4. Socioeconomic Status and Health: Old Observations and New Thoughts

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Introduction

Observations of a positive association between socioeconomic position (SES) and health are not new, extending back to the 12th century (Antonovsky 1967; Syme and Berkman 1976). Inquiry into these matters is found in the 1826 investigation of Villerme into the relationship between rent level and mortality in Paris and in Farr's work in 1851 on differences in mortality in England by occupation (Susser et al. 1985). Since these early reports, variations in disease risk by level of SES have been noted but seldom explained. Indeed, the ubiquitous nature of these findings paradoxically has led to the exclusion of SES as a subject of inquiry. Instead of being the subject of investigation in its own right, SES is often seen as a background characteristic similar to age and sex, a characteristic that must be included in analyses but that is seldom addressed directly.

The purpose of this paper is to refocus attention on the association between SES and health in hopes of stimulating explanations for this phenomenon. First, we will consider the consistency over time and place of the relationship between SES and health. From this review, we will identify features of the association that can guide a search for explanations. Finally, we will assess alternative explanations currently being proposed and offer our own. In order to facilitate discussion, this report also includes a chartbook of selected studies (Figures 1–26), which appears at the end of this chapter, and an annotated bibliography of

recent reports on SES and health, which appears at the end of this volume.

Review

We have found it both convenient and instructive to organize our review of SES and health according to stages of the life span. Not only is this organization logically clear, it illustrates the continuing influence of SES on health throughout the life span.

Infancy and Childhood

We have found evidence that the association between SES and health status persists from birth to death. With regard to infants and children, there are a large number of studies that indicate variations in health risk by level of SES. Figure 1 presents a good example from Sweden of the associations often observed between birth outcomes and SES (Ericson et al. 1979). In this study of almost 200,000 births that occurred in 1976–1977, SES was inversely associated with perinatal mortality, prematurity, low birthweight, small size for gestational age, and late (>43 weeks) birth. These results from Sweden are particularly impressive when one considers the low rates of these outcomes in Sweden relative to other countries. This study used a summary measure of SES based on census information on mother's education, family income, quality and type of housing, and number of family members.

Other studies have reported similar findings based on mother's or father's occupation (Antonovsky and Bernstein 1977; Murrells et al. 1985; Rantakallio 1979; Rush and Cassano 1983), income (Antonovsky and Bernstein 1977; Starfield and Budetti 1982), or education (Antonovsky and Bernstein 1977; Gortmaker 1979; Kitagawa and Hauser 1973; McCormick 1985; Rantakallio 1979), or on census tract or other area indicators of these measures (Antonovsky and Bernstein 1977; Brennen and Lancashire 1978; Brooks 1975; Ericson et al. 1979; Hadley and Osei 1982; Kraus and Redford 1975; Simpson 1984). Figure 2 shows the relationship between maternal and paternal education and rates of perinatal death for six states in the United States in 1973

(World Health Organization 1985). Similar results for education, social class, and income, respectively, are presented in Figures 3–5 for Finland (Rantakallio 1979); England and Wales (Morris 1979); and San Antonio, Texas (Markides and Barnes 1977). Other studies have shown that SES is related to a large number of other mortality and morbidity outcomes in children (Brennen and Lancashire 1978; Dutton 1985; Lines 1977; Mare 1982; McWhirter et al. 1983; Morgan and Chin 1983; Spiers et al. 1974; Starfield and Budetti 1982).

Adolescence and Early Adulthood

From early childhood to the forties, the major causes of death are those associated with injury, which account for more deaths than all other causes combined. Here, too, there are substantial gradients of risk associated with SES (Mare 1982; Baker et al. 1984). Figures 6 and 7 show the association between death rates from injuries and per capita income of area of residence. For deaths from unintentional injuries, which account for roughly two thirds of all injuries, there is a strong gradient of risk associated with SES, and this gradient is found for whites, blacks, and Asians. Homicides also decline with income. Motor vehicles are the major source of injury up to age 44; drowning is the next most prevalent. For each of these outcomes, there is a strong gradient of risk associated with SES (Figure 8).

A variety of other studies of those in this age group also shows a positive association between health and SES. Lower SES is associated with higher prevalence of heart disease and high blood pressure (National Center for Health Statistics 1983), higher incidence of diabetes (Medalie et al. 1974; Figure 9), and higher prevalence of orthopedic impairments associated with injury (National Center for Health Statistics 1986). In addition, for both blacks and whites, there is an association between SES level, days of restricted activity, acute conditions, bed days, and short-stay hospital days (National Center for Health Statistics 1985, 1986, 1987; Figures 10 and 11).

The Middle Years

As the incidence and prevalence of chronic conditions begin to increase from age 40 on, the association between SES and

health continues to be observed. Strong negative associations are found between SES and all-cause mortality (Antonovsky 1967; Kaplan 1987; Kitagawa and Hauser 1973; Metro Life 1977; Nagi and Stockwell 1973; Nayha 1977; Pearce et al. 1983a, 1983b, 1985; Syme and Berkman 1976). A number of studies, using different measures, have reported an association between SES and cardiovascular outcomes (Marmot et al. 1984; Rose and Marmot 1981; Salonen 1982). Figure 12 presents this relationship as observed in the Whitehall study of British civil servants (Marmot et al. 1978, 1984; Rose and Marmot 1981). Similar SES gradients for death and cardiovascular endpoints have been observed in Finland (Koskenvuo et al. 1979; Nayha 1977; Notkola et al. 1985; Salonen 1982; Figure 13), Sweden (Haglund 1985; Lapidus and Bengtsson 1986), Norway (Arnesen and Forsdahl 1985; Forsdahl, 1977; Holme et al. 1981; Leren et al. 1983), England and Wales (Black et al., 1980; Morris 1979; Figure 14), and, within the United States, in Los Angeles (Frerichs et al. 1984; Figure 15), Birmingham, Buffalo, and Indianapolis (Yeracaris and Kim 1978), Alameda County (Kaplan 1985), Baltimore (Kuller 1972), Washington County, Maryland (Comstock and Tonascia 1978), and in Australia and New Zealand (Dobson et al. 1985; Fisher 1978; Pearce et al. 1983a; Figure 16).

A negative association between SES and prevalence of disease, for those aged 40 to 60, is also found for arthritis, heart disease, ulcers, diabetes, high blood pressure, chronic bronchitis, and emphysema (National Center for Health Statistics 1983; Figure 17), gallbladder disease (Diehl et al. 1985), incidence of cervical and lung cancer (Devesa and Diamond 1980; Hakama et al. 1982; Figures 18 and 19), and mortality from all sites of cancer (Dayal et al. 1984; Hathcock et al. 1982; Holme et al. 1981a; Jenkins 1983; Moser et al. 1984; Salonen 1982). As to the burden of injury in these years, injuries from falls and house fires begin to add significantly to those due to motor vehicle accidents, and for all these there is an inverse association with SES (Baker et al. 1984).

Old Age

After age 60, a negative association between SES and all-cause mortality continues to be seen in most studies. As deaths from

malignancies begin to rise overall, SES is inversely associated with cancer of the esophagus, pancreas, lung, stomach, prostate, and other sites (Dayal et al. 1984; Devesa and Diamond 1983; Mack and Paganini-Hill 1981; Marmot et al. 1984, Steinhorn et al. 1986). Overall cancer survival is poorer in lower SES patients compared to higher SES patients (Blane 1985; Chirikos and Horner 1985; Dayal et al. 1982, 1985; Lipworth et al. 1970; Savage et al. 1984; Smith and Day 1979; Wegner et al. 1982). Kitagawa and Hauser (1973) in their study of deaths in the United States among those over 65 years of age found negative associations between SES, measured by education level, and mortality in those over 65 years old from diabetes, major cardiovascular-renal diseases, vascular lesions affecting the nervous system, arteriosclerotic and degenerative heart disease, influenza and pneumonia, and accidents. In analyses from the National Health Interview Survey, for those 65–74 years old, there is a negative association between family income and prevalence of arthritis, diabetes, hypertension, chronic bronchitis, and emphysema (National Center for Health Statistics 1983). For those 65 years old and over, there is an inverse gradient between SES and acute conditions, activity limitations due to chronic conditions (National Center for Health Statistics 1985), bed disability, days of restricted activity (National Center for Health Statistics 1987), hearing impairments, and orthopedic impairments of the back and lower extremities (National Center for Health Statistics 1986, Figure 20).

All of these studies indicate the pervasive and consistent relationship between SES and health, a relationship that is found throughout the life course and across a wide variety of diseases and organ systems. Although any one of these studies could be criticized, the consistency of the evidence clearly is sufficient to merit a serious search for explanations. This consistency is all the more impressive because it is generally observed regardless of the way in which SES is assessed. Thus, SES is linked to disease whether one uses measures of income, occupation, education, area of residence, or prestige ranking—either alone or in combination.

What Needs to be Explained?

In addition to addressing the basic observation of an association between SES and health, any proposed explanation ideally

should address four key issues. We will examine each of these in some detail.

SES GRADIENTS

The usual focus on the SES-health link has been on those at the very bottom of the SES hierarchy. This focus is understandable because occupants of the lowest position are subjected to a wider variety of and greater exposure to physical, psychological, social, and economic insults. Nevertheless, most of the evidence we have reviewed shows that the health disadvantage associated with lower SES is not restricted to those at the bottom. It appears that there is an SES gradient of health. Even persons in the upper middle class are at higher risk for disease than those at the very top of the scale. This gradient persists for different measures of SES, in different populations, and in different countries. It consistently shows that as we descend the SES hierarchy, rates of ill health increase. This observation is important because whatever single or multiple factors are responsible for the SES-health association should take this gradient into consideration.

LIFE-SPAN EFFECTS

The SES-health link persists throughout the life span, manifesting itself in differential infant mortality rates, accidental deaths among youth and young adults, in cardiovascular mortality rates among middle-aged adults, and in all-cause mortality across all ages. It may be true, as Marmot suggests in his comments in chapter 7, that the health disadvantage associated with lower SES begins at or before birth. For example, poor nutrition *in utero* or exposure to environmental lead in early childhood leads to compromised health and poorer educational performance. Poorer educational performance, in turn, could lead to continuing social and economic disadvantage and again to higher exposure to social and environmental risks.

Given that the health disadvantage associated with lower SES may begin early and may be cumulative over the life span, it is still possible that different risk factors may be responsible for the poor health of lower SES people at different stages of life. It is important for our understanding of this phenomenon to exam-

ine the *pattern* of such etiologic risk factors in addition to looking at them singly.

TEMPORAL TRENDS

The SES-health link appears to persist over time and may be increasing in strength. The pattern of risk factors that might "explain" the link must also persist over time, even though single factors composing the pattern could change. The overall pattern should also change accordingly where there have been shifts in the SES-health relationship.

INTERNATIONAL CONSISTENCY

As is clear from the introductory material and the chartbook, the association between SES and health has been observed in a wide variety of countries, cultures, and economic systems. Again SES-related patterns of risk factors should demonstrate some level of cross-national consistency and interpretability.

We think that the gradient, the persistence over the life span and over time, and the international consistency of the association are basic to our understanding of SES and health. These are central characteristics of the SES-health association, and any effort to explain it should adequately examine them. Our review has covered 300 studies on this topic. In our writing we have not attempted to account for every study of SES and health; instead we have focused on the major examples. We believe that such an "unbalanced" approach is defensible when the weight of the evidence is as convincing as it is in this case. Nevertheless, we recognize that exceptions will also need to be addressed.

CURRENT EXPLANATIONS

As noted in the *Black Report* (Townsend and Davidson 1982) and elsewhere, several major themes predominate in research and speculation about the association between SES and health. The most frequent of these themes is the idea that SES differences in behavioral risk factors such as smoking, alcohol consumption, and diet account for the link between SES and mortality. Another view is that the SES link to health reflects the influence of

health on SES. In other words, ill persons lose income and "drift" into lower social positions. Others hold that there are personal or social characteristics of individuals that keep them in poverty and that these characteristics are "cycled" from generation to generation. A fourth approach identifies socioeconomic or material factors as determinants of the SES-health link. For example, deprivation of access to medical care, to food, transportation or education are identified as the causative factors. Finally, a fifth approach theorizes that SES differences in exposures to noxious social and physical environments may lead to higher rates of morbidity and mortality in lower SES groups.

In the following sections, we will consider the usefulness of these themes in accounting for the SES-health association.

Individual Behaviors

It is often proposed that the gradients of health associated with SES are due to different levels of high risk behavior among those in different SES strata. According to this view, the SES-health link can be accounted for by differences in smoking, diet, alcohol consumption, and similar lifestyle factors. Since a substantial body of knowledge links such risk behaviors to poor health, this is an attractive proposal. Such a view, however, needs to be critically examined from a number of perspectives.

First, even if the magnitude of the association between measures of SES and health outcomes is reduced after adjusting for behavioral risk factors, the interpretation of such a finding is not without its problems. Such a pattern of results may very well indicate not that SES is irrelevant and that we should focus on these risk factors, but rather that it is an antecedent, causal variable that is involved in the adoption and maintenance of high risk behaviors.

Second, even if high risk behaviors are distributed in a graded fashion by SES strata, it still would remain to be seen whether or not the differential distribution of these factors actually did account for the increased risk in these strata.

Unfortunately, there are only a limited number of studies that have examined the association between health outcomes and SES, taking into account the practice of high risk behaviors.

