Editorial Comment

Ethnic Disparities in Risk Factors for Stroke
What Are the Implications?

In the current issue of Stroke, 2 studies report ethnic differences in stroke prevalence and risk factor prevalence and impact. Although it has been suggested that environmental–gene, socioeconomic, and behavioral interplays underlie ethnic disparities in cardiovascular risk factor profiles, reasons for observed ethnic differences in stroke incidence and mortality rates are not fully understood and little is known about ethnic disparities in stroke risk factors.

In a prevalence study in the USA, Henraya McGruder and colleagues analyzed racial and ethnic differences in the prevalence of cardiovascular risk factors among stroke survivors based on the 3 recent national household-based interview surveys. The study demonstrated substantial racial/ethnic differences in cardiovascular risk behaviors and medical history among stroke survivors. The study was focused mainly on risk factors, showing that blacks and Hispanics have a higher prevalence of obesity and are more likely to report diabetes, inadequate levels of physical activity, and hypertension (commonly blacks), and are less likely to report coronary heart disease than whites. However, the study also included important data on racial/ethnic differences in stroke prevalence, with the rates being almost 1.5 times higher in blacks compared to whites or Hispanics.

In a population-based case–control study in the UK, Cother Hajat and colleagues estimated population attributable and relative risks of ischemic stroke for 3 ethnic populations of South London: black African, black Caribbean, and white. The major strengths of this case–control study were population-based design, adjustment of risks for socioeconomic status (known to be associated with both ethnicity and risk of stroke), and estimates of population attributable risks by ethnicity. The key findings were higher prevalence of hypertension and diabetes mellitus but lower prevalence of current smoking and atrial fibrillation in black Caribbean people, and higher prevalence of hypertension but lower prevalence of atrial fibrillation, obesity, and diabetes mellitus in black African people compared to the other groups.

Before considering possible implications of these studies, it is worthwhile to assess their limitations. For example, in both papers, transient ischemic attack and coronary disease are regarded as risk factors for stroke, when in fact they are simply other manifestations of occlusive atherosclerotic disease. Hence, not surprisingly, they are extremely closely associated with stroke, since stroke is in effect just another manifestation of occlusive atherosclerotic disease. Second, analyses based on self-reported risk factor information may substantially underestimate the impact of the risk factors for a number of reasons. For example, patient self-reports of hypertension will underestimate by more than half the role of nonoptimal blood pressure, since hypertension often goes undiagnosed. Thus, for example, it has been recently shown that nonoptimal blood pressure is responsible for about 60% of stroke globally, which is substantially more than estimated in the London paper. Underestimating the impact of risk factors will underestimate their role in determining ethnic disparities. Also, in this case–control study, there are questions about the study base (eg, undersampling in some subgroups of controls). In the prevalence study in the USA, no data were available on the timing and type of stroke among survivors, thus leaving concerns about possible selection bias due to known ethnic differences in stroke incidence and survival.

Although results of these 2 studies cannot be compared directly because of differences in study designs, they do provide broad evidence for substantial ethnic disparities in risk factors for stroke. For example, cigarette smoking and atrial fibrillation are less prevalent in black and other minority populations in the USA and UK compared to white populations, while hypertension and diabetes mellitus are more prevalent. Overall, the observed ethnic differences in risk factor profiles in the 2 studies are broadly comparable with previous observations. These inequities may explain why blacks tend to have more small-vessel cerebrovascular pathology than whites. These ethnic differences in stroke risk factors, prevalence, and management are likely to determine ethnic disparities in stroke incidence and mortality observed in many countries and, together with broader demographic and socioeconomic changes, may have contributed to changing patterns of stroke epidemiology in recent decades.

As noted by Cother Hajat and colleagues, the impact of a particular risk factor in an ethnic or indeed any other population subgroup is best judged by the population attributable risk, which can be thought of simply as the amount of disease in a population that can be attributed to that risk factor. This measure is principally determined by relative risk associations (which are often remarkably consistent across ethnic and other subgroups) and prevalence (which tends to vary widely reflecting different social, cultural, and economic determinants). This mixed nature of the components means that risk factor impact is likely to change in any population subgroup, often driven by changes in prevalence. However, when subgroups are defined by ethnicity, unfortunately the reasons for differences are often wrongly ascribed to “genetic differences” between ethnicities, when in fact the
biological effects of the risk factors may be remarkably consistent.25

What then is the clinical and public health relevance of differing population attributable risks or differing prevalence levels across ethnic groups? This clearly depends on the level of the intervention that one considers. At the broadest level, there is little relevance for population-wide interventions, such as legislation to reduce salt content in processed foods, since by their very nature such interventions affect all population groups. Similarly, these findings probably have comparatively little implication for individual clinical care, since one would tend to address individual clinical risk factors or combinations directly and the marginal benefits for prognostic or therapeutics are probably modest. However, these findings do have implications for community-based programs, which typically work best when targeted to those most at risk and tailored to the relevant ethnic and cultural groups. For example, a community-based cardiovascular disease prevention program in the USA might need to focus on blacks, and should therefore have a large component focused on blood pressure relevant interventions.

Clearly, efforts to control established cardiovascular risk factors must be pursued in all populations regardless of race and ethnicity. The challenge ahead remains development and implementation of these strategies that would take into account the country-specific social, political, economic, and physical environment. Future research could well benefit from trialing differing ways to increase uptake of interventions and lifestyle changes among different ethnic groups, thus helping reverse these disparities.

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References
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