Title

Social Determinants of Risk and Outcomes for Cardiovascular Disease

Journal

Circulation, 132(9)

ISSN

0009-7322

Authors

Havranek, Edward P

Mujahid, Mahasin S

Barr, Donald A

et al.

Publication Date

2015-09-01

DOI

10.1161/cir.0000000000000228

Peer reviewed

AHA Scientific Statement

Social Determinants of Risk and Outcomes for Cardiovascular Disease

A Scientific Statement From the American Heart Association

Edward P. Havranek, MD, FAHA, Chair; Mahasin S. Mujahid, PhD, MS, Co-Chair; Donald A. Barr, MD, PhD; Irene V. Blair, PhD; Meryl S. Cohen, MD, FAHA; Salvador Cruz-Flores, MD, FAHA; George Davey-Smith, MA(Oxon), MD, BChir(Cantab), MSc(Lond); Cheryl R. Dennison-Himmelfarb, RN, PhD, FAHA; Michael S. Lauer, MD, FAHA; Debra W. Lockwood; Milagros Rosal, PhD; Clyde W. Yancy, MD, FAHA; on behalf of the American Heart Association Council on Quality of Care and Outcomes Research, Council on Epidemiology and Prevention, Council on Cardiovascular and Stroke Nursing, Council on Lifestyle and Cardiometabolic Health, and Stroke Council

An Institute of Medicine report titled *U.S. Health in International Perspective: Shorter Lives, Poorer Health* documents the decline in the health status of Americans relative to people in other high-income countries, concluding that "Americans are dying and suffering from illness and injury at rates that are demonstrably unnecessary."[1] The report blames many factors, "adverse economic and social conditions" among them. In an editorial in *Science* discussing the findings of the Institute of Medicine report, Bayer et al[2] call for a national commission on health "to address the social causes that have put the USA last among comparable nations."

Although mortality from cardiovascular disease (CVD) in the United States has been on a linear decline since the 1970s, the burden remains high. It accounted for 31.9% of deaths in 2010.[3]

There is general agreement that the decline is the result, in equal measure, of advances in prevention and advances in treatment. These advances in turn rest on dramatic successes in efforts to understand the biology of CVD that began in the late 1940s.[4,5] It has been assumed that the steady downward trend in mortality will continue into the future as further breakthroughs in biological science lead to further advances in prevention and treatment. This view of the future may not be warranted.

The prevalence of CVD in the United States is expected to rise 10% between 2010 and 2030.[6] This change in the trajectory of cardiovascular burden is the result not only of an aging population but also of a dramatic rise over the past 25 years in obesity and the hypertension, diabetes mellitus, and physical inactivity that accompany weight gain. Although there is no consensus on the precise causes of the obesity epidemic, a dramatic change in the underlying biology of Americans is not postulated. More likely culprits are changes in societal and environmental conditions that have led to changes in diet and physical activity. At the same time, there is increasing awareness that the benefits of advances in prevention and treatment have not been shared equally across economic, racial, and ethnic groups in the United States. Overall population health cannot improve if parts of the population do not benefit from improvements in prevention and treatment.

The purpose of this statement is to increase awareness of the influence of social factors on the incidence, treatment, and outcomes of CVD; to summarize the current state of knowledge about these factors; and to suggest future directions in research, particularly research on effective interventions to attenuate or eliminate these adverse social influences. The statement is not intended to be a comprehensive review;

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on February 4, 2015, and the American Heart Association Executive Committee on May 22, 2015. A copy of the document is available at http://my.americanheart.org/statements by selecting either the "By Topic" link or the "By Publication Date" link. To purchase additional reprints, call 843-216-2533 or e-mail kelle.ramsay@wolterskluwer.com.

The American Heart Association requests that this document be cited as follows: Havranek EP, Mujahid MS, Barr DA, Blair IV, Cohen MS, Cruz-Flores S, Davey-Smith G, Dennison-Himmelfarb CR, Lauer MS, Lockwood DW, Rosal M, Yancy CW; on behalf of the American Heart Association Council on Quality of Care and Outcomes Research, Council on Epidemiology and Prevention, Council on Cardiovascular and Stroke Nursing, Council on Lifestyle and Cardiometabolic Health, and Stroke Council. Social determinants of risk and outcomes for cardiovascular disease: a scientific statement from the American Heart Association. *Circulation.* 2015;132:XXX–XXX.

Expert peer review of AHA Scientific Statements is conducted by the AHA Office of Science Operations. For more on AHA statements and guidelines development, visit http://my.americanheart.org/statements and select the "Policies and Development" link.

(*Circulation*. 2015;132:00-00. DOI: 10.1161/CIR.0000000000000228.)

Table 1. Social Determinants of Health

SEP
Race, ethnicity
Social support
Culture and language
Access to care
Residential environment

SEP indicates socioeconomic position.

references are intended to be illustrative and to highlight significant knowledge in the field. The premise underlying this scientific statement is that, at present, the most significant opportunities for reducing death and disability from CVD in the United States lie with addressing the social determinants of cardiovascular outcomes. Although social determinants are most often invoked in discussions of inequalities or disparities in health, we take a broader view that social factors can and do affect cardiovascular health in all. Thus, a consideration of the role of social determinants is essential if we are to achieve the American Heart Association 2020 Impact Goals: to improve cardiovascular health of all Americans by 20% while reducing deaths from CVD and stroke by 20%.[7]

Defining Social Determinants of Health

The World Health Organization defines the social determinants of health quite broadly as "the circumstances in which people are born, grow, live, work, and age, and the systems put in place to deal with illness."[8] This definition encompasses the view that health and illness are not distributed randomly throughout human society, and neither are resources to prevent illness and its effects. Instead, they cluster at the intersections of social, economic, environmental, and interpersonal forces.

Cataloging the Social Determinants of Health

Social determinants are highly interrelated and therefore difficult to catalog. Given our focus on CVD in the United States, this statement considers socioeconomic position (SEP; encompassing wealth and income, education, employment/occupational status, and other factors), race and ethnicity, social support (including social networks), culture (including language), access to medical care, and residential environments (Table 1). We additionally consider the psychological, behavioral, and biological mechanisms through which social determinants precipitate and perpetuate CVD.

SEP and CVD

Defining SEP

There are several ways to describe and measure social and economic conditions. The terms social class, social stratification, and social or socioeconomic status are used frequently and often interchangeably. Here, we use SEP, defined as the socially derived economic factors that influence what positions individuals or groups hold within the stratified structure of a society.[9] Relations among groups within a society are determined largely by material circumstances, which in turn are determined by the relations these groups have with systems of economic production. Members of advantaged groups control resources (whether material, economic, political, social, or cultural) in a way that may exclude and dominate the disadvantaged. Unequal distribution and control over resources influence patterns of exposures, which act at different stages of the life course, resulting in unequal distribution of disease in different groups within a society. Health and SEP are seen as inextricably linked, with health itself seen as a marker of SEP in some schemes (Table 2). It is important to highlight that, although measured at the individual level, SEP is determined at least partly by structural relations between groups within society. For example, the level of education attained by an individual may be constrained by educational opportunities available to a particular group. We discuss area-level factors in more detail in the Residential Environments section.

Measuring SEP

There is no single best indicator of SEP. Each indicator of SEP emphasizes a particular aspect of social stratification, which may be more or less relevant to different health outcomes and at different stages in the life course.[9] Individual-level indicators of SEP include income, education, and occupation-based indicators, and ideally, they should be considered simultaneously. Others[10] have emphasized that SEP should consider both actual resources and status as determined by prestige or rank-related characteristics. This multidimensional nature of SEP has been emphasized in the work of the Commission on the Measurement of Economic Performance and Social Progress[11] headed by the Nobel Laureate Joseph Stiglitz. Although the Commission was focused on a critique of gross domestic product as a measure of the performance of societies or nations, some of its conclusions can be applied to individuals. Broadly, the commission concluded that well-being is determined by a number of interwoven dimensions (Table 2).

Associations With CVD

Three measures of SEP have been explored extensively with regard to their relationship to cardiovascular health: education, income, and occupation. Broadly speaking, lower SEP in the United States is associated with a greater prevalence of CVD risk factors and a higher incidence of and mortality resulting from CVD. We highlight evidence linking measures of SEP with cardiovascular health, including early childhood socioeconomic conditions. Detailed and comprehensive reviews on this topic can be found elsewhere.[12–14]

Table 2. Markers of SEP

Material conditions (based on income and wealth)
Health
Education
Access to valued personal activities (eg, work)
Political voice
Social connections
Environment
Physical insecurity (crime, violence)

SEP indicates socioeconomic position.

Education

Education, the most used indicator of SEP in the United States, provides the most consistent results in relation to CVD outcomes.[14,15] Lower levels of educational attainment are associated with a higher prevalence of cardiovascular risk factors (discussed in more detail in the Mechanisms Mediating the Relationship Between Societal Conditions and CVD section), higher incidence of cardiovascular events, and higher cardiovascular mortality, independent of sociodemographic factors.[14,16] In relation to CVD mortality, Mackenbach et al[17] examined educational differences in ischemic heart disease, cerebrovascular disease, and total CVD mortality in the United States and 11 Western European countries. They found higher mortality among individuals with lower education in all countries; however, the relative and absolute magnitude varied across countries.

Disparities associated with educational attainment have widened over time. Meara et al[18] used US Census and death certificate data to show that the disparity in life expectancy at 25 years of age between those with low educational attainment (≤12 years) and those with high educational attainment widened by 0.9 years from the 1980s to the 1990s. A widening education-based difference in cardiovascular death was responsible for 17.4% of the overall gap in life expectancy, second only to cancer. Similar increases in education disparities in life expectancy were documented between 1996 and 2006.[19]

Low health literacy and numeracy might in part mediate the relationship between lesser education and CVD, with low health literacy associated with having less than a high school education and with poor health outcomes.[20] Interventions that improve self-care behavior, risk factor control, or cardiovascular outcomes in those with low health literacy or numeracy are generally lacking. More study has been reported in heart failure, for which interventions have generally been resource intensive and results have been mixed.[21]

Income and Income Inequality

Both income and income inequality have been studied in relation to cardiovascular health. Other measures of material circumstances beyond income, particularly accumulated wealth, have not been adequately considered in the literature. Findings for associations between income and cardiovascular health parallel those for education, with the caveat that many studies document nonlinearity in the association of income and cardiovascular outcomes.[14] Data on >500 000 men and women from the National Longitudinal Mortality Study showed similar associations between education and income in relation to all-cause and cardiovascular mortality. After adjustment for sociodemographic factors, there was a 40% to 50% decrease in mortality with increasing levels of family income.[19] Whether differences in cardiovascular outcomes are becoming more or less pronounced over time is unclear because income is more unstable and difficult to measure than education.[22]

Income inequality within societies has grown in recent decades, particularly in high-income countries such as the United States, and the social consequence of this reality is becoming an important political issue. Harper et al[22] found no evidence of consistent associations between income inequality and cardiovascular health, including prevalence of CVD risk factors and CVD trends.

Employment/Occupational Status

The relationship between occupation and CVD is less clear than it is for education or income. The Whitehall study was most influential in the description of differences in cardiovascular mortality by job classification. In the first cohort initiated in the 1970s, Marmot and colleagues[23] followed up a group of 17 530 British civil servants in London, all of whom had office-based jobs and none of whom were considered to be economically disadvantaged. At the 10-year follow-up, mortality resulting from coronary heart disease was 2.2%, 3.6%, 4.9%, and 6.7% across job grade from the highest to the lowest; these differences remained significant after controlling for age, height, smoking, systolic blood pressure, cholesterol, and blood sugar. Comparable results have been documented with 25 years of follow-up.[24] In the United States, Leigh and Du[25] used data from the Health and Retirement Study to assess for an independent relationship between lifetime occupations grouped into 15 categories and prevalent hypertension, concluding that "in general, higher status occupations were associated with less hypertension." Another study using *National Health and Nutrition Examination Survey* (*NHANES*) data[26] tended to support this finding, additionally noting that protective service workers such as police and firefighters had the lowest rates of treatment for established hypertension. In general, there is a paucity of data on the relationship between occupation and cardiovascular morbidity and mortality in the United States.

In addition to these relationships between type of employment and CVD, a relationship between unemployment and CVD has been postulated. Epidemiological studies of unemployment and health are particularly difficult because of potential "effect-cause" relationships, in which unemployment is a consequence of poor health rather than the reverse, and because of confounding by factors such as low educational attainment that might predict both unemployment and poor health. Nonetheless, the preponderance of evidence supports the position that job loss leads to illness. Studies of widespread labor downsizing, in which an individuals' health is not a factor, support the causal relationship between job loss and ill health.[27] At least for behavioral health issues, long-term longitudinal studies that gather health information before job loss have also supported a causal relationship.[28] Studies specifically in CVD have been performed. Dupre and colleagues[29] used data from the prospective Health and Retirement Study to study the relationship between unemployment and incident myocardial infarction. After adjustment for risk factors and sociodemographics, the hazard ratio for myocardial infarction was highest in the first year of unemployment and increased with the number of job losses.

The possible psychological and biological mechanisms responsible for the relationship between occupation, unemployment, and CVD are discussed in the Mechanisms Mediating the Relationship Between Societal Conditions and CVD section.

Life-Course Context of SEP

For CVD, poor socioeconomic conditions in early life appear to make an important contribution to disease risk in adulthood, especially when early-life factors influence the developmental trajectories of important adult risk factors.[22] A systematic review

of 40 studies investigating associations between childhood socioeconomic circumstances and ischemic heart disease, stroke, or combined CVD in adulthood reported that the majority of studies show robust associations of poorer childhood circumstances and CVD, although findings differed across types of CVD, socioeconomic measures, and sex.[12] Galobardes et al[9] reported heterogeneity in the strength of association of SEP indicators with specific CVDs, which suggests specificity of the pathogenic links between socioeconomically patterned exposures early in life and adult disease outcomes.[22] Perhaps more important than the direct effect of early-life socioeconomic factors on CVD is their potential effect on the development of conventional risk factors.[22] Reviews of studies focusing on the role of childhood socioeconomic conditions, usually indicated by the occupation or education of the parents, have found consistent evidence of an association with CVD risk factors such as blood pressure, lipid levels, body mass index (BMI), fibrinogen, smoking, physical activity, and alcohol consumption.[22] Investigation of the impact of social mobility on social class inequalities in all-cause mortality has suggested a cumulative effect of lifetime socioeconomic experience. However, evidence that upward or downward socioeconomic mobility may play an important role in generating or substantially magnifying CVD differences is limited.[22]

Further evidence suggests that the effect of early life socioeconomic conditions may depend on interactions with other risk factors in later life.[22] Secular changes in CVD differentials are more congruent with increasing socioeconomic differences in cigarette smoking and consumption of micronutrients than with trends in socioeconomic differentials in infant mortality or height,[30,31] understood as potential markers of early-life circumstances and related outcomes such as fetal growth. Thus, the interactions among early-life socioeconomic environments and risk factor trajectories seem to influence the development and maintenance of health behaviors and their cumulative biological sequelae as a major life-course process linking early-life SEP to CVD.[22] The Mechanisms Mediating the Relationship Between Societal Conditions and CVD section provides a detailed discussion.

SEP and CVD Risk Prediction

Given the substantial evidence linking SEP and CVD and findings that suggest that the Framingham risk score overestimates the risk of coronary heart disease in high-SEP individuals and underestimates the risk in low–socioeconomic status individuals, recent studies have begun to evaluate the potential benefit of including SEP in risk prediction models.[32] Using data from the Atherosclerosis Risk in Communities (ARIC) Study and NHANES linked to the National Death Index, Fiscella and colleagues[33] documented improvements in the calibration and reduction of bias in the Framingham risk model. These types of investigations should continue in future research.

Recommendations and Conclusions: SEP

- No single parameter fully captures SEP; income, education, and occupation have been used successfully.
- SEP measures may vary by race/ethnic groups, and these synergistic effects should be considered.
- Novel markers of SEP should be investigated for broader use in understanding CVD.

Race/Ethnicity, Racism, and CVD

For this statement, we use the terms race and ethnicity as constructs with very little biological or genetic basis but as constructs shaped by the social, economic, and political forces of societies.[4,5] Differences in health by race and ethnicity are a major public health concern.[1] On the basis of projections from the US Census Bureau, the population of non-Hispanic whites will almost double by the year 2050, with Asians and Hispanics the fastest-growing populations in the United States. Racial and ethnic minorities are disproportionately burdened with poor health across a variety of different outcomes, and given the significant increase in these populations in the United States, attention to the health needs of these groups is essential.

Racial/ethnic differences in cardiovascular health have been documented extensively.[16] For example, in the Eight Americas Study, the authors identified 7 distinct groups within the United States based on race, geographic location, and income and found significant differences in life expectancy.[34] These groups are, in order of decreasing life expectancy, Asian Americans (84.9 years), whites living in rural Northern Plains/Dakotas, low-income whites in Appalachia and the Mississippi Valley, western Native Americans, middle-income blacks, southern rural blacks, and blacks in poor urban areas (71.1 years), representing a 14-year difference between the highest and lowest group. CVD was the greatest source of differences in life expectancy. On the basis of the latest report of the American Heart Association heart and stroke disease statistics, blacks are 2 to 3 times more likely to die of heart disease compared with whites, and blacks and other racial/ethnic minorities have higher rates of premature death resulting from CVD and higher CVD risk factors.[3] Declines in CVD mortality have not eliminated racial and ethnic differences in CVD; they remain constant.

Although public opinion polls show that levels of overt or explicit racism have declined over the past 5 decades, there are clear indicators that members of ethnic minority groups, particularly blacks, must endure everyday slights and offenses that undermine health. Specific to CVD, studies have investigated the links between self-reported experiences of racism and both blood pressure and cardiovascular reactivity. The evidence to date shows limited direct relationships between reported racism and hypertension diagnosis or resting blood pressure measures.[35] There is much stronger evidence, however, for ambulatory blood pressure monitoring, with all 6 known studies finding a positive relation between ambulatory blood pressure (particularly at night) and reports of racism or discrimination.[36] In the largest of these studies,[37] 357 black and Latino adults completed a measure of lifetime experiences of ethnic or racial discrimination and then wore an ambulatory blood pressure monitor until they returned the next day. Both nighttime systolic blood pressure and diastolic blood pressure were positively related to amount of reported racism, even after adjustment for patient demographics and self-reported general hostility. Higher levels of reported racism were also associated with a lower likelihood of nocturnal dipping (≥10% decrease in nighttime blood pressure). Additional research has found that past experiences of racism predict greater cardiovascular reactivity.[38] In 1 study, 165

black and white normotensive adults had their heart rates and blood pressures measured while they recalled an event that had made them angry. Participants who had earlier reported more experiences with discrimination were found to have greater heart rate and diastolic blood pressure reactivity during the recall task and slower recovery after the task, particularly if they were black and had a generally positive outlook on life (eg, low in cynicism or high in optimism).

Of great concern to society is the possibility that healthcare provider bias contributes to the problem.[39–43] Investigations of clinicians' ethnic and racial attitudes have shown that, similar to the general population, clinicians show little explicit or intentional bias but exhibit substantial bias in their implicit (unconscious) attitudes.[44–46] Theoretical models suggest that clinicians' implicit bias may affect their delivery of health care in 3 ways.[42,47,48] First, implicit bias may directly influence clinicians' decisions about their patients' medical treatment, with incorrect, often stereotypical assumptions leading to lower-quality care for minority than for white patients. A study by Schulman and colleagues[40] used scripted videotaped interviews of actors portraying patients with chest pain, finding that physicians were less likely to recommend catheterization for black women than for white men reporting the same symptoms. The authors found no difference in the rate of physician-recommended catheterization for black men and white men. Green and colleagues[45] found that resident clinicians with greater implicit bias were less likely to recommend thrombolytic therapy for a hypothetical black patient with myocardial infarction, but this did not occur when the patient was described as white. On the other hand, research on pediatric decision making[49,50] showed that some hypothetical decisions were associated with implicit bias but others were not. However, a study[51] with medical students failed to find any relation between clinical decisions in the hypothetical scenarios and the students' implicit bias. Although this work is often criticized on methodological grounds, an influential review of the literature by the Institute of Medicine[43] concluded that "bias, stereotyping, prejudice, and clinical uncertainty on the part of healthcare providers" may play a role in racial/ethnic health disparities. Thus, although proof of bias is difficult to achieve, it remains viable and of great concern.

