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## THE SLAVERY HYPERTENSION HYPOTHESIS—NATURAL SELECTION AND SCIENTIFIC INVESTIGATION: A COMMENTARY

*The Slavery Hypertension Hypothesis proposed by Wilson and Grim claims that African Americans possess genetic predisposition for life-threatening elevated blood pressure that was the consequence of intense selection during the Middle Passage and the period of enslavement. The biological changes resulted from a genetic bottleneck that was marked by mechanisms that conserved salt in those that survived the infectious diseases that caused diarrhea and vomiting. The hypothesis has gained prominence in the popular press, frequently cited in peer-reviewed articles and featured in medical textbooks that offer little genetic evidence to support the hypothesis. There is no indication of a genetic bottleneck or evidence of "racial" differences that are genetically determined. It is time to discard the myth of the Slavery Hypertension Hypothesis and begin to examine the issue from a biological and social perspective that reflects a more realistic approach to the disparities that exist in the prevalence of hypertension.*

**KEYWORDS:** *enslavement, hypertension, Middle Passage, salt conservation, genetic predisposition*

Hypertension remains one of America's most pressing health problems. The prevalence of hypertension among African Americans contributes to significant ethnic health disparities in the United States. Hypertension is the single greatest factor leading to cardiovascular disease in African Americans (Nesbitt and Victor 2004:24). Premature excess cardiovascular disease (defined as occurring prior to age 65) is a pattern in African Americans. The end-organ consequences of hypertension result in a higher incidence of stroke, increased renal disease that often leads to end-stage renal disease, greater risk of left ventricular hypertrophy, and higher risk of heart failure (Nesbitt and Victor 2004:24). African American women when compared to "White" women have twice the number of "productive years lost" (Nesbitt and Victor 2004:24). African

American men experience a fourfold greater loss of "productive years" (Nesbitt and Victor 2004:24).

Hypertension in Blacks remains "a puzzled to be solved" (Cooper 1991) and the sodium sensitivity and psychosocial stress hypotheses, the two most prominent hypotheses, are "not wholly convincing" (Cooper 1991:112). A genetic propensity for salt retention (De Wardener and MacGregor 2002; Gleiberman 1973; Kurokawa 2001; Palacios et al. 2004; Taubes 1998; Wright et al. 2003) has been suspected in hypertension in African Americans but not proven (Aviv et al. 2004; Forrester 2004; Joossens and Geboers 1983; Kurokawa 2001; Kurokawa and Okuda 1998; O'Shaughnessy and Karet 2004; Watanabe et al. 2002; Zoccali 2003). But even the speculation about a genetic basis for hypertension has been enough to fuel the search for factors that selected for metabolic changes for salt sensitivity in African Americans. Salt sensitivity is apparent in African Americans when ingesting a half teaspoon of salt; there is a rise in their blood pressure of as much as five points (Flack et al. 1991).

Lillian Gleiberman (1973) hypothesized that salt sensitivity may have been selected for in early Pleistocene hominids as they trekked across the savanna. The genetic hypertension hypothesis was developed further by Thomas Wilson in 1986 publications (Wilson 1986a, 1986b) that analyzed the role played by low salt supply in some areas of West Africa and a hypothesized natural selection for genes that resulted in salt conservation in West Africans. This genetic predisposition was then implicated in hypertension in African Americans who were exposed to a salt-rich environment. In 1991, T.W. Wilson and C.E. Grim (Wilson and Grim 1991: I-126) transformed Wilson's earlier hypothesis by arguing that New World enslaved populations were forced into the transatlantic slave trade, thrusting them "into a tragic environment" (Wilson and Grim 1991:I-126) in which they experienced high mortality and low fertility (Wilson and Grim 1991:I-126). The enslaved individuals who survived the "Middle Passage" underwent intense selective pressure "due to the possession of a special genotype" (Wilson and Grim 1991:I-126). Because of slavery, according to Wilson and Grim, "blacks in the western hemisphere" (Wilson and Grim 1991:I-126)

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may be different from “today’s blacks in Africa” (Wilson and Grim 1991:I-126). The high mortality during the Middle Passage followed by the period of enslavement was caused “largely by salt- and water-depletive diseases” (Wilson and Grim 1991:I-126) resulting in a genetic change in sodium metabolism (Wilson and Grim 1991:I-126). This “enhanced genetic-based ability to conserve salt” (Wilson and Grim 1991:I-122) is more prevalent among “western hemisphere blacks than among African blacks” (Wilson and Grim 1991:I-126) and may have subsequently resulted in a higher prevalence of hypertension in African Americans.

