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Social factors and health: The causation–selection issue revisited

(health-related selection/social mobility)

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ABSTRACT **Since social scientists rarely have access to experimental data, they rely heavily upon observational studies. As a consequence, their attempts to make causal inferences about the effects of social factors—such as occupation or marital status—on health are plagued by potential selection problems. Some researchers have addressed this selection–causation problem on the basis of the presence or absence of a particular aggregate pattern of health status. The rationale underlying this approach derives from the investigators' hypotheses that the presence of selection would lead to a particular type of pattern that is distinct from the pattern that would result in the absence of selection. Although intuitively appealing, this strategy appears to be seriously flawed. The essential weakness is that the range of patterns that can result from selection is often much broader than researchers have speculated.**

In contrast to medical scientists, who have come to rely on randomized clinical trials to study the effects of particular treatments on health outcomes, most other researchers typically analyze data from observational studies. In particular, social scientists and epidemiologists interested in the effects of social or economic factors on health are rarely in a position to determine which individuals receive the "treatment" and which do not.* Indeed, in most investigations relating social factors to health status, social experiments are both infeasible and unethical. And, even when possible, social experiments are often not the preferred strategy for causal analysis (2, 3).†

Although clearly handicapped by the absence of random assignment to the treatment and control statuses, researchers working with observational studies strive to make causal inferences about the effects of the social factors under study on health outcomes. However, unlike the analyst of a randomized trial, who can argue that any characteristic—even those not observable to the investigator—is equally likely to be present in the treatment and control groups, the researcher who uses an observational study is plagued by potential selection problems. That is, if a characteristic X (such as marital or employment status) is associated with a health outcome Y, the researcher must be convinced that persons bearing X do not differ in any way that potentially affects health from persons not bearing X before he or she can make causal inferences about the relationship between X and Y. Many researchers handle this problem by using a statistical model, in conjunction with a model of the underlying process, to control for a large number of characteristics that are associated with the characteristic X and the health outcome Y. The use of controls for factors not observable to the researcher is also possible, although more problematic (4).

The extent to which a researcher can control for selection depends to a large extent on the available data. Longitudinal data are typically superior to cross-sectional data, since they often allow the investigator to measure preexisting conditions (i.e., characteristics present at the start of the study) whose effects on health outcome may be confounded with those of the characteristic under study. Longitudinal data may offer the additional advantage of permitting the investigator to relate changes in the characteristic under study to changes in health outcome over the follow-up period. Because both explanatory factors and health outcomes are usually measured as of a single time point in cross-sectional data, these data typically offer a less convincing basis for causal inference.

A review of the social science literature reveals a recent trend toward greater reliance on longitudinal data and on more elaborate statistical and econometric techniques, both of which can serve to reduce potential selection bias. At the same time, however, there have been numerous analyses that have addressed the selection–causation problem as it relates to the influence of social factors on health on the basis of the presence or absence of a particular aggregate pattern of health status, with few if any control factors. The rationale underlying this approach derives from the investigators' hypotheses that the presence of selection would lead to a particular type of pattern with regard to health outcome that is distinct from the pattern that would result in the absence of selection. The extent of similarity between the patterns estimated from cross-sectional data and the hypothesized patterns forms the basis for conclusions about the importance of selection.

Although intuitively appealing and elegant in its simplicity, this strategy appears to be seriously flawed. The essential weakness is that the range of patterns that can result from selection can be much broader than researchers have speculated. In the next section of this paper, I summarize two examples of this type of approach that have appeared in the demographic and sociological literature, and, in the subsequent section, I describe an example from the field of psychiatric epidemiology. These examples focus on two important associations between social factors and health: (*i*) marital status and mortality and (*ii*) social status and psychiatric disorder.

Marriage and Mortality: Selection or Causation?

An issue that has intrigued social scientists for more than a century is the relationship between marital status and longevity. Since William Farr's study of the influence of mar-

*In recent years, social experiments have become more common, particularly as a strategy for evaluation of social policy related to welfare, child care, education, and health care reform (1).

†Consider, for example, an experiment designed to study the effects of marital status on health. Attempts to randomly assign persons to different marital states would be certain to face strong resistance (particularly from spouses of those assigned to the widowed state). In addition, the health-related effects of living with a randomly chosen mate are likely to be very different from the psychological and social benefits of living with one's beloved.

