Health Selection and the Process of Social Stratification: The Effect of Childhood Health on Socioeconomic Attainment*

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This study investigates whether childhood health acts as a mechanism through which socioeconomic status is transferred across generations. The study uses data from the Panel Study of Income Dynamics to track siblings and to estimate fixed-effects models that account for unobserved heterogeneity at the family level. The results demonstrate that disadvantaged social background is associated with poor childhood health. Subsequently, poor health in childhood has significant, direct, and large adverse effects on educational attainment and wealth accumulation. In addition, childhood health appears to have indirect effects on occupational standing, earnings, and wealth via educational attainment and adult health status. The results further show that socioeconomic health gradients are best understood as being embedded within larger processes of social stratification.

This paper examines the interplay between health and socioeconomic status by asking three questions. First, does poor health early in life adversely affect the amount of schooling one is able to complete, the socioeconomic position of one's occupation, labor earnings, or wealth accumulation? Second, is childhood health a mechanism for the intergenerational transmission of socioeconomic status? Third, does health selection help explain the socioeconomic gradient in health? The answers to these questions have important implications both for research on health disparities and for understanding social stratification. I address these questions using the Panel Study of Income Dynamics.

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BACKGROUND

The following review briefly summarizes previous research on health disparities, with an emphasis on issues of causality. It then draws upon sociological work on status attainment to highlight the utility of embedding health disparities within larger theories of social inequality.

The Socioeconomic Gradient in Health

Within demographic and epidemiological research, one of the most consistent empirical findings over the last 50 years has been the strong relationship between health and socioeconomic status (SES), measured along several dimensions. Those who are more educated, have higher incomes, work in more prestigious occupations, and possess more wealth have better health, lower disability, and lower mortality risk than their lower-SES counterparts (House et al. 1994; Kitigawa and Hauser 1973; Marmot 2001; Moore and Hayward 1990; Syme and Berkman 1976; Williams 1990). This social gradient has been found at the beginning of life (Singh and Yu 1995) and among the aged (Smith and Kington 1997).

For a number of reasons, health inequalities have historically occupied a less visible role in social science research than they do currently. First, there was a substantial decline in mortality over the 20th century. Simultaneously, Western industrialized countries expanded their social welfare states, usually including universal health care systems, as in the United Kingdom, or systems targeted at the poor (Medicaid) and elderly (Medicare), as in the United States. The implicit assumption was that, with declining mortality and the advent of social safety nets to provide basic medical services for the elderly and indigent, inequalities in health would disappear. Finally, despite a great deal of work historically on economic conditions and health, modern epidemiology has, until recently, usually viewed SES as a confounding variable, either outside of or hopelessly distal from disease processes. In the 1980s, spurred by the publication of the British "Black Report," health inequalities again became a focus of research (Townsend and Davidson 1982). The Black Report shattered the notion that health disparities were declining and raised serious doubts about differential access to health services as the central determinant of health disparities.

Causality and the Social Gradient in Health

Causality has long been a central issue in health disparities research. Sociological and epidemiologic studies of health inequalities almost invariably begin with an a priori assumption of social causation. That is, lower SES is thought to have detrimental causal effects on health (Ross and Wu 1995). Mechanisms by which SES is thought to affect health include differential access to and utilization of health services (Ross and Wu 1995), exposure to occupational hazards and environmental pathogens (Toscano and Windau 1994), lower levels of social support (Berkman and Syme 1979; Thoits 1995), the cumulative effects of stress (McEwen 1998; Thoits 1995), and differences in health-related risk behaviors (e.g., smoking) (Kaplan et al. 1987). Mirowsky and Ross (2003) synthesize many of these mechanisms under the conceptual paradigm of *education as learned effectiveness.* They argue that increased educational attainment improves health primarily by increasing individual agency, self-efficacy, and problem-solving capacity, all of which promote a healthy lifestyle (Mirowsky and Ross 2003). Similarly, Link and Phelan (1995) conceptualize SES as a *fundamental cause of disease,* a cause of causes, as it is a primary determinant of access to important health-related resources and because it shapes numerous health outcomes via a plethora of mechanisms.

Another class of explanations suggests that the relationship is spurious. Either there may be some unobserved factor jointly determining SES and health, creating a spurious association, or there may be differential subjective interpretations of health by social class (Kadushin 1964). Finally, in the case of mortality rates, there may be incongruence between numerator and denominator. The general consensus is that it is very unlikely that observed health disparities are due to either spurious correlations or statistical artifact (Bloor, Samphier, and Prior 1987).[1]

A third explanation, *health selection,* reverses the direction of causality so that lower SES is explained by poor health. It is possible to differentiate two variants of the health-selection hypothesis. In type I, also known as *drift,* those in poor health are selected into lower SES because of decreased labor force participation, thereby decreasing wage income and inhibiting wealth accumulation. Furthermore, health problems may necessitate spending previously accumulated wealth. Economists have presented evidence of substantial adverse effects of health shocks on labor force participation, income, and wealth (Chirikos and Nestel 1985; Luft 1975; Smith 1999, 2005). A recent report estimates that medical problems contributed to half of all bankruptcies in the United States in 2001 (Himmelstein et al. 2005). The type II or *stunting* variant argues that poor health—particularly during critical periods of childhood and adolescence—may limit an individual's initial accumulation of human capital and subsequent ascent to higher positions of prestige, power, and wealth. Thus, health selection may operate in two distinct ways: either by inducing downward social mobility or by preventing movement upward.

