

# Hypertension in african americans and the middle passage



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In 1991, Wilson and Grim (1991) sought to explain why African Americans have a higher incidence of hypertension. Their hypothesis stated that an event in single genetic selection during slave capture and transport favored slaves with salt sensitive genes because they were able to retain a sufficient hydration level for survival. This “slavery hypertension hypothesis” has been widely reported and accepted in popular media, but it has met with considerable controversy in scientific circles. Most experts have rejected this hypothesis as incompatible with documented historical data as well as with principles of population genetics, and today’s view is that it is most likely incorrect. This paper traces debates around Wilson and Grim’s (1991) hypothesis, paying particular attention to the implicit ideologies of proponents and opponents in regard to the heightened occurrence of hypertension among African Americans.

Medical science defines hypertension as resting blood pressure above 140 mm Hg during the heart stroke (systolic value) and 90 mm Hg between strokes (diastolic value). Normal blood pressure is 120/80. Doctors distinguish two types of hypertension: “essential” hypertension, of unknown etiology, which accounts for 90-95% of cases. In secondary hypertension, which accounts for about 5-10% of cases, to which the causes are known – for example, cancers of the adrenal glands, or loss of limbs.

Essential hypertension is a multi-factorial disease. Factors correlated with its incidence include lifestyle, diet, exercise, weight, age, race, gender, and general health (e. g., diseases such as diabetes, or metabolic syndrome).

There are multiple genes known to be associated with essential hypertension risk, e. g., alleles for G-protein, AGT-235, and ACE I/D (Poston 2001), but the <https://assignbuster.com/hypertension-in-african-americans-and-the-middle-passage/>

genetic basis of essential hypertension is currently not completely understood.

African Americans have higher rates of hypertension and suffer from more severe complications than non-Hispanic whites (Ferdinand, 2007). In the 2003-2004 National Health and Nutrition Examination Survey (NHANES, 2004, survey) trials, the percentage of participants having high blood pressure ( $> 140/90$  mm Hg or taking medication) was found to be 41.4% for African American females vs. 28.0% for non-hispanic white females, and 39.0% for African American males vs. 28.5 for non-hispanic white males. "With an estimated 81 percent of African American women and 78 percent of men age 50 and older having hypertension, the disease constitutes an epidemic" (Schoenberg, 2002, p. 458). The differences in morbidity and mortality from hypertension-related diseases between African Americans and the general population are even higher than these relative rates of incidence would suggest. Overall death rates from hypertension for African Americans are 49.9 for men and 40.6 for women, vs. 17.9 national average (per 100,000); African Americans have 1.8 times the rate of stroke, 4.2 times the rate of end stage renal disease, 1.7 times the rate of heart failure, and 1.5 times the rate of coronary disease mortality. Thus, hypertension-related illnesses not only have a higher incidence but also take a greater toll on African Americans than on the Caucasian Americans.

Salt uptake is known to have an effect in essential hypertension; however, the size of the effect varies from individual to individual. The physiological pathway through which salt uptake influences blood pressure is the Renin-Angiotensin-Aldosterone System (RAAS) (Fournie-Zaluski, 2004, p. 7775). <https://assignbuster.com/hypertension-in-african-americans-and-the-middle-passage/>

Angiotensin I is a peptide synthesized in the liver, which is converted to Angiotensin II in a process regulated by ACE and renin. The angiotensins determine the amount of water and salt that is retained in the body by regulating kidney activity. An increased level of Angiotensin II leads to increased re-absorption of sodium in the kidney tubules. In order to maintain homeostasis of electrolytes, the body then increases effective circulating volume, which leads to higher blood pressure.

It has been demonstrated that ethnicity is strongly correlated with the effects of dietary sodium reduction on blood pressure (Bray, 2004). In the 2004 'Dietary Approaches to Stop Hypertension' (DASH) sodium trial, patients were placed on a 2100 kcal/mole per day diet using three levels of sodium (150, 100, and 50 mmol per day). For thirty days, patients either consumed a special DASH diet or a typical American diet. At the end of the trial, the observed spread in systolic blood pressure (SBP) between the highest and lowest sodium levels was significantly higher for African Americans than for non-African Americans:

