelligence test in the early 20th century, rejected the notion that his test measured a person's inborn or fixed cognitive ability. He also declined to use his test to rank individuals according to cognitive ability. The purpose for which he devised the test, and the only purpose for which he thought it appropriate, was to measure the intellectual capacity of children who were performing poorly in school, to determine which children had cognitive deficits for which remedial instruction might be helpful. Later psychologists, particularly those in the United States, took up and modified Binet's test, and were willing to embrace the view that intelligence was an inborn and fixed attribute of a person. We can call this view the hereditarian theory of IQ.

Over the past decade, some contemporary proponents of the hereditarian theory have argued that 1) IQ is the most important determinant of academic success; 2) academic success is the most important determinant of high status and wealth-generating employment; and therefore 3) the economic elite have their positions and wealth as a matter of merit (intellectual contribution to society), and conversely, members of the economic underclass also deserve their position at the bottom of the social hierarchy. [45-47] These commentators argue that programs aimed at raising the academic achievement of disadvantaged students are misguided because those students are, on average, biologically incapable of significant academic success. Racial gaps in test scores, from IQ to the SAT, are interpreted by hereditarians as evidence of inherent and immutable racial or ethnic differences in underlying cognitive capacity.

Many claims of contemporary hereditarians have been critiqued and debunked in books such as *Measured Lies*, [48] *Inequality by Design*, [41] *Whitewashing Race*, [49] and *Intelligence and How to Get It.* [50] These books describe mistakes of fact, method and logic made by the hereditarians.

A significant problem in debates about hereditarian theories of IQ is that correlations are often treated as proof of causation. If one observes that people in lower socioeconomic brackets, on average, score lower on IQ tests than people in higher socioeconomic brackets, this does not mean that low IQ causes poverty. It could be that poverty causes low IQ, [50] or that something else causes both outcomes. If IQ test scores correlate with race (however race is defined), this does not mean that some inborn racial essence causes particular IQ test scores. One reasonable alternative explanation is that race is correlated with other factors, such as quality of schools, exposure to lead, or malnutrition, and these other factors are causing the observed differences in test scores.

Hereditarian claims are based on the alleged heritability of IQ. Heritability assess the way a trait *varies in a population*, and purports to measure how much of that variation is explained by genetic differences *within* the population. The remaining variation is attributed to all other factors (the environment and non-genetic aspects of biology). If children in a classroom score between 90 and 130 on an IQ test, a hereditarian might claim that 65 percent of the 40 point difference in IQ is due to genetic differences between the students, and 35 percent is due to other factors. Strong proponents of hereditarian theories tend to believe that genetic differences explain as much as 85 percent of the variation in adult IQ in a population, but other scholars believe that genes explain much less than 50 percent of the variation in IQ.

Heritability is a population measure and it is only valid within the group in which the trait of interest was studied. Thus, if one measured IQ scores in a group of middle class white children and a group of middle class Native American children, and found that the average IQ score for the Native American children was 3 points higher than that for the white children, one could specify a heritability score for each group, *but one could not say how much of the difference between the white and Native American children's mean score was attributable to genes*.

To illustrate why heritability within groups has nothing to do with heritability between groups, consider a hypothetical example in which researchers measure the height of men sampled randomly across the state of Wisconsin and men sampled randomly across the island of Fiji. Researchers might determine that the differences in height in Wisconsin men were primarily due to genes, and that the same was true in Fiji. However, men in Wisconsin were an average of two inches taller than the men in Fiji, researchers could not state that the two inch between-group difference was caused by genetics. Different diets might explain most or all of the between-group difference. This example should illustrate why, *by itself, a difference in the mean IQ scores between two groups of people can never justify the inference that some or all of that difference is caused by genetics*. Even scholars with a strong hereditarian bent acknowledge this point.

Many scholars question the entire enterprise of treating heritability statistics as though genes and environment are actually separable influences on IQ or any other trait. Genes always function within particular environments to shape the developing human organism. The developmental interaction among many genes, and numerous environmental factors, is complex, varies over time, and is susceptible to chance events.