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riage on French mortality (5), hundreds of investigators have attempted to determine whether married people fare better than their unmarried counterparts because mentally and physically healthier persons are more likely to get married in the first place (*marriage selection*) or because of the presumed social, psychological, economic, and environmental benefits associated with having a spouse (*marriage protection*). Most researchers readily acknowledge that both types of mechanisms are probably responsible for the observed mortality differences, but rather little is known about their relative importance.‡

In an examination of methods used by researchers to distinguish the mechanisms underlying the observed mortality differences, Goldman (7) identified one strategy used repeatedly since Durkheim's classic study of suicide (8). The general approach entails the use of aggregate patterns of mortality by marital status, estimated from cross-sectional data, as the basis for inferences about the importance of marriage selection. Two specific strategies that exemplify this approach are summarized below. The first is based on age patterns of mortality differentials and the second on the direction and strength of the relationship between the magnitude of the mortality differential and the relative size of the single population.

The general nature of the argument proposed by Durkheim and others is that, if marriage selection were operative, the levels of excess (i.e., relative) mortality§ of singles would increase throughout marriageable ages (e.g., 20–40 years) and would decline progressively after age 40 or so, when marriage rates have fallen to very low levels (8–11). This type of reasoning has been used both to support selection hypotheses when the observed age pattern of excess mortality among singles conforms to the hypothesized pattern and to reject selection in favor of causal theories when the observed pattern deviates from the hypothesized one.

A second strategy derives from the hypothesis, implicit in the work of several researchers (11–15), that populations in which the vast majority of persons marry should be characterized by greater selectivity effects among those who remain single than populations in which substantial fractions never marry. Inferences about the importance of marriage selection are typically derived from an examination of the correlation between the proportion remaining single (at an age near the end of the marriage span) and the relative mortality ratio at the same age. For example, large negative correlations—that is, high levels of excess mortality among singles in cases where relatively few individuals remain single—have been taken as evidence of the importance of marriage selection mechanisms.

To test the legitimacy of both types of inferences, Goldman (7) developed a simple simulation model of a marriage selection process defined by two subgroups of individuals (healthy and frail) with distinct marriage and mortality rates. In the model, marriage offers no health benefits over the single state (i.e., for each subgroup, the mortality rates are identical for single and married persons), but healthy persons are more likely to marry (and less likely to die) than unhealthy individuals. The model was used to generate age patterns of excess mortality among singles and patterns of the relationship between the excess mortality among singles and the relative size of the single group, under different sets of plausible parameters.¶ Details of this model were described by Goldman *et al.* (16); a similar type of model is presented in the next section.

The results indicate that, by and large, the inferences that have been drawn from the types of patterns described above are not justifiable. Although the hypothesized patterns—namely, declining relative mortality for singles beyond the upper age at marriage and a negative relationship between the magnitude of the excess mortality and the fraction remaining single—can result entirely from marriage selection, so can many contrasting patterns. For instance, relative mortality ratios can remain constant or rise through middle and older age groups, and ratios can reveal a positive correlation with the fraction remaining single or no correlation whatsoever. Distinguishing among the many possible patterns that may result from selection requires detailed information about the marriage selection process that is rarely available to the researcher. For example, the sign and magnitude of the correlation between the excess mortality of singles and the fraction remaining single depend upon the extent to which variation in the marriage rates across observations (e.g., countries or time periods) results from differing rates of voluntary singlehood among healthy persons or from varying degrees of discrimination in the marriage market against the unhealthy. Goldman (7) concludes that, without further information about the marriage process, it is indefensible either to use hypothesized patterns to support arguments in favor of selection or to use deviant patterns to refute selection arguments in favor of causal theories.

Socioeconomic Status and Psychiatric Disorders: Selection or Causation?

Dohrenwend *et al.* (17) have used an approach similar to that described above to explore the well-established negative relationship between social class and psychiatric disorder. Two theories have been proposed to explain this association. One explanation is that the relationship arises from causal mechanisms through which conditions associated with low social status (such as a disease-prone environment, poor nutrition, lack of adequate education, inadequate medical information, and stress) increase the likelihood of becoming ill. An alternative hypothesis is that social class differences in health arise from social mobility, a selection process whereby frail or diseased individuals are less able to raise their social position and are more likely to drift downward in status, as compared with the healthy. Both causal and selection processes are thought to contribute to the observed associations, but the relative importance of the two has not been firmly established (18, 19).

