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EDITORIAL

The “African gene” theory: it is time to stop teaching and promoting the slavery hypertension hypothesis

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[A] very attractive case can be constructed, all based upon a genetic defect in sodium excretion that is more prevalent among blacks. Perhaps blacks, who originally lived in hot, arid climates wherein sodium conservation was important for survival, have evolved the physiologic machinery which protects them in their original habitat but makes it difficult for them to handle the excessive sodium they ingest when they migrate (41).

During a May 2007 Oprah show, Dr. Mehmet Oz asked Oprah, “Do you know why African-Americans have high blood pressure?” Oprah promptly replied that Africans who survived the slave trade’s Middle Passage “were those who could hold more salt in their bodies.” To which Dr. Oz exclaimed, “That’s perfect!” (64, 71). According to Dr. Oz and Oprah, African-Americans today are afflicted by hypertension at higher rate than whites because of genes passed on by their ancestors, genes that favored salt retention and that, in turn, cause high blood pressure (Fig. 1) (71). Dr. Oz and Oprah were referring to the “slavery hypertension hypothesis.” The slavery hypertension hypothesis has gained prominence in the popular press and is often cited (9, 23, 28, 41, 42, 53, 74). The hypothesis is also highlighted in medical textbooks and taught in many medical schools (15, 38). However, we posit that the theory is not supported by the data and so should not be taught or promoted (15, 43– 45).

The Slavery Hypertension Hypothesis

Could an exaggerated hypertensive response to salt be related, for example, to selective effects in blacks of slavery? First came the forced recruitment of Africans from central, low salt-use areas; subsequently came a selective wastage from heat stress and salt and water deprivation during the brutal voyage across the sea. Among survivors, likely those most fit to withstand the acute stress, there followed an abrupt exposure to a poor-quality diet with heavy salting on which the southeastern American slaves subsisted. Salt-saving renal-adrenal adaptations to a low salt environment, and selection, would be, by this idea, overwhelmed in the new salt-rich environment. Excess pressor responses to sodium might result not only from

these sorts of selection processes, but from the stress of social dissonance among the slaves (8).

Interest in understanding the mechanism mediating the high prevalence of hypertension among African-Americans began in the 1960s and 1970s. Early investigators posited that blacks

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were more likely to suffer from a genetic mutation affecting natriuresis (29), because the hot and arid environment and limited salt supplies of Africa selected for sodium retention (33). This innate ability for sodium retention, however, would become maladaptive in other settings (33, 82, 83).

Thus the slavery hypertension hypothesis began by claiming that individuals living in the hot and arid African climate were subjected to limited water and salt supplies and bouts of dehydration. As a result, a genetic predisposition for salt retention evolved in individuals of African descent. A higher sodium-retention mechanism enhanced survival during these extreme weather conditions, but the same mechanism would lead to hypertension (26, 67, 84). However, the idea that African populations had limited access to salt came under intense scrutiny (15). Accordingly, the hypothesis evolved, suggesting that during the forced migration of African slaves across the Atlantic from West Africa to the New World, high mortality due to vomiting, heat, diarrhea, and dehydration selected for surviving slaves who could conserve water and salt. On reaching American plantations, similar severe conditions of low water, intense work, and profuse sweating exerted additional selection pressures for water and salt conservation (84).

Concerns Regarding the Slavery Hypertension Hypothesis

Wilson and Grim (84) published only a single peer-reviewed scientific paper on the Slavery Hypertension Hypothesis in 1991. As noted by Kaufman and Hall (45), the majority of the written work on the slavery hypertension hypothesis is limited to conference reports and nonreviewed abstracts and book chapters. Kaufman and Hall also noted that, in the textbook published by Fray and Douglas, the chapter on the slavery hypertension hypothesis contains 10 citations (28, 45), 4 of which are abstracts, and 3 of which are books or book chapters. Despite the limited data, the theory remains popular (64) and is discussed in many hypertension textbooks (9, 23, 27, 28, 41, 42, 53, 67, 74). Even today, a statement on the American Heart Association website reads, “Researchers have also found that

