Article

Teaching Diversity: The Science You Need to Know to Explain Why Race Is Not Biological

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Abstract

This article is targeted to faculty teaching race and ethnicity, racism, diversity, and multicultural courses. Many students equate race with skin color. The premise of this article is that to teach students about the social construction of race, teachers must first know enough science to teach students that race is not biological. This article examines the biology of race by showing how advances in DNA sequencing led to genetics research that supports arguments that race is not biological. DNA comparisons show that all human populations living today are one species that came from Africa. The article explains the migration of humans out of Africa about 60,000 years ago and how they populated Australia, then Asia, Europe, and the Americas. The article shows how recent research maps the timing of the migration and admixture of specific population groups into Europe and India. The article shows how a mutation in one nucleotide can result in a trait like blue eyes, or Hemoglobin S (which confers resistance to malaria), which can be subject to evolution through natural selection. DNA comparisons show how natural selection shaped the genetics of human skin color to adapt to less UV light in the northern latitudes of Europe and Asia. The article shows that there is no relation between skin color or other "racial" characteristics and complex traits like intelligence. The science in this article will help teachers explain that as race is not biological, race is socially constructed and culturally enacted.

Keywords

teaching, race, racism, anti-racism, diversity, cultural competence, multicultural, genes, genetics, genome, biology of race, social construction of race

When I began teaching race and ethnicity, I discovered that many of my students believed that race was not an idea, but a fact. Some did not accept the idea that race was socially constructed even by the end of the semester. As I tried to answer their questions, I found that I did not know enough contemporary science to be able to explain why the idea of race was not biological. Consequently, I decided to delve into the scientific literature for answers. When I used the results of my research to teach my students, I was able to reach more of them.

This article argues that to teach students how race is socially constructed, faculty must have mastered enough science to show skeptical students that the idea of race is not biological. Most educators who teach in the areas of anti-racism, cultural competence, diversity, or race and ethnicity are social scientists. Few have backgrounds in biological anthropology, population genetics, or molecular biology. Yet, knowledge that relates to race as biology has exploded with contemporary progress in molecular biology and genomics. The purpose of this article is to present faculty with accurate scientific information on the biology of race to share with their students. This will help students understand that race as biology is not scientifically based. Therefore, the ideas about race that are held as folk concepts in U.S. culture must be socially constructed.

The concept that race does not have a biological basis, but instead is socially constructed, has been accepted among social scientists for three generations (cf. Montagu, 1942). Yet, many U.S. students enter anti-racism, cultural competence, diversity, or race and ethnicity classes in disciplines ranging from business to nursing to sociology, with the underlying assumption that race is biological (Coleman, 2011). As Goodman, Moses, and Jones (2012) explained, "race seems obviously real to anyone immersed in North America's dominant culture" (p. 2).

> We live in a society saturated with race. Racial thinking has infiltrated and now influences in some way or another everyone's experiences of health, education, romance, friendship, work,

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> religion, politics—virtually every arena and aspect of our lives. (Goodman et al., 2012, p. 9)

Students absorb normative beliefs that "race" is real as part of their culture (Smedley & Smedley, 2005).

In terms of background assumptions, this article examines the idea of race from the viewpoint of social scientific realism. Social scientific realism asserts that the social world comprises "not only human beings, but also the social relations and structures that are the products of human social interaction" (Carter, 2000, p. 1). This is to be contrasted with poststructuralist or postmodernist theories of the social world. The implications of social scientific realism include the following: (a) There is a social reality that is "relatively independent of individual social actors" and (b) social structures can "constrain and influence subsequent social actors" (Carter, 2000, p. 5). As Smedley and Smedley (2005) suggest, while race as biology is fiction, racism as a social problem is real. In the United States, "there are profound and stubbornly persistent . . . differences in socioeconomic status, educational and occupational status, wealth, [and] political power" based on ascribed race (Smedley & Smedley, 2005, p. 16).

From Traits to Genes to Genomes

Since the end of World War II, biological science has exploded. In 1953, Watson and Crick published the structure of the DNA molecule—the famous double helix. Since then, whole new fields, including molecular genetics, population genetics, epigenetics, and genomics, have developed. Advances in sequencing the human genome can serve as an example of how quickly scientific technology has been developing. The classical Sanger chain-termination DNA sequencing method was introduced in 1977 (Sanger, Nicklen, & Coulson, 1977). Sanger won a Nobel Prize for it in 1980. Sanger sequencing required a lot of space, and labs could typically run only about 100 reactions at a time. However, it was the most widely used sequencing method for about 25 years. By 1981, Sanger sequencing had been used to fully sequence human mtDNA, which has only 16,569 base pairs organized in a ring, making it easier to sequence. In 1990, the international project to map the human genome was begun. By 1996, the yeast genome was sequenced, followed by the *c. elegans* roundworm genome in 1998. The first draft of the human genome was announced in 2001. Finally, the first fully sequenced genome of a single person was published in 2003. It took more than 10 years, US$3 billion, and a massive international effort to accomplish the Human Genome Project using Sanger sequencing (Hayden, 2014b). By 2000, second generation high-throughput sequencing using technologies such as reversible terminator sequencing became commercially available. Technologies have advanced so quickly that, by 2014, a single machine could automatically sequence five full human genomes a day for close to US$1,000 each (Hayden, 2014a). Exciting new methods are being developed, including a nanopore technology, which uses an enzyme to feed an intact single-stranded length of DNA through a protein nanopore and reads the bases in order in one continuous read. The intact DNA can be hundreds of kilobytes long. All of these new technologies can be brought to bear on the issue of whether race is biological.

There are six accepted scientific arguments as to why race is a biological myth (Mukhopadhyay, Henze, & Moses, 2014). When students assume that race is a real biological entity, faculty can use these scientific arguments to help students begin to question their assumptions. I typically present the science during the second or third week of the semester.

1. People cannot be reliably divided into racial groups.
2. There are no relationships between traits that are used to categorize people into races (like skin color) and associated stereotypes.
3. Over time, geography and environment influence the genetic structures of human populations through natural selection.
4. There is more diversity within racial groups than between racial groups.
5. All people living today are descended from populations that originated in Africa.
6. All people living today are one biological species.

Considerable scientific evidence that supports these arguments has been published since 2000. The new science will help faculty address student misconceptions that race is biological.

People Cannot Be Reliably Divided Into Racial Groups

Many students enter class assuming that because they can "see" race, it is biological. They assume that by using visible human biological variation like skin color, hair texture, and facial characteristics, they can reliably divide people into racial categories. But McCarthy (2009) emphasizes that "racing" people is not just a visual but also a conceptual process. He states that "real or ascribed somatic markers are taken as signs of deeper differences . . . stereotypical representations combining phenotypic features with cultural and behavioral traits" (McCarthy, 2009, p. 10). To help students see that they are "racing" people in their minds, I present three ideas.

First, in the United States, the racial categories we use are essentially the same categories used by the Swedish botanist, Linnaeus, 250 years ago. Linnaeus (1758), using his new taxonomic system for categorizing plants and animals, classified people as belonging to the Class—*Mammalia*, Order—*Primates*, Genus—*Homo*, and Species—*sapiens*. Linnaeus further subdivided *Homo sapiens* into four types, on the basis of geography and skin color: *Europaeus* (white skins),

Asiaticus (yellow skins), *Americanus* (red skins), and *Afer* (black skins; Tattersall & DeSalle, 2011). Linnaeus further suggested that each group had a characteristic temperament due to an excess of one of Galen's four humors: blood, phlegm, yellow bile, and black bile (Brace, 2005). For example, Linnaeus characterized Europeans as having an excess of phlegm and being "sanguine," "confident, muscular, and inventive." He described American Indians as having an excess of blood and therefore being "choleric," "energetic, upright, and combative." He said that Asiatics had an excess of yellow bile, which caused them to be "melancholic" and "gloomy, thoughtful, inflexible, and avaricious," while Africans were said to have an excess of black bile, which caused them to be "bilious" and as a result, "self-contented, lazy, slow, and relaxed" (Tattersall & DeSalle, 2011, p. 12). Some of these stereotypes are still in use today.