Though social causation and selection are often presented as if they were mutually exclusive, it is likely that there is a lifelong synergistic relationship between SES and health. One period's health is a function of the previous period's health both directly and indirectly via the influence of poor health on current income and wealth. For example, poor childhood health, itself a product of socioeconomic dis-

advantage, may lead to lower educational attainment and skill formation, diminished labor market outcomes, lower earned income and wealth accumulation, and fewer resources to invest in the next period's health. In this way, there is a constant interaction between health and SES via both selection and social causation over the life course. Because poor health is a consequence of social inequalities and contributes to them, it makes intuitive sense to integrate the study of health disparities into larger models of social stratification.

Health in the Status Attainment Process

Within sociology, the status attainment model is the dominant paradigm used to explain the reproduction of socioeconomic status. Stratification research is fundamentally concerned with who gets what and why (Lenski 1966). Traditionally, this has meant the study of the determinants of *life chances.* Life chances are almost always defined according to economic and status criteria. Blau and Duncan (1967) outlined a model with an emphasis on educational attainment as the principal mechanism by which parental education and occupational status influence children's socioeconomic attainment in adulthood. Sewell, Haller, and Portes (1969) and Sewell and Hauser (1975) later extended this model to include social-psychological variables such as aspirations, peer influence, and ability as causal mediators. Where does health fit into the status attainment model? In most analyses, it is ignored, even though the *chances for life* (longevity and health) are arguably the most important rewards doled out by societies.

For childhood health to be an important mechanism in the intergenerational transmission of socioeconomic status, it must both be a product of parental socioeconomic status and have an important causal impact on children's subsequent socioeconomic attainments later in life (Palloni and Milesi forthcoming). Both requirements appear to be met. Research has shown parental socioeconomic status to be an important determinant of various infant and child health outcomes, including preterm birth (Homer et al. 1990), infant and child mortality (Gortmaker 1979; Mare 1982), and illness (Case, Lubotsky, and Paxson 2005; Starfield 1991).

Research has also linked early health to factors that play important indirect roles in socioeconomic attainment. For example, poor childhood health has been linked to poor childhood cognitive development and achievement (Boardman et al. 2002; Edwards and Grossman 1979; Lichtenstein et al. 1993; Matte et al. 2001; O'Brien Caughy 1996; Richards et al. 2001; Sorensen et al. 1997; Wadsworth 1986) and lower rates of high school completion (Conley and Bennett 2000; Wadsworth 1986). A few studies have also explicitly attempted to discern the impact of early-life health on adult SES attainment and social mobility directly, though with mixed results. Wadsworth (1986) observed that British boys (but not girls) who had experienced a serious illness during childhood were more likely to belong to a lower occupational class at age 26 than those who had not, regardless of social class of origin. Serious childhood illness, particularly in the preschool years, also led to significantly lowered odds of acquiring educational credentials for both boys and girls. Power, Fogelman, and Fox (1986) use height as an indicator of childhood health and living conditions. They find a distinct social gradient in height and a lower percentage of short people among those who were upwardly mobile. Similarly, Illsley (1986) observed that women who were upwardly mobile, defined by comparing their husbands' social class to their fathers', tended to be unusually healthy.

Research in the United Kingdom and Scandinavia has often found that, compared to those in their class of origin, those who were upwardly mobile experienced better health and lower mortality. Those who were downwardly mobile experienced poorer health outcomes (Bartley and Plewis 1997; Blane, Harding, and Rosato 1999; Elstad 2001; Manor, Mathews, and Power 2003). However, compared to those in their class of destination, the downwardly mobile tend to experience better health and the upwardly mobile worse health. There are a number of limitations to these studies, though. First, by focusing exclusively on occupational mobility, they ignore the important effect of early health on educational attainment. Second, by analyzing mobility between broad occupational class categories, a fair amount of mobility may be missed.

Lundberg (1991) examined the effect of childhood living conditions and health in early adulthood on inter- and intragenerational class mobility. The experience of economic hardship in childhood significantly lowered the likelihood of ending up in a high social class in adulthood. Illness in early adulthood was posi-

tively related to labor force departure before age 65. However, there were no observed effects on intergenerational class mobility, and only weak effects were observed on intragenerational mobility. Again, by observing health in early adulthood, Lundberg would have missed any effects of selection that occurred prior to early adulthood, specifically via educational attainment. A better test of health selection would measure health earlier in the life course. This analysis does this by measuring childhood health over the period from birth to age 16.

One dimension of socioeconomic position expected to be especially sensitive to the deleterious effects of poor childhood health is wealth accumulation. Childhood health may negatively affect wealth through diminished savings. Labor earnings are the engine that drives savings (Modigliani 1988). The chronically sick and the less educated are less productive, have weaker ties to the labor market, and command lower wages than those who are healthy and well educated. To the extent that poor childhood health is associated with decreased educational attainment, poor adult health, and diminished labor market outcomes, it is likely to have adverse effects on savings and wealth. There is a growing body of research showing that poor health early in life, such as low birth weight and certain childhood diseases, has lasting effects on adult health (Blackwell, Hayward, and Crimmins 2001; Haas 2004).

Hypotheses

Based on the previous discussion, I propose the following four hypotheses: (1) Those from disadvantaged social backgrounds will experience worse infant and childhood health outcomes. (2) Poor childhood health will have substantial adverse effects on educational attainment, occupational standing, earnings, and wealth. (3) Given the relatively large impacts of health shocks on wealth observed over the short term, and the cumulative impact over the long term via effects on educational and occupational attainment, the impact of childhood health on wealth will be especially pronounced. (4) After accounting for type II health selection, education will continue to be positively associated with adult health.

To test these hypotheses, I integrate health into the process of social stratification as both a cause and a consequence of SES attainment. I also extend previous work on health disparities by (1) explicitly examining the impact of poor childhood health on socioeconomic "stunting" and (2) controlling for unobserved heterogeneity in family of origin using sibling fixed-effects models.