Researchers have, in fact, found evidence that some environmental factors are strongly associated with IQ and other measures of cognition. Malnutrition [51] and exposure to environmental toxins, such as lead from paint, [52] are strongly correlated with IQ. The quality of a person's school significantly impacts her IQ score—children who begin their education in poor quality schools and then move to better ones show increases in their IQ scores. [41,53]

A study published in 2009 found that long-term stress is negatively associated with young adults' performance on cognitive tests. [54] This study measured levels of several physiological properties associated with stress, including blood pressure, cortisol, and epinephrine levels. The researchers collected data throughout their participants' childhood years, then administered tests of cognitive performance when the children turned 17 years old. Young adults whose bodies exhibited the highest levels of chronic stress had the least effective working memories and poorer cognitive performance.

Research also undermines the hereditarian claim that IQ is the primary determinant of achievement. Many environmental variables predict achievement as well or better than IQ, [31,41,55] except for people whose IQ scores are at the abnormally low end of the scale. For instance, a person's social environment may be an important determinant of her achievement, yet variables that capture a person's social environment are often, literally, left out of the equation in work done by hereditarians. The social environment includes the expectations of one's peers, encouragement by one's parents and teachers, enrichment opportunities available in the neighborhood, etc. A decades long study that included social environment variables found that a 15 point difference in IQ scores among high school boys only explained 6 percent of the variability in their earnings at age 35. The greater the number of social factors taken into account, the less important IQ became. [56] Social context variables were still significantly correlated with earnings by age 55.

In a related analysis, Fisher *et al*. demonstrated that if all adults in the country had the same score on an IQ test the variation in household income would only decrease by about 10%. Contrary to hereditarian claims, these data suggest that differences in IQ do not explain much about professional achievement and wage inequality, including wage inequality between racial groups. [41] On the other hand, factors external to an individual can greatly influence her or his lifelong course of achievement.

Because race comprehensively structures people's lives in the United States, it is correlated with many environmental factors that can influence IQ and achievement. People of different races tend to live in different neighborhoods, so they may be exposed to different levels of lead, different quality schools, different diets and different levels or

types of stress. They may be exposed to different attitudes about achievement. People of minority groups may routinely experience racism, a kind of stress that can have long-term physiological consequences. On average, people of different races receive health care at different institutions, and the care they receive is not of the same quality. In sum, racial groups differ with respect to so many environmental factors that it is entirely plausible that environmental differences explain current racial gaps in mean IQ scores.

The environment can be modified in ways that genes cannot. When the environment is changed, the trait of interest (in this case intelligence) may also change *even though genes also play a role in shaping that trait*. For instance, in one study African-American children in Milwaukee who were thought to be at risk for cognitive disability were randomized so that half received intensive day care and early, enriched education, while the other half received ordinary day care and schooling. [50] By age five, children who received the intensive intervention averaged 110 on a standard IQ test (above average), while children in the control group averaged 83 (well below average). The effects of early, intensive education were still apparent by adolescence, when the children from the intervention group scored, on average, 10 points higher on IQ tests than the children from the control group.

There is some evidence that differing environments have influenced the entire human population's IQ scores over time. People's average IQ scores have risen by about 3 IQ points per decade over the last century. [50,57] The average IQ score from 1917 would amount to about 73 on today's tests. This effect almost certainly is not due to changes in human genetics, because there has not been enough time for new intelligence-related mutations to arise and spread throughout human populations. The most likely explanation for the rise in IQ is that some relevant environmental factors have changed, causing people to develop in ways that are reflected in higher average IQ scores.

Another piece of evidence concerning widespread environmental influences on IQ is that the mean difference between black Americans' and white Americans' test scores has narrowed since the 1970s. Using data from several different IQ tests that were administered in a standard manner to black and non-Hispanic white people, Dickens and Flynn showed that blacks have narrowed the IQ gap by one third to one half of what it was in the 1970s. [58] If IQ were a fixed, intrinsic quality of races, then the IQ gap should be stable over time, but it is not.

GENES, BRAINS AND INTELLIGENCE

It is quite difficult to study the intersection of genes, brains and cognition. Presently, scientists have only vague and preliminary ideas about how brain structures correlate with thought processes (including solving problems on intelligence tests), and scientists are only beginning to study the ways in which genes influence the development of brain structures. One can imagine various aspects of brain physiology that might influence thinking. These include the speed and efficiency of communication between brain cells or structures, the quantity of cells that can be mobilized to store a memory or solve a problem, the density of receptors for neurotransmitters, etc. To date, none of

these aspects of physiology has been demonstrated to play a role in normal human problem solving or abstract thinking ability, but it seems reasonable that many aspects of brain physiology could play a role. Hundreds or thousands of genes, operating in particular environmental contexts, encode information that is important for the development and maintenance of brain structures and physiological processes.