The second route by which implicit bias may affect care processes is by producing lower-quality clinical interactions and communication between (more biased) clinicians and minority patients. Several studies[52–54] have found associations between clinicians' implicit bias and worse clinical interactions with black patients. Most relevant to CVD is a study by Blair and colleagues[44] in which primary care providers' levels of implicit race bias predicted differences between black and white patients' reports of their clinicians' patient centeredness, with black patients reporting less patient centeredness for clinicians previously categorized as having higher levels of implicit racial bias. Numerous studies have investigated patients' perceptions of bias and discrimination while receiving health care. A review of this literature[55] found that up to 52% of blacks, 13% of Latinos, and 6% of non-Hispanic whites have reported biased treatment based on their race or ethnicity. Perceptions of biased treatment in turn have been associated with reports of lower health, lower levels of self-care or adherence, interruptions in care, mistrust of clinicians, and underuse of available services, although some studies have not found these associations.[55] LaVeist and colleagues[56] surveyed 781 black and 1003 white patients with serious chronic heart disease about their level of satisfaction with the care they received, their perceptions of trust in the healthcare system, and their perceptions of racial bias inherent in the healthcare system. In a multivariate analysis controlling for a range of demographic factors, they found a significant link between perceived racial bias in care, trust in the system, and satisfaction with care, with perceived bias predicting both lower trust and lower satisfaction.

The third means through which race could have an adverse effect on medical care is stereotype threat.[57] Stereotype threat occurs when individuals, often unconsciously, fear being judged negatively according to racial stereotypes. In the context of medical care, stereotype threat might cause a black patient to approach an ambulatory care visit concerned that he or she may be treated according to a stereotype such as being nonadherent with medications or less able to understand complex medical issues. The effect of stereotype threat on clinical interactions has seen limited study. In 1 report,[58] an intervention known to blunt the effects of stereotype threat was administered to black patients about to see a primary care physician for hypertension care. Compared with those receiving a control intervention, those in the intervention group had patient-provider communication that was more interested, friendly, responsive, interactive, and respectful and was less depressed and distressed in tone.

Recommendations and Conclusions: Race/Ethnicity, Racism, and CVD

- Race/ethnicity is a social construct with little biological or genetic basis.
- The concepts of implicit bias and stereotype threat are real phenomena that affect health and disease and may be root causes of disparate care.
- Effective interventions to improve patient-provider communication and patient satisfaction/trust across racial lines are clearly needed.

Social Support, Social Networks, and CVD

Social Support

The term social support has been defined in the literature in a variety of ways. Perhaps the best definition is one in longest use, which defines social support as "…information leading the subject to believe that he is cared for and loved, esteemed, and a member of a network of mutual obligations."[59] The key concepts in this definition are that social support involves positive emotional exchange and that the emotional exchange is bidirectional. The literature linking social support with better health, and conversely linking social isolation with poor health, is extensive. Links between social support and CVD have been particularly well studied. A comprehensive review is beyond the scope of this statement, but a few results illustrate the strength of associations reported.

In one of the largest reported studies, Kawachi and colleagues[60] assessed social support and self-reported Framingham risk factors in 32 624 male health professionals. After 4 years

of follow-up, those in the lowest stratum of social support had a relative risk of 1.90 for cardiovascular mortality and 2.21 for incident stroke compared with those in the highest stratum; risk was intermediate in the middle 2 strata. Risk for incident myocardial infarction was not associated with social support in this study. However, survival in subjects with coronary heart disease has been consistently linked to social support. Williams and colleagues[61] assessed social support in a cohort of patients with significant coronary artery disease demonstrated on angiography. They reported that unmarried individuals without a close confidant had an adjusted hazard ratio for survival of 3.34 compared with those reporting either or both. Another contemporary study by Berkman and colleagues[62] is of particular interest because of its prospective design and explicit focus on emotional support. Of 2806 community-living elderly individuals who had undergone baseline interviews, 194 had a subsequent myocardial infarction. Lack of emotional support at baseline was associated with an odds ratio of 2.9 for 6-month mortality after infarction.

The single most important gap in the literature on the relationship between social support and cardiovascular outcomes is that an intervention directed at improving social support has not been demonstrated to improve cardiovascular outcomes. The largest study to date is the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD).[63] In this study, 2861 patients who had had a myocardial infarction and who had depression or low perceived social support were treated with cognitive behavioral therapy. The aim of the intervention was to "strengthen network ties to be more functional, supportive, and satisfying." For those enrolled on the basis of low perceived social support, the intervention produced a statistically significant improvement in scores on a social support index. After a mean follow-up of 29 months, there was no difference in all-cause or cardiovascular mortality, nonfatal infarction, or need for revascularization. Several possible reasons for the negative results of ENRICHD deserve further attention. Although statistically significant, the improvement in social support may not have been clinically significant; more effective interventions may have an effect on cardiovascular outcomes. Such interventions might be directed at individual's underlying abilities to develop and maintain relationships or might be directed at a social milieu rather than at individuals. Finally, the possibility remains that low social support does not lie in the causal pathway. Further investigation of the mechanisms linking low support and CVD may be helpful.

The degree to which low social support interacts with other social determinants of cardiovascular health remains somewhat unclear but is probably low. Differences in social support by race, ethnicity, and SEP have not been shown consistently, whereas neighborhood of residence probably does affect social support adversely when conditions favor isolation. In addition, the effects of social support differ by sex, with marriage conferring a cardiovascular health benefit in men but not women and benefits from relationships with friends conferring a benefit in women but not men.[64]

Social Networks

The concept of social networks overlaps with the concept of social support but differs in that it focuses on a group of individuals rather than a single individual and extends to aspects of social relationships beyond the emotional. Social networks are characterized by their size (the number of connected individuals), density (the extent to which all individuals in the network are connected), and the characteristics of the connections themselves. Sophisticated techniques for analyzing and characterizing networks are available and are beyond the scope of this work. Social networks are thought to influence health in 2 ways: through social influence on behavior and through the resources embedded in social networks that are available to its members. Potential mechanisms for this influence are further explained in the Mechanisms Mediating the Relationship Between Societal Conditions and CVD section.

Two reports from the Framingham Heart Study illustrate the concept of social influence on behavior. Christakis and Fowler[65] studied participants in the Framingham Heart Study for whom BMI over time was available. The investigators were able to construct social networks for study participants from the contact information that the participants had supplied to facilitate long-term follow-up. The authors found that "a person's chances of becoming obese increased by 57%…if he or she had a friend who became obese in a given [time] interval." Geographic proximity did not explain the finding. Similarly, primary and secondary preventive use of aspirin was enhanced when members of one's social networks took aspirin.[66]

The concept that resources embedded in social networks may affect health is drawn from the Social Network Theory of Capital presented by Lin.[67] Individuals might use members of their social networks for material assistance with transportation, fulfilling obligations while hospitalized, or accessing health expertise. Empirical evidence for an effect of social capital on cardiovascular health is thin. The potential for social networks to benefit cardiovascular health, particularly in light of fundamental shifts in the size and nature of social networks brought about by the Internet, represents a significant gap in knowledge and a significant opportunity for future research.

The degree to which social networks vary with other social determinants of cardiovascular health may be significant. Weaknesses in social networks are notable among socioeconomically disadvantaged individuals. Marsden[68] used national survey data to demonstrate that whites had social networks of greater size and diversity compared with Latinos, who had larger and more diverse networks than blacks. One review summarized the data as showing that "…people in lower socioeconomic status tend to use local, strong, and family ties. Since these ties are usually homogeneous in resources, these networking tendencies reinforce poor social capital." In one of the few applications of the concept of network social capital in health care, Prentice[69] reported that individuals living in neighborhoods where people are more likely to help their neighbors were more likely to receive preventive services.

Recommendations and Conclusions: Social Support and Social Networks

- Although diminished social support contributes to CVD, effective interventions for low support have not been demonstrated.

- Mechanisms by which social networks affect health are unknown and a significant opportunity for future research.
- Engaging individuals and their support networks may be a powerful intervention tool and is worth future investigation.

Culture, Language, and CVD

Linguistic and cultural differences contribute to poorer cardiovascular health in some disadvantaged groups. These concepts are closely linked to the concept of ethnicity and are particularly relevant to the nation's fastest growing minority group, Latinos.

In 2013 the US Department of Health and Human Services published a revised version of its *National Standards for Culturally and Linguistically Appropriate Services in Health and Health Care*.[70] Of the revised standards, Standard 5 states that healthcare providers must "[o]ffer language assistance to individuals who have limited English proficiency and/or other communication needs, at no cost to them, to facilitate timely access to all healthcare and services." Standard 6 requires that providers "[i]nform all individuals of the availability of language assistance services clearly and in their preferred language, verbally and in writing."

These standards are based on evidence that language barriers are associated with reduced rates of receiving recommended care.[71] In a survey of a randomly selected sample of >4000 adults in the United States drawn from 15 different racial and ethnic groups, all but 2 of the 14 minority groups (Japanese American and Native American) reported having experienced discrimination in the medical care process based on their English-language ability.[72] Beyond the linguistic barriers faced by immigrant groups, the authors of this study described an additional key finding: "Interestingly, even though US-born African Americans are native speakers of English, they were significantly more likely than whites to report discrimination because of how they spoke the language."[72]

Beyond issues of mistrust rooted in past experiences of discrimination, misunderstandings rooted in differing cultural perceptions of disease can also play an important causal role in health disparities. Culture is particularly difficult to define, but as commonly used, it is perhaps best described as a system of beliefs and behaviors characteristic of a definable group that is transmitted without biological inheritance. Despite the difficulties, the concept is useful for understanding some differences in prevalence and treatment of illness.

Poor control of type 2 diabetes mellitus is particularly common among Mexican American farm workers. Researchers from Stanford University interviewed adult patients with diabetes mellitus at 2 farm worker clinics, 1 in California and 1 in Oregon, and found that many of the workers held strong cultural beliefs about the nature of diabetes mellitus that created a barrier to successful control (unpublished data, R. Gupta, BA, N. Gordon, BA, DA Barr, MD, PhD; Stanford University, Program in Human Biology; Stanford, CA; March 13, 2013). Respondents described their beliefs that strong emotional experiences such as *susto* or *coraje* can be the primary cause of developing diabetes mellitus and can exacerbate existing disease. Some respondents also indicated that they believed God was in control of the progression of their diabetes mellitus. Respondents also indicated that maintaining a traditional Mexican diet and eating together as a family were very important to them, regardless of consistency with recommended dietary restrictions.

Community health workers, trained laypeople who are often members of the target population with similar cultural and linguistic practices, have contributed to the prevention and control of CVD as members of the healthcare team.[73] Community health workers have contributed to significant improvements in community members' access to and continuity of care and adherence to treatment for the control of hypertension.[74] Community health workers assume multiple roles, including educating patients and communities, counseling patients, monitoring patient health status, linking people with health and social services, and enhancing provider-patient communication and adherence to care. The importance of addressing language and culture is further illustrated by a California study conducted in a Korean immigrant community in patients with type 2 diabetes mellitus. This study showed that a care program that provided culturally and linguistically accessible care through a bilingual nurse practitioner resulted in better self-management practices and better blood glucose control among Korean-speaking patients participating in this program.[75] Whether these types of culturally specific interventions will also be effective in other ethnic contexts will need further research to determine.

Recommendations and Conclusions: Culture, Ethnicity, and Language

- Language differences and cultural beliefs and practices affect health-seeking behaviors and access to care
- Culturally and linguistically tailored interventions for specific ethnic groups can be highly effective for improving cardiovascular outcomes.
- Ensuring policy support for funding of the community health workers role is critical to sustainability of culturally appropriate interventions.

Access to Medical Care

Access to care is a complex concept that incorporates 5 characteristics or dimensions: approachability, acceptability, availability and accommodation, affordability, and appropriateness. To generate access, these 5 dimensions have to interact with people's abilities, including the ability to perceive, which encompasses health literacy, beliefs, and expectations; ability to seek, which relates to the personal, social, and cultural values of patients; ability to reach, which pertains to the living environment, geographic location, and transportation; ability to pay, which is concerned primarily with income, economic, and insurance status; and ability to engage, which relates to empowerment, adherence, and caregiver support.[76,77] Therefore, to understand the factors that affect access to care in CVD and stroke, one must analyze each of these dimensions.

Approachability

Approachability captures one's ability to identify the existence of healthcare services and the potential health impact

of service use. It involves transparency, outreach, information, and screening.[76] Among US stroke survivors, blacks and Hispanics may have reduced access to stroke preventive care because they have lower median household incomes, have less access to high-quality health care, and are more frequently uninsured.[78] Indeed, in the National Health Interview Survey for the years 2000 through 2006, of 4864 stroke survivors >65 years of age, Mexican Americans and non-Hispanic blacks reported a lower proportion of specialist visits compared with non-Hispanic whites.[79]

Availability and Accommodation

Availability and accommodation involve the existence of healthcare services that are physically available and convenient (eg, geographic location, hours of opening, availability and timing of appointments).[76] A study that analyzed US Census Bureau data and the Atlas and Database of the Air Medical Services found that about half of the US population has timely access to a primary stroke center.[80] Similarly, a study analyzing the cardiology workforce found an uneven geographic distribution of cardiologists, with many rural regions having poor local access to cardiologists. The authors recommended that policy should focus not only on gross numbers of cardiologists but also on geographic distribution. In addition, they suggested that telemedicine could be necessary in areas where the population density does not support specialists.[81] Indeed, just as in cardiology, access to acute stroke care is even more restricted given the limited number of vascular neurologists and their geographic distribution. Telestroke has emerged in recent years as a way to increase access. This technology continues to develop as the solution to the problem of access.[82]

Affordability

Affordability represents the economic capacity of an individual to spend resources and time on health care (eg, direct, indirect, and opportunity costs).[76] The patchwork nature of the healthcare system in the United States has resulted in a wide variation in access to care. Individuals have difficulties accessing care because of lack of health insurance, lack of geographic proximity to care, and possibly discrimination. Lacking health insurance has a profound effect on health, including a striking association with increased mortality. In a study published in 1993, Franks et al[83] reported that lacking health insurance was independently associated with a significantly increased risk of mortality (hazard ratio, 1.25; 95% confidence interval, 1.00–1.55). Updating the analysis in an article published in 2009, Wilper and colleagues[84] found that little had changed, with an adjusted hazard ratio for mortality of 1.40 (95% confidence interval, 1.06–1.84). Specific to CVD, lack of insurance is associated with a lower likelihood of adequate treatment of risk factors[85] and with an increased risk of stroke and cardiovascular death.[86]

The issue of discrimination limiting access to care has been raised in regard to the difficulties Medicaid patients face in finding regular sources of primary care[87] and access to specialty care.[88,89] In a national study,[87] Medicaid patients were nearly twice as likely to report barriers to obtaining primary care as were patients with private insurance. In a survey of 439 federally qualified health centers, directors reported barriers to obtaining specialty care for ≈15% of those with private insurance, for ≈45% of those with Medicaid, and for ≈65% of the uninsured.[88] Although surveys of physicians cite low reimbursement as one reason for not seeing Medicaid patients,[90] perceptions that Medicaid patients are more likely to miss appointments and are less adherent with treatment are also cited, raising questions about whether prejudices play a role in decisions to accept patients insured under Medicaid.[91]

Provision of health insurance as a solitary intervention is probably not sufficient for improving health. A limited expansion of Medicaid enrollment in Oregon in 2008 resulted in random allocation of individuals from a waiting list to receive insurance. After 2 years, improvements in health in general and in cardiovascular risk in particular were not seen.[92]

Acceptability

Acceptability involves cultural and social factors shaping an individual's perception of the various aspects of services and appropriateness of care services.[76] Although socioeconomic status and insurance status have important roles in the use of services, it is important to highlight that there are other factors. In fact, in a sample of hypertensive Korean American immigrants, although insurance status and relevant medical history were direct factors in service use, life priorities, years of residence in the United States, and perceived income level had indirect effects on access.[93] Furthermore, other factors such as language barrier, perceived racial biases, and immigration status have a significant impact on patients' decision to seek medical attention.[94] For instance, undocumented Hispanic immigrants may be more reluctant to use health services.[95] Disparities in access were reported at different levels of acute stroke care (eg, less stroke symptom recognition by blacks or African Americans).[96] Delay in arrival at the emergency department decreases the odds of receiving acute stroke treatment.[97]

Recommendations and Conclusions: Access to Care

- Barriers to access are many and include issues involving patient beliefs, literacy, culture, and language.
- There is also a poor geographic distribution of cardiac services.
- Barriers to improving access to subspecialty care for patients with Medicaid are a critical issue for cardiovascular specialists.
- Although access to health insurance is necessary, it is not a sufficient intervention for improving cardiovascular health.
- Improving access is a multifaceted task that will require not only the provision of insurance coverage but also a better distribution of services.

Residential Environments

As a basic principle of epidemiology, it is well understood that disease varies across person, place, and time. The focus on area-based differences in health extends this tradition not only by describing differences in health across geographic locations but also by examining associations between features of these locations and health outcomes and the underlying mechanisms linking them. A focus on features of residential

environments represents a paradigm shift from the era of modern epidemiology in which multiple disease risk factors were investigated in large population-based cohort studies. A hallmark feature of these studies was a focus on individual-level risk factors, both behavioral and biological, without consideration of the contexts that shape these risk factors.

Often referred to as neighborhoods, features of residential environments have been linked to CVD outcomes in many observational studies. In ARIC, one of the first longitudinal studies of neighborhoods and CVD, Diez Roux and colleagues[98] examined neighborhood socioeconomic disadvantage, measured as an index of Census-derived indicators of socioeconomic characteristics of Census block groups, in relation to incident coronary heart disease. After an average of 9 years of follow-up, living in more disadvantaged neighborhoods compared with advantaged neighborhoods was associated with a 70% to 90% higher risk of coronary heart disease in whites and 30% to 40% higher risk in blacks independently of individual-level characteristics (demographics, SEP, health status, and behavioral risk factors). Other longitudinal studies have documented similar association between neighborhood socioeconomic resources and myocardial infarction, stroke, coronary heart disease, and CVD mortality, in addition to a variety of CVD-related health factors such as BMI and blood pressure.[99–103]

More recent studies have focused on moving beyond neighborhood socioeconomic disadvantage to an examination of specific features of neighborhood built/physical and social environments. Neighborhood built/physical environment indicators capture features of urban design and public spaces such as land use patterns, street connectivity, access to destinations and resources, and transportation systems. Neighborhood social environment indicators represent aspects of social norms and connectedness and psychosocial stressors such as safety, violence, and social cohesion. In one of the most comprehensive longitudinal investigations in this regard, the Multi-Ethnic Study of Atherosclerosis (MESA), features of neighborhood physical environments, including neighborhood healthy food access and resources for physical activity, were linked to many CVD risk factors. After an average of 5 years of follow-up, participants 45 to 84 years of age at baseline and of diverse racial and ethnic backgrounds had a 38% lower risk of type II diabetes mellitus and 10% lower risk of obesity independently of individual-level confounders and health behaviors.[104,105] These associations are supported by other cross-sectional studies, and the most consistent evidence is in relation to neighborhood built/physical environment and obesity/BMI. In a review of 20 studies investigating this link among studies published between 2002 and 2006, 17 studies found a positive association between neighborhood built environment and obesity.[106] Other reviews have also found consistent evidence that neighborhood built/physical environment is associated with anthropometric measures. Associations between aspects of the social environment and CVD have been less established and more inconsistent, but longitudinal studies have linked neighborhood safety to ischemic heart disease and stroke mortality.[107,108]

The nature of neighborhoods and community-based exposures makes it challenging to conduct experimental studies. However, in the only study of its kind, Ludwig and colleagues[109] analyzed data from the Moving to Opportunity Study, in which 4498 women from 5 cities in the United States were randomized to 1 of 3 conditions: a voucher to move to a low-poverty neighborhood, a voucher to move to any neighborhood, or no voucher (control group). After 10 to 15 years of follow-up, women receiving the low-poverty voucher were significantly less likely to have a BMI >35 kg/m^2, a BMI >40 kg/m^2, or a glycated hemoglobin >6.5% compared with women in the control group.

Prisons

Incarceration is a special-case residential environment that has been linked to high cardiovascular risk and cardiovascular mortality. Relatively little attention has been paid to the health effects of this social factor despite the fact that, according to US Bureau of Justice Statistics, the United States has the highest incarceration rate in the world and ≈10% of black men in their 30s are in prison. Binswanger and colleagues[110] studied a cohort of individuals released from prison in Washington State. Over 1.9 years of follow-up, those released from prison had a relative risk of death 3.5 times greater than that for age-, sex-, and race-matched control subjects, with CVD the second-leading cause of death behind drug overdose. Wang and colleagues[111] used data from a prospective study of Cardiovascular Risk in Young Adults (CARDIA) to assess the impact of imprisonment on subsequent cardiovascular risk. Incarceration was an independent risk factor for developing hypertension and left ventricular hypertrophy but not for hypercholesterolemia or diabetes mellitus. The effects were greater in black men and in those with less education.

Recommendations and Conclusions: Residential Environments

- Residential environments characterized by diminished socioeconomic resources, access to healthy foods and resources for physical activity have a measurable effect on CVD and the density of CVD risk factors.
- Proactive efforts to change the built environment may reduce the burden of CVD risk.

Mechanisms Mediating the Relationship Between Societal Conditions and CVD

A great deal of progress has been made in the past 10 to 15 years toward understanding the mechanisms by which social conditions result in CVD. Among these, psychological, behavioral, and biological mechanisms have been highlighted. A brief overview of these mechanisms is offered below.