Second, in class, we talk about how racial categories differ from culture to culture. For example, Barack Obama, the son of an African father from Kenya with dark skin, and a European American mother from Kansas with light skin, would typically be raced as "White" in Brazil but as "Black" in the United States, while he would have been raced as "colored" had he lived under South African apartheid. The idea that in Brazil, people with any European ancestry are raced as White, while in the United States, people who have any African ancestry are raced as Black, often drives discussion toward the "one drop rule" in the United States, and how by 1920, the same person might be raced differently depending on the state. This helps students to challenge their own culturally received notions of the fixedness of racial categories.

Third, we do a class activity to show that many human biological traits are continuous variables, rather than discrete categories. This can be done with a number of easily visible traits, such as height or curliness of hair (have students line themselves up from most straight to most curly), but I always conclude with skin color. Have students line themselves up from most pale to most dark skin (using the inner side of the upper arm works well). Then, let them try to decide where to divide themselves into specific categories. Ask, "How many categories should there be?" and "Why do there need to be categories?"

This exercise lets students see that skin color is an example of a biological cline—the gradual change of a trait across the geographical range of a species. Kittles and Weiss (2003) explain, that "if one examines only the geographic extremes, differences appear large" (p. 38). For example, if you juxtapose people from the Congo in West Africa, and people from Norway, you might conclude that the two peoples looked so different that they were of different "races." However, if you walked all the way from the Congo through Egypt and the Middle East to Norway, you would find that skin color varies very gradually—almost imperceptibly—as you travel. There is no place where one color of skin gives way abruptly to another—no place where you can draw a line and say that people on one side of the line have a different color of skin than people on the other side of the line. Famously, Livingstone (1962), a population biologist, concluded, "there are no races, there are only clines" (p. 279). Scientists have been unable to find a way to objectively and reliably divide people into the same set of racial groupings (Mukhopadhyay et al., 2014).

"Racial" Traits Do Not Correlate With Other Types of Biological Diversity

A second argument against race as biology is that the visible traits that people use to identify "race," like skin color, hair texture, or facial characteristics, do not correlate with other types of human biological diversity. Students in diversity classes sometimes assume that there is a gene for intelligence, or a gene for athletic ability, or a gene for heart disease. But that is not the case. Even a trait that seems relatively straightforward, like height (which has only one value for each person at a time) is highly complex genetically. For example, in a study of 183,727 adults, Allen et al. (2010) found that "hundreds of genetic variants, in at least 180 loci, influence adult height" (p. 832). However, the genetic variants they identified accounted for only 10.5% of the variation in height in their sample, suggesting that there are many more genes that affect height that have not yet been identified. Height is a classic polygenic trait, meaning that it is controlled by multiple genes, each of which has a very small effect (Allen et al., 2010).

If hundreds of genes control height, consider how much more complex a trait like "intelligence" is. Psychologists have spent 150 years trying to define and measure intelligence, and they still disagree (Nisbett et al., 2012). Some scientists believed that because intelligence, as measured by analytic IQ, is 40% to 80% heritable, genes affecting it would be easy to find, once the human genome had been mapped. However, "whereas 282 individual genes responsible for specific forms of mental retardation have been identified, very little progress has been made in finding the genes that contribute to normal variation" in intelligence (p. 135). In fact, Butcher, Davis, Craig, and Plomin (2008) reported that only one gene has been consistently replicated as influencing cognitive ability, and it explains less than 1% of the variance in general cognitive ability.

Cooper (2005) says that all too frequently, "during the last hundred years, the debate over the meaning of race has retained a highly consistent core . . . [built] around the same belief in Black inferiority" (p. 71). Every now and then, he says, someone uses "the latest jargon and half-truths from the margins of science" to reassert that people with the ascribed status of "Blacks" in the United States are inferior (Cooper, 2005, p. 71; see, for example, Chase, 1977; Hearnshaw, 1981; Hernstein & Murray, 1994; Rowe, 2005). However, Cooper concludes that

> despite substantial effort, no genetic polymorphism has yet been found that accounts for any significant proportion of the "racial

> differences" in the rates of common diseases, IQ, or any other similar trait—nor is there any reason . . . to expect that to be the case. Contrariwise, there is massive and highly consistent evidence of social influences. (p. 74)

In their review of the literature, Nisbett et al. (2012) agree. They conclude that "the direct evidence indicates that the difference between the races is entirely due to environmental factors" (p. 146).

Environment Influences the Genetic Structures of Human Groups via Natural Selection

A fundamental theory of biology is that over time, geography and environment influence the genetic structures of human populations through natural selection. People differ in any number of observable traits, such as height, shape of head, length of arms and legs, stockiness, and facial characteristics, just as they vary in other traits like ABO blood groups and Rh factors. Why is it that traits vary across populations?

Darwin (1876/1902) hypothesized that a single species, spread out over different environmental niches and isolated over time, would adapt to better fit new environments through natural selection. He observed that traits (such as beak shape and size in finches, or skin color or dentition in people) were heritable. However, he had no knowledge of the mechanism of heredity. (Although the work of Mendel outlining the basic mechanics of heredity had been published in an obscure journal in 1866, it was not "rediscovered" until 1900; Darwin died in 1882. So, although Mendel lived at the same time as Darwin, Darwin did not know of his work.)

Today, we know that human DNA consists of some 3.2 billion base pairs (G-C or A-T) arranged in the famous double helix model. During the process of meiosis, when human eggs or sperm cells are created, random copying errors can occur. Most are corrected, but occasionally some errors, or mutations, slip by. Mutations can occur in a single base pair, or through duplication or inversion of a gene (which can have thousands of base pairs), or they can occur through unequal crossing over or duplication of chromosomes (as in Trisomy 21, which causes Down syndrome). A mutation can be neutral, harmful, lethal, or (rarely) beneficial to the individual in whom it occurs. If the individual with the mutation successfully reproduces, the mutation may be passed on to the next generation. Mutations are important for evolution through natural selection because they introduce variety into DNA (Gould, 1980).

Mutations can be functional or nonfunctional. On one hand, mutations that are located in nonfunctional areas of the genome do not affect genes or parts of the DNA that turn nearby genes on and off. Therefore, as mutations in nonfunctional locations in the genome do not differentially affect successful reproduction, they are not subject to natural selection. However, nonfunctional mutations can be very useful as genetic markers to trace human history. These genetic markers can be used to show ancient human migration patterns; they can also be used to show how closely two population groups are related. For example, genetic markers have been used to determine how closely the Hadza (northern Tanzania), the Sandawe (central Tanzania), and the San (South Africa) peoples, all of whom speak rare click languages, are related (Tishkoff et al., 2007). Long (2003) concluded that "population history and relationships are read best from DNA sequences without function" (p. 17).

On the other hand, as functional mutations affect genes, or parts of the DNA that turn nearby genes on and off, they are subject to natural selection. Long (2003) concluded that, in contrast to mutations in nonfunctional areas, "DNA sequences that encode expressed genes will show patterns of variation that are more directly related to natural selection and human adaptation" (p. 17).

