AHA Scientific Statement

Social Determinants of Risk and Outcomes for Cardiovascular Disease

A Scientific Statement From the American Heart Association

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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;

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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.

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*Modest.

†Significant

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*Significant.

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