

Social Inequalities in Health: Disentangling the Underlying Mechanisms

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I gratefully acknowledge support from the Behavioral and Social Research Program of the National Institute on Aging (grant R01-AG16790) and the National Institute of Child Health and Human Development (P30-HD32030). I would also like to thank Maryann Belanger for her tireless assistance in tracking down bibliographic materials and Burt Singer, Anne Pebley, Angus Deaton, and Maxine Weinstein for their advice and comments.

Forthcoming: Conference Proceedings of the seminar on "Demography and Epidemiology: Frontiers in Population Health and Aging," Georgetown University, Washington D.C. February 9-10, 2001. New York Academy of Sciences.

Abstract

Differentials in health and longevity by socioeconomic status and by the nature of social relationships have been found in innumerable studies in the social and medical sciences. Three categories of explanations for the observed patterns have been proposed: causal mechanisms through which the social environment affects health status or the risk of dying; selection or reverse causal pathways whereby a person's health status affects their social position; and artifactual mechanisms such as measurement error. The general consensus among researchers is that the observed disparities in health are driven largely by a complex set of causal processes rather than by selection or by artifactual mechanisms. This paper explores the set of arguments and strategies that researchers have used to arrive at this conclusion. As part of this undertaking, we assess whether inferences regarding the minor contribution of selection to the overall association between social factors and health are justifiable. In addition, we identify current avenues of research that are providing new insights into the causal pathways linking social factors and health.

Keywords: social inequalities, socioeconomic status, health, causal, selection

Differentials in health and longevity by socioeconomic status and by the nature of social relationships have been identified in a myriad of studies, some dating as far back as the 1800s. More so than most research areas, this field has actively engaged researchers from many disciplines, including sociology, psychology, economics, demography, epidemiology, biology, and medicine. They have found that, with few exceptions, persons of higher socioeconomic status and persons who are more socially integrated experience lower rates of morbidity and mortality than their respective counterparts.¹ These associations have been identified across time, place, gender and age. Moreover, these inequalities in health are apparent for a broad set of outcome variables (such as self-perceived health, most illnesses, disability, mortality, and psychological well-being) and for alternative measures of social position (such as income, other measures of wealth, education, occupation, level of social integration, and marital status).

Findings from many studies have challenged the notion that the association between SES and health is due largely to the adversities associated with poverty. Instead of revealing a threshold effect, these associations have emerged at every level of the social hierarchy (e.g., the highest social class was shown to be healthier and have lower risks of dying than the next highest group), generating what researchers now refer to as a *social gradient* in health. For example, the widely cited Whitehall Study of British Civil Servants demonstrated that the mortality gradient was present even within a relatively homogeneous group: civil servants in one type of occupation (stable office jobs) and one

¹ There are exceptions to these patterns. For example, higher income is associated with higher rates of self-perceived morbidity in some developing countries (Murray et al., 1992). These patterns may be the consequence of differential reporting by SES. In industrialized countries, the gradient may be absent or reversed for some forms of cancer (Adler and Ostrove, 1999).

geographical location (London), but in different grades of employment (Marmot et al. 1995). Monotonic (although not necessarily linear) relationships between SES (measured by occupational class, income or education level)² and health status have been established for other outcomes, including the infant mortality rate and the prevalence of major chronic diseases (Alder and Ostrove, 1999).

Three categories of explanations for the observed patterns have been proposed. One set of hypotheses relates to a set of *causal* mechanisms through which socioeconomic status and social relationships potentially affect health status and the risk of dying. A second type of explanation, sometimes referred to as *selection* or *reverse causation*, refers to a set of pathways whereby unhealthy individuals may reduce their social position or become socially more isolated as a consequence of their inferior health status. A third, less frequently invoked explanation encompasses artifactual mechanisms, such as measurement error.

The general consensus among researchers from different disciplines is that the observed disparities in health are driven largely (although not entirely) by a complex set of causal processes, rather than by selection or artifactual mechanisms. In terms of SES and health, the claim has been that, although there is some evidence of downward social mobility among persons in poor health, this selection process makes only a minor contribution to the overall association between SES and a wide range of health indicators (Davey Smith et al., 1994; Marmot et al., 1995; Haan et al, 1989; Wilkinson, 1986; Macintyre, 1997; Hertzman, 1999; Adler and Ostrove, 1999). Similarly, scientists have argued that selection processes have a negligible impact on the observed health

² Results are less clearcut in studies that employ multiple measures of social position. For example, Fuchs (1993) argues that when health is modeled as a function of both income and schooling, the latter variable

differentials in social support, social integration and marital status (Umberson, 1987; Ross et al., 1990). Artifactual mechanisms (e.g., errors of measurement such as undercounts in the census, “numerator-denominator” problems such as inconsistencies in reports between registration and census data, or inappropriate measures of mortality or SES) are also not considered to be a powerful explanation of the observed associations (e.g., Davey Smith et al., 1990; Pappas et al., 1993).

The objective of this paper is to explore the set of arguments and strategies that researchers have used to arrive at these conclusions. Our focus here is on how social and medical scientists have attempted to disentangle the many pathways underlying the observed social inequalities in health. As part of this undertaking, we assess whether inferences regarding the small influence of selection are justifiable. In addition, we identify current avenues of research that are providing new insights into the causal pathways linking social factors and health.

We exclude artifactual explanations from this discussion for two reasons. First, the specific types of concerns in this category vary considerably from study to study, as a function of the data and measures used. Second and more importantly, given the increasing reliance on longitudinal surveys rather than cross-sectional data during the past two decades, these concerns have become progressively less salient. Many of these methodological problems stem from the use of two, potentially incomparable sources to construct the relevant rates (e.g., deaths from a registration system and exposure from a census) and the absence of adequate and accurate measures in the data sources that researchers relied on in the past. Indeed, because relatively few researchers studying social inequalities in health during the past decade have relied on cross-sectional data,

dominates, sometimes leading to a *negative* association between health and income.

particularly census data, we focus most of this paper on studies designed to identify selection or causal mechanisms from longitudinal information.³

Longitudinal data take various forms, primarily (1) the linking of administrative or clinical records, census data, or a single-round survey with information obtained at a later time point (e.g., death records); and (2) prospective or follow-up surveys in which surviving respondents are reinterviewed at periodic intervals. These data, particularly the latter type, have obvious advantages over cross-sectional information since they permit the researcher to obtain measures of the social environment that predate measures of health outcomes and to assess the stability of both sets of measures over time. Most longitudinal data sets used in studies of SES and health are based in Western populations, notably Britain, other European countries and the U.S. Among these, three investigations in Britain have been used extensively to study social inequalities in health: (1) two Whitehall studies, which have been tracking British civil servants since 1967 and 1985-88, respectively; (2) the Office of Population Censuses and Surveys (OPCS) longitudinal survey, which links data from the 1971 and 1981 Censuses to other sources, including mortality and cancer registration data; and (3) three cohort studies, which have been following births that occurred in England, Scotland and Wales during one-week periods in 1946, 1958, and 1970.

