Fundamental causes of social inequalities in mortality: A test of the theory

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"Fundamental Causes" of Social Inequalities in Mortality: A Test of the Theory*

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Medicine and epidemiology currently dominate the study of the strong association between socioeconomic status and mortality. Socioeconomic status typically is viewed as a causally irrelevant "confounding variable" or as a less critical variable marking only the beginning of a causal chain in which intervening risk factors are given prominence. Yet the association between socioeconomic status and mortality has persisted despite radical changes in the diseases and risk factors that are presumed to explain it. This suggests that the effect of socioeconomic status on mortality essentially cannot be understood by reductive explanations that focus on current mechanisms. Accordingly, Link and Phelan (1995) proposed that socioeconomic status is a "fundamental cause" of mortality disparities—that socioeconomic disparities endure despite changing mechanisms because socioeconomic status embodies an array of resources, such as money, knowledge, prestige, power, and beneficial social connections, that protect health no matter what mechanisms are relevant at any given time. We identified a situation in which resources should be less helpful in prolonging life, and derived the following prediction from the theory: For less preventable causes of death (for which we know little about prevention or treatment), socioeconomic status will be less strongly associated with mortality than for more preventable causes. We tested this hypothesis with the National Longitudinal Mortality Study, which followed Current Population Survey respondents (N = 370,930) for mortality for nine years. Our hypothesis was supported, lending support to the theory of fundamental causes and more generally to the importance of a sociological approach to the study of socioeconomic disparities in mortality.

Amid the legacy left by Durkheim is the idea that social conditions are central to the sustenance of life itself. Even the seemingly singular act of suicide is shaped by social circumstances. Looking back, we can think of Durkheim as having brought the fledgling discipline of sociology to medicine and as having created the basis for a far-reaching medical sociology. In *Suicide* (Durkheim [1897] 1966), social factors weren't just a contributor to patterns of suicide but a central, irreducible determinant of those patterns. By bringing sociological principles to bear on what is now commonly seen as a medical and psychiatric problem, Durkheim's *Suicide* represents a bold model for medical sociology with strong links to core sociological principles.

However, in medicine and epidemiology or even in behavioral medicine and social epidemiology—the disciplines that currently dominate the study of disease and mortality—the orientation is quite different from Durkheim's. Here the emphasis is on behavioral or other proximate risk factors for disease. Consider the interpretation of the consistently observed connection between indicators of socioeconomic status (education, occupation, and income) and mortality (Antonovsky 1967; Black et al. 1982; Haan, Kaplan, and Kamacho 1987; Marmot, Shipley, and Rose 1984; Sorlie, Backlund, and Keller 1995). In recent decades, medicine and epidemiology have paid little attention to socioeconomic status as a causal factor in mortality. This is exemplified by Rothman's (1986) use of socioeconomic status, in his widely read *Modern Epidemiology,* as an example of a confounding variable, stating that socioeconomic status is "causally related to few if any diseases but is a correlate of many causes of disease" (p. 90). In contrast, in recent years there has been a burgeoning interest in the relationship of socioeconomic status to mortality. However, in this line of investigation, socioeconomic status is seen as important primarily insofar as it influences more proximate risk factors such as health-related behaviors, access to health care, and psychosocial stress due to relative deprivation (Adler, et al. 1994; Black et al. 1982; Haan et al. 1987; Wilkinson 1997). Socioeconomic status is viewed as a distal cause of mortality that influences and interacts with factors closer to disease in the causal pathway, and attention and intervention efforts

generally remain focused on these intervening risk factors.

Thus, dominant approaches presume that the distal social factor can be explained by—reduced to—more proximal individual-level causes. Socioeconomic status either doesn't affect health and mortality, or it becomes largely irrelevant after intervening modifiable mechanisms are identified.[1] Durkheim's legacy is buried in debates as to which mechanisms—diet, medical care, stress—are most deserving of further scrutiny.

But isn't the emphasis on intervening mechanisms reasonable and even desirable? Clearly, biological mechanisms are involved in the association between socioeconomic status and disease. To get to disease, one must work through biological processes. Just as clearly, other mechanisms must be involved in connecting socioeconomic status to disease; disease does not leap directly from income, educational status or occupational status into the body. Nevertheless, we argue that the effect of socioeconomic status on mortality cannot be properly understood by reducing our explanations to behavioral, environmental, psychological, and biological mechanisms linking the two and that the effect of socioeconomic status on mortality cannot be eliminated by addressing the mechanisms that happen to link the two at a particular moment in time.

Our argument arises from an examination of historical patterns, which suggest that mechanisms linking socioeconomic status with mortality do not operate according to our usual expectations of causal processes. Imagining a path model with socioeconomic status as the distal factor linked to death by more proximal risk factors, we would expect that, if the proximal risk factors are eliminated as causal agents, the association between socioeconomic status and mortality should also be eliminated. On the contrary, we have seen instances in which major proximal risk factors have been eliminated, yet socioeconomic disparities in mortality remain as strong as ever. In the 19th century, overcrowding, poor sanitation, and infectious disease appeared to explain the higher mortality rates of less advantaged persons. However, after those important causes of mortality were virtually eliminated in developed countries, with dramatic declines in deadly diseases such as diphtheria, measles, typhoid fever, tuberculosis, and syphilis, socioeconomic inequalities in mortality

remained undiminished (Rosen 1979). Mechanisms "explaining" the association between socioeconomic status and mortality were dramatically modified, independent of socioeconomic status, thus severing or reducing the link between socioeconomic status and the intervening mechanism. Our path-model approach would lead us to expect a substantial reduction in the association between socioeconomic status and mortality, but this did not occur.

How can this be explained? At one level, the answer is that the risk factors that used to mediate the association have been replaced by other factors, such as health behaviors and psychosocial stress (Adler et al. 1994). This situation is reminiscent of Lieberson's (1985) "basic causes," which he said have enduring effects on a dependent variable because, when the effect of one mechanism declines, the effects of others emerge or become more prominent. House and colleagues first suggested that such a process might produce the enduring association between socioeconomic status and mortality (House et al. 1990, 1994). Lieberson's notion of basic causes, however, is a general concept that does not offer clues as to what it is about any particular basic cause that allows it to reproduce its effects despite the elimination of intervening mechanisms. What is it about socioeconomic status that renders it a basic, or as Link and Phelan (1995) called it, a "fundamental cause" of mortality?

SOCIOECONOMIC STATUS AS A FUNDAMENTAL CAUSE OF MORTALITY

Link and Phelan (1995) argued that new mechanisms arise, leading to persistent socioeconomic differences in mortality, because persons of higher socioeconomic status possess a wide range of broadly serviceable resources, including money, knowledge, prestige, power, and beneficial social connections, that can be used to one's health advantage. These resources directly shape individual health behaviors by influencing whether people know about, have access to, can afford and are motivated to engage in health-enhancing behaviors. Current examples include knowing about and asking for beneficial health procedures; quitting smoking; getting flu shots; wearing seat belts and driving a car with airbags; eating fruits and vegetables; exercising regularly; and

taking restful vacations. In addition, resources shape access to broad contexts such as neighborhoods, occupations, and social networks that vary dramatically in associated profiles of risk and protective factors. For example, low-income housing is more likely to be located near noise, pollution, and noxious social conditions and less likely to be well served by police, fire, and sanitation services; blue-collar jobs tend to be more dangerous and stressful than white-collar jobs and to carry inferior health benefits; and social networks with high-status peers are less likely to expose a person to second-hand smoke, more likely to support a health-enhancing lifestyle, more likely to inform a person of new health-related research, and more likely to connect him or her to the best physicians. Moreover, being embedded in a social context where neighbors, friends, family members, and co-workers can generally look forward to a long and healthy life surely contributes to an individual's motivation to engage in health-enhancing behaviors (Wilson and Daly 1997).[2] In short, there is a long and detailed list of mechanisms linking socioeconomic status and mortality, most of which favor people of more advantaged circumstances (Lutfey and Freese Forthcoming).