Some researchers have attempted to correlate genetic markers with people's scores on tests of cognitive ability. Thus far, these studies have yielded some claims about gene variants that are correlated with variation in cognitive ability, but no research has demonstrated a *causal* connection between a particular gene variant and a particular degree of cognitive skill (within the normal ability ranges). Even proponents of such research note that, "there are an unknown number of genetic influences on different abilities; [and] some… proportion of these may be too small to feasibly be detected." [59] When variants of particular genes have been associated with IQ, the effects reported are relatively small—in the range of a couple of IQ points—and few of the observed correlations have been replicated. In contrast, altering environmental factors, such as the quality of education and day care, has been associated with IQ variation of 10 points or more. [50]

In 2005, a group of scientists created a firestorm of controversy when they reported evidence for recent natural selection in humans of variants in two genes involved in regulating brain size. [60,61] These scientists reported that a variant of the MCPH1 gene arose approximately 37,000 years ago and rapidly became prevalent in all populations tested except those in Africa. [60] Their companion paper reported that a variant of the ASPM gene arose much more recently, approximately 5000 – 10,000 years ago, and became prevalent in Europe and the Middle East. [61] The authors noted that the spread of the ASPM variant occurred at around the time when humans domesticated crops and livestock, when urbanization occurred and written languages developed. The authors all but invited readers to infer that a particular gene variant, which had been selected for in white people but not in other racial groups, was importantly involved in the development of civilization.

There was a flurry of media discussion and controversy surrounding the "brain gene" papers. The claim that gene variants for head size had been positively selected for in people with recent ancestors from certain continents but not others was particularly notable because of the longstanding, but highly contested, claim that head or brain size is correlated with IQ. Attempts to correlate head size, IQ and race or gender go back to the 19th century, during which time European male scientists asserted that women and non-whites (particularly people from Africa and the Americas) were intellectually inferior to European men, and that this inferiority could be "objectively" demonstrated by measuring head sizes. [2,3] The science of earlier centuries has been refuted, but many people saw the 2005 papers on MCH1 and ASPM as reintroducing those old arguments dressed up in the garb of modern molecular science.

Since 2005, other researchers have evaluated the same data on MCH1 and ASPM, plus some additional data, and concluded that there is no evidence that these genes have

been under natural selection in modern humans. [62,63] These re-analyses undercut the idea that the particular variants found at high frequency among people of European descent somehow made those people better adapted for modern civilization. Additional studies have discovered that the MCH1 and ASPM variants reported in the 2005 papers do not correlate with larger (or smaller) than average head size. [64] The genes were originally described as having to do with head size because some variants of these genes can cause microcephaly (extremely small heads that lack major portions of the brain). However, those variants were not included in the studies that were published in 2005.

Finally, several research groups have tried *and failed* to show any correlation between the variants described in the 2005 papers and IQ, reading abilities or verbal abilities. [65,66] One article stated, "The results strongly did not support the hypothesis that [variants] in MCPH-related genes are related to the evolution of human language or cognition." [66, p. 689] Unfortunately, none of these follow up studies received media attention.

CONCLUSION

The binary formulation of "genes vs environment" is misleading. Cognitive abilities are complex and will likely be influenced by a myriad of environmental factors *and* genes. Given the complexity of brains and cognition, one ought not expect that a few genes will play a dominant role in shaping the normal range of human cognitive abilities; numerous genes will be involved. It is statistically implausible that variants of numerous genes relating to intelligence would be distributed among racial groups in a manner that systematically conferred cognitive advantage on one group or disadvantage on another. Furthermore, there is no evidence to support the claim that current racial differences in mean IQ scores are caused by racially distinctive patterns of genetic variation.

There is evidence that IQ scores are influenced by environmental factors that are pervasively and systematically patterned along racial lines in the U.S. Nonetheless, mean IQ differences among racial groups have been decreasing over the past few decades, perhaps in response to improved educational opportunities for some minority individuals. Taken together, the evidence suggests that differences in IQ scores are the *result* of social inequality rather than its cause.