there may be a gene that makes African-Americans much more salt sensitive.” (3). A statement on the WebMD website also supports the idea of whites and blacks as biologically distinct by asserting, “High rates of high blood pressure in AfricanAmericans may be due to the genetic make-up of people of African descent” (78). Moreover, the Center for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion’s fact sheet notes, “Researchers think there may be a gene that makes African-

leagues (14) were unable to find differences in the blood pressure response to salt by race, age, sex, or body weight. Thus salt sensitivity is not a racial problem, but rather a human problem, and the generalization that blacks are salt sensitive and whites are not should be discarded (14). It is important to note that measurements of salt retention in humans have come into serious question (50). Specifically, 24-h urinary sodium measurements are highly variable due to whole body salt sequestering, as well as simple measurement inaccuracy. Fatimah Jackson (37), a pioneering anthropologist, also challenged the slavery hypertension hypothesis based on population genetics, noting that a genetic “bottleneck” (a restriction in variability) imposed by many infectious causes may actually increase diversity. Specifically, the author wrote:

Fig. 1. Top: slave traders licked the face of captured Africans to test the salt content of the potential slave. It was postulated that individual with lower salt content would perish during the forced migration. Accordingly, only individuals with high salt content were enslaved [From Chambon (13); public domain.]

Americans more sensitive to the effects of salt, which in turn increases the risk of developing high blood pressure” (12). Similar genetic-based explanations for health disparities between whites and blacks are promoted by respected authorities on the worldwide web (20, 30, 62). However, pathophysiological, historical, and anthropological data strongly dispute the hypothesis. Specifically, regarding the pathophysiology of salt-induced hypertension, the results of sodium balance studies in humans and in animals consistently demonstrate that, in response to salt loading, normal salt-resistant subjects (individuals resistant to the pressor effects of salt)

retain just as much sodium as salt-sensitive subjects (individuals with a pressor response to salt). Despite retaining just as much salt as salt-sensitive subjects, saltresistant subjects do not develop hypertension (16, 17, 31, 32, 34, 36, 40, 46, 61, 65, 66, 68, 69, 79, 80). Thus salt retention is not the distinguishing pathophysiological response that separates salt-sensitive from salt-resistant subjects (47– 49, 58). Accordingly, even if African-Americans were genetically programmed to conserve more salt (which is highly unlikely), this alone would not elevate their blood pressure. Furthermore, available evidence suggests that the difference in salt-sensitivity between African-Americans and Caucasians (EuropeanAmericans) is significantly smaller than what the Slavery Hypertension Hypothesis suggests. In fact, Chrysant and col-

Without question, the stresses of the transatlantic Middle Passage represented a severe selective constraint on the Africans transported to slavery in the Americas. Yet, new evidence on the molecular, genetic, and biochemical effects of stress suggest that the very presence of this major constraint may have accelerated the subsequent accumulation of genetic diversity in the descendants of survivors. In addition to an increased rate of recombination, mutation, and transposable genetic elements, the survivors of this transatlantic holocaust and their offspring were subsequently exposed to new biotic and abiotic selective pressures in the American environment, to opportunities for gene flow with non-Africans, and frequently to the social conditions (i.e., isolation) for significant genetic drift. These new ecological factors may have further magnified the genetic variability of contemporary African-Americans. It is unlikely that the intense selective pressure for electrolyte conservation proposed by Wilson and Grim, although a significant constraint during the Middle Passage, can be shown to have persisted as a consistent feature of the more recent post-Middle Passage environments of African-Americans. Once the survivors were deposited in the Americas, they faced a diversity of new physical and biological challenges in various ecological settings. Under these nonuniform conditions, the descendants of these Africans not only survived but continued to evolve. The result has been an expansion of their collective genetic variability after the Middle Passage rather than a continued constriction of heterogeneity. With respect to hypertension studies among African Americans, their history suggests that, although they have been subject to often severe diversityconstricting events, they have also been exposed to diversity expanding phenomena as well. As such, it is unlikely that any single genetic event can be relied on to account for this group’s current apparent susceptibility to high blood pressure and essential hypertension. Rather, the phenotypic and genotypic diversity that characterizes this sociological construct suggests

that the Middle Passage was, more likely, a prelude to an expansion in subsequent variation within this highly heterogeneous macro ethnic group.”