In much of the ensuing discussion, we follow recent convention and use such terms as *social class*, *socioeconomic status* (SES), and *social status* interchangeably to refer to the set of measures that researchers use most often to operationalize these concepts (Liberatos et al., 1988). Many of the seminal British studies on social

³ The deficiencies of cross-sectional data for identifying the selection and causal mechanisms that underlie social inequalities in health were recognized more than a century ago (Fox et al., 1985).

inequalities in health relied on an occupational scale, developed in 1911 and revised every decade, that assigns the occupation of the head of household to one of five classes ranging from professional to unskilled. More recently, a variety of occupational scores have been developed based on the education level, income, and/or prestige associated with a wide range of occupations. In addition to occupation, education and income have been the most frequently used measures in this area of research (Liberatos et al., 1988). Unfortunately, the lack of a clear theoretical formulation or even definition of social class in many studies has sometimes prevented researchers from recognizing that the magnitude of the observed social inequalities and their underlying mechanisms may vary according to the specific measure of social class employed.⁴ In the discussion below, we emphasize some of the shortcomings of existing research that stem from the vague conceptualization of social class.

Identifying selection mechanisms

Health or health-related selection has been referred to by a variety of terms in the literature, including reverse causation, health-related social mobility, occupational or social drift, social selection, and discrimination on the basis of health (Goldman, forthcoming; West, 1991). Some of these terms reflect a direct interest in selection as a social phenomenon, while others reflect the view that selection is a nuisance mechanism that must be confronted before identifying causal processes of interest. Regardless of the perspective, researchers have recognized that different types of selection mechanisms

⁴ This variation results not only from the different economic, political and cultural dimensions that comprise the main indicators of social class, but also from varying degrees of measurement error.

may be operative, largely reflecting the nature of the criteria used to allocate individuals into different social categories and the ages at which this sorting occurs. First, selection may be direct or indirect: i.e., it may operate on health or illness status itself (e.g., the presence of a disease or disability) or on the basis of characteristics or background factors that are related to both health and social position in adulthood (such as height). Second, social mobility resulting from poor health may occur within one (intra-generational) or two generations (inter-generational).

Scientists have used different approaches for assessing the potential impact of these selection processes. To facilitate the presentation, we categorize these approaches as (1) inferences derived from irrelevant or no evidence; (2) inferences drawn from broad, simple patterns in cross-sectional or longitudinal data; and (3) inferences based on efforts to directly identify or measure health-related social mobility from longitudinal data. These three strategies are described in more detail below.

Inferences based on scant evidence

The first category includes studies in which selection has been eliminated as a potentially important mechanism in a fairly dismissive manner. With regard to both the SES-health link and the marital status-health association, some social scientists have simply noted that the observed differentials are too large to be accounted for by selection processes alone (West, 1991). Many others have rejected the selection argument on the basis of a finding suggesting that causal processes are operative – often by identifying a measure of SES that cannot be altered by the health measure under study. For example,

the finding that health differentials are present when individuals are categorized by education (and the fact that persons cannot drift downwards in terms of their own educational achievement) has been used to minimize the importance of selection processes in producing SES differences in general (Hertzman, 1999; Adler and Ostrove, 1999).⁵ Similar arguments are based on evidence that the inequalities persist when SES is measured long before the period of interest (e.g., using SES at birth or childhood to study the SES-health link among adults; using SES during working ages to study the association among retired people); when the analysis is extended to include the health status of family members; or when the health measure of interest is the incidence (i.e., onset) rather than the presence of disease (Marmot et al., 1995; Haan et al., 1989).

There are at least two problems with these arguments. First, even under the assumption that these findings confirm the existence of causal pathways, they reveal little about the potential role of selection processes. Selection and causal mechanisms should not be viewed as competing explanations in the sense that the presence of one precludes the other. Rather, to the extent that selection operates, it must do so *alongside* a complex set of causal pathways. Second, given that (1) health selection leads to *inter*-generational mobility as well as downward drift; (2) health selection operates throughout the life cycle probably beginning prior to birth; and (3) child health reveals strong associations with adult health, evidence that appears to support causal explanations may in fact reflect subtle modes of selection.

⁵ As pointed out to me by Deaton (2001), this argument also ignores the fact that selectively healthy persons can “drift upwards” in educational achievement.

Inferences based on simple, aggregate patterns

The second category of inference reflects deductions derived from simple, aggregate patterns of the observed health differentials, based on either cross-sectional or longitudinal data. The rationale underlying this approach derives from the investigator's hypothesis that the presence of selection would lead to a pattern that is distinct from the pattern that would result in the absence of selection. The most common examples relate to patterns of the gradient with age and appear in both the SES-health and the marital status-health literatures. Specifically, researchers have used information solely about whether the gradient shows a weakening or strengthening over the life cycle as evidence about the importance of selection. An immediate indicator of the potential limitations of this research strategy is that, in the case of the SES-health link, the underlying hypotheses have not even been consistent. For example, Fox et al. (1985) argue that selection effects would be expected to wear off with time, if they reflected only health at the time of selection, and hence lead to a shallower gradient at older ages. In contrast, Marmot (1992) and the authors of the *Black Report* (Black et al., 1982) suggest that social mobility (i.e., selection) should lead to widening differentials – i.e., a steeper gradient with age – as individuals accumulate more time to experience these effects. The former of these arguments closely resembles those advanced by demographers (Livi-Bacci, 1985; Zalokar, 1960; Sheps, 1961). They contend that if selection processes – in this case, the sorting of the healthiest individuals into marriage – drive the health advantage

of married persons, then we should expect to find the health gradient⁶ decreasing after an age when marriage rates fall to low levels. The underlying logic is that, once marriages cease, both the single and married populations should “improve” their composition as their frailest members die and the death rates of the two groups should converge toward one another.