One such analysis comes from the Human Population Laboratory's ongoing follow-up of a sample of residents of Alameda County, California (Kaplan et al. 1986). Figure 21 presents survival curves for county residents followed for 19 years as a function of baseline income (Figure 21–A) and education (Figure 21–B). Family income, adjusted for family size, is grouped into four categories (inadequate, marginal, adequate, very adequate) by reference to federal standards at the time (1965), and education is grouped into three levels (0–11 years, 12 years, 13 years or more). The ordinate represents the proportion surviving at each year of follow-up. With adjustment for age and sex, those in the lowest income category had 2.23 times the risk of death compared to those in the top category ($p<.00001$), and those with 0–11 years of education had 1.57 the risk of those with some college ($p<.001$). Parts C and D of the figure illustrate that the increased risk associated with both income and education remains when there is adjustment for a number of high risk variables, in this case smoking, alcohol consumption, physical activity level, relative weight, and amount of sleep. There is also adjustment for race and prevalent chronic disease (hypertension, heart trouble, cancer, diabetes). As can be seen in this figure, there is still a strong gradient associated with both measures. Those in the inadequate, marginal, and adequate categories are at 1.78 ($p<.0012$), 1.56 ($p<.004$), and 1.30 ($p<.025$) times the risk, respectively, of those with very adequate incomes. Those with 0–11 years of education are at 1.30 ($p<.03$) times the risk of those with some college. Although in each case there is some reduction in the pattern of risk associated with SES, a substantial, increased risk associated with low SES remains, and, in the case of family income, a strong gradient persists.

Several analyses of the association between SES and cardiovascular outcomes have come to similar conclusions. The Whitehall study (Marmot et al. 1984) observed a substantial gradient in cardiovascular risk associated with employment grade in its 10-year study of British civil servants. Although levels of smoking, high blood pressure, physical inactivity, and other risk factors were higher in the lower grades, adjustment for these factors accounted for less than one half of the observed association. Those in the lowest grade were at a fourfold increased risk when

these other factors were taken into account (Marmot et al. 1978; Rose and Marmot 1981). Increased cardiovascular risk associated with lower employment grade was also found for other outcomes not particularly related to smoking (Marmot et al. 1984). Similar conclusions were reached in studies in Finland and Norway. Salonen (1982) found increased risk of death from ischemic heart disease associated with low education and income in a 7-year study of men in Eastern Finland. This increased risk persisted after adjustment for age, serum cholesterol, diastolic blood pressure, and amount of smoking. In another prospective study in Norway, Holme et al. (1981b) found that less than half of the gradient in risk of coronary heart disease associated with low income and education could be explained by levels of serum cholesterol, triglycerides, blood pressure, cigarette smoking, physical activity, and other risk factors. Thus, there is a consistent body of evidence that finds that differences in the distribution of behaviors related to cardiovascular disease do not explain fully the association between SES and cardiovascular outcomes.

Turning to reproductive health outcomes, a similar picture emerges. Although smoking is associated with low birthweight (National Academy of Sciences 1985) and higher rates of perinatal and postneonatal mortality (Bakketeig et al. 1984; Rush and Cassano 1983), and although there tend to be gradients of smoking related to SES, these variations in smoking do not seem to account for the association between SES and these outcomes. Rush and Cassano (1983), for example, studied the association between SES measured by occupational grade and both low birthweight and perinatal mortality in 16,688 singleton births occurring in Britain in one week's time in 1970. Although maternal smoking was an important predictor of both outcomes, it did not fully account for the association between these outcomes and SES. For example, 63 percent of the association between occupational grade and low birthweight remained after statistical adjustment for maternal smoking, age, gravidity, height, and marital status. Similarly, 75 percent of the SES-perinatal mortality association remained after adjustment for maternal smoking and other factors. Rantakallio (1979), in a Finnish study, also found that low maternal education was associated with lower birthweight and higher perinatal and post-

neonatal mortality, as well as higher rates of hospital admissions during the first five years of life. These differences were not attributable to higher rates of smoking in low SES women.

Finally, van den Berg and Oechesli (1984), in a study of 11,000 pregnancies that went beyond 22 weeks, found that rates of prematurity (e.g., delivery in first 37 weeks) were related to maternal education. This finding held when there was statistical adjustment for maternal smoking, weight gain, first trimester bleeding, time since last pregnancy, length of gestation of previous pregnancy, sex of infant, and father's education. Again we see a picture consistent with that for cardiovascular diseases. Although certain high risk behaviors may be more prevalent among those in the lower SES strata, they do not seem to account fully for the gradient of association between SES and reproductive outcomes.

There seems to be an "independent" role for SES, even when it comes to lung cancer. Although smoking is undoubtedly the most important factor in lung cancer incidence, several studies suggest that SES is related to lung cancer incidence even when variation in levels of smoking by SES strata are taken into account. For example, Marmot et al. (1984) found a similar gradient for occupational grade and lung cancer when they compared ex-smokers and smokers who consumed 1–9, 10–19, and 20 or more cigarettes per day. (As would be expected, there were too few lung cancer deaths in nonsmokers to complete the full set of comparisons.) Similarly, Salonen (1982) found a significant association between education level and risk of cancer mortality, approximately 60 percent of which would be due to lung cancer, when there was adjustment for smoking and other variables. Comstock and Tonascia (1978), in their studies in Washington County, Maryland, also found a trend indicating that low education was associated with increased risk for lung cancer death even after adjustment for smoking status.

Finally, recent analyses of the 18-year cancer incidence experience of respondents in the Alameda County Study (Kaplan et al. 1986) have indicated a surprisingly strong relationship between SES and lung cancer incidence and mortality. Women who were in the bottom quartile of the distribution of family income adjusted for family size, had an 11.0 ($p<.03$)- and 16.6

($p<.01$)-fold increased risk of lung cancer death or incidence, respectively, when compared with women in the top quartile of adjusted family income, with adjustment for age, race, and pack-years of smoking. No such association was found for men. These results should not be taken as indicating that smoking is not important in the etiology of lung cancer. In all analyses, smoking remains an important predictor. Rather, in the case of lung cancer where tobacco consumption is such a strong predictor, the independent risk associated with SES indicates the strength of the SES-disease association.

Unfortunately, few studies have examined the joint influence of measures of SES and other risk factors, so we cannot come to any definitive answers regarding the independent influence of SES on most disease outcomes. Considering individual SES factors, however, can provide some insight. In analyses of occupational stress and ischemic heart disease, for instance, Haan (1985) reported that persons who smoked and were exposed to high levels of job strain were at a fourfold risk of ischemic heart disease compared to those who were nonsmokers and had low strain jobs. These and other analyses suggest that behavioral risk factors may interact with SES in the disease process but not account fully for the association.

Selection

Some have interpreted gradients of health associated with SES as reflecting the influence of health on SES, rather than vice versa (Illsley 1955; Stern 1984; Wadsworth 1986). Because it is undoubtedly true that illness is often accompanied by declines in some measures of SES such as income, it is important to examine this reverse causation argument thoroughly. Analyses of the dynamics of income change in a panel of 5,000 families indicated that 28 percent of married men experienced a work loss due to illness during an 11-year period (Duncan 1984). Kitagawa and Hauser (1973) in their study of all deaths in the United States in a 4-month period in 1960, reported that decedents aged 25–64 years old had worked less in the preceding year than those who survived, and thus had lower incomes.

It is also likely that changes in occupational status occur in re-

sponse to health problems. In some cases—schizophrenia, for example—there is good evidence for a drift into lower occupational groups due to such a disorder. Drift into lower occupational groups is also more prevalent in those who were seriously ill during childhood (Wadsworth 1986). In an example of the same phenomenon occurring later in life, Illsley (1955) found that women who experienced intergenerational upward mobility, defined with respect to the occupational group of their fathers compared to their husbands, were healthier before marriage than those who did not move upward via marriage. In short, health may influence social status in both a positive and negative direction.

Given that there is some impact of health on SES, it is necessary to establish to what extent this influence makes an important contribution to the observed association between SES and health. Based on our review of the evidence, we do not feel this explanation is of major importance. First, a comprehensive discussion of the contribution of health-related mobility to SES gradients observed in England and Wales has been provided by Wilkinson (1986), who concludes that the impact of such selection is unimportant.

Second, the selection argument depends on shifts in income and occupation but does not take education into account. Because educational attainment occurs relatively early in life, it is temporally impossible for it to be influenced by health status measured decades later. For this reason, when the SES-disease link is assessed in terms of education level, this link cannot be due to poor adult health influencing amount of education. For the vast majority of disease outcomes among adults, there is an inverse gradient related to level of education. Marmot (1987) has pointed out that poor health in early childhood due to low SES may lead to poorer educational and occupational attainment in adulthood. This phenomenon is not an example of initial poor health leading to downward drift but is, instead, an example of the cascading effects of lower SES on health throughout the life span.

Third, if the SES-health link were a result of drift due to poor health, then we would not expect to see an SES-health associa-

tion for family members. Yet there are a number of studies that find gradients of health for children or wives related to the SES circumstances of their fathers or husbands.

Fourth, the drift hypothesis cannot explain the observed gradients of health among retired individuals for whom income is no longer as dependent on health. Yet, in some analyses, for some measures of SES, the gradients are actually stronger among older persons.

Fifth, if these gradients reflect a bias that transfers the sick to lower and lower levels of SES, then in longitudinal studies we should expect that the gradients would be greatest during the years immediately following the beginning of follow-up, and they should decrease over time. Fox et al. (1985) in their 10-year follow-up of a 1 percent sample of the population of England and Wales in 1971, found just the opposite effect with increasing length of follow-up—that is, the SES gradient, defined with respect to occupational groupings, was actually larger in the second five years of follow-up than in the first.

The drift hypothesis is further contradicted by the observation that for many outcomes it is incidence of disease that is inversely related to SES. Given that incidence represents the time at which the disease is first diagnosed, it is less likely that there has been any major earlier impact of the disease on SES. The gradients of mortality from unintentional injuries related to SES also cannot be due to downward drift, especially since they occur with greatest frequency among the young. Survival from a number of conditions also appears to be related to SES, and better survival is associated with higher SES even when prognostic factors have been taken into account.

Finally, we were able to examine the joint impact of health changes and income changes on mortality risk using data from the Alameda County Study (Kaplan et al. 1986). In a sample of healthy persons aged 50 and over, income loss was associated with higher risk of subsequent death, and this association was not due to worsening health coincident with income loss. Even if poor health were associated with movement into lower SES, one would still want to know what antecedent conditions were associated with this poorer health. Given the pattern of evidence

surveyed, it seems likely that a good proportion of downward drift in SES due to health may in fact be due to an etiologic role in early life for SES.

In summary, although it is true that poor health can lead to lower SES, the bulk of evidence surveyed does not indicate a major role for this pathway in accounting for the graded relationship between SES and health outcomes.

Medical Care

The central question to be addressed in this section is not whether there are inequities in access to and quality of medical care by SES, as clearly there often are, but rather whether SES inequities in medical care access or utilization could explain the SES-health link. The real question is whether lack of access to medical care is the *reason why* lower SES people have higher rates of incident disease, higher mortality, and poorer survival. Even with this limited definition, it will be difficult in this brief discussion to examine in depth the impact of access to and quality of medical care on the SES-health link.

Although lack of access to medical care is frequently cited as a cause of poorer health and higher mortality in lower SES groups (Starfield and Budetti 1982), research in a number of countries where access to medical care is relatively equitable has still revealed substantial SES differences in health (Holme et al. 1981a, 1981b; Koskenvuo et al. 1979; Pearce et al. 1983a; Rantakallio 1979; Rose and Marmot 1981). Of course, as several reviewers pointed out, potential equality of access is not the same as actual equality of access. Even in countries where medical care is free to all, there may still be more subtle barriers to quality care for lower SES people. The *Black Report* (Townsend and Davidson 1982) failed to find improvement in the SES-health differential, even with improved access to medical care afforded by the British National Health Service.

The SES gradient is important to take into consideration in examining the possible role of medical care in accounting for the SES-health link. Differences in medical care access, quality, and utilization are unlikely to account for the health status difference between the top of the SES scale and the level just below it.

We have reported SES differences in both incidence and survival. In general, medical care is less efficacious than other interventions in the prevention of new chronic diseases in adults (incidence) (McKeown 1979). However, early/improved diagnosis and control of hypertension and diabetes are examples of diseases where stroke, mortality incidence or death from diabetes-related complications may be reduced or postponed to a later age through medical interventions. Earlier diagnosis of cervical cancer could be another example. Evidence from the Hypertension Detection and Follow-up Program suggests that a comprehensive hypertension treatment program can lead to an elimination of SES-related differences in mortality related to hypertension (Stamler et al. 1987).

There are many disease events that occur more frequently in lower SES people on which medical care can have very little substantial preventive impact. For example, the associations between SES and injuries (Kristiansen 1985; Sunderland 1984) and new occurrences of alcohol-related diseases (Coates et al. 1985; Edwards et al. 1978; Haberman and Baden 1974) and smoking-related diseases are clearly not alterable through medical treatment. SES differences in infant mortality do appear to be significantly influenced by access/utilization of medical care (Dott and Fort 1975; Grossman and Jacobowitz 1981; Kraus and Redford 1975; Papiernik et al. 1985; Taylor 1983; Wright 1975). For this outcome, mortality is clearly influenced by access to and quality of medical care. Starfield (1985) has exhaustively documented the efficacy of medical care in preventing infant mortality, low birthweight babies, and a variety of childhood diseases. However, SES differences in infant mortality have been reported in countries such as Sweden (Holme et al. 1981a) and Finland (Notkola et al. 1985; Salonen 1982) where equal access to prenatal/postnatal care for all SES groups is a matter of national health policy (Shin 1975).

