Health Selection in the Stratification Process

The Effect of Childhood Health on Educational Attainment, Labor Force Participation, and Earnings

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Abstract

This study investigates the role of health in the stratification process by examining the impact of childhood health on educational and occupational status attainment, labor force participation, and earnings using the Panel Study of Income Dynamics (PSID). It also tests whether childhood health acts as a mechanism through which SES is transferred across generations. The results show health in childhood to be an important determinant of educational attainment, occupational status (men only), and adult health. Through these mechanisms, childhood health exerts a significant impact on labor force participation and earnings. In contrast to a large number of studies, only a weak association is found between social background and childhood health as each was measured here. Reasons for this incongruence are discussed. The findings further highlight the need to move beyond the notion that health selection and social causation necessarily be seen as mutually exclusive causal explanations of socioeconomic differentials in health. It instead views the SES/health relationship as the result of a truly interactive process over the life course embedded within larger processes of social stratification and health attainment.

INTRODUCTION

This study begins to provide answers to questions that increasingly have become some of the most central theoretical and empirical problems posed by sociologists, demographers, economists, and health researchers. What role does one's health status in childhood play in determining the socioeconomic position one occupies in adulthood? In other words, does poor health early in life adversely affect the amount of schooling one is able to complete, the average socioeconomic position of one's occupation, or the market returns to labor one is able to command? Is childhood health a mechanism by which socioeconomic status is passed from one generation to the next? Does health selection help explain the persistent socioeconomic gradient in health?

These questions are deeply embedded in important scholarly discourses that have taken place within demography, sociology, economics, and epidemiology/population health; however, until recently, researchers from these areas have only engaged each other sporadically. At the same time, while each of these disciplines has added substantially to our understanding of the above questions, only through synthesizing what are often very complementary research traditions can we get a purchase on the questions at hand.

The Socioeconomic Gradient in Health & Mortality

Within the demographic and epidemiological literatures, one of the most consistent empirical findings over the last 50 years has been the strong relationship between SES measured along several dimensions and health outcomes such as mortality rates, disability, and morbidity. Those that are more educated, have higher incomes, work in more prestigious occupations, and possess more wealth have better health, lower

disability, and lower mortality rates than their lower SES counterparts. This social gradient in health has been found in infants, children, adults and the elderly.

Initially, research on the social gradient in health was principally concerned with determining that this association was valid and not the spurious result of differential reporting of ill health by social class (Kadushin 1966) or incongruence between numerator and nominator in mortality rates. In the 1960s, two comprehensive reviews of the scientific literature clarified that there indeed was a consistent inverse relationship between SES and mortality (Stockwell 1961; Antonovsky 1967). In their landmark study, Kitagawa and Hauser (1973) found that in the US those in lower socioeconomic groups had higher mortality than those with higher SES. This finding was consistent across measures of SES including income, occupation, and education, with each of these having independent effects on mortality (Kitagawa & Hauser 1973).

Historically inquiries into social inequalities in health and mortality occupied a less visible role in social science research than they do today for two reasons. First, an overall decline in mortality and concomitant rise in life expectancy occurred over the 20th century, particularly in the post-war period. Simultaneously, western industrialized countries experienced the development of more comprehensive and interventionist social welfare states. These often included public universal health care systems as in the UK or, in the case of the US, government-funded health systems targeted at the most vulnerable segments of the population, including the poor (Medicaid) and the elderly (Medicare). The implicit assumption was that with overall mortality declining and the development of more elaborate social safety nets to provide basic medical services for the elderly and indigent, social differentials in health would solve themselves.

In the 1980s, interest in socioeconomic inequalities in health increasingly became a central topic of concern among social and biomedical researchers alike. One of the primary impetuses for this renewed interest was the publication of the Black Report in Britain (Townsend and Davidson 1982). The Black Report challenged the notion that social disparities in health were declining. It also raised doubts about differential access to health services as the central determinant of social disparities in health. Subsequent research has attempted to track changes in health inequality over time and explicate the mechanisms that connect SES and health.

Preston and Taubman (1994) found that not only are there large SES differentials in mortality in most western countries, but that these differentials had increased rather than decreased during the 1960s and 1970s. Similarly, Preston and Elo (1995) confirmed the findings of Feldman et al. (1989) and Pappas et al. (1993), concluding that for males, educational differentials in adult mortality have likewise increased over the last thirty years. Likewise in the UK, Blaxter (1991) documents that although overall mortality declined in the fifty years since the founding of the National Health Service, social inequalities in health and mortality have persisted and in some cases have increased.

In addition to higher mortality, lower SES groups have also been found to have higher prevalence of major chronic disease and disability (Lerner 1975; Haan & Kaplan 1986; Haan, Kaplan, & Camacho 1987; Preston and Taubman 1994). Socioeconomic differentials have also been found in infant and child mortality rates (Nersesian 1988; Singh and Yu 1995), birth weight (Macfarlane and Mugford 1984), and the experience of serious illness in childhood (Wadsworth 1986).

Explanations of the Social Gradient in Health and Mortality

There is at least one potentially important problem with most previous studies of health inequality. They almost invariably begin with an *a priori* assumption of *social causation*. That is, lower SES is assumed to have detrimental causal effects on health. (Williams 1990; Ross & Wu 1995; Smith and Kington 1997). Mechanisms by which SES is thought to affect health include differential access to and utilization of health care, lower levels of social support, an external locus of control, and differences in health-related risk behaviors (smoking, sedentary lifestyle, etc.) (Williams 1990; Ross & Wu 1995). Mirowsky and Ross (2003) synthesize these mechanisms under the conceptual paradigm of "education as learned effectiveness." Under this conception, increased educational attainment improves health both by increasing the availability of resources that can be used for health-related investments and, more importantly, by increasing individual agency and self-efficacy, and enhancing general problem-solving capacity, which promotes a healthy lifestyle. However, social causation is only one of three principal explanations that have been put forward to explain the social gradient in health (Lundberg 1991).

Another category of explanations suggest several ways in which the relationship is *spurious*. For example, there may be some unobserved factor causally determinant of both SES and health creating a spurious relationship between the two. The relationship may also be the result of differential subjective interpretations of health by social class (Kadushin 1966). 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 et al. 1987; Samphier et al. 1988). A third explanation, *health selection,* reverses

the direction of causality between poor health and low SES in that it is poor health that leads to lowered SES (Lundberg 1991).

Health Selection

It is possible to differentiate between two variants of the health selection hypothesis (Lundberg 1991; Palloni and Milesi 2002). In type I, also known as "drift," those with poor health are selected into lower SES because of decreased labor force participation or by leaving paid work entirely, thereby decreasing wage income and inhibiting wealth accumulation (Townsend and Davidson 1982). Furthermore, health problems may necessitate the spending down of accumulated wealth. The type II variant argues that poor health—particularly during critical periods of childhood and adolescence—may limit an individual's initial accumulation of human capital and their subsequent ascent to higher positions of prestige, power, and wealth. Thus health selection may operate in two distinct ways—either by inspiring downward social mobility or by preventing the initial movement upward. Almost all of the empirical work to date has considered only the type I or "drift" variant of health selection with its focus on current and/or recent health. An assessment of type II selection necessitates a broader life course approach and the analysis of very rare data that assess health and SES at various points in the life course, including childhood.

It is important to point out that explanations based on social causation and selection are not necessarily mutually exclusive as they are often presented to be, but rather it is likely that there is a lifelong synergistic relationship between SES and health. While one period's health is determined directly by the previous period's health, there is also an indirect effect via the effect of earlier health on later income and wealth.