Psychological Mechanisms

Socioeconomic disadvantage may adversely affect cardiovascular risk through its impact on mental health. A vast body of literature has documented associations between emotional states and CVD risk. Depression and elevated depressive symptoms, in particular, are associated with an increased risk of morbidity, adverse cardiovascular outcomes, and mortality among patients with CVD, even after controlling for other risk factors,[112–118] and several well-controlled studies show a dose-response relationship such that the greater the

severity of depression is, the earlier and more severe cardiac events are.[115,119–121] Patients with CVD who are depressed exhibit a worse cardiometabolic profile[122] with higher levels of atherosclerosis-related biomarkers and other predictors of cardiovascular events (ie, increased inflammatory response biomarkers,[123,124] greater platelet activation,[125,126] reduced heart rate variability,[127] hypothalamic-pituitary-adrenal axis dysfunction,[128] impaired vascular function[129]) compared with nondepressed individuals. Anxiety, alone or comorbid with depression, has been associated with cardiovascular risk and outcomes,[130–133] although some studies have shown inconsistent associations between anxiety and mortality risk.[134–137] Similarly, well-established associations have been documented between elevated levels of hostility/anger and subclinical atherosclerosis,[138] incident myocardial infarction,[139–142] CVD progression,[143] and CVD and all-cause mortality[140,145] and with selected CVD risk factors, including hypertension,[146] low-density lipoprotein, inflammation (C-reactive protein),[147–149] and behavioral risk factors.[150] A meta-analytic review found that perceived stress was associated with a 27% increased risk of CVD.[151] Furthermore, there is evidence that reductions in these negative emotions can improve CVD risk factor profile and disease end points.[152] More general measures of stress and resources have shown less consistent evidence with cardiovascular risk. A cross-sectional analysis with data from the Jackson Heart Study (JHS) found that higher stress levels were weakly associated with hypertension, diabetes mellitus, and obesity and that stressors appeared to contribute to a small proportion of the income patterning of diabetes mellitus and, to a lesser extent, hypertension and obesity in black women.[153] Additional research that examines the contribution of stress and limited resources to CVD risk is needed.[154]

There is a scarcity of research examining the potential mediating role of psychological factors (ie, depression, anxiety, anger/hostility) in the relationship between socioeconomic disadvantage and cardiovascular health. However, considerable literature has found that individuals of low-SEP, nonwhite groups, people with low social capital, and those living in adverse environments have a higher prevalence of depression and overall emotional distress.[155,156] It has been hypothesized that greater exposure to selected types of stress with less availability of resources contributes to poor mental health and cardiovascular risk among socioeconomically disadvantaged groups.[157] Economic stressors such as financial hardship and precarious employment (combination of instability, low wages, lack of regulatory protection, and limited worker job control) and financial dissatisfaction are strongly associated with depression and depressive symptoms.[158–161] Job stress also has shown associations with cardiovascular health, and its influence seems to depend on the demands of work, the individual's perceived control over these demands, and the wider social support networks that can counter the negative effects of these demands.[161] A meta-analysis of 13 cohort studies from Europe[162] assessed the impact of jobs with high or low cognitive demand and high or low job control on the incidence of later CVD. After other measures of socioeconomic status and for conventional risk factors were controlled for, there was a significantly elevated risk associated with the high-demand/low-control condition. Siegrist and colleagues[163] have alternatively proposed that an imbalance between the effort expended and the reward received best characterizes the work conditions predisposing individuals to increased risk of CVD. The degree to which these findings apply to the changing, nonindustrial workplace prevalent in the United States today is not clear.

Psychological factors also may mediate associations between physical and social environments and CVD risk. Social environment characteristics such as trust and social cohesion also have been shown to be associated with depressive symptoms and overall mental health,[164,165] even after adjustment for demographic and socioeconomic variables,[164,166] and limited social support or social network and discrimination, often experienced among disadvantaged groups,[167] are associated with emotional health. Racial discrimination and other forms of discrimination that are perceived as stressful are associated with depression and anxiety.[156,168,169] Racism, discrimination, and oppression[170] in turn are associated with elevated blood pressure and markers of inflammation. In addition, a significant interaction between life-course SEP and racial discrimination in depressive symptoms has been reported.[156] Conversely, social environment factors such as social support have been shown to reduce depression symptoms and to improve health behaviors among racial/ethnic minority groups.[171–173]

Poor built environments can increase the likelihood of mental health disorders by exposing individuals residing in disadvantaged neighborhoods to daily stressors and inconveniences, social strain, exposure to crime and trauma, and limited access to green space.[174,175] For example, residential environment factors such as crime and low personal safety can lead to the experience of anxiety, which in turn can also affect physical activity. Factors such as deprived and densely populated neighborhoods, social disorganization, and poor-quality built environments have shown associations with depression,[175–179] even when neighborhood median income is accounted for. Conversely, higher levels of neighborhood green space in low-income communities have been linked to lower levels of perceived stress and a steeper diurnal decline in cortisol secretion,[180,181] increased social contact,[182] improved mental health,[183,184] and lower all-cause mortality.[185] Although a social drift explanation (ie, people with mental health problems being more likely to move into poor neighborhoods) could at least partially explain some associations between the built environment and negative emotional states or poor mental health, it also has been shown that moving to less disadvantaged neighborhoods appears to decrease psychological distress.[186]

Thus, economic, social, and physical environment factors appear to contribute to chronic negative psychological states, which may result in dysregulation of the autonomic nervous system[187] and associated increased blood pressure, greater adiposity, and insulin resistance (Biological Mechanisms section); increased likelihood of unhealthy behaviors (Behavioral Mechanisms section); additional chronic life stress[188–196]; and the development and progression of CVD. Research in this area is continuing,[197–200] and inclusion of racial/ethnic and socioeconomically diverse populations in this research will be critical.

Although at present there is no evidence that depression treatment offsets the risk of cardiovascular events,[63,201,202] a 2008 American Heart Association science advisory on depression and coronary heart disease, endorsed by the American Psychiatric Association,[203] recommended a specific stepped screening approach to identify patients who may require further assessment and treatment for depression. In an effort to best understand the impact of depression, the symptoms of which are complex, current studies are examining specific depression subtypes that most closely relate to distinct biological underpinnings.[197] For example, somatic-vegetative symptoms of depression, but not cognitive-affective symptoms, are positively associated with intima-media thickness change.[198] Future longitudinal research is needed to disentangle psychological mediators of the impact of social and economic circumstances on health and the impact of potential interventions to target depression in the context of its socioeconomic correlates. Understanding of these factors among racial/ethnic minorities is important because conditions such as depression and anxiety are more likely to be underdiagnosed and undertreated among minority patients and are more likely to be chronic,[204–209] potentially contributing to an earlier and heavier burden of CVD disparities in these groups.[210–212]

Behavioral Mechanisms

There is compelling evidence that smoking, inactivity, obesity, unhealthy diets, and medication nonadherence increase the risk for CVD risk and contribute to cardiovascular risk factors such as hypertension, lipid abnormalities, insulin resistance, and diabetes mellitus. Conversely, evidence shows that changes in health-related behaviors can reduce CVD risks. Smoking cessation reduces the risk of cardiovascular morbidity and mortality. Similarly, changes in dietary behavior (ie, reduction in calories, saturated fat, sodium), an increase in physical activity, and a 5% weight loss among high-risk individuals have been prospectively associated with a 58% reduction in the incidence of diabetes mellitus,[213] a 42% reduction in the incidence of hypertension,[214] and a 12% to 14% reduction in 10-year risk of coronary heart disease.[215] A large body of evidence indicates that differences in health behaviors account for some of the socioeconomic gradient in health.[216–218] However, prevention of CVD cannot be reduced to simply targeting unhealthy behaviors because behaviors are in turn affected by the socioeconomic circumstances in which individuals live (ie, social patterning of behaviors).[219]

Socioeconomic gradients exist for multiple health behaviors over the life course, and the combination of several unhealthy behaviors adds up to explain a large part of the socioeconomic health gap. Smoking, poor diet, inactivity, obesity, and medication nonadherence tend to be more prevalent among individuals of low SEP.[220–225] Furthermore, SEP in childhood helps account for unhealthy behaviors and health risk in the adult years. For example, a British cohort study that followed up subjects from birth to 66 years of age found that both childhood and adult SEP (ie, father's occupational class and mother's education) accounted for a significant portion of health inequalities in mortality risk by shaping exposure to smoking and other risk behaviors.[226] Medication nonadherent behavior also has been recognized as a socioeconomically determined problem, with studies showing significant associations between SEP and adherence to preventive statin therapy.[227] A study of inequalities in BMI and smoking behaviors in 70 countries concluded that a global trend exists toward an increasing burden of chronic disease risk among people of lower SEP as countries become more urban,[228] and another study showed that standard weight loss interventions may be less effective in blacks.[229,230] An English study that examined social inequalities of CVD risk factors in men found a significant increase in social inequality for smoking status and limited physical activity between 1998 and 2006, with increases in inequality over time resulting from improvements for those in higher socioeconomic classes.[231] However, other studies have suggested that differences in health behaviors may, at least partially, stem from differences in stressors and psychosocial resources associated not only with economic factors but also with race and social and residential environments.

The relationship between unhealthy diet and low SEP may represent more than a social patterning of behavior. The direct economic effect of food costs is a significant contributing factor, with the cost per calorie increasingly lower for foods high in refined sugar and saturated fat.[232] The relationship between low availability of healthier foods such as fruits and vegetables and low consumption of those foods in poor and minority neighborhoods has also been documented.[233]

Although no direct biological differences have been found to explain racial differences in CVD risk, behavioral risk profiles vary by race and ethnicity. Race-related factors have been found to influence health behaviors. Health behavior profiles are less favorable among nonwhite groups compared with non-Hispanic whites,[229,230,234] with a greater prevalence of unhealthy diets, inactivity, obesity, and medication nonadherence and lower healthcare use. Nonwhite patients are significantly more likely to be nonadherent to statins and antihypertensive medications,[235–237] and medication nonadherence has been shown to mediate the relationship between ethnicity and CVD outcomes.[238] Of interest, a comparison between black and white individuals with hypertension in terms of preferences for behavior change revealed that both exercise and fruit and vegetable consumption were the preferred changes and did not differ by race. However, implementation of these behaviors differed by race, with a majority of whites, but not blacks, engaged in exercise.[239] Although further research is needed to understand the way in which race/ethnicity may contribute to a greater prevalence of risk behaviors among black and Hispanic groups, investigators have postulated that it is not race alone but an interaction of race and other factors that contributes to elevated risk behavior profiles. In the healthcare arena, for example, features of the patient-provider dyad may reduce patient risk behaviors. For instance, racial composition of the patient-provider dyad may affect adherence. A study that examined the impact of patient-provider communication among blacks found that collaborative patient-provider communication was associated with better adherence in racially concordant patient-provider dyads.[240] In another study, self-reported racial discrimination was associated with lower medication nonadherence among blacks with hypertension, with this association being mediated partially by trust in physicians.[241] Other factors explaining medication nonadherence or other

poor self-care behaviors specifically among blacks include low health literacy,[242] cultural beliefs related to the inevitability of cardiovascular risks or to taking medication, cultural preferences for food, and social norms concerning in whom to confide.[243]

Health behaviors also may mediate the link between adverse social environment and cardiovascular risk. Social capital factors such as social support and cohesion are associated with health behaviors and CVD risk and vary across the socioeconomic spectrum.[244] For example, social support from family and friends has been cross-sectionally associated with level of physical activity,[245] and family emotional involvement and family cohesion appear to improve the impact of a weight loss intervention in blacks, with this impact not observed in whites.[246] A study of Mexican Americans found that encouragement from an older-generation member of the participant's social network was associated with higher levels of intention to screen for blood cholesterol, blood pressure, and blood glucose.[247] Cultural proxies such as language use and educational attainment are both important determinants of health among Hispanics, and there is evidence that English-language use and educational attainment are independently associated with behavior risk profiles of Hispanics.[248]

As described above, characteristics of the residential or built environment have been correlated with cardiovascular risk and risk factors such as obesity[249] and hypertension,[249] and these associations are presumed to be mediated largely by an influence of the environment on individuals' health behaviors. There is increasing evidence of associations between the built environment and physical activity, eating behaviors, and overweight/obesity among both adults and children.[250–256] Crime-related safety perceptions tend to influence physical activity.[257] Poor communities have fewer neighborhood resources such as healthy food outlets and parks and recreational facilities, less availability of healthy food options, and fewer monetary and transportation resources to access resources outside the neighborhood, putting these communities at high risk.[258–260] For example, a cross-sectional analysis of neighborhood factors and obesity in MESA showed that residents of neighborhoods with better walking environments and healthy food availability were more likely to have a lower BMI independently of age, race and ethnicity, education, and income.[261] Other studies also have shown associations of neighborhood walkability and density of fast-food restaurants with obesity prevalence and blood pressure.[262,263] An Australian study found that exposure to energy-dense snack foods and soft drinks in supermarkets was greater in socioeconomically disadvantaged neighborhoods. This may affect purchasing, consumption, and cultural norms related to eating behaviors. A limitation of the evidence is that most studies have been cross-sectional in nature, and a review of the literature on neighborhood walkability, physical activity, and obesity reported inconsistent findings.[264] However, several quasi-experimental studies have assessed the change in CVD-related risk factors (diet and physical activity) before and after improvements in neighborhood built/physical environments (creation of urban trails, opening of supermarket). For example, Fitzhugh and colleagues[265] assessed the change in the amount of directly observed physical activity in a neighborhood (and 2 control neighborhoods) before and after the development of an urban trail that provided a connection between residential and nonresidential areas of the neighborhood. They found significant increases in physical activity (as measured by 2-hour counts of directly observed total physical activity, walking, and cycling) in the intervention neighborhood. Modification of supermarket stocking practices may similarly represent an effective means of obesity prevention.[266]

Future studies are needed to further investigate longitudinal associations among neighborhood characteristics, health behaviors, and CVD risk. Those studies will need to take into consideration cumulative exposure to neighborhood characteristics over extended periods of time because cross-sectional studies provide a very limited understanding of risk increase (eg, increased body fat) over time. In addition, it is important to determine the magnitude of behavior change through various types of changes to the built environment. In sum, health behaviors such as a healthy diet and physical activity and associated weight and weight loss, smoking cessation, and medication adherence are major determinants of health and disease and are associated with risk of coronary heart disease, hypertension, type 2 diabetes mellitus, weight gain, and premature mortality.[267] Social and economic disadvantage, however, affects health behaviors and contributes to cardiovascular risk among the poor and racial/ethnic minorities. Interventions to promote behavior change thus have substantial potential for eliminating health disparities in CVD.

Biological Mechanisms

Emerging literature seeks to link social factors with biological processes that affect cardiovascular health. At this time, it might be most appropriate to refer to biological correlates rather than determinants because most of the existing literature is based on observational data, making it difficult to distinguish causal relationships from risk markers, consequences, or pure confounders.

Current thinking focuses on several areas that link socioeconomic conditions with the biology of cardiovascular health. First, socioeconomically disadvantaged populations suffer from a great burden of Framingham risk factors.[34,268–274] This is an important consideration because it may be possible to use clinical[109] and public health interventions[92] to reduce this burden, thereby narrowing health disparities. Second, social and economic stresses lead to a biological wear and tear, or allostatic stress response,[275–280] involving a number of pathways, including stimulation of stress hormones,[280–282] inflammation,[283] endothelial dysfunction, thrombosis, vascular hyperreactivity,[270,274,284–287] and metabolic disturbances.[270,285,288,289] Third, the effects of socioeconomic disadvantage in utero and in early childhood have long-term anatomical and physiological effects that lead to CVD in adulthood.

Socioeconomic Disadvantage and Risk Factors for CVD

The modifiable risk factors for CVD—smoking, diabetes mellitus, hypertension, left ventricular hypertrophy, and hypercholesterolemia—cluster with the social determinants of SEP, race, culture, and access. The available evidence suggests that differences in risk factor prevalence account for one third of the difference in relative risk in CVD mortality by SEP.[290]

This estimate, however, underestimates the potential effect of risk factor control on reducing disparities. Using data from the Whitehall study, Kivimäki and colleagues[162] estimated that the difference in cardiovascular mortality between the highest and lowest socioeconomic groups would be reduced from 3.8 per 100 to 0.5 per 100 if conventional risk factors were eliminated (systolic blood pressure <120 mm Hg, total cholesterol <193 mg/dL, never smoked, normal postprandial blood glucose) from all. Similar data for the United States are not readily available.

Cardiovascular risk is widely documented to be greater for blacks than for whites after controlling for SEP. In a contemporary study of >24 000 subjects without CVD at baseline,[272] black men and women had higher systolic blood pressures than their white counterparts and were more likely to smoke and to have diabetes mellitus; total cholesterol was not substantially different. Among Latinos, the picture is more complex. For Mexican Americans, unadjusted cardiovascular risk is intermediate between those of whites and blacks but is not significantly different from that of whites after controlling for income, education, and health insurance status.[291] However, the prevalence of risk factors rises with duration of residence in the United States and varies among cultural and ethnic subgroups within the larger group of US Latinos.[270]

By income and by educational attainment,[292] disadvantage is associated with higher risk. In a comprehensive study based on NHANES, Kanjilal and colleagues[292] showed that, although the prevalence of high blood pressure and elevated cholesterol declined over a 30-year period for all, gradients by education and income were virtually unchanged. For smoking, there were significantly greater declines for higher-income and higher-education groups. For diabetes mellitus, the difference in diabetes mellitus prevalence between the highest- and lowest-income groups increased by a factor of 3. Similar trends were observed by educational attainment.

Taken as a whole, these findings suggest that over the coming years CVD is likely to be increasingly a disease of the disadvantaged.[268] Conversely, effective public health efforts that target blacks, Latinos (particularly US-born Latinos), and the poor might substantially reduce the overall incidence of CVD.

Allostatic Load: Chronic Stress Response and Systemic Inflammation

The human body has developed an effective biological mechanism to sense and respond to stress. The roles of the sympathetic nervous system and the hypothalamic-pituitary axis in transducing social stress have been highlighted. When we perceive potential threats, the hypothalamus and pituitary gland react by sending messages to our adrenal gland to secrete stress response hormones. This hormonal response involves a rapid phase, with the secretion of epinephrine and norepinephrine and a slower yet longer-lasting phase involving the secretion of cortisol. Together, these hormones constitute our allostatic response mechanism.

A normal allostatic response helps us to react to a perceived stressor, after which our allostatic hormones return to their baseline levels. If, however, the stressor remains, the level of allostatic hormones (referred to as our allostatic load) remains elevated. Chronic elevation of one's allostatic load over a period of years can have harmful effects on many organ systems, especially the cardiovascular system.[278,293–295]

A principal site of the harm caused by chronically elevated allostatic load is the arterial circulation. Long-term elevation of cortisol and other stress response hormones triggers an inflammatory response in the endothelial cells lining the arteries and arterioles, leading to the release of inflammatory cytokines and other markers of inflammation. Over time, this inflammatory response will cause injury to vascular endothelial cells, resulting in scarring, with the deposition of fibrin and calcium. This scarring can result in thickening and stiffening of the vascular wall, with consequent narrowing of the vascular lumen.[296] The consequences of these changes include increased blood pressure (especially diastolic pressure) and reduced blood flow, as well as an increased risk of thrombosis.

Recent research has identified a number of biomarkers that provide a quantitative measure of the physiological response to elevated allostatic load. C-reactive protein, measured in the serum, reflects the level of cellular inflammation that has been triggered by the increased load. C-reactive protein has been shown to be a strong predictor of long-term risk of CVD.[297,298] The level of circulating fibrinogen, the protein that leads to the deposition of fibrin in scar tissue and blood clots, also has been linked to increased risk of CVD.[299] Finally, the amount of calcium deposited in the coronary arteries, a factor associated inversely with socioeconomic status,[300] has a clear association with the risk of subsequent coronary artery disease.[301–305] Although other biomarkers reflect increased allostatic load, these 3 biomarkers are some of the most common measures.

As might be expected, individuals who experience chronically elevated allostatic load will show increased levels of these biomarkers and increased rates of morbidity and mortality resulting from CVD. There are 3 forms of socioeconomic disadvantage that have been shown to be associated with increased allostatic load and its consequences. Children born into extremely disadvantaged socioeconomic circumstances are at risk of developing an exaggerated response to stressors as a consequence of alterations in the cellular and molecular functioning of the hypothalamic-pituitary-adrenal system.[306] Stressful environments have been shown to imprint these changes into the sensors and receptors that are part of the hypothalamic-pituitary-adrenal axis.[307] As described by Shonkoff and colleagues,[308] "toxic stress" in early childhood "disrupts brain architecture…and leads to stress-management systems that establish relatively lower thresholds for responsiveness that persist throughout life…." The social disadvantage of lower education and associated lower occupational status during adult years creates another form of chronic stress that has been shown to be associated with increased allostatic load and its health consequences.[283] Even among fully employed civil servants in the United Kingdom, lower occupational status is associated with increased allostatic load,[280] levels of cortisol,[282] and fibrinogen[309] and associated increased risk of CVD and death.