Similar types of inferences have been based on other types of patterns. Demographers and other social scientists have judged the importance of selection on the basis of (1) the strength of the association between the size of the gradient (i.e., the levels of excess mortality of single persons relative to the married) and the relative size of the single population (i.e., the proportion of the population that never marries; Fuchs, 1974; Livi-Bacci, 1985; Kisker and Goldman, 1987); and (2) variations in the gradient by causes of death (Gove, 1973; Litwak and Messeri, 1989). Psychologists have used a similar approach to explore the association between social class and mental illness. Dohrenwend et al. (1992) maintain that a straightforward comparison of the social class gradient for schizophrenia between two ethnic groups – one of which is disadvantaged vis-à-vis the other – permits the analyst to assess the importance of selection processes in producing the observed negative association between social class and schizophrenia.⁷

Although enticing in their simplicity, these arguments are seriously flawed. The weaknesses have been revealed by mathematical simulation models in which differences in health or survival status by social groups are produced entirely by selection processes (Goldman, 1993a; Goldman, 1994). These models have demonstrated that, even under

⁶ In making these arguments, most demographers have measured the gradient in terms of the relative mortality ratio: the death rate of the never-married population (for a given age group and sex) divided by the corresponding death rate of the married or ever-married population.

the simplest scenarios possible (e.g., two health statuses and two social positions), the range of patterns of the gradient that can result from selection are much broader than researchers have speculated. For example, although sorting of the healthiest individuals into marriage can result in declining gradients after marriageable age, as hypothesized, it can also lead to flat gradients or gradients that increase well beyond marriageable age. While the availability of longitudinal data about the nature of social mobility and health differentials among groups could permit researchers to narrow the range of plausible patterns of the gradient, such data would also obviate the need for the types of indirect inferential approaches described above.

Inferences based on direct measures of selection

The third type of approach used to examine the important of selection mechanisms encompasses a broad range of analyses that *directly* model social mobility related to health, typically from longitudinal surveys. Analyses focusing on identifying or measuring selection effects from these data vary considerably from one another not only in terms of the underlying data set but also the measures of health status and social status used. Moreover, some focus directly on measures of health status, such as morbidity or survival, whereas others, in their pursuit of indirect forms of social mobility, examine characteristics related to health – most frequently height, but also physical attractiveness, health-related behaviors, and background characteristics such as education and parenting behaviors. Studies also vary as to the underlying time (or age) reference pertaining to

⁷ Dohrenwend et al. (1992) argue that if causal factors are operative, then the socially disadvantaged group should experience higher rates of mental disorder than the advantaged group at every level of SES. They

social mobility. West (1991) notes that earlier research focused largely on occupational drift among adults as a social indicator and paid relatively little attention to potential effects of health among children and young adults that may cause their achieved social class to differ from their social class of origin. As a consequence, West argues, some studies have rejected the importance of selection without paying any attention to evidence of inter-generational mobility. This concern is less relevant today, because recent studies have attempted to examine both inter-generational and intra-generational mobility. For example, Power and Matthews (1998) use data from the 1958 British cohort study to examine the relationship between a set of direct and indirect indicators of health status and social class position at three points in the life cycle (birth, age 23, and age 33).

Nevertheless, despite continued efforts to measure selection from high quality longitudinal data, the bottom line today remains much the same as it did a decade or more ago, at least among epidemiologists. Wilkinson (1999, p. 48) notes that “the accumulated research evidence at the end of the 1980s seemed to show... that social selection – or reverse causality – made only a minor contribution to health inequalities,” an opinion also expressed by Wadsworth (1986), Fox et al, (1985), and Haan et al. (1989). More recent studies present the same viewpoint, i.e., that, while there is evidence of both intra- and inter-generational social mobility related to health, selection is not an important determinant of health inequalities (Power et al., 1991, 1996; Hertzman, 1999; Adler and Ostrove, 1999). Given these strong assertions by leading researchers in the field, one might well question the utility of additional efforts directed toward measuring the role of selection. However, there are several caveats that bring into question the validity of their conclusions.

maintain that the reverse pattern should occur if only selection factors are present.

First, researchers need to bear in mind that the strength of selection process necessarily varies according to the health and social indicators of interest and by age, time and place. For example, the importance of inter-generational and/or intra-generational social mobility has already been identified for several illnesses including schizophrenia (Goldberg and Morrison, 1963), chronic bronchitis (Meadows, 1961), and epilepsy (Harrison and Taylor, 1976), and is likely to include other mental illnesses and severely disabling diseases (West, 1991). If the focus is on these types of illnesses, selection may well be a very important mechanism in producing social inequalities in health. However, its significance in accounting for inequalities in broad health measures (such as self-assessed health or longevity) obviously depends on the extent to which these illnesses affect overall health or survival status. Earlier in this century, when infectious diseases and chronic ailments were common throughout the life cycle, and medicine had little impact on moderating the resulting symptoms and disabilities, it is likely that selection played a much larger role than it does today. It is also clear that the importance of selection must vary by the measure of SES or social factor under study. As noted earlier, intra-generational selection with regard to education is very unlikely after young adulthood, because most people have completed their schooling by then. In contrast, as economists recognize, wealth and especially income are subject to repeated influence throughout the life cycle (Smith, 1999). For example, Smith and Kington (1997, p. 158) observe that “there is compelling evidence that the feedbacks from health to current socioeconomic status are quantitatively strong and should not be ignored in empirical investigations.” Healthier individuals are often able to work more hours than their frailer colleagues and achieve higher earnings, while some persons in sufficiently poor health

are eligible for government transfers, presumably altering their income, or are obligated to retire, thereby relinquishing all current earnings.