Wilson (1986) suggested a hypothesis for the prevalence of hypertension in African Americans, which was expanded and re-published in a joint paper with Grim in 1991 (Wilson, 1991). Their hypothesis, which became known as the Wilson-Grim slavery hypertension hypothesis, stated that hypertension in African Americans is higher than in Africans of comparable genetic origins because the slave trade selected for high salt retention and was a "genetic bottleneck." Death rates among slaves were high during transport, and dehydration was the main cause of death. Wilson and Grim cited death rates during slave shipping from Africa to the Americas (the so-called "Middle Passage") as evidence for their hypothesis. <https://assignbuster.com/hypertension-in-african-americans-and-the-middle-passage/>

Passage”) of about 30% (Wilson, 1991, pgs. 1-125). Persons with a higher natural ability to retain salt were less sensitive to dehydration and thus more likely to survive the slave transport to the Americas, as well as the continued environment of heavy physical labor under hot conditions in the plantations. Wilson and Grim assumed that salt was scarce in the regions of Africa where the slaves originated, and that thus there were genes for its efficient use already present in the population (Wilson, 1991, pgs. 1-123).

As further proof of a difference in gene distributions, Wilson and Grim experimentally determined differences in hypertension rates between African Americans and Africans. They measured blood pressures in a West African village where salt intake was similar to that of African Americans. Hypertension rates in Africans living today in the regions where North American slaves originated are about 2.7 times lower than the rates for African Americans (Fackelmann, 1991).

In a popularization of Wilson and Grim’s hypothesis, Diamond (1991) provided a reckoning of the “ successive winnowing” of slave numbers along the stations of enslavement; of 100 slaves captured/sold in Africa, ~25 died during forced marches to the coast, ~12 died in camps waiting to be shipped (“ barackooning”), ~5 died while ships were going up and down the African coast, filling their holds, ~10 died on the Middle Passage to the Americas, ~10 died during the first three years of plantation life (“ seasoning”); sometimes many more – generally, it was cheaper for plantation owners to buy new slaves than to provide better living conditions (Diamond, 1991, p. 4). At most, 30% of the original number of captured slaves survived to pass on their genes.

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As main causes of death Diamond (1991) cites dehydration from sweating and lack of water, diarrhea (“ fluxes”) of all etiologies, seasickness/vomiting, other diseases, punishment/execution, and suicide/loss of will to live.

Diamond’s quoted death rates for the Middle passage are much lower than Wilson and Grim’s. However, in an embellishment on the original hypothesis, he assumed a high death rate for the initial “ forced marches” to the coast (Diamond, 1991, p. 4).

Supporters of the Slavery Hypertension Hypothesis have pointed to anecdotal supporting evidence. Dimsdale (2000) enlists a passage from ‘ Moby Dick’ describing sharks following slave ships to feed on the bodies thrown overboard to demonstrate the high death rates in the Middle Passage. A 1794 illustration of a slave trader licking the face of a slave in Africa was interpreted as checking whether the slave’s skin was salty (Dimsdale, 2000). More salt on the slave’s skin would mean a higher chance of death during transport, making the slave less valuable. While Dimsdale (2000) acknowledges that the Slavery Hypertension Hypothesis is “ sheer speculation” because of lack of DNA evidence for a genetic basis, he concludes that “ nevertheless, the hypothesis remains an intriguing one” (Dimsdale, 2000).

The Slavery Hypertension hypothesis was widely popularized by Diamond and others, and it found its way not only into the newspapers but also into textbooks, medical journals, and review articles (Armelagos 2005: 120; Kaufman 2001). An example how widely the hypothesis has been accepted in the general population is given by a 2007 “ Ask Dr. Oz” segment on “ Oprah”. When an audience member asked: “ Why do I sweat so much?” Dr. <https://assignbuster.com/hypertension-in-african-americans-and-the-middle-passage/>

Mehmet Oz (wearing scrubs) explained that excessive sweating can result, among other reasons, from hypertension. He then turned to Oprah to ask: “Do you know why African Americans have high blood pressure?” Oprah answered: “African Americans who survived (the slave transport) were those who could hold more salt in their body.” Dr. Oz rejoiced: “That’s perfect!”

Unfortunately, from a scientific point of view, the Slavery Hypertension hypothesis is far from perfect. Almost every single one of Wilson and Grim’s (1991) assumptions and conclusions almost immediately drew withering criticism from historians and geneticists.

Curtin (1992), a historian of the slave trade on whose work Wilson and Grim drew extensively, methodically disassembled almost every number that they assumed. His criticism concluded that their hypothesis lacks supporting evidence and “runs counter to what evidence we do have” (Curtin, 1992, p. 1686). In particular, Curtin noted that:

The regions from which American slaves came were not salt-scarce.

Slaves for the Americas were not marched for months, they came from near the coast.

