
Socioeconomic Status and Health

The Challenge of the Gradient

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Socioeconomic status (SES) is consistently associated with health outcomes, yet little is known about the psychosocial and behavioral mechanisms that might explain this association. Researchers usually control for SES rather than examine it. When it is studied, only effects of lower, poverty-level SES are generally examined. However, there is evidence of a graded association with health at all levels of SES, an observation that requires new thought about domains through which SES may exert its health effects. Variables are highlighted that show a graded relationship with both SES and health to provide examples of possible pathways between SES and health end points. Examples are also given of new analytic approaches that can better illuminate the complexities of the SES-health gradient.

Throughout history, socioeconomic status (SES) has been linked to health. Individuals higher in the social hierarchy typically enjoy better health than do those below; SES differences are found for rates of mortality and morbidity from almost every disease and condition (Antonovsky, 1967; Illsley & Baker, 1991). Despite recognition for decades of this fundamental association, the reasons for its existence remain largely obscure. Because SES is such a powerful risk factor, a search for other etiologic factors in disease end points is often regarded as suspect unless the influence of SES is controlled. As a result, SES has been almost universally relegated to the status of a control variable and has not been systematically studied as an important etiologic factor in its own right. As Marmot, Kogevinas, and Elston (1987) noted, it is generally included "with as much regularity but with as little thought as . . . gender" (p. 111).

Socioeconomic status is "a composite measure that typically incorporates economic status, measured by income; social status, measured by education; and work status, measured by occupation" (Dutton & Levine, 1989, p. 30). The three indicators are interrelated but not fully overlapping variables. Often researchers use one or another of the indicators as the measure of SES. The fact that associations between SES and health are found with each of the indicators suggests that a broader underlying dimension of social stratification or social ordering is the potent factor. In this article we consider SES effects broadly and examine studies using a variety of specific indicators.

Of those studies that have examined the health effects of SES, most have compared the health of individuals at the very bottom of the SES hierarchy either with those above the poverty level or with those at the top of the hierarchy (for reviews, see Antonovsky, 1967; Haan, Kaplan, & Syme, 1989). The effects of severe poverty on health may seem obvious through the impact of poor nutrition, crowded and unsanitary living conditions, and inadequate medical care. As important as these variables are, such an analysis underestimates the potent and pervasive effects of SES on biological outcomes. There is evidence that the association of SES and health occurs at every level of the SES hierarchy, not simply below the threshold of poverty. Not only do those in poverty have poorer health than those in more favored circumstances, but those at the highest level enjoy better health than do those just below (Adelstein, 1980; Kraus, Borhani, & Franti, 1980; Marmot et al., 1991; Marmot, Shipley, & Rose, 1984). This poses a challenge to understand the mechanisms by which SES affects health because factors associated with low SES are not likely to account for differences in health status at upper levels. Identifying factors that can account for the link to health all across the SES hierarchy may shed light on new mechanisms that have heretofore been ignored because of a focus on the more readily apparent correlates of poverty.

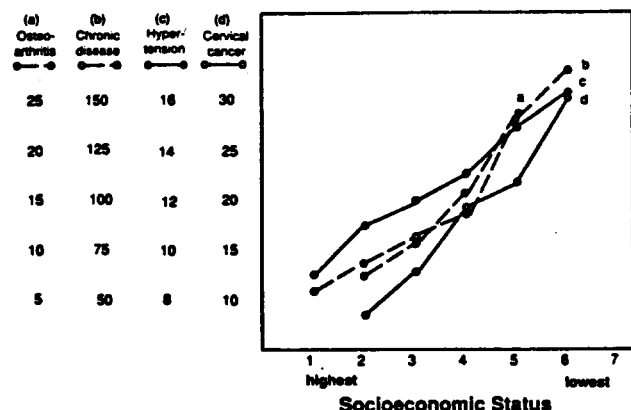
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Figure 2
Morbidity Rate by Socioeconomic Status Level



Note. (a) Percent diagnosed osteoarthritis (Cunningham & Kelsey, 1984). (b) Relative prevalence of chronic disease (Townsend, 1974). (c) Prevalence of hypertension (Kraus, Borhani, & Franti, 1980). (d) Rate of cervical cancer per 100,000 (Devesa & Diamond, 1983).

and Hauser (1973), collected in 1960. In the 26 years between the two studies, death rates declined, but the decreases were greater in more versus less educated groups. The resulting SES health gradient was, thus, steeper in 1986 than it had been in 1960.