A second concern relates to the potential role of *indirect* selection in creating health inequalities. Even if we accept that in modern industrialized societies the proportion of the young and adult population that suffers from illnesses linked to downward social mobility is small, we have little information about the extent to which selection is driven by factors related to health (rather than by health status directly). Most research on this issue pertains to the influence of height and has demonstrated that social mobility is indeed related to height, i.e., taller persons are more likely to be upwardly mobile (Illsley, 1955; Power et al., 1986, 1991; Marmot et al., 1995; Nystrom-Peck and Vagero, 1989). A few studies have examined other health-related variables such as health-related behaviors and characteristics (e.g., drug use, nutrition, exercise, and weight) and physical attractiveness. Researchers have suggested that indirect selection may also encompass background variables related to both health and social mobility – such as education and parenting styles – genetic characteristics, and such variables as time preference, self-efficacy, and coping abilities (Davey Smith et al., 1990; Fuchs, 1993; West, 1991; Hertzman, 1999). Inclusion of these variables under the rubric of indirect selection poses at least two problems for analysts. One is that many are extremely difficult to measure. Second is the fact that some of these factors are themselves the product of what we think of as social class. Education and parenting styles are the most obvious examples, but even height, which is influenced by chronic or repeated infections in childhood, is also affected by nutritional deprivation associated

with poverty. Inclusion of a broad range of variables under health-related selection undoubtedly complicates the task of distinguishing selection from causal processes.

Research on marital status differences in health points to many of the same problems noted above.⁸ With the exception of several studies,⁹ most demographers have argued that causal processes are considerably more important than marriage selection in producing the health advantages of the married population, and some have refuted altogether the importance of selection (Goldman and Hu, 1993). Several pieces of evidence presented during the past decade reveal that this conclusion is unwarranted. Goldman (1993b) demonstrates that the mate selection process was probably the major factor in generating large differences in longevity between single and married Japanese in the middle of the 20th century. In particular, the importance of excluding potential spouses with psychological ailments or illnesses *thought* to be hereditary (e.g., tuberculosis) in the family, and the use of relatives, neighbors, go-betweens, and private detectives in the arranged marriage process appear to be largely responsible for the more than 15-year disadvantage in life expectancy experienced by Japanese singles relative to married Japanese (Goldman, 1993b, Goldman et al. 1995). The health disadvantages of Japanese singles have declined considerably in recent years, probably as a result of both the progressive abandonment of arranged marriages and the decreasing prevalence of infectious diseases among the younger population. Although marriage is likely to have been more selective with regard to underlying health characteristics in Japan than

⁸ In this paper, we restrict the discussion of selection mechanisms to the transition from never-married to married. Many other selection processes are likely to characterize marital status differences in health, such as those pertaining to persons getting divorced, persons who lose their spouses, and persons who remarry. In particular, there has been considerable discussion of the potential role of selection in producing the “bereavement effect” (i.e., the poorer health status of the surviving spouse in the early stages of widowhood).

elsewhere, this study highlights the dangers of making generalizations about the role of selection across time and place.

Studies of marriage choice have also shown that indirect selection probably plays an important role in producing the health advantages of the married population. Fu and Goldman (1996) note that a serious limitation of existing research is the narrow conceptualization of health-related selection into marriage. Researchers often restrict this type of process to the existence of a specific, typically severe physical limitation and fail to consider many variables that are known to affect subsequent health status – such as smoking, drug use, drinking, emotional stability, height and weight – and may be related to the likelihood of marriage. Their analysis of data from the National Longitudinal Study of Youth reveals that young men and women with undesirable behaviors and characteristics (e.g., hard drug use, obesity, and short stature) do have lower marriage rates than their healthier counterparts. Although apparently similar to discussions about the role of indirect selection in producing the SES-health link, studies of marriage choice have an important advantage over other studies of social mobility: the transition from being single (never-married) to married occurs only once in a lifetime whereas some SES-health links involve an ongoing interaction over the life cycle.

It is precisely this potentially continuous feedback between social factors, such as occupation or income, and various measures of health status that render the disentangling of selection from causal mechanisms such a daunting task, even in the presence of rich longitudinal information. The undertaking is further complicated by the large body of research demonstrating the effects of fetal, infant and child health on adult health

⁹ For example, Mastekaasa (1992) notes that selection may play an important role in producing the association between marital status and well-being.

(Barker, 1992; Wadsworth, 1986; Hertzman, 1999) and that necessitate inclusion of these potential impacts in studies linking social factors and health. Current strategies frequently used by epidemiologists and social scientists to measure either health-related social mobility or causal connections – e.g., a statistical model that relates social status and health at two points, often decades apart – by and large fail to account for these multiple pathways. Smith (1999) recognizes these complexities in his investigation of the effects of chronic health problems on wealth accumulation. He notes (p. 161) that although economists have only recently incorporated health into their models of savings or wealth, they have the “research tools that are well-suited to model such complex feedback mechanisms and to isolate within-period innovations in the stock of health.”

Identifying causal pathways

Most research on social inequalities in health has been devoted to identifying the underlying causal linkages rather than the selection mechanisms, in part because of the belief that the latter influences are small. Many causal pathways through which financial resources, education and occupation may affect health have been postulated, including (1) access to medical care, both preventive and curative; (2) access to information regarding health risks and health care; (3) patterns of health risk behaviors (such as smoking, drinking, unhealthy diet, and inadequate exercise); (4) exposure to environments that are not conducive to good health and longevity (e.g., poor housing conditions, occupational hazards, pollution, and crime); (5) exposure to stressful situations; (6) access to resources that mediate the physiological consequences of stress;

(7) ability to control one's environment, feel secure about one's position, and adopt effective coping strategies; (8) the availability of social relationships and support; (9) preferences about the allocation of time.

Researchers who have focused on only one of these mechanisms have concluded that multiple pathways almost certainly underlie the observed associations. For example, although the higher death rates experienced by persons of lower SES in Britain and the U.S. have been shown to result in part from higher rates of smoking, poorer diets, and inadequate levels of exercise, these differences in behavior were insufficient to account for the observed mortality differentials (Marmot et al., 1995; Macintyre, 1997). Similarly, the widening of mortality differentials by SES between the 1960s and 1980s in Britain and the U.S. – despite the presence of the National Health Service in the former and Medicaid in the latter – has been taken as evidence that the explanation goes beyond inequities in access to medical care. Recognition of the multitude of pathways that need to be considered, the complexity of interactions among factors, and the deficits in our understanding of the linkages are made evident by the “*simplified* causal model” shown in Figure 1 (Marmot, 1999, p.21). Similar depictions of theoretical frameworks pertaining to social inequalities in health appear in other investigations.