Therefore poor health leads to hindered educational attainment and skill formation, diminished labor market outcomes, lower earned income and wealth accumulation, and subsequently fewer resources to reinvest in the production of the next period's health as the cycle continues. In this way, there is a constant interaction between health and SES via both selection and social causation over the life course. Though this description has a decidedly "material" or "resource" tone, Mirowsky and Ross' (2003) conception of education as "learned effectiveness" or health-related productive capacity can easily be accommodated. The non-material mechanisms by which education shapes health would essentially run parallel to the material mechanisms outlined above.

A few previous studies have attempted to discern the impact of health selection on SES. Using data from the 1946 British cohort study, Wadsworth (1986) directly tests the effect of health selection on social mobility. Wadsworth found that boys who had experienced a serious illness during childhood were more likely to belong to a lower occupational class at the age of 26 than were boys who had not experienced a serious illness. This finding held regardless of the boy's social class of origin. No effect of childhood illness on adult occupational class was found for girls. Serious childhood illness, particularly in the preschool years, also led to significantly lowered odds of acquiring educational credentials for both boys and girls.

Other studies have found indirect evidence of health selection. For example, Illsley (1955; 1986) observed that women who were upwardly mobile based on their husbands' social class relative to that of their father tend to be positively selected (via the marriage market) in regard to health. Similarly, using height as an indicator of childhood health and living conditions, Power et al. (1986) found a distinct social gradient in height

and a lower percentage of short people among those that were upwardly mobile compared to those that were downwardly mobile.

The most comprehensive examination of health selection to date was carried out by Lundberg (1991). Using data from Sweden, Lundberg tested whether childhood living conditions and health in late adolescence and early adulthood (age 15-20) affect inter-and-intra generational class mobility, including exit from the labor market. The findings suggest that the experience of economic hardship in childhood significantly lowered the likelihood of ending up in a high social class in adulthood. In addition, illness in early adulthood was positively related to labor force departure before age 65. However, there were also no observed effects on inter-generational class mobility, and only weak effects on intra-generational mobility. An important limitation to Lundberg's study was that he actually only tested the type I variant of health selection. By observing health in early adulthood he likely missed any effects of selection that may have occurred prior to early adulthood, specifically via effects on educational attainment. A more complete test of health selection would measure health earlier in the life course. The present study attempts to do this by using a measure of childhood health that covers the period of birth to age 16. In addition, shifting the focus onto childhood health and how it shapes the process of status attainment necessitates the examination of how parents invest in the health and economic success of their children.

Stratification and the Status Attainment Model

Because it has helped to elucidate the causal mechanisms by which social background shapes adult SES, the status attainment approach to social stratification provides an excellent starting point for the examination of health selection over the life

course. Moving beyond earlier work focused on describing trends in social mobility over time, a group of researchers in the late 1960s began to explore the underlying causal processes that led individuals to differential points in the educational, occupational, and earnings hierarchies. The pioneering work by Blau and Duncan (1967) and by Bill Sewell, Arch Haller, and Bob Hauser and colleagues (1969a-b; 1970; 1975) created the basis of a causal model by which social background is believed to shape the level of educational attainment and ultimately occupational prestige and earnings of offspring. Using data from the 1962 CPS, Blau and Duncan outlined a recursive model with a primary emphasis on children's educational attainment as the principal mechanism by which parental education and occupational prestige influenced that of the next generation.

Employing unique data from a longitudinal 1/3 sample of the Wisconsin high school graduating class of 1957, Sewell and colleagues (1967; 1969; 1970; 1975) extended the Blau and Duncan model to include social-psychological indicators such as aspirations, peer influence, and ability as causal mediators. This work formed the basis of what is now know as the social-psychological or "Wisconsin" model of status attainment and has resulted in hundreds of subsequent articles on status attainment (Campbell 1983). The status attainment model of social stratification provided great insight into the social processes and mechanisms that determine the placement of individuals in the social hierarchies. Both for its original contributions as well as the subsequent research agenda it fostered, the status attainment model is one of the most successful lines of sociological work of the last 50 years.

Although it contributed much to our understanding of the stratification process, the status attainment model is by no means complete. Absent entirely from almost all

work on social stratification is a discussion of the role of health either as a cause or consequence of the stratification process[1]. This is surprising given the vast and well established literatures on health inequality discussed above. One of the most important reasons for this is that stratification research almost invariably focuses on the portion of the life course between early adulthood (school completion) and midlife. Health has often been seen as the purview of those studying the aged, not those interested in young adults in the prime productive years.

Two areas of work in particular alert us as to why it is important to raise attention to the lack of discussion of health in sociological models of stratification. The first is labor and health economics, which have investigated the role played by health in such economic outcomes as labor force participation and the personal income distribution. The second is recent work that has raised the strong possibility that early life events and conditions can have lasting effects on adult health and socioeconomic outcomes. These are discussed in turn.

The Human Capital Model and Health Economics

From its inception as an explanation of the distribution of income, the human capital model focused the attention of researchers on the potentially important role of health in determining economic the outcomes of individuals and households (Mincer (1958; 1970; Schultz 1961; 1962; Becker 1962; 1964; and Mushkin 1962 see also Sahota 1978 for a comparison of economic theories of income distribution). The fundamental idea behind the human capital model in regard to the wage income distribution is that

[1] The nearly 1000-page volume edited by Grusky (2001), considered to be the definitive reader on social stratification, makes no mention of health either as a causal agent or an outcome within the stratification process.

wage income is seen to be primarily determined by the marginal productivity of labor and that certain investments in human beings improve their productivity and consequently yield economic returns. As with other forms of investment, individuals, parents, and societies make rational choice investments in humans based on the expected rate of return. Thus human capital refers to any investments in individuals such as education, skill formation, on-the-job experience, and health that increase productivity. Therefore differences in income are thought to largely reflect differential investments in human capital.

Although health has been seen as a component of human capital theory from its inception, its inclusion in actual empirical work has been much more limited in a number of important ways. 1) Human capital research has been more concerned with the market returns to education and job training than health (Fogel 1979). 2) To the extent that it is concerned with health, human capital research usually centers on the role of current health at the exclusion of other points in the life course (Luft 1975; Taubman & Wales 1974; Bartel & Taubman 1979; Lee 1982; Chirikos & Nestel 1985; Hanushek & Quigley 1985). 3) Poor health is often conceptualized narrowly as disability, particularly in how it acts as a determinant of labor force participation or otherwise limits the amount or type of work that can be performed (Scheffler & Iden 1974; Luft 1975; Chowdhury & Nickell 1985).

The human capital approach has yielded an analogous model of health. The health production function developed primarily by Grossman (1972), views health as a capital stock. Current health is a function of previous period's health stock, a genetic endowment, health-related behaviors, medical care, education, wages, and assets.

Therefore, while health economists have stressed the effect of SES on health and labor economists that of health on SES, little work has investigated how these may be simultaneously determined (see Lee 1982 for an interesting use of a simultaneous equations model of current health and wages). This study expands upon previous health econometric work by estimating a model of the effect of childhood health on adult health, non-health forms of human capital accumulation (occupational status, education), labor force participation, and current earnings.

SES: From Confounder to Casual Agent to Outcome

Over the last 250 years there has been a fundamental transformation in patterns of human disease and mortality. During this period, diseases of an infectious nature (e.g. smallpox and tuberculosis) were replaced by degenerative and chronic diseases (e.g. cancer and cardiovascular disease (CVD)) as the major causes of morbidity and mortality (Omran 1982; Vallin 1991). Unlike communicable diseases whose etiology could almost always be connected with a specific pathogenic vector, the etiology of chronic diseases is more complicated and multi-factorial. Although a few chronic diseases can be traced back to one or two pathogenic factors[2], most chronic diseases are the result of long-term exposure to a variety of risk factors.