There is one assumption and three lines of evidence that are used to support their hypothesis. While Wilson and Grim (1991:I-125) cite Grollmann’s (1978) observation that the hypothesis is “impossible to verify,” they believed that the extensive information now available on the “biohistory of blacks” (Wilson and Grim 1991:I-125) made it possible to test the hypothesis. They started with an assumption that early Paleolithic hominids living in a hot and salt-depleted savanna environment evolved an ability to conserve salt (Gleiberman 1973). This scenario assumes that water was conserved because of excessive sweating (Newman 1970). First, “Blacks,” Wilson and Grim claim, are more sensitive to salt, with an enhanced genetic ability to retain sodium. Initially, Wilson and Grim saw this as a problem with “defective kidneys” (Wilson and Grim 1991:I-123) and “renal defects that could not excrete salt effectively” (Wilson and Grim 1991:I-123). Second, the high mortality during the Middle Passage was due to “conditions that ultimately kill by salt and water depletion” (Wilson and Grim 1991:I-125). Vomiting, sweating, and diarrheal diseases were causes of sodium depletion during the Middle Passage. Third, high mortality during the period of enslavement continued to select for sodium retention because of intensive heat and punishing physical labor (Wilson and Grim 1991:I-125–I-126).

Fatimah L.C. Jackson (1991) supported the argument for the selection for salt retention during the Middle Passage.<sup>1</sup> She believed that this intense selective mortality resulted in a genetic bottleneck (a constriction of existing genetic variability [Poston et al. 2001]) for salt conservation. She offers an interesting addition to the Wilson-Grim hypothesis by suggesting that the stress experienced by enslaved populations may have increased genetic variability. Using experimental evidence from *Drosophila* that suggests environmental stress as a mechanism for increasing genetic variation, she argues that there would have been a “burst” of point mutations and an increase in genetic recombination that would increase variability in populations that survived the Middle Passage. Even if there was this unlikely “burst” of diversity in enslaved populations, there should

be evidence of salt conservation in the descendants that survived the Middle Passage. A recent genetic analysis (Poston et al. 2001) in a sample of U.S.-born African Americans and African-born immigrants used alleles associated with hypertension risk (G-protein, AGT-235, and ACE I/D) to test for evidence of a genetic bottleneck in U.S.-born African Americans that would support the Slavery Hypertension Hypothesis. In fact, the research showed an association of the AGT-235 homozygous T genotype with African-born immigrants. This finding counters the notion of a bottleneck in the survivors of the Middle Passage.

The renowned historian Phillip D. Curtin (1992) provides a devastating analysis of Wilson and Grim’s logic as well as the demographics and historical data used to bolster their argument. Curtin (1992:1686) evaluates Wilson and Grim’s arguments by saying that the Slavery Hypertension Hypothesis lacks supporting evidence and “runs counter to what evidence we do have.” Curtin questioned Wilson and Grim’s use of “African American” as a biological marker (Curtin 1992:1681); their misuse of historical sources (Curtin 1992:1683); the size of enslaved population transported during the Middle Passage (Curtin 1992:1683); the cause and the rates of mortality during the Middle Passage (Curtin 1992:1684); the availability of salt on the slave ships (Curtin 1992:1682); the misinterpretation of his (Curtin’s) accounts of the slave trade, miscalculating the availability of salt in Africa (Curtin 1992:1682); and the failure to describe the salt trade (Curtin 1992:1682). Furthermore, Curtin notes that the popularization of this story (Diamond 1991) both selectively misrepresents the evidence (Curtin 1992:1684) from Wilson and Grim and presents the hypothesis as proven. Publications that use data to distort reality increase the gap between scientific and historical knowledge, and this concerns Curtin (1992:1686).