As with the study of selection processes, it is useful to consider the types of strategies used to establish causal linkages between social factors and health. For simplicity, these strategies can be categorized along two dimensions, reflecting the nature of the design and the types of subjects: (1) experimental or quasi-experimental vs. observational studies; and (2) animal vs. human subjects. We discuss experimental and

animal studies together because most experimental research has been carried out on animals and because the two types of studies share numerous strengths and weaknesses.

Experiments, quasi-experiments and animal studies

Experiments – i.e., randomized, controlled, double blind trials, – comprise the gold standard of research in the medical profession. By ensuring that treated individuals are no different from the control subjects on average, and by having researchers manage the administration of the treatment, experiments provide a strong basis for causal inference about the effects of the treatment – although they may provide little information about the underlying physiological processes relating the treatment to the outcome.

(Quasi-experiments do not entail randomization, but also involve the researcher’s control over the intervention.) However, while experiments – often referred to as randomized clinical trials – may be an appropriate strategy for evaluating medical interventions, they are typically unfeasible and unethical in social science research, particularly when dealing with humans. For example, it is easy to imagine the obstacles involved in random assignment of persons to social class, economic position, or marital status, let alone in keeping the subjects blind to their social positions. In addition, in an effort to control confounding influences, experiments may strip “away the essential historical and social context, as well as the multiple moderating influences that constitute true causation” (Pearce, 1996, p. 682).

Much of the experimental or quasi-experimental research in the field of social factors and health pertains to the researchers’ manipulations of the presence or degree of

social contact or support experienced by subjects, rather than of SES *per se*. For example, experiments have been used to demonstrate the positive consequences of physical contact on cardiovascular functioning and on other risk factors among humans and among several species of animals (House, 1988).

Other animal studies that examine social position, rather than social contact, have provided additional evidence for causal pathways linking social factors and health. For example, higher well-being among baboons – measured in terms of several risk factors for cardiovascular disease – has been shown to be associated with higher rank, improvement in rank, and greater ability to make social connections (Sapolsky, 1998). Kaplan and Manuck (1999) review numerous studies indicating that the relative social status of monkeys is linked to atherosclerosis and hence cardiovascular disease. While many of these investigations have been observational (i.e., observing animals in their normal habitats or in captivity), others have been experimental or quasi-experimental in the sense that researchers exerted control over some part of the “intervention.” For example, in a series of experiments, Shively and colleagues manipulated the social status – i.e., dominant vs. subordinate – of monkeys (Shively and Clarkson, 1994; Shively et al., 1997) and examined the subsequent development of atherosclerosis and depression. Wilkinson (1999) notes that the investigators’ ability to manipulate the social environment in studies of nonhuman primates strengthens the implied causal inferences by minimizing the possibility of reverse causality.

There have been other types of clinical studies geared toward identifying SES or social links to health in animals and humans that might be considered as quasi-experimental. However, in these undertakings, exposure to illness rather than social

position was under the control of the researcher. Cohen (1999) describes three such studies that were designed to determine whether the greater susceptibility to infectious disease among lower SES groups is due to their higher exposure to pathogens or to better immunity. Subjects, which included both human volunteers and monkeys, were inoculated with an upper respiratory virus, quarantined, and monitored for the development of an infection (Cohen, 1999). The results for humans revealed a higher susceptibility to infection among the unemployed and among subjects with lower perceived social status. The outcomes for monkeys indicated a similar association between social rank and susceptibility to infection. Although not a true experiment, this research strategy provides fairly convincing evidence in favor of causal mechanisms because, once again, the researcher's control over the timing and nature of the exposure to disease make selection-type explanations implausible.

There are clear advantages as well as disadvantages associated with experimental-type designs for research on social factors and health. To the extent that the investigator can manipulate the relevant conditions in experiments or quasi-experiments, causal explanations become more powerful than selection-type explanations, although even the best of these studies may fail to elucidate the relevant components of SES or the pathways through which SES affects health. On the other hand, experiments are seriously limited in the range of social indicators or environmental conditions that can be studied. For example, experimental manipulation of standard SES indicators is far more difficult than of simple forms of social contact, and the studies of the cold virus cannot readily be expanded to encompass more severe or non-infectious illnesses (at least not with the approval of human subjects review boards).

Animal studies also have their benefits and drawbacks, regardless of whether these studies are experimental or observational. Hertzman (1999, p. 28) argues that insights from nonhuman primates show “remarkable similarities to human populations.” Moreover, he claims that studies based on these primates present fewer logistical complications than those involving humans, in part because we can take advantage of the natural experiments afforded by the habitats of these animals. Kaplan and Manuck (1999) note that studies of animals afford the investigator greater control over the social environment and other factors as well as permit earlier and more precise measurement of some biological parameters than is generally possible with humans. At the same time, however, there are serious limitations to generalizing from animals to humans. Kaplan and Manuck (1999, p. 157) identify two conceptual issues “that block an easy translation of results from monkeys to people...with respect to the SES gradient.” First is the finding that there is nothing pathogenic associated with dominant or subordinate social position among monkeys (i.e., either relative social position can be associated with increased health risk, depending on gender and circumstances in the social environment); in contrast, low SES (as compared with high SES) is consistently associated with a greater risk of illness in humans. Second is the lack of equivalence of the constructs of relative social status in monkeys and SES in people. More generally, animal studies – like experimental studies – can never permit us to study the full range of hypothesized linkages between the social environment and health, including such pathways as health-related behaviors and preventative and curative medical care.

