Socioeconomic Status and Health: What We Know and What We Don't

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Socioeconomic Status and Health: What We Know and What We Don't

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ABSTRACT: **In the past 15 years, we have seen a marked increase in research on socioeconomic status (SES) and health. Research in the first part of this era examined the nature of the relationship of SES and health, revealing a graded association; SES is important to health not only for those in poverty, but at all levels of SES. On average, the more advantaged individuals are, the better their health. In this paper we examine the data regarding the SES–health gradient, addressing causal direction, generalizability across populations and diseases, and associations with health for different indicators of SES. In the most recent era, researchers are increasingly exploring the mechanisms by which SES exerts an influence on health. There are multiple pathways by which SES determines health; a comprehensive analysis must include macroeconomic contexts and social factors as well as more immediate social environments, individual psychological and behavioral factors, and biological predispositions and processes.**

Our perceptions of the world are biased, and one source of bias is selective attention. Selective attention leads us to pay greater attention to certain aspects of our environment and, as a result, to overestimate the prevalence or importance of these aspects. Soon after buying a given model of a car, for example, we are attuned to how many such models are on the road, giving us the impression that almost everyone seems to have made the same choice we have. Our perception that there has been an explosion of interest in the association of socioeconomic status (SES) and health could be the result of biased perception. Having begun our work in this field in the mid-1990s, it could be that our perception of growing interest simply reflects this egocentric bias. However, a review of MEDLINE citations of studies suggests that this is not the case, or at least is not wholly the case. FIGURE 1 presents the number of articles published annually on SES and health. As the figure shows there has, indeed, been a substantial increase in the number of articles appearing on socioeconomic status and health in recent years. The increase has been especially marked in the last few years. In this paper we will examine the three "eras" of research represented in FIGURE 1.

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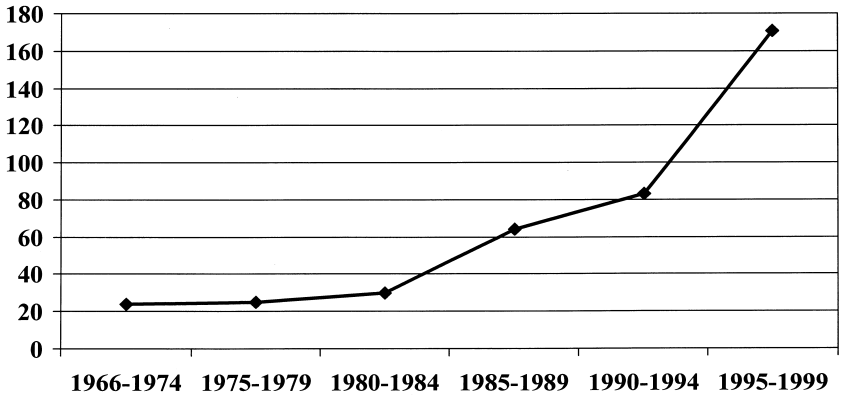

FIGURE 1. Trends in articles on "SES and health" in MEDLINE.

PRE-1985: THE POVERTY ERA

Before the mid-1980s, SES was notable largely in terms of its absence in research on health except as a control variable. Researchers were aware of the power of SES to swamp the effects of the variables in which they were interested. As a result, researchers either used subjects who were homogeneous in terms of SES, or they statistically controlled for SES before entering their variable of interest.

The most frequent measure of SES used in research was poverty status. Individuals were characterized in terms of being either above or below the poverty line. The underlying assumption appeared to be a threshold model (see FIGURE 2). Such a model assumes that increasing levels of income below the poverty line would contribute to improved health. Above the poverty line, however, increasing income would not to be expected to make a significant contribution to improved health status.

During this time there was a good deal of research on the health effects of poverty, which has continued. FIGURE 3 adds to our initial figure of publications on SES, the number of publications in MEDLINE on poverty and health for the same years. As the figure illustrates, there has been substantially more interest in poverty and health than in SES and health, both before and since 1985. Since 1985, as with SES and health, there has also been a marked increase in articles on poverty and health in the medical literature.