In a similar vein to the demographers and sociologists referred to above, Dohrenwend and colleagues argue that it is possible to use aggregate cross-sectional data to learn more about the underlying processes relating social status to disease. Specifically, they maintain that a comparison of the relationship between social class and the rate of mental disorders between two ethnic groups—one of which is disadvantaged vis-à-vis the other—permits the analyst to assess the relative role of selection and causal factors in producing the observed negative relationship. In their recent study, Dohrenwend *et al.* (17) base their inferences on a comparison of rates of psychiatric disorder (for a given level of socio-

‡Of course, the relative importance of selection and protection mechanisms is likely to vary across time and place. For example, an analysis of exceptionally high mortality among Japanese singles suggests that marriage selection was more important in Japan than elsewhere (6).

§Most analyses of marital status differences in mortality have relied upon this measure of relative risk known as the relative mortality ratio: the ratio of the death rate of a particular unmarried group (e.g., singles) to the death rate of the married group.

¶Some of the parameters in the model (such as marriage rates for healthy and for frail individuals) cannot be determined from the types of data currently available. However, in each simulation, parameters were chosen so that the overall marriage and mortality rates (i.e., for the healthy and frail groups combined) were consistent with values observed for actual populations.

economic status) between the Israelis of North African background (the disadvantaged group) and the Israelis of European ancestry. They assess the relative importance of selection factors and causal mechanisms for each of several types of psychiatric disorders, including schizophrenia and major depression.

Theoretical Formulation. The basis of the authors' inferences is depicted in Fig. 1 and can be summarized in the following way. If the relationship between psychiatric disorder and social status derives from causal factors (such as mental disorders being induced or precipitated by repeated barriers against success), then, for any level of social class, members of the disadvantaged group should be more likely to confront such obstacles than their advantaged counterparts because of discrimination. Hence, the disadvantaged should experience higher rates of mental disorder at every level of socioeconomic status (SES) and the resulting rates of mental disorder by ethnic group should resemble Fig. 1*A*. By contrast, if only selection mechanisms are operating, then members of the disadvantaged group would not face increased risks of psychiatric disorder (at a given SES level) but would still face greater barriers to improving or maintaining their social status than members of the advantaged group. Because healthy disadvantaged persons are more likely to be hindered in upward social mobility than the healthy and advantaged, the authors maintain that the disadvantaged group should contain a greater proportion of healthy people in its lower social class as compared with the advantaged group. The relatively high presence of healthy individuals should serve to "dilute" the rates of disorder among lower-SES members. By contrast, among the advantaged, the healthy should rise in social status with relative ease and "leave a relatively undiluted 'residue' of disabled among their lower SES members" (ref. 17, p. 947). The authors thus maintain that the social selection hypothesis predicts higher rates of disorder among the advantaged group, as shown in Fig. 1*B*. In their ensuing analysis, the authors defend the selection hypothesis for those psychiatric disorders that lead to a pattern resembling Fig. 1*B* and embrace causal theories for those disorders whose pattern resembles Fig. 1*A*.

Although the arguments underlying Fig. 1 are intuitively appealing, they are flawed. As I demonstrate below, even a very simple social selection mechanism—in the absence of any causal process—can yield ethnic differentials in the relationship between social class and psychiatric disorder which differ radically from that hypothesized by Dohrenwend and colleagues.

A Simple Selection Model. The weaknesses of the authors' arguments can be demonstrated with the social selection model depicted in Fig. 2. Although a considerable oversimplification of the actual mechanisms, this model captures the essential dynamics of a selection process that relates psychiatric disorder to social class mobility. Fig. 2 presents the possible transitions at each age, beginning, for example, in early adulthood (when children have a social status distinct from their parents). The transitions are identified by parameters that are assigned numerical values in each simulation. Disordered and healthy persons can raise their social status (at rates of θ_1 and θ_2, respectively) or lower their social status (at rates of δ_1 and δ_2), and healthy persons of low or high status can become disordered (at rates of β_1 and β_2). Two additional parameters not identified in the flow diagram of Fig. 2 are the proportions of low- and high-status persons (c_1 and c_2) who are disordered as of the defined starting age. For convenience, the parameters are classified into two categories: the social mobility parameters (θ_1, θ_2, δ_1, δ_2) and the incidence parameters (β_1, β_2, c_1, c_2). For simplicity, the model does not permit recovery from the disordered state, nor mortality from either mental state—simplifications which should not detract from the argument presented here, particularly since we focus on relatively young ages.