Socioeconomic status is also linked to prevalence and course of disease. Pincus, Callahan, and Burkhauser (1987) examined reports of health problems for individuals at four levels of educational attainment in a national sample and tested for a linear trend across educational levels. The frequency of 32 of 37 conditions assessed was greater the lower the educational level. The individual conditions were grouped into eight disease categories, and differences by education were analyzed separately in each of three age groups: 18–44, 45–54, and 55–64 years. There was a significant linear trend for almost all of the diseases in all three groups. The only disease category that was unrelated to education in all age groups was neoplastic disease.³ Among a group of patients with rheumatoid arthritis, Pincus and Callahan (1985) found that the lower a patient's educational level, the greater was the chance of subsequent mortality or major decline in functional capacity over a 9-year period, even when controls were entered for age, sex, smoking, functional status at baseline, treatments indicative of more severe disease, or duration of disease.

Possible Mechanisms

Having reviewed the substantial evidence for a graded association between socioeconomic position and health, we next examine three possible explanations for the basis of the association. First, the empirical link between SES and health might represent a spurious association, arising from the relationships of both SES and health outcomes to underlying, genetically based factors. For example,

physical size or intellectual capacity might lead concurrently to lower social position and poorer health. This explanation is plausible but improbable. As noted in both the Whitehall I and Whitehall II studies (Marmot et al., 1984; Marmot et al., 1991), although job status is inversely related to physical height, the association between job status and health persists even after adjustments for height and body mass index. As noted by Kohn and Schooler (1978), intelligence and cognitive flexibility are important correlates of job status; but it is less clear, beyond the known relationship of mental retardation to greater disease risk, that intelligence in a normative population is reliably linked to health. Indeed, there is evidence that health behaviors such as compliance with medical advice are unrelated to intelligence or education (Becker, Drachman, & Kirscht, 1974; Stimson, 1974). A biologically driven predisposition to both lower SES and poorer health status appears unlikely, given the evidence at hand, to offer a sound explanation for the SES–health association. Furthermore, if genetic predispositions that we have not accounted for are involved in the SES–health link, they are very likely, as are most complex genetic influences, to become important only when environmental and behavioral factors impinge on them.

A second possible explanation for the SES–health gradient, known as the *drift hypothesis*, suggests that the association reflects the influence of illness on SES, rather than of SES on illness. There is evidence, for example, that individuals with schizophrenia follow a trajectory of descending socioeconomic resources as the natural history of their disease unfolds (Goldberg & Morrison, 1963). Nonetheless, two thorough recent reviews have concluded that, although some downward drift in social position accompanies poorer health status, the phenomenon is unlikely to play an important role in accounting for the SES–health relationship (Haan et al., 1989; Wilkinson, 1986). Deteriorating health status among older adults, which has been linked to educational levels, cannot logically affect past education (Haan et al., 1989). Furthermore, if illness principally influenced SES, then no association would be expected for family members when SES is determined by income or occupation of the head of the household, or for retired individuals for whom income is no longer dependent on health. However, such SES–health associations are generally as strong as those found for working heads of households.

Finally, the third explanation for the association is that SES affects biological functions that, in turn, influence health status. Surprisingly, we know little about how SES operates to influence biological functions that determine health status. Part of the problem may be the way in which SES is conceptualized and analyzed. It is usually treated as a main effect, operating independently

³ It is interesting to note that a recent review of the contribution of psychosocial factors to disease etiology (Adler & Matthews, 1993) concluded that the evidence for the role of such factors in the etiology of cancer was much weaker than for other diseases, particularly cardiovascular disease.

with lower risk for coronary heart disease, the leading cause of death for both men and women in the United States. In this context, the interpretation of alcohol intake as a risk factor is unclear.

Psychological Characteristics

There has been increasing evidence that psychological characteristics of the individual contribute to risk of morbidity and mortality. Of these variables, depression and hostility have shown the most consistent relationship with both SES and physical health outcomes.

Depression. Depression has been studied both as a pathological state of major depression and in terms of general depressive symptoms. Socioeconomic status is inversely related to both major depression and depressive symptoms. In a Canadian community sample, the prevalence of major depression was 1.9%, 4.5%, and 12.4% in high, average, and low SES groups, respectively. Over 16 years, the inverse gradient repeated itself in annual incidence of new depression (Murphy et al., 1991). Kaplan, Roberts, Camacho, and Coyne (1987) found higher rates of new reports of depressive symptoms over a nine-year period among those lower in income and education.