1985–1995: DECADE OF THE GRADIENT

A key event in the mid-1980s was a conference organized by Dr. Alvin Tarlov at the Kaiser Family Foundation, resulting in the publication of the volume, *Pathways to Health*.[5] That volume brought together papers from a number of researchers who were suggesting that the impact of socioeconomic factors on health was broader and

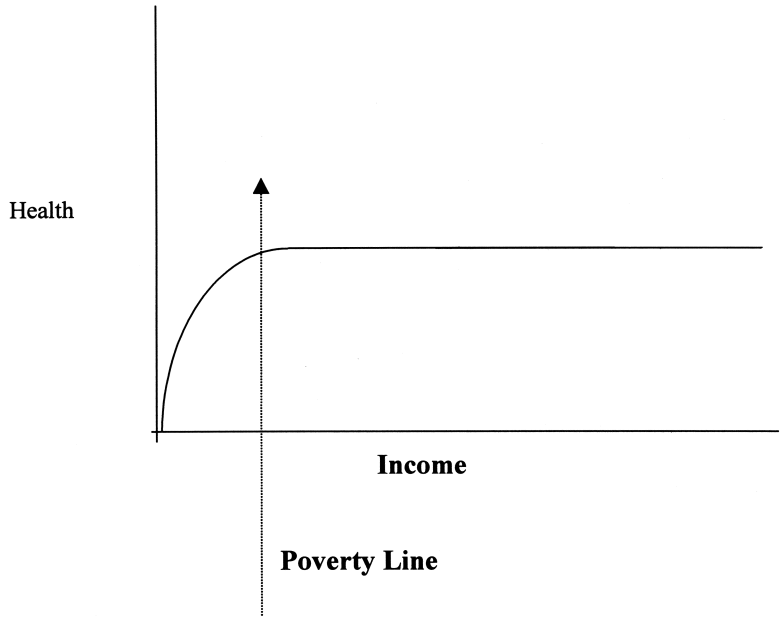

FIGURE 2. Threshold model of poverty.

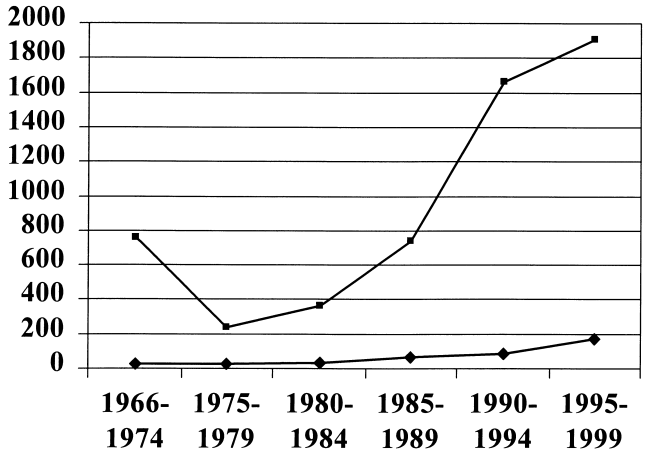

FIGURE 3. Trends in articles on "SES and health" and "Poverty and health" in MEDLINE. ◆, SES and health; ■, poverty and health.

more pervasive than the poverty threshold model represented. Their research was showing that the health effects of SES were not only due to the adversities of extreme poverty, but continued at higher levels of SES as well.

The threshold model was challenged most forcefully by the Whitehall study.[28] This research (which is described in more detail by Michael Marmot in this volume) looked at morbidity and 10-year mortality among British civil servants at each of the occupational grades within the civil service. The research revealed a gradient pattern—health improved and mortality decreased at each higher step of occupational grade. Not only did those at the bottom of the occupational grades have worse health and greater mortality than those above, but there was improvement in health status at each successive step of occupational grade up to the very top. This finding contradicted a threshold model. Not only were all the subjects employed, but also they all had access to health care. Moreover, those in the middle and higher ranks were clearly above the poverty line, yet higher occupational grade, even at this segment of the SES hierarchy, was associated with better health.