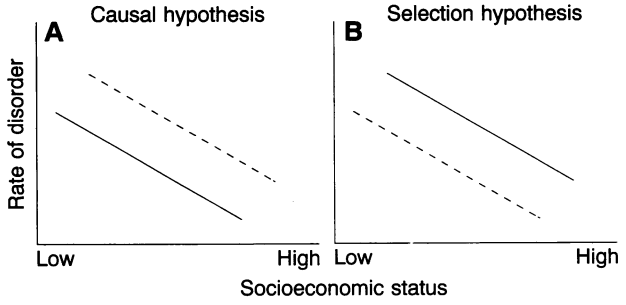

FIG. 1. Associations between rates of psychiatric disorder and social class, by ethnic group, according to causal and selection hypotheses. —, Advantaged group; ---, disadvantaged group. Adapted from ref. 17, p. 947.

Separate numerical simulations are carried out for the so-called disadvantaged and advantaged groups. It is required that, for each ethnic group, unhealthy persons be less likely to raise their social status than healthy individuals ($\theta_1 < \theta_2$) and more likely to drift downward in status ($\delta_1 > \delta_2$). Moreover, to be consistent with the authors' notion of disadvantaged status, the upward mobility rates among the disordered and the healthy members of the disadvantaged group are constrained to be lower than those of their respective advantaged counterparts, and the downward drift rates of the unhealthy and healthy disadvantaged members are constrained to be higher than those of their advantaged counterparts.

It is debatable whether the model should incorporate ethnic variation in the incidence parameters. For example, for a given level of social status, the initial proportions disordered (or the incidence rates at later ages) may differ by ethnic group because of genetic or environmental influences. Hence, we consider two sets of examples below: one in which the incidence parameters are identical between the two ethnic groups and one in which they differ. Without loss of generality, we restrict the incidence parameters to be equal for the two social classes. Nevertheless, it is important to recognize that, to the extent that selection mechanisms result in higher rates of mental illness among lower social classes and that mental disorders have a hereditary basis, we would expect children or adolescents of lower-class parents to have higher

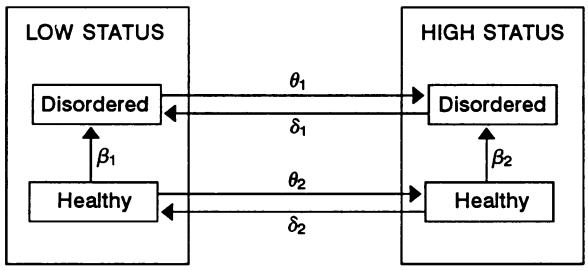

FIG. 2. Simple selection model relating state of mental health to social status mobility, for a particular ethnic group. Parameter values: $\theta_1 < \theta_2$, $\delta_1 > \delta_2$; c_1 and c_2 are proportions disordered at initial age for low- and high-status groups, respectively. The number of disordered and healthy people of low status (D_l and H_l respectively) and the number of disordered and healthy people of high status (D_h and H_h respectively) at age $t + 1$ can be obtained from the corresponding numbers at age t as follows:

$$\begin{bmatrix} D_l \\ H_l \\ D_h \\ H_h \end{bmatrix}^{t+1} = \begin{bmatrix} 1-\theta_1 & \beta_1 & \delta_1 & 0 \\ 0 & 1-\theta_2-\beta_1 & 0 & \delta_2 \\ \theta_1 & 0 & 1-\delta_1 & \beta_2 \\ 0 & \theta_2 & 0 & 1-\delta_2-\beta_2 \end{bmatrix} \begin{bmatrix} D_l \\ H_l \\ D_h \\ H_h \end{bmatrix}^{t}.$$