Blue eyes. Scientists are now able to demonstrate how a mutation in a single base pair (one of 3.2 billion) has resulted in a change in a known trait, such as blue eyes. A mutation in a single base pair is called a single nucleotide polymorphism (SNP). In 2008, Eiberg et al. were able to show how the mutation of a single base pair (from A to G) within the HERC2 regulatory gene inhibits the activity of the nearby OCA2 gene. Essentially, the mutation turns off the biosynthesis of melanin (the brown pigment that results in brown eyes) in the iris. People who have two copies of the mutation (the rs12913832*G allele) have blue eyes with no brown pigmentation. People who have two copies of the ancestral allele (the rs12913832*A allele) have brown eyes. The team's research suggests that this is a "founder mutation," meaning that everyone who has the *G allele at this position on chromosome 15q can trace their ancestry back to a single person in whom the mutation first occurred, who most likely lived in the northwest part of the Black Sea region 6,000 to 10,000 years ago (Eiberg et al., 2008). This type of work is pioneering, because it follows a specific single-point mutation (A to G), through its mechanisms of action, to show how it produces a known human trait. This is an example of the type of random mutation that has recently introduced genetic diversity into a population. However, scientists believe that the rapid increase in blue eyes in Europeans was due to sexual selection, rather than natural selection. They suggest that blue eyes may have been seen as novel, and therefore desirable, in mate selection (Eiberg et al., 2008). In contrast, let us look at how mutations affect traits that appear to have been beneficial to successful reproduction, and therefore were subject to natural selection.

Hemoglobin S. Hemoglobin S is an abnormal red blood cell which causes sickle-cell anemia. Sickle-cell anemia, a serious, often fatal disease, is also caused by a single-point mutation, from an A to a T. To get sickle-cell anemia, a person must inherit two copies of the mutation (one from each parent). The mutation alters one of the amino acids in the

hemoglobin protein which results in abnormal red blood cells, that, under certain conditions, change to a long, sickled shape which causes anemia, pain, and tissue damage (see DNA Learning Center, Cold Spring Harbor Laboratory, n.d.). Prior to modern medicine, sickle-cell anemia was invariably fatal. This should have resulted in people with sickle-cell anemia dying before they could reproduce, thus selecting against the mutation over the generations.

However, instead of being selected against, the sickle-cell mutation is very common in areas with malaria, such as India, the Mediterranean, and sub-Saharan Africa, where 10% to 40% of the population carries it. The reason is that for people who have only one copy of the sickle-cell mutation, the mutation confers tolerance to malaria—people still get malaria, but they do not die from it (Ferreira et al., 2011). Studies of the distribution of Hemoglobin S and the distribution of malaria show that the higher the prevalence of malaria, the higher the percentage of people with hemoglobin S. Where there is no malaria, 0% of the population has Hemoglobin S. Brace (1996) concluded that the frequency of the sickle-cell mutation in populations "is controlled by the intensity of infestation with falciparum malaria"—the parasite that causes malaria (p. 113). This is a classic example of natural selection. Over time, geography and the environment (areas conducive to malaria) interacted with a random mutation (Hemoglobin S) that conferred a reproductive advantage in people who carried one copy of the gene.

Researchers believe that the Hemoglobin S mutation occurred independently three times in Africa and once in either the Arabian Peninsula or central India between 70,000 and 150,000 years ago (Desai & Dhanani, 2004). While such mutations are rare, it is likely that over the last 100,000 years or so, this mutation also occurred a few times in areas of the world where there was no malaria. However, as the sickle-cell mutation conferred no advantage in the interaction between people and their (nonmalarial) environment, it died out.

Lactase persistence. All human infants are able to digest lactose, the primary carbohydrate in milk, because their intestines secrete lactase. In most human populations, the ability to secrete this enzyme goes away after weaning. This is the ancestral condition for humans (and all mammals; Itan, Powell, Beaumont, Burger, & Thomas, 2009). Accordingly, in East Asian and Southeast Asian populations, indigenous North and South American populations, and in large parts of Africa, 0% of the adult population has the lactase persistence trait. This makes sense, because, as long as human cultures were based on hunting and gathering, lactase persistence was unnecessary. However, as animals began to be domesticated, nonhuman milk (goat, cow, horse, camel, reindeer) became readily available as a source of food to pastoralists following their herds. Consequently, there was strong natural selection for lactase persistence in herding societies, and so dairying and lactase persistence co-evolved.

In Europeans, lactase persistence is due to a single C to T mutation at −13910*T, upstream from the lactase gene (Itan et al., 2009). The 13910*T allele accounts for virtually all the variance in lactase persistence frequency in Europe. It ranges in frequency from 73% to 95% in the British Isles and Scandinavia, 56% to 67% in Central and Western Europe, and 6% to 36% in Eastern and Southern Europe. Itan et al. (2009) date the origin of dairying and the co-evolving −13910*T lactase persistence mutation to about 7,500 years ago. Moreover, they conclude that this is a "recent mutation event" (Ingram, Mulcare, Itan, Thomas, & Swallow, 2009, p. 586); Bersaglieri et al. (2004) concluded that the signals of recent selection for the −13910*T lactase persistence mutation they observed were among the "strongest yet seen for any gene in the genome" (p. 1111).

However, genotype frequency comparisons have shown that the European mutation is not found in African and Middle Eastern dairying populations. Instead, different lactase persistence-associated mutations occurring in the same region of the genome have been identified. For example, Bedouins in Jordan and Saudi Arabia have the −13915*G mutation, while pastoralists in Kenya and Tanzania carry the −13907*G variant and the −14010*C variant (Ingram et al., 2009; Tishkoff et al., 2008). This is an example of convergent evolution. It is likely that over the last 10,000 years, mutations in the control area upstream of the Lactase (LCT) gene also occurred a few times in areas of the world where there was no dairying. However, as those mutations conferred no advantage to nondairying populations, they died out.

Skin color. Geography and environment also influenced the genetic structures that resulted in varied skin colors among different human populations. Unlike blue eyes or Hemoglobin S, skin color is a polygenic trait—one whose phenotype is influenced by multiple genes, each one having a relatively small effect (Sturm, 2009). Observable skin color is determined by the amount and distribution of two types of melanin pigment granules within skin cells—a red-yellow form known as pheomelanin and a black-brown form known as eumelanin (Barsh, 2003; McEvoy, Beleza, & Shriver, 2006). For example, Beaumont et al. (2007) showed that in Europeans, red hair and fair skin with freckles are associated with loss-of-function alleles of the Melanocortin Receptor (MC1R) gene. These alleles have been shown to decrease the ability of the skin to produce eumelanin (the black-brown melanin) in in vitro functional studies (Beaumont et al., 2007). Skin color is measured with a hand-held instrument using skin reflectance spectroscopy (Shriver & Parra, 2000).

Anthropologists assume that the "earliest members of the hominid lineage probably had a mostly unpigmented or lightly pigmented integument [skin] covered with dark black hair, similar to that of the modern chimpanzee" (Jablonski & Chaplin, 2000, p. 57). They also assume that after our lineage diverged from the lineage of modern chimpanzees, "coincident

with the loss of fur . . . there was strong selection for skin darkening" to protect the skin from the ultraviolet rays of the sun (McEvoy et al., 2006, p. R176). Both of these changes occurred roughly 2 to 5 million years ago (Fuentes, 2012). Therefore, anthropologists assume that the process of differentiation of skin color started from a population of *Homo sapiens* with dark skin.

On a global level, skin color in indigenous populations in the Old World tends to be darkest in the tropics. The farther north or south of the tropics, the lighter skin color becomes. The reason appears to be related to the amount of sun light, which is much greater at the equator than at the poles. Melanin absorbs and scatters light (Jablonski & Chaplin, 2000). Jablonski and Chaplin (2013) found that "86% (r = 0.927) of the variation in human skin reflectance" can be accounted for by autumn levels of ultraviolet radiation (p. 672). The reason is that humans need enough ultraviolet light to make Vitamin D, but too much ultraviolet light results in sunburn and the destruction of folate, which is necessary for healthy reproduction. The amount of pigment in human skin enables populations to balance these two needs. Therefore, on one hand, in the high latitudes like Sweden or Siberia, there was "positive directional selection" for less melanin in the skin so that people in the high latitudes could make more Vitamin D (Jablonski & Chaplin, 2000, p. 673). On the other hand, near the equator, in places like Papua New Guinea, Southern India, or the Congo, there was positive selection for more melanin in the skin, so that people could be protected from too much sun and the destruction of folate (Jablonski & Chaplin, 2000).