Because of the wide-ranging utility of these varied resources, socioeconomic status at any given time influences multiple health outcomes through multiple risk and protective mechanisms. Moreover, because of the general nature of these resources, they are adaptable to changing health-related conditions and can be used to protect health no matter what the current risks, treatments, or diseases are—whether these be crowded, vermin-infested living conditions in the 19th century, a job with high psychological demands and low decision latitude in the 20th century, or whatever health risks we face in the new millennium. Thus, even though existing mechanisms linking socioeconomic status and mortality may be eliminated, when new medical interventions become available or new knowledge about existing risk and protective factors emerges, those with greater resources are usually better able to take advantage of those interventions or information to improve health and extend life. It is the flexible and multi-purpose nature of these resources that allows the association between socioeconomic status and mortality to persist despite changes in the particular diseases, treatments, and risk and protective fac-

tors that dominate at any particular time (Lutfey and Freese Forthcoming).

From this perspective it is important to understand and address the risk factors that mediate the association between socioeconomic status and mortality at any given time, because addressing these risk factors may improve the current situation with regard to mortality disparities, and neglecting them may make it worse. However, we cannot expect these measures to lead to long-lasting reductions in socioeconomic disparities in mortality.

The "fundamental cause" explanation posits that the use of resources to benefit health, by groups and individuals, is purposeful.[3] Thus, the health advantage of high socioeconomic status is not primarily a coincidental side-effect of "the good life." Although there may be some inherent health benefits of greater social and economic resources, such as having more living space, greater opportunities for rest and relaxation, or other as yet undiscovered factors, we argue that the deliberate use of resources by individuals and groups to benefit health is essential in producing the enduring association between socioeconomic status and mortality. For example, through most of history, people with higher socioeconomic status have tended to eat a diet rich in meat, protein, and fat. Such a diet was relatively expensive, was considered both appetizing and healthful, and could have been thought of as an inherent advantage of the well-to-do. However, with the emergence of evidence that too much fat is unhealthful, individuals of high socioeconomic status have increasingly adopted a "poor person's diet" (Popkin, Siega-Riz, and Haines 1996). Similarly, freedom from physical labor has long been seen as a privilege of wealth and status, but with reports of the importance of exercise for good health, regular trips to well-appointed health clubs increasingly replace more sedentary activities previously enjoyed by individuals with high socioeconomic status. The "good life" has changed.[4]

EMPIRICALLY EVALUATING THE VALIDITY OF THE THEORY

It is clear from the historical patterns cited above that a simple mediating risk-factor model is not adequate to explain the enduring association between socioeconomic status and mortality: When important mediating risk fac-

tors have been reduced or eliminated, socioeconomic gradients in mortality have remained undiminished. However, the question remains whether the theory of fundamental causes can successfully explain the persistent association. This theory offers a specific explanation for the association between socioeconomic status and mortality. Evidence supporting or refuting the theory must be equally specific, and tests must focus on component hypotheses within the theory. Here we discuss what evidence is needed to test the theory, what evidence exists, and what evidence will be provided in this paper.

A fundamental social cause of mortality, according to Link and Phelan, has four features. First, it influences multiple disease outcomes. Second, it affects these disease outcomes through multiple risk factors. Third, the association between the fundamental cause and mortality is reproduced over time via the replacement of intervening mechanisms. Finally, the "essential feature of fundamental social causes is that they involve access to resources that can be used to avoid risks or to minimize the consequences of disease once it occurs" (Link and Phelan 1995:87). We now examine the state of the evidence regarding each of these propositions.

Association of Socioeconomic Status with Multiple Risk Factors and Multiple Disease Outcomes

The first two propositions are strongly supported by existing data. Both currently and in the past, low socioeconomic status is known to be related to numerous risk factors for potentially fatal diseases or other causes of death, for example, smoking, sedentarism, and being overweight (Lantz et al. 1998); stressful life conditions (Turner, Wheaton, and Lloyd 1995; House and Williams 2000); social isolation (House and Williams 2000; Ruberman et al. 1984); preventive health care (Dutton 1978; Link et al. 1998); and crowded and unsanitary living conditions, unsanitary water supplies, and malnutrition (Rosen 1979). Lower socioeconomic status is also related to a multiplicity of diseases and other causes of death. Two indicators of this general relationship are that low socioeconomic status is related to mortality from each of the broad categories of chronic diseases, communicable diseases, and

injuries (Pamuk et al. 1998) and from each of the 14 major causes of death in the International Classification of Diseases (Illsley and Mullen 1985).

Reproduction of the Association Between Socioeconomic Status and Mortality Through Replacement of Mechanisms

As noted, historical evidence is compelling that there has been a consistently strong socioeconomic gradient in mortality at least since the early19th century (Antonovsky 1967) and that dramatic changes in the risk factors linking socioeconomic status and mortality have occurred over this time. These broad facts are consistent with fundamental cause theory.

Moreover, there have been several important instances in which socioeconomic gradients in specific risk factors and disease outcomes have shifted as predicted by the theory, that is, following the emergence of knowledge about health-risk and protective factors, the new knowledge and benefits are utilized disproportionately by groups with high socioeconomic status, resulting in shifts in health and mortality gradients that benefit groups with higher socioeconomic status. As we have noted, when knowledge about the health benefits of a low-fat diet emerged, groups with higher socioeconomic status altered their diets more than did groups with lower socioeconomic status (Popkin et al. 1996). Similarly, as knowledge about the risks of smoking emerged, people of higher socioeconomic status were more likely to stop or not to start smoking, leading to a growing inverse association between socioeconomic status and smoking (Pierce et al. 1989). Concomitant with these changes, the association of socioeconomic status to coronary heart disease shifted dramatically, changing from a direct to an inverse association (Beaglehole 1990). Also, whereas lung-cancer mortality was not related to socioeconomic status as late as the early 1930s, a large inverse association emerged, particularly beginning in the 1950s and 1960s (Logan 1982).

HIV/AIDS is another example. Early in the epidemic, mortality from AIDS was not strongly related to socioeconomic status. However, as we learned about the transmission and prevention of HIV infection, HIV/AIDS has increasingly become concentrated in poor regions of the world and among poor and mar-ginalized groups in richer regions of the world (Fife and Mode 1992; UNAIDS/WHO 2002).

More systematic analysis is needed on this component of the theory to insure that the examples cited, although important ones, are not a biased set of cases that happen to support the theory. However, we can say that the evidence cited in this section is supportive of the dynamic aspect of fundamental cause theory and that we know of no evidence that contradicts it.

The Role of Resources

Link and Phelan regard the key feature of fundamental social causes to be the fact that the mortality advantage enjoyed by people with higher socioeconomic status lies in their greater access to and effective utilization of resources such as money, knowledge, power, prestige, and beneficial social connections. Access to and purposeful utilization of these resources to benefit health is the central mechanism by which individuals with higher socioeconomic status attain greater longevity. It is because these resources are flexible and can adapt to changing health-related conditions that the socioeconomic-status-mortality gradient persists across time and despite changing conditions. To our knowledge, this critical component of the theory—the importance of the effective utilization of resources—has not been directly tested, and it is the purpose of this paper to do so. Empirically testing the importance of resources *per se* is difficult, because it requires the identification of situations in which the ability to use socioeconomic resources can be analytically separated from socioeconomic status itself. We already know from numerous studies that higher socioeconomic status is associated with lower mortality rates—that is the fact we wish to understand. To test the theory, we need to demonstrate the role of the utilization of resources of knowledge, power, money, prestige and beneficial social connections in a way that is empirically distinguishable from socioeconomic status itself. Fortunately, although they are uncommon, situations can be identified in which socioeconomic resources cannot be used to prolong life—when even the richest or most powerful person on earth cannot use those resources to escape death. Such situations can provide useful information regarding the

importance of resources in producing socioeconomic differences in mortality. If the utilization of resources is critical in prolonging life, then, in situations in which the resources associated with higher status are of no use, high socioeconomic status should confer no advantage, and the usually robust association between socioeconomic status and mortality should be greatly reduced. One situation in which the advantages of higher socioeconomic status cannot be used to extend life occurs when the causes and cures of fatal diseases are unknown. In these circumstances, socioeconomic resources cannot be used to extend life, because it is not known how those resources should be directed. This reasoning suggests an empirically testable prediction. To the extent that the ability to utilize socioeconomic resources is critical in maintaining socioeconomic disparities in mortality, there should be strong socioeconomic gradients in mortality for causes of death that are highly preventable—for which we have good knowledge and effective measures for prevention or treatment. However, for causes of death about which we know little regarding prevention or treatment, socioeconomic gradients in mortality should be much weaker.[5]

