The Resources That Matter: Fundamental Social Causes of Health Disparities and the Challenge of Intelligence*

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A robust and very persistent association between indicators of socioeconomic status (SES) and the onset of life-threatening disease is a prominent concern of medical sociology. The persistence of the association over time and its generality across very different places suggests that no fixed set of intervening risk and protective factors can account for the connection. Instead, fundamental-cause theory views SES-related resources of knowledge, money, power, prestige, and beneficial social connections as flexible resources that allow people to avoid risks and adopt protective strategies no matter what the risk and protective factors are in a given place or time. Recently, however, intelligence has been proposed as an alternative flexible resource that could fully account for the association between SES and health and thereby find its place as the epidemiologists' "elusive fundamental cause" (Gottfredson 2004). We examine the direct effects of intelligence test scores and adult SES in two data sets containing measures of intelligence, SES, and health. In analyses of prospective data

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from both the Wisconsin Longitudinal Study and the Health and Retirement Survey, we find little evidence of a direct effect of intelligence on health once adult education and income are held constant. In contrast, the significant effects of education and income on health change very little when intelligence is controlled. Although data limitations do not allow a definitive resolution of the issue, this evidence is inconsistent with the claim that intelligence is the elusive fundamental cause of health disparities, and instead supports the idea that the flexible resources people actively use to gain a health advantage are the SES-related resources of knowledge, money, power, prestige, and beneficial social connections.

The idea that stratification systems have powerful consequences for the life chances of individuals differentially located within those systems is a core premise of the sociological enterprise. The possibility that this premise applies beyond the distribution of goods and services, beyond the assignment of honor and esteem to the biology of disease and death, is one of the most provocative implications of this idea. The robust and very persistent associations between indicators of socioeconomic status and morbidity and mortality have been a central focus of medical sociology and one that has strongly linked that sub-area to concerns within the broader discipline. Medical sociologists and social epidemiologists have sought to deepen our understanding of this connection by positing theories that might explain this association (Mirowsky and Ross 2003; Marmot 2004; Link and Phelan 1995;Wilkinson 2005; Lynch 2000). Extant theory in medical sociology and social epidemiology proposes that this robust association arises because SES affects the distribution of stress (Pearlin et al. 1981; Seeman et al. 2004; Marmot 2004; Wilkinson 2005), influences the development of healthy lifestyles (Mirowsky and Ross 2003; Cockerham 2005), or provides resources that allow people to avoid risks and adopt protective strategies (Link and Phelan 1995; Phelan and Link 2004; Lynch et al. 2000).

Relatively recently, however, research from outside medical sociology and social epidemiology has pointed to intelligence as an important but unrecognized determinant of health. Based on long-term follow-up studies of individuals initially identified in adolescence, these studies have shown significant associations between IQ scores and subsequent morbidity and mortality. A particularly provocative claim of this emerging literature is the idea that intelligence may underlie the association between SES and health, essentially revealing that association to be spurious. For example, Batty and Deary (2005) claim that "education may be a surrogate for IQ" (p. 365) in its relation to health, and Gottfredson (2004) asserts that intelligence may be the "epidemiologists' elusive 'fundamental cause' of social class inequalities in health" (p. 174). By contrast, although theories that assign prominence to SES differ with respect to exactly what it is about SES that leads to ill health, none of these theories would be congenial with the proposition that a substantial part of the association between SES and health is due to either the confounding or mediating effects of intelligence.

While the role of intelligence is potentially challenging to many sociological theories, it is particularly challenging to the theory of fundamental social causes (House et al. 1990; Link and Phelan 1995, 1996, 2000; Phelan et al. 2004; Lutfey and Freese 2005; Williams and Collins 2001). This theory seeks to understand why the association between SES and health has been so persistent across different places and times. According to the theory, the principal reason for this persistence is that SES embodies a set of flexible resources—knowledge, money, power, prestige, and beneficial social connections—that can be used in different places and at different times to avoid disease and death. Resources can be deployed at the individual level, as when people use resources to construct a healthy lifestyle, or at a contextual level, as when people use resources to gain access to salutary contexts such as good neighborhoods, safe jobs, and robust social networks. Because flexible resources are deployed to avoid whatever risks may exist and adopt whatever protective strategies may be available, the association between SES and health appears in different places and at different times.

Clearly, a central feature of fundamental-cause theory is the idea of flexible resources, and, just as clearly, intelligence is not one of the flexible resources mentioned or even considered in extant discussions of that theory. Yet, as we shall see, intelligence can also be conceived as a flexible resource that can be used in different places and historical periods to avoid risks and adopt protective strategies. This raises two questions for fundamental-cause theory: Should intelligence be added to the list of flexible resources? And might intelligence be a potent confounder in the association between SES and health?

THE ROLE OF INTELLIGENCE IN HEALTH

The Concept of Intelligence

There is no widely accepted definition of intelligence. A Task Force of the American Psychological Association formed after the publication of Herrnstein and Murray's (1994) controversial book *The Bell Curve* concluded that no conceptualization of intelligence "has yet answered all the important questions and none commands universal assent" (Neisser et al. 1996:77). However, this same report identifies the so-called psychometric approach as the dominant one because of its extensive use in research and its wide application in practical settings (Neisser et al. 1996). In this approach, intelligence is identified by test performance. While tests differ in content, a standard test like the Wechsler Adult Intelligence Scale (WAIS) includes items about general information, recall of digits, vocabulary, arithmetic problems, comprehension, word similarities, completing pictures with a missing detail, arrangement of pictures in a logical sequence, putting blocks together to match patterns on cards, copying a coding pattern, and assembling small jigsaw puzzles. A central concept of the psychometric approach derives from the fact that scores on what seem like very different tasks correlate with one another and form a common factor in factor analysis. This factor has been called "g" and is presumed to be a measure of general intelligence. One reason that these disparate components correlate could be that each is arranged from simple to complex tasks, thereby capturing a common element of cognitive complexity (Gottfredson 1997). General intelligence or "g" is thought to underlie and thus be captured by a diverse array of tests like the WAIS, the Stanford-Binet, the Henmon-Nelson, the Raven Progressive Matrixes, and many others. We use the term "intelligence" in our report rather than "cognitive ability" or "intellectual functioning" because the issue we address involves the capacity to perform on tests like these, and the term "intelligence" is the one most widely used to refer to that capacity. An American Psychological Association task force indicates that scores on intelligence tests are quite stable within individuals over time (Neisser et al. 1996). Findings from long-term follow-up studies support this assertion, showing, for example, that test scores at age 11 correlated .63 with scores at age 77; correlations for shorter time spans (ages 50 to 70) are as high as .90 (Deary et al. 2000).