Selective sweeps. With regard to how much effect natural selection has had on *Homo sapiens* in the last 50,000 years or so, at the turn of the 21st century, there were still two camps. Those in the first camp argued that cultural evolution, such as adapting to extreme cold by learning to make thick clothes out of fur skins, had lessened the effects of natural selection. Those in the second camp argued that *Homo sapiens* continued to benefit from positive natural selection during the last 50,000 years (Hawks, Wang, Cochran, Harpending, & Moyzis, 2007). Rogers (2011) states that "as recently as the year 2000, it was possible for Stephen Jay Gould to argue that 'natural selection has almost become irrelevant in human evolution. There's been no biological change in humans in 40,000 or 50,000 years'" (p. 93). Gould (1980) had previously argued that "cultural evolution is our primary innovation. It works by the transmission of skills, knowledge and behavior through learning—a cultural inheritance of acquired characters" (p. 137). Rogers states that in 2000, when Gould gave this interview, there was no way to tell which position was correct. However, had Gould lived 10 years longer, advances in genetics would have made Gould's position "untenable," and Rogers believes Gould would have changed his mind. Today, just 15 years later, there is plenty of evidence that natural selection is still affecting humans. Indeed, Hawks et al. (2007) argue that the rapidly expanding world population since the domestication of plants and animals (resulting in shifts from hunter-gatherer cultures to pastoralism and agriculture, about 10,000 years ago) has resulted in a 100-fold increase in mutations, thus increasing the amount of positive natural selection that is occurring.

The way scientists evaluate genomes for evidence of positive natural selection is to look for selective sweeps. Long (2003) states that

> a favorable allele may occasionally rise rapidly and sweep through the population replacing all other alleles at that locus. This mode of selection is often referred to as positive selection. One consequence of a selective sweep is that the level of background genetic variation in the vicinity of the favored allele is reduced. (p. 7)

Another way to explain this is that DNA tends to be inherited in chunks or blocks of base pairs grouped around a functional SNP on a chromosome. Functional mutations (SNPs) that occurred earlier in human history tend to have shorter blocks or chunks of unchanged DNA surrounding them, because they had many more generations for recombination to shorten the length of the unchanged stretches of DNA attached to them. However, functional mutations (SNPs) that have occurred relatively recently in human history have much longer chunks of unchanged DNA surrounding them. Several new statistical tests have been developed to evaluate the presence of selective sweeps and their population frequency (Huff, Harpending, & Rogers, 2010; Pickrell et al., 2009; Sabeti et al., 2002; Voight, Kudaravalli, Wen, & Pritchard, 2006). These tests are particularly effective at identifying selective sweeps at moderate frequency (~50%–80%), and at high frequency (>80% to fixation) within a population (Pickrell et al., 2009). For example, McEvoy et al. (2006) identified "a 150 kb region surrounding the SLC24A5 gene" that "shows a large drop in heterozygosity in Europeans." This means that the 150 kb region around the gene is unchanged and thus shows evidence of recent selection (p. R177).

McEvoy et al. (2006) were looking for genes within regional populations that affected skin pigmentation. The team studied DNA from three geographically distinct populations from West Africa, East Asia, and Northern Europe. Based on differences among these three groups, they developed a four-step evolutionary model for skin pigmentation. First, they found evidence of mutations in two genes (MITF and EDN3) that occurred in the ancestral human population, prior to any splits between the groups. Second, they found evidence of a split between the African group and the ancestors of Asians and Europeans, who carried the ASIP and BNC2 genes. Third, they identified five genes that showed positive selection only in the East Asian population. Fourth, they identified five different genes that showed positive selection only in the Northern European population. All the

genes identified were involved in the pathway for the biosynthesis of melanin. Both Northern Europe and East Asia are at much higher latitudes than Africa, so pale skin with less melanin benefitted humans in both environments. But since mutations are random, nature could only positively select the mutations related to the biosynthesis of melanin that actually occurred in the Northern European and East Asian populations. Thus, the genetic mechanisms that resulted in the pale skin color of East Asians and Europeans are different; this is an example of convergent evolution (McEvoy et al., 2006). The McEvoy et al. (2006) study shows clear evidence for the operation of natural selection with regard to skin color. As a result of studies like this one, molecular biologists have concluded that "skin colour as a selectable trait has likely occurred multiple times at diverse geographical sites around the globe" (Sturm, 2009, p. 13).

There Is More Diversity Within Than Between Racial Groups

The fourth argument against race as biology is that there is more diversity among people within groups than between groups. That means that there is more diversity among people within a single population, for example, among Samoans, than there is between regions (or "races") like Europe and sub-Saharan Africa. Lewontin (1972) was the first to demonstrate this. In a landmark article, he examined the frequency of alleles of 17 genes over 101 different populations. First, he established that there were differences in frequencies between populations for each of the 17 genes. For example, he found that the Duffy antigen Fy^a occurs in 66% of Europeans and 99% of East Asians but in only 10% of sub-Saharan Africans (Lewontin, 1972). Then, Lewontin divided the 101 populations into seven "races": Caucasian, African, Mongoloid, South Asian Aborigines, Amerinds, Oceanians, and Australian aborigines. For example, in his Amerind race, he included 21 North, Central, and South American populations; in his Oceanian race, he included 15 populations ranging from the Hawaiians to the Maori.

Lewontin (1972) stated that the statistical question he asked was "How much of human diversity between populations is accounted for by more or less conventional racial classification?" (p. 386). He noted that dividing world populations into seven races (instead of four, for example, as Linnaeus did) and weighting them equally maximizes "both the total human diversity and the proportion of it that is calculated between populations" (Lewontin, 1972, p. 385). In other words, his assumptions bias the statistics toward showing more diversity between regions or "races." Using the frequencies of different alleles, he calculated diversity within each population (e.g., Basques, or Koreans, or Navaho). Then, he calculated diversity between populations within a region, for example, between all 15 of the Oceanian populations. Then, he calculated the diversity between the seven regions or "races."

Lewontin (1972) concluded that "the results are quite remarkable" (p. 396). Of the total genetic diversity (100%), he found that the average within-population diversity (e.g., within Danes, or within Navaho) was 85.4%. The difference between populations within a region (e.g., between the 15 Oceanian populations) accounted for 8.3% of the total diversity. He found that only 6.3% of the total diversity was accounted for by regional or "racial classification" (Lewontin, 1972, p. 396). What this means is that most biological variability exists between you and your neighbors in the same population. Lewontin stated that "based on randomly chosen genetic differences, human races and populations are remarkably similar to each other, with the largest part by far of human variation being accounted for by the differences between individuals" (p. 397).

Lewontin's (1972) pioneering findings continue to be borne out by more recent studies. Relethford (2002) summarized six studies looking at within-group variation versus between-group variation. He reviewed studies assessing differences in blood polymorphisms, microsatellite DNA, RFLPs, *Alu* insertions, mtDNA, Y-chromosome DNA, and craniometrics. He concluded that they all showed the same pattern. Variation within local populations (e.g., Tongans) accounts for 68.9% to 85.4% of total differences found. Variation between populations within a region (e.g., populations of Europe, like the Danes and the Spanish) accounts for 1.3% to 8.4% of the total variation (Relethford, 2002). Finally, variation between regions or "races" (e.g., Europe vs. sub-Saharan Africa) accounts for 6.3% to 24.9% of total differences found (Relethford, 2002, p. 396).

Findings also suggest that the more genetic diversity there is within a single population, the older the population is. There is a correlation between frequency of variation, age, and geographic distribution (Kittles & Weiss, 2003). The greater the variation within a geographic distribution, the greater the age of that population. Using several measures of genetic variation, researchers have determined that sub-Saharan Africans have "almost twice the diversity of non-African populations" (Kittles & Weiss, 2003, p. 44). In fact, all the genetic diversity of populations from all over the world can be found within sub-Saharan Africa (Wells, 2003). The San (Bushmen) have the most within-population diversity of any group on the planet and, thus, are considered to be the oldest of all the populations living today (Li et al., 2008; Tishkoff et al., 2009; Wells, 2003).