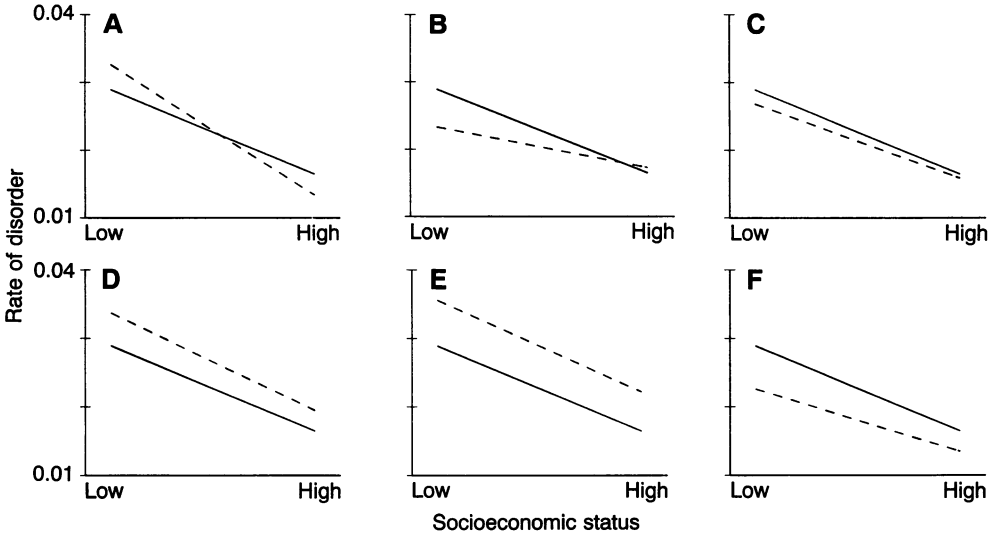

FIG. 3. Possible types of association between rates of psychiatric disorder and social class, by ethnic group, according to a simple selection model. Parameter values for β_1, θ_1, θ_2, δ_1, δ_2, c_1 ($\beta_1 = \beta_2$, $c_1 = c_2$): advantaged (all graphs), 0.001, 0.03, 0.06, 0.02, 0.01, 0.005; disadvantaged (*A*), 0.001, 0.01, 0.05, 0.04, 0.015, 0.005; disadvantaged (*B*), 0.001, 0.03, 0.045, 0.02, 0.015, 0.005; disadvantaged (*C*), 0.001, 0.02, 0.05, 0.03, 0.02, 0.005; disadvantaged (*D*), 0.001, 0.02, 0.055, 0.03, 0.025, 0.01; disadvantaged (*E*), 0.0015, 0.02, 0.055, 0.03, 0.025, 0.005; disadvantaged (*F*), 0.0008, 0.02, 0.055, 0.03, 0.025, 0.005. —, Advantaged group; ---, disadvantaged group.

rates of disorder than children of higher-class parents. The same reasoning suggests that the incidence rates of mental disorder at any age could be higher for the lower social class, even in the absence of any causal mechanism relating social status to mental disorder. Indeed, this formulation of the selection hypothesis highlights the difficulties of distinguishing causal from selection theories.

We use the simple selection model in Fig. 2 (with the equation presented in the legend) to predict the relationship between social class and the prevalence of psychiatric disorder 15 years after some defined initial age. This choice is in general agreement with the fact that the psychiatric evaluations in the study by Dohrenwend *et al.* (17) were carried out on persons about age 30 on average. We consider several different sets of parameters, each of which satisfies the above stated restrictions with regard to social mobility and ethnic discrimination and leads to overall rates of mental disorder that are of the same order of magnitude as reported in the Israeli study. Fig. 3 presents six resulting patterns of association between social class and psychiatric disorder by ethnicity. Fig. 3 *A–C* are derived from models in which only the social mobility parameters differ by ethnic group, whereas Fig. 3 *D–F* result from ethnic variation in both the social mobility and the incidence parameters.

Results. Not surprisingly, social selection results in the predicted negative relationship between social class and the rate of mental disorder in each of the illustrations.‖ However, the observed ethnic differentials may differ considerably from those in Fig. 1*B*. For example, Fig. 3 *A* and *B* demonstrate that the difference in the rate of psychiatric disorder between ethnic groups may vary substantially by social status, in contrast to the parallel lines in Fig. 1.** Indeed, the relative ordering of rates by ethnic group frequently reverses itself between the low and high social statuses. Fig. 3*C*, which most closely resembles the predicted Fig. 1*B*, suggests that, even in the presence of ethnic differences in the social selection process, the relationship between social class and the rate of mental disorder may be similar for the two ethnic groups.

