The Way They Live. 1879. Oil on canvas, Anshutz, Thomas P. (1851-1912).

THE SLAVERY HYPERTENSION HYPOTHESIS

A flawed explanation for its prevalence in African-Americans

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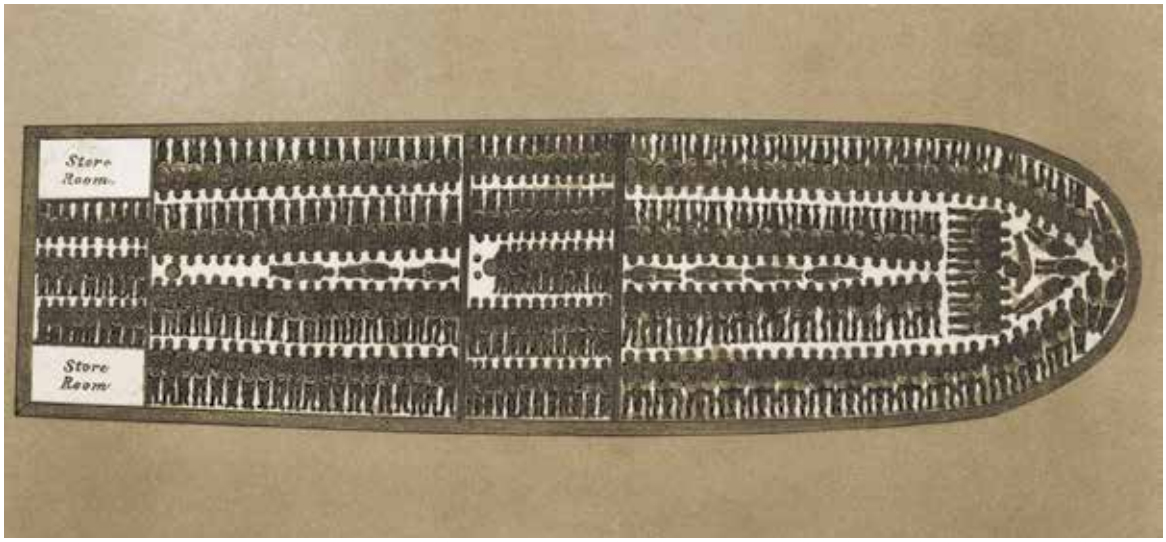
The slavery hypertension hypothesis was first proposed by University of Minnesota professors Henry Blackburn, MD, and Ronald J. Prineas, MD, PhD, in 1983,[1] and later refined by Thomas W. Wilson, PhD, and Clarence E. Grim, MD, in 1991.[2] It employed America's historical involvement in the Atlantic slave trade to explain a genetic foundation for the high prevalence of hypertension among present-day African-Americans.

Although the hypothesis was challenged in 1992 by Philip Curtin,[3] a leading historian on the Atlantic slave trade, and shown to lack corroboratory genetic support, it has continued to be prominent in the media, and cited in medical textbooks.

The hypothesis

Hypertension affects all racial and ethnic groups in the United States. According to the Third National Health Nutrition and Examination Survey, the prevalence of hypertension in the United



Lower deck of a ship with slaves packed tight. 19th century illustration. Photos/Getty Images

States in persons 18-74 years of age is 25%, with African-Americans exhibiting the highest prevalence (32.4%) among all racial groups listed.[4]

Hypertension in African-Americans has an earlier onset, and produces more target-organ damage than in other racial groups. Compared to Caucasians, African-Americans with hypertension suffer higher mortality rates from stroke, heart disease, and end-stage renal disease.[4]

Although awareness, treatment, and control of hypertension have improved in recent years, progress remains suboptimal in the general population, especially for African-Americans.[5]

While essential hypertension has been shown to be a risk factor for cardiovascular disease, the etiology of it remains uncertain.[6] Multiple hypotheses have been proposed to explain its predilection for African-Americans, such as a race-based increased sodium-sensitivity, or an upregulated tissue angiotensin II system. Other hypotheses point to socioeconomic factors, such as limited access to health care, delays in diagnosis, and iatrophobia.[4]

However, the most controversial theory, is the slavery hypertension hypothesis, which is founded on two basic assumptions:

1. The regions of Africa from which slaves came were salt-scarce; and

2. Fifty percent to 60% of the slaves taken from Africa died during transit to America via the Middle Passage, and in the first three years of bondage, and the disorders responsible for this extraordinarily high death rate were salt-depletion disorders, such as diarrhea, fevers, and vomiting.

The hypothesis argues that slaves 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."[2] The survival advantage of these salt-conserving genes transformed into a liability with the advent of the modern, high-salt Western diet by conferring a salt-sensitive hypertension phenotype on today's descendants of African slaves.

