

Article

The Myth of the Genetically Sick African

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Abstract: Western medicine has an unfortunate history where it has been applied to address the health of African Americans. At its origins, it was aligned with the objectives of colonialism and chattel slavery. The degree to which medical “science” concerned itself with persons of African descent was to keep them alive for sale on the auction block, or to keep them healthy as they toiled to generate wealth for their European owners. Medicine in early America relied upon both dead and live African bodies to test its ideas to benefit Europeans. As medicine moved from quackery to a discipline based in science, its understanding of human biological variation was flawed. This was not a problem confined to medicine alone, but to the biological sciences in general. Biology had no solid theoretical basis until after 1859. As medicine further developed in the 20th century, it never doubted the difference between Europeans and Africans, and also asserted the innate inferiority of the latter. The genomic revolution in the latter 20th century produced tools that were deployed in a biomedical culture still mired in “racial” medicine. This lack of theoretical perspective still misdirects research associated with health disparity. In contrast to this is evolutionary medicine, which relies on a sound unification of evolutionary (ultimate) and physiological, cellular, and molecular (proximate) mechanisms. Utilizing the perspectives of evolutionary medicine is a prerequisite for an effective intervention in health disparity and finally dispelling the myth of the genetically sick African.

Keywords: history of medicine; race; racism; genomics; evolutionary medicine



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1. Introduction

In 1769, Dr. Joseph Lewis, the personal physician to Dartmouth's president Eleazar Wheelock, peeled the skin from a deceased enslaved man named Cato. He boiled his body in a kettle to free the flesh from the bone. He intended to keep the skeleton for anatomical study. Cato's skin was tanned and used to dress the doctor's instrument case (Wilder 2013). This example exemplifies the early history of American medicine with regard to persons of African descent. The degree that colonial (and later American) medicine was concerned with persons of African descent was in so far as they could be tools to help heal persons of European descent, or from the same perspective as that of veterinarians taking care of domestic animals. This was evident at the very beginning of the Transatlantic trade. Slave ship's surgeons were charged with taking care of the sick, with the purpose of delivering more enslaved persons to the auction block (Mustakeem 2016). In 1798, the founding father, physician Benjamin Rush, claimed that “blackness” was a form of leprosy that physicians might in future cure (Willoughby 2018). In 1799, the physicians William H. Harvey and John Lindsey identified the disease “Cachexia Africana.” This was characterized in persons of African descent by “dirt-eating” associated with emotional distress and depression (Haller 1972). Actually, this condition was not a “pathology” but a cultural practice practiced in many tropical countries (Abrahams and Parsons 1996). Physicians during the antebellum period profited greatly from the institution of slavery. Many physicians offered discounts to slave holders for their services designed to keep their enslaved people healthy enough to continue their labors.

Both science and medicine played a role in the justification and maintenance of chattel slavery. It must be emphasized that prior to the 20th century, progress in both natural

science and medicine was uneven. Neither had a well-validated theoretical paradigm to guide their efforts. Indeed, various levels of outright quackery were commonplace in both disciplines. Thus, assumptions about the physiological and mental capacities of Africans generally went unchallenged. One of the most commonly used of these originated with Carolus Linnaeus (1707–1778) in the 10th edition of *Systema Naturae* published in 1758. Linnaeus named four varieties of humans, and considered *Homo sapiens afer*, the least capable of these mentally. Another scientific claim concerning the enslaved Africans is that they were more physiologically suited to hard labor in the subtropical climates of the southernmost English colonies compared to Europeans (Derickson 2019). While for some physical traits, such as skin color, this was undoubtedly true, apologists for slavery such as Phillip Tidyman and Samuel Cartwright invented others that were patently not true. For example, Tidyman claimed that Africans were protected from the unhealthiness of hot climates by the nature of their constitution. Cartwright claimed that the African eye was better suited for harsh sunlight, in the same way that the orangutan's eyes were (Derickson 2019). Of course, in the former case, while persons whose ancestors evolved in tropical climates were more capable of hard labor in subtropical climates than people whose ancestors evolved in northern temperate climates, this was not a justification for enslaving them and for making them work beyond anyone's physical limits. Indeed, this strange logic viewed the tropical adaptation of Africans as a mark of inferiority. This argument also did not consider the capacity for all individuals to undergo physiological acclimation to new climates. Thus, poor persons of European descent, who did not own slaves, wearing the proper clothing, taking in adequate amounts of water and salt, often worked long hours in the subtropical sun. Most of them were the descendants of indentured servants, and laws differentiating the status of these individuals from those of enslaved Africans appeared very early in the history of the English colonies (1650's). These laws had nothing to do with the purported physiological differences between the two groups.

As enslaved individuals had no control over their bodies, they served as the material that medical science used to build its understanding of anatomy as well as medical techniques. In this period the success of medical colleges was tied almost directly to their capacity to acquire bodies for anatomy. At the university of Pennsylvania in 1762, the Pennsylvania Hospital gave the director of the university's anatomy course the body of a negro who died from suicide (Wilder 2013). The Pennsylvania hospital also served many negro patients who served as the material for its research program. Similar cases occurred in New York, where King's College utilized the "Negroes Burial Ground" as source of cadavers. In 1773, the body of a Negro was used to produce the skeleton used for anatomy lessons in Providence, Rhode Island.