References

1. Anonymous. Intelligence and Genetic Determinism. *GeneWatch* **19**: 9 - 12 (2006).
2. Gould, S.J. *The Mismeasure of Man* (W.W. Norton & Company, New York, 1981).
3. Marks, J. *Human Biodiversity: Genes, Race, and History* (Aldine De Gruyter, New York, 1995).

4. Darwin, C. *The Descent of Man, and Selection in Relation to Sex* (Princeton University Press, Princeton, 1981) p. 698 (original work published in 1871).
5. Handley, L.J.L., Manica, A., Goudet, J. & Balloux, F. Going the distance: human population genetics in a clinal world. *Trends in Genetics* **23**: 432 - 439 (2008).
6. Serre, D. & Paabo, S. Evidence For Gradients of Human Genetic Diversity Within and Among Continents. *Genome research* **14**: 1679-1685 (2004).
7. International Human Genome Sequencing Consortium. Finishing the euchromatic sequence of the human genome. *Nature* **431**: 931 - 945 (2003).
8. International Human Genome Sequencing Consortium. Initial sequencing and analysis of the human genome. *Nature* **409**, 860 - 921 (2001).
9. Venter, J.C. et al. The Sequence of the Human Genome. *Science* **291**: 1304 - 1351 (2001).
10. Chakravarti, A. ...To a future of genetic medicine. *Nature* **409**: 822 - 823 (2001).
11. Tishkoff, S.A. & Kidd, K.K. Implications of biogeography of human populations for 'race' and medicine. *Nature Genetics* **36**: S21 - S27 (2004).
12. Jorde, L.B. & Wooding, S.P. Genetic Variation, Classification and 'Race'. *Nature Genetics Supplement* **36**: S28-33 (2004).
13. Buchanan, J.A. & Scherer, S.W. Contemplating effects of genomic structural variation. *Genetic Medicine* **10**: 639 - 647 (2008).
14. Cook, E.H. & Scherer, S.W. Copy-number variations associated with neuropsychiatric conditions. *Nature* **455**: 919 - 923 (2008).
15. Johansson, I. & Singelman-Sundberg, M. CNVs of human genes and their implications in phamarcogenetics. *Cyogenetic and Genome Research* **123**: 195 - 204 (2008).
16. Speleman, F., Kumps, C., Buysse, K., Poppe, B. & De Preter, K. Copy number alterations and copy number variation in cancer: close encounters of the bad kind. *Cytogenetic and Genome Research* **123**: 176 - 182 (2008).
17. Cavalli-Sforza, L.L. & Feldman, M.W. The Application of Molecular Genetic Approaches to the Study of Human Evolution. *Nature Genetics Supplement* **33**: 266-275 (2003).
18. Rosenberg, N.A. et al. Response to comment on "Genetic structure of human populations". *Science* **300**: 1877 (2003).
19. Lamason, R.L. et al. SLC24A5, a Putative Cation Exchanger, Affects Pigmentation in Zebrafish and Humans. *Science* **310**: 1782-1786 (2005).
20. Barsh, G.S. What Controls Variation in Human Skin Color. *PLoS Biology* **1**: 19-22 (2003).
21. Harpending, H.C. et al. Genetic Traces of Ancient Demography. *PNAS* **95**: 1961-1967 (1998).
22. Olson, S. *Mapping Human History: Discovering the Past Through Our Genes* (Houghton Mifflin Company, New York, 2002).
23. Rosenberg, N.A. et al. Genetic Structure of Human Populations. *Science* **298**: 2381-2385 (2002).
24. Cooper, R.S. Race and IQ: Molecular Genetics as Deus ex Machina. *American Psychologist* **60**: 71 - 76 (2005).
25. Diabetes Genetics Initiative, et al. Genome-Wide Association Analysis Identifies Loci for Type 2 Diabetes and Triglyceride Levels. *Science* **316**: 1331 - (2007).