By focusing our test on differential predictions of the association between socioeconomic status and mortality for causes of death that are more and less preventable, we distinguish between the theory of fundamental causes, which emphasizes the use of resources for health benefits, and other explanations that do not consider this health-specific use of resources to be central (Link and Phelan 2000). For example, if high socioeconomic status enhances health primarily via the psychological benefits of holding a favorable location in a social hierarchy (Adler 1994; Wilkinson 1997), or via any psychological, social, or material benefits that do not involve the direct use of resources to benefit health, there is no reason to predict a reduction in the socioeconomic-status-mortality gradient for less preventable diseases. Hierarchical status and the actual resources that a person holds are not influenced by the preventability of death from a particular cause, only the ability to use those resources to prolong life.

Some mortality benefits of high socioeconomic status may result from more general advantages associated with socioeconomic status rather than from purposive actions taken to prevent or delay death, and such benefits will attenuate the difference between high and low preventability causes of death in their association with socioeconomic status. Nevertheless, to the extent that socioeconomic resources and their utilization are important determinants of socioeconomic differences in mortality (as specified by the theory), these differences should be significantly stronger for causes of death for which effective risk or protective factors have been identified. If this is not the case, the theory is seriously challenged. If socioeconomic status predicts mortality just as strongly when the ability to utilize resources to prolong life is blocked, this fact will argue against the central role of the utilization of resources.

The inverse association between socioeconomic status and mortality has proved extremely robust across time, place, and cause of death (Antonovsky 1967; Black et al. 1982; Marmot et al. 1984; Sorlie et al. 1995). Based on the theory of fundamental causes, we identify a condition—when relatively little can be done to prevent death from a particular cause—in which this association should be substantially diminished. In this paper, we utilize data from the National Longitudinal Mortality Study and expert ratings of the preventability of death from specific causes to examine the hypothesis that socioeconomic status is more strongly inversely related to mortality from causes that are more preventable, as compared to causes that are less preventable.

METHODS

Sample

The National Longitudinal Mortality Study (Sorlie et al. 1995; Rogot et al. 1992) is a prospective study that uses samples of selected Current Population Surveys, conducted by the U.S. Bureau of the Census (1978). The Current Population Survey samples the noninstitutionalized U.S. population using a complex probability sample of households that are surveyed monthly to obtain demographic and economic information. Surveys are conducted by personal and telephone interviews. Response rates are extremely high, averaging around 96 percent. Our analysis uses the National Longitudinal Mortality Study Public Use File (release 2, October 1995), based on five

Current Population Surveys conducted between 1979 and 1981. The National Longitudinal Mortality Study matched the Current Population Survey samples to the National Death Index to determine occurrences and causes of death in the follow-up period of approximately nine years. The National Death Index is a computer file of all deaths in the United States since 1979, maintained by the National Center for Health Statistics (1990). There is evidence that the Index provides accurate information on deaths when personal identifiers are used, as they are in the National Longitudinal Mortality Study (Stampfer, Willett, and Speizer 1984). Probably because the National Longitudinal Mortality Study excludes institutionalized individuals and the National Death Index excludes some deaths, mortality rates for the National Longitudinal Mortality Study are slightly lower than for the U.S. population.

The National Longitudinal Mortality Study (release 2) includes 637,162 cases. Because educational attainment is one of our measures of socioeconomic status, we excluded persons under age 25 to minimize the number whose education was not complete at the time of interview. Table 1 shows the distribution of study variables and other characteristics for the remaining 370,930 subjects. Of these, 2,345 were missing data on education and 22,501 were missing data on family income, leaving totals of 368,585 for analyses involving education and 348,429 for those involving income. The maximum follow-up time was 3,288 days (just over nine years), and the mean was 3,111 days.

Measures

Socioeconomic status. We operationalized socioeconomic status as family income and educational attainment. Family income (adjusted to 1980 dollars using the Consumer Price Index) is the combined income of all family members during the 12 months prior to interview. The seven response categories ranged from less than $5,000 to $50,000 or more. Education is the highest grade or year of regular school completed. For our analyses, education categories were 7 or fewer years, 8 years, 9 to 11 years, 12 years, 13 to 15 years, 16 years, and 17 or more years.

Preventability of death. Testing our hypothesis requires cause-specific ratings of the pre-

ventability of death during the National Longitudinal Mortality Study follow-up period (1980s). Rutstein and colleagues (1976, 1980) published a set of ratings that sought to identify health events whose occurrence should provide an alarm that something was awry in the delivery of health care. These ratings of "sentinel events," including preventable causes of death, have been used effectively in several studies. However, given rapid changes in prevention and treatment, we wanted updated ratings. Moreover, our hypothesis applies to all causes of death, whereas Rutstein et al.'s ratings exclude some causes ("medico social problems," such as substance use and homicide, as well as accidental deaths.)

We therefore derived new ratings of death preventability as follows: The National Longitudinal Mortality Study includes the underlying cause of death from the death certificate, coded according to the International Classification of Diseases, 9th revision (ICD-9) (World Health Organization 1977). One of us (Ana Diez-Roux) reviewed the causes of

TABLE 1. Characteristics of Subjects Aged 25 Years or Older at Baseline (National Longitudinal Mortality Study)

Variable	N	%
Education		
0–7 years	32,498	8.8
8 years	30,585	8.3
9–11 years	50,531	13.7
12 years	136,761	37.1
13–15 years	56,229	15.3
16 years	35,822	9.7
17+ years	26,159	7.1
Family income		
less than $5000	35,752	10.3
$5000 to $9999	55,408	15.9
$10,000 to $14,999	59,438	17.1
$15,000 to $19,999	49,702	14.3
$20,000 to $24,999	50,023	14.4
$25,000 to $49,999	81,894	23.5
$50,000 or more	16,212	4.7
Ethnicity/race		
Black	32,757	8.8
White	328,002	88.5
Other	10,041	2.7
Female	197,055	53.1
Age at baseline		
25–44	177,323	47.8
45–64	124,594	33.6
65–99	69,013	18.6
Died during follow-up	41,554	11.2
High preventability causes	28,009	7.6
Low preventability causes	6,999	1.9
Other (unrated) causes	6,546	1.8
Total	370,930	

death (three-digit codes) in the National Longitudinal Mortality Study sample, eliminating causes she judged too heterogeneous to allow accurate ratings of death preventability and combining some codes she judged to be relatively homogeneous in terms of treatment and preventive measures. Causes accounting for fewer than 10 deaths in the National Longitudinal Mortality Study were eliminated. The 96 remaining causes cover 84 percent of deaths in the sample. These causes were independently rated by Ana Diez-Roux and Ichiro Kawachi (both physician/epidemiologists) in terms of the degree to which death was amenable to prevention or delay during the 1980s in the United States. Three ratings were made by each rater for each cause of death. First, they rated the degree to which that cause of death was preventable "by means of medical treatment or other interventions administered after the disease had been detected." Second, they rated preventability of death "by preventing the incidence of the disease—for example by good hygiene, diet, other lifestyle factors, vaccines, etc." Finally, they considered both sets of factors to make an overall rating of preventability. Causes were rated from 1 ("virtually impossible to prevent death") to 5 ("virtually all deaths preventable.") Here we employ the overall rating. Inter-rater reliability for the mean of the two raters, as assessed with the intraclass correlation coefficient, was .85. For the present analyses, causes were dichotomized into high preventability (mean preventability rating of 4 or greater; 58% of rated causes) and low preventability (mean rating of 3.5 or less; 42% of causes). We chose this cut-point to be as close as possible to the midpoint of the rating scale while maintaining roughly the same number of causes in both categories. See Appendix A for preventability ratings.