Depression is linked to health outcomes, particularly coronary heart disease. Within a sample of patients with coronary artery disease, twice as many of those with a major depressive disorder experienced at least one major cardiac event (e.g., myocardial infarction (MI), bypass surgery) in the subsequent year compared with nondepressed patients (77.8% vs. 34.9%; $p < .02$; Carney et al., 1988). In a meta-analysis of 15 studies of psychological predictors of coronary heart disease, depression was found to have a combined effect size of .21 ($p < .001$); the strongest association was with MI (combined effect size of .26, $p < .001$; Booth-Kewley & Friedman, 1987).

Hostility. Hostility—a disposition reflecting anger-proneness; a cynical, distrusting view of others; and antagonistic behavior (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989)—also relates both to SES and to disease risk. For example, in a national sample in the United States, hostility was inversely related to five levels of education ($p < .001$), occupational status ($p < .001$), and income ($p < .003$; Barefoot et al., 1991). Similarly, Scherwitz, Perkins, Chesney, and Hughes (1991) found greater hostility among less educated than among more educated adults in four urban areas ($p < .001$).

Several prospective studies have linked hostility to risk of coronary heart disease (CHD) and premature mortality. Dembroski, MacDougall, Costa, and Grandits (1989) found that among men under age 47, greater hostility measured on entry into the Multiple Risk Factor Intervention Trial (Multiple Risk Factor Intervention Group, 1976) conferred an adjusted relative risk of 2.1 for subsequent MI or coronary heart disease (CHD) or both ($p = .001$) controlling for cigarette smoking, diastolic blood pressure, and serum cholesterol. In a 25-year follow-up of a sample of medical students, Barefoot, Dahlstrom, and Williams (1983) found CHD incidence density to be .9 per 1000 person-years of follow-up for those with hos-

tility scores at or below the median versus 4.5 for those above the median. And in a 10-year follow-up of a male sample, Shekelle, Gale, Ostfeld, and Paul (1983) found the relative odds of an initial CHD event to be .68 for low versus high hostility groups after adjustment for age, systolic blood pressure, serum cholesterol, cigarette smoking, and alcohol intake ($p < .01$). In addition, cross-sectional studies have found associations between hostility and peripheral arterial disease (Joesoef, Wetterhal, DeStafano, Stroup, & Fronck, 1989), essential hypertension (reviewed in Diamond, 1982), and CHD (reviewed in Diamond, 1982; Barefoot et al., 1983).

Psychological Stress

Associations between SES and health may stem in part from differential exposure to and experience of greater stress. Stress has been characterized in two ways: (a) as exposure to life events that require adaptation, generally measured by a checklist of major events (e.g., divorce, death of a relative, job loss), or (b) as a state that occurs when persons perceive that demands exceed their abilities to cope, usually measured by self-reports of subjective experience. There is evidence for the role of both types of stress indicators in the SES-health link.

Life events presumably trigger perceptions of stress and negative emotion. These perceptions are known to alter neuroendocrine response and immune responses that may put persons at greater risk for a range of illnesses. Persons experiencing recent stressful life events have been found to be at greater risk for gastrointestinal disorders (Harris, 1991), menorrhagia and secondary amenorrhea (Harris, 1989), heart attacks (Theorell, 1974), and susceptibility to infectious agents (Cohen, Tyrrell, & Smith, 1991, 1993; Stone et al., 1992). Perceptions of stress and negative affect have been similarly linked to heart disease (Byrne & Whyte, 1980; Tofler et al., 1990), stroke (Harmsen, Rosengren, Tsipogianni, & Wilhelmsen, 1990), and susceptibility to infectious agents (Cohen et al., 1991, 1993).

Higher placement in the SES hierarchy can reduce stress and its somatic correlates in two ways. First, higher SES diminishes the likelihood that individuals will encounter negative events. In a community survey, lower income respondents were exposed to more stressful life events beyond their control than were higher income respondents (Dohrenwend & Dohrenwend, 1970). Similarly, Dohrenwend (1973) found that families whose head of the household had less than a high school education reported more stressful life events than did those headed by a high school graduate or better. This relation held both for events whose occurrences were within respondent control and for those outside of their control. McLeod and Kessler (1990) found small but consistent associations between SES and exposure to negative life events. A second way in which higher SES placement can reduce stress results because as individuals descend the SES hierarchy, they may have fewer social and psychological resources to cope with stressful life events and thus will be more susceptible to the subjective experience of stress. Those

ronal development in the preoptic area. These results suggest that the timing of central nervous system (CNS) maturational events are under social control and that dominance status within a given social context can exert profound influences on neurobiologic function.

