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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The goals of this article are threefold. First, we review evidence that the relationship of SES to health is not simply a threshold effect in which morbidity and mortality increase only at severe levels of deprivation, but is a graded relationship occurring at all levels within the spectrum of social position. Second, we begin the exploration of the gradient by considering factors that could account for this SES–health gradient. This exploration highlights the potential importance of psychosocial variables. Finally, we present a challenge to develop and apply new conceptual and statistical approaches to help understand the nature of the SES–health gradient.

Evidence for the Gradient

Although most studies of SES and health dichotomize individuals on SES or present a single correlation between gross levels of SES and a health outcome, some researchers have become aware of "finely stratified mortality differences running from the top to the bottom of the social hierarchy" (Smith & Egger, 1992, p. 1080). Figure 1 illustrates the findings of a representative subset of studies that have examined mortality rates for at least four levels of SES, and Figure 2 illustrates this for disease prevalence by SES. Because the SES indicators and the cutoff points used to define levels are not standardized, it is not possible to make direct comparisons across studies. However, these figures demonstrate that the SES differences in health occur at every level of SES, no matter what the SES indicator or cutoff point.

The most notable of the studies demonstrating the SES–health gradient is the Whitehall study of mortality (Marmot et al., 1984), which covered 17,350 British civil servants over a period of 10 years. The British Civil Service has ranked grades of employment. The lowest grade consists of unskilled workers (e.g., messengers). The next lowest consists of clerical workers, followed by the professional and executive levels, up to the top administrators. Relative risk of mortality over 10 years significantly increased as employment grade decreased. Compared to mortality risk of the top administrators and controlling for age, relative risk of mortality was 1.6 for the professional–executive grades, 2.2 for the clerical grades, and 2.7 for the lowest grades. Because the sample was relatively homogeneous, with all sharing employment in the Civil Service and having access to nationalized health care, these differences in mortality are all the more striking.

Similar findings emerge from census data in the United Kingdom. Susser, Watson, and Hopper (1985) documented a gradient between five levels of occupational status and standardized mortality rates (SMR, the ratio of observed to expected deaths) in a range of diseases including malignant neoplasms, infectious and parasitic diseases, and diseases of the respiratory, digestive, and circulatory systems. Similarly, Adelstein (1980), using census data, found that SMRs for all causes of death decreased at each of six increasing levels of SES based on occupational status. The SES gradient emerged not only in SMRs but also in the prevalence rates for most, although not all, specific diseases.[1]

Figure 1
*Mortality Rate by Socioeconomic Status Level*
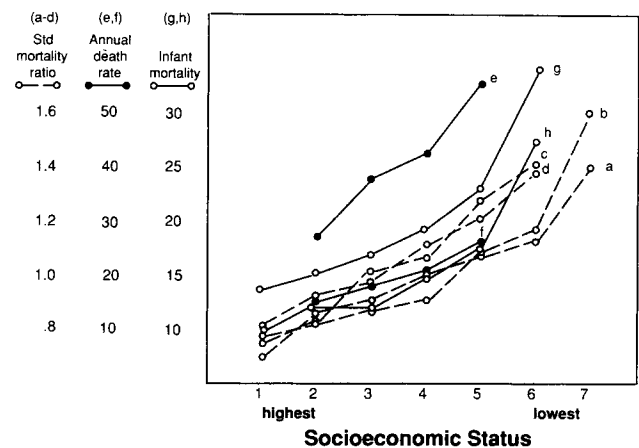
Note. (a) Standardized mortality ratio, observed to expected deaths (SMR) male (Kitagawa & Hauser, 1973). (b) SMR female (Kitagawa & Hauser, 1973). (c) SMR male (Adelstein, 1980). (d) SMR female (Adelstein, 1980). (e) Annual death rate per 1,000 (ADR) male (Feldman, Makuc, Kleinman, & Cornoni-Huntley, 1989). (f) ADR female (Feldman et al., 1989). (g) Infant mortality per 1,000 live births (IM) male (Susser, Watson, & Hopper, 1985). (h) IM female (Susser et al., 1985).