The popularization of the Slavery Hypertension Hypothesis (Kaufman and Hall 2003a, 2003b) is evident in how it was reported extensively in the newspaper, magazines (Diamond 1991), and review articles that appear in medical journals and textbooks. For example, Joel E. Dimsdale (2000) in “Stalked by the Past,” his presidential address to the American Psychosomatic Society, describes how our biological past influences the health of contemporary ethnic groups. Dimsdale (2000:164) uses the relationship between salt and slavery as one of his prime examples of how the past stalks the present. Building on his earlier research (Dimsdale et al. 1990) on salt ingestion in four groups (Blacks, Whites, normotensives, and hypertensives), Dimsdale and colleagues found that “salt sensitivity was found in only one group of individuals—black hypertensives.” While noting that the genetic basis for salt sensitivity

has reached little consensus, he nevertheless claims that there are “unique historical sources” (Dimsdale 2000:165) to support the Slavery Hypertension Hypothesis. Using the arguments derived from Wilson and Grim (1991), Dimsdale repeats the Slavery Hypertension story, saying that those who survived enslavement “would be less likely to sustain lethal electrolyte abnormalities” (Dimsdale 2000:165). While he describes the Slavery Hypertension Hypothesis as “sheer speculation because, to date, the frequency of alleles—at least for the renin-angiotensin system—is similar in Blacks from West Africa and the United States, nevertheless the hypothesis remains an intriguing one” (Dimsdale 2000:165). He then quotes a passage from *Moby Dick* describing sharks trailing slave ships (Dimsdale 2000:165), scavenging the bodies thrown overboard. He also describes a 1794 engraving in which a slave trader is licking the cheek of an “African captive” to taste his sweat (Dimsdale 2000:166). Dimsdale claims that this illustrates a test to determine if the sweat was salty, which would indicate an inability to conserve salt and making it less likely that the enslaved individual would be able to survive the Middle Passage. Dimsdale is quick to point out that these assertions are difficult to evaluate because of the “distance of time” but are suggestive “of a genetic legacy from the middle passage, a historical legacy accounting for increased salt retention and thereby accounting for increased salt retention and thereby increased salt sensitivity in blacks” (Dimsdale 2000:166).

Kaufman (2001) was quick to respond to Dimsdale’s results, saying that his analysis was a “careless repetition” of the old “slavery hypothesis” yarn. Dimsdale (2001) defends the details of his assessment, saying that his mortality estimates and the use of contemporary evidence from Melville’s description of sharks trailing slave ships support the validity of the “slavery hypothesis.” He admonishes Kaufman’s criticism as a politicization of the issue. Dimsdale states, “Race and ethnicity are too important to be ignored or politicized.” Lillian Gleiberman (Gleiberman and Dimsdale 2001) supports Dimsdale’s interpretation, saying it exemplifies the “ideas and principle of Darwinian medicine and it is important for these ideas to move into the mainstream of medical research” (Nesse and Williams 1994). While she sees the hypothesis as “intriguing,” she also notes that the genotype frequencies for the rennin-angiotensin system are similar for West Africans in the African Americans in the United States. If the African American descendants of enslaved populations went through the Middle Passage, they should show differences in genotype frequencies.

Kaufman and Hall (2003a) continued a frontal attack on the Slavery Hypertension Hypothesis. They

discuss in detail the “career” of the hypothesis from its presentation at national meetings in 1989 and 1990 to the single peer-reviewed publication (Wilson and Grim 1991) and two decades of publication in book chapters, textbooks, and popular press. They are especially concerned with the lack of experimental evidence that supports the Slavery Hypertension Hypothesis. In fact, there appears to be nearly an inverse relationship between the reports in the popular press and a definitive scientific finding that supports the Slavery Hypertension Hypothesis. Kaufman and Hall show how the language used in these popular treatments contributes to a notion that “Blacks” are inherently different by harboring genetic defects or physiological abnormalities (Kaufman and Hall 2003a). While terms such as “defective kidneys” (Wilson and Grim 1991:I-123) and “renal defects” (Wilson and Grim 1991:I-123) have been disavowed, Kaufman and Hall (2003a) describe titles in medical journals that present hypertension in Blacks as a different disease (Meggs 1985) and as “a paradigm of metabolic disarray” (Weir and Hanes 1996). Descriptions such as these essentialize race and present an image of Blacks as being fundamentally different in their genetic makeup and that this increases their risk for disease. The Slavery Hypertension Hypothesis reinforces “notions of genetic determinism” (Kaufman and Hall 2003a) and the “essential black abnormality” (Kaufman and Hall 2003a) that blames the victim and displaces economic or cultural factors from our understanding of the underlying etiology of the disease process.