In the context of persistent black/white racial differences in CVD in the United States, blacks are at increased risk for many of the above factors.[310] The toxic stress of poverty into which many children are born, the lower average educational and occupational attainment of black adults, and the persistent

effects of race bias throughout the life course can combine to increase substantially the risk of cardiovascular injury and death. Disadvantage that is associated with increased allostatic load and its adverse health consequences persists after controlling for poverty and other forms of disadvantage.[276,278,311] Recent studies have demonstrated increased carotid stiffness and associated intima-media thickness in black men compared with white men,[312] even in men as young as 18 years of age.[313]

Effect of Prenatal/Early Childhood Deprivation on CVD Incidence in Adulthood

From gestation through adulthood, the cardiovascular system appears to be particularly vulnerable to injury. Indeed, humans may be affected early in antenatal life by adverse environmental events such as maternal malnutrition, maternal chronic diseases, smoking, pollutants, and stress that result in lifelong cardiovascular risk.[314,315] In particular, low birth weight (small for gestational age) in term infants is associated with increased risk of atherosclerosis, type 2 diabetes mellitus, systemic hypertension, and metabolic syndrome. The association of intrauterine growth retardation and low birth weight with adult CVD has been called the fetal origins hypothesis, formulated by Barker and colleagues.[314] This hypothesis asserts that the fetus adapts to an abnormal environment by altering cell programming at a critical period in development. In response to an adverse antenatal insult such as malnutrition, the fetus remodels by altering the structure and function of various organs to promote survival. The modification of these organs may become permanent and thus not adaptable to a different postnatal environment. This phenomenon is known as programming. Programming can positively or negatively affect long-term survival. If a fetus is malnourished because of lack of food availability for the mother and this famine persists postnatally, then the alterations in organ regulation that occur in utero may have an advantage for the neonate who has adapted to the environment. In contrast, if food becomes plentiful after birth, the adaptive in utero mechanisms that prevent fetal demise in the setting of malnourishment may become maladaptive in childhood, resulting in obesity, diabetes mellitus, or systemic hypertension. These findings have been observed in populations that have experienced significant famines but occur in many situations in which malnutrition in the form of lack of food or access to poorly nutritious but highly caloric food exists.[316]

Numerous studies support the fetal origins hypothesis by demonstrating an association among low birth weight, placental insufficiency, and cardiovascular and other chronic diseases. Adult-onset systemic hypertension has been associated with birth weight.[317] More specifically, an inverse relationship between birth weight and blood pressure has been reported in several population-based adult studies[318,319]; this association is generally not present during the neonatal period or very early childhood.[320] There appears to be a pattern of low birth weight followed by rapid weight gain in early childhood that results in higher cardiovascular risk. This pattern has been called adiposity rebound.[321] A longitudinal study evaluating blood pressure measurements in 22-year-old men and women whose anthropometric measures had been recorded from birth through childhood found that those who were small at birth but gained weight rapidly by 5 years of age had the highest adult blood pressures.[322] Several theories exist about why systemic hypertension is linked to intrauterine growth retardation. The fetal renin-angiotensin system may become activated in this fetal milieu. Moreover, poor fetal growth may result in fewer nephrons, predisposing adults to subsequent kidney disease and systemic hypertension.[323] Insulin resistance and type 2 diabetes mellitus also appear to be more prevalent in adults with intrauterine growth retardation, and the same pattern exists.[324,325] In a population-based study from India, those individuals who developed glucose intolerance or type 2 diabetes mellitus had significantly lower birth weight and were underweight until 2 years of age, followed by an accelerated increase in BMI until young adulthood, compared with those with normal glucose tolerance.[323] A study of a US cohort reports that children exposed to intrauterine growth retardation have increased abdominal fat and increased insulin resistance biomarkers despite no differences in BMI growth patterns beyond 1 year of age.[326] Thus, the rate of gain rather than the severity of BMI appears to have the most impact. Similar to the theory behind the development of systemic hypertension, organ dysfunction likely exists in those who develop glucose intolerance. Intrauterine growth retardation is associated with a reduced number and function of pancreatic β cells, resulting in decreased insulin production.[316] This alteration leads to abnormal muscle, liver, and adipocyte insulin signaling and eventually to the development of insulin resistance.

Endothelial function, which can be measured noninvasively, reflects the presence of CVD. Adults known to have had intrauterine growth retardation have been shown to exhibit abnormalities of peripheral endothelial function.[327] Remarkably, adults with low birth weight who otherwise had a low-risk cardiovascular profile (nonsmoker, normal blood pressure, and normal weight) have been shown to have vascular dysfunction similar to the risk of individuals who smoked 4.5 cigarette pack-years.[328]

Genetic-environment interactions are ubiquitous in human development. Whether genetic, environmental, or fetal influences are the primary culprits in the epidemic of the CVD we see today remains unknown. The fetal origins hypothesis may in part explain the growing trend of CVD in areas where maternal nutrition is poor. Fetal undernutrition is multifactorial and may be a reflection of poverty, poor diet, medical causes of placental insufficiency, and abnormal uterine blood flow. It is important to recognize the influence of maternal nutrition and well-being on the fetus. The effects of an abnormal in utero environment have socioeconomic and global health implications for generations to come. It is thus critical that we strive to promote healthy pregnancies to maximize each individual's potential for normal growth and development. Better understanding of the origins of these disease states will bring with it enhanced preventive and targeted therapies.

Recommendations and Conclusions: Psychological, Behavioral, and Biological Mechanisms

- Psychological factors such as depression and a comprehensive set of psychosocial stressors may mediate associations between social determinants and cardiovascular outcomes and should be investigated more in future studies.

- Although cardiovascular health behaviors vary across social groups, they do not fully account for social group differences in cardiovascular outcomes.
- Physiological and anatomical effects of early disadvantage affect risk for CVD in adulthood.
- Effective interventions to reduce the impact of early disadvantage will require organizational partnerships that currently are uncommon.

Future Directions

Although there is substantial evidence linking social factors and CVD risk and outcomes, many unanswered questions remain. Below, we provide recommendations for future research. This is not an exhaustive list of recommendation but instead illustrative of research that may help to unpack the inherent complexities represented by the interrelationships of social determinants and their impact on cardiovascular health.

- Create standardized measures of social group categories that disaggregate the social determinants of health into modifiable risk factors and promote continued monitoring and investigations of the differences between and within these categories.
- Conduct observational studies that examine the complex interactions between social factors in relation to cardiovascular health.
- Incorporate nontraditional measures of social determinants that are difficult to operationalize and measure such as wealth/privilege and institutionalized racism.
- Prioritize research that investigates the intergenerational transmission of social disadvantage and the subsequent cardiovascular health consequences.
- Continue to investigate the psychosocial, behavioral, biological, and epigenetic pathways linking social and economic factors to cardiovascular outcomes and explore the promise of epigenetics.
- Create linguistically and culturally appropriate care for diabetes mellitus and other CVD risk conditions for Hispanics and other racial/ethnic minority groups shown to be at increased risk for CVD and assess their effectiveness critically.

Our primary recommendation for future research is the design and evaluation of interventions, programs, and policies that address the social determinants of health. Interventions should include a combination of both population- and individual-level approaches to shift the entire distribution of cardiovascular risk to lower levels and to target high-risk individuals.[329] A focus on population-level approaches is consistent with the "American Heart Association Guide for Improving Cardiovascular Health at the Community Level, 2013 Update."[330] With the use of the health impact pyramid, interventions that change the context to make individuals' default decisions healthy were endorsed as having the potential for the greatest impact on population-wide health promotion and risk reduction.[330,331] Such strategies have the potential to shift the entire distribution of cardiovascular risk to lower levels and to allow a larger segment of the population to be healthier for a longer period of time for less cost. Future research is needed to identify what modifiable environmental attributes and policies have the strongest or most widespread effects in promoting healthy lifestyles and preventing CVD in socioeconomically disadvantaged populations.

Considerable creativity and willingness to accept new ways of thinking will be required. For example, 1 publication[332] documented improved Framingham risk scores in individuals ≈30 years after having been randomized to an intensive preschool daycare program for disadvantaged children. The impact of social programs on cardiovascular health has generally not been considered by cardiovascular professionals.

Despite calls from the Institute of Medicine for multilevel interventions,[333] relatively few interventions to reduce CVD thus far have targeted social and environmental contextual factors at the city, neighborhood, or community level. Theoretically based interventions using social and environmental variables as a focal point for tailored interventions to affect population cardiovascular health are needed. Essential to these efforts is the use of a community-based participatory research framework in which academic institutions and community organizations and members work in partnership to address a significant health concern of the community, marrying research and action to improve community health and to eliminate health disparities.[334] This approach will ensure that interventions are socially and culturally appropriate. An example is the Racial and Ethnic Approaches to Community Health (REACH 2010), a federal initiative of the Centers for Disease Control and Prevention.[335] The overall goal of REACH is to use community-based participatory research methods to identify, develop, and disseminate effective strategies to address health disparities within 6 priority areas that include CVD and management of diabetes mellitus. Community and academic partners work together to develop culturally tailored interventions and services for black, American Indian, Hispanic/Latino, Asian American, Alaska Native, and Pacific Islander communities. The Charlotte, NC, REACH 2010 program developed individual- and community-level interventions for 20 000 blacks residing in its recruitment communities.[336] Individual interventions targeted CVD health behaviors such as physical activity, diet, and smoking cessation. Community-level interventions included the following: launching of culturally specific mass media campaigns to raise awareness on healthy behavior, healthful food labeling in schools and restaurants, and introduction of local farmer's markets to increase healthy food access. Over a 7-year period, statistically significant improvements in physical activity, smoking, and diet were found among individuals residing in communities.[337] Support and resources for similar efforts should continue and be prioritized.

In a similar vein, the Centers for Disease Control and Prevention Guide to Community Preventive Services (http://www.thecommunityguide.org/) provides recommendations for effective evidence-based intervention for implementation in communities in the United States. The Community Preventive Services Task Force endorsed team-based care as an example of a system-level intervention that incorporates

a multidisciplinary team to improve the quality of hypertension care for patients. This recommendation was based on a review of 52 intervention studies from 2003 through 2012 in which the total body of work suggests a 12-percentage-point increase in the proportion of patients with controlled blood pressure and other CVD risk factors (hemoglobin A_{1c}, blood glucose, and cholesterol levels) in those who received integrated care compared with those who did not.[292] However, the extent to which these interventions are effective in diverse communities remains unknown because studies were conducted only in white and black communities. There is also uncertainty about whether effectiveness will vary across other social group indicators such as education and income. Interventions, even those with a strong evidence base for effectiveness, require further investigations in diverse settings and more integration of the social determinants of health.

Although population-based approaches are useful, we also endorse a continuation of individual-level approaches that target high-risk individuals, especially those from socially and economically disadvantaged backgrounds. Individual-level interventions that aim to motivate a person to change in an environment that poses many barriers are unlikely to produce long-lasting change. Similarly, although emerging evidence suggests that environmental change is associated with an improvement in health behaviors,[338,339] providing a supportive environment in the absence of behavioral interventions to engage and promote the use of environmental improvements may also have limited impact. Targeting high-risk individuals is also consistent with the American Heart Association community guide in focusing on interventions that address socioeconomic factors.[330] Existing intervention studies designed to focus on improving cardiovascular health behaviors should include a diverse sample of participants across social groups and should examine how results vary among subgroups. Moreover, because health behaviors vary among subgroups in the population based on SEP, race, and social and built environments, understanding the modifiable determinants of these behaviors is critical for designing appropriate programs that will effectively reduce CVD disparities. Systematically assessing and quantifying modifiable CVD risk factors by race/ethnicity and socioeconomic factors will help clinicians and public health professionals best identify intervention targets and develop culturally sensitive interventions, prevention programs, and services for bridging the health gap between people in disadvantaged economic, social, and physical environment circumstances and those in advantaged circumstances.[340,341] In addition, an evaluation of the potential unintended consequences of existing interventions to disadvantaged groups is needed.

Conclusions

Despite declines in CVD mortality over the past several decades, it remains the leading cause of death in the United States, and many disadvantaged groups are disproportionately burdened with poor cardiovascular health. In this statement, we provided an overview of the substantial body of work documenting the influence of social factors on the incidence, treatment, and outcomes of CVD and the potential behavioral, biological, and psychological pathways linking them. We argued that, although we have traditionally considered CVD the consequence of certain modifiable and nonmodifiable physiological, lifestyle, and genetic risk factors, we must now broaden the focus to incorporate a third arm of risk, the social determinants of health. Failure to demonstrate awareness of this third dynamic will result in a growing burden of CVD, especially in those with the least means to engage in the healthcare system.

Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
Edward P. Havranek	Denver Health	AHRQ†; NIH/NHLBI†	None	None	None	None	None	None
Mahasin S. Mujahid	University of California, Berkeley	None	None	None	None	None	None	None
Donald A. Barr	Stanford University	None	None	None	None	None	None	Receives royalties from Johns Hopkins University for a book on health disparities, health policy, and premedical education*
Irene V. Blair	University of Colorado, Boulder	NIH*	None	None	None	None	National Cancer Institute*	University of Colorado, Boulder†
Meryl S. Cohen	The Children's Hospital of Philadelphia	None	None	None	None	None	None	None
Salvador Cruz-Flores	Texas Tech University Health Sciences Center, El Paso	None	None	None	None	None	Novo Nordisk*; Lilly*; Biotronik*	None
George Davey-Smith	University of Bristol	None	None	None	None	None	None	None
Cheryl R. Dennison-Himmelfarb	Johns Hopkins University	NIH*	None	None	None	None	None	None
Michael S. Lauer	NHLBI	None	None	None	None	None	None	None
Debra W. Lockwood	Provident Resources Group, Inc	None	None	None	None	None	None	None
Milagros Rosal	University of Massachusetts Medicine	CDC†; NIH/NIMHD†; NIH/NIMH†; NIH/NCAM†; NIH/NIDDK†; NIH/NCI†; Pfizer†	None	None	None	None	None	None
Clyde W. Yancy	Northwestern University	None	None	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant

Reviewer Disclosures

Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
Kirsten Bibbins-Domingo	University of California, San Francisco	NIH (PI on 2 center grants examining social determinants as contributors to chronic disease burden, including cardiovascular disease)*	None	None	None	None	None	None
Yoshihiro Kokubo	National Cerebral and Cardiovascular Center	None	None	None	None	None	None	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Significant.

References

1. Woolf SH, Aron L. *U.S. Health in International Perspective: Shorter Lives, Poorer Health*. Washington, DC: National Research Council and Institute of Medicine; 2013.
2. Bayer R, Fairchild AL, Hopper K, Nathanson CA. Public health: confronting the sorry state of U.S. health. *Science*. 2013;341:962–963. doi: 10.1126/science.1241249.
3. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, Dai S, Ford ES, Fox CS, Franco S, Fullerton HJ, Gillespie C, Hailpern SM, Heit JA, Howard VJ, Huffman MD, Judd SE, Kissela BM, Kittner SJ, Lackland DT, Lichtman JH, Lisabeth LD, Mackey RH, Magid DJ, Marcus GM, Marelli A, Matchar DB, McGuire DK, Mohler ER 3rd, Moy CS, Mussolino ME, Neumar RW, Nichol G, Pandey DK, Paynter NP, Reeves MJ, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Wong ND, Woo D, Turner MB; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2014 update: a report from the American Heart Association. *Circulation*. 2014;129:e28–e292. doi: 10.1161/01.cir.0000441139.02102.80.
4. Krieger N. A glossary for social epidemiology. *J Epidemiol Community Health*. 2001;55:693–700.
5. Williams DR. Race and health: basic questions, emerging directions. *Ann Epidemiol*. 1997;7:322–333.
6. Heidenreich PA, Albert NM, Allen LA, Bluemke DA, Butler J, Fonarow GC, Ikonomidis JS, Khavjou O, Konstam MA, Maddox TM, Nichol G, Pham M, Piña IL, Trogdon JG; American Heart Association Advocacy Coordinating Committee; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular Radiology and Intervention; Council on Clinical Cardiology; Council on Epidemiology and Prevention; Stroke Council. Forecasting the impact of heart failure in the United States: a policy statement from the American Heart Association. *Circ Heart Fail*. 2013;6:606–619. doi: 10.1161/HHF.0b013e318291329a.
7. Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, Greenlund K, Daniels S, Nichol G, Tomaselli GF, Arnett DK, Fonarow GC, Ho PM, Lauer MS, Masoudi FA, Robertson RM, Roger V, Schwamm LH, Sorlie P, Yancy CW, Rosamond WD; American Heart Association Strategic Planning Task Force and Statistics Committee. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. *Circulation*. 2010;121:586–613. doi: 10.1161/CIRCULATIONAHA.109.192703.
8. Marmot M, Friel S, Bell R, Houweling TA, Taylor S; Commission on Social Determinants of Health. Closing the gap in a generation: health equity through action on the social determinants of health. *Lancet*. 2008;372:1661–1669. doi: 10.1016/S0140-6736(08)61690-6.
9. Galobardes B, Lynch J, Smith GD. Measuring socioeconomic position in health research. *Br Med Bull*. 2007;81-82:21–37. doi: 10.1093/bmb/ldm001.
10. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health*. 1997;18:341–378. doi: 10.1146/annurev.publhealth.18.1.341.
11. Stiglitz JE, Sen A, Fitoussi J-P. Mismeasuring Our Lives: Why GDP Doesn't Add Up. The Report by the Commission on the Measurement of Economic Performance and Social Progress. New York, NY: The New Press; 2010.
12. Galobardes B, Smith GD, Lynch JW. Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. *Ann Epidemiol*. 2006;16:91–104. doi: 10.1016/j.annepidem.2005.06.053.
13. Pollitt RA, Rose KM, Kaufman JS. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: a systematic review. *BMC Public Health*. 2005;5:7. doi: 10.1186/1471-2458-5-7.
14. Kaplan GA, Keil JE. Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation*. 1993;88(pt 1):1973–1998.
15. Winkleby MA, Jatulis DE, Frank E, Fortmann SP. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *Am J Public Health*. 1992;82:816–820.
16. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. *Circulation*. 2005;111:1233–1241. doi: 10.1161/01.CIR.0000158136.76824.04.
17. Mackenbach JP, Cavelaars AE, Kunst AE, Groenhof F. Socioeconomic inequalities in cardiovascular disease mortality: an international study. *Eur Heart J*. 2000;21:1141–1151. doi: 10.1053/euhj.1999.1990.
18. Meara ER, Richards S, Cutler DM. The gap gets bigger: changes in mortality and life expectancy, by education, 1981-2000. *Health Aff (Millwood)*. 2008;27:350–360. doi: 10.1377/hlthaff.27.2.350.
19. *Health, United States 2011: With Special Feature on Socioeconomic Status and Health*. Hyattsville, MD: US National Center for Health Statistics; 2012.
20. Berkman ND, Sheridan SL, Donahue KE, Halpern DJ, Crotty K. Low health literacy and health outcomes: an updated systematic review. *Ann Intern Med*. 2011;155:97–107. doi: 10.7326/0003-4819-155-2-201107190-00005.
21. Evangelista LS, Rasmusson KD, Laramee AS, Barr J, Ammon SE, Dunbar S, Ziesche S, Patterson JH, Yancy CW. Health literacy and the patient with heart failure: implications for patient care and research: a consensus statement of the Heart Failure Society of America. *J Card Fail*. 2010;16:9–16. doi: 10.1016/j.cardfail.2009.10.026.
22. Harper S, Lynch J, Smith GD. Social determinants and the decline of cardiovascular diseases: understanding the links. *Annu Rev Public Health*. 2011;32:39–69. doi: 10.1146/annurev-publhealth-031210-101234.
23. Marmot MG, Shipley MJ, Rose G. Inequalities in death: specific explanations of a general pattern? *Lancet*. 1984;1:1003–1006.
24. Marmot MG, Shipley MJ. Do socioeconomic differences in mortality persist after retirement? 25 Year follow up of civil servants from the first Whitehall study. *BMJ*. 1996;313:1177–1180.
25. Leigh JP, Du J. Hypertension and occupation among seniors. *J Occup Environ Med*. 2009;51:661–671. doi: 10.1097/JOM.0b013e31819f1d85.
26. Davila EP, Kuklina EV, Valderrama AL, Yoon PW, Rolle I, Nsubuga P. Prevalence, management, and control of hypertension among US workers: does occupation matter? *J Occup Environ Med*. 2012;54:1150–1156. doi: 10.1097/JOM.0b013e318256f675.
27. Kivimäki M, Vahtera J, Pentti J, Ferrie JE. Factors underlying the effect of organisational downsizing on health of employees: longitudinal cohort study. *BMJ*. 2000;320:971–975.
28. Montgomery SM, Cook DG, Bartley MJ, Wadsworth ME. Unemployment pre-dates symptoms of depression and anxiety resulting in medical consultation in young men. *Int J Epidemiol*. 1999;28:95–100.
29. Dupre ME, George LK, Liu G, Peterson ED. The cumulative effect of unemployment on risks for acute myocardial infarction. *Arch Intern Med*. 2012;172:1731–1737. doi: 10.1001/2013.jamainternmed.447.
30. Watterson PA. Infant mortality by father's occupation from the 1911 census of England and Wales. *Demography*. 1988;25:289–306.
31. Kuh DL, Power C, Rodgers B. Secular trends in social class and sex differences in adult height. *Int J Epidemiol*. 1991;20:1001–1009.
32. Brindle PM, McConnachie A, Upton MN, Hart CL, Davey Smith G, Watt GC. The accuracy of the Framingham risk-score in different socioeconomic groups: a prospective study. *Br J Gen Pract*. 2005;55:838–845.
33. Fiscella K, Tancredi D, Franks P. Adding socioeconomic status to Framingham scoring to reduce disparities in coronary risk assessment. *Am Heart J*. 2009;157:988–994. doi: 10.1016/j.ahj.2009.03.019.
34. Murray CJ, Kulkarni SC, Michaud C, Tomijima N, Bulzacchelli MT, Iandiorio TJ, Ezzati M. Eight Americas: investigating mortality disparities across races, counties, and race-counties in the United States [published correction appears in *PLoS Med*. 2006;3:e545]. *PLoS Med*. 2006;3:e260. doi: 10.1371/journal.pmed.0030260.
35. Brondolo E, Rieppi R, Kelly KP, Gerin W. Perceived racism and blood pressure: a review of the literature and conceptual and methodological critique. *Ann Behav Med*. 2003;25:55–65.
36. Brondolo E, Love EE, Pencille M, Schoenthaler A, Ogedegbe G. Racism and hypertension: a review of the empirical evidence and implications for clinical practice. *Am J Hypertens*. 2011;24:518–529. doi: 10.1038/ajh.2011.9.
37. Brondolo E, Libby DJ, Denton EG, Thompson S, Beatty DL, Schwartz J, Sweeney M, Tobin JN, Cassells A, Pickering TG, Gerin W. Racism and ambulatory blood pressure in a community sample. *Psychosom Med*. 2008;70:49–56. doi: 10.1097/PSY.0b013e31815ff3bd.
38. Williams DR, Mohammed SA. Discrimination and racial disparities in health: evidence and needed research. *J Behav Med*. 2009;32:20–47. doi: 10.1007/s10865-008-9185-0.
39. Finucane TE, Carrese JA. Racial bias in presentation of cases. *J Gen Intern Med*. 1990;5:120–121.
40. Schulman KA, Berlin JA, Harless W, Kerner JF, Sistrunk S, Gersh BJ, Dubé R, Taleghani CK, Burke JE, Williams S, Eisenberg JM, Escarce JJ. The effect of race and sex on physicians' recommendations for cardiac catheterization [published correction appears in *N Engl J Med*. 1999;340:1130]. *N Engl J Med*. 1999;340:618–626. doi: 10.1056/NEJM199902253400806.
41. Thomson GE. Discrimination in health care. *Ann Intern Med*. 1997;126:910–912.