Observational studies of humans

Thus, we must ultimately rely on observational studies of humans to learn more about the causal linkages between SES and health, in spite of the inferential shortcomings of these types of investigations. Classic methodological and statistical problems – such as measurement error, simultaneity bias or endogeneity, and omitted variable bias – characterize observational studies of SES and health, as they do all fields of research, rendering inferences of causality suspect. There is little doubt that improvements in the type and quality of data collected and the analytical procedures used over the past couple of decades have resulted in more convincing evidence in support of causal mechanisms. For example, detailed prospective data permit the researcher to minimize effects of reverse causality by ensuring that measures of social factors precede measures of health outcomes and by controlling for health status at the start of the relevant follow-up period. On the other hand, it is also important to recognize that while the correct ordering of events made possible by prospective data reduces the likelihood of selection-type explanations, the resulting analyses are not sufficient for identifying causal linkages. For example, underlying causal pathways may become obscured by researchers' efforts to control for earlier events with which later causal processes are associated (Power and Matthews, 1998). Although researchers studying the linkages between SES and health are unlikely to agree upon a set of criteria sufficient for the establishment of causality, at

minimum they need to recognize that these criteria must include more than the presence of a temporal relationship.¹⁰

Given the vast number of observational studies related to social gradients in health that have been conducted over the past several decades, even if we restrict consideration to those based on longitudinal data, it is difficult to provide a useful summary in terms of the underlying mode of causal inference. This task is complicated further by fact that existing studies encompass many disciplinary focuses and hence vary considerably from one another in theoretical framework, design, data sources, and analytical procedures. Instead, we attempt to highlight below the major changes in research strategy over the past decade that are elucidating the linkages between social factors and health.

Early research related to social gradients in health expended enormous energy in repeatedly demonstrating the presence of these gradients, with little or no effort devoted to understanding the underlying reasons for their presence. Other research attempting to identify causal pathways – especially the large body of work by epidemiologists – was for the most part atheoretical. Data were typically available on relatively few components of the hypothesized pathways, generally those aspects that were most straightforward to measure with interview surveys. As a result, a set of explanatory variables was often tossed into a multivariate model, with little attention paid to the fact

¹⁰ For example, a temporal relationship was only one of five features used by a committee appointed by the Surgeon General to explain the relationship between smoking and health (Surgeon General, Advisory Committee of the USPHS, 1964). Similarly, the virologist Robert Heubner (1957) noted that although the isolation of a viral agent temporally related to the disease process was necessary and important, it provided evidence “of very low order” for the establishment of causality. More generally, Evans (1978) describes how the criteria for establishing causality of disease have been under constant revision as technology advanced and new illnesses emerged. Of particular relevance for the identification of the social determinants of illness is the acknowledgment that definitions of causation have broadened over time as

that these variables operated at different levels of causal influence. For example, explanatory variables typically encompassed a hodgepodge of what the British refer to as material conditions or material circumstances (e.g., assets, housing, car ownership), health-related behaviors (such as smoking), use of medical services, and an occasional biological parameter (such as blood pressure). Variables were usually measured at a single point in time (in adulthood) prior to the outcome of interest (illness or death).

This is not to say that such types of analyses failed to yield valuable findings. Indeed, the large number of studies that identified social gradients in health, regardless of the measure of SES or health outcome used or the social and cultural setting, has elevated the importance of this area of research. In addition, earlier research demonstrated the failure of simple models to explain the existing gradients: (e.g., SES gradients in health could not be accounted for by existing variation in health-related behaviors or in medical care) and, hence, inspired researchers to expand their horizons. However, it appears that many of these studies were more successful at ruling out possible explanations than they were at identifying environmental and behavioral factors that serve as critical linkages in the pathways connecting SES and health.

One notable change in the past decade has been a recognition and understanding of the influence of psychosocial influences on health. These factors include sense of control or mastery, anxiety, shame, stress and strain, depression, and hostility (Marmot, 1999; Wilkinson, 1999). This awareness has led researchers to move beyond a focus on the material conditions associated with different levels of occupation or social class to consideration of how different occupations or social positions vary in terms of acute and

scientists have come to recognize that the development of most (infectious) diseases is influenced not only by pathogenic agents, but also by the susceptibility of the host, the environment, and numerous cofactors.

chronic stresses, ability of individuals to control their lives, demands and rewards, etc. (Marmot, 1999).

Another research development has been an increasing focus on characteristics of the individual's broader social environment. Some of the interest in community or societal context has been motivated by studies demonstrating that the steepness of the social gradient in a given society reveals a stronger relationship with the degree of income inequality than with the average level of income (Kawachi et al., 1999). Although the legitimacy of these findings has recently been questioned,¹¹ studies of income equality have encouraged scientists to pay attention to how characteristics of these societies affect the well-being of individuals in all positions of the socioeconomic spectrum. For example, Hertzman (1999) cites the importance of factors at the level of "civil society" and the state in understanding SES influences on health. Robert (1999) reviews studies suggesting that the socioeconomic context of communities affects individual health above and beyond the impact of individual SES. The potential significance of income inequality for health has prompted researchers to focus on the *relative* rather than the absolute deprivation of individuals and to consider the health consequences of low social status, lack of social integration and cohesiveness, social anxiety, and discrimination (Wilkinson, 1998, 1999).

Advances in the previous two areas of focus (i.e., psychosocial influences and the broader social context) have been especially important for an understanding of racial differences in health in the United States. Although SES differences among racial groups account for a substantial fraction of the racial disparities in health, adjustment for SES

rarely eliminates the differences.¹² Racial discrimination, operating partly through residential segregation, affects health through numerous pathways – such as access to resources and opportunities, environmental conditions, and psychosocial factors (such as stress and low self-worth) associated with stigmas of inferiority (Williams, 1999).

Important progress in understanding causal linkages between social factors and health has also come about through increasing recognition that social gradients in health are formed continuously throughout the life cycle and that many critical influences occur early in life – at birth if not before (Power et al., 1991; Power et al., 1998; Keating and Hertzman, 1999). For example, in a recent volume, Keating and Hertzman (1999, p. 7) note that “differing social circumstances at the time of disease expression are not a sufficient explanation for the robust population patterns connecting socioeconomic gradients to health outcomes. These patterns of population gradients, especially their longitudinal nature, suggest a potentially important role for the organism’s experience, particularly early experience, in shaping coping skills, resiliency, and thus neuroimmune and neuroendocrine response at the individual level.” A substantial body of research has now established that factors associated with a child’s early life, such as parent-child interactions, childhood abuse, and the stability of the home environment, can affect a wide range of behavioral and physical outcomes in subsequent years (Wadsworth and Kuh, 1997; Power and Hertzman, 1999; Felitti et al., 1998). Moreover, indicators of physical development during the prenatal period, at birth, and during early childhood are

¹¹ For example, Judge et al. (1998) find little support for the proposition that income inequality is associated with average levels of health when the analysis is restricted to rich industrial countries with the highest quality and most comparable data available.