In short, SES-related differences in access or utilization of preventive medical treatment may result in SES differences in survival from disease. Secondly, it may postpone the onset of some diseases or reduce the incidence of some diseases through the treatment of risk factors. If people of lower SES have differential access to medical care, their survival and age at mortality

may indeed be affected. However, the fact that people of lower SES have higher incidence of many important diseases and injuries both in childhood and adulthood suggests that the SES link is unlikely to be explained *entirely* by lack of medical care access or poorer quality of medical care. It also seems unlikely that medical care could account for the SES gradient or for SES differences in health and injury outcomes not alterable by medical treatment. Nevertheless, our review of this issue does not rule out medical care as a possible and important intervention that would be useful in lessening the impact of SES on health.

Personal and Social Characteristics of Individuals

This approach to explaining the relationship between SES and health argues that the poor come to adopt and transmit through generations patterns of thought and action that place them at higher risk of poor health. There are a number of reasons for believing that such an approach cannot explain the SES-health link. It is hard to see, for example, exactly what patterns are consistently arrayed throughout SES strata in a way that could account for the gradients of health that have been observed. Furthermore, as pointed out, although high risk behaviors may be more prevalent in lower SES strata, they do not seem to account for the elevated disease rates in these groups.

Increasingly, references are made to the relationship between such concepts as control, efficacy, coherence, hardiness, coping style, and other related measures and health outcomes. Is it possible that these constructs provide a link between SES and health? There is some evidence that lower SES persons may perceive themselves as less in control of external events, and there is evidence that such beliefs are associated with poorer health (Rodin 1986). However, it is important to question whether these measures are psychological, reflecting characteristics of individuals, or whether instead they represent cognitive assessments of social process. In general, this latter hypothesis has not been well studied.

At a conceptual level, the observation that these perceptions of control and efficacy are differentially distributed by SES strata argues for viewing these characteristics as outcomes, reflecting

differences in environment and experience at different levels of SES. Kohn and Schooler's (1974) classic study of the impact of job characteristics on an individual's personality and cognition is an example of such influences. In other words, the observation that beliefs and attitudes vary by SES strata should not be taken as convincing evidence that these beliefs and attitudes "explain" the SES-health link. Instead, they should prompt us to examine aspects of the lives of people in different SES strata that could lead to these different patterns of cognition and personality.

Other Considerations

Trends Over Time

Persistence or change over time of the SES-health association is of interest because examination of patterns of change may help to identify explanatory factors. Persistence over time also supports the notion that the SES-health association is not an artifact of a particular time period or data set.

Any attempt to characterize changes in SES differences in health over time should address several key questions:

- First, a descriptive matter: Have SES differences in mortality changed over time; and, if so, are there differences in the trends by cause of death?
- Can changes in exposure to behavioral or environmental risk factors account for these trends in SES differences?
- What roles do early selection or competing risk play in trends in SES differentials in mortality?

Several researchers have examined changes over time in the SES gradient of mortality. In general, SES differentials in all-cause mortality have remained stable or have possibly increased. Blaxter (1983) has reported that class differentials are widening even as overall mortality declines. Pamuck (1985) has reported in her analysis of mortality trends from 1921–1971 in England and Wales, that SES inequalities in health declined in the 1920s, increased in the 1950s and 1960s, and by the 1970s were greater than in the 1920s. Similarly, Fox et al. (1985), in their examination of SES mortality differentials, report that the SES differential in the Office of Population Censuses and Surveys (OPCS)

Longitudinal Study increased between 1971–1976 and 1976–1981 in England and Wales (Figure 22). Susser et al. (1985) have also reported a steepening differential in this association between 1931 and 1971. Wilkinson (1986) has also noted an increase in the all-cause differential between 1951–1971 (Figure 23). It should be noted that explanations for the observed changes primarily invoke shifts in lifestyle—especially in smoking, drinking, and diet—as the likely factors. However, Susser et al. (1985) and others have also noted that changes in environmental exposures and in the efficacy of medical care may contribute to SES-related mortality trends. For example, if occupational exposure to industrial pollution has increased and if those of lower SES are more highly exposed, such environmental changes could lead to increasing SES differences over time.

Relatively little research has been reported on trends in cause-specific mortality by SES. In general, the evidence supports an increasing SES mortality differential for major causes of death, consistent with the findings for all-cause mortality. For example, Yeracaris and Kim (1978) and Lerner and Stutz (1978) both report an increase in the SES-mortality differential between 1960 and 1970 for cancer and heart diseases.

When we examine cancer outcomes in more detail, SES differences in mortality appear to have persisted over time and increased. Lerner (1986), reporting on income differentials in cancer mortality separately for blacks and whites, found an SES differential for both races throughout the period between 1949 and 1971. In other words, low income whites and blacks had higher cancer mortality rates than high income whites and blacks when compared within race. This was true not only for all sites, but for many specific sites as well. The effect of income was especially strong for respiratory cancers. Similarly, Marmot et al. (1984) have reported that the SES differential in mortality from lung cancer increased between 1970–1972 and 1979–1983. Unfortunately, most data on cancer incidence and mortality are not reported by SES, but only by race. Data on race suggest that for lung, gastrointestinal, and reproductive cancers (excluding breast), incidence is higher among blacks and has increased.

Attempts to explain the SES-health association can benefit by examination of changes in trends. A particular example of the

way that etiologic factors may be identified can be found in the possible reversal in the coronary heart disease (CHD)-SES association reported in three studies. Specifically, Marmot et al. (1978a) and Morgenstern (1980) have both reported that the association between SES and CHD reversed from a positive to a negative association between 1950 and 1960. Susser et al. (1985) have also reported a rise in age-specific CHD mortality between 1931 and 1971, with a similar reversal occurring about 1955 for men only. However, in this latter report, the picture for women is quite different: it shows no reversal in CHD, with low SES women remaining at higher risk throughout the 1931–1972 period. In fact the SES differential for women increased sharply after 1960. Other sources have reported that the SES gradient for CHD has persisted over time and may have increased in recent years.

Marmot et al. (1978a), Morgenstern (1980), and others (e.g., Pamuck 1985) have argued that the reversal in the CHD-SES-association may be explained by the diffusion of high risk behaviors to the lower classes at the same time that the upper classes were abandoning such behaviors. In the United States, however, the major reduction in prevalent risk behaviors *postdates* the reversal in CHD. The U.S. Surgeon General's report on smoking was not issued until 1964, and public consciousness and avoidance of these risk factors is relatively recent (U.S. Surgeon General 1985). Figure 24 shows the annual rate of decline in smoking prevalence by occupational class and birth cohort in the United States from 1950 to 1978 (Carstairs 1981). Birth cohort is presented in order to account for the rather substantial cohort differences in smoking. These data suggest that the greatest decline in smoking prevalence occurred between 1970 and 1978 and that the least change occurred between 1950 and 1960, the period during which the CHD-SES reversal may have occurred. Other data show that smoking prevalence increased for all classes until 1950 in the United States. In short, the greatest changes in high risk behavior did not begin until *after* the reversal in the CHD-SES association. Therefore, it would be temporally improbable for diffusion of high risk behavior to have accounted for this reversal.

In any event, the bulk of evidence on the association of smok-

ing, cholesterol, and other risk factors and CHD-SES mortality suggests that these factors cannot fully explain SES-health differences. As we have mentioned previously, cholesterol, smoking, and other risk factors account for less than half of the CHD-SES association. While in some studies this lack of effect may be due to inadequate measurement of major risk factors, in several studies adequate measurement of these risk factors has not altered the observed association.

The role of selection and competing risk are important when examining SES-related trends in mortality. If the crossover reported by Marmot et al. cannot be accounted for by changes in smoking and diet, an alternative explanation may be found in SES differentials in competing risk. If higher SES groups not only have a general survival advantage but are also the first to receive the benefits of medical, behavioral, or environmental improvements, then conversely one may presume that the lower SES groups continue to get sick and die for a longer period from diseases that are on the decline in general.

If lower SES persons are dying younger, they are also probably dying at greater rates from causes characteristic of younger ages such as injuries, cirrhosis, and acute infectious diseases, since cardiovascular disease (CVD) and cancer are very much phenomena of old age. Thus trends in the SES-mortality link may be a product of both differential selection and competing risk. In other words, until 1960 the life expectancy of lower SES persons was such that they had less likelihood of living long enough to get CVD. The all-cause mortality-SES differential then could remain stable or increase while CVD mortality, once a disease of the privileged, became a disease more prevalent in lower SES groups. It must be said that Fox et al. (1985) did not find evidence for early selection in their analysis of recent trends (1971–1981), but they did not examine age-specific rates across different birth cohorts. However, no research has been done to examine SES and patterns of competing risk over time.

In short, trends in the SES-health association may be due to a number of factors that seem to be linked to behavioral changes, environmental changes, or changes in medical interventions. Changes in behavioral risk factors or changes in the social and physical environment, or both, may help to explain changes in

the SES-health association. Appropriate examination of these changes will require comparisons of two or more cohorts of persons on whom SES, behavioral, environmental, and health data are collected over time. This approach will need to take account of the early selection and competing risk problems we have discussed.

SES and Minority Health

In the United States, minority status (particularly being black, Hispanic, or Native American) and SES are so closely linked that no examination of the relationship between SES and health is complete without a discussion of minority health. The question, simply put, is whether or not the association between SES and higher rates of disease simply reflects the higher percentage of minority persons in lower SES strata. Although little data are available to answer this important question, what is available suggests that the answer is no. For example, Figure 25 presents such an analysis for blacks and whites in the Alameda County Study (Haan and Kaplan 1985; Kaplan et al. 1986). Figure 25-A indicates the poorer mortality experience of blacks compared to whites. As can be seen, there is a substantial difference of approximately 30 percent in risk of death. Figure 25-B indicates what happens when black/white differences in income are taken into account. In these analyses, the difference between black and white mortality was substantially reduced and became statistically nonsignificant when income differences were taken into account. Adjustment for education reduced the black/white risk difference by 8 percent, but the association between race and mortality was still significant ($p = .02$). Thus, these analyses indicate that it is income, not being black, that is largely responsible for the observed mortality differentials.

This discussion has not attempted to address whether the poorer health seen in many minority groups is due exclusively to the lower SES predominant in these groups. It is possible that the simple measures of SES used in most analyses do not fully capture all the cultural and historical experiences that are relevant to the health experience of a given minority group. As Nickens points out in his comments in Chapter 7, these features

point where they engage in high risk behaviors as a response to the stressors. However, the bulk of environmental studies lack data on individual characteristics or health status and rely instead upon the correlation between ecological measures of health and ecological measures of the social and/or physical environment.

Two, the measurement of the environment is ecological; that is, no direct link can be made between individual-level exposures or individual health status and the measures of the environment, leading thus to the so-called ecological fallacy. The statistical and inferential errors that may arise from trying to impute individual risk from ecological data have been pointed out by many.

An exhaustive review of these environmental studies is neither possible nor necessary for this discussion. The usual study design involves the use of socioenvironmental data derived from the Census, Housing Survey, Environmental Protection Agency air pollution monitoring stations, and other data bases that assess area characteristics. These data are linked to area mortality rates. For example, two well-known studies by Jenkins et al. (1977) linked elevated area rates of CHD mortality and cancer mortality to residence in lower SES neighborhoods. Similarly, Devesa and Diamond (1983) found elevated rates of lung cancer in lower SES neighborhoods. Neither of these studies was able to link individual characteristics or health status with the area measures.

These limitations have been addressed in research currently being carried out by Haan and Kaplan (1987, in preparation) in the Alameda County Study. Specifically, it has been possible to link area measures taken from the Census to individual-level measures taken from survey data on individuals, and to follow those individuals prospectively. In research by Haan et al. (1987, Figure 26), residence in a poverty area was significantly associated with subsequent nine-year all-cause mortality in Oakland, California. Current analyses of the same data have expanded to include all of Alameda County. More detailed measures of the social environment have been formulated based upon 1960 Census data and linked to data on individuals. At this time, four major factors have been identified that are consistently and persistently associated with all-cause, cardiovascular, and cancer

may also interact differently with the lower socioeconomic status predominant in that group.

An Alternative Approach

In previous sections of this paper, we have outlined and discussed a variety of factors that may describe and/or account for the link between SES and health status. No factor or set of factors clearly emerges as a likely candidate. In this section, we attempt to summarize and discuss the associations between the social and physical environment and SES and health. This final approach differs from previous approaches because it is less well developed as a research area and because it is more controversial in its implications. In our discussion of the social and physical environment, we will separately discuss the community at large and the work environment since they comprise separate areas of research and have different implications for intervention.

Community Environment, SES, and Health

A number of studies (Brooks 1975; Dayal et al. 1982, 1984; Devesa and Diamond 1980, 1983; Harburg et al. 1973; Hathcock et al. 1982; Jenkins et al. 1977; Kasl and Harburg 1975; Kendrick 1980; Kraus and Redford 1975; Lawton and Nahemow 1979; Miller 1982; Taylor and Emery 1983) have examined the influence of the social and physical environment upon health. Somewhat less has been written about SES differentials in environmental exposures that might account for the SES-health link (Beresford 1981; Blair et al. 1980; Blot and Fraumeni 1976; Kuller et al. 1972; West 1977). For example, a number of epidemiological studies have used area characteristics to examine the influence of SES on health. Others have investigated the influences of SES-related differences in exposures to physical environmental hazards such as lead and air pollution. These studies have been criticized from at least two perspectives:

One, if there is an environment-health link, it is believed to be through the medium of individual behaviors (for example, smoking) that are induced by or influenced by environmental factors. These factors are thought to stress individuals to the

mortality after adjustment for behavioral risk factors, race, income, education, social networks, baseline health status, political activity levels, depression, and personal uncertainty. These factors were identified through principal component analysis and represent clusters of variables that characterize the sociophysical environment of neighborhoods.