42. van Ryn M, Fu SS. Paved with good intentions: do public health and human service providers contribute to racial/ethnic disparities in health? *Am J Public Health*. 2003;93:248–255.
43. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. Washington, DC: Institute of Medicine Committee on Understanding and Eliminating Racial and Ethnic Disparities in Health Care; 2003.
44. Blair IV, Havranek EP, Price DW, Hanratty R, Fairclough DL, Farley T, Hirsh HK, Steiner JF. Assessment of biases against Latinos and African Americans among primary care providers and community members. *Am J Public Health*. 2013;103:92–98. doi: 10.2105/AJPH.2012.300812.
45. Green AR, Carney DR, Pallin DJ, Ngo LH, Raymond KL, Iezzoni LI, Banaji MR. Implicit bias among physicians and its prediction of thrombolysis decisions for black and white patients. *J Gen Intern Med*. 2007;22:1231–1238. doi: 10.1007/s11606-007-0258-5.
46. Sabin J, Nosek BA, Greenwald A, Rivara FP. Physicians' implicit and explicit attitudes about race by MD race, ethnicity, and gender. *J Health Care Poor Underserved*. 2009;20:896–913. doi: 10.1353/hpu.0.0185.
47. Blair IV, Steiner JF, Havranek EP. Unconscious (implicit) bias and health disparities: where do we go from here? *Perm J*. 2011;15:71–78.
48. Dovidio JF, Penner LA, Albrecht TL, Norton WE, Gaertner SL, Shelton JN. Disparities and distrust: the implications of psychological processes for understanding racial disparities in health and health care. *Soc Sci Med*. 2008;67:478–486. doi: 10.1016/j.socscimed.2008.03.019.
49. Sabin JA, Rivara FP, Greenwald AG. Physician implicit attitudes and stereotypes about race and quality of medical care. *Med Care*. 2008;46:678–685. doi: 10.1097/MLR.0b013e3181653d58.
50. Sabin JA, Greenwald AG. The influence of implicit bias on treatment recommendations for 4 common pediatric conditions: pain, urinary tract infection, attention deficit hyperactivity disorder, and asthma. *Am J Public Health*. 2012;102:988–995. doi: 10.2105/AJPH.2011.300621.
51. Haider AH, Sexton J, Sriram N, Cooper LA, Efron DT, Swoboda S, Villegas CV, Haut ER, Bonds M, Pronovost PJ, Lipsett PA, Freischlag JA, Cornwell EE 3rd. Association of unconscious race and social class bias with vignette-based clinical assessments by medical students. *JAMA*. 2011;306:942–951. doi: 10.1001/jama.2011.1248.
52. Blair IV, Steiner JF, Fairclough DL, Hanratty R, Price DW, Hirsh HK, Wright LA, Bronsert M, Karimkhani E, Magid DJ, Havranek EP. Clinicians' implicit ethnic/racial bias and perceptions of care among Black and Latino patients. *Ann Fam Med*. 2013;11:43–52. doi: 10.1370/afm.1442.
53. Cooper LA, Roter DL, Carson KA, Beach MC, Sabin JA, Greenwald AG, Inui TS. The associations of clinicians' implicit attitudes about race with medical visit communication and patient ratings of interpersonal care. *Am J Public Health*. 2012;102:979–987. doi: 10.2105/AJPH.2011.300558.
54. Penner LA, Dovidio JF, West TV, Gaertner SL, Albrecht TL, Dailey RK, Markova T. Aversive racism and medical interactions with black patients: a field study. *J Exp Soc Psychol*. 2010;46:436–440. doi: 10.1016/j.jesp.2009.11.004.
55. Shavers VL, Fagan P, Jones D, Klein WM, Boyington J, Moten C, Rorie E. The state of research on racial/ethnic discrimination in the receipt of health care. *Am J Public Health*. 2012;102:953–966. doi: 10.2105/AJPH.2012.300773.
56. LaVeist TA, Nickerson KJ, Bowie JV. Attitudes about racism, medical mistrust, and satisfaction with care among African American and white cardiac patients. *Med Care Res Rev*. 2000;57(suppl 1):146–161.
57. Burgess DJ, Warren J, Phelan S, Dovidio J, van Ryn M. Stereotype threat and health disparities: what medical educators and future physicians need to know. *J Gen Intern Med*. 2010;25(suppl 2):S169–S177. doi: 10.1007/s11606-009-1221-4.
58. Havranek EP, Hanratty R, Tate C, Dickinson LM, Steiner JF, Cohen G, Blair IA. The effect of values affirmation on race-discordant patient-provider communication. *Arch Intern Med*. 2012;172:1662–1667. doi: 10.1001/2013.jamainternmed.258.
59. Cobb S. Presidential Address, 1976: social support as a moderator of life stress. *Psychosom Med*. 1976;38:300–314.
60. Kawachi I, Colditz GA, Ascherio A, Rimm EB, Giovannucci E, Stampfer MJ, Willett WC. A prospective study of social networks in relation to total mortality and cardiovascular disease in men in the USA. *J Epidemiol Community Health*. 1996;50:245–251.
61. Williams RB, Barefoot JC, Califf RM, Haney TL, Saunders WB, Pryor DB, Hlatky MA, Siegler IC, Mark DB. Prognostic importance of social and economic resources among medically treated patients with angiographically documented coronary artery disease [published correction appears in *JAMA*. 1992;268:2652]. *JAMA*. 1992;267:520–524.
62. Berkman LF, Leo-Summers L, Horwitz RI. Emotional support and survival after myocardial infarction: a prospective, population-based study of the elderly. *Ann Intern Med*. 1992;117:1003–1009.
63. Berkman LF, Blumenthal J, Burg M, Carney RM, Catellier D, Cowan MJ, Czajkowski SM, DeBusk R, Hosking J, Jaffe A, Kaufmann PG, Mitchell P, Norman J, Powell LH, Raczynski JM, Schneiderman N; Enhancing Recovery in Coronary Heart Disease Patients Investigators (ENRICHD). Effects of treating depression and low perceived social support on clinical events after myocardial infarction: the Enhancing Recovery in Coronary Heart Disease Patients (ENRICHD) Randomized Trial. *JAMA*. 2003;289:3106–3116. doi: 10.1001/jama.289.23.3106.
64. Stansfeld SA. Social support and social cohesion. In: Marmot M, Wilkinson R, eds. Oxford, UK: Oxford University Press; *Social Determinants of Health*. 2006:155–174.
65. Christakis NA, Fowler JH. The spread of obesity in a large social network over 32 years. *N Engl J Med*. 2007;357:370–379. doi: 10.1056/NEJMsa066082.
66. Strully KW, Fowler JH, Murabito JM, Benjamin EJ, Levy D, Christakis NA. Aspirin use and cardiovascular events in social networks. *Soc Sci Med*. 2012;74:1125–1129. doi: 10.1016/j.socscimed.2011.12.033.
67. Lin N. Inequality in social capital. *Contemp Sociol*. 2000;29:785–795.
68. Marsden PV. Homogeneity in confiding relations. *Soc Networks*. 1988;10:57–76.
69. Prentice JC. Neighborhood effects on primary care access in Los Angeles. *Soc Sci Med*. 2006;62:1291–1303. doi: 10.1016/j.socscimed.2005.07.029.
70. *National Standards for Culturally and Linguistically Appropriate Services in Health and Health Care*. Washington, DC: US Department of Health and Human Services Office of Minority Health; 2013.
71. Ponce N, Gatchell M, Brown R. *Cancer Screening Rates Among Asian Ethnic Groups (Health Policy Fact Sheet)*. 2003. UCLA Center for Health Policy Research. http://healthpolicy.ucla.edu/publications/Documents/PDF/Cancer%20Screening%20Rates%20Among%20Asian%20Ethnic%20Groups.pdf. Accessed July 1, 2015.
72. Blendon RJ, Buhr T, Cassidy EF, Perez DJ, Hunt KA, Fleischfresser C, Benson JM, Herrmann MJ. Disparities in health: perspectives of a multiethnic, multi-racial America. *Health Aff (Millwood)*. 2007;26:1437–1447. doi: 10.1377/hlthaff.26.5.1437.
73. Brownstein JN, Bone LR, Dennison CR, Hill MN, Kim MT, Levine DM. Community health workers as interventionists in the prevention and control of heart disease and stroke. *Am J Prev Med*. 2005;29(suppl 1):128–133. doi: 10.1016/j.amepre.2005.07.024.
74. Allen JK, Dennison-Himmelfarb CR, Szanton SL, Bone L, Hill MN, Levine DM, West M, Barlow A, Lewis-Boyer L, Donnelly-Strozzo M, Curtis C, Anderson K. Community Outreach and Cardiovascular Health (COACH) Trial: a randomized, controlled trial of nurse practitioner/community health worker cardiovascular disease risk reduction in urban community health centers. *Circ Cardiovasc Qual Outcomes*. 2011;4:595–602. doi: 10.1161/CIRCOUTCOMES.111.961573.
75. Choi SE, Rush EB. Effect of a short-duration, culturally tailored, community-based diabetes self-management intervention for Korean immigrants: a pilot study. *Diabetes Educ*. 2012;38:377–385. doi: 10.1177/0145721712443292.
76. Levesque JF, Harris MF, Russell G. Patient-centred access to health care: conceptualising access at the interface of health systems and populations. *Int J Equity Health*. 2013;12:18. doi: 10.1186/1475-9276-12-18.
77. Penchansky R, Thomas JW. The concept of access: definition and relationship to consumer satisfaction. *Med Care*. 1981;19:127–140.
78. *National Healthcare Disparities Report 2008*. Washington, DC: Agency for Healthcare Research and Quality (AHRQ); 2009:1–300.
79. Levine DA, Neidecker MV, Kiefe CI, Karve S, Williams LS, Allison JJ. Racial/ethnic disparities in access to physician care and medications among US stroke survivors. *Neurology*. 2011;76:53–61. doi: 10.1212/WNL.0b013e318203e952.
80. Albright KC, Branas CC, Meyer BC, Matherne-Meyer DE, Zivin JA, Lyden PD, Carr BG. ACCESS: acute cerebrovascular care in emergency stroke systems. *Arch Neurol*. 2010;67:1210–1218. doi: 10.1001/archneurol.2010.250.
81. Aneja S, Ross JS, Wang Y, Matsumoto M, Rodgers GP, Bernheim SM, Rathore SS, Krumholz HM. US cardiologist workforce from 1995 to 2007: modest growth, lasting geographic maldistribution especially in rural areas. *Health Aff (Millwood)*. 2011;30:2301–2309. doi: 10.1377/hlthaff.2011.0255.
82. Hess DC, Audebert HJ. The history and future of telestroke. *Nat Rev Neurol*. 2013;9:340–350. doi: 10.1038/nrneurol.2013.86.
83. Franks P, Clancy CM, Gold MR. Health insurance and mortality: evidence from a national cohort. *JAMA*. 1993;270:737–741.

84. Wilper AP, Woolhandler S, Lasser KE, McCormick D, Bor DH, Himmelstein DU. Health insurance and mortality in US adults. *Am J Public Health*. 2009;99:2289–2295. doi: 10.2105/AJPH.2008.157685.
85. Ayanian JZ, Zaslavsky AM, Weissman JS, Schneider EC, Ginsburg JA. Undiagnosed hypertension and hypercholesterolemia among uninsured and insured adults in the Third National Health and Nutrition Examination Survey. *Am J Public Health*. 2003;93:2051–2054.
86. Fowler-Brown A, Corbie-Smith G, Garrett J, Lurie N. Risk of cardiovascular events and death: does insurance matter? *J Gen Intern Med*. 2007;22:502–507. doi: 10.1007/s11606-007-0127-2.
87. Cheung PT, Wiler JL, Lowe RA, Ginde AA. National study of barriers to timely primary care and emergency department utilization among Medicaid beneficiaries. *Ann Emerg Med*. 2012;60:4–10.e2. doi: 10.1016/j.annemergmed.2012.01.035.
88. Cook NL, Hicks LS, O'Malley AJ, Keegan T, Guadagnoli E, Landon BE. Access to specialty care and medical services in community health centers. *Health Aff (Millwood)*. 2007;26:1459–1468. doi: 10.1377/hlthaff.26.5.1459.
89. Felland L, Lechner A, Sommers A. *Improving Access to Speciality Care for Medicaid Patients: Policy Issues and Options*. 2013:1–24. The Commonwealth Fund, June 2013. http://www.commonwealthfund.org/publications/fund-reports/2013/jun/improving-access-to-speciality-care. Accessed July 1, 2015.
90. Cunningham P, May J. Medicaid patients increasingly concentrated among physicians. *Track Rep*. 2006:1–5.
91. LaVeist TA, Rolley NC, Diala C. Prevalence and patterns of discrimination among U.S. health care consumers. *Int J Health Serv*. 2003;33:331–344.
92. Baicker K, Taubman SL, Allen HL, Bernstein M, Gruber JH, Newhouse JP, Schneider EC, Wright BJ, Zaslavsky AM, Finkelstein AN; Oregon Health Study Group, Carlson M, Edlund T, Gallia C, Smith J. The Oregon experiment: effects of Medicaid on clinical outcomes. *N Engl J Med*. 2013;368:1713–1722. doi: 10.1056/NEJMsa1212321.
93. Song HJ, Han HR, Lee JE, Kim JY, Kim KB, Ryu JP, Kim M. Does access to care still affect health care utilization by immigrants? Testing of an empirical explanatory model of health care utilization by Korean American immigrants with high blood pressure. *J Immigr Minor Health*. 2010;12:513–519. doi: 10.1007/s10903-009-9276-1.
94. Blanchard JC, Haywood YC, Scott C. Racial and ethnic disparities in health: an emergency medicine perspective. *Acad Emerg Med*. 2003;10:1289–1293.
95. Nandi A, Galea S, Lopez G, Nandi V, Strongarone S, Ompad DC. Access to and use of health services among undocumented Mexican immigrants in a US urban area. *Am J Public Health*. 2008;98:2011–2020. doi: 10.2105/AJPH.2006.096222.
96. Lutfiyya MN, Lipsky MS, Bales RW, Cha I, McGrath C. Disparities in knowledge of heart attack and stroke symptoms among adult men: an analysis of behavioral risk factor surveillance survey data. *J Natl Med Assoc*. 2008;100:1116–1124.
97. Barber PA, Zhang J, Demchuk AM, Hill MD, Buchan AM. Why are stroke patients excluded from TPA therapy? An analysis of patient eligibility. *Neurology*. 2001;56:1015–1020.
98. Diez Roux AV, Merkin SS, Arnett D, Chambless L, Massing M, Nieto FJ, Sorlie P, Szklo M, Tyroler HA, Watson RL. Neighborhood of residence and incidence of coronary heart disease. *N Engl J Med*. 2001;345:99–106. doi: 10.1056/NEJM200107123450205.
99. Brown AF, Liang LJ, Vassar SD, Stein-Merkin S, Longstreth WT Jr, Ovbiagele B, Yan T, Escarce JJ. Neighborhood disadvantage and ischemic stroke: the Cardiovascular Health Study (CHS). *Stroke*. 2011;42:3363–3368. doi: 10.1161/STROKEAHA.111.622134.
100. Chaix B, Rosvall M, Lynch J, Merlo J. Disentangling contextual effects on cause-specific mortality in a longitudinal 23-year follow-up study: impact of population density or socioeconomic environment? *Int J Epidemiol*. 2006;35:633–643. doi: 10.1093/ije/dyl009.
101. Chaix B, Rosvall M, Merlo J. Neighborhood socioeconomic deprivation and residential instability: effects on incidence of ischemic heart disease and survival after myocardial infarction. *Epidemiology*. 2007;18:104–111. doi: 10.1097/01.ede.0000249573.22856.9a.
102. Chaix B, Rosvall M, Merlo J. Recent increase of neighborhood socioeconomic effects on ischemic heart disease mortality: a multilevel survival analysis of two large Swedish cohorts. *Am J Epidemiol*. 2007;165:22–26. doi: 10.1093/aje/kwj322.
103. Baibas N, Trichopoulou A, Voridis E, Trichopoulos D. Residence in mountainous compared with lowland areas in relation to total and coronary mortality: a study in rural Greece. *J Epidemiol Community Health*. 2005;59:274–278. doi: 10.1136/jech.2004.025510.
104. Auchincloss AH, Mujahid MS, Shen M, Michos ED, Whitt-Glover MC, Diez Roux AV. Neighborhood health-promoting resources and obesity risk (the Multi-Ethnic Study of Atherosclerosis). *Obesity (Silver Spring)*. 2013;21:621–628. doi: 10.1038/oby.2012.91.
105. Auchincloss AH, Diez Roux AV, Mujahid MS, Shen M, Bertoni AG, Carnethon MR. Neighborhood resources for physical activity and healthy foods and incidence of type 2 diabetes mellitus: the Multi-Ethnic Study of Atherosclerosis. *Arch Intern Med*. 2009;169:1698–1704. doi: 10.1001/archinternmed.2009.302.
106. Papas MA, Alberg AJ, Ewing R, Helzlsouer KJ, Gary TL, Klassen AC. The built environment and obesity. *Epidemiol Rev*. 2007;29:129–143. doi: 10.1093/epirev/mxm009.
107. Clark CJ, Guo H, Lunos S, Aggarwal NT, Beck T, Evans DA, Mendes de Leon C, Everson-Rose SA. Neighborhood cohesion is associated with reduced risk of stroke mortality. *Stroke*. 2011;42:1212–1217. doi: 10.1161/STROKEAHA.110.609164.
108. Fujino Y, Tanabe N, Honjo K, Suzuki S, Shirai K, Iso H, Tamakoshi A; JACC Study Group. A prospective cohort study of neighborhood stress and ischemic heart disease in Japan: a multilevel analysis using the JACC study data. *BMC Public Health*. 2011;11:398. doi: 10.1186/1471-2458-11-398.
109. Ludwig J, Sanbonmatsu L, Gennetian L, Adam E, Duncan GJ, Katz LF, Kessler RC, Kling JR, Lindau ST, Whitaker RC, McDade TW. Neighborhoods, obesity, and diabetes: a randomized social experiment. *N Engl J Med*. 2011;365:1509–1519. doi: 10.1056/NEJMsa1103216.
110. Binswanger IA, Stern MF, Deyo RA, Heagerty PJ, Cheadle A, Elmore JG, Koepsell TD. Release from prison: a high risk of death for former inmates [published correction appears in *N Engl J Med*. 2007;356:536]. *N Engl J Med*. 2007;356:157–165. doi: 10.1056/NEJMsa064115.
111. Wang EA, Pletcher M, Lin F, Vittinghoff E, Kertesz SG, Kiefe CI, Bibbins-Domingo K. Incarceration, incident hypertension, and access to health care: findings from the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Arch Intern Med*. 2009;169:687–693. doi: 10.1001/archinternmed.2009.26.
112. Rutledge T, Reis VA, Linke SE, Greenberg BH, Mills PJ. Depression in heart failure: a meta-analytic review of prevalence, intervention effects, and associations with clinical outcomes. *J Am Coll Cardiol*. 2006;48:1527–1537. doi: 10.1016/j.jacc.2006.06.055.
113. Barth J, Schumacher M, Herrmann-Lingen C. Depression as a risk factor for mortality in patients with coronary heart disease: a meta-analysis. *Psychosom Med*. 2004;66:802–813. doi: 10.1097/01.psy.0000146332.53619.b2.
114. van Melle JP, de Jonge P, Spijkerman TA, Tijssen JG, Ormel J, van Veldhuisen DJ, van den Brink RH, van den Berg MP. Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: a meta-analysis. *Psychosom Med*. 2004;66:814–822. doi: 10.1097/01.psy.0000146294.82810.9c.
115. Frasure-Smith N, Lespérance F. Reflections on depression as a cardiac risk factor. *Psychosom Med*. 2005;67(suppl 1):S19–S25. doi: 10.1097/01.psy.0000162253.07959.db.
116. Sørensenf C, Friis-Hasché E, Haghfelt T, Bech P. Postmyocardial infarction mortality in relation to depression: a systematic critical review. *Psychother Psychosom*. 2005;74:69–80. doi: 10.1159/000083165.
117. van Reedt Dortland AK, Giltay EJ, van Veen T, Zitman FG, Penninx BW. Longitudinal relationship of depressive and anxiety symptoms with dyslipidemia and abdominal obesity. *Psychosom Med*. 2013;75:83–89. doi: 10.1097/PSY.0b013e318274d30f.
118. Frasure-Smith N, Lespérance F, Talajic M. Depression and 18-month prognosis after myocardial infarction. *Circulation*. 1995;91:999–1005.
119. Carney RM, Freedland KE. Depression, mortality, and medical morbidity in patients with coronary heart disease. *Biol Psychiatry*. 2003;54:241–247.
120. Nicholson A, Kuper H, Hemingway H. Depression as an aetiologic and prognostic factor in coronary heart disease: a meta-analysis of 6362 events among 146 538 participants in 54 observational studies. *Eur Heart J*. 2006;27:2763–2774. doi: 10.1093/eurheartj/ehl338.
121. Rugulies R. Depression as a predictor for coronary heart disease. a review and meta-analysis. *Am J Prev Med*. 2002;23:51–61.
122. Kronish IM, Carson AP, Davidson KW, Muntner P, Safford MM. Depressive symptoms and cardiovascular health by the American Heart Association's definition in the Reasons for Geographic and Racial Differences in Stroke (REGARDS) study. *PLoS One*. 2012;7:e52771. doi: 10.1371/journal.pone.0052771.