The exception to this pattern is skin color. Relethford (2002) found that 88% of the total variation in worldwide skin color is found between regions or "races" (e.g., Europe vs. sub-Saharan Africa), while only 9% of the total variation is within group (e.g., among Tongans, or among San, or among Danes; p. 396). As stated earlier, the observable trait of skin color is not correlated with other traits. What this means is that while there is a relationship between continental ancestry and skin color, the classifications we call "race" are meaningless when it comes to genes and DNA for human traits other than skin color. "Race" is truly only skin deep.

All Humans Living Today Are Descended From Africa

The fifth argument against race as biology is that all humans living today are descended from a small group of ancestors who lived in Africa. This is the "out of Africa" or "recent African origin" model (Veeramah & Hammer, 2014). In the past, some anthropologists had "argued for the origin of human races through a process of separate speciation events from ape-like ancestors in many parts of the world. This hypothesis became known as multi-regionalism" (Wells, 2003, p. 33). The multiregionalism hypothesis has not been supported. Evidence from genetics supports the idea that we are all one people, whose ancestors lived in Africa.

The more recently two species have branched from a common ancestor, the more similar their DNA sequences will be. When random mutations occur in nonfunctional areas of DNA, and are passed down to offspring, they stay in the DNA. Thus, they can serve as markers for our ancestry. Wells (2003) explains that

> our DNA carries, hidden in its string of four simple letters, a historical document stretching back to the origin of life . . . the differences we see when we compare DNA from two or more individuals . . . are the historical language of the genes. (p. iii)

Molecular biologists compare DNA to find differences, called genetic markers, which they can then use to calculate the genetic distance between two populations (Cavalli-Sforza, 2000). In some cases, they can use the genetic markers to estimate how long it has been since the populations split into two groups.

For an example of how comparison of DNA can be used, take the Neanderthals. The fossil record shows that they lived in Europe and Asia from about 400,000 years ago to 30,000 years ago, and overlapped with modern humans from about 45,000 to 30,000 years ago (Green et al., 2010). There have been many questions about how Neanderthals and *Homo sapiens* were related (Cavalli-Sforza, 2000; Sykes, 2001). The team that analyzed the first Neanderthal genomes found that humans and Neanderthals share about 99.7% of their DNA, and that humans and Neanderthals shared common ancestors about 500,000 years ago (Green et al., 2010). The results showed clearly that the Neanderthals were not our direct ancestors (Cavalli-Sforza, 2000). Instead, the Neanderthals were classified as our "sister" species (*Homo sapiens neandertales*, whereas we are *Homo sapiens*). However, the team found that 1% to 4% of DNA is shared between Neanderthals and Eurasian (but not African) *Homo sapiens* (Green et al., 2010). This suggests that there was some interbreeding (Green et al., 2010; Stewart & Stringer, 2012).

By comparison, the first study to sequence and compare the genome of a chimpanzee with a human genome determined that we share 96% of our DNA with chimpanzees, who are our closest living relatives (Cheng et al., 2005; Lovgren, 2005). The findings of this team suggested that the ancestors of *Homo sapiens* (including Neanderthals) and chimpanzees diverged about 6.5 million years ago (Lovgren, 2005).

The same methods of comparing DNA can be used to tell how closely related two human populations are (Fairbanks, 2012; Jorde et al., 2000). A decade ago, three teams, led by Cavalli-Sforza, by Sykes and by Wells, reported on years of work looking at relationships among human populations. In 2000, Cavalli-Sforza summarized research conducted over 40 years. His team analyzed gene frequencies on 110 blood system groups and proteins from samples of members of about 2,000 aboriginal populations throughout the world. He concluded that all modern humans are members of one species which originated in Africa. He calculated that some modern humans left Africa between 80,000 and 50,000 years ago and reached Australia by 50,000 years ago. His model of human migration from Africa suggests that after Australia, humans settled Asia, then Europe, and finally, via East Asia, the Americas (Cavalli-Sforza, 2000).

MtDNA. In 2001, Sykes summarized 25 years of work on mitochondrial DNA (mtDNA), which has the advantage of being passed intact, directly from mother to child. As mtDNA is found within the mitochondria, small "energy factories" within cells, it is not involved in sexual reproduction. When a mutation, such as a change from a G to an A, occurs in the DNA of a mitochrondrion in a woman's germ cell, if that germ cell is passed on to a daughter, the mutation will continue in the daughter's DNA. In other words, your mtDNA is an exact copy of your mother's mtDNA, which is an exact copy of her mother's DNA, which is an exact copy of her mother's DNA, and so on. A mutation occurs in mtDNA about once every 10,000 years, which enables scientists to estimate how long ago two lines shared a common maternal ancestor (Sykes, 2001).

After analyzing thousands of samples from all over Europe, Sykes (2001) found that nearly all native Europeans could trace their maternal ancestry to one of just seven different women, who lived between 45,000 and 10,000 years ago. Remarkably, 47% of Europeans have mtDNA that traces back to one woman, who lived about 20,000 years ago, most likely in the area of the Rhone River in France. Sykes explained that each of these women was certainly not the only woman living in her hunting and gathering band at the time. But if a woman has no children or only boys, her mtDNA line dies out.

Using mtDNA sequences from all over the world, Sykes (2001) identified 33 women who are the mothers of all people living today, and 13 of them originated in Africa. His findings suggest that a small group from one of the African mtDNA clans left Africa to populate the rest of the world. The 20 women whose mtDNA clans are found in the rest of the world are all descendants of that single woman whose descendants left Africa. Sykes writes,

> Incredibly, even though the African clans are easily the most ancient in the world, we are still able to reconstruct the genetic relationships among them . . . the ancestors of the ancestors . . . One by one the clans converge until there is only one ancestor, the mother of all of Africa and of the rest of the world . . . "Mitochondrial Eve." (Sykes, 2001, p. 276)

Sykes (2001) concluded that "we are all direct maternal descendants" of Mitochondrial Eve, who lived about 150,000 years ago in Africa (pp. 276-277).

Y-chromosome DNA. In 2003, Wells released a book on his research team's findings using DNA from the Y-chromosome. The Y-chromosome is passed down from fathers to sons just as mtDNA is passed down from mothers to daughters. As with maternal lineages, if a man has no children or no sons, his Y-chromosome lineage is lost. The Wells (2003) team studied a "worldwide sample of men, from dozens of populations on every continent" (p. 53).

Wells (2003) concluded that all modern humans originated in Africa. He also concluded that all men alive today can trace their paternal lineage back to one man—"Y-chromosome Adam"—who lived in Africa about 60,000 years ago. While there were other men alive then, their lineages have died out. He noted that the difference between the estimated age of "Mitochondrial Eve" and "Y-chromosome Adam" (who were real people) means that more female lineages survived, unbroken, into the present day. As a result, scientists could trace the maternal line much further back into prehistory (150,000 years) than they could trace the paternal line of all people living today. Wells explained that the greater the diversity, as shown by the accumulation of additional genetic markers, the older the lineage. As a result, he said that "you are more likely to sample extremely divergent genetic lineages within a single African village than you are in the whole of the rest of the world" (Wells, 2003, p. 39). He emphasized that the real surprise is that as all modern humans lived in Africa until at least 60,000 years ago, our species has only had 60,000 years to populate the globe (Wells, 2003).