These analyses have supplied more information about the association between the social and physical environment and health. It appears that clusters of social and physical area characteristics collectively influence health status, whereas they do not do so when used as individual measures. Thus, for example, residence in a census tract simultaneously characterized by high proportions of men over 65, widowers, persons without cars, deteriorated housing, and housing without heat is associated with elevated all-cause and cardiovascular mortality, and with cancer incidence. Taken one at a time, these characteristics are not related to these outcomes. Taken together, they describe an ecological setting and are strong predictors of mortality. It seems clear from these findings and from research by others that socioenvironmental risk factors cluster and that they include measures of social network disruption, age of population, housing conditions, income, racial composition, and access to transportation. Ultimately, they are strongly associated with the health of individuals who live in their midst.

Research on SES and exposures to noxious physical environments is a long-standing tradition in public health, and investigations of poor sanitation and unsafe housing are common. More recent research has attempted to link air pollution exposure (Lave and Seskin 1977; Lloyd et al. 1985; Phillipp and Hughes 1985) and, in particular, exposure to environmental lead, with SES and health. For example, a recent study by Schwartz et al. (1985) found an association between blood lead levels and hypertension. Other analyses of leaded gasoline sales and hypertension have reported significant associations (Pirkle and Schwartz 1985). The supposition in these latter analyses has been that residential proximity to freeways (often a marker of low SES) is associated with increased exposure to environmental lead. In addition, some studies of soil lead levels have revealed that lower SES areas have higher levels of lead than suburban

areas. Studies of elevated blood lead in children have found a threefold higher prevalence in children of lower SES (Mahaffey et al. 1982). The effect of lead is also potentiated by undernutrition and iron deficiency anemia, both of which occur commonly in children of lower SES. Residence in polluted areas tends to be strongly associated with other factors such as low income and poor housing.

Environmental pollution often covaries with sociodemographic factors such as area of residence and may indeed add to the overall burden of social and physical exposures encountered by persons of lower SES. However, no research has been done that examines the association between environmental pollution and health while adjusting for SES or in interaction with SES.

Work Environment, SES, and Health

Occupation is commonly used as a measure of SES. As with other measures of SES, little is understood about why occupation and health are linked. Research on this topic generally falls into the category of exposures to toxics on the job or into the category of psychosocial stress. It is clearly understood that persons of different SES have different kinds of jobs. In what ways job characteristics vary consistently by SES may be important in understanding the SES-health link. Much work examining psychosocial factors has already been done by researchers on job stress, health, and organizational environments. Little work has been done to examine the hypothesis that the physical environment of jobs could help to account for the SES-health link.

Considerable research has been conducted examining the association between job characteristics and health, particularly coronary heart disease. Although these findings are not always consistent, the bulk of evidence points to an association between CHD and jobs characterized by low variety and high demands accompanied by low control over pace and schedule. Haan (1985), Karasek et al. (1981), and Theorell and Rahe (1982) have all reported similar findings, taken mostly from prospective studies. For example, in a 10-year prospective study of ischemic heart disease in Finland, Haan found a strong association between education and income and job strain, and also found that

job strain is a stronger predictor of CHD incidence than is education. Karasek et al. (1981) similarly reported that job strain accounted for more of the variance in a United States sample than did the Duncan SES scale (a commonly used measure of SES, composed of income, education, and occupation). The level of job strain to which an individual is exposed is not entirely independent of education. Job strain exposure may in fact be a consequence of education. In part, lifelong exposure to job strain may account for the association between education and health. Haan (1985) has found that in analyses that included adjustment for age, sex, six cardiovascular risk factors, and education, adjustment for job strain accounted for the association between education and ischemic heart disease. The same results were found for all-cause mortality. In general, it appears that the job strain model is promising as an explanatory factor that varies in a consistent manner across job categories, is strongly associated with more traditional measures of SES, and is associated with health. Furthermore, there is a gradient of health associated with job strain.

Although the physical work environment is often included in the measure of job strain discussed above, some researchers have suggested that exposures to noxious occupational physical or chemical agents could account for SES differences in health (Haan 1985). Although there is little research examining this supposition, it is not entirely implausible to think that persons in lower SES will be exposed to more hazardous agents on the job. However, the SES gradient in health cannot be accounted for by variations in such exposures since persons in the higher SES levels are not likely to be more exposed to physical or chemical agents than those in the highest SES. In addition, the unemployed, who are presumably not exposed at all, generally suffer the highest mortality and morbidity rates of all occupational groups. On the other hand, some evidence exists (Haan 1985) suggesting that workplace exposure to noise co-occurs with job strain and may interact with job strain to enhance the risk of coronary heart disease. This area deserves much more attention.

This discussion has attempted to sift through and summarize an expanding field of research on the sociophysical environmen-

tal causes of ill health, looking for a link between SES and health. Several findings seem to emerge from this examination:

1. Sociodemographic environmental factors may be associated with health status, and this association may not be due to the mediation of individual-level behavioral risk factors. These factors may be useful in accounting for the SES-health link.

2. Job strain is associated with the incidence of nonfatal and fatal coronary heart disease and with all-cause mortality and may provide additional explanatory material concerning the association between education and CHD and all-cause mortality.

3. There is a need for a general conceptual model to help organize thinking on this topic. The demands-control model developed for job strain research might be translated to wider settings such as the community and be used to examine the SES-health links in general. The central focus of the job strain model is that *control* over work pace, scheduling, and hours interacts with physical and psychosocial *demands* such that persons experiencing low control and high demands are at highest risk. Presumably, in a low control–high demands situation, individuals may be inhibited from mobilizing the personal and social resources they need to cope with and meet those demands. In addition, the availability of these resources may be an important factor in such responses. Although this approach is relatively well developed in the job stress literature, it has not been adapted or applied to the investigation of SES and health. We will expand upon this approach in our final section.

Conclusions

We have reviewed evidence in this paper and elsewhere that SES and health are associated, that the SES-health link is graded, and that this gradient exists in many industrialized countries. We have seen also that the SES differences in health have persisted over time and in fact have probably increased. Further, they are not likely to be primarily accounted for by behavioral risk factors, by medical care factors, by selection, by racial differences, or by personality differences. We have reviewed evidence that SES and health are linked by a variety of possible routes, includ-

ing individual, social, and environmental, but no single factor or set of factors seems completely to account for this association. Clearly, what is needed is an approach to thinking about this problem that integrates and organizes what is known so that a coherent program of research and intervention can be designed.

Any attempt to provide a detailed discussion of such a theoretical approach is beyond the limits of this paper. However, we would like to summarize our thinking in this area. The job strain research discussed in the previous section may provide us with a new direction if it can be expanded and translated to a more general model. As noted, this research suggests that disease rates are higher when people are exposed to high levels of job demands coupled with few resources to cope with those demands. Extending this concept beyond the job circumstances, it is possible to argue that disease rates will be high when people live in high demands situations and where they have a smaller repertoire of resources. The fact that those in lower SES positions have a higher burden of demands is clear; that they have fewer resources for dealing with those demands seems clear as well.

It should be pointed out, however, that resources are not simply characteristics of individuals but are also, and perhaps predominantly, characteristics of the physical and social environment. Thus, while individuals in lower SES positions may have less training and experience in dealing with demands and in taking advantage of opportunities, they also are faced with a variety of important structural constraints that limit access to options and opportunities. A proper translation of the demands/resources model to the problem of SES and health, therefore, will require a far broader approach to risk factors than is generally utilized. As our research (Haan et al. 1987) has begun to demonstrate, community or area characteristics may be important determinants of the health of those who live in them.

One central disagreement among those researching SES and health seems to be whether different factors are responsible at different levels of SES for the link or whether a single "big factor" is responsible. It does seem that a consistent pattern of association exists between SES and health. There are also exceptions to the pattern, as noted by Harris in his discussion of breast cancer and malignant melanoma in Chapter 6. However, it is the prac-

tice of epidemiologists in investigations of disease to focus first on the commonalities of exposure for identification of an etiologic agent rather than on the exceptions. In fact, we have generally found that the various specific factors proposed as candidates for explaining the SES-health link frequently covary with one another and do form an underlying "big factor," statistically speaking. Since we have observed a consistency of effect, it is not unreasonable, and may in fact be enlightening, to search for commonalities across social classes that can "explain" the association. If, as some reviewers have commented, these commonalities are really a multiplicity of different factors, then some general model may be useful for organizing the pieces into a coherent picture. We have attempted to introduce one such possible model in our discussion of job strain and the demands-control concept. This model implies that different elements of the physical and social environment may *interact* with behavioral risk factors and may also, at the same time, *covary* with such factors.

This is not to say that interventions directed at specific factors may not be important. Rather, we would like to explore an approach to intervention that focuses on the social and physical environment. Such an approach could allow extension and generalization beyond single factors to the entire SES-health relationship.

Current research by Haan and Kaplan in this area suggests that the social and physical environment plays a significant role in the SES-health link. This approach may provide a model that incorporates and permits integration of the individual, interactive, and environmental levels at which the SES-health link is seen. The central focus in this approach would be the demands-resources model previously discussed. This model permits us to examine simultaneously the physical, economic, social, and psychological demands placed upon individuals by their environment while relating these same demands to the availability, quality, and nature of the resources that individuals can command in response to them. It also allows consideration of the characteristic ways in which individuals act upon their environment to obtain resources.

To explore this approach a program of research is needed that includes the following:

1. Conceptualization and measurement of physical and social environmental resources and demands which can be linked to individuals *over time*. Such measures should include the working, home, and neighborhood environment and should consider not only people's perceptions of their environment but also the objective circumstances within which people live.

2. Conceptualization and measurement of characteristic interactions *over time* between individuals and their environments. Again, such measures should include interactions among all three of the environments to which the individual is exposed.

3. Conceptualization and measurement of individual-level psychosocial, social, physical-biological, and behavioral resources and demands through self-report and observation.

The SES-health link is one of the most profound and pervasive observations ever made in public health. The reasons for this link are not clear. Previous efforts to explain it have left many questions unanswered. We believe that a focus on an approach that integrates environmental and individual levels, and centers on demands and resources at both levels, holds promise for clarifying and advancing understanding in this area.

References

Antonovsky, A. 1967. Social class, life expectancy, and overall mortality. *Milbank Memorial Fund Quarterly* 45:31–73.

———, Berstein, J. 1977. Social class and infant mortality. *Social Sciences and Medicine* 11:453–70.

Arnesen, E., Forsdahl, A. 1985. The Tromso heart study: coronary risk factors and their association with living conditions during childhood. *Journal of Epidemiology and Community Health* 39:210–14.

Baker, S. P., O'Neill, B., Karpf, R. S. 1984. *The injury fact book.* Lexington, MA: Lexington Books.

Bakketeig, L. S., Hoffman, H. J., Oakley, A. R. 1984. Perinatal mortality. In Bracken, M. B., ed. *Perinatal epidemiology*, pp. 99–151. New York: Oxford University Press.

Beresford, S. A. 1981. The relationship between water quality and health in the London area. *International Journal of Epidemiology* 10:103–115.

Black Report. 1980. *Inequalities in health: report of a research working group*. Great Britain: Department of Health and Social Services.

Blair, A., Fraumeni, J. F., Mason, T. J. 1980. Geographic patterns of leukemia in the United States. *Journal of Chronic Diseases* 33:251–260.

Blane, D. 1985. An assessment of the Black Report's 'explanations of health inequalities.' *Sociology and Health Illness* 7:423–445.

Blaxter, M. 1983. Health Services as a defence against the consequences of poverty in industrialized societies. *Social Sciences in Medicine* 17:1139–1148.

Blot, W. J., Fraumeni, J. F., 1976. Geographic patterns of lung cancer: industrial correlations. *American Journal of Epidemiology* 103:539–550.

Brennen, M. E., Lancashire, R. 1978. Association of childhood mortality with housing status and unemployment. *Journal of Epidemiology and Community Health* 32:28–33.

Brooks, C. H. 1975. Path analysis of socioeconomic correlates of county infant mortality rates. *International Journal of Health Services* 5:499–514.

Carstairs, V. 1981. Multiple deprivation and health state. *Community Medicine* 3:4–13.

Chirikos, T. N., Horner, R. D. 1985. Economic status and survivorship in digestive system cancers. *Cancer* 56:210–217.

Coates, R. A., Corey, P. N., Ashley, M. J., Steele, C. A. 1985. Alcohol consumption and blood pressure: analysis of data from the Canada health survey. *Preventive Medicine* 14:1–14.

Comstock, G. W., Tonascia, J. A. 1978. Education and mortality in Washington County, Maryland. *Journal of Health and Social Behavior* 18:54–61.

Cunningham, L. S., Kelsey, J. L. 1984. Epidemiology of musculoskeletal impairments and associated disability. *American Journal of Public Health* 74:574–579.

Dayal, H. H., Chui, C. Y., Sharrar, R. et al. 1984. Ecologic correlates of cancer mortality patterns in an industrialized urban population. *Journal of the National Cancer Institute* 73:565–574.

Dayal, H. H., Polissar, L., Dahlberg, S. 1985. Race, socioeco-

nomic status, and other prognostic factors for survival from prostate cancer. *Journal of the National Cancer Institute* 74:1001–1006.

Dayal, H. H., Power, R. N., Chui, C. 1982. Race and socioeconomic status in survival from breast cancer. *Journal of Chronic Diseases* 35:675–683.

Devesa, S. S., Diamond, E. L. 1980. Association of breast cancer and cervical cancer incidences with income and education among whites and blacks. *Journal of the National Cancer Institute* 65:515–528.

———. 1983. Socioeconomic and racial differences in lung cancer incidence. *American Journal of Epidemiology* 118:818–831.

Diehl, A. K., Rosenthal, M., Hazuda, H. P. et al. 1985. Socioeconomic status and the prevalence of clinical gallbladder disease. *Journal of Chronic Disease* 38:1019–1026.