As a validity check, we correlated our ratings with Rutstein et alia's (1980). Comparing our dichotomous measure (high vs. low preventability) to Rutstein et alia's measure (also dichotomous), there were 59 agreements and 17 disagreements (phi = .57). Thirteen causes were rated highly preventable by us but not by Rutstein's group. Four causes were rated as sentinel events (i.e., highly preventable) by Rutstein et al. but low in preventability by us: cancer of the rectum, cancer of the uterus, aplastic anemia, and chronic pulmonary heart disease. Each of these four was rated at or just below our cutoff for low preventability (3.5 or 3.0). Our five-point scale was correlated .57

with Rutstein et alia's ratings. Given our broader scope and more recent time period, we consider the correspondence between the two sets of ratings to be good and the pattern of disagreements expectable.

Analysis

Life table analyses were performed by dividing the follow-up period into six-month intervals and dividing education and family income into three categories (0 to 11 years, 12 to 15 years, and 16 or more years of education; less than \$10,000, \$10,000 to 24,999, and \$25,000 or more of income). For each of three age groups (25 to 44, 45 to 64, and 65 to 98) and for each socioeconomic-status category, survival curves were plotted for high and low preventability causes of death. When death from high preventability causes was the event of interest, individuals who died of low preventability causes or unrated causes or who did not die were censored. When death from low preventability causes was the event of interest, individuals who died of high preventability causes or unrated causes or who did not die were censored.

We used Cox proportional hazards models to estimate the age-adjusted relative risk of mortality from high preventability causes and from low preventability causes associated with decreasing levels of education and family income for each age group. Education (in seven categories as described above) and family income (in seven categories as described above) were treated as sets of six dummy variables, with the highest socioeconomic-status category omitted in each case. Individuals who did not die or who died of causes other than the type being predicted were censored.

Our key test involves comparing the magnitude of the mortality risk associated with socioeconomic status for two different categories of events (deaths from high and from low preventability causes). In standard Cox regressions, these two types of events must be predicted in separate equations, providing no opportunity for a significance test for the difference in magnitude of the effect of socioeconomic status. To obtain a significance test, we conducted polytomous conditional logistic regression (Levin 1987, 1990), with death from high preventability causes, low preventability causes, other causes, and no death

as outcome categories. For each socioeconomic-status measure, the equivalent of a random-effects meta-analysis was prepared, treating each of the 74 age cohorts (25 to 99 years old at baseline) as individual studies. The meta-analysis produces an overall weighted average (across age cohorts) of the differences between log relative risk associations (with education or income) for high vs. low preventability causes of death. The weighted average difference in log relative risks and its standard error test the null hypothesis that the relative risks associated with socioeconomic status are equal for high and low preventability causes.

RESULTS

Socioeconomic Status, Mortality, and Preventability of Death

Figures 1 through 6 provide graphical displays of mortality rates. Figure 1 shows cumulative survival from high and low preventability causes of death, for three levels of education, for persons who were between 25 and 44 years of age at baseline. Figures 2 and 3 show comparable results for ages 45 to 64 and 65 to 98, and Figures 4, 5, and 6 show those for family income.

The figures show, first, that cumulative sur-vival declines more steeply for high preventability causes. Although we dichotomized causes of death above the midpoint of our scale, more people died from high preventability than from low preventability causes. Second, cumulative survival declines more rapidly at low socioeconomic levels for both high and low preventability causes of death. Our primary question is whether socioeconomic status is more strongly related to survival when death is more preventable. Visual inspection of Figures 1–6 suggests this is so: For each age group and socioeconomic-status measure, gaps between survival curves for different levels of socioeconomic status are much larger for high preventability causes of death.

Perhaps, however, the low preventability curves are more tightly packed together simply because they do not fall as low as those for high preventability causes. Perhaps the *relative* risk associated with low socioeconomic status is just as great for low preventability as for high preventability causes. Table 2 shows that this is not true. Table 2 reports the results of Cox proportional hazards models predicting high and low preventability deaths from age and education and from age and family income, for each of the three age groups. We report the risk of death for someone in each education category relative to someone in the highest category and for someone in each fam-

FIGURE 1. Cumulative Survival by Education and Preventability of Death Ages 25 to 44 at Baseline
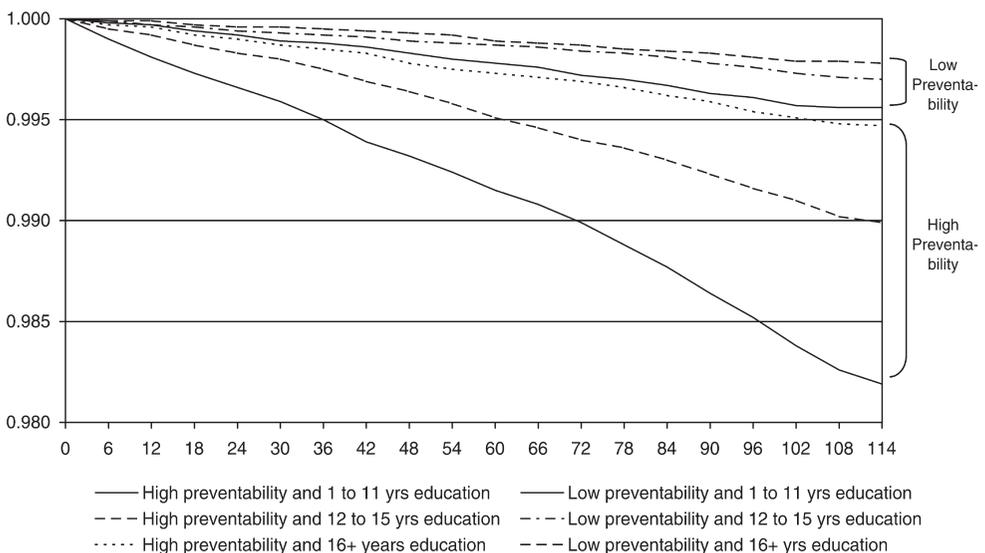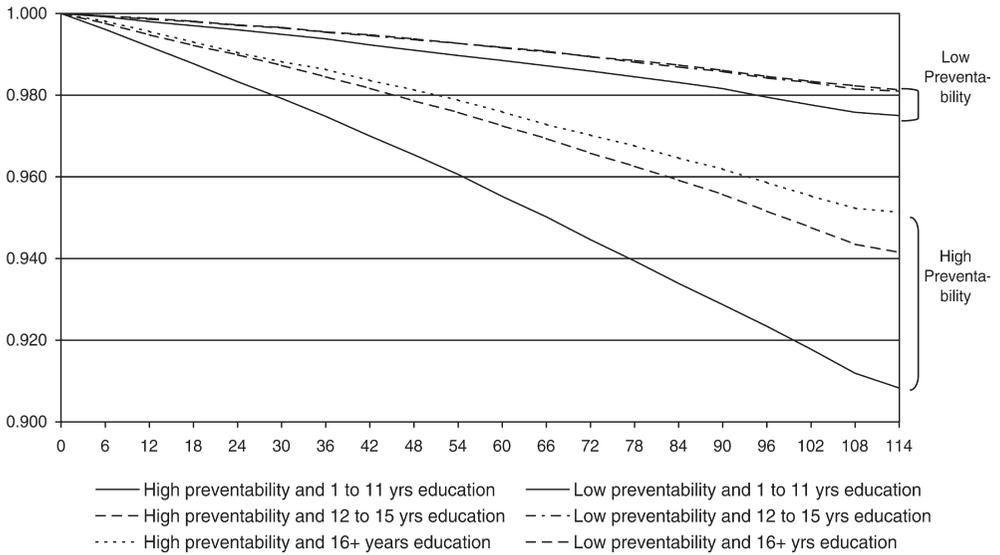
FIGURE 2. Cumulative Survival by Education and Preventability of Death Ages 45 to 64 at Baseline

ily income category relative to someone in the highest category.

