Editorial Commentary

Why Do Black Americans Have Higher Prevalence of Hypertension?
An Enigma Still Unsolved

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Studies have consistently reported a higher prevalence of hypertension in blacks than in whites, a main reason for the higher incidence of cardiovascular disease in blacks.1 The long list of putative causes for this higher prevalence suggests that the real reasons are still unknown. Biological differences in the mechanisms of blood pressure control or in the environment and habits of whites and blacks are among the potential causes. The higher prevalence of hypertension in blacks living in the United States instead of Africa2 demonstrates that environmental and behavioral characteristics are the more likely reasons for the higher prevalence in blacks living in the United States. They could act directly or by triggering mechanisms of blood pressure increase that are dormant in blacks living in Africa.

Kaplan and Victor3 listed 18 genotypes and intermediate phenotypes that were implicated with an increase of blood pressure in blacks. A higher sensitivity to alcohol could be added to that list.4 Some of these mechanisms, such as a higher renal retention of sodium by blacks, are attractive. The “slavery hypertension hypothesis”5 states that the higher prevalence of hypertension among blacks could have resulted from an enhanced ability to conserve salt by slaves, protecting them from fatal salt-depletive diseases during the stormy Atlantic passage, such as diarrhea and vomiting.5 This condition would induce hypertension when they and their descendants consumed the much higher sodium content in American compared with African foods. This hypothesis is hard to confirm or refute.

The association between genetic traits and renal outcomes has been reported in black Americans versus white Americans. Kao et al6 and Kopp et al7 reported an association between a genetic variation of the MYH9 region on chromosome 22 with focal segmental glomerulosclerosis, a condition previously attributed to “hypertensive nephrosclerosis” and found much more frequently in black patients with end-stage renal disease than in whites with end-stage renal disease. The attributable risk for carriage of this MYH9 haplotype was found in 74% of blacks and in only 4% of whites with focal segmental glomerulosclerosis. More recently, using much more complete genomic sequences, Tzur et al8 reported missense mutations in the APOL1 gene, which neighbors the MYH9 gene on chromosome 22 in patients with focal segmental glomerulosclerosis. These genetic defects may account for the higher propensity of blacks to develop end-stage renal disease, which may then induce hypertension, rather than hypertension being responsible for the renal damage. Nonetheless, the possible role of these or other genetic defects in the large majority of blacks without focal segmental glomerulosclerosis or end-stage renal disease remains unknown, particularly with regard to differences in the renal handling of sodium between normotensive black subjects living in Africa and the United States.

Differences in exposure to the environment and habits between blacks and whites have also been proposed to explain their differential prevalence of hypertension. Many potential reasons have been reported, such as socioeconomic status, dietary habits, social network, stress, and health behaviors. Among the consequences of differential dietary habits, excess adiposity emerges as a natural candidate to explain the higher prevalence of hypertension in blacks, who have a 51% greater prevalence of obesity than whites.9 Nonetheless, excess of adiposity does not fully account for the higher prevalence of hypertension in blacks. Based on data of National Health and Nutrition Examination Survey (NHANES) III, Okosun et al10 demonstrated that the population-attributable fraction of hypertension attributed to abdominal obesity was 24.9% in black men and 15.9% in black women. The risk of black ethnicity for hypertension persisted after adjustment for abdominal obesity and other confounders: the odds ratios in black men were 1.80 (95% CI: 1.54 to 2.11) and in black women 2.70 (95% CI: 2.07 to 3.28). There is no conclusive evidence that other nutritional factors could explain the higher prevalence of hypertension in blacks.

In this issue of Hypertension, Redmond et al11 present the results of an analysis of the data from the NHANES 2001–2006 survey, a nationally representative cohort of US adults, aiming to identify health behaviors that could explain the high prevalence and lower rate of hypertension control among black people. They found that the rate of blood pressure control was worse among non-Hispanic blacks (27.4% with uncontrolled blood pressure) compared with non-Hispanic whites (17.0%) and Mexican Americans (20.2% with uncontrolled blood pressure). Odds ratios for poorly controlled blood pressure were higher in non-Hispanic...
blacks (1.88 [95% CI: 1.53 to 2.32]) compared with non-Hispanic whites, despite adjustment for a large number of potential confounders, including race/ethnicity, age, sex, education, insurance status, financial status, general health status, functional status, body mass index, diabetes mellitus, smoking status, physical activity, and sodium, fiber, alcohol, and total daily calorie intake. The rate of blood pressure control was similar in non-Hispanic whites and in Mexican Americans. In an analysis restricted to individuals with hypertension, the authors included adherence to medications to the full model, and the degree of control remained poorer for non-Hispanic blacks (odds ratio: 1.49 [95% CI: 1.12 to 1.98]) in comparison with non-Hispanic whites. Redmond et al11 rightly concluded that racial/ethnic disparities in blood pressure control were not explained by differences in health behaviors that they identified within the data collected by the NHANES. Nonetheless, measurement bias of some health behaviors cannot be fully discarded.

Nutritional aspects, in particular, seem to be more important, because in the NHANES 2003–2006, nutrients were identified by just one 24-hour dietary recall. At least the record of 3 days or the use of validated food-frequency questionnaires are nowadays recommended to better establish individual dietary patterns. A lower adherence to a Dietary Approaches to Stop Hypertension–like pattern of diet by black Americans, a diet that has been associated with lower prevalence of hypertension,12 may have been missed by the 1-day questionnaire. The authors were not able to investigate the frequency of consumption of dairy products, and they used fiber content as a proxy for ingestion of fruits and vegetables.

The evaluation of adherence to treatment is another potential source of bias. Hypertensives classified as nonadherent were those who answered “yes” to the question, “Because of your high blood pressure/hypertension, have you ever been told to take prescribed medicine?” and “no” to the question, “Are you now taking prescribed medicine?” Evaluation of adherence to treatment has been a complex issue, and an unknown proportion of participants may have been misclassified by the criteria used in this analysis. Other potential biases, such as occupational-related physical activity, exposure to psychosocial stressors, discrimination, socioeconomic position in early life, social networks, and other unknown confounders, which may vary by ethnic background, were not investigated in the NHANES.

And where are we going now? Redmond et al11 recommend that future research should further assess the relationships of gene-environment interactions, job-related stress, racism, and other psychosocial factors to racial/ethnic disparities in the prevalence of hypertension. In other words, they are saying what has been repeatedly stated by articles on the subject, that the enigma over the reasons for the higher prevalence and lower rate of control of hypertension among black Americans still remains to be deciphered.

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