METHODS

Data

This analysis utilizes the Panel Study of Income Dynamics (PSID) (Institute for Social Research 2001). Begun in 1968, the PSID is a long-running, nationally representative, longitudinal survey that tracks the economic and demographic dynamics of households. Because of its duration, breadth, and high quality, it is among the most widely used social science data resources. Between 1968 and 1996, all individuals from core households were interviewed yearly, regardless of whether they had remained in the same household. The PSID therefore follows the children of original 1968 households who grow up, leave the family home, and establish their own households. In this manner, new households and individuals are constantly being added to the sample, while others leave through death and sample attrition. In 1997, follow-up was changed to every other year, and the core sample was reduced to 6,168 households.

Of the 4,802 households interviewed in 1968, 88.5 percent were reinterviewed in 1969. Subsequent response rates have hovered in the high nineties (Hill 1992). Despite overall yearly response rates at or above 95 percent, a survey that collects data on subjects for more than 30 years will eventually have problems with attrition. By 1988, almost 45 percent of the original 1968 sample had been lost, either through mortality or nonresponse. Previous research has examined sample attrition in the PSID and the potential bias introduced. In general, this work has found that attrition is more likely among those with low or high income and those receiving public assistance, while married individuals and those who were children in the original households are more likely to remain (Becketti et al. 1988; Duncan and Hill 1989; Fitzgerald, Gottschalk, and Moffitt 1998a, 1998b). Though attrition is correlated with observed characteristics, these variables explain only a very small amount of the variation, so attrition appears to be largely stochastic. Previous research also suggests that sample attrition bias in analyses of important out-

comes such as earnings, education, marital status, and welfare participation is minimal in the PSID (Becketti et al. 1988; Fitzgerald et al. 1998a, 1998b).

The PSID has data on approximately 10,000 respondents who are household heads or "wives." As a convention, the PSID uses the term "wives" to refer to both the legal spouses of the male household heads as well as cohabiting female partners. Though it uses information gathered in various waves, the present analysis is limited to employed heads and wives present in both the 1999 and 2001 waves, as the measure of childhood health was gathered only from these respondents in these years. This reduces the sample to roughly 8,500 individuals. As the analyses are concerned with earnings and occupational standing, they are limited to those currently working for pay and to those ages 64 and under. Also, due to their small numbers in the PSID, Asians and Hispanics are excluded.

Finally, the sample is limited to those respondents for which information on birth weight status is known. These respondents represent the employed children of original 1968 household heads and wives who have since established their own households. Besides helping to validate the childhood health measure, this subsample is also composed largely of sets of siblings and therefore permits the use of fixed-effects models to control for unobserved heterogeneity.[2] After these exclusions, there is only a small amount of missing data on most variables. With the exception of the variables discussed below, missing data were deleted listwise. The final analytic sample for the structural equation model (SEM) used in this analysis (n = 2,805) is on average seven years younger than the full sample of heads and wives, with a mean age of about 39. Descriptive characteristics for the sample along with a correlation matrix can be found in the Appendix. In the fixed-effects analysis, the sample is further constrained to those who have a sibling in the sample. The fixed-effects models are based on 2,279 cases drawn from 762 families. The sample characteristics for the fixed-effects analysis are essentially identical to those in the SEM analysis described in the Appendix.

Measurement

Infant and childhood health. Infant health is assessed by an indicator of low birth weight based on mothers' reports from fertility histories collected in 1986. Such indicators have been shown to be a reliable and valid measure of actual birth weight (Walton et al. 2000). A strong relationship between birth weight and objective measures of childhood health has been consistently reported (Brooks et al. 2001; Vohr et al. 2000). For these PSID respondents, birth weight is only available in a form that compares those who weighed less than 88 ounces to those who weighed more.

In the United States, the investigation of the effects of childhood health on later-life outcomes has been limited by the scarcity of prospective life course data. Researchers must therefore find alternative ways of assessing these effects. This analysis utilizes retrospective reports. Respondents were asked, "Consider your health while you were growing up, from birth to age 16. Would you say that your health during that time was excellent, very good, good, fair, or poor?" Values of 4 (excellent) to 0 (poor) were assigned to these categories. Retrospectively reported childhood health is potentially subject to recall bias and measurement error, but previous research suggests that the quality of retrospective measures of childhood health is reasonably good. It has been shown using data from the PSID and the Health and Retirement Study that retrospective reports of overall childhood health are reliably reported over time (Goodman-Kruskal gamma = 0.6), especially when the measure was dichotomized into a good/very good/excellent vs. fair/poor comparison (Goodman-Kruskal gamma = 0.9) (Haas 2004). Quality of measurement did not vary substantially by gender or age. However, those with higher levels of education were more consistent reporters of childhood health. Elo's (1998) analysis of these measures further demonstrated a high level of internal consistency between the report of general health and reports of specific long-term childhood health limitations.

Krall and colleagues (1988) validated retrospective self reports of childhood communicable diseases, accidents, hospitalizations, and surgeries. In the first year of life, subjects were examined by a pediatrician every three months. Examinations and parental interviews were then performed twice a year between age 1 and age 10 and yearly until age 18. Retrospective childhood health questionnaires were administered at ages 30, 40, and 50. With the exception of German measles, illnesses were recalled

with a very high level of accuracy, averaging 85 percent at age 50. Accidents and surgeries were recalled correctly 75 percent and 89 percent of the time at age 50, respectively. Reliability did not change much between age 30 and age 50, nor was recall accuracy correlated with education (Krall et al. 1988).