The role that African American bodies played in the founding of gynecology has been recently brought to light (Owens 2017). However, in this period, the Irish had not yet achieved "whiteness" and so poor Irish women were utilized for medical experimentation as well. In addition, ideas of racialized differences between African American and Irish women made them excellent material for experimentation. Their "promiscuity" and insensitivity to pain justified experimenting on them without use of anesthesia (Paugh 2017). Both of these traits, promiscuity and insensitivity to pain, were thought to result from the biological inferiority of these groups.

Throughout the 19th century, American medicine continued to contribute to the ideology of African racial inferiority. Samuel Morton (1799–1851) amassed a skull collection of ~1000 individuals to study the differences in human cranial capacity. He did so within the paradigm of polygeny (Graves 2005a). This held that the various "races" of humanity were in reality different biological species. His collection contained disproportionate numbers of African and American Indian skulls. This resulted simply because these groups had no means to protect themselves from wanton death and murder at the hands of Europeans. At least 51 of these skulls were taken by grave robbers from Cuba (Wade 2021). Not surprisingly, Morton found that the cranial volumes of these inferior species were less than those of Europeans (Gould 1981). Samuel Cartwright (1793–1863) is best known (and

often ridiculed) for his theories of racial difference, particularly his coining of the uniquely African diseases of drapetomania (insane desire to run away), rascality (unruliness), and dysaesthesia ethiopica (performing tasks in a haphazard manner). Yet, Cartwright's views were thoroughly in line with the racial thinking of Europeans in this period (Willoughby 2018). Indeed, it was felt that all southern physicians should be evaluated by their capacity to judge the resale value of enslaved persons by being fully aware of the diseases of the Negro (Owens and Fett 2019). Modern medicine has still not completely escaped the principles of racial difference that Cartwright established. In his 1851 "Report on the Diseases and Physical Peculiarities of the Negro Race", he complained about the lack of attention paid to race by northern and southern medical educators. His approach to the importance of racial difference was largely unexceptional to the tens of thousands of American medical students in the 19th century, and is still a foundational orientation of many within medicine in the 21st century. For example, the physician Sally Satel claimed that she was a "racially profiling" doctor in 2002 (Graves 2011). Other studies indicated that Satel was not alone. In 2016, a survey of medical students at the prestigious University of Virginia Medical School indicated that many entered with false views of human biological difference. For example, 19.4% of second-year medical students felt that black nerve endings were less sensitive than white, 38.9% of second years thought that blacks age more slowly than whites, and 37.3% of third years thought that black skin was thicker than white (Hoffman et al. 2016). The former belief concerning nerves and pain perception began during chattel slavery (Haller 1972). Worse was the fact that several of the claims of human difference that the authors of the 2016 paper thought were true, were in fact false (Graves 2021).

2. Why Has Medicine Always Gotten Race Wrong?

For most of its history, medicine got much more than race wrong. This was because there was no consistent integration of natural science in the discipline. Scientific principles that we take for granted today were not widely accepted prior to the 20th century (e.g., circulation, germ theory of disease). It was still common in the 19th century for physicians to bleed people to "balance their humors." Medical leech therapy was still being widely used for balancing humors in Europe and the United States in 1850 (Singh and Rajoria 2020). The medical mantra of "do no harm" had particular relevance at this time because more often than not, medical intervention killed the patients. There were no national standards for medical training until well into the 20th century. The salaries of professors of medicine were dependent upon the number of students that they trained. For this reason, there was necessarily a great deal of variability between the skill and knowledge of physicians (Barry 2018). It may seem ironic, but Samuel Cartwright was an internationally recognized figure in medicine. He received his medical degree from Transylvania University (Kentucky) in 1826. He was widely cited on topics as diverse as pneumonia, croup, fever, and cholera. He received an award from Harvard University for his works on veins and absorption, and a medal from the Boston Medical Society for his essay on resuscitating drowning victims (Willoughby 2018).

Medicine got race wrong in the 19th century because all disciplines got it wrong. Graves 2005 summarizes the views of naturalists concerning race in the 18th and 19th centuries. In the 18th century, naturalists generally agreed that there was one human species but disagreed about whether the observed differences between human groups resulted from innate (genetic) or environmental differences. Additionally, in the Western world, all theories of human variation were special creationist. This meant that all humans were the result of a supernatural act of creation and all their attributes were determined by God's plan. God, of course was "white", and Jesus was routinely depicted as having European features. This of course was hard to reconcile given that these people realized that he was born the child of a Middle Eastern Levantine woman. Thus, more is learned about 18th and 19th century views of human variation by understanding their theology than by understanding their science (Keel 2019). The collusion of theology and science to

understand nature was dubbed “natural theology”, and in natural theology all things in nature had to relate to God’s plan which revolved around human beings. In the mind of the Europeans, God clearly planned for white people to dominate the world.