One might try to explain away the findings of the Whitehall study by arguing that the British (and especially British civil servants) are so attuned to social class differences in occupational grade or other SES indicators that they would be more profoundly affected by these differences than would individuals in the United States. The Whitehall study challenged us to see whether we could find data to test the gradient in the U.S. Such data were not easy to find, because obtaining them required

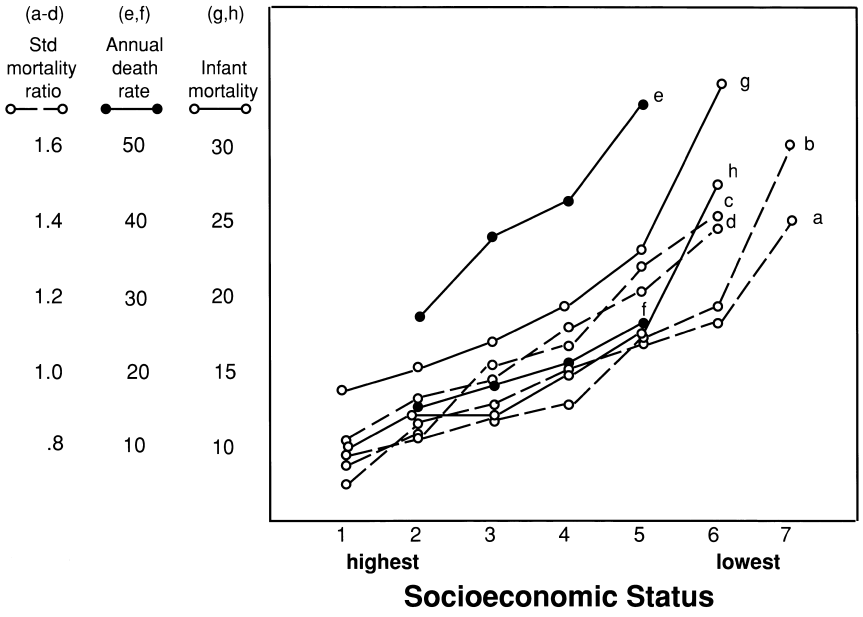

FIGURE 4. Mortality rate by socioeconomic status level. (Taken from the *American Psychologist,* January, 1994, **49**(1): 15–24; used with permission.)

reporting health outcomes at several levels of SES, not simply at the poverty level. Fortunately, several such studies existed. The data, summarized in Adler *et al.,*[1] showed a gradient effect for both mortality and morbidity. FIGURE 4 presents findings from four studies of mortality at different levels of SES. Each study used a different indicator of mortality and different SES measures. As a result, the specific numbers on the *x*-axis do not have real meaning, and their absolute value may be different across studies. Nevertheless the figure clearly shows a gradient relationship between different levels of SES and mortality. The gradient is not perfectly linear, particularly for infant mortality; there is a sharper drop in infant mortality with increases in SES at the lower end of the hierarchy than at the upper end. However, it is clear that even infant mortality continues to drop as one goes up the SES ladder to the highest levels.

The relationship between prevalence of chronic diseases and SES shows an even clearer linear gradient. FIGURE 5 shows data from four studies on prevalence of various chronic diseases by SES level. At each higher level of SES, prevalence of chronic disease decreases. As SES increases, there are drops in the prevalence of osteoarthritis, hypertension, cervical cancer, and having any chronic disease. In addition to morbidity and mortality, risk factors for disease also show a gradient with SES. Rates of smoking, cholesterol levels, and prevalence of sedentary lifestyle are lower the higher one goes on the SES hierarchy, and these occur in a gradient relationship[39] (see also Winkleby[40]).

