Biohistory of Slavery and Blood Pressure Differences in Blacks Today

A Hypothesis

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Genetic factors are known to play an important role in the variations in blood pressure levels. However, genetic factors that explain the higher average blood pressure levels of western hemisphere blacks when compared with African blacks have not been seriously considered. Because the genetic makeup of a population is largely determined by biological and ecological forces in the past, an examination of the biohistory of blacks, specifically the slavery era, was conducted. An overview of the salient findings of that investigation is included in this article. The published historical evidence on the transatlantic slave trade and New World slavery (from the 16th century to the 19th century) reveals that conditions existed for "natural selection," and therefore, genetic changes were virtually inevitable in the slave populations. During this period of history, mortality was extremely high, and fertility (or reproductive success) was so low among the survivors that most plantation societies in the western hemisphere depended on a constant importation of captives (over 12 million) from Africa for the viability of the plantation communities. Because the major causes of death were salt-depletive diseases such as diarrhea, fevers, and vomiting, it is argued that individuals with an enhanced genetic-based ability to conserve salt had a distinct survival advantage over others and were, therefore, more likely to bequeath their genotype to subsequent generations of Western hemisphere blacks. Thus, it is predicted that blacks in the Americas have a greater frequency of individuals with an enhanced genetic-based ability to conserve salt than African blacks. Moreover, it is surmised that this trait is partially responsible for the higher incidence of hypertension today among western hemisphere blacks than among African blacks. Ways of testing this hypothesis are suggested by the authors. (Hypertension 1991;17[suppl I]:I-122-I-128)

The distribution of blood pressure is different in black communities in Africa than in black communities in the western hemisphere. In his 1984 article on the world epidemiology of hypertension in blacks, Akinkugbe emphasized that although there have been at least six low blood pressure populations reported in Africa, none have ever been reported in the western hemisphere. In this review he also wrote that "in both men and women, mean systolic and diastolic arterial pressures were generally lower in Nigerians than in comparable black populations in the United States and the Caribbean." Indeed, at least three compilations of blood pressure studies have reported that the average blood pressure is higher in western hemisphere black populations than in the black population in Africa.

Most of the conjectures for blood pressure differences between black populations center around environmental factors such as nutrition and psychosocial stress. The failure to discuss genetic factors is due, no doubt, to the overriding concern with higher levels of hypertension and higher blood pressure among blacks when compared with whites, and if genetic factors are discussed, they are usually in reference to white-to-black differences, rather than black-to-black differences. However, familial and twin studies reveal a strong genetic component to blood pressure variability in black communities in the West Indies and the United States. Some of this variability could be due to genetic factors.
Outside the hypertension research field, genetic differences among geographically separated black populations are well documented. For example, in sub-Saharan Africa there are numerous groups differentiated by genetic markers; in addition, there are major blood group differences between West African blacks and American blacks. Some of the blood group differences are explained by admixture with European or Amerindian gene pools, but some differences are thought to be due to natural selection processes. It is possible that some of this variability of blood pressure between populations is due to genetic variations in the sensitivity to environmental factors. Thus, we agree with Akin-kugbe that “as the search for causal factors goes on [for hypertension], we must be cognizant of the role of genetic factors as well.”

This article is an overview of salient historical/epidemiological materials of the slavery period of western hemisphere history and well-accepted physiological concepts of fluid and electrolyte metabolism. The authors hypothesize that genetic differences between blacks in Africa and those in the western hemisphere are related to the higher blood pressure in the latter group.

Salt, Hypertension, and Heredity

Although the role of salt (NaCl) in high blood pressure is controversial, there is strong evidence that the blood pressure of some individuals, even some populations, may be sensitive to the sodium portion of the molecule. The blood pressure of American blacks has been shown to be more sensitive to increases of dietary salt than that of American whites. Furthermore, American blacks retain an intravenous sodium load longer than whites, and black hypertensive individuals are more likely to normalize their blood pressure with salt-excreting diuretics than are their white counterparts. All of these observations suggest that American blacks have an enhanced ability to retain salt, which affects blood pressure when compared with American whites. Unfortunately, no studies have been published comparing African blacks with western hemisphere blacks, but since the blood pressure of African blacks is closer to American whites than American blacks, salt-retentive abilities among African black populations may be similar to American whites as well.