26. Helgason, A., Yngvadottir, B., Hrafnkelsson, B., Gulcher, J. & Stefansson, K. An Icelandic example fo the impact of population structure on association studies. *Nature Genetics* **37**: 90 - 95 (2005).
27. Haga, S.B. & Venter, J.C. FDA Races in the Wrong Direction. *Science* **301**: 466-467 (2003).
28. Schwartz, R.S. Racial Profiling in Medical Research. *New England Journal of Medicine* **344**: 1392-1393 (2001).
29. Massey, D.S. Residential Segregation and Neighborhood Conditions in U.S. Metropolitan Areas. in *America Becoming: Racial Trends and Their Consequences*, Vol. I, eds. Smelser, N.J., Wilson, W.J. & MItchell, F. (National Academies Press, Washington, 2001).
30. Massey, D.S. & Denton, N. *American Apartheid: Segregation and the Making of the Underclass* (Harvard U. Press, Cambridge, 1993).
31. Orfield, G. & Lee, C. Why Segregation Matters: Poverty and Educational Inequality (Harvard Civil Rights Project, Cambridge, 2005).
32. Blank, R.M. An Overview of Trends in Social and Economic Well-being by Race. in *America Becoming: Racial Trends and Their Consequences*, Vol. I, eds. Smelser, N.J., Wilson, W.J. & MItchell, F. (National Academies Press, Washington, 2001).
33. Omi, M. & Winant, H. *Racial Formation in the United States, 2nd Edition* (Routledge, New York, London, 1994).
34. Smelser, N.J., Wilson, W.J. & MItchell, F. (eds.). *America Becoming: Racial Trends and Their Consequences,* (National Academy Press, Washington, 2001).
35. Ossorio, P. About Face: Forensic Genetic Testing for Race and Visible Traits. *Journal of Law, Medicine & Ethics* **34**: 277 - 287 (2006).
36. Lopez, I.H. *White By Law* (New York University Press, New York, London, 1996).
37. Omi, M.A. The Changing Meaning of Race. in *America Becoming: Racial Trends and Their Consequences*, Vol. I, eds. Smelser, N.J., Wilson, W.J. & MItchell, F. (National Academies Press, Washington, 2001).
38. Lopez, I.F.H. Race on the 2010 census: Hispanics & the shrinking white majority. *Daedalus* **134**: 42-52 (2005).
39. Prewitt, K. Racial classification in America: where do we go from here? *Daedalus* **134**: 5 - 17 (2005).
40. Smedley, A. *Race in North America: Origin and evolution of a worldview,* (Westview Press, Boulder, CO, 1999).
41. Fisher, C.S. et al. (eds.) *Inequality By Design: Cracking the Bell Curve Myth,* (Princeton University Press, Princeton, NJ, 1996).
42. Gardiner, H. *Frames of Mind: The Theory of Multiple Intelligences* (Basic Books, New York, 1983).
43. Goleman, D. *Working with Emotional Intelligence* (Bantam Books, New York, 1998).
44. Sternberg, R.J. *The Triarchic Mind: A New Theory of Human Intelligence* (Viking, New York, 1986).
45. Hernstein, R.J. & Murray, C. *The Bell Curve: The reshaping of American life by difference in intelligence* (Free Press, New York, 1994).

