

Invited Commentary

Socioeconomic Status and Heart Health—Time to Tackle the Gradient

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*Averages are no consolation to those who have been left behind.*Angus Deaton¹

One of the most remarkable achievements of the 20th century is the dramatic reduction in death rates from cardiovascular diseases (CVDs) in the United States and several high-income countries. For example, in 1950, the age-adjusted mortality rate per 100 000 population for heart disease was 699 in men and 487 in women in the United States.² By 2017, these rates had declined to 209 in men and 130 in women, representing more than a 70% reduction.³ Life expectancy at birth for the total US population also improved substantially over that period from 68.2 years in 1950 to 78.6 years in 2017.³ Although invaluable, these summary measures of health are national averages that can mask important differences for population groups defined by age, sex, race/ethnicity, geography, rural or urban residence, neighborhood characteristics, and socioeconomic status (SES).

For example, in 2017, the age-adjusted mortality rate for heart disease was highest in non-Hispanic black men (n = 265) and women (n = 166), nearly 2.5-fold greater than the rates in non-Hispanic Asian or Pacific Islander men (n = 108) and women (n = 68).³ Examination of trends in premature CVD mortality (defined as heart disease and stroke deaths in adults aged 25-64 years) from 2000 to 2015 showed substantial increases in age-standardized mortality rates among American Indian or Alaska Native men and women aged 25 to 47 years; in white women aged 25 to 49 years, the rates predominantly increased in rural counties and counties with low-SES markers.⁴ The common thread in these populations with adverse CVD mortality findings is not their race/ethnicity but the social, economic, and environmental determinants of health as well as the comorbid CVD risk factor burden. But how do SES markers get under the skin, so to speak, to directly or indirectly change these adverse CVD mortality trends?

Mechanisms and Pathways of SES and Heart Disease

Several mechanisms have been examined and proposed as the basis for the observed associations between low-SES markers and heart health. Examined markers include those at the individual level (ie, educational attainment, income, employment status, occupation type, adverse childhood experiences, and differential exposure to threat and stress in both the work and home environment) and those at the social or population level such as income inequality, neighborhood deprivation, structural racism, and adverse environmental exposures.

In an extensive review of mechanisms that underlie the association between low SES in childhood and diseases in adulthood, Morris et al⁵ cited evidence to support a role for enhanced peripheral inflammation; a persistent hypothalamic-pituitary-adrenal axis dysregulation, especially in the setting

of children with low SES in inadequate housing conditions; and the implication of a low-SES for epigenetic changes, particularly in DNA methylation.⁵ Other recent data also show that telomere shortening may be a biomarker for neighborhood deprivation and a mediator of bad health outcomes and increased mortality.⁶ Another important advancement in the understanding of these mechanisms is the concept of allostatic load, a cumulative measure of physiological dysregulation and a marker of the overall wear and tear on physiological regulatory systems.⁷ These mechanisms are by no means comprehensive; however, they provide a glimpse of the biological plausibility for the role of low SES in adverse CVD outcomes.

SES Association With Coronary Heart Disease Mortality

A question that remains unanswered is, what proportion of CVD morbidity and mortality is attributable to low SES, beyond the contributions of traditional CVD risk factors? In this issue of *JAMA Cardiology*, Hamad et al⁸ used the Cardiovascular Disease Policy Model to estimate the excess burden of early coronary heart disease (CHD) among adults with low SES and to identify the proportion of this excess burden attributable to traditional CHD risk factors and to other factors associated with low SES. The authors defined low SES as income below 150% of the federal poverty level or less than a high school educational attainment. An important strength of the study is the use of model inputs derived from nationally representative US data and cohort studies of incident CHD.⁸

The study by Hamad et al⁸ has several important findings. First, the 31.2 million US adults aged 35 to 64 years with low SES had double the rate of myocardial infarction and CHD deaths compared with those with higher SES. Second, the higher burden of traditional CHD risk factors in adults with low SES explained only 40% of the excess events, suggesting that the remaining 60% of excess events were attributable to factors associated with low SES. Third, in a simulated cohort of 1.3 million adults with low SES who were 35 years old in 2015, the models estimated that 250 000 adults would develop CHD by age 65 years, and nearly half of this burden would occur in excess of that expected for persons with higher SES.⁸ Hamad et al⁸ concluded that the proportion of the US population aged 35 to 64 years with low SES is substantial and that an explicit focus on this group is needed to address the cardiovascular disparities associated with low SES.

Tackling SES in Disparities Elimination

Reducing and eliminating disparities in cardiovascular health require a multidisciplinary, collaborative approach that engages patients, health care practitioners, health systems, policy makers, and entire communities at multiple levels. Also required is a focus on identifying individuals and communities at greatest risk, such as those with low-SES markers, and then matching the dose and intensity of interventions to the mag-

nitude of the disparities. Providing equal access and equal care to all is important but, by itself, will not eliminate disparities. The persisting challenges in eliminating CVD disparities may be attributed, in part, to the paucity of interventions that address social determinants of health.⁹

The health impact pyramid framework of the Centers for Disease Control and Prevention suggests that, although implementing interventions at all levels of the ecological model can deliver the maximum possible sustained public health benefit, interventions with the greatest value for population health are those that focus on the socioeconomic factors of health.¹⁰ In addition, taking a life-course perspective in designing interventions to tackle low-SES factors and the social gradient is crucial because the antecedents of adult chronic diseases, including CHD, begin in childhood and are associated with low SES and social and environmental adversity experienced in childhood.

Leaving No One Behind

As Nobel laureate Angus Deaton pointed out in the book *The Great Escape: Health, Wealth, and the Origins of Inequality*,¹ life is better now than at almost any time in history, but he also emphasized that his thesis is far from complete. We have evidence that the dramatic decreases in CHD mortality seen in the United States in the past century have now stalled, and for many African American men and women, American Indian or Alaska Native men and women, and middle-aged white women in rural counties and counties with low-SES markers, CVD mortality rates remain too high or are increasing and not declining as in the rest of the country. Addressing traditional CVD risk factors is necessary but not sufficient for preventing premature CVD mortality. The time has now come to also tackle the social, environmental, and socioeconomic determinants of health and associated cardiovascular health disparities.

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