123. Lespérance F, Frasure-Smith N, Théroux P, Irwin M. The association between major depression and levels of soluble intercellular adhesion molecule 1, interleukin-6, and C-reactive protein in patients with recent acute coronary syndromes. *Am J Psychiatry*. 2004;161:271–277.
124. Empana JP, Sykes DH, Luc G, Juhan-Vague I, Arveiler D, Ferrieres J, Amouyel P, Bingham A, Montaye M, Ruidavets JB, Haas B, Evans A, Jouven X, Ducimetiere P; PRIME Study Group. Contributions of depressive mood and circulating inflammatory markers to coronary heart disease in healthy European men: the Prospective Epidemiological Study of Myocardial Infarction (PRIME). *Circulation*. 2005;111:2299–2305. doi: 10.1161/01.CIR.0000164203.54111.AE.
125. Pollock BG, Laghrissi-Thode F, Wagner WR. Evaluation of platelet activation in depressed patients with ischemic heart disease after paroxetine or nortriptyline treatment. *J Clin Psychopharmacol*. 2000;20:137–140.
126. Serebruany VL, Glassman AH, Malinin AI, Sane DC, Finkel MS, Krishnan RR, Atar D, Lekht V, O'Connor CM. Enhanced platelet/endothelial activation in depressed patients with acute coronary syndromes: evidence from recent clinical trials. *Blood Coagul Fibrinolysis*. 2003;14:563–567. doi: 10.1097/01.mbc.0000061336.06975.52.
127. Carney RM, Blumenthal JA, Stein PK, Watkins L, Catellier D, Berkman LF, Czajkowski SM, O'Connor C, Stone PH, Freedland KE. Depression, heart rate variability, and acute myocardial infarction. *Circulation*. 2001;104:2024–2028.
128. Taylor CB, Conrad A, Wilhelm FH, Neri E, DeLorenzo A, Kramer MA, Giese-Davis J, Roth WT, Oka R, Cooke JP, Kraemer H, Spiegel D. Psychophysiological and cortisol responses to psychological stress in depressed and nondepressed older men and women with elevated cardiovascular disease risk. *Psychosom Med*. 2006;68:538–546. doi: 10.1097/01.psy.0000222372.16274.92.
129. Sherwood A, Hinderliter AL, Watkins LL, Waugh RA, Blumenthal JA. Impaired endothelial function in coronary heart disease patients with depressive symptomatology. *J Am Coll Cardiol*. 2005;46:656–659. doi: 10.1016/j.jacc.2005.05.041.
130. Strik JJ, Denollet J, Lousberg R, Honig A. Comparing symptoms of depression and anxiety as predictors of cardiac events and increased health care consumption after myocardial infarction. *J Am Coll Cardiol*. 2003;42:1801–1807.
131. Frasure-Smith N, Lespérance F. Depression and anxiety as predictors of 2-year cardiac events in patients with stable coronary artery disease. *Arch Gen Psychiatry*. 2008;65:62–71. doi: 10.1001/archgenpsychiatry.2007.4.
132. Tully PJ, Baker RA, Knight JL. Anxiety and depression as risk factors for mortality after coronary artery bypass surgery. *J Psychosom Res*. 2008;64:285–290. doi: 10.1016/j.jpsychores.2007.09.007.
133. Shibeshi WA, Young-Xu Y, Blatt CM. Anxiety worsens prognosis in patients with coronary artery disease. *J Am Coll Cardiol*. 2007;49:2021–2027. doi: 10.1016/j.jacc.2007.03.007.
134. Ahern DK, Gorkin L, Anderson JL, Tierney C, Hallstrom A, Ewart C, Capone RJ, Schron E, Kornfeld D, Herd JA, Richardson DW, Follick MJ; The CAPS Investigators. Biobehavioral variables and mortality or cardiac arrest in the Cardiac Arrhythmia Pilot Study (CAPS). *Am J Cardiol*. 1990;66:59–62.
135. Frasure-Smith N, Lespérance F. Depression and other psychological risks following myocardial infarction. *Arch Gen Psychiatry*. 2003;60:627–636. doi: 10.1001/archpsyc.60.6.627.
136. Kornerup H, Zwisler AD, Prescott E; DANREHAB Group, Copenhagen, Denmark. No association between anxiety and depression and adverse clinical outcome among patients with cardiovascular disease: findings from the DANREHAB trial. *J Psychosom Res*. 2011;71:207–214. doi: 10.1016/j.jpsychores.2011.04.006.
137. Lane D, Carroll D, Ring C, Beevers DG, Lip GY. Mortality and quality of life 12 months after myocardial infarction: effects of depression and anxiety. *Psychosom Med*. 2001;63:221–230.
138. Ohira T, Diez Roux AV, Polak JF, Homma S, Iso H, Wasserman BA. Associations of anger, anxiety, and depressive symptoms with carotid arterial wall thickness: the Multi-Ethnic Study of Atherosclerosis. *Psychosom Med*. 2012;74:517–525. doi: 10.1097/PSY.0b013e31824f6267.
139. Williams JE, Paton CC, Siegler IC, Eigenbrodt ML, Nieto FJ, Tyroler HA. Anger proneness predicts coronary heart disease risk: prospective analysis from the Atherosclerosis Risk in Communities (ARIC) study. *Circulation*. 2000;101:2034–2039.
140. Everson SA, Kauhanen J, Kaplan GA, Goldberg DE, Julkunen J, Tuomilehto J, Salonen JT. Hostility and increased risk of mortality and acute myocardial infarction: the mediating role of behavioral risk factors. *Am J Epidemiol*. 1997;146:142–152.
141. Lahad A, Heckbert SR, Koepsell TD, Psaty BM, Patrick DL. Hostility, aggression and the risk of nonfatal myocardial infarction in postmenopausal women. *J Psychosom Res*. 1997;43:183–195.
142. Barefoot JC, Larsen S, von der Lieth L, Schroll M. Hostility, incidence of acute myocardial infarction, and mortality in a sample of older Danish men and women. *Am J Epidemiol*. 1995;142:477–484.
143. Goodman M, Quigley J, Moran G, Meilman H, Sherman M. Hostility predicts restenosis after percutaneous transluminal coronary angioplasty. *Mayo Clin Proc*. 1996;71:729–734. doi: 10.1016/S0025-6196(11)64836-2.
144. Deleted in proof.
145. Matthews KA, Gump BB, Harris KF, Haney TL, Barefoot JC. Hostile behaviors predict cardiovascular mortality among men enrolled in the Multiple Risk Factor Intervention Trial. *Circulation*. 2004;109:66–70. doi: 10.1161/01.CIR.0000105766.33142.13.
146. Rutledge T, Hogan BE. A quantitative review of prospective evidence linking psychological factors with hypertension development. *Psychosom Med*. 2002;64:758–766.
147. Marsland AL, Prather AA, Petersen KL, Cohen S, Manuck SB. Antagonistic characteristics are positively associated with inflammatory markers independently of trait negative emotionality. *Brain Behav Immun*. 2008;22:753–761. doi: 10.1016/j.bbi.2007.11.008.
148. Graham JE, Robles TF, Kiecolt-Glaser JK, Malarkey WB, Bissell MG, Glaser R. Hostility and pain are related to inflammation in older adults. *Brain Behav Immun*. 2006;20:389–400. doi: 10.1016/j.bbi.2005.11.002.
149. Yan LL, Liu K, Matthews KA, Daviglus ML, Ferguson TF, Kiefe CI. Psychosocial factors and risk of hypertension: the Coronary Artery Risk Development in Young Adults (CARDIA) study. *JAMA*. 2003;290:2138–2148. doi: 10.1001/jama.290.16.2138.
150. Calhoun PS, Bosworth HB, Siegler IC, Bastian LA. The relationship between hostility and behavioral risk factors for poor health in women veterans. *Prev Med*. 2001;33:552–557. doi: 10.1006/pmed.2001.0921.
151. Richardson S, Shaffer JA, Falzon L, Krupka D, Davidson KW, Edmondson D. Meta-analysis of perceived stress and its association with incident coronary heart disease. *Am J Cardiol*. 2012;110:1711–1716. doi: 10.1016/j.amjcard.2012.08.004.
152. Rozanski A, Blumenthal JA, Davidson KW, Saab PG, Kubzansky L. The epidemiology, pathophysiology, and management of psychosocial risk factors in cardiac practice: the emerging field of behavioral cardiology. *J Am Coll Cardiol*. 2005;45:637–651. doi: 10.1016/j.jacc.2004.12.005.
153. Gebreab SY, Diez-Roux AV, Hickson DA, Boykin S, Sims M, Sarpong DF, Taylor HA, Wyatt SB. The contribution of stress to the social patterning of clinical and subclinical CVD risk factors in African Americans: the Jackson Heart Study. *Soc Sci Med*. 2012;75:1697–1707. doi: 10.1016/j.socscimed.2012.06.003.
154. Matthews KA, Gallo LC, Taylor SE. Are psychosocial factors mediators of socioeconomic status and health connections? A progress report and blueprint for the future. *Ann NY Acad Sci*. 2010;1186:146–173. doi: 10.1111/j.1749-6632.2009.05332.x.
155. Lemstra M, Neudorf C, D'Arcy C, Kunst A, Warren LM, Bennett NR. A systematic review of depressed mood and anxiety by SES in youth aged 10-15 years. *Can J Public Health*. 2008;99:125–129.
156. Hudson DL, Puterman E, Bibbins-Domingo K, Matthews KA, Adler NE. Race, life course socioeconomic position, racial discrimination, depressive symptoms and self-rated health. *Soc Sci Med*. 2013;97:7–14. doi: 10.1016/j.socscimed.2013.07.031.
157. Lantz PM, House JS, Mero RP, Williams DR. Stress, life events, and socioeconomic disparities in health: results from the Americans' Changing Lives Study. *J Health Soc Behav*. 2005;46:274–288.
158. Butterworth P, Olesen SC, Leach LS. The role of hardship in the association between socio-economic position and depression. *Aust NZ J Psychiatry*. 2012;46:364–373. doi: 10.1177/0004867411433215.
159. Vives A, Amable M, Ferrer M, Moncada S, Llorens C, Muntaner C, Benavides FG, Benach J. Employment precariousness and poor mental health: evidence from Spain on a new social determinant of health. *J Environ Public Health*. 2013;2013:978656. doi: 10.1155/2013/978656.
160. Quesnel-Vallée A, Taylor M. Socioeconomic pathways to depressive symptoms in adulthood: evidence from the National Longitudinal Survey of Youth 1979. *Soc Sci Med*. 2012;74:734–743. doi: 10.1016/j.socscimed.2011.10.038.
161. Clark AM, DesMeules M, Luo W, Duncan AS, Wielgosz A. Socioeconomic status and cardiovascular disease: risks and implications for care. *Nat Rev Cardiol*. 2009;6:712–722. doi: 10.1038/nrcardio.2009.163.

162. Kivimäki M, Shipley MJ, Ferrie JE, Singh-Manoux A, Batty GD, Chandola T, Marmot MG, Smith GD. Best-practice interventions to reduce socioeconomic inequalities of coronary heart disease mortality in UK: a prospective occupational cohort study. *Lancet*. 2008;372:1648–1654. doi: 10.1016/S0140-6736(08)61688-8.
163. Siegrist J, Starke D, Chandola T, Godin I, Marmot M, Niedhammer I, Peter R. The measurement of effort-reward imbalance at work: European comparisons. *Soc Sci Med*. 2004;58:1483–1499. doi: 10.1016/S0277-9536(03)00351-4.
164. Bassett E, Moore S. Social capital and depressive symptoms: the association of psychosocial and network dimensions of social capital with depressive symptoms in Montreal, Canada. *Soc Sci Med*. 2013;86:96–102. doi: 10.1016/j.socscimed.2013.03.005.
165. Araya R, Dunstan F, Playle R, Thomas H, Palmer S, Lewis G. Perceptions of social capital and the built environment and mental health. *Soc Sci Med*. 2006;62:3072–3083. doi: 10.1016/j.socscimed.2005.11.037.
166. Sturm R, Cohen DA. Suburban sprawl and physical and mental health. *Public Health*. 2004;118:488–496. doi: 10.1016/j.puhe.2004.02.007.
167. Smith TW, Traupman EK, Uchino BN, Berg CA. Interpersonal circumplex descriptions of psychosocial risk factors for physical illness: application to hostility, neuroticism, and marital adjustment. *J Pers*. 2010;78:1011–1036. doi: 10.1111/j.1467-6494.2010.00641.x.
168. Bower KM, Thorpe RJ Jr, LaVeist TA. Perceived racial discrimination and mental health in low-income, urban-dwelling whites. *Int J Health Serv*. 2013;43:267–280.
169. Choi KH, Paul J, Ayala G, Boylan R, Gregorich SE. Experiences of discrimination and their impact on the mental health among African American, Asian and Pacific Islander, and Latino men who have sex with men. *Am J Public Health*. 2013;103:868–874. doi: 10.2105/AJPH.2012.301052.
170. Lewis TT, Aiello AE, Leurgans S, Kelly J, Barnes LL. Self-reported experiences of everyday discrimination are associated with elevated C-reactive protein levels in older African-American adults. *Brain Behav Immun*. 2010;24:438–443. doi: 10.1016/j.bbi.2009.11.011.
171. Fortmann AL, Gallo LC, Philis-Tsimikas A. Glycemic control among Latinos with type 2 diabetes: the role of social-environmental support resources. *Health Psychol*. 2011;30:251–258. doi: 10.1037/a0022850.
172. O'Neal CW, Wickrama KA, Ralston PA, Ilich JZ, Harris CM, Coccia C, Young-Clark I, Lemacks J. Examining change in social support and fruit and vegetable consumption in African American adults. *J Nutr Health Aging*. 2014;18:10–14. doi: 10.1007/s12603-013-0376-1.
173. Shaya FT, Chirikov VV, Howard D, Foster C, Costas J, Snitker S, Frimpter J, Kucharski K. Effect of social networks intervention in type 2 diabetes: a partial randomised study. *J Epidemiol Community Health*. 2014;68:326–332. doi: 10.1136/jech-2013-203274.
174. Ochodo C, Ndetei DM, Moturi WN, Otieno JO. External built residential environment characteristics that affect mental health of adults. *J Urban Health*. 2014;91:908–927. doi: 10.1007/s11524-013-9852-5.
175. Galea S, Ahern J, Rudenstine S, Wallace Z, Vlahov D. Urban built environment and depression: a multilevel analysis. *J Epidemiol Community Health*. 2005;59:822–827. doi: 10.1136/jech.2005.033084.
176. Lorenc T, Clayton S, Neary D, Whitehead M, Petticrew M, Thomson H, Cummins S, Sowden A, Renton A. Crime, fear of crime, environment, and mental health and wellbeing: mapping review of theories and causal pathways. *Health Place*. 2012;18:757–765. doi: 10.1016/j.healthplace.2012.04.001.
177. Guite HF, Clark C, Ackrill G. The impact of the physical and urban environment on mental well-being. *Public Health*. 2006;120:1117–1126. doi: 10.1016/j.puhe.2006.10.005.
178. Walters K, Breeze E, Wilkinson P, Price GM, Bulpitt CJ, Fletcher A. Local area deprivation and urban-rural differences in anxiety and depression among people older than 75 years in Britain. *Am J Public Health*. 2004;94:1768–1774.
179. Latkin CA, Curry AD. Stressful neighborhoods and depression: a prospective study of the impact of neighborhood disorder. *J Health Soc Behav*. 2003;44:34–44.
180. Roe JJ, Thompson CW, Aspinall PA, Brewer MJ, Duff EI, Miller D, Mitchell R, Clow A. Green space and stress: evidence from cortisol measures in deprived urban communities. *Int J Environ Res Public Health*. 2013;10:4086–4103. doi: 10.3390/ijerph10094086.
181. Thompson CW, Roe J, Aspinall P, Mitchell R, Clow A, Miller D. More green space is linked to less stress in deprived communities: evidence from salivary cortisol patterns. *Landscape and Urban Planning*. 2012;105:221–229.
182. Maas J, van Dillen SM, Verheij RA, Groenewegen PP. Social contacts as a possible mechanism behind the relation between green space and health. *Health Place*. 2009;15:586–595. doi: 10.1016/j.healthplace.2008.09.006.
183. Barton J, Pretty J. What is the best dose of nature and green exercise for improving mental health? A multi-study analysis. *Environ Sci Technol*. 2010;44:3947–3955. doi: 10.1021/es903183r.
184. van den Berg AE, Maas J, Verheij RA, Groenewegen PP. Green space as a buffer between stressful life events and health. *Soc Sci Med*. 2010;70:1203–1210. doi: 10.1016/j.socscimed.2010.01.002.
185. Mitchell R, Popham F. Effect of exposure to natural environment on health inequalities: an observational population study. *Lancet*. 2008;372:1655–1660. doi: 10.1016/S0140-6736(08)61689-X.
186. Leventhal T, Brooks-Gunn J. Moving to opportunity: an experimental study of neighborhood effects on mental health. *Am J Public Health*. 2003;93:1576–1582.
187. Christensen AJ, Smith TW. Cynical hostility and cardiovascular reactivity during self-disclosure. *Psychosom Med*. 1993;55:193–202.
188. Lett HS, Blumenthal JA, Babyak MA, Sherwood A, Strauman T, Robins C, Newman MF. Depression as a risk factor for coronary artery disease: evidence, mechanisms, and treatment. *Psychosom Med*. 2004;66:305–315.
189. Carney RM, Freedland KE, Miller GE, Jaffe AS. Depression as a risk factor for cardiac mortality and morbidity: a review of potential mechanisms. *J Psychosom Res*. 2002;53:897–902.
190. Thomas AJ, Kalaria RN, O'Brien JT. Depression and vascular disease: what is the relationship? *J Affect Disord*. 2004;79:81–95. doi: 10.1016/S0165-0327(02)00349-X.
191. Kubzansky LD, Kawachi I. Going to the heart of the matter: do negative emotions cause coronary heart disease? *J Psychosom Res*. 2000;48:323–337.
192. Everson-Rose SA, Lewis TT. Psychosocial factors and cardiovascular diseases. *Annu Rev Public Health*. 2005;26:469–500. doi: 10.1146/annurev.publhealth.26.021304.144542.
193. DiMatteo MR, Lepper HS, Croghan TW. Depression is a risk factor for noncompliance with medical treatment: meta-analysis of the effects of anxiety and depression on patient adherence. *Arch Intern Med*. 2000;160:2101–2107.
194. Ziegelstein RC, Fauerbach JA, Stevens SS, Romanelli J, Richter DP, Bush DE. Patients with depression are less likely to follow recommendations to reduce cardiac risk during recovery from a myocardial infarction. *Arch Intern Med*. 2000;160:1818–1823.
195. Ades PA, Waldmann ML, McCann WJ, Weaver SO. Predictors of cardiac rehabilitation participation in older coronary patients. *Arch Intern Med*. 1992;152:1033–1035.
196. Glazer KM, Emery CF, Frid DJ, Banyasz RE. Psychological predictors of adherence and outcomes among patients in cardiac rehabilitation. *J Cardiopulm Rehabil*. 2002;22:40–46.
197. Hasler G, Drevets WC, Manji HK, Charney DS. Discovering endophenotypes for major depression. *Neuropsychopharmacology*. 2004;29:1765–1781. doi: 10.1038/sj.npp.1300506.
198. Stewart JC, Janicki DL, Muldoon MF, Sutton-Tyrrell K, Kamarck TW. Negative emotions and 3-year progression of subclinical atherosclerosis. *Arch Gen Psychiatry*. 2007;64:225–233. doi: 10.1001/archpsyc.64.2.225.
199. Dorr N, Brosschot JF, Sollers JJ 3rd, Thayer JF. Damned if you do, damned if you don't: the differential effect of expression and inhibition of anger on cardiovascular recovery in black and white males. *Int J Psychophysiol*. 2007;66:125–134. doi: 10.1016/j.ijpsycho.2007.03.022.
200. Doster JA, Purdum MB, Martin LA, Goven AJ, Moorefield R. Gender differences, anger expression, and cardiovascular risk. *J Nerv Ment Dis*. 2009;197:552–554. doi: 10.1097/NMD.0b013e3181aac81b.
201. Thombs BD, de Jonge P, Coyne JC, Whooley MA, Frasure-Smith N, Mitchell AJ, Zuidersma M, Eze-Nliam C, Lima BB, Smith CG, Soderlund K, Ziegelstein RC. Depression screening and patient outcomes in cardiovascular care: a systematic review. *JAMA*. 2008;300:2161–2171. doi: 10.1001/jama.2008.667.
202. Shaffer JA, Whang W, Shimbo D, Burg M, Schwartz JE, Davidson KW. Do different depression phenotypes have different risks for recurrent coronary heart disease? *Health Psychol Rev*. 2012;6:165–179. doi: 10.1080/17437199.2010.527610.
203. Lichtman JH, Bigger JT Jr, Blumenthal JA, Frasure-Smith N, Kaufmann PG, Lespérance F, Mark DB, Sheps DS, Taylor CB, Froelicher ES. Depression and coronary heart disease: recommendations for screening, referral, and treatment: a science advisory from the American Heart Association Prevention Committee of the Council on Cardiovascular Nursing, Council on Clinical Cardiology, Council on Epidemiology