Kaufman and Hall’s criticism that Grim and Robinson essentialize race was interpreted by Grim and Robinson as their being called racist (Grim and Robinson 2003). Racism was never used or implied in any of the discussion. In their continuing defense of the Slavery Hypertension Hypothesis, Grim and Robinson (2003) cite more demographic evidence that enumerates the mortality involved in the process of enslavement. While they claim to be testing a hypothesis, their approach reflects a strategy of advocacy to make their case by marshaling more evidence about the impact of the Middle Passage. A primary problem is that they avoid the essential point of defining a racial (genetic) difference in hypertension risk among African Americans. This point is brought home when they ask the question, “Do African populations metabolize sodium differently than Europeans or African Americans?”; they answer, “We do not know.” Any scientific test should begin with evidence that there is a genetic difference in salt sensitivity that is differentially distributed among African Americans before hypothesizing its cause.

Grim and Robinson claim that Kaufman and Hall are suggesting that any genetic research that would demonstrate group difference should cease. Following

Risch et al. (2002), they claim that categorization by race is a useful tool in biomedical research. While the use of race as a biological variable in biomedicine has been challenged (CDC 1993; Cooper et al. 2003; Geiger 1997; Goodman 2000; Pearce et al. 2004; South-Paul 2001; Williams and Warren 1994) and supported (Burchard et al. 2003; Dimsdale 2000; Polednak 1989; Risch et al. 2002), its use continues. One of the problems with analyses that demonstrate a disease risk in a racial group is that researchers “quit early.” They assume that race explains the risk and do not attempt to tease out the social and biological factors that underlie the association.

In his commentary on Kaufman and Hall’s discussion, K.M. Weiss (2003:124), borrowing from Swiss jurisprudence, says that “until given a rigorous test” the verdict on the Slavery Hypertension Hypothesis should be “judged plausible, but not proven by reason of doubt.” He acknowledges that there is abundant evidence that African American populations are more susceptible to hypertension than individuals in “other ethnic groups” are and suggests that this genetic hypothesis may “have the legs [it does] because at best there is no consensus that such patterns can be entirely accounted for by differences in environmental exposure.” The first step should be establishing a genetic relationship in salt sensitivity and hypertension in African American before perpetuating the Slavery Hypertension Hypothesis. It also begs the question why racial explanations are given primacy. Since the genetic evidence has been problematic, why not give “legs” to environmental factors in hypertension in African Americans?

Henry Blackburn (2003) notes that the “ethnic paradigm” has not been helpful in explaining population differences in hypertension and that “social epidemiological models remain confounded.” He comments that while the “Slavery Hypothesis of Hypertension” is speculative, it may not be any more speculative than looking at today’s health risks as the result of rapid change in face of the inertia built into the genetic legacy developed during the Paleolithic. Evolutionary medicine, Blackburn argues, provides a “fresh view of many common afflictions of modern society” (Blackburn 2003:118), and he implies that to be critical of that endeavor may inhibit innovative ideas. Darwinian or evolutionary medicine is not immune to criticism. The hyperadaptive perspective of Darwinian medicine often obscures long-term evolutionary processes by focusing on short-term adaptation (Armelagos 1997). When the evidence to support “just so stories” is shown to be inadequate, the importance of any evolutionary explanations will be minimized.

There is a political aspect to the debate. Blackburn (2003) says that there are differences in worldviews of scientist and physicians who see the uniqueness of individuals or races and those who see “vast commonalities” in *Homo sapiens*. Blackburn finds equating the uniqueness of an individual and a race to be a similar phenomenon. Races are arbitrary classifications that have limited explanatory power. Dimsdale (2001:325) see this as an even more political act and describes critics of the Slavery Hypertension Hypothesis as “left-thinking people.” Kaufman and Hall’s (2003b) rebuttal argues that the politicalization of the issue misses the point. They argue that they are trying to encourage scientific consensus, which should be based on “evidence rather than mere assertion.” Blackburn (2003) suggests that we need to cool the “temperature” and lower the “temper” to develop some resolution to the problem.

Cooper (1991:112) notes that blood pressure control is a highly redundant system that involves cardiovascular, renal, endocrine, and neural systems. Therefore, unraveling the ethnic component of such a complex system is a “daunting” undertaking. It may be time to take on the “daunting” task of unraveling the genetic and social factors of hypertension in African American and other groups. Discarding the Slavery Hypertension Hypothesis may be the first step in our efforts to understand this important health disparity. If genetic racial differences are found in African Americans with respect to a “hypertension” gene, then the Slavery Hypertension Hypothesis can be raised from its grave.

#### NOTES

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1. Kaufman and Hall claim that Jackson is critical of Wilson and Grim’s hypothesis. In actuality she supports the hypothesis by claiming a bottleneck occurred, but she suggests that the bottleneck was obscured by her dubious argument that stress-induced genetic change increased the variability in African Americans.

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