Wilson&Grim’s death rate numbers for the Middle Passage were too high.

Diarrhea/water loss were not key death factors for slaves.

Curtin (1992) seems to have been particularly incensed by Jared Diamond’s popularization of Wilson and Grim’s hypothesis (Diamond, 1991), which he

perceived to present the hypothesis as proven and to “selectively misrepresent the evidence” (Armelagos, 2005).

Geneticists have argued that population dynamics do not allow the Wilson/Grim hypothesis: even the death rates during slave transport cannot influence gene distributions to that extent. However, this view is not uncontested: Fatimah Jackson supported the concept of the genetic bottleneck in a 1991 paper, and further postulated that stress experienced by slave populations lead to increased genetic variability (Jackson, 1991). Others have argued that African Americans have ~15-20% admixture of Caucasian genes, so any genetic effects should be diluted. A 2001 study comparing African Americans and African-born immigrants examined the known alleles associated with increased hypertension risk (G-protein, AGT-235, and ACE I/D) found that the AGT-235 homozygous T genotype was more prevalent among African-born immigrants, the opposite of what would be expected from the Slavery Hypertension Hypothesis (Poston, 2001). However, it is clear that our understanding of the genetic basis for hypertension is at best incomplete. Luft (2001) lists seven genes known to be associated with hypertension but concludes that “In terms of genetically explaining blood pressure variance for specific genes, we have a long way to go” (Luft, 2001, p. 503).

Kaufman was particularly outspoken in criticizing Wilson and Grim’s hypothesis in a series of articles and letters. He initially attacked Dimsdale’s (2000) summary of the hypothesis as a “careless repetition of the old ‘Slavery Hypothesis’ yarn,” calling it a “medical myth” and “pseudoscientific canard” that “unwittingly plays into the hands of racial essentialists and <https://assignbuster.com/hypertension-in-african-americans-and-the-middle-passage/>



biological determinists,” and relegating it to “fantasy,” not “sensible and respectable science” (Kaufman, 2001). Dimsdale (2001) responded to this by noting that “Race and ethnicity are too important to be ignored or politicized” (Dimsdale, 2001). In subsequent publications (Kaufman & Hall, 2003), Kaufman (2001) was particularly concerned with the ideology that he perceived to underlie the Slavery Hypertension Hypothesis, and accused its proponents to foster the notion that “Blacks” are “inherently different by harboring genetic defects or physiological abnormalities” (Armelagos, 2005, p. 121). Certainly Wilson and Grim’s initial language referring to “defective kidneys” and “renal defects” (Wilson 1991: I-123) was ill chosen in that regard, although they note in the same article that “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” (Wilson, 1991, pgs. I-126). Kaufman (2001) also questioned the use of race (as defined by skin color) or ethnicity as physiologically useful criteria, despite the epidemiological studies quoted above that have shown measurable differences, but it is not clear what he would suggest to use in its place.

It could be said that Kaufman’s (2001) attacks added a political and ideological dimension to the Slavery Hypertension debate, but is more accurate to say that they only brought a previously hidden dimension into full view. In their 2003 paper, Kaufman and Hall attacked the concept of “genetic determinism” and “essential black abnormality” they saw as underlying Wilson and Grim’s work (Kaufman & Hall, 2003). They saw it as an example of a worldview that “blames the victim and displaces economic or

cultural factors from our understanding of the underlying etiology of the disease” (Armelagos, 2005, p. 121). While Grim and Robinson (2003) interpreted this criticism as being called racist (Grim & Robinson 2003), racism was not really implied in it. Instead it is a difference in worldview between the reductionism of the exact sciences and the approach of the social sciences that attempts to synthesize scientific evidence and social context into a broader picture.

Both approaches have their inherent fallacies: the ‘ pure science’ approach may focus on pieces of the puzzle, maintaining that no area of inquiry is off limits to study by unbiased and objective scientists. Never mind that there is no such thing: scientists cannot escape the mental context and preconceived notions of the societies they live in, and objectivity is a more of a lofty goal than reality. Too often in the history of science, prejudice has masqueraded as scientific conclusion. One example that comes to mind is the notion of female intellectual inferiority widely accepted in the 19th century which was based on no more evidence than lower average brain weights of women, yet was widely accepted because all scientists were male and everybody (male) already ‘ knew it was true.’ On the other hand, preoccupation with the social consequences of a result can taint scientific inquiry and prevent a clear view of what is true or not. Kaufman (2001) disavows “ genetic determinism” without disproving it based on what would be considered ‘ hard science.’ In the final analysis, the difference in worldviews comes down to whether there is such a thing as an objective truth, dissociated from social context, or not.