¹² For example, many studies have demonstrated that, within a given level of SES, blacks have poorer health than whites. In addition, in spite of high levels of poverty, some Hispanic groups have indicators of health status (such as infant mortality) that are comparable to whites (Williams and Collins, 1995).

strongly associated with later health (Barker, 1994; Wadsworth and Kuh, 1997; Wadsworth, 1991).

Two theoretical models, which are currently the subject of considerable investigation, are based on these notions of critical influences early in human development and continuous influences of SES over the life cycle. The *latency* model underscores the potentially critical impacts of early experiences on facets of adult life, whereas the *pathways* model highlights the cumulative and interactive effects of socioeconomic and psychosocial factors throughout the life cycle (Keating and Hertzman, 1999). Although not expressed explicitly in terms of the pathways model, several recent investigations have focused on the importance of examining cumulative adversity as well as the potentially compensating influences of cumulative advantage – in lieu of single isolated circumstances – in efforts to understand health outcomes (Marmot et al., 1991; Singer and Ryff, 1997, 1999; Beckett et al., 1998). In a similar way, some economists have recognized the need to consider the health consequences of long-term economic deprivation, permanent income, or wealth in lieu of the effects of annual income (Williams and Collins, 1995). Some researchers have also underscored the importance of identifying the *multiple* pathways, across different life domains, through which individuals may achieve a set of health outcomes (Seeman et al., forthcoming).

Related to the emphasis on early childhood development, recent research has demonstrated that social inequalities extend beyond the already broad measures of wellbeing, illness and survival examined in the literature on social factors and health. Keating and Hertzman (1999) note that socioeconomic gradients also characterize a wide range of development outcomes, such as academic achievement, behavioral problems,

mental health and social adaptation. Expansion of current research on social inequalities in health to include these measures of “development health” may ultimately enhance our understanding of how factors at the level of the society, family, and individual interact to produce a broad set of socioeconomic gradients during early childhood development.

A final, major advance in understanding the pathways linking the social environment and health has been the incorporation of physiological parameters into social science surveys. The rationale for this is clear and convincing: if differences in the social environment are causally related to health, then scientists should be able to identify how differences in income, jobs, social support and other social dimensions express themselves in terms of variations in biological parameters that are linked to health. To the extent that researchers are successful in this effort, the findings will undoubtedly enhance our understanding of the underlying causal linkages. Attempts to elucidate the connections between the social environment and health by identifying the underlying physiological pathways are not new. For example, Bovard (1985) reviews a large number of animal and human studies that point to the role played by hypothalamic mechanisms, and mediation by the amygdala, in explaining how social networks and support protect individuals against illness. However, most of these earlier analyses were based on small-scale clinical studies and typically focused on a narrow range of biological mechanisms.

In contrast, current research in this area is attempting to (1) incorporate biological measures in (large-scale) population-based health and social science surveys; and (2) include a broad range of biological markers across different physiological systems that

form important pathways linking social factors and health.¹³ The benefits to be gained from the inclusion of biomarkers (such as the collection of blood and urine samples or physician's exams) in population-based surveys, especially longitudinal ones, are clear. Such surveys are generally based on large-representative samples and some already contain detailed information on individuals' life histories, including the social environment – the kind of data lacking in most clinical-type studies. The costs of incorporating biological measurement, however, may be substantial, in terms of financial expenditures, logistical complications, and ethical considerations (Weinstein and Willis, 2000). During the past few years, several data collection efforts along these lines have been undertaken– e.g., the MacArthur Successful Aging Study (Seeman et al., 1997), the Wisconsin Longitudinal Study (Singer and Ryff, 1999), and the Social Environment and Biomarkers of Aging Study in Taiwan (Goldman et al., 1999) – and it is highly probable that similar efforts will be carried out in the near future.

A major consideration in the design of these data collection efforts is determination of what biological variables should be measured. These choices depend upon both the underlying biology (what markers are sensitive to social and economic conditions) and practical considerations (what can be measured in a population-based survey without compromising respondent participation, budgetary constraints, etc.).¹⁴ Research pertaining to the first of these issues has borrowed many of its ideas from the extensive body of work over several decades pertaining to the health consequences of stress (see, for example, Weiner, 1992). The justification for doing so is that

¹³ Another distinction between current and earlier research based on physiological parameters is that scientific advances are now allowing researchers to obtain measures of critical biomarkers (such as urinary cortisol) that were previously unavailable.

socioeconomic status is known to affect both exposure to stress and mechanisms for coping with stress.

McEwen has provided an alternative formulation to what he considers a poorly defined concept of stress (McEwen and Stellar, 1993; McEwen, 1998). This representation, described in terms of allostasis and allostatic load, is particularly appropriate for examining the health impacts of the social environment, because social position and social networks entail long-term or repeated challenges (as well as advantages) and because complex social factors are likely to affect multiple biological systems. “Allostasis” refers to the balance among physiological systems within the body that fluctuate to meet demands from external forces. “Allostatic load” refers to the cost of adaptation to heightened physiological responses resulting from repeated or chronic environmental challenges – i.e., the “wear and tear” on the body from continuous cycles of allostasis. Involved in allostatic load are elevated levels of neuroendocrine, immunological, and sympathetic nervous system reactivity and accompanying strain on multiple organs and tissues. The accumulation over long periods of high allostatic load can lead to organ-system breakdown, impaired immune responses, elevated cortisol and insulin secretion, and ultimately, a range of chronic disease outcomes including coronary heart disease, diabetes, depression, and musculo-skeletal problems.