Therefore, the prerequisite to getting race right involved learning how variation within species actually came about. This required solving the species problem. It was becoming more apparent by the middle of the 18th century that the Earth was much older than Christian scripture could allow for, and that species that no longer walked the Earth had existed in its past (Eiseley 1958). Jean Baptiste Lamarck was the first consistent evolutionist, arguing that species evolved through geological time along specific species plans. These plans had originated in the mind of the creator. By the middle of the 19th century, it was also clear that Lamarck’s ideas about evolution within species plans could not be correct. In 1850, Charles Lyell (the most respected person in English science) felt that the “species” problem was the most significant issue in biology (Desmond and Moore 1991). Charles Darwin solved the species problem and published the solution in 1859 (Darwin 1859). Darwin held that variation within species was real and important (variation), that variants were transmitted from parents to offspring (heredity), and that some variants were more significant in the struggle for life than others (natural selection). It was natural selection that gave rise to the diversity of variation within species and drove the production of new species through geological time. This meant that all species resulted from descent with modification, meaning that they shared common ancestry.

However, Darwin carefully stayed away from discussing humans in *The Origin*. This was because he realized that his most strident opposition would come from those who rejected that Europeans shared ancestry with their African servants (even though this is what the Christian Bible said as well.) Chief amongst his concerns was the anticipated response from the polygenist Louis Agassiz (one of the four horsemen of polygeny, Graves 2005a). However, in 1871, Darwin returned specifically to the question of humans in *The Descent of Man and Selection in Relation to Sex* (Darwin 1871). His chapter on the “Races of Man” was specifically designed to debunk the claims of the polygenists and establish that there was only one species of humanity. Furthermore, Darwin argued that based upon how naturalists had treated variation within other species, that our species did not have biological races (Graves 2005a).

Darwin’s understanding of evolution was not all correct, and further development of its theory took place in the later portion of the 19th and early 20th century. Specifically, the unification of population genetics with natural selection (known as the Neo-Darwinian synthesis) was required to develop a full understanding of how genetic variation accumulated within populations. The Darwinian impact on anthropology was slow coming as well, but by the mid-20th century the argument about the lack of biological races within the human species was beginning to take shape. Ashley Montagu first published *Man’s Most Dangerous Myth: The Fallacy of Race*, in 1942 (Montagu 1997). The latter portion of the 20th century saw further developments in both theory and data, further casting doubt on the legitimacy of classifying biological races within the human species (Lewontin 1972; discussed more fully in Graves 2021).

The significance of evolutionary biology for medicine was almost completely ignored in the 20th century (Neese and Williams 1994; Graves 2011). Of all the contributions of evolutionary biology, the only one that has had significant impact on medical thinking was microbial evolution and the concept of antibiotic resistance (Dodds 2017). This followed due to the widespread acceptance of the germ theory of disease in the early 20th century, and the observation of the rapid spread of antibiotic resistance beginning in the 1950’s. However, even today in 2021, the appreciation of the fact that all diseases have both ultimate (evolutionary) and proximate (physiological, cellular, molecular) bases is generally not understood by physicians (Graves et al. 2016; Stearns and Medzhitov 2016). There are several reasons for this, including the fact the evolution is not a required course for entry in medical training, and even when it is taught, its significance for medicine (and understanding of human biological variation) is not emphasized (Donovan 2015; Graves

et al. 2016). In addition, those who enter medical training without serious challenges to the racial smog they inhabit simply never get around to recognizing the fallacies of racial ideas in medicine. The 10th edition of one of the most widely used textbooks in pathology (*Robbins Basic Pathology*) was rife with racial errors (Evans et al. 2021; Deyrup and Graves 2021). In another pathology text, *Robbins and Cotran Pathologic Basis of Disease, Professional Edition (8th Edition)*, it was found that nearly two-thirds of the assertions that African Americans have a unique disease profile compared to other racialized groups could not be supported by the literature. Similarly, one of the most widely selling texts in pediatrics also represents consistent racial misconceptions (Li et al. 2022). Finally, the religiosity of physicians around the world mediated against a strong adoption of evolutionary ideas to clinical practice. One survey of American family practice physicians found that 79.1% rated themselves as being somewhat strong, or strongly religious (Daaleman and Frey 1999). There is an inverse relationship between religiosity and acceptance of evolution (Mead et al. 2018). This relationship is particularly strong in African American communities (Bailey et al. 2011).

3. Racial Medicine and the Genetically Sick Negro

The myths of the genetically sick African began in chattel slavery and have persisted to the current day. In the late 19th century, this was well-represented in the “Negro Extinction hypothesis.” This was articulated in the work of Frederick Hoffman in 1896 (Hoffman 1896). In short, Hoffman cataloged a variety of sources of morbidity and mortality amongst the Negro race and showed that for multiple infectious diseases (e.g., consumption—tuberculosis, pneumonia, venereal disease, malaria, typhoid, smallpox, measles), behavioral problems (neglect of children, alcoholism, insanity and lunacy), and chronic disease (cancer), that Negroes had higher rates than whites. These were in turn reducing the reproductive rates of the Negro, such that Hoffman felt that the inevitable extinction of the Negro must follow in short order.