We hypothesize that socioeconomic status is more strongly inversely related to mortality when death is more preventable. Thus, our main interest lies in the contrast between the relative risk of mortality for high and low preventability causes of death. Consistent with this hypothesis, for each age group and socioe-conomic-status measure, the risk associated with low socioeconomic status is larger for high preventability deaths than for low preventability. As we progress from high to low socioeconomic status, risk ratios generally increase for both high and low preventability causes, but the risk ratios become larger for high preventability causes, usually much larger.[6] For high preventability causes, many more

FIGURE 3. Cumulative Survival by Education and Preventability of Death Ages 65 to 99 at Baseline
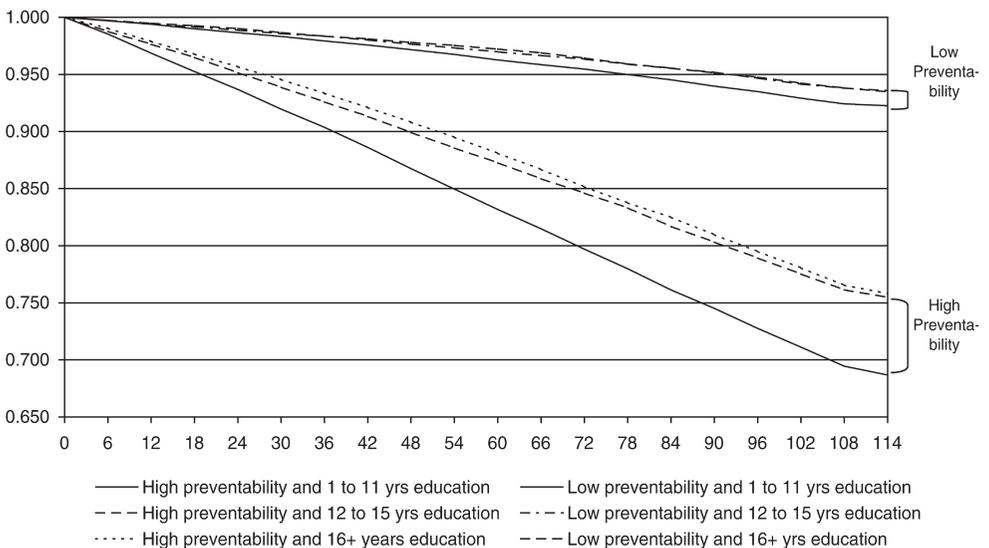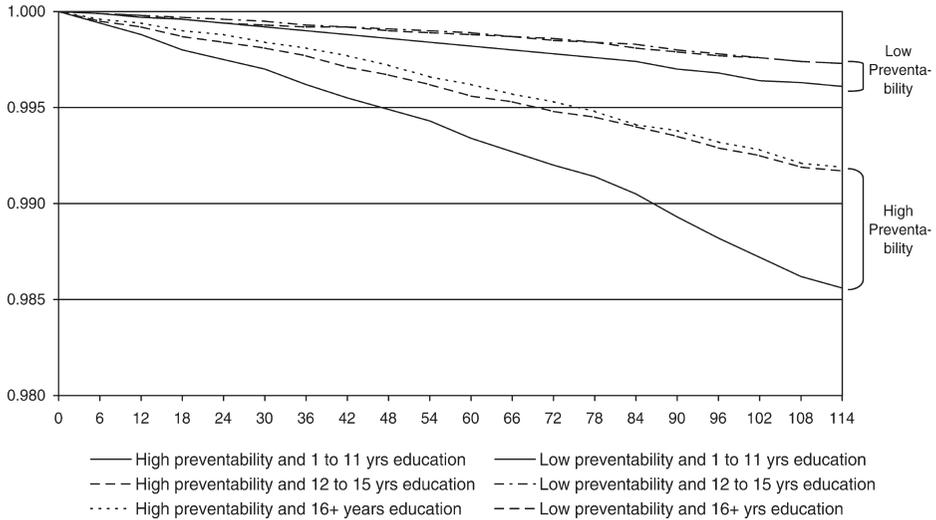
FIGURE 4. Cumulative Survival by Family Income and Preventability of Death Ages 25 to 44 at Baseline

of the lower socioeconomic-status categories differ significantly from the high-socioeconomic status comparison category in terms of morality risk. In fact, for three of the six sets of analyses (education for the middle and old age groups and family income for the old age group), none of the socioeconomic-status categories differed significantly from the highest for low preventability causes, but 14 of the possible 18 comparisons were significant for high preventability causes. In addition, the risk ratios usually progress in a nearly monotonic manner for high preventability causes, whereas this is much less true for the low preventability causes.

These contrasts between high and low preventability were tested formally for statistical significance with polytomous conditional

FIGURE 5. Cumulative Survival by Family Income and Preventability of Death Ages 45 to 64 at Baseline
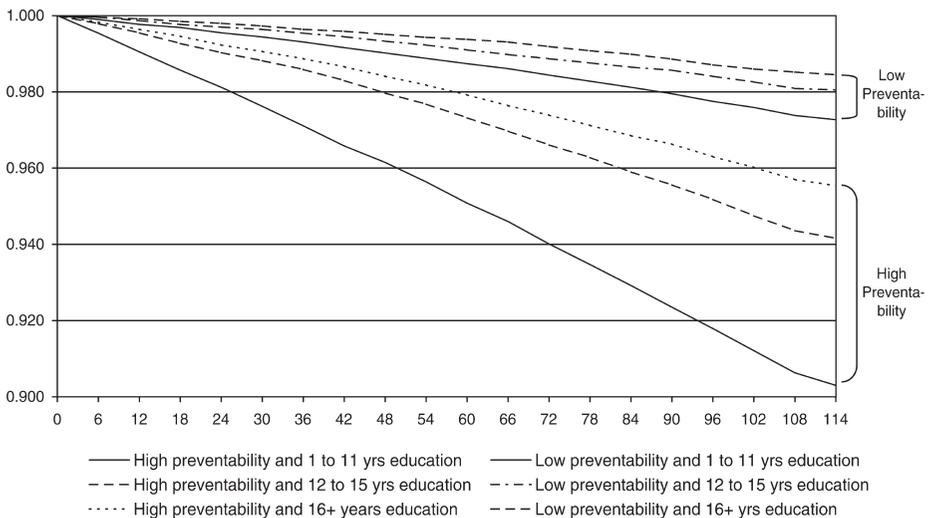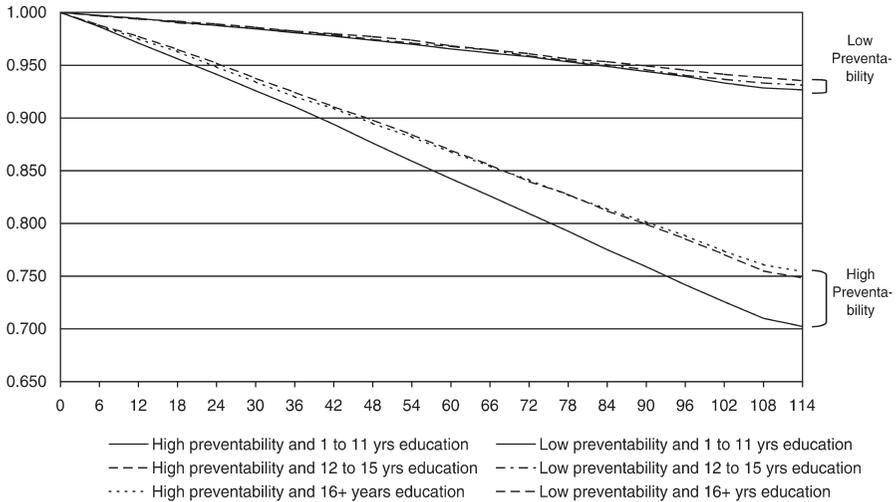
FIGURE 6. Cumulative Survival by Family Income and Preventability of Death Ages 65 to 99 at Baseline

logistic regressions (Levin 1987, 1990). The weighted mean (across age cohorts) of the relative risk of death per unit decrement in education level for high preventability causes is 4.9 percent greater than the mean relative risk for low preventability causes. The mean relative risk of death per $10,000 decrement in family income across age cohorts for high preventability causes is 2.9 percent greater that

for low preventability causes. Both these effects are highly significant ($p < .000001$). A 95 percent confidence interval for the percentage excess for education is 2.9 percent to 6.9 percent and for income is 1.8 percent to 4.1 percent.