Dobson, A. J., Gibberd, R. W., Leeder, S. R., O'Connell, D. L. 1985. Occupational differences in ischemic heart disease mortality and risk factors in Australia. *American Journal of Epidemiology* 122:283–290.

Dott, A. B., Fort, A. T. 1975. The effect of availability and utilization of prenatal care and hospital services on infant mortality rates. *American Journal of Obstetrics and Gynecology* 123:854–860.

Duncan, G. J. 1984. *Years of poverty/years of plenty*. Ann Arbor, MI: University of Michigan.

Dutton, D. B. 1985. Socioeconomic status and children's health. *Medical Care* 23:142–156.

Edwards, G., Kyle, E., Nicholls, P., Taylor, C. 1978. Alcoholism and correlates of mortality. *Journal of Studies in Alcohol* 39:1607–1617.

Ericson, A., Eriksson, M., Zetterstrom, R. 1979. Analysis of perinatal mortality rate in the Stockholm area. *Acta Paediatrica Scandinavica Supplement* 275:35–40.

Fisher, S. 1978. Relationship of mortality to socioeconomic status and some other factors in Sydney in 1971. *Journal of Epidemiology and Community Health* 32:41–46.

Forsdahl, A. 1977. Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart

disease? *British Journal of Preventive and Social Medicine* 31:91–95.

Fox, A. J., Goldblatt, P. O., Jones, D. R. 1985. Social class mortality differentials: artefact, selection or life circumstance. *Journal of Epidemiology and Community Health* 39:1–8.

Frerichs, R. R., et al. 1984. *Cardiovascular disease in Los Angeles.* Los Angeles: American Heart Association, Greater Los Angeles Affiliate.

Gortmaker, S. L. 1979. The effects of prenatal care upon the health of the newborn. *American Journal of Public Health* 69:653–660.

Grossman, M., Jacobowitz, S. 1981. Variations in infant mortality rates among counties of the United States: the roles of public policies and programs. *Demography* 18:695–713.

Haan, M. N. 1985. *Organizations and ischemic heart disease: an epidemiologic study of employees in a Finnish metal fabrication company.* Doctoral dissertation. Berkeley, CA: University of California.

———, Kaplan, G. A. 1985. The contribution of socioeconomic position to minority health. In Heckler, M., ed. *Report of the secretary's task force on black and minority health. Vol. II. Cross-cutting issues in minority health*, pp. 69–103. Washington, D.C.: Department of Health and Human Services.

Haan, M., Kaplan, G. A., Camacho, T. 1987. Poverty and health: prospective evidence from the Alameda County Study. *American Journal of Epidemiology* 125(6):989–998.

Haberman, P. W., Baden, M. M. 1974. Alcoholism and violent death. *Quarterly Journal of Studies on Alcohol* 35:221–231.

Hadley, J., Osei, A. 1982. Does income affect mortality? An analysis of the effects of different types of income on age/sex/race-specific mortality rates in the United States. *Medical Care* 20(9):901–914.

Haglund, J. A. 1985. Geographical and socioeconomic distribution of high blood pressure and borderline high blood pressure in a Swedish rural county. *Scandinavian Journal of Social Medicine* 13:53–66.

Hakama, M., Hakulinen, T., Pukkala, E., Saxen, E., Teppo, L. 1982. Risk indicators of breast and cervical cancer on ecologic

and individual levels. *American Journal of Epidemiology* 116:990–1000.

Harburg, E., Erfurt, J. C., Chape, C. et al. 1973. Socioecological stressor areas and black-white blood pressure: Detroit. *Journal of Chronic Diseases* 26:595–611.

Hathcock, A. L., Greenberg, R. A., Dakan, W. A. 1982. An analysis of lung cancer on a microgeographical level. *Social Science and Medicine* 16:1235–1238.

Holme, I., Helgeland, A., Hjermann, I., Leren, P. 1981a. The Oslo study: social indicators, risk factors and mortality. In Bostrum, H., and Ljungstedt, N., eds. *Medical aspects of mortality statistics*. Stockholm: Almquist Wiksell.

Holme, I., Helgeland, A., Hjermann, I., Leren, P., Lund-Larsen, P. G. 1981 b. Physical activity at work and at leisure in relation to coronary risk factors and social class. *Acta Medicina in Scandinavica* 209:277–283.

Illsley, R. 1955. Social class selection and class differences in relation to stillbirths and infant deaths. *British Medical Journal* (Dec 24):1520–1524.

Institute of Medicine. 1985. *The prevention of low birth weight*. Washington, D.C.: National Academy Press.

Jenkins, C. D. 1983. Social environment and cancer mortality in men. *New England Journal of Medicine* 308:395–398.

———, Tuthill, R. W., Tannenbaum, S. I., Kirby, C. R. 1977. Zones of excess mortality in Massachusetts. *New England Journal of Medicine* 296:1354–1356.

Kaplan, G. A., 1985. Psychosocial aspects of chronic illness: direct and indirect associations with ischemic heart disease mortality. In Kaplan, R. M., and Criqui, M., eds., pp. 237–269. *Behavioral epidemiology and diseases prevention*. New York: Plenum Press.

———, Haan, M. N. Sept. 1986. *Socioeconomic position and health: prospective evidence from the Alameda County Study*. Presented at 114th Annual Meeting of the American Public Health Association, Las Vegas.

Kaplan, G. A., Haan, M. N., Syme, S. L., Miszcynski, M. 1987. Socioeconomic position and health. Invited paper, Carter Center Conference on "Closing the Gap." Supplement to *American Journal of Preventative Medicine* 1987:3(5).

Karasek, R., Baker, D., Marxer, F., Ahlbom, A., Theorell, T. 1981. Job decision latitude, job demands, and cardiovascular disease: a prospective study of Swedish men. *American Journal of Public Health* 71:694–705.

Kasl, S. V., Harburg, E. 1975. Mental health and the urban environment: some doubts and second thoughts. *Journal of Health and Social Behavior* 16:268–282.

Kendrick, B. L. 1980. A spatial environmental and socioeconomic appraisal of cancer in New Zealand. *Social Science and Medicine* 14d:205–214.

Kitagawa, E. M., Hauser, P. M. 1973. *Differential mortality in the United States: a study in socioeconomic epidemiology*. Cambridge, MA: Harvard University Press.

Kohn, M., Schooler, C. 1974. Job conditions and personality: a longitudinal assessment of their reciprocal effects. *American Journal of Sociology* 6:1257–1286.

Koskenvuo, M., Sarna, S., Kaprio, J., Lonnqvist, J. 1979. Cause-specific mortality by marital status and social class in Finland during 1969–1971. *Social Science and Medicine* 13a:691–697.

Kraus, J., Redford, R. J. 1975. Some ecological correlates of perinatal mortality in a metropolis. *Medical Journal of Australia* 2:16–18.

Kristiansén, C. M. 1985. Seat belt legislation and seat belt use: effects on differences related to sex, social class, and smoking. *Accident Analysis and Prevention* 17:75–78.

Kuller, L. H., Seltser, R. 1967. Cerebrovascular disease mortality in Maryland. *American Journal of Epidemiology* 86:442–450.

Kuller, L. H., Cooper, M., Perper, J., Fisher, R. 1973. Myocardial infarction and sudden death in an urban community. *Bulletin of the New York Academy of Medicine* 49(6):532–543.

Lapidus, L., Bengtsson, C. 1986. Socioeconomic factors and physical activity in relation to cardiovascular disease and death: a 12-year follow-up of participants in a population study of women in Gothenburg, Sweden. *British Heart Journal* 55:295–301.

Lave, L. B., Seskin, E. P. 1977. *Air pollution and human health*. Baltimore: The Johns Hopkins University Press for Resources for the Future.

Lawton, P. M., Nahemow, L. 1979. Social areas and the well-being of tenants in housing for the elderly. *Multivariate Behavioral Research* 14:463–484.

Lerner, M. 1986. Cancer mortality differentials in Baltimore, 1959–1961 to 1979–1981. In *Cancer in the economically disadvantaged*. New York: American Cancer Society.

———, Stutz, R. N. 1978. Mortality by socioeconomic status, 1959–1961 and 1969–1971. *Maryland State Medical Journal* (Dec):35–43.

Leren, P., Helgeland, A., Hjermann, I., Holme, I. 1983. The Oslo study: CHD risk factors, socioeconomic influences and intervention. *American Heart Journal* 106:1200–1206.

Lines, D. R. 1977. An Auckland high school health survey. *Australia and New Zealand Journal of Medicine* 7:143–147.

Lipworth, L., Abelin, T., Connelly, R. R. 1970. Socioeconomic factors in the prognosis of cancer patients. *Journal of Chronic Diseases* 23:105–116.

Lloyd, O. L., Smith, G., Lloyd, M. M., Holland, Y., Gailey, F. 1985. Raised mortality from lung cancer and high sex ratios of birth associated with industrial pollution. *British Journal of Industrial Medicine* 42:475–480.

Mack, T. M., Paganini-Hill, A. 1981. Epidemiology of pancreas cancer in Los Angeles. *Cancer* 47:1474–1483.

Mahaffey, K. R., Annest, J. L., Roberts, J., Murphy, R. S. 1982. National estimates of blood lead levels: United States 1976–1980: association with selected demographic and socioeconomic factors. *New England Journal of Medicine* 307:573–579.

Mare, R. D. 1982. Socioeconomic effects on child mortality in the United States. *American Journal of Public Health* 72:539–547.

Markides, K. S., Barnes, D. 1977. A methodological note on the relationship between infant mortality and socioeconomic status with evidence from San Antonio, Texas. *Social Biology* 24:38–44.

Marmot, M. G. 1986. Social inequalities in mortality: the social environment. In Wilkinson, R. G., ed. *Class and health—research and longitudinal data*, pp. 1–20. London: Tavistock Publications.

———, Adelstein, A. M., Robinson, N., Rose, G. A. 1978a.

Changing social-class distribution of heart disease. *British Medical Journal* 2:1109–1112.

Marmot, M. G., Rose, G., Shipley, M., Hamilton, P. J. S. 1978b. Employment grade and coronary heart disease in British civil servants. *Journal of Epidemiology and Community Health* 32:244–249.

Marmot, M. G., Shipley, M. J., Rose, G. 1984. Inequalities in death-specific explanations of a general pattern. *Lancet* (May 5):1003–1006.

McCormick, M. C. 1985. The contribution of low birth weight to infant mortality and childhood morbidity. *New England Journal of Medicine* 312:82–90.

McKeown, T. 1979. *The role of medicine: dream, mirage, or nemesis?* Princeton, NJ: Princeton University Press.

McWhirter, W. R., Smith, H., McWhirter, K. M. 1983. Social class as a prognostic variable in acute lymphoblastic leukemia. *Medical Journal of Australia* 2:319–321.

Medalie, J. H., Papier, C., Herman, J. B. et al. 1974. Diabetes mellitus among 10,000 adult men. I. Five-year incidence and associated variables. *Israel Journal of Medical Sciences* 10:681–697.

Metropolitan Life Insurance Company. 1977. Socioeconomic mortality differentials by leading causes of death. *Metropolitan Life Insurance Company Statistical Bulletin* 58:5–8.

Miller, A. B. 1982. Risk factors from geographic epidemiology for gastrointestinal cancer. *Cancer* 50:2533–2540.

Morgan, M., Chinn, S. 1983. ACORN group, social class, and child health. *Journal of Epidemiology and Community Health* 37:196–203.

Morgenstern, H. 1980. The changing association between social status and coronary heart disease in a rural population. *Social Science and Medicine* 14a:191–201.

Morris, J. N. 1979. Social inequalities undiminished. *Lancet* 87–90.

Moser, K. A., Fox, A. J., Jones, D. R. 1984. Unemployment and mortality in OPCS longitudinal study. *Lancet* 2:1324–1330.

Murrells, T. J., Catford, J. C., Smith, T. M. F., Machin, D. 1985. The use of logit models to investigate social and biolog-

ical factors in infant mortality. II: Stillbirths. *Statistics in Medicine* 4:189–200.

Nagi, M. H., Stockwell, E. G. 1973. Socioeconomic differentials in mortality by cause of death. *Health Services Reports* 88:449–456.

National Academy of Sciences. 1985. *Institute of medicine prevention of low birth weight.* Washington, D.C.: National Academy Press.

National Center for Health Statistics. 1986. *Current estimates from the National Health Interview Survey: United States, 1983. Series 10, No. 154.* Hyattsville, MD.: Department of Health and Human Services.

———. 1985. *Health characteristics according to family and personal income. Series 10, No. 147.* Hyattsville, MD.: Department of Health and Human Services.

———. 1986. *Types of injuries and impairments due to injuries. Series 10, No. 159.* Hyattsville, MD.: Department of Health and Human Services.

———. 1986. *Disability days: United States, 1983. Series 10, No. 158.* Hyattsville, MD.: Department of Health and Human Services.

———. 1972–73. *Vital and Health Statistics. Series 10, No. 94 and No. 109.* Hyattsville, MD.: Department of Health and Human Services.

Nayha, S. 1977. Social group and mortality in Finland. *British Journal of Preventive and Social Medicine* 31:231–237.

Notkola, V., Punsar, S., Karvonen, M. J., Haapakoski, J. 1985. Socioeconomic conditions in childhood and mortality and morbidity caused by coronary heart disease in adulthood in rural Finland. *Social Science and Medicine* 21:517–523.

Pamuck, E. R. 1985. Social class inequality in mortality from 1921 to 1972 in England and Wales. *Population Studies* 29:17–31.