While Hoffman did not make any explicitly genetic arguments in his work, the notion that these conditions resulted from the innate inferiority of the Negro followed without need of explanation. Within 20 years of Hoffman, individuals such as Charles B. Davenport of the Eugenics Record Office (ERO) would “geneticize” the inferiority of Negroes, as well as that of poor whites. The work of the ERO around misrepresenting the cause of the vitamin deficiency disease pellagra is an important example. Pellagra was rampant in the American south by 1900. In 1916, the epidemiologist Joseph Goldberger began serious studies of the disease. He began by demonstrating that it was not caused by an infectious agent. He also noted that there were no cases of this disease amongst the wealthy and reasoned that it had something to do with poverty. Goldberger conducted a study to show how pellagra was related to poor-quality diets by convincing convict volunteers to subsist on a high carbohydrate diet without protein or fresh green vegetables, and was able to produce the disease in all of them in 6 months. He also demonstrated how to cure the disease by providing people with access to high-quality, balanced diets (Chase 1980). However, this research was ignored by the ERO as it lobbied congress to view pellagra as a genetic disease. The difference of viewing pellagra as resulting from poverty versus resulting from genes had important policy implications. The ERO argued for eugenic sterilization as the solution. If pellagra was a disease caused by poverty, it required measures to alleviate that condition, such as quality jobs and higher wages. Over the course of the pellagra epidemic, records showed that African Americans died from this condition at much higher rates than European Americans—more evidence of the genetically sick Negro.

The ideology of the inferior African was resisted by African Americans themselves. We see evidence of this as early as the writings of Frederick Douglass (Douglass 1854). In 1869, African American physicians found themselves shut out of professional medical societies. The founding of medical schools at the newly formed black colleges such as Howard and Fisk allowed for the training of more black physicians. This increased need for a national

medical association that dealt with the needs of the African American community was apparent by the end of the century. In 1895, the National Medical Association (NMA) was founded to better unite and educate African American physicians. The NMA was at odds with the white medical associations on a variety of issues, especially the inferiority of the Negro (Morris 2007). W.E.B DuBois spent a great deal of time on the idea that differential exposure to the environmental sources of disease in African American communities was responsible for their elevated morbidity and mortality, as discussed in his monumental work on the Philadelphia Negro published in 1897.

By the 1960's, patterns of mortality and morbidity for biological sources of death were still disproportionately higher in African Americans compared to European Americans (Graves 2013). However, by this point, African American birth rates were surpassing those of European Americans. This meant that the Hoffman extinction thesis had failed, and it was replaced with the dysgenesis arguments of people such as Arthur Jensen (Graves 2005a). Medicine still relied on biological (mainly genetic) arguments for health disparity. This is well-illustrated by the case of hypertension. By the 1930's, reports of elevated blood pressure in African Americans were widespread. In the 1960's, similar reports of this condition amongst Afro-Caribbean populations were also common. Many in the medical community argued that this was a racial condition of the Negro. This idea was bolstered by the "slavery hypertension" hypothesis. This came into vogue in the late 1980's and was prefaced on the idea that salt was rare in Western Africa, and for that reason Western Africans had evolved the capacity to retain salt in their tissues. This was further exacerbated by survival in the Transatlantic slave trade (specifically Middle passage). Of course, the core assumptions that supported the hypothesis had no evidence supporting them. For example, salt was abundant in the Western African societies that produced enslaved individuals, and records of enslaved cargoes could not be used to identify heat exhaustion as a primary cause of death on Transatlantic voyages (Graves 2005a, 2005b).

A common error resulting from the genetically sick African is to assume that population-based differences in gene frequency are the primary factors driving health disparities. This can be seen in arguments about the prostate cancer differential observed between persons of West African and European ancestry. However, all genes are expressed in the context of environment. This means that in this case, the genetic variants associated with an increased risk of prostate cancer do so as a function of the environment experienced by males in question. As the environments faced by males of African descent across the world are generally harsher than those faced by those of European descent, the degree to which these variants are associated with prostate cancer may be strongly mediated by environmental context.

A recent review of genome-wide association studies of genetic variants within the 8q24 locus showed that these variants are found in all populations (Tong et al. 2018). However, of 48 studies listed in this review, only one of these was conducted on an African population living in Africa (Cameroon). The studies found 24 SNPs that were commonly associated with prostate cancer risk worldwide. However, of these 24, 15 were shared by both African- and European-American men (although in different frequencies); whereas there were only 1 of the 27 that were found in African- but not European-Americans, and 6 of the 27 that were found only in European- but not African-Americans. While these numbers suffer from ascertainment bias, as most of the studies of prostate cancer have been conducted on Europeans, European Americans, and E. Asians, on the face of it these numbers do not construct a strong case for the African-/European-American prostate cancer disparity primarily resulting from allelic frequency differences. More prostate cancer-associated variants exist that are unique to European- as opposed to African-Americans. If genetics were the main causal factor determining the prevalence of prostate cancer, we should expect more European Americans to display prostate cancer, but the data show the exact opposite pattern.