To explain this salt-retentive ability, several researchers have suggested that “blacks” (presumably meaning American blacks) may have inherited a “renal defect in the handling of sodium.” In most people, with or without this defect, an influx of sodium will result in a temporary increase in arterial blood pressure. This rise in blood pressure triggers the kidney to increase sodium excretion, a process termed “pressure-natriuresis,” until a new steady state is achieved. In this new equilibrium, sodium excretion again equals sodium intake, but in those with a “defective” kidney, the blood pressure is notably higher than before and causes, in the nomenclature, a “resetting” of the pressure-natriuresis curve. This “renal defect” (a term that we will qualify below) in the handling of sodium may be under genetic control.

A rat model developed by Dahl et al demonstrated that only three generations of great “selection pressure” that was based, in part, on sodium intake was enough to produce severe hypertension in all future generations of rats when their diet contained high levels of sodium. Rapp et al have recently demonstrated that this sensitivity to salt is associated with inherited variations in the renin gene. The experiments of Dahl et al were classic examples of genetic changes, or evolution, on the systems controlling sodium homeostasis that were induced by selection related to sodium intake. Although this was due to experimental, or artificial, selection, selection pressures influencing sodium metabolism may have also occurred in the biohistory of humans.

More than 20 years ago, Helmer offered an evolutionary explanation for the variability of blood pressure and sodium retentive abilities in the human species. He speculated that sub-Saharan African populations undoubtedly experienced high salt losses from sweating (sweat can contain over 80 mmol Na+/L) and thus adapted genetically to their environment. The specific adaptation would be an enhanced ability of the kidney to retain or conserve salt.

Of course, as stated before, such a trait would be disadvantageous in today’s world of high salt intake because it may contribute to high blood pressure; however, under conditions of a high salt output in sweat, the same trait would be beneficial to survival and reproduction. Since Helmer’s provocative idea, Gleiberman added the possible influence of low dietary salt on the evolution of salt conservation. Since then, others have discussed this African-based biohistorical hypothesis of the etiology of hypertension. In 1983, Blackburn and Prineas suggested that the transatlantic slave trade voyage may have induced further selection. All of these authors followed Helmer and Gleiberman and focused only on excessive salt losses through sweating or low salt consumption, thereby ignoring the most dramatic source of salt loss: that in diarrheal stools or vomit, a fact that Grim first alluded to in connection with the “slavery hypothesis of hypertension.” Although normal stools contain only 2.5–5.0 mmol Na+/day, diarrheal stools average about 100 mmol Na+/L, and vomit can contain up to 60 mmol Na+/L. Taking these tremendous potential sodium losses into consideration, as well as modern evolutionary theory, a clearer statement of this genetic-environmental interactive theory of the etiology of salt-sensitive high blood pressure would be as follows: An enhanced genetic-based ability to retain or conserve salt would increase in populations in which reproductive success (fertility) is decreased by high mortality from Na+ losses in sweat, stools, or vomit. In other words, in historical human ecologies with higher salt losses than salt intake, differential mortality based on one’s genetic-based ability to retain salt (genotype) would
cause differential fertility (i.e., the ability to transmit one's genotype to offspring) and, therefore, natural selection. Put plainly, the variability of salt retention or salt conservation (and blood pressure) among human beings today may be due to natural selection in the past.

Although this hypothesis was considered intriguing, it was also, in the words of Grollmann,25 “impossible to verify.” We disagree with Grollmann; our knowledge of the extensive material available on the biohistory of blacks makes it possible to test the hypothesis against the historical record.

**Slave Death Rates**

A historic era in which mortality was substantial and natural selection was possible was the slavery period of history (from the 16th to the 19th century). This period included the transatlantic trade in African captives and the institution of slavery in the western hemisphere plantation economies. During this period, more than 12 million young men and women were imported into the Western hemisphere from their homeland in Africa.33 The intended destination of the captors was the North or South American continent or the Western Indies, and most who came to the United States and the West Indies departed from West Africa.44 However, many never arrived, because they died first. Of those who did disembark from the ships in the New World, many died within a few years. Thus, only a few Africans captured in the interior of Africa survived long enough to bequeath their genotype to subsequent generations.