Papiernik, E., Maine, D., Rush, D., Richard, A. 1985. Prenatal care and the prevention of preterm delivery. *International Journal of Gynaecology and Obstetrics* 23:427–433.

Pearce, N. E., Davis, P. B., Smith, A. H., Foster, F. H. 1983a.

Mortality and social class in New Zealand I: overall male mortality. *New Zealand Medical Journal* 96:281–285.

———. 1983b. Mortality and social class in New Zealand II: male mortality by major disease groupings. *New Zealand Medical Journal* 96:711–716.

———. 1985. Social class, ethnic group, and male mortality in New Zealand, 1974–1978. *Journal of Epidemiology and Community Health* 39:9–14.

Phillipp, R., Hughes, A. 1985. Population sampling for studies of long-term residential exposure to air-borne lead: migration problems identified from electoral registrations. *Public Health London* 9:302–306.

Pirkle, J. L., Schwartz, J., et al. 1985. The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. *American Journal of Epidemiology* 121:246–258.

Rantakallio, P. 1979. Social background of mothers who smoke during pregnancy and influence of these factors on the offspring. *Social Science and Medicine* 13a:421–429.

Rodin, J. 1986. Aging and health: effects of the sense of control. *Science* 233:1271–1276.

Rose, G., Marmot, M. G. 1981. Social class and coronary heart disease. *British Heart Journal* 45:13–19.

Rush, D., Cassano, P. 1983. Relationship of cigarette smoking and social class to birth weight and perinatal mortality among all births in Britain, 5–11 April 1970. *Journal of Epidemiology and Community Health* 37:249–255.

Salonen, J. T. 1982. Socioeconomic status and risk of cancer, cerebral stroke, and death due to coronary heart disease and any disease: a longitudinal study in eastern Finland. *Journal of Epidemiology and Community Health* 36:294–297.

Savage, D., Lindenbaum, J., Van Ryzin, J., Struening, E., Garrett, T. J. 1984. Race, poverty, and survival in multiple myeloma. *Cancer* 54:3085–3094.

Schwartz, J. 1985. *The relationship between blood lead levels and blood pressure.* Washington, D.C.: EPA.

Shin, E. H. 1975. Economic and social correlates of infant mortality: a cross-sectional and longitudinal analysis of 63 selected countries. *Social Biology* 22:315–325.

Simpson, S. P. 1984. Causal analysis of infant deaths in Hawaii. *American Journal of Epidemiology* 119:1024–1029.

Smith, J. P., Day, T. G. 1979. Review of ovarian cancer at the University of Texas Systems Cancer Center, M. D. Anderson hospital and tumor institute. *American Journal of Obstetrics and Gynecology* 136:984–993.

Spiers, P. S., Schlesselman, J. J., Wright, S. G. 1974. Sudden infant death syndrome in the United States: a study of geographic and other variables. *American Journal of Epidemiology* 100:380–389.

Stamler, R., Hardy, R. J., Payne, G. H., et al. 1987. Education level and five-year all-cause mortality in the hypertension detection and follow-up program. *Hypertension* 9:641–646.

Starfield, B. 1982. Family income, ill health and medical care of U.S. children. *Journal of Public Health Policy* 3:244–259.

———. 1985. *The effectiveness of medical care: validating clinical wisdom*. Baltimore: The Johns Hopkins University Press.

———, Budetti, P. P. 1985. Child health status and risk factors. *Health Services Research* 19(6):817–886.

Steinhorn, S. C., Myers, M. H., Hankey, B. F., Pelham, V. F. 1986. Factors associated with survival differences between black women and white women with cancer of the uterine corpus. *American Journal of Epidemiology* 124:85–93.

Stern, J. 1983. Social mobility and the interpretation of social class mortality differentials. *Journal of Social Policy* 12:27–49.

Sunderland, R. 1984. Dying young in traffic. *Archives of Disease in Childhood* 59:754–757.

Susser, M., Watson, W., Hopper, K. 1985. *Sociology in medicine*. New York: Oxford University Press.

Syme, S. L., Berkman, L. F. 1976. Social class, susceptibility, and sickness. *American Journal of Epidemiology* 104:1–8.

Taylor, E. M., Emery, J. L. 1983. Family and community factors associated with infant deaths that might be preventable. *British Medical Journal* 287:871–874.

Theorell, T., Rahe, R. H. 1972. Behavior and life satisfactions characteristic of Swedish subjects with myocardial infarction. *Journal of Chronic Diseases* 25:139–147.

Townsend, P., Davidson, N., eds. 1982. *Inequalities in health: the Black Report*. Harmondsworth, England: Penguin Books.

U.S. Surgeon General. 1985. The health consequences of smoking: cancer and chronic lung disease in the workplace, a report of the Surgeon General. Rockville, MD: Department of Health and Human Services.

van den Berg, B. J., Oechsli, F. W. 1984. Prematurity. In Bracken, M. B., ed. *Perinatal epidemiology*, pp. 69–85. New York: Oxford University Press.

Wadsworth, M. E. J. 1986. Serious illness in childhood and its association with later-life achievement. In Wilkinson, R. G., ed. *Class and health—research and longitudinal data*, pp. 50–74. London: Tavistock Publications.

Wegner, E. L., Kolonel, L. N., Nomura, A. M. Y., Lee, J. 1982. Racial and socioeconomic status differences in survival of colorectal cancer patients in Hawaii. *Cancer* 49:2208–2216.

West, R. R. 1977. Geographical variation in mortality from ischaemic heart disease in England and Wales. *British Journal of Preventative and Social Medicine* 31:245–250.

Wilkinson, R. G. 1986. Socioeconomic differences in mortality: interpreting the data on their size and trends. In Wilkinson, R. G., ed. *Class and health—research and longitudinal data*, pp. 1–20. London: Tavistock Publications.

World Health Organization. 1985. *A WHO report on social and biological effect of perinatal mortality*. Budapest, Hungary: WHO.

Wright, N. H. 1975. Family planning and infant mortality rate decline in the United States. *American Journal of Epidemiology* 101:182–187.

Yeracaris, C. A., Kim, J. H. 1978. Socioeconomic differentials in selected causes of death. *American Journal of Public Health* 68:342–351.

Chartbook

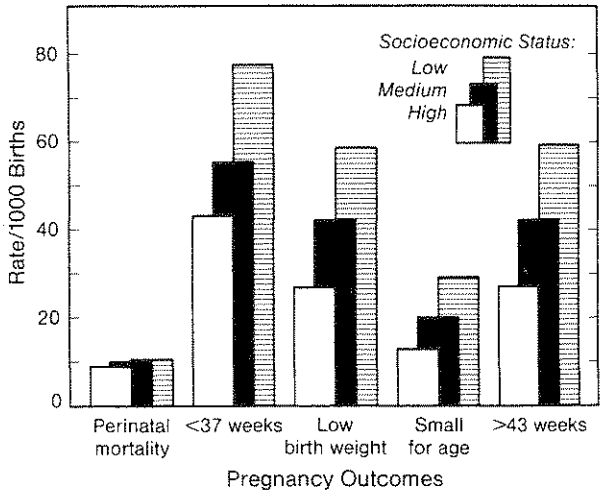


FIGURE 1. Pregnancy Outcomes and Socioeconomic Status, Sweden, 1976–1977. *Source: adapted from Ericson, et al. (1984).*

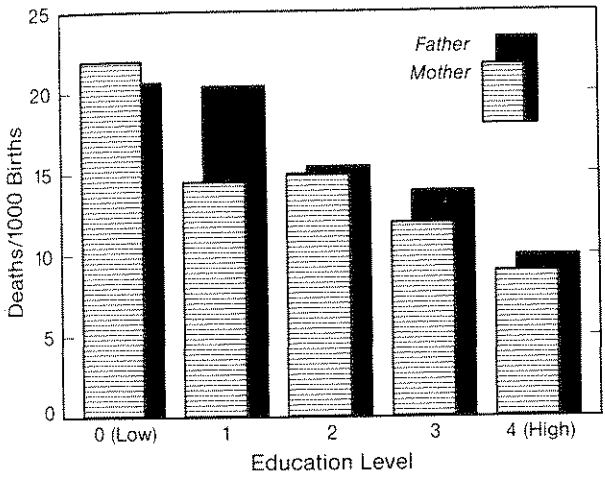


FIGURE 2. Perinatal Death Rates by Parental Education for Six States in 1973, United States. *Source: adapted from World Health Organization (1985).*

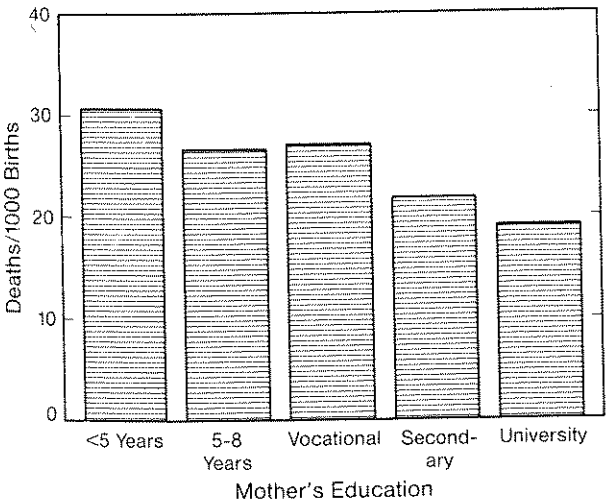


FIGURE 3. Perinatal Mortality and Mother's Education in Finland. *Source: adapted from Rantakallio (1979).*

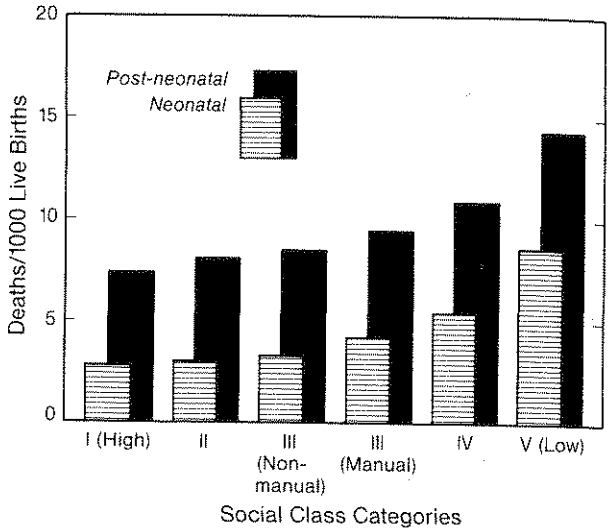


FIGURE 4. Infant Mortality Rates, by Social Class of Patients, England and Wales, 1975–1976. *Source: adapted from Morris (1979).*

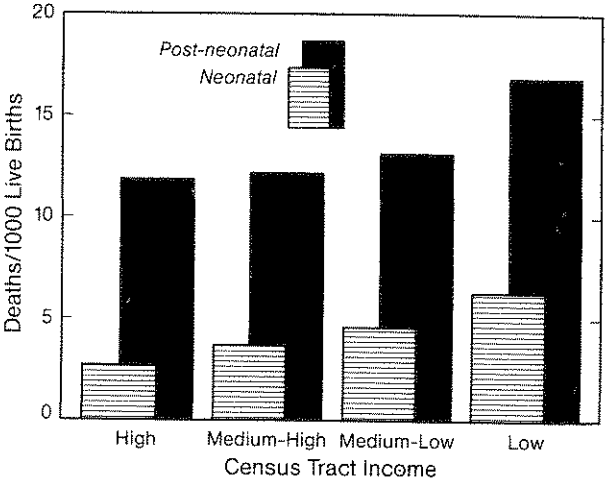


FIGURE 5. Neonatal and Post-Neonatal Mortality Rates by Income of Census Tract of Residence, San Antonio, Texas, 1970–1974. *Source: adapted from Markides and Barnes (1977).*

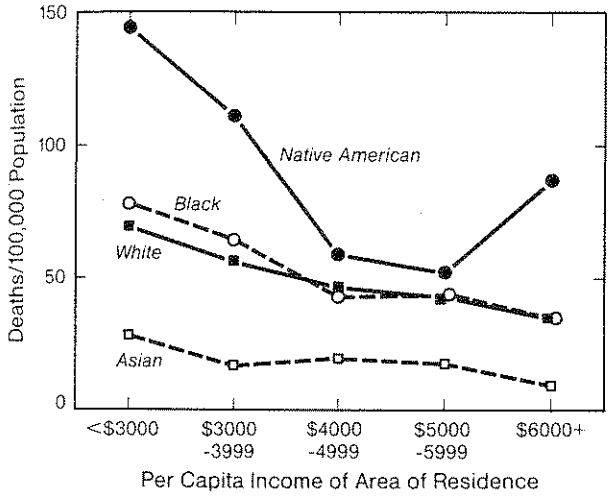


FIGURE 6. Death Rates from Unintentional Injury by Per Capita Income of Area of Residence and Race, United States, 1977–1979. *Source: adapted from Baker, et al. (1984).*

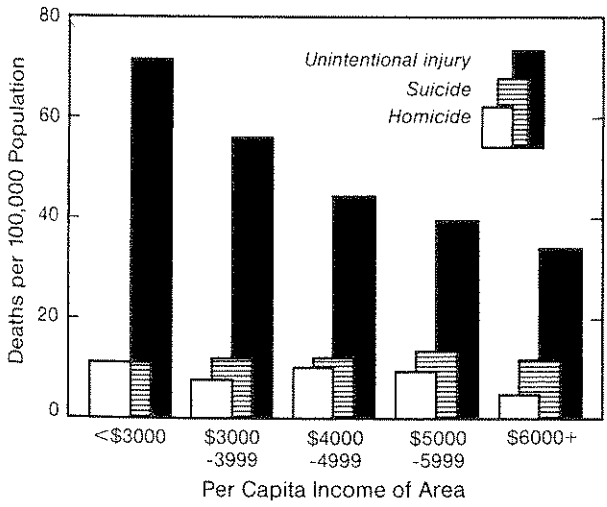


FIGURE 7. Death Rates from Unintentional Injury, Suicide, and Homicide by Per Capita Income of Area of Residence, United States, 1977–1979. *Source: adapted from Baker, et al. (1984).*