An Explanation for Why Intelligence Influences Health

Theory to support the influence of intelligence on health begins with the observation that the management of health, like so many other aspects of modern life, has become exceptionally complex. Massive amounts of new health-relevant information have become available, and gaining access to that information, absorbing its content, assessing its salience, and constructing a plan to act on the information received is a daunting task. Simply put, a strain is placed on the capacity to fully grasp and effectively deploy health-relevant information, whether it is to address a health crisis or to prevent one from occurring (Nielsen-Bohlman, Panzer, and Kindig 2004). It follows that individuals who are more intellectually adept are better able to grasp any health situation they confront, ferret out the relevant information required to address the circumstance they experience, and creatively construct a plan to maximize their chances for a healthy outcome (Gottfredson 2004; Batty and Deary 2005). As Gottfredson (2004) puts it, "health self-management is inherently complex and thus puts a premium on the ability to learn, reason and solve problems" (p. 189).

According to the foregoing account, intelligence is critical because people actively use it to gain a health advantage. It does not lie behind nor is it fully mediated by SES-related resources, but is instead actively deployed to bring on good things and avoid bad ones in the health arena. If the theory were different and asserted instead that intelligence—along with parental resources, hard work, and the good fortune to be born in a wealthy country—lay behind the acquisition of SES-related resources, and if SES-related resources were actively deployed to achieve a health advantage, intelligence would pose little challenge to fundamental-cause theory. In such a scenario, SES-related resources would remain the key flexible resources central to fundamental-cause theory.

However, a theory like the one presented above—one that claims that intelligence is actively and directly employed to obtain beneficial health circumstances— poses two critical challenges to fundamental-cause theory. If general intelligence not only directly affects health but also lies prior to and strongly influences the attainment of SES, intelligence could be a confounder in the SES-health association. If intelligence is to some degree the consequence of SES, then intelligence could mediate the effect of SES. Both of these roles for intelligence are possible, and both conflict with predictions from sociologically inspired theories concerning the association between SES and health.

Intelligence as a Potential Confounder in the SES-Health Association

If intelligence confounds the association between SES and health, it should precede both SES and health. Some investigators find support for this possibility in the very high estimates of heritability (.8) for IQ (Neisser 1996) and in the stability of test scores across the life course. In addition, because IQ is related to both health and SES, it could be that IQ is a potent confounder in estimating an SES-to-health causal relationship. As mentioned above, studies have shown that IQ measured early in life is significantly related to morbidity and mortality measured later in life. In addition, scores on IQ-type tests are highly predictive of socioeconomic outcomes (e.g., Jencks et al. 1972, 1979). Relationships between intelligence and health and intelligence and SES raise the possibility that controls for IQ could reveal the association between SES and health to be entirely spurious. Such an outcome would provide a strong challenge to sociologically inspired theories that argue that socioeconomic status plays a causal role in influencing health.

Intelligence as a Potential Mediator of the SES-Health Association

Not everyone believes that intelligence is entirely antecedent to both socioeconomic status and health. Many contend that nutrition, the timing and quality of education, neighborhood circumstances, and occupational conditions play a role in determining an individual's intellectual functioning (Alwin and McCammon 2001; Breslau et al. 2001; Guo 1998; Guo and Harris 2000; Richards and Wadsworth 2004; Schooler, Mulatu, and Oates 1999). Moreover, in light of evidence that intelligence influences the attainment of SES, a plausible view is that there are reciprocal effects between SES and intellectual functioning across the life course, much like Kohn and Schooler's (1982) demonstration of reciprocal effects between occupational conditions of substantive complexity and intellectual flexibility. This raises the possibility that intelligence may partly mediate the association between SES and health.

While a mediating role for intelligence is plausible, it has not been embraced by sociological theories concerning the connection between SES and health. These theories posit mediating factors that are different from intelligence, such as material circumstances (Lynch et al. 2000; Link and Phelan 1995), lifestyle (Mirowsky and Ross 2003), social psychological orientations (Schnittker and McLeod 2005), relative deprivation (Marmot 2004; Wilkinson 2005), and stress (Pearlin et al. 1981; Seeman et al. 2004). Mirowsky and Ross (2003) are unusual in addressing intelligence at all, and they describe it is as one of a number of "specious" explanations for the connection between education and health. It is clear, then, that existing theories have not sought to incorporate intelligence as a mediating factor, and it is thus safe to say that if intelligence proved to be a major mediator of the SES-health association, some rethinking of these theories would be necessary.

The issue is even more sharply drawn for fundamental-cause theory because of its emphasis on flexible resources in accounting for the presence of an SES-health association in different places and during different historical periods. To be sure, if intelligence plays some modest role in mediating the effect of SES on health, intelligence might simply be added to the list of flexible social and economic resources: knowledge, money, power, prestige, beneficial connections, and intelligence. However, if intelligence was the principal factor mediating the SES-health association, accounting for a large proportion or all of that association, it would require a rethinking of the role of the other listed resources. Consider for a moment the possibility that intelligence mediates all of the SES-health association. In such a scenario, it would be difficult to argue that SES-related resources are the flexible resources that people deploy in health relevant situations. While they might be thought of as sources of the active ingredient—intelligence—they would not be the active ingredient themselves. Put a slightly different way, inducing a positive change in people's social and economic resources would play little role in changing health outcomes unless doing so also led to a boost in intelligence. Clearly, then, the role of intelligence in the relationship between SES and health has substantial bearing on theories of social influence on health.

EXISTING LITERATURE ON INTELLIGENCE, SES, AND HEALTH

In contrast to the rapidly expanding literature focused on SES and health, relatively little research has addressed the role of intelligence in health using population-based samples of adults. In the literature, cross-sectional studies with adequate measures of SES show a strong role for SES and a weak role for intelligence, whereas prospective studies that have design advantages over cross-sectional studies but weaker measures of SES point to a different conclusion. We found two cross-sectional studies, one by Singh-Manoux et al. (2005) and another by Schnittker (2005). In the first, Singh-Manoux and colleagues use data from the Whitehall sample of British civil servants (phase 5, n = 5,838). Age-adjusted results show that intelligence is significantly associated with multiple indicators of health in both men and women. However, controls for SES sharply diminish this effect and render it nonsignificant in 9 of 10 comparisons. Schnittker (2005) uses General Social Survey data from 14,905 respondents interviewed in multiple cross-sectional surveys from 1974 to 2000. As with the study by Singh-Manoux et al. (2005), this study finds appreciable bivariate associations between verbal ability and health (self-rated), but it also finds that these effects are substantially weakened when controls for SES are entered. These two studies provide evidence supportive of the primacy of SES resources, but the cross-sectional nature of the findings are problematic, as both income and scores on tests of intelligence can be influenced by current health status.

