I
n the last few years, much attention has been given to racial and ethnic differences in health measures. Not surprisingly, much of the work in this area is being done in the United States. Not only does our unique racial and ethnic diversity offer opportunities for study, but the social and political issues related to this diversity have driven a need to understand the disparities in health related to these differences.

These racial and ethnic differences in health measures are seen clearly in cardiovascular disease risk factors and outcomes for Americans of African descent, or African Americans (AAs), compared with those of European descent, or European Americans (EAs). Compared with EAs, AAs have higher mortality rates for most cardiovascular diseases, including coronary heart disease and stroke. These differences are magnified at younger ages.

Prevalence rates for key risk factors differ for AAs, with higher rates for hypertension, obesity, and diabetes mellitus, and lower rates for dyslipidemia. Additionally, the cardiovascular consequences imposed by various risk factors differ by race. Compared with hypertensive EA men and women and AA men, AA women with hypertension have a substantially greater relative risk for heart disease. Conversely, AA women have a substantially lower relative risk for diabetes mellitus than EA women.

The disparities in cardiovascular outcome are large and significant, and the disparities in outcome have worsened in the last 2 decades. Appropriately, much attention is being given to understanding these differences.

For a number of years, this important area of study was hampered by a lack of interest and a lack of funding. Now that a commitment is being made in this area, other challenges of understanding the causes of these differences are becoming clearer. The science is complex.

The challenges to understanding the science of racial differences and disparities in cardiovascular disease are substantial. Race and ethnicity are difficult to define and classify. The biology is driven by a complex set of gene/gene, environment/environment, and gene/environment interactions. Among the environmental influences are social issues that may induce stress and potentially influence multiple physiological functions. These are difficult to measure, and understanding the biological consequences is difficult. Other social issues such as access to care have less interaction with biology, but these issues drive some of the differences in outcome.

Research in this area is hampered by the small knowledge base. Most of what is known has been gained through observational studies. Moving from the knowledge gained in observations to experimental design has not been easy. This area of study does not lend itself easily to biological exploration through animal models. And, just as clinical trials exploring gender differences are challenged by design issues, trials exploring racial differences are difficult to design.

Most of what is known about racial differences in hypertension and cardiovascular disease comes from studies in adults. Understanding the root causes of vascular disease in adult patients is often complicated by the vascular changes brought on by the disease. This is especially true in studying hypertension because blood pressure patterns are often impacted by the vascular disease, which may occur as a consequence of the blood pressure abnormalities.

This has been particularly problematic in studying hypertension and vascular disease in AAs. In young adults, cardiovascular disease rates are substantially higher in AAs than in EAs. The onset of vascular changes occurs earlier, and risk factors also are seen earlier, especially obesity, hypertension, and diabetes mellitus.

The study by Wang et al in this issue of Circulation makes an important contribution to our understanding of racial differences in blood pressure. The authors report that the blunting of nocturnal blood pressure declines in AAs begins by age 10 years and that this blunted decline is exacerbated with age during the years of adolescence.

In addition to racial differences in prevalence rates of hypertension, differences in the blood pressure pattern demonstrated through 24-hour ambulatory blood pressure monitoring have been reported. Compared with EAs, AAs have higher nocturnal blood pressure and a smaller difference between daytime pressures. That is, compared with EAs, AAs have less “dip” in blood pressure at night. This “nondipping” status is strongly associated with higher rates of vascular disease.

The finding that this lack of nighttime decline in blood pressure is seen in AAs at a very young age and accelerates during adolescent years is important. The authors appropriately suggest that this increased blood pressure burden may
be an important cause of the excessive cardiovascular disease rates in AAs and may account for the frequent early onset of vascular disease.