Taken together, the studies on social order suggest the following general and preliminary observations regarding possible health effects of social dominance status *per se*: First, responses to hierarchical position may be encoded into the behavioral repertoire of individual organisms to protect the survival of the group and may be expressed at times even at the expense of individual well-being. Second, hierarchical position may have direct effects on physiological processes and neuroanatomic structures, which may in turn influence an individual's biologic vulnerability to agents of disease. Finally, the health effects of dominance status may be largely dependent on characteristics—particularly stability—of the larger social context in which position is assigned.

Issues of Methodology and Analysis

Research on SES and health has been limited by several conceptual and methodological constraints. First, as noted earlier, the vast majority of studies of SES and health have failed to examine the whole range of the SES hierarchy. Differentiations at upper as well as lower levels need to be examined.

Second, SES is typically measured by a single variable, such as income or education. Although various components of SES are intercorrelated, they are not identical. Socioeconomic status may function most powerfully in terms of combinations of variables. In studying psychiatric disorders, Rutter (1985) found that no single adverse condition affected risk but that "psychiatric risk went up sharply when several adversities co-existed" (p. 601). In many studies, moreover, race is used as a proxy for SES. Yet there is evidence that SES may operate differently within racial groups and may interact with race to affect health. For example, the association of race and health appears to be particularly strong among low SES Blacks, for whom the burden of discrimination may be more powerful (Klag, Whelton, Coresh, Grim, & Kuller, 1991).

Third, SES indicators have generally been measured at only one level. For example, income has generally been assessed either at the individual level (e.g., family income) or the aggregate level (e.g., mean income within a census tract). We know little about how these levels may function together to affect health outcomes. It may be that the health implications of low income are quite different for individuals living in relatively more affluent areas than in those residing in poorer areas. For example, Haan, Kaplan, and Camacho (1987) found that residing in a neighborhood that was federally designated as a poverty area (characterized by a high proportion of low-income families, substandard housing, many unskilled male laborers, etc.) was a risk factor for subsequent mortality above and beyond the characteristics of the individual. Using data from the Alameda County study, they found

that residing in a poverty area predicted nine-year mortality rates even controlling for the individual's own socioeconomic characteristics (e.g., income or education). Similarly, neighborhood residence continued to predict subsequent mortality when controls were entered for access to health care, for health behaviors, or for social isolation. Similarly, Krieger (1992) has shown that "contextual analyses" in which neighborhood (block group, a subdivision of a census tract, encompassing about 1,000 individuals) and census tract information is used in addition to individual data provides a better understanding of health behaviors and outcomes.

Fourth, almost all studies have used either simple correlation or regression analysis to examine the main effects of SES on a health outcome. Regression analysis is severely limited in its ability to disentangle the SES-health gradient. Only a small set of variables can be analyzed in a regression model, particularly if the goal is to evaluate the interactions as well as the separate effects of the variables. For example, Haan et al. (1987), cited above, examined individual and neighborhood data as independent predictors, assessing the contribution of the latter once a given individual-level variable was controlled for. However, this does not inform us about the joint and individual functioning of these factors. Because of the complexity of the expression of SES, we need more complete measures and use of statistical procedures to analyze complex, interrelated variables. One such approach is use of tree-structured regression that examines combinations of conditions associated with poorer health outcomes (Segal & Bloch, 1989). This approach partitions populations into subgroups and then identifies different paths to given outcomes. It may be that individuals who have less than a high school education *and* who smoke *and* who are depressed *and* who live in poor neighborhoods show dramatically worse health outcomes. Taken individually these factors may have relatively weak associations with health outcomes, but their combination may be strongly associated.

Alternatively, "grade of membership" (GOM) analysis provides a way to deal with large numbers of variables. Clive, Woodbury, and Siegler (1983) demonstrated that this technique, which uses "fuzzy sets," better portrayed health status over time than did conventional models. GOM analyses develop profiles or "ideal types" either theoretically or empirically. Individuals can then be classified in terms of how closely they match these profiles. For example, Berkman, Singer, and Manton (1989) identified four profiles based on multiple indicators of health and functioning in a community sample of elderly individuals and compared how well Blacks and Whites were characterized by these profiles. An advantage of GOM analysis is that it becomes more precise as more variables are added, rather than becoming more unstable, as in regression.

A deeper understanding of the SES-health gradient may emerge if we examine how variables across multiple dimensions and levels co-occur and interact. Ideally, we would assess variables that characterize various aspects

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