A handful of prospective, population-based studies have also focused on the role of intelligence in health. Most of these employ a design in which intelligence tests administered early in life are related to health in adult life (Deary et al. 2004; Hart et al. 2003; Hart et al. 2004; Hemmingsson et al. 2006; Kuh et al. 2004; Martin et al. 2004; Osler et al. 2003; O'Toole and Stankov 1992; Whalley and Deary 2001). Of nine studies of this type, all found some statistically significant connection between test scores and subsequent morbidity and mortality. However, the relationships were not always monotonic, and in one analysis (for women) the relationship was not present at all (Kuh et al. 2004). All of the studies make some effort to control for SES, and when they do the effect of intelligence is generally only minimally attenuated. But the adequacy of the SES measures can be questioned. Four studies include only childhood measures of SES (O'Toole and Stankov 1992; Osler et al. 2003; Whalley and Deary 2001; Deary et al. 2004). Four other published reports include at least some measure of adult socioeconomic circumstances (Hart et al. 2003; Hart et al. 2004; Hemmingsson et al. 2006; Martin et al. 2004; Kuh et al. 2004), but the available measures are generally crude classifications of occupations (two to seven categories) or the economic level of the individual's area of residence. Only one of these studies included an assessment of educational attainment, and that study only followed cohort members to a maximum age of 39 (Martin et al. 2004). None of these studies used either adult income or wealth in its assessment of SES.

Two other studies (Batty et al. 2006; Pavlik et al. 2003) report results from prospective studies with a different design and better measures of SES. The study by Batty and colleagues followed a cohort of middle-aged persons for 16 years and found associations between indicators of SES (education, income, and occupation) and all-cause mortality, coronary artery disease mortality, self-reports of chronic illness, self-rated health, mental health, and respiratory function. While most of these associations remained significant after controls for test scores (20 of 25 did so), the effects of SES indicators were dramatically reduced with these controls. The study does not report effect sizes for the IQ test scores, but one presumes from the extent to which such scores reduce the effect of SES that they must be substantial. The study by Pavlik and colleagues (2003) followed a cohort of middle-aged respondents for 10 years and found associations between test scores and subsequent mortality that were diminished but not eliminated by controls for education and occupation.

Although the measurement of SES is better in these two studies than in the cohort studies mentioned above, there is a potentially serious bias in their design for the purpose we wish to address. Performance on tests is impaired by severe illness, thereby producing an inappropriately low score in a person who is sick. If a person is sick at initial interview, they are also more likely to be sick at follow-up or to die in the intervening years. In the absence of a sound approach to addressing this important problem, test scores carry the effect of illness, making IQ seem more important than it is. To address this potential problem, we require a design strategy that begins with individuals who are not ill or an analytic strategy that comprehensively controls for baseline illness. Because neither study did this, their results concerning the connections among SES, intelligence, and health probably overestimate the effect of IQ.

RESEARCH STRATEGY

An ideal design to address the questions we pose would be prospective from birth, with multiple measurements of the key variables of SES, intelligence, and health. Such a design would allow the evaluation of reciprocal effects across the life course. In the absence of these ideal data, we are interested in implementing a prospective design that follows disease-free persons forward to determine who develops poor/fair self-rated health, who develops a life-threatening illness, and who dies during a follow-up period. As each one of these outcome variables is dichotomized (see below), we use logistic regression and report the log-odds and odds ratios of predictor variables on these outcomes. Implementing such a prospective design allows us to place the putative causal variables (intelligence and SES) before health problems in time, thereby substantially reducing the plausibility of reverse causation (health to intelligence or health to SES). This is important because education and especially income and test performance might be affected by the development of disease, thereby creating an association that could be misinterpreted as a causal effect of SES or intelligence.

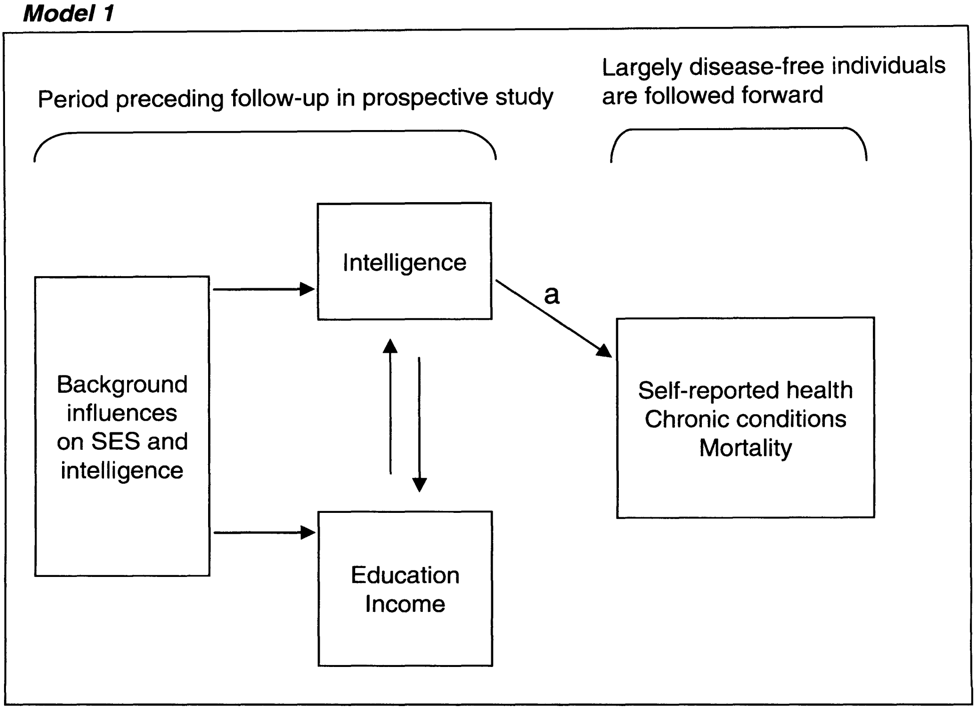
We located two large public-access data sets: the Wisconsin Longitudinal Study and the Health and Retirement Survey. Each provides the requisite measures and also allows us to implement a prospective design. While each data set is appropriate for our purposes, each also has relative strengths and weaknesses when compared to the other with respect to the timing and comprehensiveness of the assessment of intelligence and the generalizability of the sample. It will thus be important to determine whether and to what extent these two data sets provide consistent evidence regarding the issue that lies before us.

Figure 1 depicts two models that highlight the critical associations at issue in the current analysis (arrows labeled "a" and "b"). Before turning to these, we consider aspects of Figure 1 that relate to the study design and are common to both models. First, on the left-hand side of both models we depict relationships that are presumed to have occurred before follow-up begins in our prospective design. Here we provide boxes to indicate that there are likely multiple background influences on SES and on intelligence. Also in this section of both model 1 and model 2 we portray arrows going from intelligence to SES and from SES to intelligence. Some argue that because intelligence is subject to substantial genetic influence and also quite stable over the life course (Neisser et al. 1996), most of the effect goes from intelligence to SES. But others argue that SES-related exposures across the life course influence scores on intelligence tests (Breslau 2001; Richards and Wadsworth 2004). Thus, we portray (but do not seek to empirically assess) mutual influence between the two constructs up until the point that the prospective follow-up begins.