Research in this decade addressed several questions about the nature of the relationship between SES and health. One question had to do with causal direction—

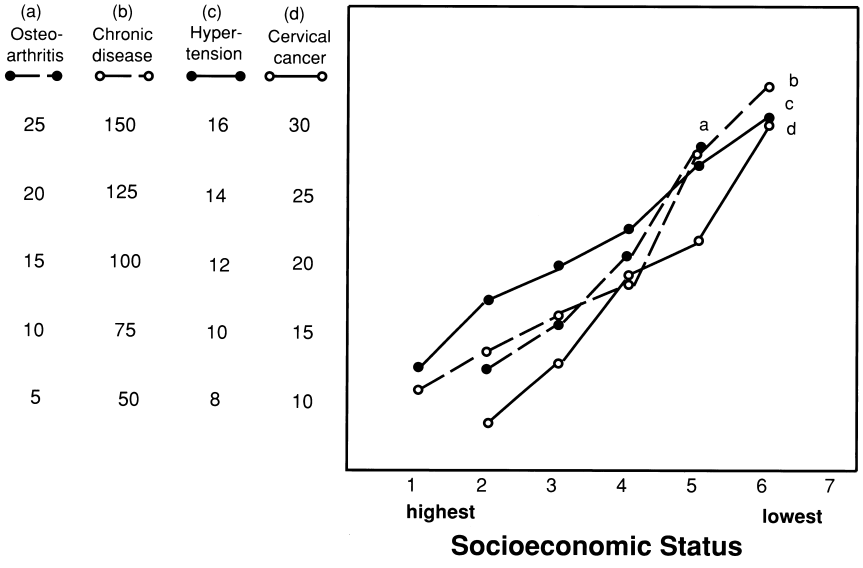

FIGURE 5. Prevalence of chronic diseases by socioeconomic status level. (Taken from the *American Psychologist,* January, 1994, **49**(1): 15–24; used with permission.)

whether SES influences health or vice versa. Other questions had to do with the generalizability of the gradient: Does it occur for all diseases? Does it occur for all populations? Does it occur for all indicators of SES? Each of these questions is discussed briefly below.

Causal Direction

There are two alternative explanations for the association of SES and health. One is that SES influences health status (social causation). The other is that health status contributes to socioeconomic status (social drift or selection). Social drift is more likely for diseases with early onset that have more profound effects on life trajectories (e.g., schizophrenia). Although there is some reciprocal influence of SES and health,[36] the data are more compelling for social causation than for social drift[13,15] For example, we find effects of education acquired through young adulthood on health problems that emerge many years later, suggesting that educational attainment is determining later health. We also find effects of childhood SES on adult health, apart from adult socioeconomic level.[41] Although some childhood diseases are sufficiently debilitating that childhood health may determine educational attainment and later socioeconomic status, these are sufficiently rare that they are unlikely to account for the substantial SES association later in life in general populations. One aspect of the research agenda on SES and health should be to understand how SES plays a role in health across the life cycle and how the cumulative effect of socioeconomic disadvantage operates to influence health.

Diseases

The answer to the question of whether one finds an SES gradient for all diseases is "no." It does not occur for all diseases, but does occur across a wide range of diseases and for many diseases that carry a heavy burden of morbidity and premature mortality. In terms of specific diseases and syndromes, there is a strong and consistent SES gradient for cardiovascular disease, diabetes, metabolic syndrome, arthritis, tuberculosis chronic respiratory disease, gastrointestinal disease, and adverse birth outcomes as well as for accidental and violent deaths.[6,8,18,29,32,35]

There are complicated relationships between SES and cancer. The direction and strength of the association depends on the type of cancer and whether one is looking at incidence or survival. Two diseases for which the gradient in incidence is in the opposite direction (i.e., rates are higher among higher SES individuals) are breast cancer and malignant melanoma. In both cases, behaviors that differ by SES play a role in the reversal of the gradient: delayed childbearing in relation to breast cancer and recreational tanning in relation to malignant melanoma.[10,16] For breast cancer, the familiar SES gradient reappears in looking at survival; once diagnosed, higher SES women show better survival. Longer survival is only partly a function of early diagnosis; the survival advantage for higher SES women remains even when controlling for histology and stage of disease.[7,9]