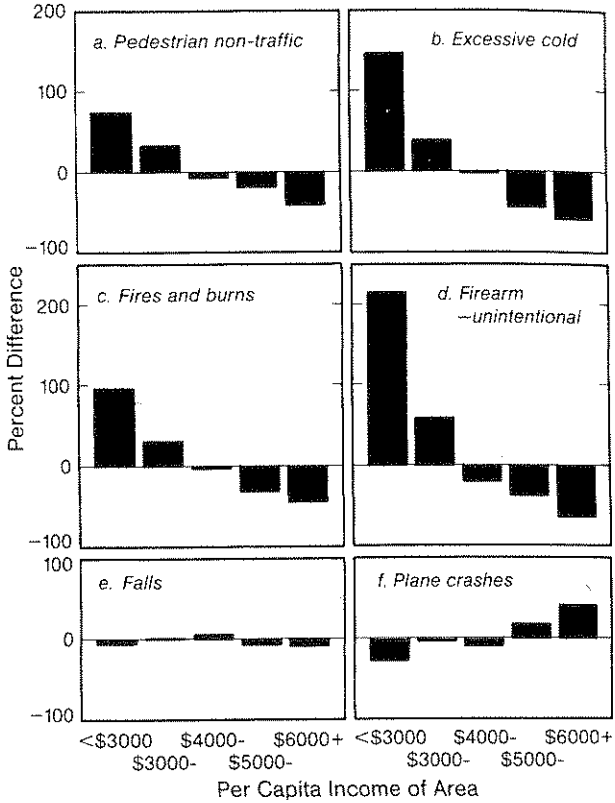


FIGURE 8. Percent Difference from the Average Death Rates from Unintentional Injury by Per Capita Income of Area of Residence and Cause, 1977–1979. *Source: adapted from Baker, et al. (1984).*

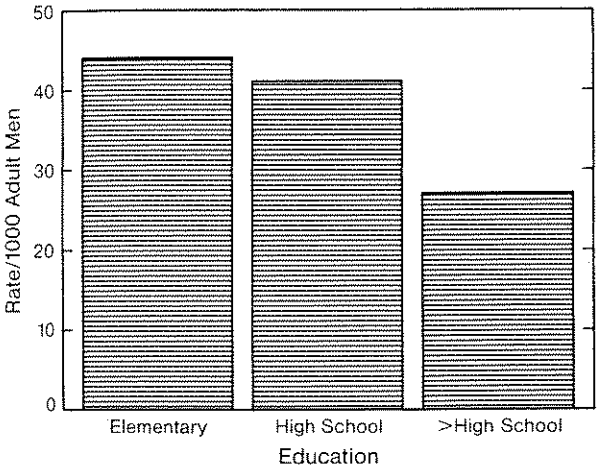


FIGURE 9. Education and 9-Year Incidence of Diabetes Mellitus in Adult Men. *Source: adapted from Medalie, et al. (1974).*

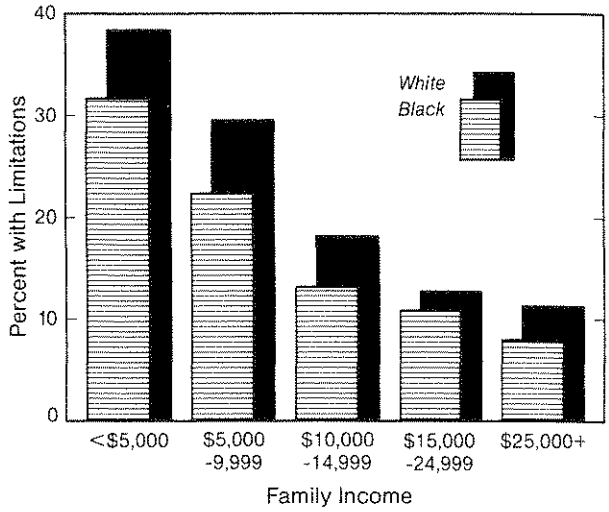


FIGURE 10. Family Income, Race and Activity Limitations due to Chronic Conditions, United States, 1979–1980. *Source: adapted from National Center for Health Statistics (1985).*

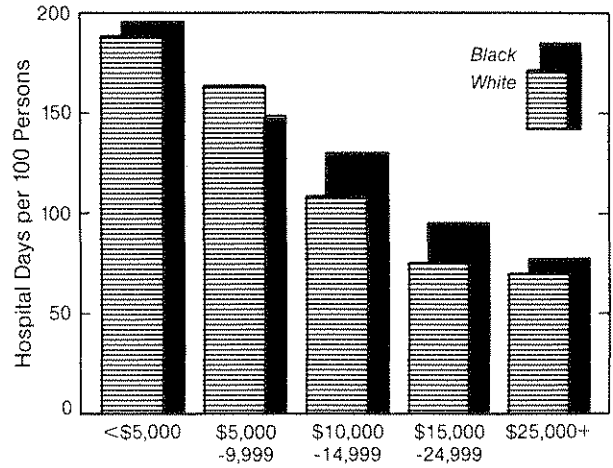


FIGURE 11. Family Income, Race and Short-Stay Hospital Days, United States, 1979–1980. *Source: adapted from National Center for Health Statistics (1985).*

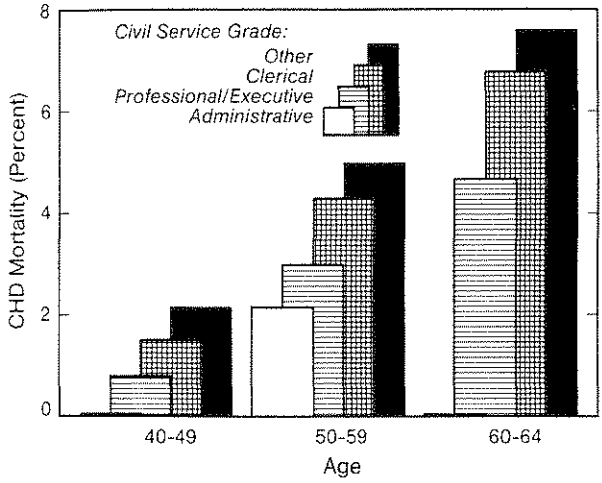


FIGURE 12. Coronary Heart Disease Mortality Rates in 7½ Years by Civil Service Grade and Age for British Civil Servants. *Source: adapted from Marmot, et al. (1978).*

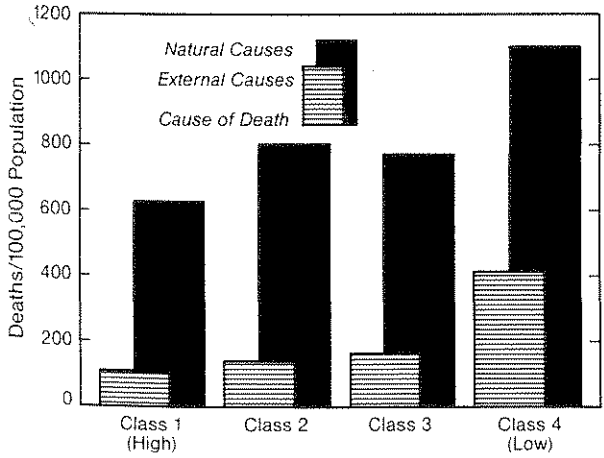


FIGURE 13. Social Class and Mortality by Natural and External Causes in Finland, 1969–1971. *Source: adapted from Koskenvuo, et al. (1979).*

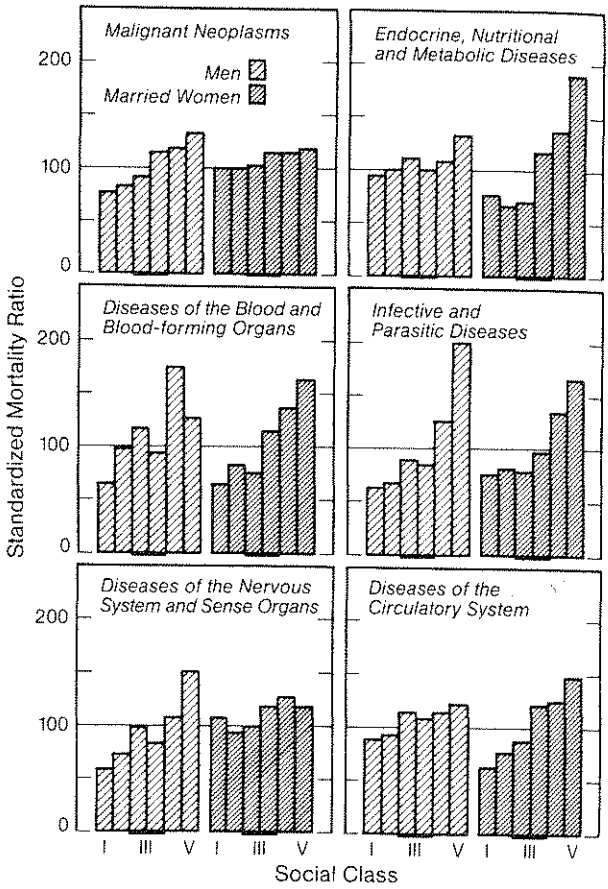
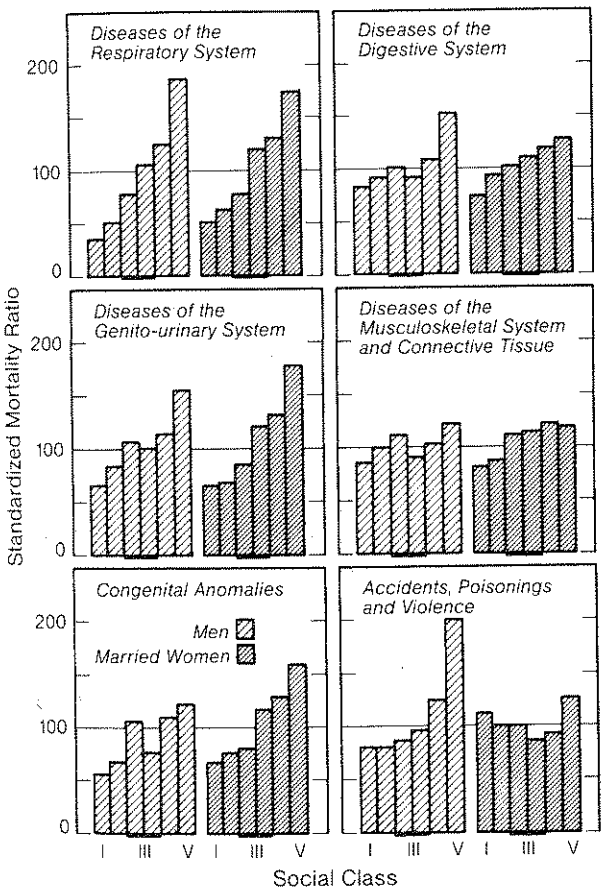


FIGURE 14. Occupation Class and Mortality from Several Causes in Adult Men and Married Women by Husband's Occupational Grade. *Source: Susser (1985).*


Diseases of the Respiratory System
Diseases of the Digestive System
Diseases of the Genito-urinary System
Diseases of the Musculoskeletal System and Connective Tissue
Congenital Anomalies
Accidents, Poisonings and Violence
Men
Married Women
Standardized Mortality Ratio
0
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I III V I III V
Social Class

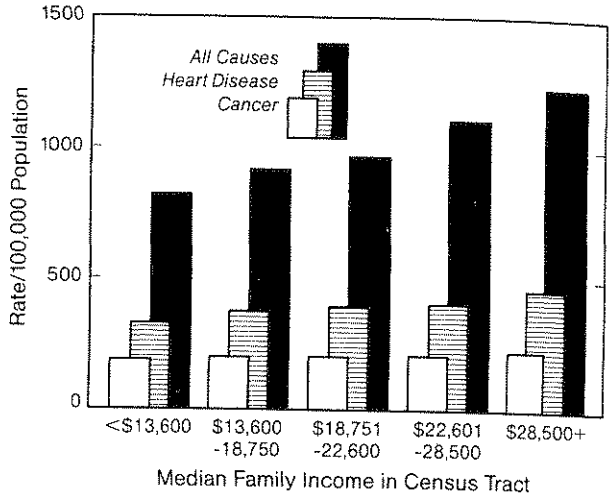

FIGURE 15. Mortality from All Causes, Heart Disease, and Cancer, for Males, by Median Family Income of Census Tract of Residence, Los Angeles, 1979–1981. *Source: adapted from Frerichs, et al. (1984).*

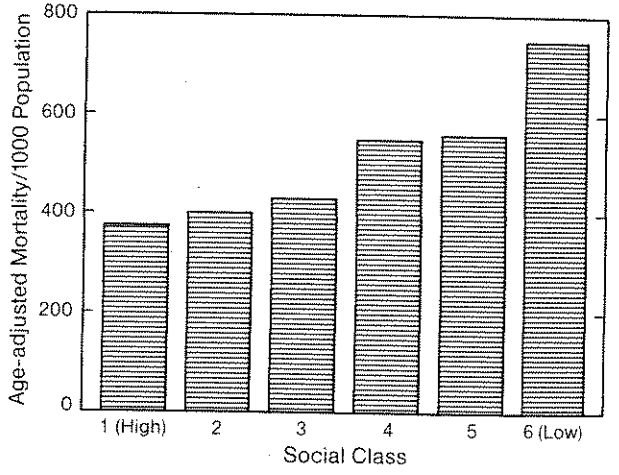

FIGURE 16. Mortality from All Causes for New Zealand Males by Social Class. *Source: adapted from Pearce, et al. (1983a).*