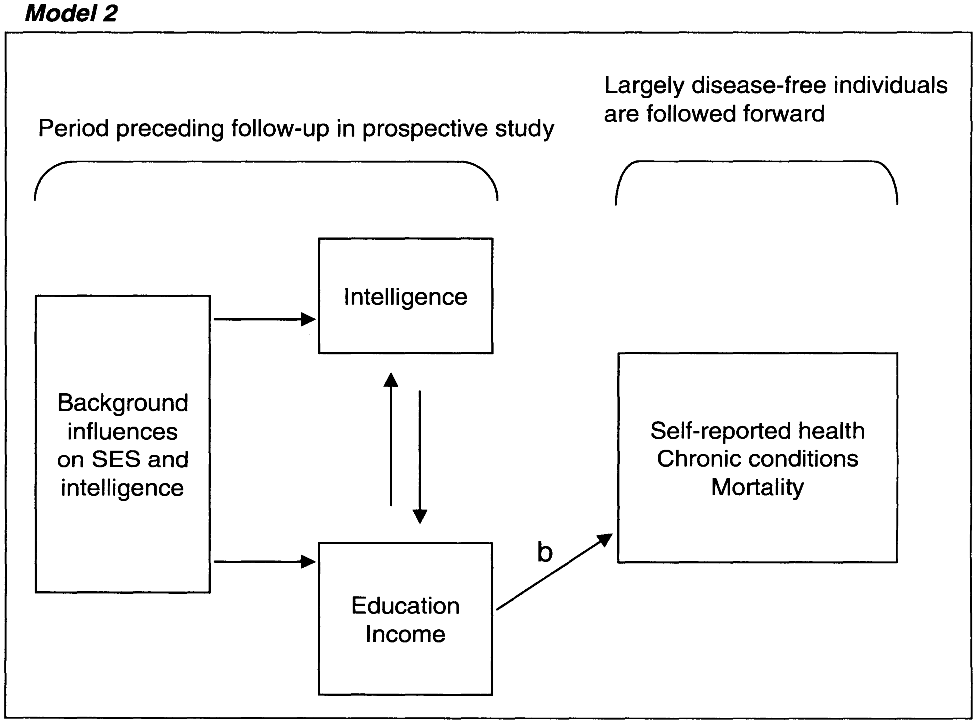
Finally, moving to the right-hand side of Figure 1, observe that the arrows are drawn from intelligence (model 1) and SES (model 2) to health outcomes rather than vice versa. The rationale for this depiction is that a prospective study begins with individuals who are free of the condition(s) under study. Despite these similarities, model 1 is different from model 2 in two important ways. First, consistent with the idea that people use intelligence to craft a healthy life style and absorb complex information about disease management, model 1 depicts a direct effect of intelligence on health in the follow-up period (arrow labeled "a"). Second, model 1 denies a direct effect of SES-related resources on health in the follow-up period. Model 2 posits just the opposite set of circumstances—a direct effect of SES resources on health (arrow labeled "b") but no direct ef-

FIGURE 1. Depiction of the Critical Associations at Issue in a Prospective Study of the Effects of Intelligence and SES on Health

Model 1



Model 2



fect of intelligence. Model 2 is therefore consistent with the idea that people use resources of knowledge, money, power, prestige, and beneficial social connections to construct a healthy lifestyle and to gain access to contexts that are beneficial for health.

To the extent that results are consistent with model 1, the fundamental-cause approach will be refuted in favor of an explanation that asserts that intelligence is the primary flexible resource people use for health benefit. In contrast, to the extent that results are consistent with model 2, the fundamental-cause perspective can retain its emphasis on the use of SES-related resources in health-relevant situations. Clearly, the models are not necessarily mutually exclusive, and it is entirely possible that both SES and intelligence could have independent direct effects on health. Thus, the results of our analysis could support the fundamental social causes explanation, with its emphasis on SES; the alternative explanation, giving primacy to intelligence; or both perspectives. As such, the empirical analysis will provide important information concerning the future development of explanations for the commonly observed association between socioeconomic status and health.

METHODS

Samples

The Wisconsin Longitudinal Study (WLS) is based on a random sample of 10,317 men and women who graduated from Wisconsin high schools in 1957. The survey collected data from the original respondents in 1964, 1975, and 1992–1993. The present study employs the full sample for analyses of mortality, with the exception that individuals with missing data on educational attainment (n = 208) are excluded, leaving a sample of 10,109 for analysis. For analyses of life-threatening health conditions and self-rated health, we use the 1992–1993 wave of data collection, which included 8,493 telephone interviews of approximately 9,750 surviving men and women and a supplemental mail survey on health, sent only to members who completed the telephone interview. The health survey was returned by 6,875 respondents, representing a response rate of 70.5 percent of the surviving cohort. Men (67.0%) were slightly less likely to respond than women (71.3%). Responders scored somewhat higher on IQ tests (mean 105 vs. 97), had more years of education (13.5 vs. 13.1), and reported higher incomes in 1974 ($16,600 vs. $14,800). While all of these differences were statistically significant, none was greater than half a standard deviation difference, suggesting some but not extreme sample selection bias for analyses of the mail survey. One weakness of the WLS for our purposes is that everyone in the primary sample graduated from high school, thereby truncating the distribution of SES. Sewell and Hauser (1975) have estimated that about 75 percent of Wisconsin youth graduated from high school in the late 1950s. Another weakness is that there are only a handful of African American, Hispanic, or Asian persons in the WLS, a fact that precludes our ability to generalize to those groups. (The data employed are based on the 10th public release of the 1957–1977 surveys and the 9th public release of the 1992–1993 surveys.)

The Health and Retirement Survey (HRS) is based on a nationally representative, multistage area probability household sample of adults born during the years 1931 to 1941, and it includes data on those individuals' spouses. African Americans, Hispanics, and residents of Florida were oversampled. A total of 15,497 individuals were eligible to be interviewed, and 12,654 interviews were conducted, yielding a response rate of 81.7 percent. The survey has collected data from respondents during a home interview in 1992 and during telephone interviews every two years thereafter. Because the sampling design uses clustering and stratification, we employ SUDAAN software (Research Triangle Institute 2001) that allows us to use weights to restore population representativeness and to obtain standard errors that take the complex survey sampling design into account.