Furthermore, to lay to rest the myth of the genetically sick African, it is important to have a deeper understanding of the genetic foundations of complex disease. We now know that these diseases result from the action of many genetic variants each contributing small

amounts to the resultant phenotype. The modern technique for analyzing these genetic influences on any phenotype (including diseases) is called genome-wide association study (GWAS). This method is designed to reveal associations between variants and traits. The association is a statistical relationship between the co-occurrence of alleles and phenotypes. Association can have many causes:

1. Direct causation: allele A causes phenotype B;
2. Epistatic effect: people who display phenotype B are more likely to survive and reproduce if they have allele A;
3. Population subdivision: the population has several subpopulations, and phenotype B and allele A happen to be at high frequency in some of the subpopulations;
4. Type I error: GWAS studies test large numbers of markers; by chance alone, 5% will be significant at $p = 0.05$ and 1% will be significant at $p = 0.01$;
5. Linkage disequilibrium (LD): allele A marks a chromosome segment that contains a genetic marker that causes phenotype B.

GWAS as a method to describe the underlying basis of complex disease has had variable success. This is well-illustrated by a trait such as height. The trait has very high heritability (offspring resemble parents, h^2). Yet, recent GWAS studies of height have only explained a small fraction of heritable variation for the trait. This results from the small effect size of individual variants, low frequencies of the variants within populations, population size, marker density, and the rate at which LD diminishes with map distance (Yang et al. 2010). While h^2 for height is 0.80, 180 single nucleotide polymorphisms (SNPs) were identified with $p < 5 \times 10^{-8}$ which together accounted for about 10% of variation in the trait (the study comprised over 180,000 individuals; Hill 2012). This means that each significant SNP accounted for 5.5% or 0.00055 of the variation in height. Around this same time, a meta-analysis of variation in blood pressure, that examined results from 34,433 individuals of northern European ancestry, identified 10 SNPs within the following loci: ATP2B1, CYP17A1, PLEKHA7, SH2B3 for systolic BP and ATP2B1, CACNB2, CSK/ULK3, SH2B3, TBX3/TBX5, and ULKK4 for diastolic BP. The 10 SNPs in total accounted for 1% of the variation in measured blood pressure in this sample. This means that the 99% of variation in blood pressure is determined by genetic variants whose statistical association never rose to the required $p < 1.0 \times 10^{-8}$, and environmental factors. Seven years later, a much larger study of northern Europeans ($N = 342,415$) identified 66 loci at $p < 1 \times 10^{-8}$. Those associated with systolic BP accounted for 3% of the variation. Over the last 20 years, GWAS studies have improved their capacity to predict phenotypic outcomes by dramatically increasing the number of individuals tested, e.g., >1,000,000 individuals. Such studies can often account for >10% of the variation depending upon the trait. However, all these studies are influenced strongly by what population is being studied, such that results for GWAS in Europeans do not always predict the same set of genetic markers operating in non-Europeans. For example, early studies of the genomics of viral set point for HIV uncovered different SNP markers for African Americans and Europeans (Chapman and Hill 2012).

Finally, there is absolutely no reason to have believed that Africans have more deleterious genetic variants than Europeans do. Studies that examine the frequency of deleterious variants genome-wide suggest that it is Europeans, not Africans who maintain more deleterious variants in their genome (Lohmueller et al. 2008). That study showed that there was no statistical difference between Africans and Europeans in the frequency of genetic variants that were possibly deleterious, but that there was a significant statistical difference favoring Africans for having less variants that were probability deleterious. All human populations show some level of local adaptation. The number of traits for which there is very strong evidence of local adaptation is amazingly small. These are also traits that most people would guess should be locally adapted, such as those related to: arctic environment, high-fat food (found in arctic populations), lactase persistence (found in populations that domesticated cows), high altitude (found in the East Africa plateau, Andes, and Himalayas), adaptation to diet (starchy foods), adaptation to infectious disease (such as malaria and try-

panosomes), and skin pigmentation (along the gradient of solar intensity from the tropics to the arctic, Fan et al. 2016). Furthermore, adaptation to these traits is generally the result of single or small groups of loci. In general, we do not have any well-validated examples of adaptation resulting from complex genetics; notable exceptions are selection for greater height in some groups of northern Europe and the rainforest phenotype (short stature) found in tropical Africa, Asia, and South America (Fan et al. 2016).