Lung cancer associations with SES differ by age and gender, reflecting the changing patterns of smoking. Smoking used to be more common among higher SES individuals. As the health effects of smoking became known, particularly through publication of the Surgeon General's report, smoking rates dropped. The decline was

greatest among higher SES men so that there are now higher rates of smoking among lower SES individuals, particularly among men.[35] Lung cancer mortality is now greater among lower SES men than higher SES men, and the association is stronger for those under age 65. The gradient of lung cancer by SES is weaker for women; among women over age 65 there is actually higher mortality among higher SES women.[35]

Looking at the difference in associations of SES with incidence versus survival, there are some suggestions that SES plays a different role in survival for those cancers for which health care makes more of a difference. Research in Canada reveals that the association of SES with survival is strongest for cancers of the head and neck region, uterus and cervix, and bladder. These are cancers in which local symptoms often antedate development of metastatic disease and allow early treatment. In contrast, SES is less strongly related to survival for cancer of the lung and pancreas where diagnosis often comes with systemic symptoms, at which time the disease may be incurable.[27] Kogevinas and Porta[22] reviewed over 40 studies of cancer survival, finding both consistent SES differences in survival and greater differences for cancers in which prognosis is more favorable (e.g., breast, bladder and colon cancer, and cancer of the corpus uteri).

Total mortality from any given disease will reflect both incidence and survival. Analyses of which diseases show the SES gradient in mortality and the extent to which this reflects SES differences in incidence and/or in survival may help identify the more specific pathways by which SES increases premature mortality. Differences in incidence are unlikely to be related to differences in medical care. Currently, most medical care systems allocate relatively few resources to prevention and so access to care in these systems will do little to prevent onset of disease. To the extent that prevention becomes a greater focus of medical care and such care is more available to higher SES individuals, differential access will contribute more to SES gradients in incidence of disease. SES differences in survival may be more closely linked to health care disparities. Even here, health care may play only a partial role. Our research agenda needs to include both studies of the pathways by which SES influences etiology of diseases and their incidence, as well as studies of differential recovery and survival that may involve other pathways and mechanisms.

Populations

The second issue of generalizability has to do with the populations in which the SES gradient has been found: Is it the same in all populations? A gradient between SES and health has been found in almost every industrialized nation in which it has been studied. However, the strength of the association is not uniform. The gradient has been shallower in more egalitarian countries such as the Scandinavian countries,[12] although recent data presented by Mackenbach, Kunst, Cavelaars *et al.*[26] show more complicated patterns of differences across countries in Western Europe. In addition, the familiar gradient may not be found in nonindustrialized countries, at least in terms of cardiovascular disease.[30] For example, research on Nigerian civil servants parallel to the Whitehall studies of British civil servants found a reversal of the gradient. Among the Nigerian civil servants, it was those at higher rank who had a greater incidence of risk factors for cardiovascular disease such as obesity, high-fat diet, and high blood pressure.[4]

Even within the United States, there may be differences in the strength and shape of the gradient in different populations, particularly by race/ethnicity and gender. There is a large literature on racial and ethnic differences in health and within that literature there has been increasing attention to SES.[20,37] Reflecting the history of discrimination in this country, African-Americans and other people of color are more heavily concentrated at lower socioeconomic levels. Some studies have examined and discussed racial differences in health without considering the extent to which these might reflect socioeconomic differences. This attribution to race rather than to socioeconomic differences has been fostered by the nature of available data, particularly in the area of mortality. It is only in recent years that information on education was collected on death certificates in addition to information on race; some states still do not collect data on education. Analyses based on earlier death certificates and those from states that do not collect information on education can therefore only address racial differences. Attributions of differences to race may ignore the contribution of socioeconomic status to differences in health of various racial/ethnic groups.