The SES–health gradient has been shown in U.S. studies as well.[2] For example, Kitagawa and Hauser (1973) found a graded relationship between mortality and years of education. The ratio of observed to expected deaths within subgroups among White men aged 25 to 64 years was .70 for those with a college education or better, .85 for those with some college, .91 for high school graduates, 1.03 for those with some high school, 1.07 for those completing eight years of schooling, 1.13 for those with five to seven years of education, and 1.15 for those with four years or less. Comparable ratios for White women of this age were .78, .82, .87, .91, 1.08, 1.18, and 1.60 for each of the education levels. In brief, the more years of education, the lower is the ratio of observed to expected deaths. The gradient for both income and education also emerged in more recent analyses of a national sample reported by Pappas, Queen, Hadder, and Fisher (1993). Pappas et al. compared the degree of association of mortality with education and with income in their data, collected in 1986, with that in the data analyzed by Kitagawa

[1] In a few diseases such as malignant melanoma and breast cancer, a reverse gradient is found. In addition, the gradient may not emerge in every country or epoch. Although beyond the scope of this paper, study of the variation in the direction and degree of association of SES with specific diseases across time and countries would be valuable.

[2] In the United States, research has tended to focus on health differences by race rather than by SES. Research has been hampered by limitations on national data. Although the census provides data on SES, this allows only for analyses of aggregate rather than individual data. Until last year, death certificates provided information only on race; they will now also include years of education. In contrast, Britain provides a ranking of occupational status, which provides a more uniform indicator of social standing (Smith & Egger, 1992).

Figure 2
Morbidity Rate by Socioeconomic Status Level
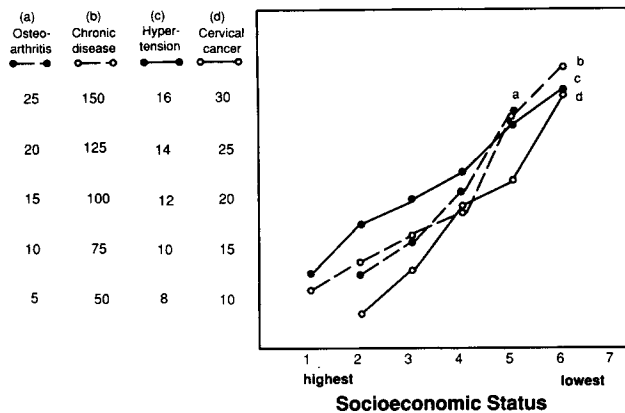
Note. (a) Percent diagnosed osteoarthritis (Cunningham & Kelsey, 1984). (b) Relative prevalence of chronic disease (Townsend, 1974). (c) Prevalence of hypertension (Kraus, Borhani, & Franti, 1980). (d) Rate of cervical cancer per 100,000 (Devesa & Diamond, 1983).

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

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[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.

of other variables to predict health. In reality, however, components of SES, including income, education, and occupation, shape one's life course and are enmeshed in key domains of life, including (a) the physical environment in which one lives and works and associated exposure to pathogens, carcinogens, and other environmental hazards; (b) the social environment and associated vulnerability to interpersonal aggression and violence as well as degree of access to social resources and supports; (c) socialization and experiences that influence psychological development and ongoing mood, affect, and cognition; and (d) health behaviors.

Within these domains, many specific candidate variables may contribute to the SES–health gradient. In this review we have selected those variables that could operate at the upper as well as at the lower end of the hierarchy, although the mechanisms or their relative impact may well differ at different levels. We have focused on variables for which there is empirical evidence of a linear relationship both with SES and with important health outcomes. This review is not exhaustive, but rather it is suggestive of the types of variables and approaches that can be taken to understanding the SES–health gradient. Elsewhere, we have considered the role of access to care in explaining the SES–health gradient and concluded that access alone could not explain the gradient (Adler, Boyce, Chesney, Folkman, & Syme, 1993). Here, we place particular emphasis on psychological and behavioral variables that have largely been overlooked because of the predominant focus on material aspects of SES differences.

Health Behaviors

Health risk behaviors such as cigarette smoking, physical inactivity, poor diet, and substance abuse are closely tied to both SES and health outcomes. Despite the close ties, the association of SES and health is reduced but not eliminated when these behaviors are statistically controlled (Marmot et al., 1984).