In the present study we include respondents who were born between 1931 and 1941 (n = 9,773). In order to implement a prospective design and thereby reduce the possibility of reverse causation (see above), we exclude 3,011 people who reported having a life-threatening illness in 1992. An additional 479 persons are excluded because of missing data on the intelligence measures in 1992, leaving 6,283 persons for analysis. Follow-up for mortality was possible for all 6,283 respondents, but for life-threatening illness and self-reported health the number of cases available for analysis is smaller. With respect to life-threatening illness, 203 (3.2%) of the 6,283 persons had died, and 797 (12.7%) were lost to follow-up, leaving 5,283 for analysis. With respect to self-reported health, 748 persons who reported fair or poor health at baseline were excluded in order to allow a prospective design for the analysis of this variable. Of the remaining 4,535 persons, 149 (2.7%) died and 701 (12.7%) were lost to follow-up, leaving 3,685 for analysis. While constructing a prospective design from the sample has important benefits, it also induces some sample selection biases that we consider as limitations in the discussion section.

The WLS and the HRS are appropriate for testing our hypotheses because they allow us to implement prospective designs in which SES and intelligence are ascertained before the health events under study have occurred. This places the variables of interest in time order and protects measures of SES and intelligence from being contaminated by the effects of illness. Further, the samples have different strengths and weaknesses for addressing the issues we have posed. One strength of the HRS in comparison to the WLS is that the HRS sample includes individuals who did not graduate from high school. Another relative strength is its national representativeness and its oversample of African American and Hispanic persons. Strengths of the WLS in comparison to the HRS are its longer follow-up period, the capacity it provides to analyze mortality that occurs before late middle age, and the quality of the measures of intelligence (see below).

Measures of Health

Mortality was assessed at each wave of the WLS as part of the study team's efforts to locate and keep in contact with interview respondents. As of 2002, 911 of the initially sampled subjects had died. Mortality in the HRS was assessed at each point of contact with respondents using the National Death Index. The current study data are from the 1998 assessment. Because the follow-up period is longer for the WLS (1957–2002) than for the HRS (1992–1998), there is a higher percentage of deaths in the WLS.

Life-threatening health conditions. In the WLS and the HRS, respondents were asked to indicate whether a medical professional (WLS) or a doctor (HRS) had told them they had any of a list of specific conditions. We selected and then empirically assessed whether several self-reported health conditions were life threatening. In the WLS, the selected conditions included bronchitis/emphysema, cancer, chronic liver trouble, diabetes, and heart trouble; in the HRS, the selected conditions included lung problems, cancer, diabetes, heart problems, and stroke. In the WLS, an opportunity existed to screen out reports of health condition that were likely to have little consequence. For each condition identified, respondents reported how much (not at all, very little, some, quite a bit, a great deal) the illness or condition currently interfered with what they liked to do. Based on this information we included health conditions that interfered some, quite a bit, or a great deal as potentially life-threatening conditions.

In both studies, the designation of these conditions as life-threatening is supported by the fact that each one is strongly predictive of subsequent death. In the WLS, relative risks for death following the 1992 health survey indicate that people reporting one of these conditions were between about 3.5 times (liver trouble) to more than 15 times (cancer) as likely to die by 2002. In the HRS, the relative risks of death in the six-year follow-up period (1992–1998) ranged from 2.5 (lung) to 3.5 (diabetes) across the five life-threatening conditions. Respondents who reported one or more of these conditions were coded 1; those who reported none were coded 0.

Poor self-rated health. In both the WLS and the HRS, respondents were asked to rate their health as excellent, good, fair, or poor. In both studies, respondents who rated their health as less than good (fair or worse) are scored 1, and all others as 0. As with reports of specific conditions, people who rate their health as less than good are more likely to have died on follow-up in each study (relative risk WLS = 4.1, HRS = 4.2).

Measures of Intelligence

Intelligence was assessed in the WLS using the 90-item Henmon-Nelson test, which covers vocabulary, sentence completion, disarranged sentences, classification, logical selection, series completion, directions, analogies, anagrams, proverb interpretation, and arithmetic problems (Buros 1970). Data were taken from school records, and scores for every respondent were ascertained. For most respondents the data are the average of scores on tests taken in the freshman and junior years. For the relatively small number of respondents who had data from only the freshman or junior years, that single score was used as the best measure of intelligence. In order to ease interpretation, we centered the intelligence measure by subtracting the mean, and we also divided by 10 to move the decimal point one digit to the left, so that coefficients and standard error values would be visible in the tables. Because of this, coefficients for intelligence in the WLS should be interpreted in terms of the effects of a 10-unit change in intelligence.

In the HRS, three tests were administered during the 1992 in-person survey that provide a usable, if not ideal, measure of intelligence. The first is taken from the word-similarities component of the WAIS. This subscale is considered a good measure of general intelligence because of its .77 loading on a factor that identifies "g" (Kaufman 1994), and it is also considered a good representation of the full scale score, as it correlates .75 with the parent instrument in adults 45–54 (Wechsler 1955). In this assessment, participants were asked seven of thirteen questions from the word-similarities test that ask how words are alike (e.g., "How are a banana and an orange alike?"), with possible scores of 0, 1, or 2 on each. To augment this assessment, we also include two word-recall measures. For immediate word recall, each participant was read a list of 20 words and then immediately asked to recall as many as possible. For delayed recall, participants were asked again after a 10-minute lag to name as many of the same 20 words as they could. The score on each of these measures was the number of words recalled. Memory tasks such as these are common elements in IQ tests, and we decided to include them in a composite measure.

For respondents with complete data, we constructed a composite measure of intelligence (alpha = .73) by standardizing each of the three measures, summing the standardized values, and dividing by three. The word-recall measures correlate highly with one another (.74) and moderately with the word-similarity measure (.36 and .33 for immediate and delayed recall, respectively). Because the word-similarity and memory components are correlated but distinct, we repeated all analyses using separate memory and word-similarity measures, with no change in the conclusions reported below. For respondents missing one of the three measures (5.8% of respondents), we added the standardized values of the two available scores and divided by two. If two or more measures were missing, respondents were assigned missing values on the intelligence measure in the HRS.

Measures of Socioeconomic Status

Educational attainment in both the WLS and the HRS represents the respondent's self-reported years of formal schooling.

Income in the WLS was the total earnings of the respondent and, if married, his or her spouse for the tax year 1974. Data on income were ascertained through questions about farm and business income, as well as salary and wages. Of the 10,317 cohort members, 1,203 (11.7%) had missing values on income in 1974, whereas 338 (4.9%) of the 6,875 who participated in the 1992 mail survey were missing on this variable. To address missing data on income, we employed a multiple-imputation strategy suggested by Allison (2002). We report results for income based on this multiple imputation procedure but also check the robustness of inference by excluding all cases missing on income (listwise deletion) and imputing a fixed predicted value based on a regression of education on income. While the results vary slightly, none of these approaches leads us to a different conclusion about the main issue addressed in the paper.

In the HRS, household income in 1991 is based on the sum of earnings, unemployment and workers' compensation, pensions and annuities, social security and welfare, capital income, disability income, other income received by respondents, and income of other household members. To address missing values, the HRS provides imputed data using a hot deck approach. The measure of income employed in both the HRS and the WLS is the natural log of income in thousands of dollars.