Wells (2003) divided the descendants of men who left Africa into a genealogical tree with 11 lineages. Each genetic marker represents a single-point mutation (SNP) at a specific place in the genome. First, genetic evidence suggests that a small band with the marker M168 migrated out of Africa along the coasts of the Arabian Peninsula and India, through Indonesia, and reached Australia very early, between 60,000 and 50,000 years ago. This very early migration into Australia is also supported by Rasmussen et al. (2011). Second, a group bearing the marker M89 moved out of northeastern Africa into the Middle East 45,000 years ago. From there, the M89 group split into two groups. One group that developed the marker M9 went into Asia about 40,000 years ago. The Asian (M9) group split three ways: into Central Asia (M45), 35,000 years ago; into India (M20), 30,000 years ago; and into China (M122), 10,000 years ago. The Central Asian (M45) group split into two groups: toward Europe (M173), 30,000 years ago and toward Siberia (M242), 20,000 years ago. Finally, the Siberian group (M242) went on to populate North and South America (M3), about 10,000 years ago.

Whole genomes. While the earlier studies compared frequencies of gene alleles for blood proteins, or genetic markers in parts of the mtDNA or the Y-chromosome, second generation sequencing now enables teams to compare whole genomes. For example, Li et al. (2008) analyzed 650,000 base pairs that had been previously identified as having mutations (SNPs), in the genomes of 1,064 individuals from 51 populations. The genomes of members of 51 populations were compared with each other and with chimpanzees. (The chimpanzee genomes were assumed to represent "ancestral allele frequencies"—the frequencies of alleles before humans and chimpanzees began to diverge from a common ancestor.) Li et al. constructed a phylogenetic tree diagram showing the relationships among the populations. The least different from the ancestral allele frequencies were the San (Bushmen) and other sub-Saharan African populations, followed by North African populations, European, Middle Eastern, Central Asian, East Asian, Amerindian, and Oceanian populations. (Australian aboriginal populations were not studied.) They concluded that their findings were consistent with a "serial founder effect, a scenario in which population expansion involves successive migration of a small fraction of individuals out of the previous location, starting from a single origin in sub-Saharan Africa" (Li et al., 2008, p. 1103). Their study fully supports the recent African origin model.

Second-generation population history studies. Recent work with second generation sequencing and new statistical modeling methods adds much finer detail on migration and admixture of populations. For example, in Europe, Stewart and Stringer (2012) show a cyclical pattern of interglacial expansion of *Homo sapiens* out of Africa and retreat back to Africa or warmer refuges during ice ages. They showed that populations of Homo sapiens moved out of Africa during warm interglacial periods, then retreated from Europe and Asia into southern areas like Spain, Italy, the Balkans, and the Caspian Sea during the last ice age (about 27,000-11,700 years ago), and then expanded again to repopulate Europe after the climate warmed. Behar et al. (2006) supported these findings by showing that the Basques are a remnant of the Paleolithic hunter-gatherer population that retreated into the area around the Bay of Biscayne during the last ice age. The hunter gatherers then expanded to resettle Central and Western Europe after the end of the last ice age. Using genome-wide data from 94 ancient Europeans who lived 8,000 to 3,000 years ago, Haak et al. (2015) modeled the genetic admixture among the indigenous hunter gatherers, Neolithic farmers expanding into Europe from Anatolia (starting about 8,000 years ago), and the Yamnaya or kurgan culture—a pastoralist culture from the

steppes north of the Black and Caspian Seas. The Yamnaya culture had horses, wheeled vehicles, and carried the gene for lactase persistence. Haak et al.'s study showed a massive migration of the Yamnaya people into central Europe about 4,500 years ago, one that could have spread an early form of the Indo-European language, which is the basis of most of today's European languages.

Much of northern India also speaks Indo-European languages. In the first major work on population history in India, Reich, Thangaraj, Patterson, Price, and Singh (2009) used second generation sequencing technology and statistical modeling to compare the genomes of 25 diverse groups from India to the genomes of people from Western Asia, Europe, and the Andaman Islands (who are thought to be closest to the ancestral populations of southern India). They found that the "Ancestral North Indians" (ANI) form a clade with Europeans (meaning that they are closely related), but are genetically distinct from the "Ancestral South Indians" (ASI), who form a clade with the Andaman Islanders (Reich et al., 2009). Using admixture modeling, they found that different Indian groups inherited different proportions of ancestry from the ANI who are related to western Eurasians, and the ASI, who are related to the Andaman Islanders (p. 491). This parallels the language data—Northern Indian groups speak Indo-European languages, while Southern Indian groups speak Dravidian languages. However, Reich's team called for further investigation to date the beginning of the admixture of Northern Indian and Southern Indian populations.

Thus, the work of teams examining blood group allele frequencies, mtDNA, Y-chromosome DNA, and whole genomes, all shows the same pattern. By comparing the DNA of people from around the world, scientists can determine where their ancestors came from and approximately when migrations took place. And in doing so, they have found that the ancestors of all humans living today came from Africa (Veeramah & Hammer, 2014). We are all Africans, no matter the color of our skin.

All Humans Living Today Are One Biological Species

The sixth argument against race as biology is that all humans living today are one biological species. The most common definition of a biological species is "a group of interbreeding natural populations that are reproductively isolated from other such groups" (Fuentes, 2012, p. 108). In other words, one criterion that defines populations as part of the same species is that they can mate and reproduce successfully. By that criterion, it is clear that all humans today are part of the same species. Different species, for example, fox and gray squirrels east of the Mississippi River in the U.S., do not recognize each other as potential mates. All humans, from any population group, can and do mate and reproduce successfully (unlike, for example, horses and donkeys, which can mate, but produce mules, which are infertile). Mukhopadhyay et al. (2014) state that "Contemporary humans are, and always have been, one species, with roots in Africa. There are no subspecies of humans" (p. xvi).

Conclusion

On its face, race seems to be "a concept that is intuitively biologically based" (Kittles & Weiss, 2003, p. 34). Yet, during the 18th century, the idea that humans could be divided into different biological races that were correlated with different traits and characteristics, such as "intelligent and hard-working" versus "unintelligent and indolent," seems to have appeared about the same time that chattel slavery was rapidly expanding as a money-making opportunity for Europeans (Smedley & Smedley, 2005). This suggests that the idea of race had a social genesis tied to the justification of slavery. However, even during the time of Linnaeus, there were some authors, e.g., Blumenbach, who recognized that "no absolutely sharp distinction could be expected between different peoples and [that] . . . the varieties of mankind . . . seemed to have been arbitrarily chosen as to both number and definition" (Boyd, 1950, as cited by Kittles & Weiss, 2003, p. 35). Since then, despite the fact that humans may look very different from each other on the surface, scientists have found that under the skin, we are all alike.

In summary, there are a number of evidence-based reasons why race is not biological. First, there are no objective, identifiable traits by which people can be reliably divided into racial groups. Second, skin color, which is the primary basis of folk conceptions of race in the United States, has been shown to be the result of natural selection; it is a result of the interaction between geographical environment and random mutations in genes that affect the biosynthesis of melanin in the skin. Third, visible traits like skin color do not correlate with traits like blood groups, much less with complex traits like intelligence, athletic ability, or musical ability. Fourth, another way to show that skin color does not correlate with other human traits is to statistically compare within-population variation versus between-region variation. Researchers find that there is much more variation within a single population, like the Yoruba, than there is between geographical regions or "races" (e.g., between Europeans and sub-Saharan Africans).

Fifth, by comparing the genetic markers (mutations or SNPs) of people in populations all over the world, scientists can determine population expansion patterns of the past. Using "molecular clocks," they can also roughly estimate how many thousands of years ago an out-migration or admixture occurred. By comparing genetic markers in mtDNA, Y-chromosome DNA, autosomal DNA or whole genomes, researchers concluded that all humans living today are descended from Africa. Furthermore, all human populations outside of Africa are descended from one hunting and gathering band that migrated from Eastern Africa, probably about 60,000 years ago. Within Africa, the ancestry of sub-Saharan

African populations can be traced back to the San, who appear to have the most similarities to the ancestors of all people living today (Li et al., 2008; Tishkoff et al., 2009). There is no research that supports the theory of polygenesis or multiregionalism, which posited that different "races" arose independently in different geographic regions.