Adult health. Current self-rated health status, measured as 4 (excellent) to 0 (poor), was ascertained in the 2001 wave. While self-rated health is not a perfect measure of current health status, previous research has shown it to be a reliable and valid measure of general physical well-being that is highly correlated with objective measures of physical health, including mortality risk and physicians' assessments (Idler et al. 2004; Idler and Kasl 1991).[3]

Socioeconomic status. The analysis investigates multiple dimensions of SES. Educational attainment is measured as years of completed schooling. Earnings data for tax year 2000 were collected in 2001 and are the sum of several labor income components including wages, salaries, bonuses, overtime, tips, commissions, and professional practice or trade. The analysis uses a logarithmic transformation of labor earnings. For most individuals and families, labor earnings are the primary source of income. In addition, unlike household income, labor earnings can be directly linked to an individual and thus provide greater insight into the relationship between health and SES at the individual level. Wealth is measured as the log of net total household assets, subtracting all debts. This includes all real estate, vehicles, farm or business assets, equities and bonds, savings and checking accounts, IRAs, pensions, trusts, and inheritances.

Occupational standing of the respondent's first full-time and current occupation is measured by occupational education, which is defined as the percentage of incumbents within a specific occupation with at least one year of college education (Hauser and Warren 1997). Occupational education reflects the average socioeconomic status of occupations. Hauser and Warren (1997) have demonstrated that occupational differentiation occurs primarily by differences in educational attainment and that the contribution of wage rates is negligible. About 6 percent of respondents had missing data on first occupation. Education-specific mean imputation was used to assign values to these cases. This assigns the mean level of occupational education of those with the equivalent level of education as the respondent.

Parental socioeconomic status. Father's occupational standing is also measured by occupational education based on the respondent's report of father's usual occupation during the period between the respondent's birth and age 16. Approximately 12 percent of respondents have missing data on father's usual occupation. For these cases, father's education-specific mean imputation was performed. This assigns the mean level of occupational education of those with the same level of education as the father. In addition, social background is assessed using father's and mother's educational attainment as a set of three dummy variables each for mother and father. These include less than high school (0–11 years) (referent), high school and beyond (12+ years), and missing.

Analysis

The effects of childhood health on SES are first analyzed within a recursive SEM. The effect of exogenous variables (Γ) and between endogenous outcomes (β) are estimated by the following structural equation,

$$\eta = \beta\eta + \Gamma\xi + \varsigma\,,$$

where η is a vector of endogenous outcomes, ξ is a vector of exogenous predictors, and ς is a vector of the random disturbances in η. The measurement models for Y and X are

$$Y = \Lambda_y\,\eta + \varepsilon$$

and

$$X = \Lambda_x\,\xi + \delta\,,$$

where Λ_x and δ are the identity and zero matrix, respectively. As childhood health was assessed twice, each of these is included in the model to allow for measurement error in the latent childhood health construct. Therefore,

$$\Lambda_y = \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ * & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix} \text{ and } \varepsilon = \begin{bmatrix} 0 \\ * \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}.$$

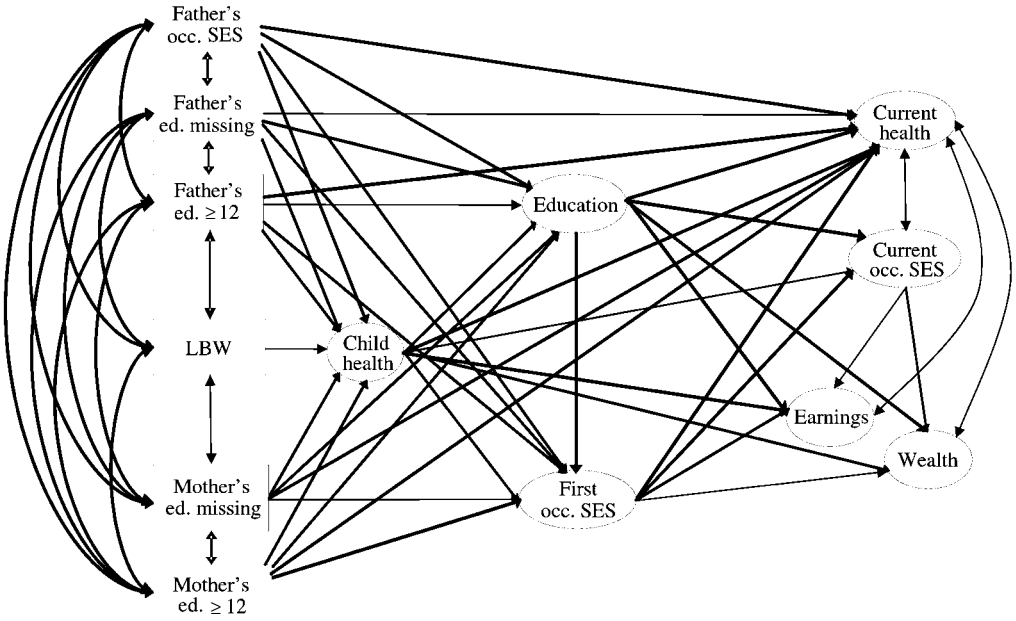
The causal structure of the model is presented in Figure 1. Childhood health is allowed to have direct effects on adult SES and current adult health and indirect effects via education and early occupational standing. The effects of parental SES on respondents' later attainments (current occupational standing, earnings, and wealth) are constrained to act exclusively through early attainments (childhood health, educational attainment, and early occupational standing). As there is substantial empirical evidence to suggest that adult health is both a cause and consequence of employment, income, and wealth, it does not make sense to privilege either a selection or causation specification for these variables. Therefore, the model is agnostic as to the direction of causation between health and current occupational status, earnings, and wealth. These three associations are estimated by allowing their disturbances (ς) to covary. Though not presented in Figure 1 for the sake of clarity, the model also includes controls for age, race, gender, and marital status. The results presented below are from unweighted analyses because the sample selection criteria may make the available weights questionable. However, the substantive findings are robust to weighting.