Not all differences in health among racial/ethnic groups are necessarily due to socioeconomic differences. Members of minority groups face substantial discrimination (see Williams[43]). Personal experiences of discrimination have been found to be associated with greater prevalence of hypertension.[23] In addition, economic and social discrimination may change the association of traditional socioeconomic indicators with health. Additional years of education, particularly at the upper level, appear to "buy" more improvement in health for white men than for white women or for African-American men or women.[3]

We still have very little understanding of how SES is both affected by race/ethnicity and gender or how aspects of SES may operate in conjunction with race/ethnicity and gender to influence health. Thus, another part of the research agenda for the future is developing a greater understanding of the joint and separate functioning of SES, gender, and race/ethnicity in influencing health.

SES Indicators

A final issue of generalizability of the SES–health gradient has to do with the components of SES. SES reflects different aspects of social stratification, and the traditional indicators at the individual level have been income, education, and occupation. These are often used interchangeably even though they are only moderately correlated with one another.[33,39] In some studies in which more than one SES indicator is used, health outcomes may be more highly correlated with one indicator than another. Such studies are useful in identifying specific resources associated with education, income, or occupation that have implications for health. At the same time, similar associations with health have been found no matter which SES indicator is used. Together with the animal literature on the effects of dominant versus subordinate status (see Kaplan and Manuck[43]), this suggests that there may be some common element of social ordering that may be operating to influence health.

One common element among the SES indicators is social status, and a direct measure of subjective social standing may capture this. We have been testing a new measure of subjective social standing and are finding that it has very strong associations with health outcomes, even stronger than the associations of health with objective indicators of SES.[2,4,34]

The measures described above are all individual indicators, but SES also operates at the social level. The interesting work on income inequality has shown that the distribution of income within areas, be they countries, states, or cities, is associated with mortality.[19,21,38] Populations living in areas with greater income inequality have shorter life expectancies, independent of median levels of income. Other studies have shown that the socioeconomic characteristics of neighborhoods in which individuals are living (e.g., the average income of residents, percent unemployed, and residence in a poverty area) predicts morbidity and mortality above and beyond individual SES characteristics.[11,14] Another part of the research agenda is understanding how these multiple levels of influence work together and separately to influence health. These are not alternative explanations but, rather, begin to fill in the complex puzzle of how health is affected by both individual characteristics and the environments in which individuals are living.

1995 AND BEYOND: DECADE OF MECHANISMS

Finally, we come to the most recent era, where there has been another inflection point in publications (see FIG. 1). This might be termed the beginning of the decade of mechanisms. The more recent studies have been addressing the pathways by which SES influences health, examining social, psychological, behavioral, and biological mechanisms. One example of a conceptual model setting out possible pathways was developed by the MacArthur Network on SES and Health. This was done to guide our research on the mechanisms by which SES can get "under the skin" to influence health (see FIG. 6).

The figure indicates that one pathway from SES to health is through exposure to different environments and adaptations to these environments. One aspect of environments with health consequences is differential exposure to pathogens and carcinogens. Equally important are the social and interpersonal aspects of environments, particularly differential exposure to threat and stress in both the work and the home environment. Environments associated with different SES levels may vary in how much control is afforded to individuals, the degree of emotional and instrumental support provided, and exposure to conflict and threat.

Environmental demands and supports can shape psychological responses that, in turn, become more frequent modes of responding. For example, individuals in social environments that are consistently threatening are more likely to develop a sense of distrust and fear of others. Over time this may develop into a more chronic sense of hostility that can place the individual at increased risk for cardiovascular disease (see, e.g., Helmers, Posluszny, and Krantz[17] for evidence on the association of hostility and cardiovascular disease).

The environment also shapes health behaviors. For example, low income neighborhoods have more liquor stores and afford fewer opportunities for exercise and less access to nutritious foods.[25] The combination of individual characteristics and the environmental demands and constraints will affect the likelihood of enacting health-related behaviors such as tobacco use, alcohol use, exercise, and dietary practice.