This lack of support for widespread adaptation in traits with underlying complex genetics is to be expected. Methods have been developed to assess the signature of natural selection on polygenic traits (Zhang et al. 2013). Classic quantitative genetics tells us that selective sweeps reduce the heterozygosity of nearby neutral polymorphisms and provides a strong genetic signature of selection (Sabeti et al. 2006). However, in eukaryotes there is increasing evidence that “soft” selective sweeps, particularly adaptation resulting from changes in allele frequencies of standing genetic variation, could be the major mechanism of adaptive events in most organisms (Pritchard et al. 2010; Zhang et al. 2013; Graves et al. 2017). It is particularly hard to detect these signals in humans due to incomplete knowledge of their genetic architecture and weak selection signals of individual loci. To overcome these difficulties, Zhang et al. in 2013 utilized a combination of population subdivision (F_{ST}) and branch analysis on 38 complex traits with at least 15 index SNPs to determine if there was evidence of directional selection in this set. The traits fell into seven categories: quantitative physical traits, quantitative physiological traits, inflammatory or autoimmune disorders, mental disorders, cancers, and miscellaneous traits related to menstruation. They concluded the following points: first, that natural selection on standing variants associated with complex traits is common in humans; second, that signatures of selection for any particular trait that is enriched in different human lineages indicating recent adaption are most likely associated with *temporary and geographically specific environmental challenges* (e.g., malaria adaptation); there is a strong correlation between the effect sizes and the strength of selection (obvious examples are height, urate level—physiological trait); for traits without correlation between effect size and the estimated strength of selection (34/38 of the traits they studied), the selection on the trait is acting through selection on a correlated trait. Their two final conclusions have relevance to whether we should expect consistent directional selection for any complex trait. Their data indicated that the genetic response to natural selection (change in allele frequencies) could be highly driven by chance, meaning that we should not expect any consistent or predictable changes for allele frequencies. This is also since the evolutionary forces impacting any specific trait are heterogeneous (multiple mechanisms operating at the same time.) These conclusions are entirely consistent with my assertion that it is highly unlikely that directional selection acted consistently on any traits that made Africans generally less capable than Europeans, no matter how defined. Indeed, the greater genetic diversity of Africans would argue for the exact opposite result.

4. Word on the Street Says . . .

If the word on the street says Africans are not genetically sick, why is health disparity so prevalent? The answer to this is as obvious as you might think, oppression (in this case, structural racism) makes people sick (Graves 2005b, 2015; Graves and Goodman 2022). To understand how it makes people sick, one must have a correct theory of disease. As surprising as that sounds, many practitioners within biomedical research and clinical practice do not have such a perspective. This again results from the fact that they do not appreciate how ultimate (evolutionary) and proximate (physiological, cellular, and molecular) mechanisms interact. Thus, evolutionary medicine identifies seven classifications of disease (Stearns and Medzhitov 2016). Structural racism can differentiate and exacerbating the impacts of these mechanisms.

4.1. Diseases with Genetic Causes

Socially defined race and biological variation operate differently depending upon whether the underlying genetic mechanisms of the diseases are caused by single genes of

high penetrance or are influenced by complex genetics. It is the latter type of causation that has been the primary concern of health disparity (heart disease, cancer, stroke, metabolic disease, psychiatric disorders). Clearly, if humans do not have biological races, there can be no genetically based racial differences in the prevalence of diseases caused by high-penetrance alleles. On the other hand, the prevalence of alleles associated with such diseases such as cystic fibrosis, sickle cell anemia, thalassemia's, and severe combined immune deficiency (SCID) can differ dramatically in populations across the world, but they do not differ in a way consistent with social racial classifications. For example, α - and β -thalassemia range from southern Spain, through the Mediterranean, Eastern and Southern Africa, through to New Guinea. There are over 500 variants associated with these diseases. The elevation of their frequencies is thought to result from their role in resistance to malaria via heterozygote advantage (Shang and Xu 2017). It is also clear that specific populations have different frequencies of the possible α - and β -thalassemia variants, but similarly to sickle cell anemia variation, their distributions do not correspond to socially defined race (López et al. 2010).

Genetic drift can also play a role in dramatically altering disease predisposition variants in populations. Mutations in the genes that encode *RAG1* and *RAG2* are one cause of severe combined immune deficiency (SCID). The prevalence of this condition in the general population is 1/1,000,000 but it is 1/2000 in the Navajo and Apache nations. Similarly, in SCIDA (Athabascan-type), a nonsense mutation in the *DCLRE1C* gene abrogates the function of a protein (Artemis) involved in V(D)J recombination and DNA repair (Li et al. 2002). Additionally, in SCIDA, a nonsense mutation in the *DCLRE1C* gene abrogates the function of a protein (Artemis) involved in V(D)J recombination and DNA repair (Li et al. 2002). The 1/1,000,000 frequency of affected individuals indicates that the frequency of this variant in humans is determined by mutation–selection balance, however it elevated many orders of magnitude above its expected frequency in the Navajo and Apache nations. This resulted from genetic drift due to genocidal action by the United States against these tribes. From 1863–1868, the Navajo population was interned at Fort Defiance in Arizona from which multiple treks of 250–400 miles (depending on the route), including “The Long Walk”, were made to Fort Sumner in New Mexico territory (Kwan et al. 2015). Approximately 10,000 Navajo began the journey, but many died on the way or at the New Mexican camp (<https://americanindian.si.edu/nk360/navajo/long-walk/long-walk.cshtml>, accessed on 15 October 2021). It is thought that about 8000 survived. From that small population, the Navajo now number about 330,000. The Jicarilla Apache declined to a population of approximately 600 in the early 20th century and now only number about 3,300. For more information on American Indian history in the United States, see <https://www.nlm.nih.gov/nativevoices/timeline/index.html> (accessed on 15 October 2021) (Dunbar-Ortiz 2014).