Dismantling the historical evidence

While the slavery hypertension hypothesis has had considerable public appeal, its historical underpinnings have been dismantled by Philip Curtin, a historian of the slave trade on whose work the hypothesis is based.[3]

Wilson and Grim suggest that slave mortality rates of 10% to 15% occurred during capture and delivery of slaves to the African coast; while awaiting transport to America; and during their time at sea.[2] Curtin argues, it would be impossible to know the mortality rates that occurred during capture, imprisonment, and transport as African slave traders kept few, if any, records. Therefore, estimates of mortality rates are, at best, guesses.[3]

Curtin continues, "while some deaths from dehydration and salt-depletion could have occurred, there is no evidence that either was a significant cause of death on the slave ships."[3]

Wilson and Grim's contention that limitations in the amount of salt fed to slaves increased the number of deaths due to salt-depletion disorders is also at odds with the available historical data. At the time of the Middle Passage, salt was both abundant and cheap, and salted meat was a main staple of the slave diet.[7-10]

However, water-deprivation could have been a major contributor. In testimony given by abolitionists before the British Parliament in the mid-1800s, slaves were said to have endured ineffable suffering from lack of water

during the Middle Passage. A captain of a slave transport ship described slaves "labouring under the most famishing thirst…being in very few instances allowed more than a pint of water a day."[10] Abolitionist Thomas Buxton alleged that there was "nothing which slaves during the Middle Passage suffer from so much as want of water."[10]

In addition, it is estimated that the temperature below deck where the slaves were housed during transport reached 120°-130°F. In those temperatures, sweating could reach levels as high as 2.5 liters/hour. Although additional water was provided to slaves during transport in the form of a daily quart of soup, it's not likely that any slave could have endured the 35 day to 70 day voyage without succumbing to dehydration.[10]

The historical evidence, although limited, suggests that the diet of slaves during the Middle Passage was likely high in salt, but severely limited in water. Since the overall mortality among slaves transported to America was no higher than 10%, the daily water ration must have been considerably greater than that reported by abolitionists testifying before the British Parliament. And, given the fact that slaves were fed considerable amounts of salt in the form of cured meat, and as medicine,[9] those with an enhanced genetic-based ability to conserve salt should not, as suggested by the slave hypertension hypothesis, have "had a distinct survival advantage."[3]

A different hypothesis

The enforced immobility of slaves chained below deck during voyages would have placed them at considerable risk of deep vein thrombosis. Hemoconcentration resulting from the chronic dehydration described above would have increased this risk. Therefore, deep vein thrombosis may have been responsible for "edematous swelling of the legs"[11] of slaves, which Guinea surgeons (surgeons assigned to slaves ships) attributed at the time to scurvy.[9]

However, deep vein thrombosis would have led to pulmonary emboli as a cause of at least some of the deaths during the Middle Passage. It is recorded that Guinea surgeons believed that slaves were capable of committing suicide by holding their breath,[10] but a more likely explanation would be fatal pulmonary emboli, a disorder unknowingly mitigated by slavers who later began to force slaves "to exercise themselves with dancing"[11] at least once a day for one to two hours, with the belief that exercise reduced mortality.

About James Comotto

A Maryland native, I graduated in 2014 with a major in biology from Washington College where I learned to question theories of thought in science and philosophy. As a second year medical student at the University of Maryland School of Medicine, I have used history to answer the questions of modern medicine. My hope is that researching and writing in the humanities will help hold medical science accountable for the people it is meant to serve.

If pulmonary emboli were responsible for some of the slave deaths during the Middle Passage, the diagnosis could not have been made by slave surgeons since its existence was not recognized until the German physician Rudolf Virchow (1821-1902) elucidated its pathophysiology in 1856, almost a half century after the slave trade ended.

A British ship surgeon in 1790 suggested "fully two-thirds of slave deaths during the Middle Passage stemmed from [Melancholia]," which is an example of archaic diagnoses of the time.[10]

Flawed scientific evidence

Genetic analyses conducted to date tend to refute the existence of the evolutionary bottleneck responsible for the propensity proposed by the slavery hypertension hypothesis. In 2001, an article published in the *Journal of Human Hypertension*, an analysis on a sample of U.S.-born African-Americans and African-born immigrants for alleles associated with hypertension risk (G-protein, AGT-235 and ACE I/D), the AGT-235 homozygous T genotype was found to be more common among African-born immigrants than among U.S.-born African-Americans.[12,13]

In addition, the physicians who cared for slaves residing on plantations most often diagnosed diseases of the lungs, particularly pneumonia and tuberculosis, though malaria and gastroenteritis were also common. According to Curtin's analysis of the historical evidence, salt-depleting diseases were not the principal cause of death among recently captured slaves. Rather, they were disorders which accounted for mortality rates of 6.3 deaths to 16.5 deaths per thousand.[3]

Evolutionary biologists also question the slave

hypertension hypothesis based on the fact that the suggested evolutionary bottleneck is much too rapid to have had such long-term effects, and different genes likely influence salt retention and excretion.[14,15] Even if there were a significant level of assortative mating within the race, it would be expected that any divergence caused by a short-term bottleneck would fade out rapidly during subsequent generations. In order to have maintained the salt-retaining phenotype, there would have had to be "almost complete genetic homogeneity…achieved during selection."[15]

While a genetic defect in salt excretion would be physiologically appealing, and "more consistent with salt-retention as a basis for hypertension, again, it is difficult to understand why selection would favor such a trait."[15]

Why so persistently popular?

Deterministic biological explanations, such as that embodied in the slavery hypertension hypothesis, absolve both society and individual of responsibility for a health problem that is, at least in part, behavioral.[16] African-Americans, have not been shown to metabolize salt differently from other racial groups, rather, the bulk of evidence suggests that hypertension in general, and its predilection for African-Americans, is mediated by a host of factors such as low birth weight, low-protein maternal diet, excess glucocorticoids, vitamin A deficiency, undernourishment, and enhanced growth in childhood due to modern diet and lifestyle.[16,17]

Social determinates of health and health disparities are the real factors for increased hypertension in African-Americans.

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