and Prevention, and Interdisciplinary Council on Quality of Care and Outcomes Research. *Circulation*. 2008;118:1768–1775. doi: 10.1161/CIRCULATIONAHA.108.190769.
204. Alegría M, Chatterji P, Wells K, Cao Z, Chen CN, Takeuchi D, Jackson J, Meng XL. Disparity in depression treatment among racial and ethnic minority populations in the United States. *Psychiatr Serv*. 2008;59:1264–1272. doi: 10.1176/appi.ps.59.11.1264.
205. Jimenez DE, Alegría M, Chen CN, Chan D, Laderman M. Prevalence of psychiatric illnesses in older ethnic minority adults. *J Am Geriatr Soc*. 2010;58:256–264. doi: 10.1111/j.1532-5415.2009.02685.x.
206. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication [published correction appears in *Arch Gen Psychiatry*. 2005;62:768]. *Arch Gen Psychiatry*. 2005;62:593–602. doi: 10.1001/archpsyc.62.6.593.
207. Waldman SV, Blumenthal JA, Babyak MA, Sherwood A, Sketch M, Davidson J, Watkins LL. Ethnic differences in the treatment of depression in patients with ischemic heart disease. *Am Heart J*. 2009;157:77–83. doi: 10.1016/j.ahj.2008.08.013.
208. Jonas BS, Franks P, Ingram DD. Are symptoms of anxiety and depression risk factors for hypertension? Longitudinal evidence from the National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study. *Arch Fam Med*. 1997;6:43–49.
209. Jonas BS, Mussolino ME. Symptoms of depression as a prospective risk factor for stroke. *Psychosom Med*. 2000;62:463–471.
210. Lewis TT, Everson-Rose SA, Colvin A, Matthews K, Bromberger JT, Sutton-Tyrrell K. Interactive effects of race and depressive symptoms on calcification in African American and white women. *Psychosom Med*. 2009;71:163–170. doi: 10.1097/PSY.0b013e31819080e5.
211. Williams JE, Couper DJ, Din-Dzietham R, Nieto FJ, Folsom AR. Race-gender differences in the association of trait anger with subclinical carotid artery atherosclerosis: the Atherosclerosis Risk in Communities Study. *Am J Epidemiol*. 2007;165:1296–1304. doi: 10.1093/aje/kwm001.
212. Troxel WM, Matthews KA, Bromberger JT, Sutton-Tyrrell K. Chronic stress burden, discrimination, and subclinical carotid artery disease in African American and Caucasian women. *Health Psychol*. 2003;22:300–309.
213. West DS, Elaine Prewitt T, Bursac Z, Felix HC. Weight loss of black, white, and Hispanic men and women in the Diabetes Prevention Program. *Obesity (Silver Spring)*. 2008;16:1413–1420. doi: 10.1038/oby.2008.224.
214. Stevens VJ, Obarzanek E, Cook NR, Lee IM, Appel LJ, Smith West D, Milas NC, Mattfeldt-Beman M, Belden L, Bragg C, Millstone M, Raczynski J, Brewer A, Singh B, Cohen J; Trials for the Hypertension Prevention Research Group. Long-term weight loss and changes in blood pressure: results of the Trials of Hypertension Prevention, phase II. *Ann Intern Med*. 2001;134:1–11.
215. Maruthur NM, Wang NY, Appel LJ. Lifestyle interventions reduce coronary heart disease risk: results from the PREMIER Trial. *Circulation*. 2009;119:2026–2031. doi: 10.1161/CIRCULATIONAHA.108.809491.
216. Kershaw KN, Mezuk B, Abdou CM, Rafferty JA, Jackson JS. Socioeconomic position, health behaviors, and C-reactive protein: a moderated-mediation analysis. *Health Psychol*. 2010;29:307–316. doi: 10.1037/a0019286.
217. Lantz PM, Golberstein E, House JS, Morenoff J. Socioeconomic and behavioral risk factors for mortality in a national 19-year prospective study of U.S. adults. *Soc Sci Med*. 2010;70:1558–1566. doi: 10.1016/j.socscimed.2010.02.003.
218. van Oort FV, van Lenthe FJ, Mackenbach JP. Material, psychosocial, and behavioural factors in the explanation of educational inequalities in mortality in The Netherlands. *J Epidemiol Community Health*. 2005;59:214–220. doi: 10.1136/jech.2003.016493.
219. Stringhini S, Sabia S, Shipley M, Brunner E, Nabi H, Kivimaki M, Singh-Manoux A. Association of socioeconomic position with health behaviors and mortality. *JAMA*. 2010;303:1159–1166. doi: 10.1001/jama.2010.297.
220. Mullie P, Clarys P, Hulens M, Vansant G. Dietary patterns and socioeconomic position. *Eur J Clin Nutr*. 2010;64:231–238. doi: 10.1038/ejcn.2009.145.
221. Giskes K, Avendano M, Brug J, Kunst AE. A systematic review of studies on socioeconomic inequalities in dietary intakes associated with weight gain and overweight/obesity conducted among European adults. *Obes Rev*. 2010;11:413–429. doi: 10.1111/j.1467-789X.2009.00658.x.
222. Gilman SE, Abrams DB, Buka SL. Socioeconomic status over the life course and stages of cigarette use: initiation, regular use, and cessation. *J Epidemiol Community Health*. 2003;57:802–808.
223. Cameron AJ, Ball K, Pearson N, Lioret S, Crawford DA, Campbell K, Hesketh K, McNaughton SA. Socioeconomic variation in diet and activity-related behaviours of Australian children and adolescents aged 2-16 years. *Pediatr Obes*. 2012;7:329–342. doi: 10.1111/j.2047-6310.2012.00060.x.
224. Hanson MD, Chen E. Socioeconomic status and health behaviors in adolescence: a review of the literature. *J Behav Med*. 2007;30:263–285. doi: 10.1007/s10865-007-9098-3.
225. Power C, Graham H, Due P, Hallqvist J, Joung I, Kuh D, Lynch J. The contribution of childhood and adult socioeconomic position to adult obesity and smoking behaviour: an international comparison. *Int J Epidemiol*. 2005;34:335–344. doi: 10.1093/ije/dyh394.
226. Giesinger I, Goldblatt P, Howden-Chapman P, Marmot M, Kuh D, Brunner E. Association of socioeconomic position with smoking and mortality: the contribution of early life circumstances in the 1946 birth cohort. *J Epidemiol Community Health*. 2014;68:275–279. doi: 10.1136/jech-2013-203159.
227. Wallach-Kildemoes H, Andersen M, Diderichsen F, Lange T. Adherence to preventive statin therapy according to socioeconomic position. *Eur J Clin Pharmacol*. 2013;69:1553–1563. doi: 10.1007/s00228-013-1488-6.
228. Fleischer NL, Diez Roux AV, Hubbard AE. Inequalities in body mass index and smoking behavior in 70 countries: evidence for a social transition in chronic disease risk. *Am J Epidemiol*. 2012;175:167–176. doi: 10.1093/aje/kwr314.
229. Hollis JF, Gullion CM, Stevens VJ, Brantley PJ, Appel LJ, Ard JD, Champagne CM, Dalcin A, Erlinger TP, Funk K, Laferriere D, Lin PH, Loria CM, Samuel-Hodge C, Vollmer WM, Svetkey LP; Weight Loss Maintenance Trial Research Group. Weight loss during the intensive intervention phase of the Weight-Loss Maintenance Trial. *Am J Prev Med*. 2008;35:118–126. doi: 10.1016/j.amepre.2008.04.013.
230. Kumanyika S. Ethnic minorities and weight control research priorities: where are we now and where do we need to be? *Prev Med*. 2008;47:583–586. doi: 10.1016/j.ypmed.2008.09.012.
231. Rumble C, Pevalin DJ. Widening inequalities in the risk factors for cardiovascular disease amongst men in England between 1998 and 2006. *Public Health*. 2013;127:27–31. doi: 10.1016/j.puhe.2012.09.003.
232. Drewnowski A, Darmon N. Food choices and diet costs: an economic analysis. *J Nutr*. 2005;135:900–904.
233. Morland K, Wing S, Diez Roux A. The contextual effect of the local food environment on residents' diets: the Atherosclerosis Risk in Communities study. *Am J Public Health*. 2002;92:1761–1767.
234. Diaz VA, Mainous AG 3rd, Koopman RJ, Carek PJ, Geesey ME. Race and diet in the overweight: association with cardiovascular risk in a nationally representative sample. *Nutrition*. 2005;21:718–725. doi: 10.1016/j.nut.2004.11.010.
235. Zhang Y, Baik SH, Chang CC, Kaplan CM, Lave JR. Disability, race/ethnicity, and medication adherence among Medicare myocardial infarction survivors. *Am Heart J*. 2012;164:425–433.e4. doi: 10.1016/j.ahj.2012.05.021.
236. Lewey J, Shrank WH, Bowry AD, Kilabuk E, Brennan TA, Choudhry NK. Gender and racial disparities in adherence to statin therapy: a meta-analysis. *Am Heart J*. 2013;165:665–678, 678.e1. doi: 10.1016/j.ahj.2013.02.011.
237. Ndumele CD, Shaykevich S, Williams D, Hicks LS. Disparities in adherence to hypertensive care in urban ambulatory settings. *J Health Care Poor Underserved*. 2010;21:132–143. doi: 10.1353/hpu.0.0259.
238. Wu JR, Lennie TA, De Jong MJ, Frazier SK, Heo S, Chung ML, Moser DK. Medication adherence is a mediator of the relationship between ethnicity and event-free survival in patients with heart failure. *J Card Fail*. 2010;16:142–149. doi: 10.1016/j.cardfail.2009.10.017.
239. Wexler R, Feldman D, Larson D, Sinnott LT, Jones LA, Miner J; Ohio State University Primary Care Practice-Based Research Network. Adoption of exercise and readiness to change differ between whites and African-Americans with hypertension: a report from the Ohio State University Primary Care Practice-Based Research Network (OSU-PCPBRN). *J Am Board Fam Med*. 2008;21:358–360. doi: 10.3122/jabfm.2008.04.070175.
240. Schoenthaler A, Allegrante JP, Chaplin W, Ogedegbe G. The effect of patient-provider communication on medication adherence in hypertensive black patients: does race concordance matter? *Ann Behav Med*. 2012;43:372–382. doi: 10.1007/s12160-011-9342-5.

241. Cuffee YL, Hargraves JL, Rosal M, Briesacher BA, Schoenthaler A, Person S, Hullett S, Allison J. Reported racial discrimination, trust in physicians, and medication adherence among inner-city African Americans with hypertension. *Am J Public Health*. 2013;103:e55–e62. doi: 10.2105/AJPH.2013.301554.
242. Kaplan RC, Bhalodkar NC, Brown DL, White J, Brown EJ Jr. Differences by age and race/ethnicity in knowledge about hypercholesterolemia. *Cardiol Rev*. 2006;14:1–6.
243. Siegel K, Karus D, Schrimshaw EW. Racial differences in attitudes toward protease inhibitors among older HIV-infected men. *AIDS Care*. 2000;12:423–434. doi: 10.1080/09540120050123828.
244. Gallo LC, de Los Monteros KE, Shivpuri S. Socioeconomic status and health: what is the role of reserve capacity? *Curr Dir Psychol Sci*. 2009;18:269–274.
245. Teychenne M, Ball K, Salmon J. Correlates of socio-economic inequalities in women's television viewing: a study of intrapersonal, social and environmental mediators. *Int J Behav Nutr Phys Act*. 2012;9:3. doi: 10.1186/1479-5868-9-3.
246. Samuel-Hodge CD, Gizlice Z, Cai J, Brantley PJ, Ard JD, Svetkey LP. Family functioning and weight loss in a sample of African Americans and whites. *Ann Behav Med*. 2010;40:294–301. doi: 10.1007/s12160-010-9219-z.
247. Ashida S, Wilkinson AV, Koehly LM. Motivation for health screening: evaluation of social influence among Mexican-American adults. *Am J Prev Med*. 2010;38:396–402. doi: 10.1016/j.amepre.2009.12.028.
248. Echeverría SE, Pentakota SR, Abraído-Lanza AF, Janevic T, Gundersen DA, Ramirez SM, Delnevo CD. Clashing paradigms: an empirical examination of cultural proxies and socioeconomic condition shaping Latino health. *Ann Epidemiol*. 2013;23:608–613. doi: 10.1016/j.annepidem.2013.07.023.
249. Ewing R, Schmid T, Killingsworth R, Zlot A, Raudenbush S. Relationship between urban sprawl and physical activity, obesity, and morbidity. *Am J Health Promot*. 2003;18:47–57.
250. Feng J, Glass TA, Curriero FC, Stewart WF, Schwartz BS. The built environment and obesity: a systematic review of the epidemiologic evidence. *Health Place*. 2010;16:175–190. doi: 10.1016/j.healthplace.2009.09.008.
251. Fields R, Kaczynski AT, Bopp M, Fallon E. Built environment associations with health behaviors among Hispanics. *J Phys Act Health*. 2013;10:335–342.
252. Gilliland JA, Rangel CY, Healy MA, Tucker P, Loebach JE, Hess PM, He M, Irwin JD, Wilk P. Linking childhood obesity to the built environment: a multi-level analysis of home and school neighbourhood factors associated with body mass index. *Can J Public Health*. 2012;103(suppl 3):eS15–eS21.
253. Mujahid MS, Diez Roux AV, Borrell LN, Nieto FJ. Cross-sectional and longitudinal associations of BMI with socioeconomic characteristics. *Obes Res*. 2005;13:1412–1421. doi: 10.1038/oby.2005.171.
254. Do DP, Dubowitz T, Bird CE, Lurie N, Escarce JJ, Finch BK. Neighborhood context and ethnicity differences in body mass index: a multilevel analysis using the NHANES III survey (1988-1994). *Econ Hum Biol*. 2007;5:179–203. doi: 10.1016/j.ehb.2007.03.006.
255. Wang MC, Kim S, Gonzalez AA, MacLeod KE, Winkleby MA. Socioeconomic and food-related physical characteristics of the neighbourhood environment are associated with body mass index. *J Epidemiol Community Health*. 2007;61:491–498. doi: 10.1136/jech.2006.051680.
256. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. *Science*. 1998;280:1371–1374.
257. Foster S, Giles-Corti B. The built environment, neighborhood crime and constrained physical activity: an exploration of inconsistent findings. *Prev Med*. 2008;47:241–251. doi: 10.1016/j.ypmed.2008.03.017.
258. Black JL, Macinko J. The changing distribution and determinants of obesity in the neighborhoods of New York City, 2003-2007. *Am J Epidemiol*. 2010;171:765–775. doi: 10.1093/aje/kwp458.
259. Gordon-Larsen P, Nelson MC, Page P, Popkin BM. Inequality in the built environment underlies key health disparities in physical activity and obesity. *Pediatrics*. 2006;117:417–424. doi: 10.1542/peds.2005-0058.
260. Oreskovic NM, Kuhlthau KA, Romm D, Perrin JM. Built environment and weight disparities among children in high- and low-income towns. *Acad Pediatr*. 2009;9:315–321. doi: 10.1016/j.acap.2009.02.009.
261. Mujahid MS, Diez Roux AV, Shen M, Gowda D, Sánchez B, Shea S, Jacobs DR Jr, Jackson SA. Relation between neighborhood environments and obesity in the Multi-Ethnic Study of Atherosclerosis. *Am J Epidemiol*. 2008;167:1349–1357. doi: 10.1093/aje/kwn047.
262. Li F, Harmer P, Cardinal BJ, Vongjaturapat N. Built environment and changes in blood pressure in middle aged and older adults. *Prev Med*. 2009;48:237–241.
263. Boehmer TK, Hoehner CM, Deshpande AD, Brennan Ramirez LK, Brownson RC. Perceived and observed neighborhood indicators of obesity among urban adults. *Int J Obes (Lond)*. 2007;31:968–977. doi: 10.1038/sj.ijo.0803531.
264. Durand CP, Andalib M, Dunton GF, Wolch J, Pentz MA. A systematic review of built environment factors related to physical activity and obesity risk: implications for smart growth urban planning. *Obes Rev*. 2011;12:e173–e182. doi: 10.1111/j.1467-789X.2010.00826.x.
265. Fitzhugh EC, Bassett DR Jr, Evans MF. Urban trails and physical activity: a natural experiment. *Am J Prev Med*. 2010;39:259–262. doi: 10.1016/j.amepre.2010.05.010.
266. Cameron AJ, Thornton LE, McNaughton SA, Crawford D. Variation in supermarket exposure to energy-dense snack foods by socio-economic position. *Public Health Nutr*. 2013;16:1178–1185. doi: 10.1017/S1368980012002649.
267. *Diet, Nutrition and the Prevention of Chronic Diseases*. Geneva, Switzerland: World Health Organization; 2003. WHO Technical Report Series.
268. Danaei G, Rimm EB, Oza S, Kulkarni SC, Murray CJ, Ezzati M. The promise of prevention: the effects of four preventable risk factors on national life expectancy and life expectancy disparities by race and county in the United States [published correction appears in PLoS Med. 2011;8]. *PLoS Med*. 2010;7:e1000248. doi: 10.1371/journal.pmed.1000248.
269. Murray CJ, Kulkarni S, Ezzati M. Eight Americas: new perspectives on U.S. health disparities. *Am J Prev Med*. 2005;29(suppl 1):4–10. doi: 10.1016/j.amepre.2005.07.031.
270. Daviglus ML, Talavera GA, Avilés-Santa ML, Allison M, Cai J, Criqui MH, Gellman M, Giachello AL, Gouskova N, Kaplan RC, LaVange L, Penedo F, Perreira K, Pirzada A, Schneiderman N, Wassertheil-Smoller S, Sorlie PD, Stamler J. Prevalence of major cardiovascular risk factors and cardiovascular diseases among Hispanic/Latino individuals of diverse backgrounds in the United States. *JAMA*. 2012;308:1775–1784. doi: 10.1001/jama.2012.14517.
271. Lynch JW, Kaplan GA, Cohen RD, Tuomilehto J, Salonen JT. Do cardiovascular risk factors explain the relation between socioeconomic status, risk of all-cause mortality, cardiovascular mortality, and acute myocardial infarction? *Am J Epidemiol*. 1996;144:934–942.
272. Safford MM, Brown TM, Muntner PM, Durant RW, Glasser S, Halanych JH, Shikany JM, Prineas RJ, Samdarshi T, Bittner VA, Lewis CE, Gamboa C, Cushman M, Howard V, Howard G; REGARDS Investigators. Association of race and sex with risk of incident acute coronary heart disease events. *JAMA*. 2012;308:1768–1774. doi: 10.1001/jama.2012.14306.
273. Teo K, Chow CK, Vaz M, Rangarajan S, Yusuf S; PURE Investigators-Writing Group. The Prospective Urban Rural Epidemiology (PURE) study: examining the impact of societal influences on chronic noncommunicable diseases in low-, middle-, and high-income countries. *Am Heart J*. 2009;158:1–7.e1. doi: 10.1016/j.ahj.2009.04.019.
274. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L; INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*. 2004;364:937–952. doi: 10.1016/S0140-6736(04)17018-9.
275. Bird CE, Seeman T, Escarce JJ, Basurto-Dávila R, Finch BK, Dubowitz T, Heron M, Hale L, Merkin SS, Weden M, Lurie N. Neighbourhood socioeconomic status and biological "wear and tear" in a nationally representative sample of US adults. *J Epidemiol Community Health*. 2010;64:860–865. doi: 10.1136/jech.2008.084814.
276. Chyu L, Upchurch DM. Racial and ethnic patterns of allostatic load among adult women in the United States: findings from the National Health and Nutrition Examination Survey 1999-2004. *J Womens Health (Larchmt)*. 2011;20:575–583. doi: 10.1089/jwh.2010.2170.
277. Duru OK, Harawa NT, Kermah D, Norris KC. Allostatic load burden and racial disparities in mortality. *J Natl Med Assoc*. 2012;104:89–95.
278. Gruenewald TL, Karlamangla AS, Hu P, Stein-Merkin S, Crandall C, Koretz B, Seeman TE. History of socioeconomic disadvantage and allostatic load in later life. *Soc Sci Med*. 2012;74:75–83. doi: 10.1016/j.socscimed.2011.09.037.
279. McEwen BS, Gianaros PJ. Central role of the brain in stress and adaptation: links to socioeconomic status, health, and disease. *Ann NY Acad Sci*. 2010;1186:190–222. doi: 10.1111/j.1749-6632.2009.05331.x.