4.2. Diseases with Environmental Causes

Throughout much of the existence of our species, infectious disease has been a significant cause of morbidity and mortality. The environmental cause is due to a wide variety of pathogens that infect humans: viruses, bacteria, and unicellular and multicellular eukaryotes. Human activity has played crucial roles in creating the environments that make pathogens so successful. For example, it is thought that malaria evolved the capacity to infect humans due to both the domestication of animals and the crowding of humans into settlements, making transmission easier to accomplish (Hedrick 2011, 2012). These conditions worked the same way in facilitating viral and bacterial transmission. What is not generally understood is how social conditions of oppression played a role in differentially visiting pathogen transmission in various communities throughout human history. Some of the best-known cases of this were the transmission of smallpox by Europeans to Amerindian populations in the 17th century (e.g., 1616 smallpox in New England). These epidemics had both a genetic (lower genetic HLA genetic diversity in Amerindian populations via founder effect) and a social component (disruption of Amerindian societies due to

warfare against them). In another example, during American chattel slavery, even though enslaved West Africans had various antimalarial adaptations within their populations, they still died differentially from malaria due to exposure, poor nutrition, and overwork resulting from their enslaved condition (Savitt 2005). The early differential in COVID-19 infections amongst African Americans and Latino/a's in the United States was driven by environmental conditions such as crowding and inability to work from home (Abbasi 2020).

Environments also play a key role in complex disease. In 2019, the leading complex biological causes of death in the United States were heart disease, cancers, respiratory disease, and cerebrovascular disease (Centers for Disease Control 2019). Heart disease in economically privileged nations is the poster case for environmental mismatch. Well-known environmental and behavioral risk factors include poor diet, sedentarism, ambient air and noise pollution, sleep deprivation, psychosocial stress (which are primarily environmental); as well as body composition, cardiovascular fitness, muscle strength, and functionality of the intestinal microbiome (which may be more influenced by genetic factors). There is strong evidence that psychosocial stress resulting from structural racism differently predisposes African Americans to heart disease (Churchwell et al. 2020). Recent studies have shown greater levels of CpG methylation in promoter regions of metabolic genes in heart muscle associated with heart muscle failure differentially by socially defined race and lower socioeconomic status (Pepin et al. 2021). Thus, the historical presence of structural racism in America (both a social and physical environmental factor) is consistent with the long-standing disparity in heart disease by socially defined race.

4.3. Diseases of Homeostasis

Diseases of homeostasis affect physiological systems that were designed to be plastic and adjustable. As such, these have both genetic and environmental causes and are particularly revealed by evolutionary mismatch. These are often the result of inappropriate adjustment of homeostatic set points. Phenotypic plasticity is the developmental counterpart of adjustable homeostatic set points (Stearns and Medzhitov 2016). Both these mechanisms allow for the flexibility required to deal with variable environments. However, both mechanisms allow for physiological systems to be fixed in the wrong state. Diseases of homeostasis include addictive behavior, obesity, type-2 diabetes, atherosclerosis, hypertension, and cardiovascular disease.

4.4. Diseases Resulting from Lack of Maintenance

The evolutionary basis of aging is conserved in all animals, and this results from the declining force of natural selection on the age of the adult soma (Medawar 1952; Williams 1957). The population genetic mechanisms consistent with the evolution of aging are antagonistic pleiotropy (AP) and mutation accumulation (MA). Variants governed by AP should be fixed in all human populations, whereas MA should allow for some variation specific with ancestry. The proximate mechanisms associated with aging are multiform, occurring at the physiological, cellular, and molecular levels (e.g., wear and tear, hormonal controls, oxidative stress, somatic mutation, DNA methylation; Graves 1997). Diseases that result from lack of maintenance include neurodegeneration, cancers, osteoporosis, cardiovascular disease, type 2 diabetes, and arthritis.

Similarly to diseases of homeostasis, the prevalence of these conditions will be influenced by genetic and environmental factors. It is highly unlikely that differences in prevalence that we see in these conditions between socially defined racial groups are driven by genetic differences. For example, the rise in the prevalence of allergies and asthma has increased across the industrialized world in conjunction with changes in hygiene. Studies of pediatric asthma have examined risk factors associated with socially defined race. Hughes et al. (2017) found that material hardship (poor housing quality, housing crowding, lack of amenities, absence of a vehicle) were risk factors explaining asthma diagnosis and emergency room visits. Genome-wide analysis for asthma risk variants have identified different loci in African- and European-Americans (11q21 in African Americans and 6p21

in European Americans; [Xu et al. 2001](#)). A more recent study found that the percentage of African ancestry was a predictor of the prevalence of nocturnal asthma ([Levin et al. 2014](#)). However, it should be noted that this study did not examine any possible covariates with African ancestry such as SES, or material hardship, which is differentiated in this country with percentage of African ancestry. So once again we are left with the overarching question of the lack of environmental equity complicating any analysis of genetic causation associated with this byproduct of defense condition.