Kaufman and colleagues (43– 45) have also questioned the hypothesis on many historical distortions, especially concerns regarding the reported 30% mortality rate during the Atlanticpassage (15).

The Tendency to Accept Genetic Explanations of “Racial” Differences

So many medical conditions are differentially distributed to African Americans— heart disease, diabetes, hypertension, low birth weight babies—are we to believe that Black people were so evolutionarily unlucky that they got all the genes that predisposed them to every malady? [Anthropologist Robert

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Dressler, phone conversation with Larry Adelman, April 22, 2001 (71)]

Studies dating back to 1932 (1), and continuing to this day, have consistently reported a higher prevalence of hypertension among African-Americans than Caucasians (11, 51, 59). The concern with these reports, as suggested by Dr. Oz and Oprah (64), is that many support the view of whites and blacks as fundamentally distinct in terms of innate biology or physiology. In this context, it is important to note that race itself is not a biological category. Rather, race is a social construction, defined by region-specific cultural and historical ideas rather than inherent biological characteristics (2, 24, 63). Accordingly, we should not consider racial groups as genetically homogenous. For example, numerous groups with African descent have low hypertensive rates (56). Furthermore, African-derived groups in South America experienced a great deal of genetic drift and incorporated a large proportion of Amerindian genes (54). In contrast, African-derived groups in North America incorporated a large proportion of European genes (54). Thus populations of African descent in the New World are varied genetically because of their own unique evolutionary trajectory (55–57). Accordingly, most genetic variation is found within, not between, ethnic groups.

Furthermore, although it is well accepted that ancestral alleles can affect disease rates and medication efficacy (5, 85), these alleles do not align with racial groupings. Specifically,

admixture and migration have produced broad variation so that race categories cannot substitute for genetic ancestry (6, 10, 21, 22, 25, 35, 39, 75), and there are no sharp boundaries dividing the human species, as gene frequency differences between populations found in one polymorphism are not paralleled in others (7, 55, 70, 76). The concern is that the tendency to represent race as biology may cause physicians to employ racial signifiers as clinically meaningful without full examination or understanding of their complex formation (18, 77).

Although the American Heart Association and the National Heart Lung and Blood Institute are making considerable efforts and investments into defining the role of “epigenetics” in the development of common forms of hypertension and to consider environmental factors (food, stressors of all kind, the air we breathe, etc.), and, most recently, the microbiome, which can clearly modify the expression of our inherited genome, the fact that there is vastly more research on genomic mechanisms than on the social determinants of health merits a national debate (52).

The Impact on Our Students

There is unequivocal scientific evidence that no matter into what racial category a person is placed, each of our complex physiological and behavioral phenotypes are determined by both the genes/alleles that we inherit and the environment into which we are placed, since our milieu alters the expression of these genes. It is not possible to categorize an entire racial group as being genetically or physiologically distinct from one another because of the vast genetic heterogeneity of our populations. This is especially true in the United States, given that there are few indigenous people surviving, and we are a nation of immigrants from everywhere, shaped by both our genes and environment.

Despite these truths, the slavery hypertension hypothesis provides a rationale for treating African-Americans as a group that had been genetically altered and transformed (45). The hypothesis supports our tribal bias (38, 52) for an AfricanAmerican inferiority that is innately pathological. This has a deep negative impact on our students. However, overcoming this bias and refusing to treat African-Americans as a genetically homogenous group is critical for our nation’s health because minorities receive less and lower quality healthcare than do Caucasians (4, 19, 72, 73). We should consider health disparities within the broader social context and consider social determinants, including the stress of racial discrimination (60, 63, 81). To do this, we should reject the premise of genetic determinism and essential black abnormality and not think of racial groups as fundamentally distinct in terms of biology or physiology.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

H.L.L. and S.E.D. conceived and designed research; H.L.L. and S.E.D. prepared figures; H.L.L. and S.E.D. drafted manuscript; H.L.L. and S.E.D. edited and revised manuscript; H.L.L. and S.E.D. approved final version of manuscript.

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