Singer (1996) addresses these and other issues. He specifically refers to “

Cartesianism, that Western tendency to see independent parts as composing  
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and determining a summary whole” (Singer, 1996, p. 499), and argues that the a simple one-way Darwinian adaptive response of organisms to the environment (a view that he calls “ adaptionism”) needs to be replaced with a dialectical view in which organisms shape the environment even as they are adapting to it. Since the human environment is significantly determined by societal and economic forces, he argues that humans undergo “ unnatural selection” (Singer, 1996, p. 506). He then goes on to re-capitulate Wilson and Grim’s (1991) hypothesis as an example of the genetic component of such a process, and further embellishes their emphasis on the effect of gastrointestinal disorders on slave mortality with a reference to cholera. However, cholera was “ completely absent from the Atlantic basin during the period of the slave trade to North America” (Curtin, 1992, p. 1684).

Setting aside this small factual inaccuracy, Singer’s (1996) argument that environment and organisms co-evolve is undoubtedly correct, although it is not clear exactly who would argue otherwise. Natural history is full of examples where organisms have fundamentally changed the environment and then were in turn changed by it: one example that comes to mind is the evolution of early oxygen generating organisms that fundamentally changed the earth’s atmosphere and enabled animal life, but then could not compete with new life forms and became extinct. Singer’s (1996) argument has more merit with respect to modern humans and the economical and societal aspects of their evolution, and it is indeed true that the natural sciences may overlook societal contributions in their effort to establish simple cause and effect relationships. However, it is not clear whether a truly dialectical perspective is needed or whether the recognition that humans have

increasingly shaped their own environment and that today their natural environment is largely man-made would suffice. In any case, it would appear that the slave trade is a poor example against the adaptationist view, since slaves were essentially powerless to change their environment, so that any adaptations caused by slavery could only be one-way.

Beyond such general philosophical considerations, the Slavery Hypertension hypothesis undoubtedly has a lot of issues as a 'hard science' hypothesis. In order to analyze Wilson and Grim's (1991) hypothesis more closely, it is necessary to examine the history and economics of the slave trade. The slave trade from Africa began around 1517 and ended in 1888, when Brazil outlawed slavery (Diamond, 1991, p. 4). The slave trade to the United States effectively ended in 1807. Originally in Portuguese hands, the slave trade became part of the "triangular trade", in which ships carried cotton and other plantation crops from the Americas to Europe, manufactured goods from Europe to Africa, and slaves from Africa to the Americas in the infamous "Middle Passage" (Boddy-Evans). Overall, about ten to twelve million slaves were brought to the Americas from Africa.

Slaves originated mostly from the West coast of Africa: Senegambia, Upper Guinea, the Windward and Gold Coasts, the Bights of Benin and Biafra, and West Central Africa. A smaller region of slave origins is found in South East Africa (Boddy-Evans, 2010). In the time of interest for slave transport to North America, slaves were captured or bought mostly in regions no more than 100 miles from the coast. Diamond's (1991) death rate of 25% during "forced marches" to the coast seems to be excessive for this distance.

Conditions during slave transport were appalling but death rates were lower than the 30% quoted by Wilson and Grim (1991). The duration of ships' voyages for the Middle Passage decreased from about three to six months at the beginning of the slave trade to six to eight weeks towards its end. The best existing estimates give death rates of about 24% around 1680, close to Wilson and Grim's number, but dropping to less than 6% around 1790 (Curtin, 1992, p. 1684). For all slaves transported to North America, the number-averaged death rate is estimated to be about 10 to 12%, close to Diamond's number but much lower than the one assumed by Wilson and Grim.

As an aside, death rates for the ships' crews were not very different from those of slaves. This seems to indicate that slave ship captains were not completely indifferent to slave mortality figures but followed their economic interest in providing a level of care reasonably possible under the technical and logistical constraints of their time.

Wilson and Grim's (1991) argument that African populations with similar salt intake have lower incidence of hypertension can also not be considered conclusive since it does not take other differences between the populations into account, in particular prevalence of obesity, diabetes and metabolic syndrome between the populations, differences in diets and lifestyles, and the impact of psychological factors (low socioeconomic status, effects of discrimination).