We assessed the generality of these findings in three ways. First, we assessed generalizability across racial/ethnic and gender groups. We

TABLE 2. Cox Proportional Hazards Models Predicting Death from Age and Educational Attainment and from Age and Family Income (National Longitudinal Mortality Study)

	Ages 25 to 44 (n = 177,323) Death Preventability		Ages 45 to 64 (n = 124,594) Death Preventability		Ages 65 to 99 (n = 69,013) Death Preventability	
	High	Low	High	Low	High	Low
Years of Education:						
< 8	3.53***	1.31	2.27***	1.15	1.34***	1.14
8	3.82***	2.17**	2.00***	1.21	1.39***	1.12
9–11	3.24***	1.99***	1.93***	1.14	1.29***	1.11
12	2.18***	1.52*	1.41***	.96	1.19***	1.03
13–15	2.00***	1.25	1.36***	.98	1.13*	.96
16	1.24	1.14	1.14	.92	1.10	1.04
17+	1.00	1.00	1.00	1.00	1.00	1.00
Family Income:						
< $5,000	3.56***	3.17***	2.81***	1.86***	1.21**	1.17
$5,000–$9,999	2.39***	2.09*	2.15***	1.58***	1.29***	1.21
$10,000–$14,999	1.73***	2.52***	1.90***	1.39**	1.17*	1.19
$15,000–$19,999	1.38*	1.82*	1.54***	1.24	1.17*	1.30
$20,000–$24,999	1.11	1.47	1.39***	1.30*	1.10	1.09
$25,000–$49,999	1.07	1.46	1.26***	1.10	1.12	1.15
$50,000+	1.00	1.00	1.00	1.00	1.00	1.00

* $p < .05$; ** $p < .01$; *** $p < .001$
Note: Age-adjusted risk ratios shown.

divided the sample into six groups (black, non-hispanic white, and hispanic men and women) and used Cox proportional hazards models to predict high and low preventability deaths from education and from family income, combining the three age groups and controlling age at baseline. In 11 of the 12 comparisons (two socioeconomic-status measures for each of six ethnic-gender groups), socioeconomic status was more strongly related to mortality from high preventability than from low preventability causes, indicating that the results are largely generalizable across racial/ethnic and gender groups. (In the 12th comparison, for black men, the magnitude of the association was slightly larger for low preventability causes, but the significance of the association was greater for high preventability causes).

Second, we repeated the Cox proportional hazards models, as shown in Table 2, substituting Rutstein et alia's preventability ratings. In all six comparisons, socioeconomic status was more strongly related to mortality from high preventability than from low preventability causes.

Third, we wanted assurance that our results were not driven by a few very common causes of death. Ischemic heart disease (high preventability) is the only cause that accounts for a large proportion (27 percent) of the deaths. Next most common were cerebrovascular diseases, accounting for 7 percent of deaths. Because ischemic heart disease weighs so heavily in the overall results, we repeated the Cox proportional hazards models as shown in Table 2, this time omitting it. Our central finding—that socioeconomic status is more strongly associated with mortality from high preventability causes—was slightly more pronounced in these analyses.

Age Patterns

Figures 1–6 and Table 2 indicate that associations between socioeconomic status and mortality vary by age. These patterns were analyzed more systematically in two ways. First, we ran Cox proportional hazards models, pooling all age groups and predicting all-cause mortality from socioeconomic status, age, and their interaction. These analyses indicated that the association between socioeconomic status and mortality varied significantly with age ($p < .01$ for both education and family income)

and that the survival advantage conferred by higher socioeconomic status diminished with age and actually disappeared at age 85 for education and at age 83 for family income. Similarly, including a quadratic term for age in our polytomous conditional logistic regressions indicated a quadratic relationship such that the greater socioeconomic advantage for high preventability causes of death diminished with age and disappeared at age 81 for education and 84 for income. The quadratic age-by-education interaction term for the difference in log relative risks for high versus low preventability causes of death was significant ($p < .05$). The quadratic age-by-income term was smaller, with $p = .081$.

DISCUSSION

This paper argues for the necessity of sociological theory and analysis in answering a life and death question—the strong, longstanding, and pervasive association between socioeconomic status and mortality. Research and thinking on this question are currently dominated by medicine and epidemiology and are usually approached in a way that denies a central irreducible causal role for socioeconomic status. Commonly, socioeconomic status is either dismissed as not causally relevant, or researchers focus on mechanisms that mediate between socioeconomic status and mortality in the hopes of reducing mortality disparities by intervening on those mechanisms. However, the fact that the strong association between socioeconomic status and mortality has proved impervious to major changes in intervening mechanisms over history suggests that socioeconomic status plays an irreducible and "basic" (Lieberson 1985) causal role in mortality. Consequently, we have pursued an approach that focuses squarely on socioeconomic status rather than on mediating variables.

Support for Fundamental Cause Theory

According to the theory of fundamental causes (Link and Phelan 1995), a strong association between socioeconomic status and mortality has persisted over time despite radical changes in diseases and their risk factors, because people of higher socioeconomic status

can avail themselves of a wide range of socioeconomic resources that can be used to protect health and prolong life no matter what the current profile of risks, treatments, and diseases. In this paper, we tested what Link and Phelan (1995) identified as the key explanatory feature of the theory—the idea that resources and their utilization play a central role in maintaining socioeconomic differences in mortality. To do so, we sought to identify a situation in which individuals with higher socioeconomic status should not be able to utilize their resources to delay death. We reasoned that this would occur when little is known about the causes and cures for particular diseases. We predicted that for causes of death about which we know little in terms of prevention and treatment, the near ubiquitous association between socioeconomic status and mortality would be strongly diminished. Our results confirmed this prediction. Mortality from causes of death that are more preventable, as reliably rated by a pair of physician-epidemiologists, were substantially and significantly more strongly related to socioeconomic status than mortality from causes that are less preventable. These findings held across gender and racial/ethnic groups, using an alternate measure of preventability, and omitting the one cause of death that accounted for a large number of deaths in the sample.

Our confidence in the validity of the findings is strengthened by the quality of our data. The National Longitudinal Mortality Study is very large and nationally representative, and response rates are excellent. The measures of socioeconomic status come from the Current Population Survey conducted by the Census Bureau for the express purpose of tracking economic and labor-force characteristics. Deaths are ascertained through the National Death Index, which provides a comprehensive coverage of deaths that occur in the United States. While there may be inaccuracies in the underlying cause of death recorded on death certificates, the present analyses are based on high and low preventability groupings of causes of deaths, each of which comprises over 40 individual causes. For inaccuracies in recorded cause of death to affect our results, there would have to be a significant amount of systematic misclassification across the cut-point for our dichotomized measure, which seems unlikely.

