The Socioeconomics of Hypertension

How $50 000 May Buy a Drop in Blood Pressure

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The Socioeconomic status (SES) is a known risk factor for cardiovascular disease. However, unlike traditional Framingham risk factors, SES does not directly impact the cardiovascular system but exerts its cardiovascular effects via a complex interaction of biobehavioral factors, such as exercise and diet. Because these mediating behavioral factors are potentially modifiable, elucidating the pathways by which SES influences cardiovascular disease offers important opportunities for preventive interventions that may, in turn, help to address health disparities among social groups.

It was toward this aim of understanding the role of mediating factors in the association between SES and hypertension that Brummett et al. conducted a large cross-sectional study using data from Wave IV (2007–2009) of the National Longitudinal Study of Adolescent Health. Using path models, they found that, in 14 299 “nationally representative” Americans aged 24 to 35 years: (1) higher household income and being married were independently associated with lower systolic blood pressure (SBP), whereas older age, male sex, black ethnicity, higher body mass index (BMI), greater waist circumference, smoking, and higher alcohol intake were independently associated with higher SBP in multivariable modeling; (2) higher household income was associated with lower SBP by way of lower resting heart rate (offsetting the SBP-raising effects of increased alcohol consumption) and remained inversely associated with SBP even after adjusting for all measured covariates, and, in fact, each $50 000 increase was related to a decrease in SBP of 0.61 mm Hg; and (3) higher education level was similarly associated with lower SBP by way of lower BMI, smaller waist circumference, and lower resting heart rate, but was no longer significantly related to SBP after accounting for these indirect effects. The authors concluded that increased BMI, particularly central obesity, and higher resting heart rate were important mediators of the association between lower SES and higher SBP.

Higher heart rate was identified by Brummett et al. as another important link between lower SES and higher SBP (Figure). Higher heart rate may be a surrogate measure of increased psychosocial stress or reduced physical fitness. A host of adverse psychosocial factors have been found to be increased in lower SES groups, including hostility, depression, and social isolation, and these stressors may lead to higher resting heart rates and increased SBP via sympathetic overactivity. Although psychosocial factors were not directly measured in the current study, the “protective” effect of being married suggested a role for social support, and previous studies have shown that perceived stress and depression were significant mediators of the association between lower SES and increased SBP. The significance of these findings lies in the potential for preventive interventions, such as promoting healthy stress coping skills and regular aerobic exercise.

Taken together, it is tempting to oversimplify the association between SES and SBP as such: socially disadvantaged individuals face job and neighborhood stressors, which, coupled with lack of knowledge and limited resources, drive these individuals to low-cost, high-calorie fast food options, resulting in obesity, the metabolic syndrome, and hypertension; chronic ill health may then limit job opportunities, creating even more stress and perpetuating the vicious cycle. The association may not, however, be that straightforward for the following reasons. Firstly, the SES-obesity-SBP link can change in its direction of association—in undeveloped or developing countries, BMI increases with higher SES and contributes to a direct association between higher SES and...
higher SBP. Obesity is not, therefore, an inevitable consequence of social deprivation, and much depends on the state of development of the country. Second, variations in SES may have different effects depending on sex and ethnicity. The relationship between lower SES and higher SBP is stronger in women than in men. Among women alone, SES-SBP inverse associations are stronger in black Americans than in Mexican Americans. Third, even if SES disparities were eliminated among sexes and ethnicities, the prevalence of hypertension would still be greater in black than in white Americans, suggesting inherent genetic predisposition or other unmeasured explanatory factors. Some of these unmeasured factors are sodium and potassium intake, psychosocial stress factors not captured by heart rate, stress coping mechanisms, and complex neighborhood-specific social effects not reflected in education level or household income (Figure).

It is intriguing that income, but not education, retained an independent effect on SBP after adjusting for all measured covariates in the study by Brummett et al: earning $50,000 more was related to having a 0.6-mm Hg lower SBP. As a measure of SES, education has the advantages of being reliably recalled and unaffected by later adult health so that reverse causality is less of an issue (poor adult health cannot change previous educational attainment in healthy adolescence). Income, on the other hand, may be affected by adult health in that poor health may lead to lower income and vice versa (problem of reverse causality). Conversely, higher income may reflect not only greater occupational earning and purchasing power but may incorporate more proxy effects of better SES and better overall health, explaining how earning more may buy a lower SBP or, perhaps, how a lower SBP can buy greater earnings.

Low SES should be recognized as a potent risk factor for hypertension, even among young adults in the United States. Although SES, per se, is not regarded as a “treatable” risk factor, its impact on blood pressure may be modified by better understanding of, and intervening on, its mediating mechanisms. The study by Brummett et al has contributed to this understanding, revealing obesity and increased heart rate (as a measure of psychosocial stress or poor physical fitness, both potentially leading to increased sympathetic traffic to the heart and vasculature) as key modifiable correlates of lower SES and higher SBP. Given the cross-sectional nature of these data, however, causality cannot be proven, and the impact of losing weight or reducing heart rate (via better stress management or aerobic exercise) on SES-related differences in SBP cannot be conclusively determined. Nonetheless, the Hypertension Detection and Follow-Up Program has demonstrated that intensive blood pressure control has the greatest positive impact in hypertensive individuals of the lowest SES, attenuating the SES-related excess mortality in hypertension. Thus, regardless of mechanism for hypertension, clinicians should be reminded that aggressive blood pressure control is an available and effective means to address social disparities in outcomes among hypertensive individuals.

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References

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