An issue that looms large is potential unobserved heterogeneity at the family level, in which unobserved characteristics of families may act as a determinant of both childhood health and adult SES, revealing a spurious association where no causal association exists. Take the following equation,

$$Y = \alpha + \beta C + \gamma \boldsymbol{F} + u \,,$$

where Y is the number of years of completed schooling, C is a measure of childhood health, and $\boldsymbol{F}$ is a vector of unobserved family characteristics. If C and $\boldsymbol{F}$ are correlated and $\boldsymbol{F}$ is not included in the model, then estimates of the effect of childhood health on educational attainment (β) will be biased (Griliches 1979). Sociologists typically try to solve this problem by including as many variables to account for $\boldsymbol{F}$ as possible. However, to the extent that unknown or unmeasured family-level characteristics are excluded, estimates are still subject to omitted variable bias. An alternative approach takes advantage of sibling sets in the PSID. By conditioning on family of origin, sibling fixed-effects models (otherwise known as within-family models) have the effect of controlling for all $\boldsymbol{F}$ variables. Thus, sibling fixed-effects models compare siblings along the characteris-

FIGURE 1. Path Diagram of Recursive Structural Equation Model



Notes: SES = socioeconomic status. LBW = low birth weight.

tics in which they differ while holding fixed all the stable background and family characteristics (both observed and unobserved) that they share. For example, in this analysis, the indicators of parental socioeconomic status are not included in the model, as they are invariant across siblings.

Estimation is accomplished by including a set of family-specific dummy variables. Thus, the outcome of interest is regressed upon the predictors while including (k – 1) indicator variables corresponding to k original 1968 families within which the siblings are clustered (for similar applications of this method, see Conley and Bennett 2000, Currie and Thomas 1995, and Jencks et al. 1972). This estimation utilizes one degree of freedom to estimate each of these family-specific means, with one family excluded as the referent (these 761 parameters are not presented, as they are not of substantive interest).

One issue with this type of sibling model is making sure that there is enough within-family variation to get credible parameter estimates. In the present study, there is substantial within-family variation. For all of the outcome variables (including childhood health), more than half of the observed variation occurs within families, rather than between them. The variable with the lowest proportion of within-family variation is educational attainment, in which 54 percent of variation is within families and 46 percent occurs between families. All other variables have more than 70 percent of their variation occurring within families. Adult health, earnings, and wealth have more than 80 percent of their variation occurring within families.

RESULTS

Table 1 presents the unadjusted mean levels of SES by childhood health status. Though these estimates are based on small numbers of cases for some categories, and they do not control for important covariates, the magnitude of the differences is nonetheless quite dramatic. Those who reported poor childhood health completed approximately 1.8 fewer years of schooling; are almost half as likely to work in an occupation in which the average incumbent has been to college; earn on average $13,000 less annually; and have a staggering $165,000 (77%) less in assets compared to their peers who reported excellent health. Childhood health appears to exert a stronger impact on later life attainments than earlier ones (education and initial occupational standing). It also appears that earlier attainments may be less monotonically associated with childhood health than are later attainments. However, this may be due to the small n's, especially in the poor childhood health category.

Childhood Health as a Mechanism of Intergenerational SES Transmission

The results of the multivariate model are presented in Table 2. Regarding the role of early life health in the transmission of socioeconomic status, it is necessary to establish two conditions: that childhood health is a function of parental SES (hypothesis 1) and that it in turn affects adult SES (hypothesis 2). As to the first condition, there is a statistically significant direct effect of father's education on childhood health. Those whose fathers completed 12 or more years of schooling had childhood health scores 0.12 higher than those whose fathers did not complete high school. This is equivalent to a 0.06 standard deviation increase in childhood health. The effects of mother's education and father's occupational standing, though in the expected direction, were not statistically significant. In addition, there was also an indirect effect of parental SES on childhood health via low birth weight. High-SES parents were significantly less likely to have low-birth-weight children (the z scores for the covariance between low birth weight and fa-

TABLE 1. Unadjusted Distribution of Adult SES by Childhood Health

	Childhood Health (1999)					
	Excellent (n = 1,482)	Very Good (n = 849)	Good (n = 392)	Fair (n = 73)	Poor (n = 9)	Ratio Exc./Poor
Years of schooling	13.65	13.38	12.86	13.33	11.89	1.15
Occupational SES first job	26.85	23.69	20.53	22.23	23.22	1.16
Occupational SES current job	37.87	35.41	29.55	29.52	20.67	1.83
Earnings in $	41,615	34,931	28,032	27,734	28,512	1.46
Household wealth in $	215,270	137,596	110,754	54,834	49,617	4.34

Note: SES = socioeconomic status.

ther's occupational standing and maternal education are 3.33 and 2.64, respectively). Those who were born with low birth weight subsequently had significantly lower childhood health scores (–0.13). This is approximately a 0.04 standard deviation decrease in child health. Also, given the consistent relationship between birth weight and objective measures of childhood health, the fact that a similar relationship is found using retrospective reports lends support to their validity.

