A strong association between socioeconomic status (SES) and cardiovascular disease (CVD) has been established in both cross-sectional and prospective studies. There are suggestions of gender and racial/ethnic variation in SES differences in CVD, and strong evidence exists that while lower-SES groups have been characterized by higher morbidity and mortality from all causes in most populations for at least a century, the currently observed excess of CVD morbidity and mortality at lower SES levels is a relatively new phenomenon. In the early 20th century, CVD morbidity and mortality in the economically most developed nations were more prevalent at higher SES, but in the past 30–50 years, low-SES groups in these countries have borne the greater CVD burden. This secular trend is important for understanding the pathways through which SES leads to CVD.

Significant SES differences in access to preventive and therapeutic medical care for CVD and other diseases clearly continue to exist in the United States—and perhaps even, to a lesser degree, in countries with programs of national health insurance or health services. However, a variety of evidence indicates that access to medical care explains only a small portion (perhaps less than 10 percent) of the currently observable SES differences in CVD, just as changes in medical care have accounted for only a minor part of the dramatic declines in CVD morbidity and mortality of the past 25 years (1,2). Examples include the changing SES gradient in CVD and the relative imperviousness of the current SES gradient in CVD to the introduction of national health care (e.g., in the United Kingdom) and insurance (e.g., in Canada).

Increasing evidence shows that all or most of the major physiological variables (e.g., diabetes, blood pressure, blood lipids, blood glucose) and behavioral or lifestyle factors (e.g., smoking, alcohol consumption, diet, body mass, and physical activity) for CVD are differentially distributed by SES. Low-SES groups tend to have higher levels of each of these risk factors, especially the psychosocial ones. These variables appear to play a larger role than medical care in explaining both recent declines in CVD and SES differences in CVD, but controlling for them still accounts for only a moderate portion (about 25 percent) of observed SES differences in CVD (3).

Thus, access to medical care and almost all of the conventional CVD risk factors relate to SES in a way that is consistent with the observed excess of CVD in lower SES strata. However, multivariate analysis indicates that they may in combination account for or explain only about one-third of the observed SES differences in CVD morbidity and mortality. A growing body of research over the past 25 years suggests that a range of other psychosocial variables may explain the remaining two-thirds of the relationship between SES and CVD. In order to determine the contribution of these variables to the SES–CVD relationship, it is important to consider what evidence is necessary, how much evidence we now have, what future research is needed, and the implications of these considerations for research and public health policy.
Psychosocial Variables as Pathways Between SES and CVD

To establish that any psychosocial variable is a pathway explaining the impact of SES on CVD requires four kinds of evidence:

- Evidence, especially from prospective studies, that the variable is a risk factor for CVD morbidity and mortality;
- Evidence that the variable varies by SES in a manner that may account for or explain the relationship of SES to CVD (i.e., higher levels of risk should characterize lower-SES groups);
- Evidence that controlling for the variable reduces the relationship between SES and CVD;
- Logical or empirical evidence that the variable is caused or determined by SES, not by some third factor that produces a spurious association between the variable and CVD. The psychosocial variable, in turn, must cause or determine CVD through plausible biobehavioral mechanisms.

Psychosocial Factors as Potential Pathways Between SES and CVD

A variety of psychosocial variables have been proposed as potential additional pathways between SES and CVD. For all those reviewed here, evidence exists that they relate to SES in a manner that might account for the SES–CVD relationship. However, the extent to which each has been already established as a risk factor for CVD varies and the extent to which they have been clearly shown to account for some portion of the SES–CVD relationship varies even more. In most cases, plausible confounding factors that might produce a spurious association between the psychosocial variable and either CVD or SES have been appropriately controlled, and the necessary causal ordering and mechanisms among the variables are plausible. However, as in any nonexperimental research, the search for variables that produce spurious relationships is never complete, and must be evaluated largely in terms of how plausible the existence of currently unmeasured, unevaluated, or unknown confounding factors is.

Evidence for CVD Risk Factor Status

As in the case for more conventional risk factors, the key evidence in establishing these psychosocial variables as CVD risk factors comes from prospective studies. Such studies are either ongoing community and national studies that measured indicators of psychosocial risk factors even before they were identified as putative risk factors, or newly initiated studies that tend to be more limited in scope or duration.