280. Steptoe A, Feldman PJ, Kunz S, Owen N, Willemsen G, Marmot M. Stress responsivity and socioeconomic status: a mechanism for increased cardiovascular disease risk? *Eur Heart J*. 2002;23:1757–1763.
281. Steptoe A, Wardle J, Marmot M. Positive affect and health-related neuroendocrine, cardiovascular, and inflammatory processes. *Proc Natl Acad Sci USA*. 2005;102:6508–6512. doi: 10.1073/pnas.0409174102.
282. Steptoe A, Kunz-Ebrecht S, Owen N, Feldman PJ, Willemsen G, Kirschbaum C, Marmot M. Socioeconomic status and stress-related biological responses over the working day. *Psychosom Med*. 2003;65:461–470.
283. Loucks EB, Pilote L, Lynch JW, Richard H, Almeida ND, Benjamin EJ, Murabito JM. Life course socioeconomic position is associated with inflammatory markers: the Framingham Offspring Study. *Soc Sci Med*. 2010;71:187–195. doi: 10.1016/j.socscimed.2010.03.012.
284. Khang YH, Lynch JW, Jung-Choi K, Cho HJ. Explaining age-specific inequalities in mortality from all causes, cardiovascular disease and ischaemic heart disease among South Korean male public servants: relative and absolute perspectives. *Heart*. 2008;94:75–82. doi: 10.1136/hrt.2007.117747.
285. Albert MA, Glynn RJ, Buring J, Ridker PM. Impact of traditional and novel risk factors on the relationship between socioeconomic status and incident cardiovascular events. *Circulation*. 2006;114:2619–2626. doi: 10.1161/CIRCULATIONAHA.106.660043.
286. Thomas KS, Nelesen RA, Ziegler MG, Bardwell WA, Dimsdale JE. Job strain, ethnicity, and sympathetic nervous system activity. *Hypertension*. 2004;44:891–896. doi: 10.1161/01.HYP.0000148499.54730.0d.
287. Marmot MG. Socio-economic factors in cardiovascular disease. *J Hypertens Suppl*. 1996;14:S201–S205.
288. Whisman MA. Loneliness and the metabolic syndrome in a population-based sample of middle-aged and older adults. *Health Psychol*. 2010;29:550–554. doi: 10.1037/a0020760.
289. Abraham NG, Brunner EJ, Eriksson JW, Robertson RP. Metabolic syndrome: psychosocial, neuroendocrine, and classical risk factors in type 2 diabetes. *Ann NY Acad Sci*. 2007;1113:256–275. doi: 10.1196/annals.1391.015.
290. Marmot M, Wilkinson R. *Social Determinants of Health*. Oxford, UK: Oxford University Press; 2006:80.
291. Crimmins EM, Kim JK, Alley DE, Karlamangla A, Seeman T. Hispanic paradox in biological risk profiles. *Am J Public Health*. 2007;97:1305–1310. doi: 10.2105/AJPH.2006.091892.
292. Kanjilal S, Gregg EW, Cheng YJ, Zhang P, Nelson DE, Mensah G, Beckles GL. Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US adults, 1971-2002. *Arch Intern Med*. 2006;166:2348–2355. doi: 10.1001/archinte.166.21.2348.
293. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med*. 1998;338:171–179. doi: 10.1056/NEJM199801153380307.
294. Brunner E. Stress mechanisms in coronary heart disease. In: Stansfeld SA, Marmo M, eds. *Stress and the Heart*. London, UK: BMJ Books; 2002:181–191.
295. Seeman T, Epel E, Gruenewald T, Karlamangla A, McEwen BS. Socio-economic differentials in peripheral biology: cumulative allostatic load. *Ann NY Acad Sci*. 2010;1186:223–239. doi: 10.1111/j.1749-6632.2009.05341.x.
296. Lemelin ET, Diez Roux AV, Franklin TG, Carnethon M, Lutsey PL, Ni H, O'Meara E, Shrager S. Life-course socioeconomic positions and subclinical atherosclerosis in the Multi-Ethnic Study of Atherosclerosis. *Soc Sci Med*. 2009;68:444–451. doi: 10.1016/j.socscimed.2008.10.038.
297. Emerging Risk Factors Collaboration, Kaptoge S, Di Angelantonio E, Pennells L, Wood AM, White IR, Gao P, Walker M, Thompson A, Sarwar N, Caslake M, Butterworth AS, Amouyel P, Assmann G, Bakker SJ, Barr EL, Barrett-Connor E, Benjamin EJ, Bjorkelund C, Brenner H, Brunner E, Clarke R, Cooper JA, Cremer P, Cushman M, Dagenais GR, D'Agostino RB Sr, Dankner R, Davey-Smith G, Deeg D, Dekker JM, Engstrom G, Folsom AR, Fowkes FG, Gallacher J, Gaziano JM, Giampaoli S, Gillum RF, Hofman A, Howard BV, Ingelsson E, Iso H, Jorgensen T, Kiechl S, Kitamura A, Kiyohara Y, Koenig W, Kromhout D, Kuller LH, Lawlor DA, Meade TW, Nissinen A, Nordestgaard BG, Onat A, Panagiotakos DB, Psaty BM, Rodriguez B, Rosengren A, Salomaa V, Kauhanen J, Salonen JT, Shaffer JA, Shea S, Ford I, Stehouwer CD, Strandberg TE, Tipping RW, Tosetto A, Wassertheil-Smoller S, Wennberg P, Westendorp RG, Whincup PH, Wilhelmsen L, Woodward M, Lowe GD, Wareham NJ, Khaw KT, Sattar N, Packard CJ, Gudnason V, Ridker PM, Pepys MB, Thompson SG, Danesh J. C-reactive protein, fibrinogen, and cardiovascular disease prediction. *N Engl J Med*. 2012;367:1310–1320. doi: 10.1056/NEJMoa1107477.
298. Deverts DJ, Cohen S, Kalra P, Matthews KA. The prospective association of socioeconomic status with C-reactive protein levels in the CARDIA study. *Brain Behav Immun*. 2012;26:1128–1135. doi: 10.1016/j.bbi.2012.07.017.
299. Brunner E, Davey Smith G, Marmot M, Canner R, Beksinska M, O'Brien J. Childhood social circumstances and psychosocial and behavioural factors as determinants of plasma fibrinogen. *Lancet*. 1996;347:1008–1013.
300. Matthews KA, Schwartz JE, Cohen S. Indices of socioeconomic position across the life course as predictors of coronary calcification in black and white men and women: Coronary Artery Risk Development in Young Adults study. *Soc Sci Med*. 2011;73:768–774. doi: 10.1016/j.socscimed.2011.06.017.
301. Yan LL, Liu K, Daviglus ML, Colangelo LA, Kiefe CI, Sidney S, Matthews KA, Greenland P. Education, 15-year risk factor progression, and coronary artery calcium in young adulthood and early middle age: the Coronary Artery Risk Development in Young Adults study. *JAMA*. 2006;295:1793–1800. doi: 10.1001/jama.295.15.1793.
302. Detrano R, Guerci AD, Carr JJ, Bild DE, Burke G, Folsom AR, Liu K, Shea S, Szklo M, Bluemke DA, O'Leary DH, Tracy R, Watson K, Wong ND, Kronmal RA. Coronary calcium as a predictor of coronary events in four racial or ethnic groups. *N Engl J Med*. 2008;358:1336–1345. doi: 10.1056/NEJMoa072100.
303. Weintraub WS, Diamond GA. Predicting cardiovascular events with coronary calcium scoring. *N Engl J Med*. 2008;358:1394–1396. doi: 10.1056/NEJMe0800676.
304. Polonsky TS, McClelland RL, Jorgensen NW, Bild DE, Burke GL, Guerci AD, Greenland P. Coronary artery calcium score and risk classification for coronary heart disease prediction. *JAMA*. 2010;303:1610–1616. doi: 10.1001/jama.2010.461.
305. Grayburn PA. Interpreting the coronary-artery calcium score. *N Engl J Med*. 2012;366:294–296. doi: 10.1056/NEJMp1110647.
306. Miller GE, Chen E, Fok AK, Walker H, Lim A, Nicholls EF, Cole S, Kobor MS. Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. *Proc Natl Acad Sci USA*. 2009;106:14716–14721. doi: 10.1073/pnas.0902971106.
307. McEwen BS. Brain on stress: how the social environment gets under the skin [published correction appears in *Proc Natl Acad USA*. 2013;110:1561]. *Proc Natl Acad Sci USA*. 2012;109(suppl 2):17180–17185. doi: 10.1073/pnas.1121254109.
308. Shonkoff JP, Boyce WT, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities: building a new framework for health promotion and disease prevention. *JAMA*. 2009;301:2252–2259. doi: 10.1001/jama.2009.754.
309. Brunner EJ, Marmot MG, Nanchahal K, Shipley MJ, Stansfeld SA, Juneja M, Alberti KG. Social inequality in coronary risk: central obesity and the metabolic syndrome: evidence from the Whitehall II study. *Diabetologia*. 1997;40:1341–1349. doi: 10.1007/s001250050830.
310. Das A. How does race get "under the skin"?: Inflammation, weathering, and metabolic problems in late life. *Soc Sci Med*. 2013;77:75–83. doi: 10.1016/j.socscimed.2012.11.007.
311. Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health*. 2006;96:826–833. doi: 10.2105/AJPH.2004.060749.
312. Heffernan KS, Jae SY, Wilund KR, Woods JA, Fernhall B. Racial differences in central blood pressure and vascular function in young men. *Am J Physiol Heart Circ Physiol*. 2008;295:H2380–H2387. doi: 10.1152/ajpheart.00902.2008.
313. Thurston RC, Matthews KA. Racial and socioeconomic disparities in arterial stiffness and intima media thickness among adolescents. *Soc Sci Med*. 2009;68:807–813. doi: 10.1016/j.socscimed.2008.12.029.
314. Barker DJ, Bull AR, Osmond C, Simmonds SJ. Fetal and placental size and risk of hypertension in adult life. *BMJ*. 1990;301:259–262.
315. Barker DJ. In utero programming of cardiovascular disease. *Theriogenology*. 2000;53:555–574.
316. Calkins K, Devaskar SU. Fetal origins of adult disease. *Curr Probl Pediatr Adolesc Health Care*. 2011;41:158–176. doi: 10.1016/j.cppeds.2011.01.001.
317. Ingelfinger JR, Nuyt AM. Impact of fetal programming, birth weight, and infant feeding on later hypertension. *J Clin Hypertens (Greenwich)*. 2012;14:365–371. doi: 10.1111/j.1751-7176.2012.00660.x.
318. Leon DA, Lithell HO, Vâgerö D, Koupilová I, Mohsen R, Berglund L, Lithell UB, McKeigue PM. Reduced fetal growth rate and increased risk

of death from ischaemic heart disease: cohort study of 15 000 Swedish men and women born 1915-29. *BMJ*. 1998;317:241–245.
319. Barker DJ, Martyn CN. The maternal and fetal origins of cardiovascular disease. *J Epidemiol Community Health*. 1992;46:8–11.
320. Law CM, de Swiet M, Osmond C, Fayers PM, Barker DJ, Cruddas AM, Fall CH. Initiation of hypertension in utero and its amplification throughout life. *BMJ*. 1993;306:24–27.
321. Bhargava SK, Sachdev HS, Fall CH, Osmond C, Lakshmy R, Barker DJ, Biswas SK, Ramji S, Prabhakaran D, Reddy KS. Relation of serial changes in childhood body-mass index to impaired glucose tolerance in young adulthood. *N Engl J Med*. 2004;350:865–875. doi: 10.1056/NEJMoa035698.
322. Law CM, Shiell AW, Newsome CA, Syddall HE, Shinebourne EA, Fayers PM, Martyn CN, de Swiet M. Fetal, infant, and childhood growth and adult blood pressure: a longitudinal study from birth to 22 years of age. *Circulation*. 2002;105:1088–1092.
323. Brenner BM, Garcia DL, Anderson S. Glomeruli and blood pressure: less of one, more the other? *Am J Hypertens*. 1988;1(pt 1):335–347.
324. Rich-Edwards JW, Colditz GA, Stampfer MJ, Willett WC, Gillman MW, Hennekens CH, Speizer FE, Manson JE. Birthweight and the risk for type 2 diabetes mellitus in adult women. *Ann Intern Med*. 1999;130(pt 1):278–284.
325. Forsén T, Eriksson J, Tuomilehto J, Reunanen A, Osmond C, Barker D. The fetal and childhood growth of persons who develop type 2 diabetes. *Ann Intern Med*. 2000;133:176–182.
326. Crume TL, Scherzinger A, Stamm E, McDuffie R, Bischoff KJ, Hamman RF, Dabelea D. The long-term impact of intrauterine growth restriction in a diverse U.S. cohort of children: the EPOCH study. *Obesity (Silver Spring)*. 2014;22:608–615. doi: 10.1002/oby.20565.
327. Leeson CP, Kattenhorn M, Morley R, Lucas A, Deanfield JE. Impact of low birth weight and cardiovascular risk factors on endothelial function in early adult life. *Circulation*. 2001;103:1264–1268.
328. Leeson CP, Whincup PH, Cook DG, Donald AE, Papacosta O, Lucas A, Deanfield JE. Flow-mediated dilation in 9- to 11-year-old children: the influence of intrauterine and childhood factors. *Circulation*. 1997;96:2233–2238.
329. Rose G. Sick individuals and sick populations. *Int J Epidemiol*. 1985;14:32–38.
330. Pearson TA, Palaniappan LP, Artinian NT, Carnethon MR, Criqui MH, Daniels SR, Fonarow GC, Fortmann SP, Franklin BA, Galloway JM, Goff DC Jr, Heath GW, Frank AT, Kris-Etherton PM, Labarthe DR, Murabito JM, Sacco RL, Sasson C, Turner MB; on behalf of the American Heart Association Council on Epidemiology and Prevention. American Heart Association guide for improving cardiovascular health at the community level, 2013 update: a scientific statement for public health practitioners, healthcare providers, and health policy makers. *Circulation*. 2013;127:1730–1753. doi: 10.1161/CIR.0b013e31828f8a94.
331. Frieden TR. A framework for public health action: the health impact pyramid. *Am J Public Health*. 2010;100:590–595. doi: 10.2105/AJPH.2009.185652.
332. Campbell F, Conti G, Heckman JJ, Moon SH, Pinto R, Pungello E, Pan Y. Early childhood investments substantially boost adult health. *Science*. 2014;343:1478–1485. doi: 10.1126/science.1248429.
333. Smedley BD, Syme SL; Committee on Capitalizing on Social Science and Behavioral Research to Improve the Public's Health. Promoting health: intervention strategies from social and behavioral research. *Am J Health Promot*. 2001;15:149–166.
334. Wallerstein N, Duran B. Community-based participatory research contributions to intervention research: the intersection of science and practice to improve health equity. *Am J Public Health*. 2010;100(suppl 1):S40–S46. doi: 10.2105/AJPH.2009.184036.
335. Giles WH, Tucker P, Brown L, Crocker C, Jack N, Latimer A, Liao Y, Lockhart T, McNary S, Sells M, Harris VB. Racial and Ethnic Approaches to Community Health (REACH 2010): an overview. *Ethn Dis*. 2004;14(suppl 1):S5–S8.
336. Plescia M, Groblewski M, Chavis L. A lay health advisor program to promote community capacity and change among change agents. *Health Promot Pract*. 2008;9:434–439. doi: 10.1177/1524839906289670.
337. Plescia M, Herrick H, Chavis L. Improving health behaviors in an African American community: the Charlotte Racial and Ethnic Approaches to Community Health project. *Am J Public Health*. 2008;98:1678–1684. doi: 10.2105/AJPH.2007.125062.
338. Proia KK, Thota AB, Njie GJ, Finnie RKC, Hopkins DP, Mukhtar Q, Pronk NP, Zeigler D, Kottke TE, Rask KJ, Lackland DT, Brooks JF, Braun LT, Cooksey T. Community Preventive Services Task Force. Team-based care and improved blood pressure control: a community guide systematic review. *Am J Preventive Med*. 2004;47:86–99.
339. TenBrink DS, McMunn R, Panken S. Project U-Turn: increasing active transportation in Jackson, Michigan. *Am J Prev Med*. 2009;37(suppl 2):S329–S335. doi: 10.1016/j.amepre.2009.09.004.
340. Kurian AK, Cardarelli KM. Racial and ethnic differences in cardiovascular disease risk factors: a systematic review. *Ethn Dis*. 2007;17:143–152.
341. Mulder BC, de Bruin M, Schreurs H, van Ameijden EJ, van Woerkum CM. Stressors and resources mediate the association of socioeconomic position with health behaviours. *BMC Public Health*. 2011;11:798. doi: 10.1186/1471-2458-11-798.

KEY WORDS: AHA Scientific Statements ■ cardiovascular diseases ■ healthcare disparities ■ health services accessibility ■ prejudice ■ psychosocial deprivation ■ social determinants of health ■ socioeconomic factors

Social Determinants of Risk and Outcomes for Cardiovascular Disease: A Scientific Statement From the American Heart Association

Edward P. Havranek, Mahasin S. Mujahid, Donald A. Barr, Irene V. Blair, Meryl S. Cohen, Salvador Cruz-Flores, George Davey-Smith, Cheryl R. Dennison-Himmelfarb, Michael S. Lauer, Debra W. Lockwood, Milagros Rosal and Clyde W. Yancy

Circulation. published online August 3, 2015;
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539