Childhood Health and Status Attainment

The evidence that poor childhood health in turn adversely affects socioeconomic attainment (hypothesis 2) is also quite strong. Childhood health has significant and relatively large effects on educational attainment. Being in excellent rather than poor health in childhood is associated with 0.52 additional years of education. The magnitude is roughly equivalent to having one parent who completed 12 or more years of schooling (0.62 for fathers and 0.57 for mothers). Those who were healthy in childhood also were more likely to be healthy in adulthood, whereas those in poor health in childhood also experienced poor health in adulthood. A standard deviation increase in childhood health is associated with a 0.29 standard deviation increase in current self-rated adult health. Poor childhood health is also associated with significantly diminished initial and current occupational status. Those who experienced poor rather than excellent childhood health worked in first and current occupations in which about 4 percent fewer of the incum-

TABLE 2. Parameter Estimates of Recursive Structural Equation Model

	Outcome						
	Childhood Health	Educational Attainment	Occ. SES, 1st Job	Adult Health	Occ. SES, Current Job	Log Earnings	Log Wealth
	Gamma coefficients (robust standard error)						
Low birth weight	–.13*	—	—	—	—	—	—
	(.06)						
Father's occupational SES	.00	.02***	.11***	.00	—	—	—
	(.00)	(.00)	(.02)	(.00)			
Father missing education	.05	–.32*	1.56	.00	—	—	—
	(.07)	(.15)	(1.58)	(.07)			
Father ≥ 12 years education	.12*	.62***	3.39**	.04	—	—	—
	(.05)	(.10)	(1.05)	(.04)			
Mother missing education	–.11	–.32	1.22	.17	—	—	—
	(.09)	(.20)	(2.13)	(.09)			
Mother ≥ 12 years education	.09	.57***	.37	.11*	—	—	—
	(.05)	(.10)	(1.05)	(.04)			
Male	.09**	–.18*	–4.91***	.08*	–4.61***	.47***	.11
	(.03)	(.07)	(.76)	(.03)	(.82)	(.04)	(.16)
Black	–.10*	–.17*	–.66	–.21***	–4.41***	–.04	–1.51***
	(.04)	(.09)	(.91)	(.04)	(.91)	(.04)	(.17)
Age (years)	.00*	.04***	.14**	–.02***	.02	.01**	.12***
	(.00)	(.00)	(.05)	(.00)	(.05)	(.00)	(.01)
Married	.10**	.33***	2.02*	.07*	1.72*	–.03	2.05***
	(.04)	(.07)	(.80)	(.03)	(.86)	(.04)	(.16)
	Beta coefficients (robust standard error)						
Childhood health	—	.13**	.90*	.30***	.97*	.06**	.23**
		(.04)	(.43)	(.02)	(.46)	(.02)	(.08)
Educational attainment	—	—	5.49***	.04***	5.40***	.05***	.20***
			(.20)	(.01)	(.23)	(.01)	(.05)
Occupational SES, first job	—	—	—	.00**	.28***	.00**	.01
				(.00)	(.02)	(.00)	(.00)
Occupational SES, current job	—	—	—	—	—	.01***	–.00
						(.00)	(.00)
Log earnings	—	—	—	—	—	—	.53***
							(.08)
R^2	.03	.23	.34	.19	.40	.17	.25

* $p < .05$; ** $p < .01$; *** $p < .001$ (two–tailed tests)

Notes: N = 2,805. $\chi^2_{(df)} = 391.14_{(37)}$. SES = socioeconomic status. Root mean square error approximation = .057. Goodness-of-fit index = .98.

bents in that occupation had at least some college.

The effects of childhood health on the financial dimensions of SES (earnings and wealth) are perhaps the most dramatic. Reporting excellent rather than poor childhood health is associated with 22 percent higher earnings (((e$^{.05}$ – 1) * 100) * 4) and 104 percent more assets (((e$^{.23}$ – 1) * 100) * 4). Poor childhood health also influences adult occupational SES, earnings, and wealth indirectly through diminished educational attainment and adult health. This diminished human capital accumulation has a negative impact on the returns to labor and the process of wealth accumulation over the life course. Finally, as expected, the correlations between adult health and occupational status, earnings, and wealth (0.03, $p < 0.05$; 0.06, $p < 0.001$; 0.03, $p < 0.05$, respectively) are all positive and significant (not shown).

It is also important to note that, despite the relatively strong evidence of health selection presented here, the strong positive association between SES and adult health predicted in hypothesis 4 remains. Those with more completed education report significantly better current self-rated health. Similarly, those whose first jobs were in higher-status occupations also reported significantly better current health. Thus, in the SEM analysis, there is evidence that health inequalities result from both selection and social causation.

Sibling Fixed Effects

The question remains, however, whether unobserved family characteristics are driving the association between childhood health and SES attainment. To address this issue, the results of sibling fixed-effects analysis are presented in Table 3. These results largely echo those in the SEM analysis, though with a few notable differences. Those born with low birth weight have substantially diminished childhood health outcomes relative to their heavier siblings. Poor relative to excellent childhood health continues to be associated with about a half a year less of completed schooling. Those who experienced excellent childhood health have accumulated more than 104 percent more wealth than their sickly siblings (((e$^{.23}$ – 1) * 100) * 4). They also have better self-rated health in adulthood.