RESULTS

SES and Health

Table 1 shows the association between SES (with education in four categories [less than high school, high school, some college, and college or more] and income in quartiles) and the incidence of each of the three health indicators during the follow-up period. As the table shows, education and income are significantly associated with each of the three health indicators in both studies.[1] In all 12 tests, the lowest category of SES is less healthy than the highest, and in 9 of 12 the association shows a monotonic decrease in the incidence of disease and death with increasing education and income. Thus, evidence for an association be-

TABLE 1. Bivariate Associations between Education, Income, Intelligence, and Ill Health in the Wisconsin Longitudinal Study (WLS) and the Health and Retirement Study (HRS)

	WLS			HRS[a]		
	% Dying	% Reporting LTI	% Reporting Fair or Poor SRH	% Dying	% Reporting LTI	% Reporting Fair or Poor SRH
n	10,109	6,875	6,862	6,283	5,283	4,685
Education						
Less than high school	—	—	—	4.2*	24.3***	26.6***
High school	8.9***	6.4***	13.7***	2.7	17.3	12.8
Some college	9.5	4.8	10.6	2.7	16.3	9.7
College or more	6.2	3.4	7.0	2.6	16.0	6.1
	—					
Income						
Lowest quartile	10.3*	6.3**	14.9***	4.7***	22.5***	20.8***
Second quartile	7.6	6.2	11.6	3.0	17.5	13.3
Third quartile	7.5	5.4	11.2	2.9	19.2	13.1
Highest quartile	8.1	3.9	8.8	1.5	14.3	7.1
	—					
Intelligence						
Lowest quartile	8.9	6.3**	14.8***	4.1**	21.4***	19.9***
Second quartile	8.1	5.7	11.4	2.7	18.0	14.6
Third quartile	8.7	5.6	9.9	3.0	18.6	11.1
Highest quartile	7.7	4.0	10.0	2.3	15.5	8.3

* $p < .05$; ** $p < .01$; *** $p < .001$ (chi-square significance of linear trend)

[a] HRS weighted percentages are reported. LTI = life-threatening illness. SRH = self-reported health.

tween SES and health and a gradient in that association are present in both samples.

Intelligence and SES

As indicated above, the literature has shown that intelligence predicts the attainment of socioeconomic status, particularly education. In order to provide an assessment of the magnitude of the association in the WLS and HRS, we calculated the percent graduating from college by quartiles of intelligence. In the WLS, where intelligence is ascertained before the completion of education, the percent graduating from college by quartile of intelligence from lowest to highest is 4.9 percent, 15.7 percent, 28.5 percent, and 50.2 percent. Comparable figures in the HRS are 5.9 percent, 16.5 percent, 26.3 percent, and 36.1 percent. While substantial, these associations are not so strong as to preclude a test of the separate effects of intelligence and education (WLS r = .434; HRS r = .441) or intelligence and income (WLS r = .175; HRS r = .268).

Intelligence and Health

The association between intelligence and health has not been as well established, particularly with respect to the incidence of ill health in general population samples. As Table 1 shows, intelligence in the WLS and in the HRS is significantly associated with health in five of six tests, the only exception being the association between intelligence and mortality in the WLS. In every instance, health is worse in the lowest category of intelligence than it is in the highest.

Intelligence, SES, and Health

We have established that in the WLS and the HRS, both SES and intelligence are related to the onset of ill health. Further, intelligence is associated with both education and income. This pattern of associations presents the possibility that intelligence accounts for the association between SES and health and brings to the fore the question of whether model 1 or model 2 is more consistent with the data. Table 2 presents results that address these issues. In the first four columns we ascertain whether the effect of SES on health is either sharply diminished or completely eliminated when intelligence is controlled. All coefficients shown are adjusted as appropriate for sociodemographic variables of sex, age, marital status, and race/ethnicity. Because the WLS is a cohort of almost entirely white high school graduates, age and race/ethnicity are held constant by design and are not entered as control variables in the analysis.

As Table 2 shows, controlling for intelligence results in relatively small changes in associations between SES and health indicators; the logistic regression coefficients after controlling intelligence are only modestly smaller (average of 13.2%) than they were before controlling it. In fact, in none of the 11 instances in which education or income is significantly related to health before entering intelligence does the effect become nonsignificant as a result of controlling for intelligence.[2]

To convey the magnitude of the associations we created dummy variables to capture categories of education (less than high school, high school graduate, some college, and college or more) and income (quartiles). Adjusting for sociodemographic variables and IQ, odds ratios comparing the lowest category of education to the highest category vary from 1.37 for mortality in the HRS to 3.94 for self-reported health in the same data set. With respect to income, adjusted odds of ill health comparing the lowest quartile to the highest vary from 1.36 for mortality in the WLS to 3.09 for mortality in the HRS.

The last two columns of Table 2 tell us whether the effect of intelligence is either sharply diminished or completely accounted for by controlling educational attainment and income. As the table shows, four of the five initially significant associations between intelligence and health are rendered nonsignificant with controls on education and income. Moreover, the logistic regression coefficients after controls are substantially smaller, on average 69.4 percent smaller than they were before controls. The only effect that remains significant, intelligence on self-reported health in the HRS, shows a dramatic decline in the logistic regression coefficient (over 50%) from before to after controlling education and income. Further, we found little evidence to indicate that intelligence interacts with education or income in influencing health. Only one of the possible 12 interaction terms was significant (education by intelligence on self-rated health in the WLS), and there was no consistent direction to the effects (seven coefficients for cross-product terms were positive and five negative). Thus the results suggest that any ef-

TABLE 2. Effects of Education, Income, and Intelligence on Health Outcomes Before and After Controls in the Wisconsin Longitudinal Study (WLS) and the Health and Retirement Study (HRS)—Logistic Regression Coefficients (standard errors in parentheses)[a]

	Education		Income		Intelligence	
	Before Controlling Intelligence	After Controlling Intelligence	Before Controlling Intelligence	After Controlling Intelligence	Before Controlling Education & Income	After Controlling Education & Income
Mortality						
WLS	–.105***	–.107***	–.234*	–.204*	–.056*	.009
	(.019)	(.021)	(.103)	(.104)	(.024)	(.027)
HRS	–.044	–.028	–.541***	–.512***	–.201	–.108
	(.024)	(.031)	(.148)	(.148)	(.116)	(.136)
Life-threatening illness						
WLS	–.144***	–.129***	–.361**	–.295*	–.123***	–.043
	(.029)	(.032)	(.141)	(.144)	(.037)	(.041)
HRS	–.054***	–.046**	–.331***	–.306***	–.145**	–.049
	(.013)	(.016)	(.073)	(.078)	(.042)	(.053)
Self-rated health						
WLS	–.158***	–.143***	–.511***	–.448***	–.130***	–.038
	(.021)	(.023)	(.104)	(.106)	(.026)	(.029)
HRS	–.184***	–.152***	–.704***	–.594***	–.534***	–.258***
	(.017)	(.021)	(.085)	(.084)	(.072)	(.081)

* $p < .05$; ** $p < .01$; *** $p < .001$

[a] Equations from the WLS control for gender and marital status; equations from the HRS control for age, gender, race/ethnicity, and marital status.

fect of intelligence on health operates largely through the attainment of education and income. People with strong test scores who happen not to attain high education and income receive few health benefits from their intellectual abilities. Similarly, people with low intelligence who nevertheless attain high education or income benefit substantially from such social and economic resources.