Our findings also cohere with those of other studies that have focused on death from causes amenable to medical intervention (a subset of Rutstein et alia's 1980 sentinel events) and on avoidable hospitalization. Charlton and Velez (1986), looking at changes in mortality rates for six western countries from 1950 to 1980, found that mortality from "amenable causes" declined faster than mortality from all other causes, suggesting that the ability to intervene medically reduces mortality. Mackenbach, Stronks, and Kunst (1989), using data for England and Wales between 1931 and 1981, found that mortality from conditions that became amenable to medical intervention declined more in relative terms among higher occupational classes than among lower ones, suggesting that the ability to intervene disproportionately benefits the higher social classes. To our knowledge, the previous analysis that is most similar to ours was conducted by Marshall et al. (1993). They compared socioeconomic-status-mortality gradients for amenable and other causes using a sample of employed men in New Zealand. Marshall and colleagues found that occupational class inequalities were more pronounced for amenable causes of mortality than for all-cause mortality. The gradient for amenable mortality was about twice as steep as for all-cause mortality, a magnitude of difference that is consistent with our findings. Although all these studies differ from ours in using a somewhat narrower measure of death preventability that excludes accidental deaths and "medicosocial problems" such as substance use and homicide, the consistency of their findings with ours provides reassurance as to the validity and generalizability of our results. Our findings are also consistent with studies (Pappas et al. 1997) that have found significant socioeconomic disparities in rates of potentially avoidable hospitalizations.

Although it was not the focus of this paper, another clear pattern in the findings is that socioeconomic inequalities in mortality diminish at older ages. These findings are consistent with those of previous investigations (Antonovsky 1967; Haan et al. 1987; Sorlie et al. 1995; Sorlie, Backlund, and Keller 1995). Two explanations for these findings are currently considered to be the most plausible (see Lauderdale 2001). First, House et al. (1990) argue that socioeconomic disparities in health may be most pronounced in middle and early old age because socioeconomic variations in exposure to and impact of environmental and

psychosocial risk factors are greatest during this age period. Second, some analyses suggest that the diminished association between education and mortality at older ages that are typically found in cross-sectional or relatively short-term longitudinal studies are due to period effects rather than age effects (e.g., Lauderdale 2001). Lauderdale (2001) and Mirowsky and Ross (2003) argue that, over time, health-enhancing advances are developed that disproportionately favor those with more education. Because, at a given time, younger people have lived more of their lives in a period in which health-enhancing knowledge and technology is more advanced, and because highly educated people benefit most from that knowledge, those who are younger at a given time have benefitted more from education than those who are older. The same argument could be made for family income (which shows essentially the same pattern of diminishing associations with mortality at older ages), if one assumes that relative income stays fairly stable across the lifetime. We offer a third possibility: As when we do not know how to prevent or treat life-threatening illness, old age can be seen as an instance in which socioeconomic resources are of limited use in prolonging life. In the case of low preventability causes of death, available resources are not useful because it is unclear how to direct them. In the case of old age, it is known how resources should be directed, but at some point, the growing frailty of the body places limits on the effectiveness of interventions. Each of these explanations is also consistent with our finding (not addressed in previous studies) that socioeconomic advantage diminishes more for high preventability causes, such that the incremental advantage of socioeconomic status for high preventability causes, as compared to low preventability, also diminishes with age and disappears when people reach their early 80s. The current data do not allow us to adjudicate between these three explanations. We note, however, that each explanation is consistent with fundamental-cause theory.

Qualifications and Limitations

First, although associations between mortality and socioeconomic status were much weaker for low preventability causes of death, those associations were still statistically significant in many cases. Because only one relatively uncommon cause of death (malignant neoplasm of gallbladder and extrahepatic bile ducts) was rated as completely unpreventable by both raters, we based our test on a comparison of more- and less-preventable causes of death rather than preventable and unpreventable causes. Thus, it is expected that socioeconomic resources are helpful in delaying death, even from many of those causes we categorized as low preventability, and an association between death from these causes and socioeconomic status is not surprising. As noted earlier, it is also possible that some of the association between socioeconomic status and mortality from low preventability causes results because conditions of a more advantaged life, such as clean, uncrowded living conditions, may benefit health in as yet unknown ways.

Second, we wish to clarify the unique contribution of this paper and which aspects of fundamental cause theory the present analyses do and do not address. Our results show a strong mortality gradient for both educational attainment and family income. This, however, is not a new or unique finding. Such gradients are seemingly ubiquitous, and in fact it is the persistence of these gradients that we wish to explain.

The results reported in this paper do not bear on the plausibility of a traditional risk factor approach to understanding the association of socioeconomic status to mortality, and in fact are perfectly compatible with such an approach. The resources associated with socioeconomic status certainly affect mortality through a variety of more proximate mechanisms, mechanisms that were not the focus of the present research. What is incompatible with a risk-factor approach is the fact that the association between socioeconomic status and mortality has persisted despite radical changes in the risk factors connecting them at any given time. The present analyses were devised not to pit fundamental cause theory against a risk factor approach, but rather to test a critical component of the theory of fundamental causes.

The theory has four component propositions. The analyses described here tested and supported the key explanatory proposition—the role of resources—but they do not constitute a comprehensive test of all four propositions. As mentioned earlier, support is fairly

strong for the first two propositions (socioeconomic status affects mortality through multiple mechanisms and multiple disease outcomes). The remaining proposition is that the association between a fundamental cause and mortality is reproduced over time via the replacement of intervening mechanisms. There is some justification for inferring from our present results that differences between diseases in the preventability of death at a single period in time mimic the situation that would occur when preventability changes over time. That is, we observed that socioeconomic gradients in mortality were stronger for highly preventable deaths during a relatively brief period of time. We might infer from that fact that, in general, at a time in history when death from a particular cause was less preventable, socioeconomic differences in mortality would have been smaller, and that if a disease becomes more preventable in the future, socioeconomic inequalities in mortality will become larger.[7] The validity of this inference is supported by historical examples such as coronary artery disease, lung cancer, and AIDS. In each case, advances in knowledge about prevention or treatment of the disease were accompanied by an increased mortality advantage for high-socioeconomic status individuals (Beaglehole 1990; Fife and Mode 1992; Link et al. 1998; Logan 1982). Nevertheless, this remains an inference rather than a direct test, because we only assessed the importance of socioeconomic resources in a single historical context. A more complete test of the dynamic aspect of the theory would systematically assess changes in gradients between socioeconomic status and mortality over time in relation to emerging health information and technology.

CONCLUSION

Despite the qualifications we have noted, the findings clearly support our a priori hypothesis that socioeconomic inequalities in mortality are substantially larger for causes of death that are more preventable. The reasoning behind that hypothesis was that the importance of socioeconomic resources for longevity can be demonstrated if, in situations in which persons with higher socioeconomic status cannot utilize their resources, socioeconomic inequalities in mortality are diminished. Because the association between socioeconomic status and mortality has been so consistent and pervasive,

the fact that we found exceptions to this association that were predicted by the theory significantly increases our confidence in the theory's validity. We believe these findings strongly suggest that material and social resources and the deliberate use of them are critical factors in maintaining socioeconomic differentials in mortality and that they should redirect attention toward the fundamental importance of the societal distribution of resources—who gets what and how much of it—in shaping the strong socioeconomic gradients in mortality.

Despite increasing our confidence in the theory, these results clearly do not constitute the final word on the theory of fundamental causes. Particularly needed is a systematic test of the responsiveness of socioeconomic-status-mortality patterns to changing health conditions over time (e.g., new diseases, new treatments). It is possible that further research will disconfirm this specific theory attempting to explain how it is that high socioeconomic status has allowed people to live much longer lives—over the centuries and around the world. However, there is no question that the currently dominant risk factor approach—that the association between socioeconomic status and mortality can be explained by, reduced to, and eliminated through addressing mediating factors such as crowding and sanitary conditions, health behaviors, medical care or relative deprivation—is wrong. While at any given time and place attention to mediating risk factors is certainly important for the short-term ameliorization of mortality disparities, the fact of a persistent mortality advantage over time for people with high socioeconomic status, despite changes in the intervening mechanisms, makes this type of explanation untenable in a broader perspective.