Epidemiology has been successful in identifying some of the more salient risk factors associated with cancer and CVD, the leading causes of death in western industrialized countries. Most of these risk factors, which include smoking, excessive alcohol consumption, sedentary life style, high-fat/low fiber diet, and stress, can be categorized as lifestyle or behavioral factors. These risk factors are no doubt important

[2] For example, lung cancer is very rare among those who have never smoked or been exposed to asbestos.

determinants of chronic disease, yet they explain only about 20-30% of social class variation (Marmot et al 1978).

Because of persistent social differentials in health and the inability of known lifestyle risk factors to explain them, researchers have begun to investigate a more comprehensive model of health determination than the traditional focus on health behaviors and medical care. Evans, Barer, and Marmor (1994) have introduced a framework for analyzing the multiple determinants of health. The Evans model introduces some important features into the traditional biomedical model of disease. 1) It brings in the social and physical environment as well as the individual's genetic endowment into the model as proximate determinants. Traditionally, epidemiologists have viewed SES more as a confounding variable either outside of or hopelessly distal to the disease process. They acknowledged that SES may be an important upstream variable, but in practical application it was seen as something that clouded up the otherwise clear waters of causal association between etiologic agent and disease. In the Evans model, SES is seen as an important causal agent, although they continue to seek the more proximate physiological mechanisms by which it manifested its effects. 2) The Evans model also broadened the outcomes of interest to include functioning and overall well-being, not just the presence of specific disease sequelae. 3) In addition, the Evans model incorporates the economic concept of marginal cost. Resources are scarce and interventions to increase the well-being of populations must be judged by their marginal costs and benefits. Whereas health care investments have been the traditional instrument in attempts to increase health and well-being within populations, the marginal return of these must be compared with alternative investments such as education that may yield

higher marginal benefits. By implication this suggests a comprehensive re-examination of how health is achieved and points to the potentially important role played by such factors as education, child care, and early childhood health and social conditions.

It is not a coincidence then that an increasingly strong body of evidence has recently pointed to the role of early childhood and even prenatal health and social conditions in shaping both socioeconomic status and health later in life. This area of research has found that poor health early in life such as low birth weight and particular childhood diseases can have lasting effects on adult health (Elo 1998; Blackwell et al. 2001), cognitive development, and high school completion (Conley and Bennett 2000; Rich-Edwards et al. 1997; Sorensen et al. 1997).

In the concept of "biological embedding" Hertzman (1999a; 1999b) offers a generalized framework that incorporates early childhood development into a life course model of health. Biological embedding asserts that the conditions under which early cognitive, emotional, and psychosocial development occur shape and condition the central nervous system (Cynader & Frost 1999). An early childhood environment not conducive to healthy development may lead to developmental delays and poor psychosocial coping mechanisms and thus higher lifetime levels of stress and subsequently poor health. These processes are also all deeply imbedded in the process of academic achievement and educational attainment.

From the previous discussion several important empirical and theoretical regularities may be drawn, which form the basic justification for the current study. 1) There is a strong and persistent inverse relationship between SES and morbidity and mortality. 2) This relationship is likely the product of both social causation as well as

selection mechanisms particularly as they interact over the life course. 3) SES is transmitted across generations through various mechanisms. 4) There is increasing evidence of the potential importance of early life events and circumstances as determinants of both adult health and SES. 5) Health in childhood may be an important link between adult health and socioeconomic status. Therefore, embedding health inequalities within the stratification process as both a cause and a consequence of social inequality underscores the life course interconnection of health and SES.

Research Questions

This study employs an analytic approach similar to that of Lundberg (1991), although I focus on SES broadly rather than occupational class mobility specifically. The empirical questions are threefold: Is childhood health a mechanism of intergenerational transmission of socioeconomic position? What role does one's health status in childhood play in determining SES and health in adulthood? Does health selection help explain the persistent relationship between SES and health?

DATA

Data for this analysis come from the *Panel Study of Income Dynamics* (PSID). The PSID is a nationally representative longitudinal survey of households begun in 1968. The PSID was designed to investigate the effects of poverty and policies directed at its amelioration. The data detail the economic and demographic dynamics of households and include information on topics such as work, income, wealth, and housing. This analysis focuses on data derived from the 1999 and 2001 waves because the measure of childhood health was only gathered in these waves. I use a subset of the more than 50,000 individuals on which data have been collected, namely those that were heads and "wives" in the 2001

wave. Because the substantive focus is on labor force outcomes and earnings, I further limited the analysis to non-students and those aged 18-64. With the exception of a few variables discussed below, cases with missing data on either explanatory or outcome variables were deleted in a listwise fashion. These restrictions result in two separate subsamples used in the analysis.

In the analysis of the effect of childhood health on labor force participation, the first subsample of 8,187 respondents includes 4,465 women and 3,722 men. There are a larger number of women due presence of female-headed households in the sample. To analyze the effect of childhood health on inter-and intra-generational transmission of SES, the sample is further limited to those currently in the labor force. This sample of 6,527 includes 3,266 men and 3,261 women.

It is important to point out that labor force participation and earnings are modeled separately, so those not in the labor force are excluded in the earnings analysis. For this reason, the estimated effects of health on earnings are net of labor force participation. The indirect effects via labor force participation are likely the most important mechanism by which current health affects earnings.

METHODS

Measures

Childhood Health

The investigation of the effects of childhood health on later-life health, mortality, and SES has thus far been limited primarily by the lack of true life course data. Due to these data constraints, researchers must find alternative and indirect ways of assessing these effects. The method used in this analysis and by others is to use measures based on

retrospective reports (Elo 1998; Blackwell, Hayward, and Crimmins 2001). Asking adults to retrospectively report on their health in childhood may seem like a reasonable and relatively straightforward thing to do; however, these reports are potentially subject to large amounts of recall bias and measurement error. Unfortunately, to date, retrospective reports are only available in a very small number of surveys and fewer still collect the repeated measures needed to assess their reliability.

The measure of childhood health used in this study is based on response to the following question: "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 1(excellent) - 5 (poor) were then assigned to these categories. A similar question was asked in an experimental module in wave 3 of the Health and Retirement Study (HRS). Elo's (1998) analysis of these data found that responses had a high level of internal consistency between the report of general health and reports of specific long-term health limitations in childhood. While global measures like these are often dichotomized, because of the use of structural equation models I keep it in its original ordinal metric. To maximize sample size, the childhood health measure is based on the 2001 report except for a small number of cases that had missing data in 2001. These cases use the 1999 reports.

My analysis of retrospective reports of childhood health across the two waves suggests that the retrospective reports used here are fairly reliable over time, especially when the measure is dichotomized into a good/very good/excellent vs. fair/poor comparison (Haas 2003)[3]. For approximately 1/3 of cases, a maternal report of birth

[3] Across all groups the gamma measure of association was 0.597 for the 5-level variable. When dichotomized, the gamma increases to 0.904.

weight was available. The analysis shows that those who where born with low birth weight (less than 88 ounces) reported significantly worse health than those who were normal weight at birth (Haas 2003). Given the consistent relationship between birth weight and objective measures of childhood health (Brooks et al. 2001; Vohr et al.; Stevenson et al.), the fact that a similar relationship is found using retrospective reports lends support for their validity.