Smoking. Cigarette smoking is strongly linked to indexes of SES, including education, income, and employment status, and it is significantly associated with morbidity and mortality, particularly from cardiovascular disease and cancer (Adelstein, 1980; Centers for Disease Control, 1987; Devesa & Diamond, 1983; Escobedo, Anda, Smith, Remington, & Mast, 1990; Kraus et al., 1980; Marmot et al., 1991; Pugh, Power, Goldblatt, & Arber, 1991; Remington et al., 1985; Seccareccia, Menotti, & Prati, 1991; U.S. Department of Health, Education, and Welfare [DHEW], 1979; Winkleby, Fortmann, & Barrett, 1990). Smoking rates vary inversely with SES. In a U.S. community-based survey of 3,349 adults, approximately 41% of men with 12 years' education or less smoked, versus 30% of those with 13–15 years' education, 25% of those with 16 years' education, and 18% of those with more than 16 years' education. Comparable rates of smoking among women at each educational level were 36%, 24%, 15%, and 17%, respectively (Winkleby et al., 1990). A linear gradient between education and smoking

prevalence was also shown in a community sample of middle-aged women: Forty-three percent of women with less than a high school education were current smokers, versus 30% of those with some college, 23% of those with a college degree, and 19% of those with advanced degrees. Additionally, among current smokers the number of cigarettes smoked was related to SES (Matthews, Kelsey, Meilahn, Kuller, & Wing, 1989).

Significant employment grade differences in smoking were found in the Whitehall II study, which examined a new cohort of 10,314 subjects from the British Civil Service beginning in 1985 (Marmot et al., 1991). Moving from the lowest to the highest employment grades, the prevalence of current smoking among men was 33.6%, 21.9%, 18.4%, 13.0%, 10.2%, and 8.3%, respectively. For women, the comparable figures were 27.5%, 22.7%, 20.3%, 15.2%, 11.6%, and 18.3%, respectively. Social class differences in smoking are likely to continue because rates of smoking initiation are inversely related to SES and because rates of cessation are positively related to SES (Escobedo et al., 1990; Kaprio & Koskenvuo, 1988; Pugh et al., 1991).

Physical activity. Involvement in physical activity has both a direct association with health outcomes and an indirect effect insofar as it is associated with obesity. Both lack of physical activity and obesity are positively associated with poor health outcomes (U.S. Department of Health and Human Services [DHHS], 1989; Bouchard, Shepard, Stephens, Sutton, & McPherson, 1990) and are inversely related to SES (Cauley, Donfield, LaPorte, & Warhaftig, 1991; Ford et al., 1991; Kahn, Williamson, & Stevens, 1991; Marmot et al., 1991; Sobel & Stunkard, 1989).

The association of both obesity and lack of physical activity with SES emerged in the Whitehall II study for men but less strongly for women. Among the men but not among the women, those at lower employment grades were significantly more likely to report getting no moderate to vigorous exercise. In a U.S. study, Ford et al. (1991) found an association of physical activity and SES in an urban community sample. Higher SES women spent significantly more time than did their lower SES counterparts in leisure time, job-related, and household physical activity. The men showed qualitative differences in physical activity by SES: Lower SES men spent significantly more time doing household chores and walking, whereas higher SES men spent more time engaged in leisure physical activity.

Alcohol. Alcohol consumption shows the opposite pattern to smoking and other risk behaviors. Several studies (Cauley et al., 1991; Marmot et al., 1991; Matthews et al., 1989) have found a positive correlation of alcohol consumption with SES as measured by education or job status. The relationship between alcohol consumption and health outcomes, however, is not uniform across diseases. For example, although alcohol may increase risk of some cancers (e.g., cancer of the larynx) and alcohol abuse increases risk of cirrhosis of the liver, moderate levels of alcohol consumption are associated

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, & Fronek, 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

lower in the hierarchy may have less opportunity to form, maintain, and access social networks that can buffer the effects of stressful life events (Cohen & Wills, 1985; House et al., 1991; McLeod & Kessler, 1990). In an analysis of 720 persons interviewed in a New Haven mental health catchment area, Kessler (1979) found that persons of lower SES were exposed to more stressful events than were upper SES persons and that, given equal exposure, emotional functioning was more affected among lower than among upper SES individuals.