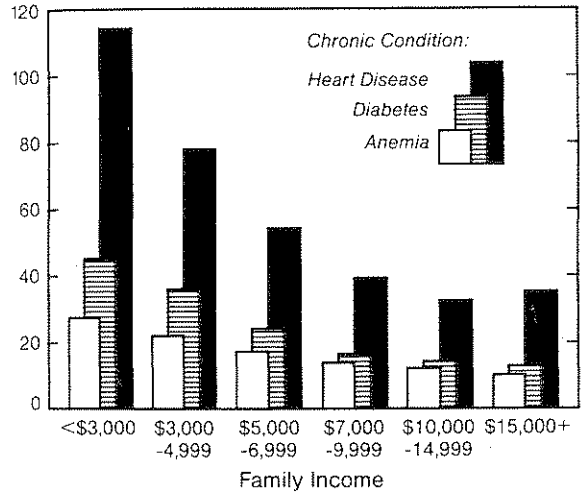

FIGURE 17. Annual Income and the Prevalence of Selected Chronic Diseases, United States, 1972–1973. *Source: adapted from National Center for Health Statistics (1972, 1973).*

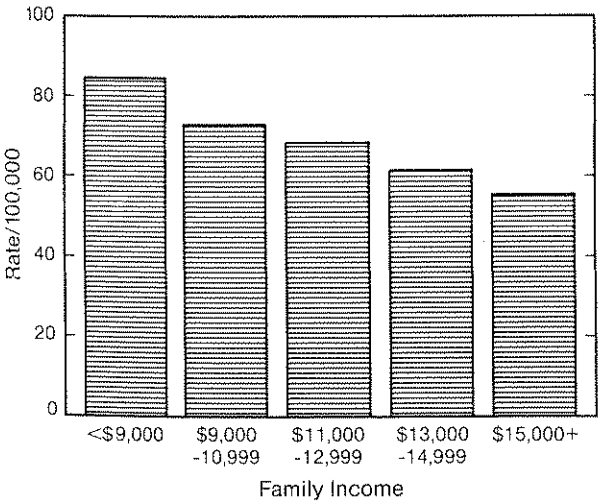

FIGURE 18. Lung Cancer Incidence and Family Income, Third National Cancer Study, 1969–1971. *Source: adapted from Devesa and Diamond (1980).*

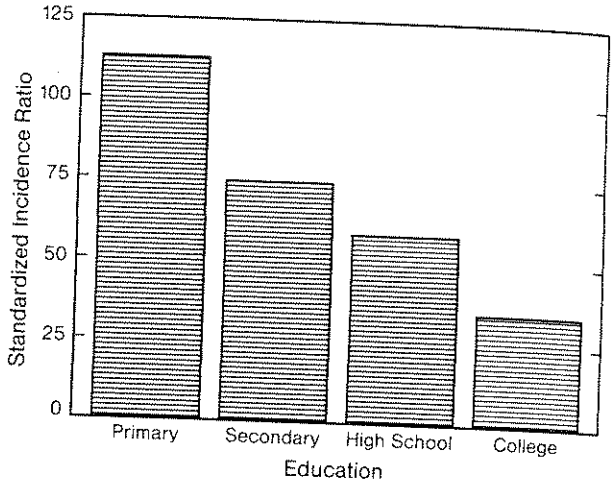

FIGURE 19. Cervical Cancer Incidence and Education in Finland, 1971–1975. *Source: adapted from Hakama, et al. (1982).*

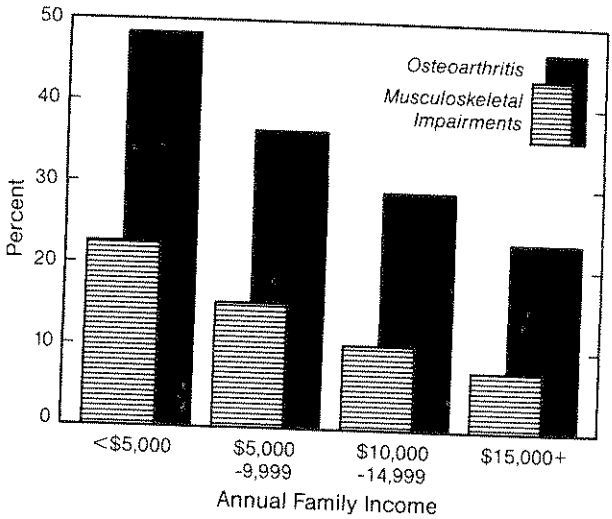

FIGURE 20. Prevalence of Musculoskeletal Impairments and Osteoarthritis by Annual Family Income, National Health and Nutrition Examination Study, 1971–1975. *Source: adapted from Cunningham and Kelsey (1984).*

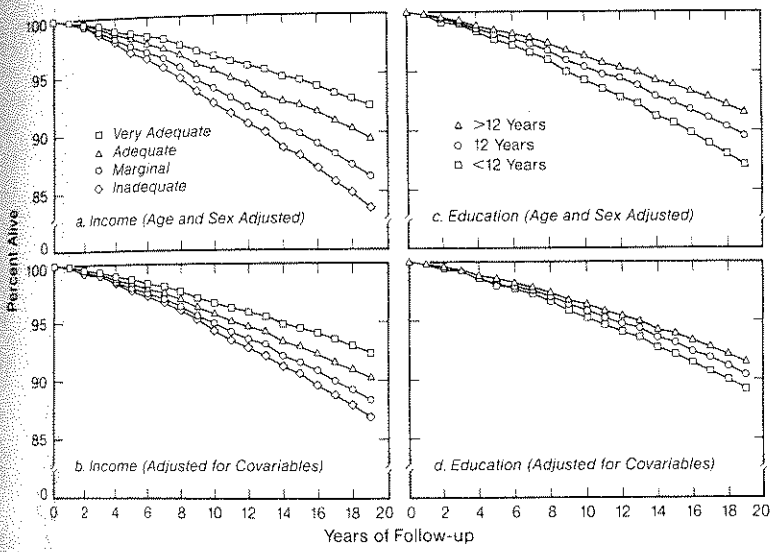

FIGURE 21. Age and Sex-Adjusted 19-year Mortality by Education and Income, with and without Adjustment for Race, Smoking, Alcohol Consumption, Leisure-time Physical Activity, Relative Weight, Sleep Habits, and Prevalence of Chronic Conditions (Hypertension, Heart Trouble, Cancer, Diabetes), Alameda County Study. *Source: adapted from Kaplan, et al. (1986).*

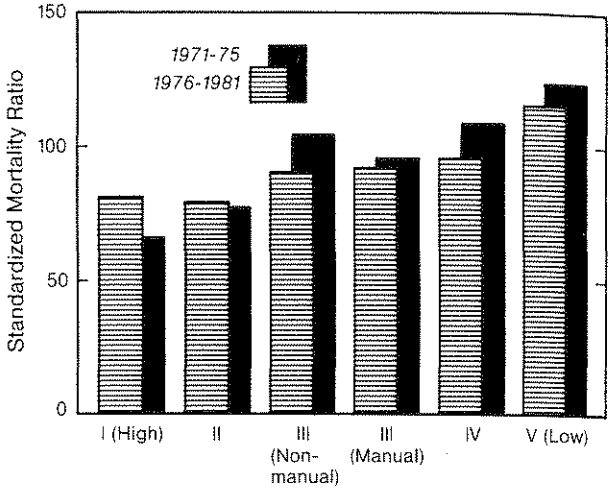

FIGURE 22. Social class and Mortality from All Causes, England and Wales, 1971–1975 and 1976–1981. *Source: adapted from Fox, et al. (1985).*

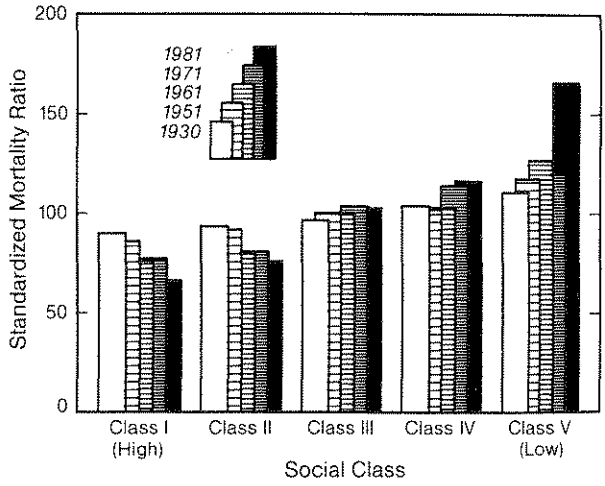

FIGURE 23. Trends in Mortality from All Causes by Social Class, England and Wales, 1931–1981. *Source: adapted from Wilkinson (1986).*

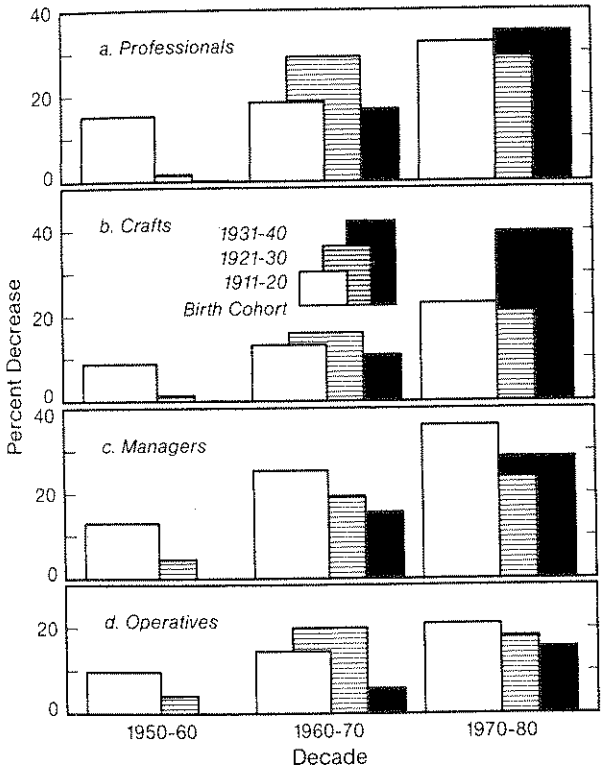

FIGURE 24. Changes in Smoking Rates by Birth Cohort, Year, and Occupation Class. *Source: adapted from Carstairs (1981).*

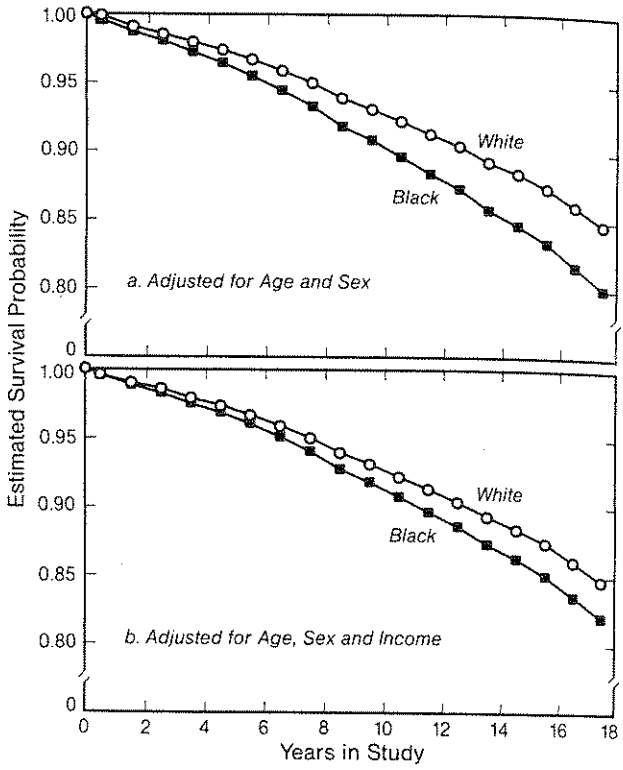

FIGURE 25. Age and Sex-Adjusted 18-year Survival, with and without Adjustment for Income, for Blacks and Whites, Alameda County Study. *Source: adapted from Haan and Kaplan (1985).*

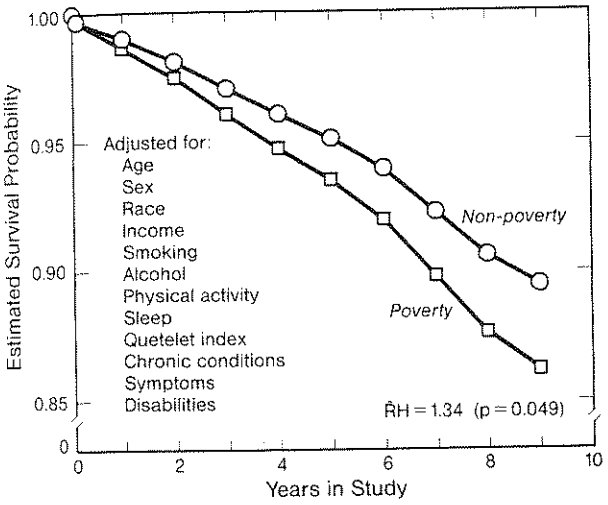

FIGURE 26. 9-year Survival from All Causes by Poverty Area Residence with Adjustment for Risk Factors, Alameda County Study. *Source: adapted from Haan, et al. (1987).*