Using the extensive quantitative records available, 20th century historians have attempted to estimate the number of people who died as a result of slavery. Current estimates suggest that the average death rate in transit on the transatlantic “middle passage,” the most carefully studied part of the slave trade, was about 12–15%. The death rate from the point of capture to delivery on the coast was about 10%, and mortality during confinement along the African coast about 12%. Thus, about one of three Africans who were captured in their homeland never set foot in the western hemisphere.35 Of those who disembarked from the ships, 10–30% or more did not survive the first 3 years of their slavery; they never became, in the typical words of the slave master, “seasoned” to their new environment.36 Moreover, mortality remained excessive during the postseasoning period as well.37,38

It was said that “if a Negro lasted a certain time his death was accounted nothing. This time was fixed at seven years by some planters, by others at less.”39

While mortality rates were high, reproduction success rates were low. Infant mortality rates of approximately 500 per 1,000 were partially responsible for the lack of “natural increase” (i.e., births exceeding deaths) of most populations.40,41 Of course, those who could have left descendants were those who survived the seasoning process (conservatively estimated at ~40–50% of those who left Africa).

The high death rate and low fertility rate made it necessary to continue the transatlantic slave trade in most slave societies. The new arrivals from Africa, of course, also experienced selection pressure. This tragedy was repeated year after year, century after century, and unless these deaths occurred randomly, natural selection was virtually inevitable throughout the entire slavery period. Of course, this probable selection would have a profound effect on the genotypes of future generations of western hemisphere blacks. The key question for this hypothesis becomes: How were the survivors of slavery different from those who died? Or, in other words, what traits were selected?

**Cause of Death of Slaves**

An examination of the causes of death during the transatlantic slave trade and of slaves on the plantations reveals that mortality was often due to conditions that ultimately kill by salt and water depletion (i.e., volume depletion).

The slave trade itself was characterized by “horrible” sweating, vomiting, and diarrhea. Chroniclers of slave expeditions speak of heat and excessive sweating on forced marches from inland Africa to the coast and in the confined, unventilated barracoons along the coast and in the ship holds of the middle passage.42-43 On the slave ships, vomiting due to seasickness was common,44 and the most common causes of ileal (the “flux”) and febrile diseases, both salt depletors.45 A dramatic example of the disastrous results of diarrhea (dissenterie) is revealed by the surgeon’s journal of the French slave ship Le Jeune Louis, an example of the kind of historical document that is available for biohistorical research46 (see Figure 1). On the plantations, excessive salt losses continued: sweating from hard work in the fields was a constant salt drain, and a major cause of death in the plantation societies continued to be diarrheal

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**FIGURE 1.** This is a photograph of pages 6 and 7 of the log of the 1824–1825 “African trade voyage” of the French ship Le Jeune Louis. The first four lines on the left page indicate the final African captives (five males and two females) who were loaded aboard ship before departure for Cuba on April 19, 1825. This made a total of 385 who had been loaded on board since January. The log also indicates that the cause of the deaths (mort) in a great majority of the cases was diarrhea (dissenterie), a condition that results in severe sodium losses. We surmise that the survivors were also in contact with the causative agent of the dissenterie and that one's ability to conserve sodium would offer protection against fatality from the disorder. Before the voyage ended on June 14, 1825, in Havana, Cuba, a total of approximately 30% (n = 113) of the captives died. An example on how to read the log: Glance down the first column until you see “22” (this is “April 22, 1825”) and then read across the row; this phrase can be easily translated as “3 female Negroes died from diarrhea.” Used with the permission of the Henry E. Huntington Library in San Marino, California.
disorders. In the 19th century, a new form of diarrhea, cholera, attacked the black populations in the western hemisphere. The several cholera pandemics that swept through the Americas and the Caribbean killed thousands of blacks. Thus, salt-depleting conditions and diseases seemed to be ubiquitous throughout the slavery period. It is important to note that the standard life-saving therapy for diarrhea is fluid and electrolyte replacement. Clearly, individuals with an enhanced ability of fluid and electrolyte conservation would have an advantage during diarrheal episodes. Professor Derek Denton, a leading expert on the physiology of salt, remarked:

It is worth noting that with infectious diseases involving electrolyte loss, the initial defence for survival will be in the area of biochemical and endocrinological regulation since the animal needs to withstand the impact for some days before immunological mechanisms become effective.