Type A behavior and anger/hostility. Type A behavior was the first major psychosocial variable to be established as a risk factor for CVD (4). Subsequent research, however, suggests that the key aspect of Type A responsible for its impact on CVD is a chronic disposition toward anger or hostility, and this disposition has been quite compellingly established as a risk factor for CVD (5).

Lack of social relationships and supports. In the past 20 years, the relative lack of social relationships or supports in circumstances such as social isolation has consistently been found to constitute a significant risk factor for all-cause mortality (6). In all studies where CVD deaths could be isolated, social
relationships were as predictive of CVD as they were of other major causes of death and all-cause mortality.

**Lack of personal efficacy/control.** A variety of evidence suggests that the lack of a sense of personal efficacy/control is associated with poor general and cardiovascular health and is predictive of all-cause and CVD mortality (7). However, broad community or national prospective studies such as those available for Type A/anger/hostility and social supports are scarce for this variable and constitute a major research need.

**Depression/negative affect.** Findings from the past 10 years increasingly suggest that depression or negative affect is a risk factor for CVD and probably for other diseases as well. This prospective research is probably at least as abundant as that for personal efficacy/control, but has largely been limited to very selective samples, often of patients with coronary heart disease (5). Again, broader community or national prospective studies are needed to study the impact of depression and negative affect on CVD and other diseases. Initial results in this regard support the risk factor status of depression (8).

**Acute and chronic stress.** The study of "life events" was a major impetus for research on the relationships of stress and psychosocial factors to health. Early studies linked the occurrence of life events to increased risk of physical and mental disorders, albeit often relatively mild or transient disorders. Later work showed that major negative events are the ones responsible for these associations, and also demonstrated that such events can be predictive of CVD and other forms of morbidity and mortality (9). Again, evidence from broader community and national prospective studies is still lacking and should be a major direction for future research.

Conceptual and empirical analysis clearly suggest that chronic stress or deprivation caused by lack of income or chronic problems in work, marriage, or parenting may be at least as important a determinant of health as acute or life event stressors. Recent developments in stress measurement highlight the need to broaden the assessment of stress beyond life events and chronic stressors. This research indicates that, independent of the effects of recent life events and chronic stressors, other sources of stress such as childhood traumas, earlier adult life events, daily hassles, and even nonevents (desired events that fail to occur) can affect physical and mental health. Moreover, this comprehensive measurement of stress accounts for substantially more variability in health status than previous studies of stress have suggested (10). This full range of stress variables needs to be incorporated into research on the relationship between SES and CVD.

Finally, there is new interest in characterizing the stressfulness or deprivation of areas as well as individuals and evaluating their impact on all forms of health. This is another direction for future CVD research (8).

**Evidence for SES Determination of Psychosocial Risk**

Although the evidence linking putative psychosocial risk factors to CVD and other forms of morbidity and mortality is somewhat variable, relationships of these psychosocial risk factors to SES are remarkably consistent. As with more conventional CVD risk factors, it is almost always the case that lower-SES groups are exposed to more psychosocial risk. This holds for education and other SES indicators that are clearly antecedent to the psychosocial risk factors, and for income and other SES variables that are more proximal to the psychosocial risk variables, yet probably also antecedent to them.
Figure 1.—Psychosocial Risk Factor Status in U.S. Residents, Ages 45 to 64, by Education and Income. Hostility data are taken from the 1984-86 MPI-II Restandardization Study. Data for other variables come from the 1986 Americans' Changing Lives Study, a national probability sample survey conducted by the University of Michigan.

Figure 1 shows that indicators of each of the major psychosocial variables just discussed vary almost exactly as expected by both education and income among the U.S. population, 45 to 64 years of age. Results tend to be similar for other age groups, but weaker for some variables at older ages (11).

The prevalence of each of the putative psychosocial risk factors in Figure 1 is always greatest among those with the lowest level of education or income and almost always least among those with the highest level of education and income. The rate ratios of the lowest education and income level to the highest levels range from a low of 1.1 to a high of 3.8, and average around 2.0. Thus, as with the more conventional biomedical and lifestyle/behavioral risk factors, the putative psychosocial risk factors tend to be more prevalent in lower-SES groups.