Evidence for a gradient relation between SES and appraisals of life as stressful was reported in an analysis of a national probability sample collected by the Harris Poll; perceptions of stress decreased in a dose–response fashion in relation to both increased household income and education (Cohen & Williamson, 1988). In summary, higher social economic status is associated with decreases in stressful events and stress perceptions, both of which may affect risk for illness.

Only one study has examined associations among education, stress, and mortality. Ruberman, Weinblatt, Goldberg, and Chaudhary (1984) examined mortality among 2,572 male survivors of MI who were assigned to the treatment condition in a clinical trial of a beta blocker for prevention of subsequent attacks. Educational level, social isolation, and life stress (as measured by questions involving occurrence and evaluation of events or circumstances such as experiencing major financial difficulties, not enjoying one's work, being in a low-status occupation, experiencing a divorce or violent event and reacting by being very upset, etc.) each showed an inverse gradient with mortality over a three-year period. Educational level itself was inversely related to life stress and to social isolation. Moreover, when both life stress and social isolation were high or low, education was no longer linked to differential mortality, suggesting that the zero-order association was due to the linkage of education with stress and social isolation. However, the measure of stress included measures of occupational status, which may have confounded the association with education, and replication of these findings with a better measure of stress is needed.

Effects of Social Ordering

Hierarchical position may have a direct effect on health as well as indirect effects through SES-related differences in the physical and social environment, health behaviors, or personality. In other words, one's relative position in the SES hierarchy, apart from the material implications of one's position, may affect risk of disease. Wilkinson (1992) has shown that among developed countries, per capita income is not as strongly related to life expectancy as is income distribution, with longer life expectancy associated with a greater proportion of income received by the least well-off 70% of the population. Effects of SES hierarchies are most strongly shown within countries rather than across countries, particularly in terms of life satisfaction, suggesting that relative status as opposed to absolute status may be most critical. Provocative research

findings in both animals and humans provide evidence for this proposition.

Hierarchical social structures emerge in virtually all human social groups and serve to reduce intragroup aggression (LaFreniere & Charlesworth, 1983). These structures are stable over time and are present as early as the second year of life (Strayer, 1989; Strayer & Trudel, 1984; Vaughn & Waters, 1978). Dominance hierarchies in primate and subprimate groups have been inferred from observations of antagonistic, aggressive behaviors and were initially assumed to be driven primarily by survival-related competition for limited resources (e.g., food; Bernstein, 1976). More recently, evidence for stable, observable patterns of social dominance has appeared even within primate groups artificially constructed in laboratory settings with universal availability of food and other resources (Manuck, Kaplan, Adams, & Clarkson, 1988).

Hierarchical status in animal models in the laboratory and the natural environment relates to health end points and risk factors for disease. Sapolsky and Mott (1987), for example, found decreased levels of high density lipoprotein cholesterol—a protective factor in CHD—among subordinate wild baboons. Other work in the same laboratory (Sapolsky, 1989) has revealed significant associations between social rank and serum cortisol levels, secretion of gonadal steroids, and immune function.

Conditions in the larger social environment will affect the direction and magnitude of status-related health effects. Manuck and colleagues (Kaplan, Manuck, Clarkson, Lusso, & Taub, 1982; Manuck et al., 1988) found decreased coronary atherosclerosis in socially dominant cynomolgus macaques, but only under stable social conditions. Under unstable conditions that presented recurrent threats to dominance status, dominant animals showed more atherosclerosis than did submissive animals. The atherogenic effects of dominance under unstable social conditions were reversed with a beta antagonist, propranolol, implying that autonomic arousal and cardiovascular reactivity may underlie the observed association. Similarly, Sapolsky (1989) showed that high social rank was protective in the context of a stable hierarchy but was a risk factor for disease under conditions of instability. This work has also produced limited evidence that profiles of protective versus pathogenic physiology change over time with changes in rank, suggesting that physiologic status is a function of hierarchical position rather than the reverse.