Dressler (1993) looked at health effects in the African American community. He developed the idea of status incongruence, which here means having a

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more expensive lifestyle than people think one is entitled to. African Americans with darker skin color and higher lifestyle have about three times higher hypertension rates than those with lower skin color and lifestyle. However, any social effects of skin color are convoluted with possible genetic contributions because lighter skin color could mean more Caucasian genes. However, the hypertension rates for African Americans with higher education (> 12 years of school) are about a factor of two higher than for those with lower education (≤ 12 years) and there it is almost impossible to imagine that slave transport has any impact on whether a person goes to school longer or not. Dressler's (YR) study indicates that other factors can have very large influences on hypertension, as large as the observed difference between African Americans and Caucasians.

Armstrong (2005) gave an extensive and somewhat unbiased historical review of both sides of the "Slavery Hypertension story". Armstrong (2005) came down squarely in the camp of the hypothesis' opponents, stating that "there is no indication of a genetic bottleneck or evidence of 'racial' differences that are genetically determined" and that "It is time to discard 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" (Armstrong, 2005, p. 119). His conclusion appears to be shared by most experts today, and it is certainly true that the factual basis of the hypothesis is disputed or weak. However, Armstrong (2005) accepts the arguments of the hypothesis' opponents, and nagging questions remain.

Curtin's criticism was particularly damaging in view of the fact that Wilson and Grim drew so heavily on his work. While most of Curtin's arguments are valid, some stretch the evidence:

His view that diarrhea was not a factor is based on plantation and British army records for African conscripts, which show pulmonary diseases and fevers as main killers. However, these death rates are much lower (<5%), and thus not representative of the situation of slaves during transport. It is also disputed by other historians who state that "gastrointestinal disturbances were responsible for the greatest annual death rates in slaves during Middle Passage transit" (Dimsdale 2001: 235, quoting Steckel and Jensen 1986).

He argues that vomiting due to seasickness is a 48 hour long phenomenon that cannot have a genetic impact generations later. However, it can if it leads to a permanent effect (death from dehydration).

His argument that the regions from which slaves originated were no salt scarce is limited to recent history and does not rule out a genetic contribution left over from pre-historic times.

Armelagos (2005) also does not address Dimsdale's analysis (Dimsdale 2001: 235) of Eltis et al.'s (1999) work on mortality rates during the Middle Passage (Eltis 1999) that Kaufman (2001) drew on when questioning Wilson and Grim's numbers. The Eltis database contains information on only 5, 130 of their 27, 233 voyage data set, and their death rates are imputed. While Dimsdale's (2001) argument that this means that "the death rates in the

Middle Passage are uncertain to everyone” is somewhat defensive and self-serving, it contains a kernel of truth.

Lastly, the fact remains that hypertension in African Americans is real, but it seems that it cannot completely be explained by other known factors even after adjusting for health-related behaviors. This may reflect our ignorance, but it leaves a loophole for the genetic contribution postulated by the Slavery Hypertension hypothesis,

Kaufman notes that “ the seductive nature of Dr. Grim’s fairytale” is in itself an interesting sociological phenomenon” (Kaufman 2001). The Wilson and Grim slave transport/hypertension hypothesis is seductive because it provides a simple, mono-causal explanation for a complex phenomenon. It also fits in well with the concept of ‘ Darwinian medicine’ that has been successful in explaining the prevalence of sickle cell disease as a protective factor against malaria, or of Tay-Sachs disease against tuberculosis. As in these cases, adaptation to a specific environment carries a cost, and it becomes maladaptive under altered circumstances. The difference to the Slavery Hypertension hypothesis is that the molecular genetics base for these diseases is comparatively simple and well understood. The genetic basis for salt sensitivity is not well understood, and it can be assumed to be far more complex.

The widespread and ready acceptance of the Slavery Hypertension Hypothesis in the African American community is on the face of it a puzzling phenomenon when viewed in the context of Kaufman’s assertions that it perpetuates myths of Black inferiority. However, it fits in with the “ cult of

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victimology” (a term coined by John McWhorter) (McWhorter) practiced by some in the African American community in which all ills befalling them are rooted in slavery or discrimination, and it exonerates African Americans from any behavioral or diet-based contributions to their hypertension disease load.

Today, the Slavery Hypertension Hypothesis is widely seen as disproved. However, a final answer will have to await a complete understanding of the genetic basis for essential hypertension, and a comparison of the disease markers in the African American and original African populations. If such data were to show that there is indeed a genetic difference in genes regulating salt metabolism between African and African American populations, the Slavery Hypertension Hypothesis would have to be resurrected. Until these data are available, it must be considered unproven and at variance with much of the historical record. Dare we say it should at least be taken with a large grain of salt?