46. Lynn, R. *Dysgenics in Modern Populations* (Praeger, Westport, CT, 1996).
47. Lynn, R. & Vanhanen, T. *IQ and the Wealth of Nations* (Praeger, Westport, CT, 2002).
48. Kincheleo, J.L., Seteinberg, S.R. & Gresson, A.D. (eds.) *Measured Lies* (St. Martin's Press, New York, New York, 1996).
49. Brown, M.K. et al. *Whitewashing Race: The Myth of a Colorblind Society* (University of California Press, Berkeley and Los Angeles, CA, 2003).
50. Nisbett, R. *Intelligence and How to Get It: Why Schools and Cultures Count* (W.W. Norton & Company, New York, 2009).
51. Isaacs, E.B. et al. The Effect of Early Human Diet on Caudate Volumes and IQ. *Pediatric Research* **63**: 308 - 314 (2008).
52. Lidsky, T.I. & Schneider, J.S. Adverse effects of childhood lead poisoning: The clinical neuropsychological perspective. *Environmental Research* **100**: 284 - 293 (2006).
53. Ceci, S.J. How much does schoolign influence general intelligence and its cognitive components? A reassessment of the evidence. *Developmental Psychology* **27**: 703 722 (1991).
54. Evans, G.W. & Schamberg, M.A. Childhood poverty, chronic stress, and adult working memory. *Proceedings of the National Academy of Sciences, Early Edition* **10.1073/pnas.0811910106**: 1 - 5 (2009).
55. Sirin, S.R. Socioeconomic Status and Academic Achievement: A Meta-Analytic Review of Research. *Reivew of Educational Research* **75**: 417 - 453 (2005).
56. Zax, J.S. & Rees, D.I. IQ, Academic Performance, Environment, and Earnings. *The Review of Economics and Statistics* **84**: 600 - 616 (2002).
57. Flynn, J.R. The mean IQ of Americans: Massive gains 1932 to 1978. *Psychological Bulletin* **14**: 623 - 628 (1981).
58. Dickens, W.T. & Flynn, J.R. Black Americans Reduce the Racial IQ Gap. *Psychological Science* **17**: 913 - 920 (2006).
59. Deary, I.J. & Smith, P. Intelligence Research and Assessment in the United Kingdom. in *international Handbook of Intelligence*, ed. Sternberg, R.J. (Cambridge University Press, Cambridge, 2004).
60. Evans, P.D. et al. Microcephalin, A Gene Regulating Brain Size Continues to Evolve Adaptively in Humans. *Science* **309**: 1717 - 1720 (2005).
61. Mekel-Bobrov, N. et al. Ongoing Adaptive Evolution of ASPM, a Brain Size Determinant in Homo sapiens. *Science* **309**: 1720 - 1722 (2005).
62. Currat, M. et al. Commnet on "Ongoing Adaptive Evolution of ASPM, a Brain Size Determinant in Homo sapiens" and "Microcephalin, a Gene Regulating Brain Size Continues to Evolve Adaptively in Humans". *Science* **313**: 172(a) (2006).
63. Yu, F. et al. Comment on "Ongoing Adaptive Evolution of ASPM, a Brain Size Determinant in Homo sapiens". *Science* **316**: 370(b) (2007).
64. Rushton, J.P., Vernon, P.A. & Bons, T.A. No evidence that polymorphisms of brain regulator genes Microcephalin and ASPM are associated with general mental ability, head circumference or altruism. *Biology Letters* **3**: 157 - 160 (2007).

65. Mekel-Bobrov, N. et al. The ongoing adaptive evolution of ASPM and Microcephalin is not explained by increased intelligence. *Human Molecular Genetics* **16**: 600 - 608 (2007).
66. Bates, T.C. et al. Recently-derived variants of brain-size genes ASPM, MCPH1, CDK5RAP and BRCA1 not associated with general cognition, reading or language. *Intelligence* **36**: 689 - 693 (2008).

Pilar Ossorio is Associate Professor of Law and Bioethics at the University of Wisconsin at Madison, and Program Faculty in the Graduate Program in Population Health at the UW. Prior to taking her position at UW, she was Director of the Genetics Section at the Institute for Ethics at the American Medical Association, and taught as an adjunct faculty member at the University of Chicago Law School.

Dr. Ossorio received her Ph.D. in Microbiology and Immunology in 1990 from Stanford University. She went on to complete a post-doctoral fellowship in cell biology at Yale University School of Medicine. Throughout the early 1990's Dr. Ossorio also worked as a consultant for the federal program on the Ethical, Legal, and Social Implications (ELSI) of the Human Genome Project, and in 1994 she took a full time position with the Department of Energy's ELSI program. In 1993 she served on the Ethics Working Group for President Clinton's Health Care Reform Task Force.

Dr. Ossorio received her JD from the University of California at Berkeley School of Law (Boalt Hall) in 1997. While at Boalt she was elected to the legal honor society Order of the Coif and received several awards for outstanding legal scholarship.

Dr. Ossorio is a fellow of the American Association for the Advancement of Science's (AAAS), a member of the editorial board of the American Journal of Bioethics, chair of an NHGRI advisory group on ethical issues in large scale sequencing, and a member of UW's institutional review board for health sciences research. She is a past member of AAAS's Committee on Scientific Freedom and Responsibility, a past member of the National Cancer Policy Board (Institute of Medicine), and has been a member or chair of several working groups on genetics and ethics.