4.5. Diseases from Stochastic Developmental Problems

The fact that these conditions result from chance mutations suggest that they are not racialized. For example, Turner Syndrome results from a deletion of an X chromosome resulting in the karyotype (45, X). Individuals with this condition generally display female external genitalia and internal ducts, but rudimentary ovaries. Other phenotypes associated with this karyotype are short stature (under 5 feet), a webbed neck, and a broad shield like chest. As the reproductive potential of these individuals is severely reduced, the frequency of this trait in populations would be governed by mutation/selection balance. Thus, we would expect that in stable populations the frequency should be roughly that of the mutation rate. In Canadian populations, the frequency of Turner syndrome was 1/2500 live female births ([Bouet et al. 2016](#)). Genetic drift can alter the frequency of stochastic developmental phenotypes. For example, the frequency of intersex karyotypes in native African populations within South Africa is elevated compared to other African populations ([Wiersma 2004](#)).

4.6. Diseases Resulting from Maternal–Offspring and Maternal–Paternal Conflicts

The equilibrium of maternal–offspring and maternal–paternal conflicts occur at the species level. For example, invasive placentas are a primitive trait of placental mammals. The hominids have particularly invasive placentation compared to nonhuman primates ([Stearns and Medzhitov 2016](#)). Human fetuses can increase their provisioning via expanding spiral arteries and increasing maternal blood pressure. If this equilibrium is disrupted, this can result in dangerously high blood pressure in the mother. The condition preeclampsia is characterized by a rise in blood pressure during pregnancy that is also associated with signs of damage to other organs. In this case, placental blood vessels are often abnormally narrow, resulting in increasing blood pressure. The prevalence of preeclampsia in the United States was 2–5% of live births in 2008. However, there was a notable disparity between African- and European-American women (49.2/1000 compared to 44.0/1000). Given the complexity of the physiological mechanisms involved in this condition, genetic, environmental, and chance effects clearly play a role. A recent meta-analysis examined 9515 cases and 157,719 controls of Eurasian descent for genomic variants associated with risk of preeclampsia ([Steinthorsdottir et al. 2020](#)). They found five SNPs at $-\log_{p10} > 8$ of the 11,796,347 examined that accounted for ~0.67% of the variance in preeclampsia risk. It was suggested that these genetic effects impacted maternal hypertension and BMI. Results such as this do not make a strong case for strong genetic causation of health disparity between African- and European-American women. On the other hand, studies of psychosocial stress differences between these women and their association with differential rates of this condition are far more promising ([Giurgescu et al. 2015](#)). Psychosocial stress activates the HPA axis inducing the secretion of corticotrophin (CRH). This in turn is associated with systemic inflammation which is a known risk factor in preeclampsia (which is also a leading cause of premature birth.) Numerous studies have shown that African American women face greater levels of psychosocial stress than European Americans ([Giurgescu and Misra 2018](#)). However, at least one study has shown that controlling for psychosocial risk reduces but does not eliminate the difference in preeclampsia incidence in these groups ([Grobman et al. 2018](#)).

5. The Last Word

My birth certificate lists my race as “colored.” I was born in the mid-1950’s. It is not clear whether my mother filled in the form, or the nurses handling my birth did so. At that time, the official word was still “Negro.” As a child, I could not have known that my life chances were still being determined by the simmering boil of American racism. Like most African Americans, this was determined by my physical features demonstrating obvious African ancestry. While I have never had my ancestry tested by a DNA ancestry company, I fall right around the partitioning of ancestry found in most persons who are descendants of mid-Atlantic chattel slavery (11/16 African, 4/16 European, 1/16 Amerindian, as determined by family oral tradition). In America, the non-African portions of my ancestry (whatever they are) never mattered. On the street I was colored, Black, Afro-American, and now African American. This street identity worked against my progress in the academy—I discuss this in my forthcoming work: *Voice in the Wilderness* (Graves 2022). My career came into existence and persisted against all odds. I was the first African American to earn a PhD in evolutionary biology, and the first person to consistently problematize health disparity from an evolutionary perspective. I had dedicated my career to not being the last African American in the field, which is now just beginning to realize the impact of my labors. All the work I have done is for the streets where I was born and nurtured my struggle. The same vicious streets that continue to conspire to kill my people daily.

At least now we have a better understanding of why and how this happens. With this knowledge we are also prepared with a tool to stop the ongoing carnage. It’s not rocket science: eliminate racism, and health disparity will disappear. This is accomplished by amazingly simple methods: provide people with quality childcare, education, universal health care, and meaningful employment. Without these kinds of changes, studies designed to outline the genetic causes of disease will always be compromised, due to the lack of equal environments that racialized subjects inhabit. I predict that equalizing racial environments will do more to reduce health disparities than any other intervention. These are all things that everyone should be willing to accept, but to implement them requires calling into question the socioeconomic system (capitalism) that prevents these things from coming into existence. This is the challenge that faces those who will walk the future streets of our world. In *Racism, Not Race* (Graves and Goodman 2022) we argue that the path to an antiracist future cannot be divorced from making fundamental changes in our political/economic system. At the end, it comes down to what streets you want to walk, ones that target people by their socially defined appearance, or those which are free for all.

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