In the HRS, we were able to check whether effects of intelligence, education, and income on health varied substantially by race/ethnicity. We did this by testing for the presence of an interaction between race/ethnicity and each of the variables for each of the three measures of ill health. None of these tests revealed a significant interaction.

DISCUSSION

We set out to assess whether and to what extent intelligence might serve as an alternative or adjunctive flexible resource in a fundamental cause approach to understanding socioeconomic disparities in health. In our analyses of prospective data, we found two things: (1) the effects of SES variables on health outcomes remained significant and were changed little by controls for intelligence; (2) significant effects of intelligence were eliminated by controls for education and income (in four of five tests) or sharply diminished in magnitude (for the effect of intelligence on self-reported health in the HRS).

Potential Limitations

The measurement of health. Our analysis relies in part on self-reports of health conditions and self-rated health. Ideally one would also include measures from physical exams using physicians and biomedical tests. Nevertheless, our analysis is somewhat strengthened by two considerations. First, the measures we use range in objectivity from self-rated health (which is entirely subjective) to mortality (which is not subjective at all). The fact that our conclusions about SES and intelligence were consistent across measures that varied in terms of their objectivity gives us more confidence in the validity of our conclusion. Second, recent evidence has shown that self-reports of health conditions predict mortality at least as strongly as physician-assessed measures do, and perhaps more strongly (Ferraro and Farmer 1999). Thus, while an ideal study would include both physician-assessed and self-report measures, we believe the measures used were adequate for our purposes.

Timing of the IQ assessment in the WLS. In the WLS, we required intelligence measured in adolescence to compete with education and income, which were each assessed at a later time. Because (other things held equal) variables assessed closer to one another in time are more likely to correlate, our analysis of the WLS could be challenged for a failure to use a measure of adult intelligence. However, while the SES variables are ascertained after intelligence, they were measured long before the health outcomes. For example, family income was assessed in 1974, some 18 years before the health conditions were assessed. Of course the best solution to this problem would be to have multiple assessments of both SES and IQ. However, even in the absence of such an ideal circumstance, the HRS measure of intelligence, unlike the WLS measure, was assessed in adulthood at the beginning of a six-year follow-up period in which the onset of ill health was ascertained.[3] The fact that the pattern of results obtained in the HRS was consistent with findings in the WLS leads us to downplay the importance of this potential limitation regarding the analysis of the WLS.

The measurement of intelligence in the HRS. The assessment of intelligence in the HRS was not ideal in that it relied on relatively short tests administered in the context of a survey interview situation. In their defense, the HRS word-similarity measure is a component of the WAIS and correlates highly with the parent instrument. In addition, not only did the three available measures correlate with each other and form an adequately reliable scale, but the resulting composite measure also showed the expected strong association with educational attainment and significant bivariate associations with each of the measures of ill health. Still, the most compelling consideration in this regard comes from the fact that the same criticism cannot be raised concerning the assessment of intelligence in the WLS. The assessment used in that study contained 90 items covering most of the domains believed to be important in the measurement of general intelligence. Again, the fact that findings concerning the role of intelligence were relatively consistent across the two studies places constraints on the explanatory power of a potential weakness that applies to only one.

Sample selection. Both the WLS and the HRS are affected by sample selection bias, albeit in different ways. The WLS sampled individuals relatively early in life, before the cohort was substantially influenced by mortality —a relative strength. At the same time, the WLS used a mail survey to collect data in 1992, and, while the response rate was quite good for this mode of data collection, there was some nonresponse bias (see above). The HRS sampled individuals at late midlife and therefore could not include people who were born in the same period (1931–1941) but did not survive long enough to be entered into the study. In addition, we chose to construct a prospective design from the HRS that followed healthy persons forward to the development of fair/poor self-rated health, life-threatening illness, or death. In doing so, it was necessary to exclude persons with life-threatening illness in 1992 and, for the analysis of self-rated health, persons with fair/poor health in 1992. The sample is, therefore, selected for health to some degree. To the extent that low intelligence, education, or income played a role in generating the ill health or death of those who were excluded, this effect is not reflected in our analyses. An additional concern is that people of low SES (or low intelligence) who remain healthy, and who therefore remain in the sample, may be particularly robust individuals compared to individuals of higher SES (or higher intelligence) who have not been as strenuously challenged by adverse health circumstances. To the extent that these selection factors were operative, they should result in an underestimation of the effect of SES and intelligence on ill health in the HRS. Even so, both samples produce the expected associations between SES and health and between intelligence and health, and as such contain the key associations that are at issue. Because of this, it was possible to address the central theoretical issue: whether it is intelligence or SES that has direct effects on health in these two samples.

Possibility of unmeasured reciprocal effects. The prospective design does not allow us to take into account reciprocal effects between IQ and SES during adulthood. Substantial evidence indicates that IQ affects SES, and that SES simultaneously also affects IQ and cognitive ability, at least during the primary and secondary school years (Winship and Korenman 1997; Ceci 1991). The role of reciprocal effects in adulthood is less clear, but the results of the study suggest that whatever their influence, it is ultimately SES that plays the stronger direct role in health and that IQ is not a dominant health predictor when SES is controlled. Consequently, we are able to draw conclusions about our main research question without measuring reciprocal effects directly: while we acknowledge that SES and IQ are interrelated, the results of this study indicate that the substantial association of SES with health is not substantially mediated by IQ, nor is the association completely explained away as a spurious consequence of IQ.

Generalizability. The sample for the WLS was ascertained in one state and is almost entirely non-Hispanic white. The HRS, in contrast, was constructed to be nationally representative and to oversample African American and Hispanic respondents to ensure adequate statistical power in these groups. The convergence in findings between the two samples enhances the generalizability of the conclusions. Further, our check for statistical interaction between race/ethnicity and education, income, and intelligence in the HRS led us to the conclusion that no large differences in the effects of the key variables of interest to us were evident in different racial/ethnic groups.

Both samples are limited in terms of generalizability, as results cannot be extrapolated to early periods in the life course before follow-up began. In addition, we cannot generalize to cohorts that were not sampled. All members of the WLS graduated in a single year (1957) and are therefore roughly the same age. The HRS sample of persons born between 1931 and 1941 is somewhat broader. Our analysis of the HRS showed that age did not interact significantly with education, income, or intelligence, suggesting that at least within this ten-year band, the effects of these variables on health are relatively constant.