The possibility that dominance status can affect physiological and anatomic characteristics is further supported by research on the African cichlid fish, *Haplochromic burtoni*. Davis and Fernald (1990) showed that young, submissive male fish displayed slower phenotypic maturation, hypogonadism, and undersized neuronal cell bodies among the preoptic neurons responsible for secretion of gonadotropin-releasing hormone. Delayed phenotypic and anatomic maturation was found, however, only under rearing conditions in which older, territorial males were also present; in peer-rearing conditions, earlier maturation occurred as a result of more accelerated neu-

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

of SES, including education, income, and occupational status; individual-level variables, such as depression, hostility, sense of control, and health behaviors; and social-level variables, such as characteristics of one's residential neighborhood (e.g., % poverty, air quality), communities (health access, community norms regarding health-relevant behaviors), and work environments. Data analytic strategies that can accommodate multiple correlated variables would allow us to determine which profiles or combinations of variables were associated with better health and lower morbidity and mortality.

Conclusion

Individuals in lower social status groups have the highest rates of morbidity and mortality within most human populations. Moreover, studies of the entire SES hierarchy show that differences in social position relate to morbidity and mortality even at the upper levels of the hierarchy. This observation calls into question traditional explanations for the relationship between SES and health, which pertain primarily to the lower SES levels and the health effects of poverty.

The review presented in this article suggests a series of analytic and conceptual steps that should be taken in an effort to elucidate the impact of SES on health. As a first step in increasing our understanding of the SES gradient, SES should be examined in terms of a set of variables beyond the standard SES indicators. On the basis of existing studies, we have suggested several domains of such factors, which include health behaviors, psychological factors, and perceptions of social ordering. Although not reviewed here, variables in the physical and social environments, such as crowding, pollution, and access to health care, should also be included (Stokols, 1992). The range of individual variables should be broad and should include those that may lower as well as increase risk of morbidity and mortality. It is very likely that some variables and domains will be more potent at lower levels, whereas others may be more relevant to the SES–health association at the upper levels. For example, Margolis et al. (1992) found that the prevalence of both acute and persistent respiratory symptoms in infants showed dose-response relationships with SES. When risk factors such as crowding and exposure to smoking in the household were adjusted for, relative risk associated with SES was reduced but still remained significant. The data further suggest that risk factors operated differently for different SES levels; being in day care was associated with somewhat reduced incidence in lower SES families but with increased incidence among infants from high SES families.

Many of the variables linking SES to health may be dynamically intertwined. Standard analytic methods such as linear regression cannot do justice to the complex relationships among these variables. Their impact on the SES–health gradient may therefore be best described by statistical methods such as regression trees and GOM that can disentangle the effects of variables that co-occur and interact. The application of these methods will enable us to build on the foundation provided by the analysis of individual factors and increase knowledge of the ways these factors directly and in interaction affect health outcomes at different points along the SES–health gradient.

We should not expect, however, that the results of these first stages of analysis will exhaustively explain the SES gradient. Alternatively, they may point to higher order variables, which will account for aspects of the gradient not explained by the subordinate variables that interact and co-occur. The concept of individual control over existing life circumstances, for example, might be a higher order variable that synthesizes or renders coherent a number of the factors reviewed here. There is evidence, largely from older populations, that the experience of control contributes to lower morbidity and mortality (Rodin, 1986a, 1986b). Individuals higher on the socioeconomic ladder may have more frequent or more significant opportunities to influence the events that affect their lives, compared with people at lower levels. This sense of control could affect education, occupation, housing, nutrition, health behaviors, medical care, and other aspects of social class experience not previously discussed. New conceptualizations and measures of control will be needed to capture this type of cross-domain influence.

Social class is among the strongest known predictors of illness and health and yet is, paradoxically, a variable about which very little is known. Psychologists have an important role to play in unraveling the mystery of the SES–health gradient. Several plausible explanations for the puzzling and challenging gradient, including the role of stress, have been proposed, and it will be important to explore these possibilities in more depth in future research. Resolution of the conceptual and analytic dilemmas that have been the focus of this review will be key elements in the continuing, and we hope advancing, efforts to improve health and prevent disease.

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