Adult Health

Adult health in the form of self-reported global health status measured on the same Likert scale with 1 (excellent) – 5 (poor) is also included. Current health was ascertained in both the 1999 and 2001 waves.

Labor Force Participation

Labor force participation is modeled as a binary outcome (1 = out of labor force) based on the response to the question: "We would like to know about what you do. Are you working now, looking for work, retired, keeping house, a student, or what?" Respondents were allowed to report up to three activities, and I use the first as the primary activity. With the exception of those who listed their primary activity as students and were consequently dropped from the analysis, all those not currently working for pay were coded as out of the labor force. In the analysis of earnings I also include the average weekly hours of work as an indicator of full-time versus part-time employment.

Education & Earnings

Educational attainment is measured as years of completed schooling. Earnings data were collected in 2001 about tax year 2000. Earnings are the sum of several labor

income components including not only wages and salaries, but also any separate reports of bonuses, overtime, tips, commissions, professional practice or trade, market gardening, and miscellaneous labor income. It excludes farm and unincorporated business income. This analysis uses a started log transformation (natural log ($Earnings_{2000}$ +1000)) of labor earnings. Labor earnings are used here for several important reasons. First, labor earnings are the primary component of total income. Second, unlike a broad measure of household income, labor earnings can be directly linked to an individual and thus provide greater purchase on the direct relationship between health and SES at the individual level. Third, household income is likely to be more resilient to health shocks because others in the household can increase their labor force participation to compensate for lost earnings of another. While this compensation is good in that it may ultimately insulate individuals and households, it is also likely to lead to an underestimate of the effects of health shocks on economic resources.

Occupational Status

The analysis includes the occupational status of the respondent's first full-time and current occupation. Occupational status is measured in terms of occupational education, which is defined as the percentage of incumbents within a specific occupation and industry with at least one year of college education (Nakao & Treas 1994; Hauser & Warren 1997). Occupational education reflects the average socioeconomic status of occupations. In their analysis of the relationship between a composite index of occupational status (SEI) and occupational education and wage, Hauser and Warren (1997) demonstrate that occupational differentiation occurs primarily by differences in educational attainment across occupations and that the contribution of wage rates is

negligible. In the PSID, occupations are classified by 3-digit 1970 basis census occupational and industrial codes. Using these 1970 basis codes, I import values of occupational education by occupation and industry[4]. About 6% of respondents had missing data on first occupation. Mean imputation was used to assign values to these cases.

Likewise, father's occupational status is 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% of respondents have missing data on father's usual occupation. For these cases mean imputation was performed. Analyses that include occupational status also include a dummy variable to denote imputed cases.

Parental Education

In addition to father's occupational status, social background is assessed using father and mother's educational attainment. Parental education is measured as a set of 6 dummy categories each for mother and father. These categories include less than high school (0-11 years), high school graduates (12 years) (reference group), some post-secondary school (13-15 years), college graduates (16 years), post-graduate education (17+), and a missing category.

Socio-Demographic Controls

A standard set of socio-demographic controls are also used in the analysis. For labor force participation, age is measured using 5 dummy variables corresponding to the

[4] 1970 basis occupational education values were calculated by John Robert Warren in conjunction with others working on the Wisconsin Longitudinal Survey. While I differentiate occupations by industry I do not also differentiate by class of worker. The PSID does not allow for adequate mapping by occupation, industry and class of worker. This is not likely to bias estimates of the effects of occupational education much as the majority of variance occurs across occupations and industries rather than between classes of worker.

age groups 18-25, 26-35, 36-45 (reference), 46-55, and 55 and above. In the earnings models, age is measured both in years and a quadratic term. Current marital status (1=married, 0=not), and a set of race and ethnic dummy variables (non-Hispanic white=reference group; black=1, not=0; Latino=1, not=0; Asian=1, not=0; other=1, not=0) are also included. Descriptive statistics for all variables used in the analysis can be found in table 1. All analyses are conducted separately for men and women because the PSID is based on a household sample. If observations were pooled, the standard assumption of independence between observations would be violated. Because only one man (head) and one woman ("wife" or single "head") is analyzed for each household, separating the sample by gender would presumably create two samples within which observations are independent. However, because some household heads and wives are members of original 1968 households that have since spilt off to form new ones there is still clustering in the sex-specific samples as some of the heads and wives are the children and/or sibling of other heads and wives. Standard errors have been corrected for this clustering.

[Table 1 about here]

Analytic Models

Two different statistical models are employed in this analysis. The first models the probability of labor force participation using a binary logistic regression model estimated by the following equation-

$$\ln\left(\frac{p}{1-p}\right) = \beta_0 + \sum_{j=1}^{k} \beta_j X_j \qquad (1)$$

where p is the probability of being out of the labor force and β_j are estimates of the effects of X_j, a series of k covariates.

The effects of childhood health on status attainment and earnings are analyzed within a set of nested recursive structural equation models estimated within the Lisrel framework (Jöreskog and Sörbom 2003). The exogenous effects (**Γ**) and between endogenous outcomes (**B**) are estimated by the following structural equation of the model-

$$\boldsymbol{\eta} = \mathbf{B}\boldsymbol{\eta} + \boldsymbol{\Gamma}\xi + \zeta \qquad (2)$$

where $\boldsymbol{\eta}$ is a 9 x 1 vector of endogenous outcome variables, ξ is a 19 x 1 vector of exogenous predictor variables, and ζ is a 9 x 1 vector of the random disturbances in $\boldsymbol{\eta}$. Because there are no latent or unobserved variables, the measurement models for **Y** and **X** are-

$$\mathbf{Y} = \boldsymbol{\Lambda}_y \boldsymbol{\eta} + \boldsymbol{\varepsilon} \qquad (3)$$

$$\mathbf{X} = \boldsymbol{\Lambda}_x \xi + \boldsymbol{\delta} \qquad (4)$$

Where $\boldsymbol{\Lambda}_y$ and $\boldsymbol{\Lambda}_x$ are the identity matrix and $\boldsymbol{\varepsilon}$ and $\boldsymbol{\delta}$ are zero matrices.

The causal structure of the endogenous variables in model 1, which serves as a baseline for the earnings analysis, is presented in figure 1. In this model, childhood health is allowed to have direct effects on educational attainment, occupational status of both first and current job, and subsequent adult health. There are no direct effects of childhood

health on earnings or average weekly work hours. Furthermore, there are no effects (direct or indirect) of earnings in 2000 on health in 2001.

Different constraints are imposed on this baseline model to test various hypotheses about the social processes involved. To test the hypothesis that childhood health is not determined by social background, the effects of parental education and father's occupational status on childhood health are fixed to zero in model 2. This test is important because if childhood health is not strongly related to social background, then it is not likely to be an important mechanism of intergenerational SES transmission. Models 3-5 impose null constraints on the effects of childhood health on early occupational status, educational attainment, and current occupational status, respectively. These models test whether childhood health affects intragenerational social mobility.

In models 6 and 7, the direct effect of childhood health first on health in 1999 and then in 2001 are constrained testing the long-term health impact of health in childhood. Model 8 allows a free effect of earnings in 2000 on subsequent health in 2001. The addition of this parameter examines whether the observed relationship between current health and earnings is due to their common causal factors (namely previous human capital accumulation). Model 9 tests the direct effect of childhood health on current earnings net of the indirect effects via education, occupational status, and adult health by freeing this parameter. Finally, based on the results of the previous models, I present the parameter estimates from the best-fit model for men (10a) and women (10b).