Put differently, even if the theory of fundamental causes turns out to be incorrect in its specification of access to broadly serviceable resources as being the critical factor in maintaining socioeconomic inequalities in mortality, it will still be true, as noted by House and colleagues (House et al. 1990, 1994), that socioeconomic status is a basic cause of mortality in Lieberson's (1985) terms, in that the association reproduces itself despite changes in mechanisms. Consequently, whether it is the theory of fundamental causes or other theories that prove to offer better explanations for the enduring and basic association between socioeconomic status and mortality, an ade-

quate explanation for this association will have to give appropriate recognition to socioeconomic status as a central and irreducible social influence on health. Thus, this critically important question cannot be adequately addressed by reductionistic inclinations. Its adequate understanding must engage the sociological imagination.

NOTES

1. It has also been proposed that the inverse association between socioeconomic status and mortality is due to social selection rather than social causation. Although poor health can affect socioeconomic status (Smith and Kington 1997), several types of data suggest that only a small proportion of the association between socioeconomic status and health/mortality is due to selection processes (Blane et al. 1993; Case, Lubotsky, and Paxson 2002; House et al. 1994).

2. We reason that motivation to protect one's health derives from optimism that one will have the opportunity to enjoy a long and healthy life and that this optimism derives in turn from resources related to socioeconomic status. If life chances are poor for people similar to oneself, motivation to engage in health-enhancing behaviors may be low (Wilson and Daly 1997). Thus, we do not see motivation or optimism as a resource but as a closely related mechanism through which those resources, in part, operate.

3. This does not mean there is a purposeful attempt on the part of high socioeconomic-status groups to harm the health of people with lower socioeconomic status, only to produce good health for themselves. Mortality differentials result from the unequal distribution of resources available to benefit one's own health and from zero-sum qualities.

4. This theory was developed in an attempt to understand a particular social fact—the persistent association between socioeconomic status and mortality—and it consequently focuses on these two variables. However, the theory might be generalized in terms of both the independent and dependent variables. Other social statuses, such as race and ethnicity, are associated with many resources in a manner similar to socioeco-

nomic status and may also be fundamental causes of mortality. The theory might also be broadened to explain the enduring association between socioeconomic status and the attainment of desirable life outcomes, of which longevity is only one. In this broader formulation, there might sometimes be situations in which higher socioeconomic-status individuals would knowingly put themselves at risk of earlier death in order to achieve some other desideratum. An example might be the postponement of childbearing by higher socioeconomic-status women with attendant increase in the risk of developing breast cancer. At the same time, the theory predicts that, in this situation, societal resources will be marshaled to improve detection and treatment of breast cancer so that individuals with higher socioeconomic status may attain both desired outcomes—family size and timing of their choice as well as long and healthy lives.

5. There may be numerous ways in which death from a particular cause is preventable, including preventive measures and medical interventions once a disease develops. According to the theory of fundamental causes, people with higher socioeconomic status are more likely, on average, to employ all of these life-extending mechanisms. Thus, preventability of a particular cause of death should strengthen the association between socioeconomic status and mortality regardless of the particular way or ways in which death can be prevented.

6. Our hypothesis concerns the association between socioeconomic status and mortality, and not the potential role in that association played by other sociodemographic factors. However, we repeated the analyses shown in Table 2, controlling for gender, urban residence, marital status, race/ethnicity and, for analyses involving family income, number in household. As in Table 2, the association between socioeconomic status and mortality was stronger for high-preventability than for low-preventability causes in every case. In these analyses, the contrast between high- and low-preventability was, if anything, even more pronounced.

7. This principle may not hold in every instance. We thank an anonymous reviewer for suggesting conditions in which it may not, i.e., if the condition is initially more

prevalent in lower socioeconomic groups, and the preventive remedy is relatively easy to apply. For example, the introduction of anti-tuberculosis drugs in the 1930s appear to have reduced disparities because of their widespread use and the differential prevalence of tuberculosis before the introduction of the drugs.

APPENDIX A. Causes of Death, Death-preventability Ratings, and Number of Deaths in the National Longitudinal Mortality Study Sample (ages 25 and older at baseline)

Preventability rating	Cause of death (number of deaths)
1.0	Malignant neoplasm of gallbladder and extrahepatic bile ducts (93)
1.5	Multiple sclerosis (28)
2.0	Anterior horn cell disease (80), cardiomyopathy (296), disorders of lipoid and plasma protein metabolism (28), leukemia of unspecified cell type (107), lymphosarcoma and reticulosarcoma (81), malignant neoplasm of brain (211), malignant neoplasm of ovary and other uterine adnexa (281), malignant neoplasm of pancreas (504), multiple myeloma and immunoproliferative neoplasms (184), myeloid leukemia (169), myoneural disorders, muscular dystrophies and other myopathies (18), polyarteritis nodosa and allied conditions (12)
2.5	Malignant neoplasm of stomach (325), malignant neoplasm of thymus, heart, and mediastinum (11), sarcoidosis (12), senile and presenile organic psychotic conditions (83), valvular heart disease excluding that of rheumatic origin (244)
3.0	Acute glomerulonephritis, nephrotic syndrome, chronic glomerulohephritis, and nephritis and nephropathy, not specified as acute or chronic (44), acute pulmonary heart disease (229), aplastic anemia (30), benign neoplasm of brain and other parts of nervous system (15), chronic pulmonary heart disease (35), chronic renal failure (107), lymphoid leukemia (82), malignant neoplasm of body of uterus (65), Parkinson's disease (118)
3.5	Accidents due to natural and environmental factors (29), aortic aneurysm (333), arrhythmias (1031), coagulation defects (21), congenital anomalies of urinary system (13), epilepsy (23), malignant neoplasm of esophagus (191), malignant neoplasm of female breast (885), malignant neoplasm of liver and intrahepatic bile ducts (142), malignant neoplasm of prostate (607), malignant neoplasm of rectum, rectosigmoid junction and anus (179), malignant neoplasm of skin (102), suicide and self-inflicted injury by hanging, strangulation, suffocation and jumping from high place (53)
4.0	Accident to powered aircraft (23), acute and subacute endocarditis (16), bulbus cordis anomalies and anomalies of cardiac septal closure, other congenital anomalies of heart and circulatory system (19), cerebrovascular diseases (3037), diabetes mellitus (776), gastrointestinal hemorrhage (118), congestive heart failure (753), ischemic heart disease (11407), late effects of accidental injury (14), liver abscess and sequelae of chronic liver disease (80), malignant neoplasm of bladder (223), malignant neoplasm of colon (1081), malignant neoplasm of oropharynx, nasopharynx, and hypopharynx (31), pneumoconiosis due to external agents (141), septicemia (38)
4.5	Accidental falls (196), accidents caused by submersion, suffocation, and foreign bodies (129), alcohol dependence syndrome (72), atherosclerosis (496), bacterial meningitis (9), chronic liver disease and cirrhosis (577), homicide (188), hypertensive heart and renal disease (51), hypertensive heart disease (479), hypertensive renal disease (76), infections of kidney (35), malignant neoplasm of lip, tongue, major salivary glands, gum, floor of mouth (70), motor vehicle traffic accidents (424), nondependent abuse of drugs (22), obesity and other hyperalimentation (23), pneumonia and influenza (1182), rheumatic heart disease (154), suicide and self-inflicted injury by firearms and explosives (266), Suicide and self-inflicted poisoning by liquid, solid, gas or vapor (92)
5.0	Accidental poisoning (79), accidents caused by fire and flames (23), acquired hypothyroidism (13), acute appendicitis (12), acute laryngitis and tracheitis, acute upper respiratory infections of multiple or unspecified sites, acute bronchitis and bronciolitis (16), calculus of kidney and ureter (13), cholelithiasis (32), chronic obstructive pulmonary disease (1575), diverticula of intestine (58), gastritis and duodenitis (14), hernia of abdominal cavity (24), injury undetermined whether accidentally or purposely inflicted (38), malignant neoplasm of cervix uteri (102), malignant neoplasm of larynx (63), malignant neoplasm of trachea, bronchus and lung (2862), misadventures during surgical and medical care and medical procedures as the cause of abnormal reaction or later complication (45), other accidents (171), peptic ulcer (139), tuberculosis (27), viral hepatitis (23), water transport accidents (10)

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