Finally, because members of all human populations living today can successfully interbreed, by definition, humans constitute one species. Although some populations lived long enough in certain geographic environments for natural selection to alter characteristics like skin color, these populations were not isolated long enough to become new species. There is a great deal of both prehistoric and historic evidence for extensive global trading routes, which led to people from different groups meeting and mating over thousands of years of human history (Fuentes, 2012).

Many students in the United States equate race with skin color. The idea that race is not skin color, or a set of discrete biological types, but instead is socially constructed, is one of the most important ideas of our time. Omi and Winant (2015) conclude as follows:

> Race is not something rooted in nature, something that reflects clear and discrete variations in human identity. But race is also not an illusion. While it may not be "real" in a biological sense, race is indeed real as a social category with definite social consequences. (p. 110)

For many students, moving from race-as-biology to race-as-socially-constructed is as profound as learning that the earth is round, when they always knew it was flat (Goodman et al., 2012). It requires a paradigm shift. While this paradigm shift will not be sufficient to eliminate racism in our time, it is surely a necessary step in the right direction.

When I used these six scientific arguments as to why race is not biological to teach my students, I found that more of them accepted the idea that race is socially constructed. I suspect that I am not the only social scientist teaching diversity classes whose teaching may benefit from increased biological science literacy. Understanding how 21st century science supports arguments against race-as-biology will help educators assist students to make the necessary paradigm shift.

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References

Allen, H. L., Estrada, K., Lettre, G., Berndt, S. I., Weedon, M. N., Rivadeneira, F., . . . J. N. (2010). Hundreds of variants clustered in genomic loci and biological pathways affect human height. *Nature*, *467*, 832-838. doi:10.1038/nature09410

Barsh, G. S. (2003). What controls variation in human skin color? *PLoS Biology*, *1*, 19-22. doi:10.1371/journal.pbio.0000027

Beaumont, K. A., Shekar, S. L., Newton, R. A., James, M. R., Stow, J. L., Duffy, D. L., & Sturm, R. A. (2007). Receptor function, dominant negative activity and phenotype correlations for MC1R variant alleles. *Human Molecular Biology*, *16*, 2249-2260.

Behar, D. M., Harmant, C., Manry, J., van Oven, M., Haak, W., Martinez-Cruz, B., . . . The Genographic Consortium (2006). The Basque paradigm: Genetic evidence of a maternal continuity in the Franco-Cantabrian region since Pre-Neolithic times. *The American Journal of Human Genetics*, *90*, 486-493.

Bersaglieri, T., Sabeti, P. C., Patterson, N., Vanderploeg, T., Schaffner, S. F., Drake, J. A., . . . Hirschhorn, J. N. (2004). Genetic signatures of strong recent positive selection at the lactase gene. *American Journal of Human Genetics*, *74*, 1111-1120.

Boyd, W. C. (1950). *Genetics and the races of man: An introduction to modern physical anthropology*. Boston, MA: Little, Brown.

Brace, C. L. (1996). A four-letter word called "race." In L. T. Reynolds & L. Lieberman (Eds.), *Race and other misadventures: Essays in honor of Ashley Montagu in his ninetieth year* (pp. 106-141). Dix Hills, NY: General Hall.

Brace, C. L. (2005). *"Race" is a four-letter word: The genesis of the concept*. New York, NY: Oxford University Press.

Butcher, L. M., Davis, O. S. P., Craig, I. W., & Plomin, R. (2008). Genome-wide quantitative trait locus association scan of general cognitive ability using pooled DNA and 500K single nucleotide polymorphism microarrays. *Genes, Brain and Behavior*, *7*, 435-446.

Carter, B. (2000). *Realism and racism: Concepts of race in sociological research*. London, England: Routledge.

Cavalli-Sforza, L. L. (2000). *Genes, peoples, and languages*. Berkeley: University of California Press.

Chase, A. (1977). *The legacy of Malthus: The social costs of the new scientific racism*. New York, NY: Knopf.

Cheng, Z., Ventura, M., She, X., Khaitovich, P., Graves, T., Osoegawa, K., . . . Eichler, E. E. (2005). A genome-wide comparison of recent chimpanzee and human segmental duplications. *Nature*, *437*, 88-93. doi:10.1038/nature04000

Coleman, S. (2011). Addressing the puzzle of race. *Journal of Social Work Education*, *47*, 91-108.

Cooper, R. S. (2005). Race and IQ: Molecular genetics as Deus ex Machina. *American Psychologist*, *60*, 71-76. doi:10.1037/0003-066X.60.1.71

Darwin, C. (1902). *The origin of species by means of natural selection*. New York, NY: Collier. (Original work published 1876)

Desai, D., & Dhanani, H. (2004). Sickle cell disease: History and origin. *The Internet Journal of Hematology, 1*(2), 1540.

DNA Learning Center, Cold Spring Harbor Laboratory. (n.d.). *Sickle cell anemia, 3D animation with narration*. Retrieved from http://www.dnalc.org/view/15532-Sickle-cell-anemia-3D-animation-with-narration.html

Eiberg, H., Troelsen, J., Nielsen, M., Mikkelsen, A., Mengel-From, J., Kjaer, K. W., . . . Hansen, L. (2008). Blue eye color in

humans may be caused by a perfectly associated founder mutation in a regulatory element located within the HERC2 gene inhibiting OCA2 expression. *Human Genetics*, *123*, 177-187. doi:10.1107/s00439-007-0460-x

Fairbanks, D. J. (2012). *Evolving: The human effect and why it matters*. Amherst, NY: Prometheus Books.

Ferreira, A., Marguti, I., Bechmann, I., Jeney, V., Chora, A., Palha, N. R., . . . Soares, M. P. (2011). Sickle hemoglobin confers tolerance to Plasmodium infection. *Cell, 145*, 298-409.

Fuentes, A. (2012). *Biological anthropology: Concepts and connections* (2nd ed.). New York, NY: McGraw-Hill.

Goodman, A. H., Moses, Y. T., & Jones, J. L. (2012). *Race: Are we so different?* Malden, MA: American Anthropological Association.

Gould, S. J. (1980). *The panda's thumb: More reflections in natural history*. New York, NY: W.W. Norton.

Green, R. E., Krause, J., Briggs, A. W., Maricic, T., Stenzel, U., Kircher, M., . . . Paabo, S. (2010). A draft sequence of the Neandertal genome. *Science*, *328*, 710-722. doi:10.1126/science.1188021

Haak, W., Lazaridis, I., Patterson, N., Rohland, N., Mallick, S., Llamas, B., & Reich, D. (2015). Massive migration from the steppe was a source for Indo-European languages in Europe. *Nature*, *522*, 207-211. doi:10.1038/nature14317

Hawks, J., Wang, E. T., Cochran, M., Harpending, H. C., & Moyzis, R. K. (2007). Recent acceleration of human adaptive evolution. *Proceedings of the National Academy of Sciences of the United States of America*, *104*, 20753-20758.

Hayden, E. C. (2014a, 15 January). Is the $1,000 genome for real? *Nature*. doi:10.1038/nature.2014.14530

Hayden, E. C. (2014b). Technology: The $1,000 genome. *Nature*, *507*, 294-295. doi:10.1038/507294a

Hearnshaw, L. S. (1981). *Cyril Burt, psychologist*. New York, NY: Vintage Books.

Hernstein, R., & Murray, C. (1994). *The bell curve: Intelligence and class structure in American life*. New York, NY: Free Press.

