Socioeconomic Status and Subclinical Atherosclerosis
Are We Closing Disparity Gaps?

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Editorial

In the recent decades, we have experienced large reductions in the prevalence of several major cardiovascular disease (CVD) risk factors, including hypertension and dyslipidemia along with improved medical management for stroke and CVD, and large reductions in CVD-related morbidity and mortality. These health improvements have been accompanied by gains in income and education although a widening of the socioeconomic status (SES) gap has occurred. However, the improved stroke CVD health status may not exist across all segments of society and which groups lack the health benefits largely remains unknown. In addition to traditional CVD risk factors and genetics, SES is likely one of the most critical determinants of CVD, but the mechanism by which SES influence CVD health remains unexplained. Furthermore, trends in SES factors have yet to be carefully examined in relation to health plan appropriate actions to close gaps in SES-related health disparities.

Education, occupation, and income are some of the important SES indicators that have been associated with CVD in numerous studies. Adverse socioeconomic position across the life-course increases CVD risk; therefore, identification of preclinical disease is important for early detection and prevention of CVD and stroke. Yet little information is available on the relationship between SES and early manifestations of atherosclerotic vascular disease across the life-course.

In this issue of Stroke, Thurston et al have demonstrated that low SES overall and particularly consistently low SES across midlife was associated with increased carotid intima-media thickness and a greater extent of carotid plaque among women free of overt CVD in the Study Women’s Health Across the Nation (SWAN) study, a large community-based, multicenter, multiethnic, longitudinal study designed to characterize the menopausal transition. Low education, low income, and financial strain were among the SES measures examined in this study. The SES status was measured annually >12 years, which was a unique aspect of this research. The main results showed that low education, sustained financial strain, and low income were each related to subclinical atherosclerosis. These results complemented previous research conducted in the Atherosclerosis Risk in Communities (ARIC) Study where lower cumulative individual-level SES across the life-course was associated with a greater burden of subclinical atherosclerosis. Financial strain including affordability of basics, such as food, housing, and medical care, better likely than other measures accounts for the adequacy of financial resources to meet basic needs and protect against income distresses. These results address a considerable gap in our understanding of the role of SES on vascular health disparity and especially among women.

The significant relationship between individual-level SES and subclinical atherosclerosis has been reported more frequently in men than in women. Because the relationship between SES factors and subclinical atherosclerosis can differ by sex, sex-specific or sex-stratified results are appropriate avenues of investigations. Thurston et al presented the data for middle-aged women (42–52 years). Midlife is the critical life phase for women. It includes many fluctuations, such as the menopausal transition, changing social roles with caretaking for aging parents, retirement, all of which may affect financial status. Therefore, midlife health status is particularly relevant for vascular health in women, as shown in this study that economic factors in addition to biological factors are associated with an increased risk of subclinical atherosclerosis.

Numerous pathways linking SES and CVD may exist. Traditional vascular risk factors and health-related behavior may be moderators or mediators of this association. Although Thurston et al found the association between SES and subclinical atherosclerosis while adjusting for many traditional vascular risk factors, other important and less traditional vascular factors not accounted for in this study may have an important role. Although the psychological factors, such as depressive symptoms, anxiety, positive and negative affect, were considered in the SWAN study, their association with subclinical atherosclerosis was not observed except for positive affect. However, these factors do not represent the full spectrum of psychosocial factors. Additional factors, such as anger, sustained anxiety, and alexithymia, have been associated with increased carotid intima-media thickness in the Multi-Ethnic Study of Atherosclerosis (MESA) and in HIV-infected patients. This SWAN analysis did not assess the mechanistic pathway for the relationship between low SES and subclinical atherosclerosis. Many potential not only health-damaging factors but also health-promoting factors, such as social support, are important predictors of stroke and CVD. The exact mechanism by which these factors affect development of atherosclerosis is not well understood but is likely to be complex.
Alterations in the autonomic nervous system as a result of chronic health-damaging stressors is one of the common mechanisms proposed to link SES and other psychological and cultural factors to cardiovascular responses and to an increased risk of atherosclerosis. These factors may play a similar or even a larger role in the life-course of stroke and CVD than traditional risk factors and need to be considered in future studies.

SES is among the most fundamental determinant of health status. The relationships among SES, health, and disease can be attributed to the combined effects of disparities in age, sex, race/ethnicity, health-related behaviors, environmental factors, social structures, and the access and delivery of healthcare. Most of the health-related behavioral factors are modifiable. Reducing disparities in SES and disease-modifiable factors is a priority of public health efforts. Studies like Thurston et al will help us understand the cumulative SES effects on preclinical CVD and ultimately help us close the gaps in health disparities. Clearly defined conceptual frameworks of the life-span causal pathways leading to disparities in preclinical CVD that consider early-life factors, parental SES, individual-level and neighborhood SES, inequalities in health services, work-related factors, biological, and psychological factors are critically needed. Only then will we start to remedy health inequalities while respecting cultural diversity.

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References

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