However, there are a few important differences between the results of the fixed-effects analysis and the SEM analysis. First, the adverse effect of low birth weight on childhood health is almost 2.5 times larger in the fixed-effects model compared to standard estimates, suggesting that unobserved family characteristics may be masking the true effect of low birth weight on childhood health. The direct effect of childhood health on occupational status and

TABLE 3. Parameter Estimates of Sibling Fixed-Effects Model

	Outcomes						
	Childhood Health	Educational Attainment	Occ. SES, 1st Job	Adult Health	Occ. SES, Current Job	Log Earnings	Log Wealth
Low birth weight	–.32***	—	—	—	—	—	—
	(.09)						
Male	.11*	–.21*	–4.71***	.07	–6.45***	.47***	.07
	(.04)	(.08)	(1.02)	(.04)	(1.11)	(.05)	(.21)
Age (years)	–.00	.00	–.00	–.02***	.04	.00	.12***
	(.00)	(.01)	(.07)	(.00)	(.07)	(.00)	(.01)
Married	.08	.02	1.54	.03	2.07	–.07	1.84***
	(.05)	(.09)	(1.09)	(.05)	(1.18)	(.05)	(.22)
Childhood health	—	.12*	–.21	.40***	.27	.03	.23*
		(.05)	(.60)	(.03)	(.65)	(.03)	(.12)
Educational attainment	—	—	4.75***	.02	4.66***	.02	.05
			(.32)	(.01)	(.37)	(.01)	(.07)
Occupational SES, first job	—	—	—	.00**	.26***	.00**	.01
				(.00)	(.03)	(.00)	(.01)
Occupational SES, current job	—	—	—	—	—	.01***	.00
						(.00)	(.01)
Log earnings	—	—	—	—	—	—	.36***
							(.10)
R^2	.49	.71	.66	.63	.70	.58	.62

* $p < .05$; ** $p < .01$; *** $p < .001$ (two-tailed tests)
Notes: N = 2,279; clusters = 762. SES = socioeconomic status.

earnings is no longer statistically significant. However, poor childhood health continues to adversely affect labor market outcomes indirectly via diminished educational attainment. Another important difference between the SEM and fixed-effects estimates is that the estimated effect of educational attainment on current health is cut in half and is no longer statistically significant. Once unobserved heterogeneity is taken into account, there do not appear to be significant educational differences in adult health. Early occupational standing continues to exert a significant impact on current health, so there is some evidence that social causation mechanisms are still at work.

DISCUSSION

This analysis suggests that conceiving of health as a *cause* and a *consequence* of larger processes of social stratification can provide insight both into how health disparities emerge over the life course and how they contribute to the placement of individuals in socioeconomic hierarchies. In contrast to previous studies that have found mixed evidence of health selection, this study presents relatively strong evidence that health selection does occur. The results show childhood health to be an important mechanism in attainment processes. Disadvantaged social background leads to poorer childhood health both directly and indirectly through increased risk of poor birth outcomes. In turn, those who experienced poor health in childhood complete significantly less education, occupy lower occupational niches, and have substantially diminished economic returns in the form of labor earnings and especially in the form of accumulated wealth. The results also confirm recent findings that poor childhood health is associated with subsequent poor health in adulthood (Blackwell et al. 2001; Haas 2004).

The sizes of these associations are not trivial, and in the case of wealth accumulation are quite dramatic. The fixed effects estimates also demonstrate that the effects of child health on educational attainment and wealth are robust to the influence of stable unobserved family characteristics. The results presented here also likely reflect a lower bound estimate of the true economic impacts of childhood health. One of the primary mechanisms by which childhood health would be expected to affect adult economic outcomes (particularly wealth) is as a determinant of adult health and subsequently of labor force participation. Economists have repeatedly shown current health to be a major determinant of labor force status. Therefore, as poor childhood health adversely affects adult health and labor force participation, it also has a large impact on life cycle savings. By excluding those who are not currently in the labor market, this analysis is thus excluding those for whom childhood health likely has the largest impact.

Disentangling the mechanisms by which childhood health affects educational attainment is difficult. Work by Meijer et al. (2000) hints at social functioning as a possible mechanism. They find that chronically ill children are more submissive and have more restricted social activities than their healthy peers. Illness may also be associated with increased periodic or prolonged school absences and impaired cognitive development. Wadsworth (1986) observed that children who had been seriously ill before age 10 had significantly lower achievement scores at age 15. Similarly, using height as a proxy for childhood health, Douglas, Ross, and Simpson (1968) found that high academic achievers were taller than lower achievers. Another possible explanation for the effect of childhood health on educational attainment is that the experience of chronic poor health in childhood may alter individual preferences for educational achievement and attainment. Further research is needed to uncover the mechanisms by which poor health in childhood leads to lowered educational attainment.

The findings also raise important questions about the relative importance of selection versus causal mechanisms in generating socioeconomic disparities in health. The SEM analysis suggests that both phenomena are operating, though perhaps to different degrees at different points in the life course. The less educated and those in lower-status occupations continue to experience poorer health outcomes than their more educated peers. However, diminished adult SES is itself partly a function of previous insults to health that stunt socioeconomic attainment. Therefore, there is clear evidence that health and SES are deeply interconnected, with constant interaction and reciprocal feedback mechanisms over the life course.

Interestingly, once unobserved family characteristics are controlled for in the fixed-effects analysis, there is no longer a significant educational gradient in health. This would suggest that once the shared factors that lead the

children from one family to get more education than those from another are taken into account, those with more education do not necessarily experience better subsequent health. However, as early occupational standing continues to exert a significant impact on adult health, social causation mechanisms continue to be present.

The lack of an educational gradient in health in the fixed-effects results is at odds with substantial empirical research, so caution is clearly warranted. It is also important to note that the fixed-effects analysis is based on a relatively small sample of siblings. Constraints on sample size are especially important in sibling models, as there tends to be less variation among siblings than among random individuals. Though the majority of the observed variation in the analysis occurs within families, the fixed-effects estimates may be imprecise to the extent that the total observed variance is constrained by the size of the sample. Further research is needed to replicate this finding in other, larger data sets on siblings and with other health outcomes.