Huff, C. D., Harpending, H. C., & Rogers, A. R. (2010). Detecting positive selection from genome scans of linkage disequilibrium. *BMC Genomics*, *11*(8), 1-9. Retrieved from http://www.biomedcentral.com/1471-2164/11/8

Ingram, C. J. E., Mulcare, C. A., Itan, Y., Thomas, M. G., & Swallow, D. M. (2009). Lactose digestion and the evolutionary genetics of lactase. *Human Genetics*, *124*, 579-591. doi:10.1007/s00439-008-0593-6

Itan, Y., Powell, A., Beaumont, M. A., Burger, J., & Thomas, M. G. (2009). The origins of lactase persistence in Europe. *PLoS Computational Biology*, *5*(8), E1000491. doi:10.1371/journal.pcbi.1000491

Jablonski, N. G., & Chaplin, G. (2000). The evolution of human skin coloration. *Journal of Human Evolution*, *39*, 57-106.

Jablonski, N. G., & Chaplin, G. (2013). Epidermal pigmentation in the human lineage is an adaptation to ultraviolet radiation. *Journal of Human Evolution*, *65*, 671-675.

Jorde, L. B., Watkins, W. S., Bamshad, M. J., Dixon, M. E., Ricker, C. E., Seielstad, M. T., & Batzer, M. A. (2000). The distribution of human genetic diversity: A comparison of mitochondrial, autosomal and Y-chromosome data. *American Journal of Human Genetics*, *66*, 979-988.

Kittles, R. A., & Weiss, K. M. (2003). Race, ancestry, and genes: Implications for defining disease risk. *Annual Review of Genomics and Human Genetics*, *4*, 33-67. doi:10.1146/annurev.genom.4.070802.110356

Lewontin, R. C. (1972). The apportionment of human diversity. In T. Dobzhansky, M. K. Hecht, & W. C. Steere (Eds.), *Evolutionary biology* (Vol. 6, pp. 381-398). New York, NY: Appleton-Century-Crofts.

Li, J. Z., Absher, D. M., Tang, H., Southwick, A. M., Casto, A. M., Ramachandran, S., . . . Myers, R. M. (2008). Worldwide human relationships inferred from genome-wide patterns of variation. *Science*, *319*, 1100-1104.

Linnaeus, C. (1758). *Systema naturæ per regna tria naturæ, secundum classes, ordines, genera, species, cum characteribus, differentiis, synonymis, locis* [System of nature through the three kingdoms of nature, according to classes, orders, genera and species, with characters, differences, synonyms, places] *(Tomus I. Editio decima, reformata, 1-824)*. London, England: Salvius.

Livingstone, F. B. (1962). On the non-existence of human races. *Current Anthropology*, *3*, 279-281.

Long, J. C. (2003, November 21). *Human genetic variation: The mechanisms and results of microevolution*. Paper presented at the session "Exploring the Nature of Human Biological Diversity: Myth v. Reality" at the American Anthropological Association (AAA) 2003 Annual Meeting, Chicago, IL.

Lovgren, S. (2005, August 31). Chimps, humans 96 percent the same, gene study finds. *National Geographic News*. Retrieved from http://news.nationalgeographic.com/news/2005/08/0831_050831_chimp_genes.html

McCarthy, T. (2009). *Race, empire, and the idea of human development*. Cambridge, UK: Cambridge University Press.

McEvoy, B., Beleza, S., & Shriver, M. D. (2006). The genetic architecture of normal variation in human pigmentation: An evolutionary perspective and model. *Human Molecular Genetics*, *15*, R176-R181.

Montagu, A. (1942). *Man's most dangerous myth: The fallacy of race*. New York, NY: Columbia University Press.

Mukhopadhyay, C. C., Henze, R., & Moses, Y. T. (2014). *How real is race? A sourcebook on race, culture, and biology* (2nd ed.). Lanham, MD: Rowman & Littlefield.

Nisbett, R. E., Aronson, J., Blair, C., Dickens, W., Flynn, J., Halpern, D., & Turkheimer, E. (2012). Intelligence: New findings and theoretical developments. *American Psychologist*, *67*, 130-159. doi:10.1037/a0026699

Omi, M., & Winant, H. (2015). *Racial formation in the United States* (3rd ed.). New York, NY: Routledge.

Pickrell, J. K., Coop, C., Novembre, J., Kudaravalli, S., Li, J. Z., Absher, D., . . . Pritchard, J. K. (2009). Signals of recent positive selection in a worldwide sample of human populations. *Genome Research*, *19*, 826-837.

Rasmussen, M., Guo, X., Wang, Y., Lohmueller, K. E., Rasmussen, S., Albrechtsen, A., . . . Willerslev, E. (2011). An aboriginal Australian genome reveals separate human dispersals into Asia. *Science, 334*, 94-98.

Reich, D., Thangaraj, K., Patterson, N., Price, A. L., & Singh, L. (2009). Reconstructing Indian population history. *Nature*, *461*, 489-495. doi:10.1038/nature08365

Relethford, J. H. (2002). Apportionment of global human genetic diversity based on craniometrics and skin color. *American Journal of Physical Anthropology*, *118*, 393-398.

Rogers, A. R. (2011). *The evidence for evolution*. Chicago, IL: University of Chicago Press.

Rowe, D. C. (2005). Under the skin: On the impartial treatment of genetic and environmental hypotheses of racial differences. *American Psychologist*, *60*, 163-173.

Sabeti, P. C., Reich, D. E., Higgins, J. M., Levine, H. Z. P., Richter, D. J., Schaffner, S. F., . . . Lander, E. S. (2002). Detecting recent positive selection in the human genome from haplotype structure. *Nature*, *419*, 832-837.

Sanger, F., Nicklen, S., & Coulson, A. R. (1977). DNA sequencing with chain-terminating inhibitors. *Proceedings of the National Academy of Sciences of the United States of America*, *74*, 5463-5467.

Shriver, M. D., & Parra, E. J. (2000). Comparison of narrow-band reflectance spectroscopy and tristimulus colorimetry for measurements of skin and hair color in persons of different biological ancestry. *American Journal of Physical Anthropology*, *112*, 17-27.

Smedley, A., & Smedley, B. D. (2005). Race as biology is fiction, racism as a social problem is real: Anthropological and historical perspectives on the social construction of race. *American Psychologist*, *60*, 16-26.

Stewart, J. R., & Stringer, C. B. (2012). Human evolution out of Africa: The role of refugia and climate change. *Science*, *335*, 1317-1321.

Sturm, R. A. (2009). Molecular genetics of human pigmentation diversity. *Human Molecular Genetics*, *18*, R9-R17. doi:10.1093/hmg/ddp003

Sykes, B. (2001). *The seven daughters of Eve*. New York, NY: W.W. Norton.

Tattersall, I., & DeSalle, R. (2011). *Race? Debunking a scientific myth*. College Station: Texas A&M University Press.

Tishkoff, S. A., Gonder, M. K., Henn, B. M., Mortensen, H., Knight, A., & Mountain, J. L. (2007). History of click-speaking populations in Africa inferred from mtDNA and Y chromosome genetic variation. *Molecular Biology and Evolution*, *24*, 2180-2195. doi:10.1093/molbev/msm155

Tishkoff, S. A., Reed, F. A., Friedlaender, F. R., Ehret, C., Ranciaro, A., Froment, A., . . . Williams, S. M. (2009). The genetic structure and history of Africans and African Americans. *Science*, *324*, 1035-1044. doi:10.1126/science.1172257

Tishkoff, S. A., Reed, F. A., Ranciaro, A., Voight, B. F., Babbitt, C. C., Silverman, J. S., . . . Deloukas, P. (2008). Convergent adaptation of human lactase persistence in Africa and Europe. *Nature Genetics*, *39*, 31-40.

Veeramah, K. R., & Hammer, M. F. (2014). The impact of whole-genome sequencing on the reconstruction of human population history. *Nature Reviews Genetics*, *15*, 149-162.

Voight, B. F., Kudaravalli, S., Wen, X., & Pritchard, J. K. (2006). A map of recent positive selection in the human genome. *PLoS Biology*, *4*(3), e72.

Wells, S. (2003). *The journey of man: A genetic odyssey*. New York, NY: Random House.

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