One measure that may have provided some relief during salt-depleting episodes was dietary salt. Unfortunately, at present there is not enough published information on salt intake to determine if the slave ship operators provided adequate salt supplies to their captives. Some sources suggest that salt was plentiful, but others wrote that only "a little salt sometime" was fed to the slaves. After landing in the New World, however, the major protein sustenance of slaves was either salted meat or salt fish, and salt was said to be the "only seasoning slaves saw regularly." One thing is certain: just like other food supplies, these salt provisions were in short supply during climatic disasters, wars, and famines.

Regardless of salt intake, however, individuals with an enhanced genetic-based ability to conserve salt (Na⁺ conservers) would have had a decided advantage over others under the severe salt-depleting conditions of slavery. Even if salt were readily available, the daily dose would be retained longer in the Na⁺ conservers than in others, thus offering protection from acute salt losses in stools, sweat, or vomit. We suggest, therefore, that one important difference between those who lived and those who died was an enhanced genetic-based ability to retain salt.

Thus, the kidneys of American blacks may be only defective in relation to sodium-induced high blood pressure in today's environment, but under conditions of excessive fluid and electrolyte loss, such as those that existed during the slavery period of history, the ability to retain sodium would be crucial to survival. Therefore, if this hypothesis is correct, it would be more accurate to state that American blacks simply respond differently, sometimes better and sometimes worse (depending on the circumstances), to sodium than do whites.

In summary, the evidence presented fits the requirement of a hypothesis: it accounts for a set of facts that can be tested by further investigation. More than 300 years ago, a forcible split occurred in the African black population that was the direct result of the transatlantic slave trade and New World slavery. Those Africans who became captives and, later, slaves were thrust into a tragic environment that was characterized by high mortality and low fertility. If survival under such selection pressure was due to the possession of a special genotype, the opportunity for striking shifts in the gene pool was present. Therefore, because of slavery, blacks in the western hemisphere today could be quite different genetically from today's blacks in Africa. Because the high mortality during slavery was caused largely by salt- and water-depleting diseases, we have suggested that one genetic difference between the two populations may relate to sodium metabolism. We hypothesize that an enhanced genetic-based ability to conserve salt is more prevalent among western hemisphere blacks than among African blacks and may, in part, explain the higher blood pressure levels in the former group.

**Future Research**

There are many opportunities to test aspects of this hypothesis today. Historical research should include a focus on the relation between salt supplies and mortality in Africa during the slave trade and in the slave communities in the western hemisphere. Extensive quantitative historical records on the amount of salt loaded on 17th century slave ships are available in the United Kingdom. These records should be accessed and then correlated with the known death records to test the role of salt supplies in mortality on slave ships. Similar records from the slave plantations are also available. Historical demographic data would also assist in predicting different levels of salt sensitivity in different western hemisphere black populations based on differential selection pressures in their past.

Future epidemiological investigations should systematically compare present-day black populations in Africa, with a focus on West Africa, to present-day black populations in the western hemisphere. Controlling for the confounding effects of admixture with European or Amerindian gene pools would be necessary. In addition, studies within present-day Liberia might be especially powerful because of the existence of African blacks as well as descendants of US slaves (transported to Liberia in the early 19th century). To address the role of environment, we would suggest migrant studies of West Africans to the United States and of African-Americans to West Africa. Finally, we encourage the search for low blood pressure black populations outside of Africa, because these populations may hold important clues to the roles of genes and environment in the variation in blood pressure in blacks worldwide. The key outcome measures in all of these studies would be blood pressure levels and the prevalence of sensitivity of blood pressure to dietary salt. We recommend that population-based studies follow the standardized protocol by the INTERSALT study. The salt sensitivity protocol would require careful standardization of
daily sodium intake and then induction of a standardized sodium-depletion and sodium-loading protocol such as the one developed by Grim and Weinberger.53 Of course, environmental factors should also be examined because high dietary salt intake44 or biobehavioral stress55,56 may affect sodium retention as well.

In conclusion, we have made the hypothesis, based on biomedical theory and biohistorical knowledge, that there is a strong likelihood of genetic differences that are related to sodium metabolism and blood pressure between blacks in African and those in the western hemisphere, and we have suggested ways to test (disprove) this hypothesis. It is likely that understanding the roles of genes and environment in black populations will lead to new insights into the causes of high blood pressure in all populations.

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