RESULTS

Childhood Health and Labor Force Participation

Tables 2 and 3 present the parameter estimates (odds ratios) from logistic regression models of labor force participation. In the bivariate model, childhood health has significant yet moderate effects on labor force participation. Those who experienced worse health in childhood were significantly more likely to be out of the labor force than those who experienced healthy childhoods. A one unit increase in childhood health (higher values denoting worse health) is associated with a 31% and 24% increase in the odds of being out of the labor force for men and women respectively.

[Table 2 about here]

[Table 3 about here]

Whereas in the bivariate model poor childhood health appears to have significant negative effects on labor force participation, as would be expected subsequent analysis shows these effects to be indirect, working primarily through educational attainment and current health. The inclusion of socio-demographic and social background variables attenuates the effect of childhood health somewhat, yet it remains statistically significant. Similar attenuation occurs when controls for educational attainment and early occupational status are added to the model. When an indicator of more current health is added to the model, childhood health no longer has a significant effect on labor force participation.

Both educational attainment and current health, and to a lesser degree early occupational status, appear to act as the primary mechanisms by which health in childhood influences adult labor force participation. Educational attainment accounts for about 30-40% of the effect of childhood health on labor force participation with current health accounting for the rest. As a result, those who experienced poor health in

childhood have substantially lower levels of educational attainment and poorer current health, which in turn increases the likelihood of premature exit from the labor force.

In addition to the effect of poor current health, this analysis also confirms the effect of age on labor force departure. In general, labor force participation increases monotonically with age, though in the full model only those 55 years and older are different from the 46-55 year old reference group to a statistically significant degree. These men and women between the ages of 55 and 64 have 5.8 and 2.8 greater odds of being out of labor force respectively. Married men and non-married women are also more likely to still be working. Black men have 1.5 times greater odds of being out of the labor force than white men. For women there are no observed differences in labor force participation by race and ethnicity, although in a less inclusive model Latino women appear to be more likely to be out of the labor force. These differences disappear when education is added to the model. Also, even though the general pattern of the effects of childhood health on labor force participation is invariant by gender, health—both current and in childhood—appears to be more strongly associated with labor force participation for men than for women. As would be expected, those whose early occupational status was imputed due to missing data on first full-time occupation had significantly higher odds of being currently out of the labor force. It would seem to confirm that those with missing data are not a random selection of respondents; instead, they likely include those that have either never worked for pay or have generally less stable attachments to the labor force. Other than those with imputed values, there is no significant effect of early occupational status on labor force participation.

Childhood Health, Social Background, and Status Attainment

Model Comparisons

The evidence as to whether health in childhood acts as a mechanism of intergenerational transmission of SES is mixed. The results suggest a cautious affirmative. As seen in table 4, constraining the effects of all social background variables on childhood health (model 2) results in a significant decline in model fit. However, this is not the case when father's occupational status, education, and mother's education are constrained separately. With the exception of maternal education among women, none of these constraints leads to a significant decline in model fit when imposed separately (not shown). Reasons for this apparent weak association with social background on childhood health are presented in the discussion.

[Table 4 about here]

Models 3-5 test the effect of childhood health on early occupational status, educational attainment, and current occupational status respectively. The results show relatively large direct effects of childhood health on educational attainment. Fixing the effect on education to zero (model 4) causes the fit of the model to deteriorate substantially (L^2 increases by 36.46 for men and 29.59 for women with one additional degree of freedom). The effects on occupational status are less clear. The results of models 3 and 5 suggest that there are no direct effects of childhood health on early occupational status and only effects on current occupational status among men. This is not to say that poor health in early life does not have significant impacts on occupational attainment; rather, these effects act indirectly, primarily through lowered educational attainment.

The effects of health in childhood on health in later life are tested in model 6 (health in 1999) and model 7 (health in 2001). Confirming the findings of Elo (1998) and Blackwell et al. (2000), childhood health is strongly and directly related to later measures of health. The imposition of null constraints on the effect of childhood health on adult health has massively negative effects on the fit of the model. With only one additional degree of freedom, the fit of model 6 more than triples and the ratio of L^2 to degrees of freedom increases from about 1.6 to over 5. Similarly, in model 7, L^2 and L^2/degree of freedom nearly quadruples.

In looking at the results from model 8, the observed relationship between earnings in 2000 and health in 2001 differs for men and women. For men, the lack of a significant change in the overall fit of the model suggests that this relationship is essentially due to their shared upstream causal determinants such as educational attainment and the previous period's health. For women, freeing the effect of earnings on health significantly improves the fit of the model. L^2 increases by 14.34 at the cost of one degree of freedom. Therefore, for women, shared structural determinants do not completely explain the relationship between earnings and health in the following year. While men and women differ in this regard, they are in concordance with respect to the direct effect of childhood health on adult earnings. The freeing of this effect yields a statistically trivial improvement in model fit for both men and women. Although childhood health may have lasting effects on earnings, these are completely accounted for by the indirect effects in the model.

In models 10a and 10b, I present the best fit model for men and women respectively. The best fit model for men ends up being identical to model 3, which

constrains the effect of childhood health on early occupational status to zero. For women, the best fit model starts with model 3 but imposes an additional null constraint on the effect of childhood health on current occupational status. It also frees the effect of earnings on health in 2001 as suggested by model 8.

Childhood Health as a Mechanism of Intergenerational SES Transmission

Tables 5-8 present the maximum likelihood estimates from the best fit model for men and women respectively. As was the case in the model comparison, an examination of the parameter estimates also leads to a conclusion that social background appears to be only a weak determinant of childhood health as they are both measured here. Only 2-3% of the variance in childhood health is explained by the model. There is no statistically significant effect of social background on childhood health for men, although the observed effects are generally all in the expected direction. For women, while there is not a significant effect of father's occupational status, there are significant differences in childhood health by parental education. Women whose mother or father had less than a high school diploma had significantly worse health in childhood than those whose parents completed high school. Similarly, women whose mother's had post-baccalaureate education had significantly better childhood health in relation to the daughters of high school graduates. These results, particularly for men, contradict a large volume of literature that has consistently found a strong relationship between social background and child health outcomes.

[Tables 5-8 about here]

Childhood Health and Status Attainment

While the results provide only modest support for childhood health as a mechanism of intergenerational social mobility, they provide rather strong evidence for its role in intragenerational status attainment and thus for health selection as an important component in explanations of health inequalities. For both men and women, childhood health has significant and relatively large effects on educational attainment. Being in poor rather than excellent health in childhood is associated with 1.38 fewer years of education for men and 1.08 fewer years of education for women. The magnitude of the difference between excellent and poor health is roughly equivalent to having two college educated parents. For men, childhood health also has significant yet much more modest effects on current occupational status. Based on the results of model 5, these effects were not estimated for women.

The Effect of Childhood Health on Later Adult Health

As with educational attainment, there is also a strong relationship between childhood health and both instances of adult health. Those that were healthy in childhood tend to be healthy in adulthood, whereas those in poor health in childhood also experienced poor health in adulthood. Echoing the findings of Barker (1994), Blackwell et al. (2000), and Elo (1998) these results suggest that health in childhood is critically important to health over the life course. In addition, these direct effects are only a lower bound estimate for several reasons. First, they don't include the indirect effects on adult health via lowered lifetime SES. Lowered educational attainment exerts additional downward effect on adult health. Second, these are estimates of the effect of childhood health only among those still in the labor force. Those who have left the labor force, often as a result of disability or otherwise poor health, are not included in this analysis.