Evidence That Psychosocial Risk Factors Help Explain SES-CVD Relationship

Too few studies, especially prospective ones, have evaluated the ability of these various psychosocial risk factors, taken singly or together, to explain the relationship of SES to CVD or other health outcomes. Several studies, however, find that controlling for a broad range of these variables along with physical environmental exposures and more conventional risk factors can explain a substantial portion of the relation of SES to CVD or other health outcomes (11,12). These results should be considered quite preliminary, though promising and consistent with Redford Williams' model for explaining the relationship of SES to CVD (13). More prospective studies of large community populations are necessary to measure the individual and combined contributions of a broad array of psychosocial, physiological, and behavioral risk factors to the relationships between SES and the incidence and course of CVD.

Spurious Associations

It is plausible that SES shapes or determines exposure to almost all risk factors for CVD. This is especially true for education, which in most adult populations is completed well before the assessment of risk factors. Greater potential for reciprocal causality exists between more contemporaneous measures of SES such as income and adult health or risk factor measures, but a variety of evidence suggests that causality generally flows from SES to risk factors to morbidity and mortality (3).
Perhaps the main challenge to this model of causal pathways lies in the argument that genetic factors, environment, or their interactions early in life determine the later life trajectory of both socioeconomic attainment and health. Only long-term longitudinal studies can resolve these concerns. At this point it appears that although genes and environmental conditions in early life can and do affect SES and health in later life, these effects tend to be smaller and diminishing over the life course compared to more proximal or contemporaneous SES, psychosocial, or biomedical influences. Moreover, there is relatively little evidence to suggest that such genetic or early life variables can account for the observed relationships of SES to psychosocial risk factors and CVD or other health outcomes in middle or later life (3,12). The need remains, however, for more and better research on these issues, especially on gene–environment interactions over the life course and the ways in which early life deprivations or trauma may affect SES, risk factors, and CVD in later life.

Implications

The psychosocial risk factors discussed here, and others that remain to be identified, are critical to understanding the pathways linking SES to CVD and other health outcomes. For all of these psychosocial variables, plausible biobehavioral mechanisms have been posited to explain their effects on CVD or other health outcomes (3,5,6). However, it is important to recognize that with or without full understanding of these biological pathways, SES and associated psychosocial risk factors remain important determinants of CVD and other health outcomes, and hence are important potential avenues for efforts at CVD prevention and control. For example, we do not yet fully understand the biological mechanisms by which cigarette smoking produces CVD morbidity and mortality, but we have been able to reduce smoking and hence have a significant preventive effect on CVD and other health problems.

Most important, SES is a kind of "master status" that influences exposure to almost all major physiological, behavioral, and psychosocial risk factors for CVD. Thus, we need to examine the role of SES as a "fundamental cause" of CVD (14). As such, changes in the SES of individuals or larger subgroups of the population could result in the simultaneous reduction of a broad array of risk factors and hence diseases, as general improvements in nutrition and public sanitation have done. Addressing these broad and fundamental or "upstream" determinants of health has the likely advantage of affecting not only current or known risk factors but unknown or future ones as well. Thus, for example, as CVD has become a more important cause of morbidity and mortality and its risk factors better understood, persons of higher SES have been increasingly able and likely to reduce their exposure to these risk factors and hence to CVD.

The SES of individuals and groups can and does change in response to unplanned or natural SES cycles or events and as a result of social policy. The greater success of many other industrialized countries, relative to the United States, in reducing SES deprivation and inequality has been posited as a major reason that population health and life expectancy in those countries has come over the last several decades to improve more rapidly and reach higher absolute levels than in this country, despite the fact that none of the other countries approach the United States in either spending for or advances in medical care and biomedical research (15). Thus, SES policy may increasingly be a major instrument of health policy. Both researchers and public health policy makers must pay increasing attention to understanding the pathways by which SES, or changes in SES at the level of both individuals and populations, affects CVD. This knowledge will serve as the basis of preventive efforts to reduce not only risk factor pathways but also SES deprivation and inequality.
REFERENCES


