Editorial Commentary

Epidemiological Issues Are Not Simply Black and White

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National health statistics and prospective epidemiological studies have reaffirmed the conclusion that hypertensive cardiovascular disease is more prevalent in the black population than in the white.1 Furthermore, strokes and coronary heart disease are also more common in black individuals.2 In addition, development of cardiac and renal failure continues to increase in prevalence, especially in the black race. As early as in 1939, individuals with blood pressures of 120 to 139/80 to 89 mm Hg (defined then as prehypertension) were more likely to develop established essential hypertension.3 This term “prehypertension” was later adopted in the Joint National Committee’s seventh report, although the term “high normal” was used in the Joint National Committee’s third report on the advice of the insurance industry’s representative, because the earlier term (of 1939) might suggest potential insurability concerns.3

These epidemiological issues have since been confirmed; and a report in this issue of Hypertension further demonstrates that a faster rate of progression from the condition of prehypertension to the diagnosis of established essential hypertension exists in black individuals as compared with white.4 To explore this important question further, the investigators initiated a prospective cohort study of 18 865 nonhypertensive persons (composed of 30.4% black and 69.5% white subjects who were 19 to 85 years of age) using electronic health records that were obtained from 197 community-based outpatient clinics in the Southeastern United States.

The time elapsing from study entry to the establishment of the diagnosis of essential hypertension in the subjects who were under careful scrutiny was one of the major issues addressed by the investigators.4 Cox regression modeling was used to examine conversion to hypertension. Not only did the authors of this study clearly demonstrate the inherent risk that is associated with the state of prehypertension, but they also found that age (ie, 35 to 54 years), diastolic pressure (ie, 80 to 89 mm Hg), and overweight and obesity, as well as carbohydrate intolerance (ie, diabetes mellitus), are potential risk factors related to a significantly faster rate of conversion from the condition of prehypertension to established essential hypertension in black individuals. These findings were included with other pathophysiological factors that had already been identified in predisposed black individuals with essential hypertension, thereby demonstrating their greater risk.5–9 These earlier reports demonstrated that among those clinical pathophysiological alterations was hemodynamic evidence of impaired cardiovascular and renal structure and function. The reported pathophysiological changes included a relatively greater degree of intravascular (ie, plasma) volume expansion for any level of increased arterial pressure or total peripheral resistance increase associated with a lesser plasma renin activity in the black patients as compared with white patients having essential hypertension.3 In addition, at any level of arterial pressure, renal blood flow was lower and renal vascular resistance was higher in the black patients.6 Moreover, the left ventricular mass was greater in black patients and was directly and significantly correlated with their systolic and diastolic pressures, as well as their total peripheral resistance levels.7 In addition, another report suggested that the pathological findings of the renal vascular lesions appeared similar to the gross appearance of keloid formation, which is more commonly encountered in black patients.8 These earlier pathophysiological comparisons were neither pursued further nor have they been related to other clinical findings also found to be more common in black patients. Among those observations were reports of greater antihypertensive therapeutic responsiveness to diuretic therapy, increased daily dietary sodium intake, and, perhaps, lesser potassium intake in the black population. Why those foregoing clinical reports were not pursued further seems to have been relegated to political or social issues that may be more likely subject material in after-dinner discussions.

There are a number of other important basic concerns that demand attention by investigators committed to serious translational investigation. For example, well documented are the findings reported in each of the more recent Joint National Committee reports that demonstrate a highly significant relationship that exists between the ubiquity of antihypertensive treatment and diminished morbidity and mortality relating to strokes and coronary heart disease.2 However, in contrast to that highly significant relationship, there are no reports detailing similar statistical relationships relating that same increase in antihypertensive treatment with the occurrence of cardiac or renal failure. In fact, with increased antihypertensive treatment, there has been a temporally direct and significant relation to an increasing prevalence of both cardiac and renal failure in both black and white hypertensive patients.2

We have suggested that one possible explanation for that disturbing relationship may be based on the greater dietary intake of salt during one’s lifetime, especially in black individuals who are genetically predisposed to these severe target organ complications of hypertension. There is ample...
experimental and clinical evidence available that clearly shows that prolonged dietary excess of salt is responsible for the structural and functional impairment of the heart and kidneys and is associated with an ever-increasing clinical prevalence of cardiac failure and end-stage renal disease.9,10 To this end, we should be stimulated by the present exciting report of Selassie et al in this journal.4 Certainly, in this era when electronic medical databases are used so imaginatively with large clinical populations by primary care clinics with hypertensive specialists reported in this journal, these and other important areas should prove to be extremely fertile fields for further detailed clinical investigation too long neglected. Ultimately, tangible and fundamental answers must be forthcoming to explain why the black population develops hypertension more frequently and rapidly and, of course, which underlying mechanisms and risk factors exist that will explain the more rapid and devastating natural history and course of their disease.

Disclosures
None.

References
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Hypertension. 2011;58:546-547; originally published online September 12, 2011;
doi: 10.1161/HYPERTENSIONAHA.111.178541

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