The results in Fig. 3 *D–F* indicate that, with relatively modest differences in the incidence parameters between ethnic groups, the predicted selection pattern (Fig. 3*F*) can occur. However, so can the predicted causal pattern (Fig. 3 *D* and *E*). Further knowledge about which of the very different patterns shown in Fig. 3 is most apt to result from social selection requires further information about the social mobility process for European and North African Israelis and about ethnic variation in the incidence of the (particular) psychiatric disorder for a given social class. Although the patterns in Fig. 3 depend to some extent on the specific selection model used here, a more realistic model or a relaxation of some of the numerical constraints would almost certainly lead to an even greater variety of possible patterns.††

The analysis presented above casts serious doubt on the authors' professed success in having identified circumstances in which causation and selection theories make different predictions. Since the authors failed to provide any information which would enable us to quantify the social selection (or causal) processes in Israel, we must conclude instead that social selection can lead to numerous distinct patterns of ethnic differences in the association between social status and psychiatric disorder. Some of these patterns are virtually indistinguishable from those hypothesized to result from causal mechanisms.

Conclusions

The limitations of the Israel study are virtually the same as those of the studies based on aggregate mortality patterns by marital status. In each case, the range of plausible selection mechanisms is so broad as to encompass patterns hypothe-

‖Since we restrict the model to two social classes, the resulting associations are necessarily linear. However, a linear (or even monotonic) relationship between social class and psychiatric disorder would not necessarily result from a model with three or more social classes.

**Dohrenwend and colleagues note that the resulting association may not be linear and that "this oversimplification does not undermine the test unless the trace lines cross in the center" (ref. 17, p. 947). Fig. 3*A* shows that it is indeed possible for the lines to cross near the center of the socioeconomic scale.

††The authors of the Israel study actually considered two distinct selection models, one based on years of education (which can never be reduced) and one based on occupation status, although they do not distinguish them in any way.

sized by the investigators to be identified only with causal mechanisms.

Perhaps the most important difference between the Israel study and the marital status analyses relates to the availability of data on the respective selection processes. Since sociologists have invested relatively little effort in directly examining the extent to which an individual's likelihood of marriage is affected by health-related characteristics, they have little scientific basis on which to assess the contribution of selection to the observed marital status differences in health and mortality. In contrast, social scientists have undertaken considerable research to evaluate the contributions of social mobility to social class differences in health and mortality. For example, based on data from several long-term longitudinal surveys in Britain and the United States, researchers have demonstrated both that social mobility is selective for characteristics related to health and that social mobility makes a relatively modest contribution to the overall mortality differentials by social class (18, 20–22).‡‡

It is important to recognize, however, that existing longitudinal surveys may not provide the requisite information to the authors of the Israel study or of similar investigations. Undoubtedly, the social mobility process varies across social and cultural settings as well as by the nature of the health limitation. Nevertheless, longitudinal data in their particular setting may ultimately provide the only means for Dohrenwend and colleagues to strengthen their inferences about the impact of social selection. At the same time, the availability of such data would obviate the need for the indirect inferential approaches described above.

One final note of caution is in order. The authors of the Israel study present their approach as a *natural experiment* by which one can assess the relative role of selection and causal factors in producing the observed negative relationship between social class and psychiatric disorders. The relative scarcity of experimental data in the social sciences has resulted in more frequent exploitation of natural experiments. For example, in recent years, economists have used various forms of exogenous shocks in the labor market (such as factory closings, disease-induced population declines, birthrate-generated changes in worker supply, and the introduction of affirmative action policies) as natural experiments to strengthen analysis of the labor market (25). While the researcher should take advantage of such naturally arising comparisons, he or she must also be careful not to use this designation to enhance the credibility of research in the face of serious limitations. The approaches described in this paper appear to fall far short of the standards necessary for even cautious causal inference, and the resulting inferences about selection and causal processes are at best premature.

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‡‡Blaxter (23) provides a description of 20 longitudinal studies in Britain that relate to inequalities in health. For example, the Office of Population Censuses and Surveys longitudinal study, which is based on a follow-up from the 1971 census, continues to collect health and social data from death records, censuses, cancer registers, and hospitals. Much more detailed health and social data are available on a smaller sample from the National Survey of Health and Development (24). These and other longitudinal studies have expanded our knowledge about social class differences in physical and mental health in general and about the role of social selection in particular.