Overall assessment of limitations. As indicated above, limitations associated with the measurement of health, the timing of the assessments in the life course, the measurement of intelligence, the nature of sample selection, and issues of generalizability apply to differing degrees to one or the other of the two samples we used. By using two samples with different strengths and limitations, we sought to mitigate the consequences of these limitations on our ability to make conclusions. At the same time, the fact that across these limitations we need to counterbalance the strengths of one sample to offset the weaknesses of another underscores the fact that neither sample is ideal in and of itself. Thus, we propose that these findings provide useful but not definitive evidence concerning the issue we have studied.

IMPLICATIONS AND CONCLUSION

Direct Effect of Intelligence on Health

Investigators who assign a prominent role to intelligence argue that it has direct effects on health independent of SES. Direct effects will be present because the critical factor people use to construct a healthy lifestyle, make the most of health information, and navigate the health care system is intelligence. While previous studies have shown effects of intelligence on morbidity and mortality, these studies either failed to measure adult SES or failed to implement a prospective design. The results of this study do not support assertions that education may be a mere "surrogate for IQ" (Batty and Deary 2005) or that intelligence can stand as "the epidemiologists' elusive 'fundamental cause' of social class inequalities in health" (Gottfredson 2004:174). We found no evidence of a consistent linear effect of intelligence on health once education and income were held constant. In our samples, respondents with low intelligence but high education and respondents with robust intelligence but low education experience health outcomes consistent with their educational attainment and irrespective of their intellectual abilities. The fact that the results replicated in two samples with different strengths and limitations increases our confidence in this conclusion.

But why is it that intelligence has little independent effect on health? One possible reason is that it is not so much intelligence that is utilized to gain a health advantage as the SES-related resources themselves; not so much being intellectually adept as knowing the simple steps that can be taken to prevent sudden infant death syndrome or avoid an attack of asthma; not so much one's level of intelligence as having enough money to buy health insurance or afford life-saving screening tests; not so much being nimble-witted as having a powerful advocacy group that demands safer work conditions; not so much how smart one is as knowing someone who can provide appropriate help for an urgent health need. While intelligence contributes to the acquisition of SES-related resources, it is not the key flexible resource people use to obtain beneficial health circumstances. As such, intelligence cannot be the factor in fundamental-cause theory that explains why indicators of SES are re-expressed as causes of morbidity and mortality in different places and at different times.

A second, and perhaps even more important, reason is the possibility that SES-related resources operate to enhance health at contextual levels. Socioeconomic status affects health not just because it facilitates and encourages healthy behaviors but because associated resources of knowledge, money, power, prestige, and beneficial social connections also shape access to salutary contexts. For example, a person with many resources can afford to live in a high-status neighborhood where neighbors are also of high status and where, collectively, enormous clout is exerted to ensure that crime, noise, violence, pollution, traffic, and vermin have been kept at a minimum and that the best health care facilities, parks, playgrounds, and grocery stores are conveniently located nearby. The idea is that once a person has used SES-related resources to locate in an advantaged neighborhood, a host of health-enhancing circumstances comes along as a sort of "package deal." The person benefits in numerous ways that do not depend on his or her own initiative or ability to personally construct a healthy situation: It is an added benefit of the context. And, of course, the same sort of added benefit can be enjoyed in other important contexts such as social networks, work settings, families, and marriages. To the extent that SES operates to benefit health at this contextual level, one would expect intelligence to have little direct impact on health outcomes because it is not individual initiative or savvy that leads to better health in this scenario.

Indirect Effects of Intelligence

Our results are compatible with the possibility of indirect effects of intelligence on health that operate through the attainment of social and economic resources. Future research might seek to understand these indirect effects more fully. In particular, research focused on issues other than health has shown that there is a dynamic interplay between intelligence and educational and occupational experience across the life course (e.g., Farkus et al. 1997; Guo 1998; Guo and Harris 2000; Hauser and Huang 1997; Kohn and Schooler 1982; Schooler et al. 1999; Shavit and Featherman 1988), and it would be interesting to assess this dynamic interplay in relation to health. Our study cannot address such possibilities because we do not have the requisite data concerning intelligence, SES, and health over the life course that would allow us to further probe such effects. As a result, we leave this intriguing and important issue to future research.

SES and the Fundamental-Cause Approach

This study has significance for the theory of fundamental causes because it subjected that theory to a challenge that could have disconfirmed the approach or called for its significant modification. Fundamental-cause theory proposes that what people use to garner a health advantage are social and economic resources. The theory is challenged by an explanation that holds that intelligence is the key flexible resource people actually use to achieve a health benefit. The fact that fundamental-cause theory survived this particular challenge does not, of course, inoculate it from other potential challenges. Still, we become more convinced of the validity of an explanation as it survives successive challenges from competing explanations.

Support for the fundamental-cause theory presented here and elsewhere (Phelan et al. 2004; Lutfey and Freese 2005) directs attention to the broadly serviceable resources that SES confers and to the power that such resources hold in creating and recreating SES gradients in health. It opens the possibility for the future development of a more fully explicated sociological explanation for health disparities. Progress toward this end has been limited by an orientation that assigns irrelevance to social conditions based on the belief that any health effects they confer can be accounted for by more proximal and seemingly more modifiable risk and protective factors. The theory of fundamental causes provides an antidote to this line of thinking by indicating that access to flexible social and economic resources creates and recreates associations between social circumstances and risk and protective factors. The theory tells us that when sociologists study how status is attained, power allocated, knowledge conferred, income acquired, and beneficial social connections configured, they simultaneously provide essential information for understanding the social distribution of disease and death.

NOTES

1. When listwise deletion is employed, the association between income and mortality in the WLS is not significant; otherwise, the pattern of significant associations in Table 1 is identical, regardless of the imputation approach employed.
2. Controlling the effects of education and income for each other generally leads to a reduction in the magnitude of the unique effect of each. Three of the 11 significant effects of one of these variables is reduced to nonsignificance (1 for education and 2 for income) when the other is controlled. The three that dip below significance are: in the HRS, the education effect on life-threatening illness; and in the WLS, the effects of family income on mortality and life-threatening illness. Because there is no consistent pattern indicating that one variable dominates the other, we conclude that both are important and frequently share explanatory power.
3. A potential concern with the adult assessment of intelligence in the HRS is that such abilities may decline with age. If this were true, especially the older members of the HRS cohort may have enjoyed better functioning during most of their lives than their scores suggest. However, Alwin and MacCammon (1999, 2001) have found that age effects are relatively small and that any decline that does occur tends to begin after age 60 and thus occurs after the age of enrollment into the HRS.

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