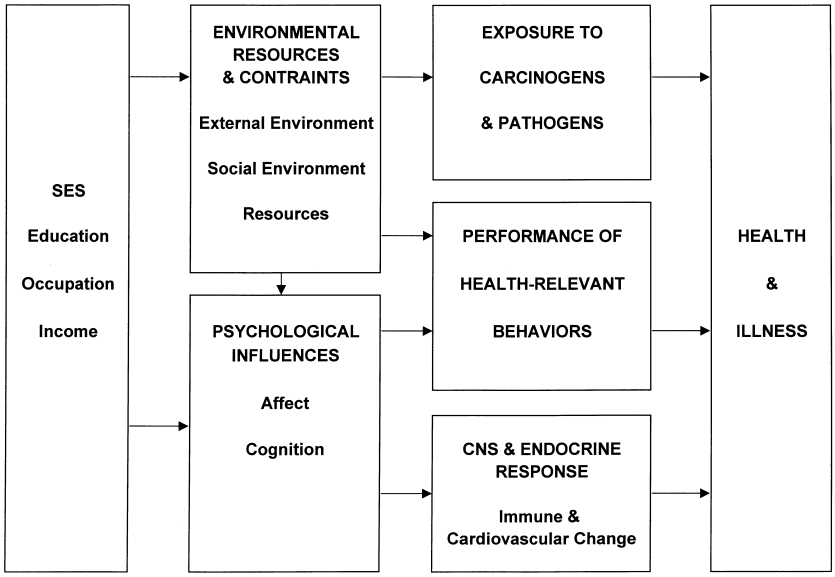

FIGURE 6. Model of the pathways by which SES influences health.

The combination of environmental and individual factors will determine the extent to which the individual experiences repeated stress responses. The CNS and endocrine responses associated with repeated exposures to stress may have long-term effects on the immune and cardiovascular systems, leading to an increased risk of disease onset or more rapid progression of diseases once established. These processes are described in McEwen's chapter in this volume, which examines the development of allostatic load.[44]

The impact of environmental threats and individual responses may be modified by the same health behaviors that are also shaped by socioeconomic forces. For example, exercise may reduce some of the adverse biological effects of stress exposure.[31] At lower positions on the SES hierarchy, one may not only be more subject to chronic stressors that can lead to allostatic load, but also may have fewer opportunities to engage in exercise that could help to buffer the adverse effects of stress responses. Thus, enhanced risk of disease at lower levels of SES is due both to greater exposure to stress and reduced resources for buffering its impact.

This model is oversimplified. The arrows have been drawn in one direction to suggest the pathways by which socioeconomic factors can play out in various ways to influence onset and progression of disease. In fact, there are likely to be feedback loops and interaction effects (e.g., interaction of exercise, stress exposure, and hostility). As research accumulates in this field, we will be better able to establish the pathways and refine our models.

Research on the gradient in the second half of the decade will add to our knowledge of the mechanisms by which SES influences health. Research establishing the pathways from SES to health will be important for developing policies and interventions at the federal, state, or local level to reduce SES disparities in health; some potential approaches are addressed in the final section of this volume (see chapters by Anderson,[45] Lee,[46] and Tarlov[47]). Mechanisms that emerge out of the social environment and broader economic context (e.g., income distribution) may be most effectively addressed by legislation. Other mechanisms may emerge out of more immediate social environments and require more direct action (e.g., building social institutions, empowering communities, modifying working conditions). Still other mechanisms may be at the individual level, so interventions aimed at individual-level change (e.g., health behavior change, parenting support) could help ameliorate some of the adverse effects of SES differentials. Ideally, change can be undertaken at all of these levels to reduce the disparities in health associated with the various determinants. Such change will benefit from a strong science base, and it is our hope that this volume will help to build that foundation.

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