Initial attempts to operationalize allostatic load in terms of ten indicators of risk (pertaining to the cardiovascular system, hypothalamic-pituitary-adrenal (HPA) axis, sympathetic nervous system, and metabolic processes) have been predictive of future mortality, cardiovascular disease, and declines in cognitive and physical functioning

¹⁴ Kelly et al. (1997) provide an excellent review of potential physiological markers of chronic stress that are candidates for incorporation into population-based surveys.

(Seeman et al., 1997; McEwen and Seeman, 1999; Seeman et al., in press).¹⁵ The finding that the socioeconomic gradient for allostatic load resembles that for mortality (McEwen and Seeman, 1999) suggests that allostatic load, or alternative summary measures of risk, may provide useful insights into the causal connections between social factors and health. Beyond the physiological systems encompassed in current measures of allostatic load, scientists are constantly discovering additional pathways through which responses to stress affect the immune system, thereby influencing susceptibility to a broad range of illnesses (Sternberg, 2000). Undoubtedly, the specification of measures of health risk will undergo constant refinement, as scientific advances provide new information about poorly understood biological and genetic markers and identify additional ones.

Conclusions

Researchers from diverse disciplines have employed a variety of data sources and analytic strategies in their attempts to disentangle the selection and causal mechanisms between social factors and health. Social and medical scientists are likely to be correct in their consensus that the observed disparities in health are driven largely by causal processes rather than by selection – even though they have done a relatively poor job of

¹⁵ These ten indicators, along with additional biological markers obtained from blood and urine samples, were collected in the MacArthur, Wisconsin, and Taiwan studies described above. The ten parameters (and the associated physiological system or process) are: systolic and diastolic blood pressure (cardiovascular activity); waist-hip ratio (metabolism); serum HDL and total cholesterol (atherosclerotic risk); blood plasma levels of glycosylated hemoglobin (glucose metabolism); DHEA-S (HPA axis); overnight urinary cortisol (HPA axis); and overnight urinary norepinephrine and epinephrine (sympathetic nervous system). In studies to date, allostatic load has been calculated as the number of parameters (among the 10 listed above) for which the subject falls into the quartile of highest risk (i.e., the top quartile for all markers except HDL cholesterol and DHEA-S).

defining and analyzing selection. Only during the most recent decade have researchers begun to seriously devote themselves to pinpointing the salient causal mechanisms.

The extensive body of research to date has revealed how intricate the linkages between the social environment and health are likely to be. As researchers are expanding their theoretical frameworks in the ways described above to accommodate these complexities, they are also revising their data collection efforts in corresponding directions. The implications for future longitudinal surveys are vast, suggesting, for example, that prospective surveys should begin at birth, follow respondents at regular intervals throughout the life cycle, obtain detailed life histories regarding social/SES, psychological and health dimensions, consider not only the individual and family but the broader social environment, and include biological measurements along the way. The resulting data will provide serious challenges for social statisticians who will need to take into account the two-way interactions between the social environment and health throughout the life cycle as well as influences between a given set of factors at one time and the same set of factors at a later time. Standard regression models will not suffice in this case. More sophisticated econometric tools based on structural equations models may be more appropriate, but even these procedures depend heavily on simplifying assumptions as well as on the notion that the essence of individuals can be succinctly captured by a small set of generally independent characteristics (variables). Alternative statistical methodologies (sometimes referred to as *person-centered* approaches in contrast to *variable-based* methodologies) that use individual life histories to construct multiple pathways relating the social environment to health are being developed and may prove useful in this field of study (see, for example, Singer et al., 1998). Regardless of

the type of analytical approach, however, researchers will have to learn how to manage complex reams of data in ways that incorporate the richness of information into their procedures.

This type of research will call not only for the involvement of academics in different disciplines but more importantly for interdisciplinary collaboration among scientists to articulate how environmental or psychosocial influences are embedded in biological processes. Anderson (1999) argues similarly that a full understanding of the linkages between the social environment and health will require the integration of research across the multiple levels of analyses inherent in health research: the social/environmental, behavioral/psychological, organ systems, cellular, and molecular levels. These efforts should occupy social and medical scientists attempting to explicate the linkages between social factors and health well into the new millennium.

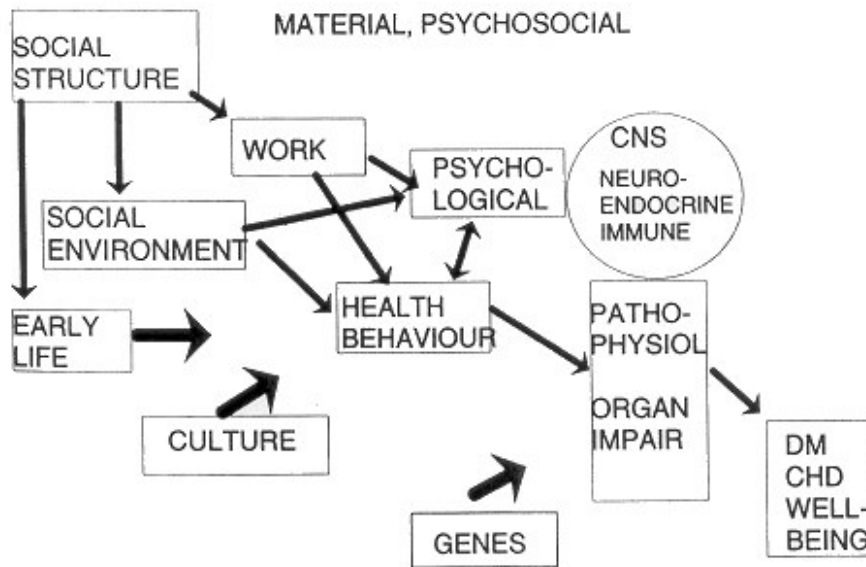


Figure 1. Simplified causal model of inequalities in health. Abbreviations: CNS, central nervous system; DM, diabetes mellitus; CHD, coronary heart disease.

Source: Marmot, M. (1999). Epidemiology of socioeconomic status and health: are determinants within countries the same as between countries? In *Socioeconomic Status and Health in Industrial Nations: Social, Psychological and Biological Pathways*, eds. N.E. Adler, M. Marmot, B.S. McEwen and J. Stewart. *Annals of the New York Academy of Sciences*. Vol. 896, pp. 16-29. New York: The New York Academy of Sciences.

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