If this finding is found to be robust, then more research is needed to explore the precise mechanisms that may lead children from certain families to both acquire more education and experience better health. While childhood health is one mechanism that links adult health and SES, other early environmental factors and endowments, which are likely to be shared by siblings, may also be important. These include nutrition, exposure to infectious diseases, and toxic physical and social environments. For example, in incorporating childhood development into a life course model of health, Hertzman (1999) asserts that the conditions under which early cognitive, emotional, and psychosocial development occur may condition the central nervous system (see also Cynader and Frost 1999). Thus, an early life environment not conducive to healthy development may lead to cognitive and psychosocial developmental delays and poor psychosocial coping mechanisms, and therefore to higher lifetime levels of stress and subsequent poor health. All of these processes are also deeply embedded in the process of academic achievement and educational attainment.

This study also has important limitations. First, it is important to point out that this sample is still comparatively young (about 39 years old on average) and thus has not yet experienced the brunt of health decline associated with aging. This likely makes the results conservative, as a major portion of the deleterious impact of poor childhood health has yet to be experienced. Another limitation is that, with the exception of occupational attainment, the analysis only estimates the impact of early life health on SES at one point in adulthood. However, it is likely that childhood health shapes trajectories of SES as well. For example, through its effects on educational attainment, we would expect early life health to alter the level and shape of earnings and life cycle savings curves. More work is needed to examine the impact of childhood health on dynamic patterns of employment, earnings, and wealth over the long run.

A final limitation is that the validity of the most important variable—childhood health—has not been established conclusively. However, previous analysis shows that these reports are reliable over time. The analysis above explicitly accounts for the effect of measurement error and gives further indication of their validity. The results are also consistent with findings from prospective studies using the British cohorts (Wadsworth 1986; Case, Fertig, and Paxson 2005). More work is needed to conclusively establish the validity of retrospective childhood health reports. However, given the dearth of life course data available, we may have to rely on less-than-perfect measures. Another problem with using a retrospective health measure collected in adulthood is that only those who survive to answer the questionnaire are in the survey. Those who did not survive are more likely to be those who experienced poor childhood health. Thus, mortality selection has the likely effect of biasing my results downward, again making the estimates obtained here conservative.

This study highlights the need to move beyond the notion that health selection and social causation be seen as mutually exclusive causal explanations. Instead, it demonstrates the importance of conceptualizing and measuring health-SES relationships as resulting from interactive mechanisms over the life course embedded within larger processes of social stratification and health attainment.

APPENDIX. Descriptive Statistics and Correlation Matrix for Variables Used in the Analysis

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Mean	.10	.65	26.57	.05	.74	.47	38.70	.33	.61	.07	3.28	3.33	13.45	24.85	35.69	10.13	8.86	2.82
Standard deviation	.30	.48	24.78	.21	.44	.50	8.60	.47	.49	.26	.89	.83	2.10	24.07	27.23	1.05	4.45	.93
1. Father education missing	1.0																	
2. Father ≥ 12 years education	–.45***	1.0																
3. Father's occup. SES	–.10***	.36***	1.0															
4. Mother education missing	.48***	–.23***	–.07***	1.0														
5. Mother ≥ 12 years education	–.27***	.50***	.27***	–.36***	1.0													
6. Male	–.06**	.07***	.08***	.03	.09***	1.0												
7. Age	.00	–.19***	–.06***	.05**	–.13***	.05**	1.0											
8. Black	.29***	–.34***	–.30***	.16***	–.33***	–.19***	.00	1.0										
9. Married	–.12***	.08***	.07***	–.02	.10***	.12***	.17***	–.25***	1.0									
10. Low birth weight	.01	–.02	–.06***	.01	–.05**	–.08***	–.02	.06**	–.01	1.0								
11. Childhood health 1999	–.06***	.11***	.07***	–.06**	.11***	.08***	.03	–.12***	.09***	–.05**	1.0							
12. Childhood health 2001	–.05**	.09***	.08***	–.04*	.09***	.11***	.00	–.11***	.07***	–.06**	.48***	1.0						
13. Education (years)	–.20***	.31***	.36***	–.15***	.29***	.03	.12***	–.24***	.16***	–.05**	.12***	.12***	1.0					
14. Occup. SES 1st job	–.12***	.24***	.31***	–.09***	.20***	–.06***	.09***	–.17***	.13***	–.01	.10***	.09***	.55***	1.0				
15. Occup. SES current job	–.16***	.24***	.29***	–.13***	.19***	–.07***	.08***	–.21***	.14***	–.03	.11***	.11***	.58***	.50***	1.0			
16. Earnings (log $)	–.09***	.14***	.15***	–.06**	.12***	.22***	.10***	–.15***	.08***	–.00	.11***	.09***	.28***	.23***	.30***	1.0		
17. Wealth (log $)	–.17***	.11***	.11***	–.08***	.11***	.11***	.30***	–.26***	.34***	–.01	.12***	.10***	.24***	.18***	.18***	.23***	1.0	
18. Adult self-rated health (2001)	–.09***	.20***	.16***	–.05**	.19***	.09***	–.14***	–.22***	.10***	–.05*	.33***	.43***	.20****	.17***	.18***	.16***	.11***	1.0

Note: SES = socioeconomic status. * $p < .05$; ** $p < .01$; *** $p < .001$ (two-tailed tests)

NOTES

1. Within economics, there is more support for the spurious explanation. Victor Fuchs (1982) has suggested different preferences in time horizons as a possible common cause of both health and economic outcomes.
2. The results of the structural equation model (SEM) analysis are robust to this sample constraint. Using the entire sample of heads and wives does not change the substantive results.
3. Economists often object to the use of self-rated health in studies because it has been shown to bias the relationship between health and economic variables in retirement models (Bound 1991). The results of the analysis are robust to alternative measures of adult health, including work-limiting disability and the number of physician-diagnosed chronic conditions.

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