Childhood Health and Earnings

Although the direct effect on earnings was constrained to zero, childhood health continues to be a determinant of adult labor earnings. These effects work primarily through the process of human capital formation. Poor childhood health has a detrimental impact on adult health, the ability to acquire additional schooling, and, for men, the ability to occupy higher status occupational niches. This diminished human capital accumulation in turn has a negative impact on the returns to labor. Similar to the effect on adult health, the effects estimated here 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 impact adult economic outcomes (particularly earnings) is as a determinant of adult health, and subsequently, labor force participation.

DISCUSSION

While a few previous studies of social inequalities in health have found evidence of health selection (Wadsworth 1986), others have found only indirect (Illsley 1955;1986; Power et al. 1986), or little evidence at all (Lundberg 1991). In contrast to the latter, this study presents relatively strong and direct evidence that health selection does occur particularly in the case of educational attainment. Those who experienced poor health in childhood complete significantly less education than their healthy peers. They also experience substantially worse health over the life course. As a result they are more likely to leave the labor market prematurely, occupy lower status occupational niches (men only), and have diminished economic returns in the form of labor earnings.

Disentangling the specific mechanisms by which childhood health impacts 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 their social activities restricted relative to their healthy peers. In addition to impaired social functioning, chronically ill children may have altered developmental trajectories. Illness may be associated with increased periodic and/or prolonged school absences. Consequently, their cognitive development may also be impaired. Wadsworth (1986) observed just such an effect, finding that children who had been seriously ill under age 10 scored significantly lower on achievement tests at age 15. Similarly, Douglas et al. (1968) provides indirect evidence of child health on school achievement. Using height as a proxy for childhood health and nutrition, they found that those children who were high academic achievers also tended to be taller than non-high achievers. Another possible explanation 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.

In contrast to a large number of studies linking socioeconomic characteristics of parents and families to health outcomes in children (Macfarlane and Mugford 1984; Wadsworth 1986; Nersesian 1988; Singh and Yu 1995 among many), this study finds only a weak association between social background and childhood health for women and none among men. This would suggest a limited role for childhood health as a mechanism through which parents pass on their SES to their children. Because of the strong evidence of the social determinants of childhood health, I am skeptical of this result. Three explanations for this weak association readily come to mind.

The first is that measures of social background tend to be highly intercorrelated with each other. Because of this high intercorrelation when the effect of one is constrained, its covariance with childhood health can be still be transmitted via alternate paths leading from the others. When all of these effects are constrained, this shifting is no longer possible and the model cannot reproduce the observed covariance structures leading to a significant decline in model fit. This would seem to explain why the model has a significantly poorer fit when all of the effects of social background are constrained, but imposing these constraints separately has negligible effects. If this were the case, then we would expect the parameter estimates to ultimately reflect the "true" important effect of social background. However, while the observed effects of social background on childhood health are all in the expected direction, only a few are statistically significant.

A second explanation concerns the period of reference for the retrospective reports themselves. Socioeconomic differentials in childhood health tend to be strongest during the prenatal and neonatal periods and in infancy (Aber et al. 1997; Singh and Yu 1996) and weakest among adolescents (West et al. 1990; Ford et al. 1994). It is generally assumed that very early childhood is a period of life in which individuals have poor if any memory. In addition, recall ability is higher for events in the near term relative to those in the long term (Ebbinghaus 1964). Asking respondents to recall their health in general from birth to age 16 naturally requires them to average their health experience over their entire childhood. It may be likely that in making this mental average, respondents give relatively less weight to periods of childhood with the highest level of health inequality (early childhood) and more to those with the lowest (adolescence). This would have the effect of decreasing the observed effect of social background on childhood health.

Lastly, the particular indicators of social background used here may not adequately capture the real effects of social background. For example, permanent income or poverty status during childhood is likely to be more strongly and directly related to childhood health outcomes than are father's occupational status and parental education. There is likely some truth in this assessment, even though maternal education is consistently a strong predictor of child health (see for example Sastry 1996; HHS 2003). Future work will more adequately account for permanent income, poverty status, and income shocks.

Another limitation of this study is that for its most important variable it relies on an un-validated retrospective measure of childhood health. Although the analysis of Haas (2003) shows that these reports tend to be fairly reliable, and there is some indication of their validity, further work is needed to better determine how accurately these retrospective reports correspond to objective measures of childhood health. However, because of the severely limited life course data available on health and SES, researchers are forced to find alternative methods of getting at these relationships. As is often the case, if work is to progress, we may have to rely on less-than-perfect measures.

This study shows that the role of health selection is clearly not-trivial. However, I do not assert that health selection is the only or even the most important explanation of the social gradient in health. On the contrary, this study highlights the need to move beyond the notion that health selection and social causation necessarily be seen as mutually exclusive causal explanations. Instead, it demonstrates the importance of conceptualizing and measuring the health/SES relationship as the result of a truly

interactive process over the life course embedded within larger processes of social stratification and health attainment.

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Table 1 Descriptive Statistics

	Men		Women	
Variable	Mean	S.D.	Mean	S.D.
Father's SES				
ED Missing	0.161	0.402	0.160	0.386
ED 0-11	0.257	0.473	0.283	0.508
ED 12	0.352	0.478	0.334	0.472
ED 13-15	0.080	0.296	0.081	0.303
ED 16	0.091	0.366	0.088	0.331
ED 17+	0.059	0.288	0.054	0.267
Occ SES	26.66	32.05	25.31	29.59
Occ SES Imputed	0.130	0.360	0.133	0.353
Mother's SES				
ED Missing	0.113	0.333	0.093	0.289
ED 0-11	0.224	0.461	0.279	0.532
ED 12	0.459	0.498	0.424	0.494
ED 13-15	0.094	0.324	0.103	0.354
ED 16	0.077	0.326	0.073	0.302
ED 17+	0.033	0.207	0.029	0.185
Demographic				
White	0.678	0.467	0.602	0.490
Black	0.229	0.656	0.302	0.761
Asian	0.016	0.134	0.016	0.125
Latino	0.055	0.260	0.049	0.212
Other	0.021	0.152	0.031	0.178
Age (years)	42.43	9.16	41.22	9.32
Age2	1903.00	757.49	1804.55	760.70
18-25	0.045	0.208	0.060	0.238
26-35	0.225	0.418	0.243	0.429
36-45	0.329	0.470	0.347	0.476
46-55	0.293	0.455	0.256	0.437
55+	0.107	0.310	0.093	0.291
SES				
Education (years)	13.10	3.33	12.99	2.88
Occ SES 1st Job	22.73	28.98	26.55	27.45
Occ SES 1st Job Imputed	0.073	0.266	0.097	0.267
Occ SES Current	33.00	34.32	36.82	31.89
Out of Labor Force	0.123	0.328	0.270	0.444
Earnings 2000 (Log $)*	10.40	1.18	9.96	1.03
Health				
Childhood Health	1.68	0.847	1.79	0.873
Health 1999	2.16	0.986	2.34	1.04
Health 2001	2.24	1.06	2.40	1.05
N	3722		4465	