Understanding this potential explanation for excessive vascular disease in AAs is important, and the opportunity to explore the causes of blood pressure differences among young AAs could be highly important in understanding how better to prevent premature onset of disease. The authors note that the lack of nocturnal dipping is not seen in groups of African descent outside the United States. This leads the authors to conclude that this blunting of nocturnal decline seen in AAs is not purely genetic in origin but is likely the result of a gene/environment interaction. Depending on the Guyton model of long-term blood pressure control through pressure natriuresis, they suggest that some environmental factor causes sodium retention, leading to a need for a higher nocturnal pressure to excrete the sodium load.

The authors report some evaluation of potential causes through their analysis of environmental associations with the nondipping status. In their interpretation of the findings, the authors point to the possibility of social factors, including stress-induced sodium retention.

Among the social factors considered, the study used a tool to measure John Henryism. This scale measures active coping responses to stress. It may be surprising to some that this stress-related association with blood pressure abnormalities was present at a very young age. Importantly, the authors note that the impact of stress on blood pressure as measured by this John Henryism scale was seen in both AAs and EAs.

Understanding the relationship between stress, blood pressure, and vascular disease is complicated. The methodologies are complex and controversial. Most of the human data demonstrating a link are observational. Most experimental data that seem to support a cause-and-effect relationship are from animal models. In humans, it has been difficult to show long-term benefit on blood pressure with available therapies for management of stress. On the basis of the current evidence, major guidelines do not list stress as a risk factor for hypertension or cardiovascular disease, and the guidelines offer no specific recommendations for stress management as a tool for improving blood pressure.

The other major association with nondipping status noted was that of adiposity, or body mass. The authors report a strong association with blood pressure status and body mass index (BMI), as have many other studies.

As explanations of the early blood pressure differences between AAs and EAs are explored, body weight and BMI deserve further exploration. Racial differences in BMI have been noted at an early age in other studies, and this difference is also seen in the study by Wang et al. The mean BMI of 24.8 in AA females in the Wang et al study indicates that about half of this cohort, with an average age of 14.4 years, was overweight. Mean BMI for AAs was about 10% higher than for EAs at this early age in both males and females. This contrasts with almost identical scores for John Henryism across race.

Both BMI and blood pressure are continuous variables. The arbitrary cutoff points are necessary for clinical decision making, but they are artificial. The relationship between BMI and blood pressure is roughly linear and extends well into the lean range. Understanding that this relationship does not depend on adiposity, overweight, or obese status suggests that body weight may be an important cause of the early differences seen in blood pressure profiles across race. Also, the location of excess fat seems to be an important factor in determining the impact of adiposity on cardiovascular risk, with excess visceral adiposity conveying much greater risk than subcutaneous adiposity. However, the importance of these differences in contributing to racial and ethnic disparities in hypertension and cardiovascular disease is still poorly understood and should be explored further.

In the study by Wang et al, data on the intake and excretion of sodium and potassium were not available. Racial differences in intake and handling of sodium and potassium have been noted in other studies. This may be another area that might be helpful for understanding blood pressure differences at an early age.

Although there are substantial challenges in the area of racial disparities in hypertension and cardiovascular disease, there is some very good news. We have strong evidence that the treatment options available for managing hypertension have similar benefit in lowering blood pressure and in reducing cardiovascular events in EAs and AAs. There are subtle differences in response across race, most notably in degree of blood pressure decreases in response to individual drug classes. But, with hypertension therapy, there are very similar and impressive benefits to reduction of both heart disease and stroke in AAs and EAs.

Wang et al have made an important contribution to our understanding of an important and complex topic. As they note, further study of this area is warranted. Causes of early racial differences in blood pressure profile must be understood. Gene/environment interactions should be explored. Target environmental elements should include stress, body weight, and sodium and potassium intake and handling.

It is important to move forward to expand our knowledge base through further research, but it is equally important to assure appropriate treatment across race. We must take advantage of the good hypertension-management tools available to us now. Applying our current knowledge in the management of hypertension to all, regardless of race, will save many lives and go a long way toward eliminating the existing racial disparities.

Disclosures

None.

References


Racial and Ethnic Differences in Blood Pressure: Biology and Sociology
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