* Among those in the Labor Market

Table 2 Logistic Regression (Odds-Ratios) of Labor Force Participation-Men

	1		2		3		4		5		6	
Health												
Childhood Health	1.312	***	1.236	***	1.282	***	1.223	***	1.014		1.005	
Current Health	_		_		_		_		2.165	***	1.865	***
Parent's SES												
Dad's Occ. Edu	_		1.001		_		_		_		1.004	
Dad's Occ Imputed	_		0.982		_		_		_		0.873	
Dad HS Grad (Ref)	_		1.000		_		_		_		1.000	
Dad Ed Missing	_		1.205		_		_		_		0.897	
Dad < 12	_		1.039		_		_		_		0.922	
Dad 13-15	_		0.862		_		_		_		0.813	
Dad 16	_		0.747		_		_		_		0.777	
Dad 17+	_		0.554		_		_		_		0.529	
Mom HS Grad (Ref)	_		1.000		_		_		_		1.000	
Mom Ed Missing	_		1.127		_		_		_		1.019	
Mom < 12	_		1.437	*	_		_		_		1.323	
Mom 13-15	_		0.679		_		_		_		0.665	
Mom 16	_		0.846		_		_		_		0.902	
Mom 17+	_		1.698		_		_		_		1.992	
Demographic												
Age												
18-25	_		0.715		_		_		_		0.728	
26-35	_		0.819		_		_		_		0.873	
36-45 (Ref)	_		1.000		_		_		_		1.000	
46-55	_		1.501	**	_		_		_		1.317	
55+	_		6.579	***	_		_		_		5.794	***
White (Ref)	_		1.000		_		_		_		1.000	
Black	_		1.667	***	_		_		_		1.504	**
Asian	_		0.508		_		_		_		0.612	
Latino	_		1.023		_		_		_		0.587	
Other	_		0.534		_		_		_		0.555	
Married	_		0.350	***	_		_		_		0.381	***
SES												
Education (Years)	_		_		_		0.886	***	_		0.926	**
1st Job Occ Edu	_		_		0.993	**	_		_		1.002	
1st Job Imputed					1.407						1.559	**
-2 Log Likelihood	2744.77		2410.89		2732.39		2694.74		2514.55		2258.94	
df	1		22		3		2		2		26	
N	3722		3722		3722		3722		3722		3722	

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

Table 3 Logistic Regression (Odds-Ratios) of Labor Force Participation-Women

	1		2		3		4		5		6	
Health												
Childhood Health	1.236	***	1.193	***	1.207	***	1.146	***	1.078		1.049	
Current Health	_		_		_		_		1.462	***	1.350	***
Parent's SES												
Dad's Occ. Edu	_		1.000		_		_		_		1.002	
Dad's Occ Imputed	_		0.961		_		_		_		0.870	
Dad HS Grad (Ref)	_		1.000		_		_		_		1.000	
Dad Ed Missing	_		1.576	***	_		_		_		1.247	
Dad < 12	_		1.178		_		_		_		1.058	
Dad 13-15	_		1.189		_		_		_		1.266	
Dad 16	_		1.324		_		_		_		1.466	*
Dad 17+	_		1.129		_		_		_		1.316	
Mom HS Grad (Ref)	_		1.000		_		_		_		1.000	
Mom Ed Missing	_		1.274		_		_		_		0.987	
Mom < 12	_		1.190		_		_		_		1.040	
Mom 13-15	_		0.843		_		_		_		0.909	
Mom 16	_		0.584	**	_		_		_		0.615	**
Mom 17+	_		0.752		_		_		_		0.816	
Demographic												
Age												
18-25	_		1.277		_		_		_		1.087	
26-35	_		1.130		_		_		_		1.166	
36-45 (Ref)	_		1.000		_		_		_		1.000	
46-55	_		1.027		_		_		_		0.983	
55+	_		3.035	***	_		_		_		2.777	***
White (Ref)	_		1.000		_		_		_		1.000	
Black	_		1.073		_		_		_		1.044	
Asian	_		1.078		_		_		_		0.792	
Latino	_		1.820	***	_		_		_		1.094	
Other	_		0.879		_		_		_		0.805	
Married			1.365	***							1.452	***
SES												
Education (Years)	_		_		_		0.851	***	_		0.876	***
1st Job Occ Edu	_		_		0.995	**	_		_		1.003	
1st Job Imputed	_		_		2.240	***	_		_		2.088	***
-2 Log Likelihood	5172.86		4967.82		5103.98		5026.97		5057.41		4792.27	
df	1		22		3		2		2		26	
N	4465		4465		4465		4465		4465		4465	

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

Table 4 Model Comparisons

			Men			Women		
	Model	*df*	L^2	L^2 / *df*	*BIC*	L^2	L^2 / *df*	*BIC*
1	Baseline	71	113.65	1.60	-460.83	115.59	1.63	-458.79
2	1- Parent's SES on Child Health	83	145.27	1.75	-526.31	158.65	1.91	-512.80
	2 vs 1	12	31.62**		-65.48	43.06***		-54.01
3	1- Child Health on Early OccSES	72	116.18	1.61	-466.40	115.68	1.61	-466.78
	3 vs 1	1	2.53		-5.57	0.09		-7.99
4	1- Child Health on Education	72	150.11	2.08	-432.47	145.18	2.02	-437.28
	4 vs 1	1	36.46***		28.36	29.59***		21.51
5	1- Child Health on Current OccSES	72	120.78	1.68	-461.80	118.71	1.65	-463.75
	5 vs 1	1	7.13**		-0.97	3.12		-4.96
6	1- Child Health on $Health_{1999}$	72	367.49	5.10	-215.09	400.99	5.57	-181.47
	6 vs 1	1	253.84***		245.74	285.40***		277.32
7	1- Child Health on $Health_{2001}$	72	449.77	6.25	-132.81	440.61	6.12	-141.85
	7 vs 1	1	336.12***		328.02	325.02***		316.94
8	1+ Earnings on $Health_{2001}$	70	113.40	1.62	-452.99	101.25	1.45	-465.04
	8 vs 1	1	-0.25		7.85	-14.34***		-6.25
9	1+ Childhood Health on Earnings	70	112.98	1.61	-453.41	114.49	1.64	-451.80
		1	-0.67		7.42	-1.10		6.99
10a	Final Model-Men	72	116.18	1.61	-466.40			
		1	2.53		-5.57			
10b	Final Model-Women	72				104.47	1.45	-477.99
		1				-11.12***		-19.20
	N			3266			3261	

* $p<.05$, ** $p<.01$, *** $p<.001$ *BIC= L2- (ln(N) * df)*

Table 5 Beta Coefficiants for Final Model-Men

	Outcome								
	Father's OccSES	Childhood Health	OccSES 1st Job	Educational Attainment	Health 1999	OccSES Current Job	Average Work Hrs	Log Earnings	Health 2001
Father's OccSES	_	-0.008 (0.006)	0.102 *** (0.019)	0.136 *** (0.021)	_	0.102 *** (0.016)	_	_	_
Childhood Health	_	_	_	-0.344 *** (0.057)	0.305 *** (0.019)	-0.153 ** (0.057)	_	_	0.328 *** (0.017)
OccSES 1st Job	_	_	_	_	-0.011 (0.006)	0.337 *** (0.019)	_	0.025 ** (0.008)	-0.017 ** (0.006)
Educational Attainment	_	_	0.362 *** (0.016)	_	-0.059 *** (0.006)	0.397 *** (0.018)	_	0.032 *** (0.008)	-0.027 *** (0.005)
Health 1999	_	_	_	_	_	-0.046 (0.051)	-0.276 (0.210)	-0.070 *** (0.021)	0.480 *** (0.016)
OccSES Current Job	_	_	_	_	_	_	0.246 *** (0.071)	0.061 *** (0.008)	_
Average Work Hrs	_	_	_	_	_	_	_	0.010 *** (0.002)	_
Log Earnings	_	_	_	_	_	_	_	_	_
Health 2001	_	_	_	_	_	_	_	_	_
R^2	.49	.02	.28	.34	.18	.42	.02	.12	.42

** p<.05, ** p<.01, ***p<.001*

Table 6 Gamma Coefficents For Final Model-Men

	Father's OccSES		Childhood Health		OccSES 1st Job		Educational Attainment		Health 1999	
Father's Education										
Missing	-0.416	**	0.094		-0.084		-1.163	***	_	
	(0.145)		(0.053)		(0.157)		(0.172)			
0-11 Years	-0.801	***	0.070		-0.220		-0.457	***	_	
	(0.108)		(0.040)		(0.117)		(0.129)			
13-15 Years	1.255	***	-0.170		0.343	*	0.766	***	_	
	(0.149)		(0.055)		(0.162)		(0.179)			
16 Years	3.217	***	-0.030		0.464	**	0.781	***	_	
	(0.126)		(0.051)		(0.149)		(0.164)			
17 + Years	5.569	***	-0.001		0.280		0.941	***	_	
	(0.169)		(0.071)		(0.210)		(0.231)			
Mother's Education										
Missing	-0.028		0.061		0.264		-1.255	***	_	
	(0.163)		(0.060)		(0.178)		(0.194)			
0-11 Years	-0.001		0.032		-0.007		-0.410	**	_	
	(0.106)		(0.039)		(0.115)		(0.126)			
13-15 Years	0.565	***	-0.015		0.219		0.404	**	_	
	(0.136)		(0.050)		(0.147)		(0.162)			
16 Years	0.499	***	-0.050		-0.049		0.643	***	_	
	(0.141)		(0.052)		(0.152)		(0.168)			
17 + Years	0.550	*	-0.072		0.765	**	0.925	***	_	
	(0.218)		(0.080)		(0.235)		(0.259)			
Father's OccSES imputed	0.978	***	0.036		0.083		0.115		_	
	(0.124)		(0.046)		(0.136)		(0.149)			
R's Early OccSES Imputed	_		_		0.356	*	_		0.073	
					(0.171)				(0.060)	
Demographic										
Age	0.102	**	0.003		-0.032		0.130	**	0.024	
	(0.034)		(0.013)		(0.037)		(0.041)		(0.013)	
Age^2	-0.107	**	-0.003		0.069		-0.102	*	-0.004	
	(0.041)		(0.015)		(0.045)		(0.049)		(0.016)	
Black	-0.412	***	0.058	*	-0.121		-0.092		0.100	***
	(0.067)		(0.025)		(0.073)		(0.081)		(0.025)	
Latino	-0.043		0.146	*	1.093	***	-3.306	***	0.077	
	(0.167)		(0.061)		(0.187)		(0.199)		(0.066)	
Asian	0.847	**	0.207		1.899	***	1.31	***	0.037	
	(0.304)		(0.112)		(0.328)		(0.362)		(0.119)	
Other	0.176		0.160		0.056		-0.495		-0.094	
	(0.270)		(0.099)		(0.291)		(0.321)		(0.104)	
Married	_		_		0.228	*	0.363	**	-0.100	**
					(0.103)		(0.114)		(0.037)	

** p<.05, ** p<.01, ***p<.001*

Table 6 Cont. Gamma Coefficents For Final Model-Men

	OccSES Current Job		Average Work Hrs		Log Earnings		Health 2001	
Father's Education								
Missing	–		–		–		–	
0-11 Years	–		–		–		–	
13-15 Years	–		–		–		–	
16 Years	–		–		–		–	
17 + Years	–		–		–		–	
Mother's Education								
Missing	–		–		–		–	
0-11 Years	–		–		–		–	
13-15 Years	–		–		–		–	
16 Years	–		–		–		–	
17 + Years	–		–		–		–	
Father's OccSES imputed	-0.146		–		–		–	
	(0.136)							
R's Early OccSES Imputed	0.044		–		-0.103		0.061	
	(0.179)				(0.074)		(0.053)	
Demographic								
Age	0.021		0.374	*	0.080	***	0.018	
	(0.039)		(0.168)		(0.017)		(0.012)	
Age^2	-0.002		-0.473	*	-0.097	***	-0.007	
	(0.047)		(0.203)		(0.020)		(0.014)	
Black	-0.499	***	-1.316	***	-0.012		0.053	*
	(0.077)		(0.318)		(0.031)		(0.022)	
Latino	0.407	*	0.361		-0.159		0.009	
	(0.194)		(0.783)		(0.082)		(0.060)	
Asian	-0.159		-0.232		0.256		0.187	
	(0.348)		(1.490)		(0.147)		(0.106)	
Other	0.187		2.073		-0.003		-0.199	*
	(0.306)		(1.308)		(0.129)		(0.093)	
Married	0.288	**	1.689	***	0.135	**	-0.029	
	(0.110)		(0.473)		(0.047)		(0.034)	

** p<.05, ** p<.01, ***p<.001*

Table 7 Beta Coefficiants for Final Model-Women

	Outcome								
	Father's OccSES	Childhood Health	OccSES 1st Job	Educational Attainment	Health 1999	OccSES Current Job	Average Work Hrs	Log Earnings	Health 2001
Father's OccSES	_	-0.005 (0.007)	0.060 **	0.126 ***	_	0.052 **	_	_	_
Childhood Health	_	_	_	-0.269 ***	0.329 ***	_	_	_	0.310 *** (0.017)
OccSES 1st Job	_	_	_	_	-0.022 ** (0.007)	0.274 *** (0.019)	_	0.015 * (0.006)	-0.020 *** (0.006)
Educational Attainment	_	_	0.428 *** (0.017)	_	-0.056 *** (0.007)	0.422 *** (0.020)	_	0.047 *** (0.006)	-0.008 (0.006)
Health 1999	_	_	_	_	_	-0.207 *** (0.046)	-0.273 (0.218)	-0.054 *** (0.014)	0.450 *** (0.015)
OccSES Current Job	_	_	_	_	_	_	0.520 *** (0.071)	0.062 *** (0.006)	_
Average Work Hrs	_	_	_	_	_	_	_	0.040 *** (0.001)	_
Log Earnings	_	_	_	_	_	_	_	_	-0.055 *** (0.015)
Health 2001	_	_	_	_	_	_	_	_	_
R^2	.46	.03	.30	.29	.19	.37	.03	.41	.43

** p<.05, ** p<.01, ***p<.001*

Table 8 Gamma Coefficients for Final Model-Women

	Father's OccSES		Childhood Health		OccSES 1st Job		Educational Attainment		Health 1999	
Father's Education										
Missing	-0.058		0.022		-0.128		-0.839	***	_	
	(0.137)		(0.054)		(0.146)		(0.154)			
0-11 Years	-0.582	***	0.099	*	-0.088		-0.573	***	_	
	(0.095)		(0.038)		(0.101)		(0.107)			
13-15 Years	1.353	***	0.092		0.167		0.462	**	_	
	(0.138)		(0.056)		(0.148)		(0.157)			
16 Years	3.217	***	0.010		0.387	*	0.685	***	_	
	(0.132)		(0.057)		(0.152)		(0.161)			
17 + Years	4.048	***	0.053		0.413	*	0.987	***	_	
	(0.165)		(0.074)		(0.199)		(0.210)			
Mother's Education										
Missing	-0.187		0.117		0.123		-0.499	**	_	
	(0.170)		(0.067)		(0.181)		(0.190)			
0-11 Years	-0.232	**	0.086	*	-0.103		-0.614	***	_	
	(0.088)		(0.035)		(0.094)		(0.099)			
13-15 Years	0.222		-0.029		-0.136		0.597	***	_	
	(0.118)		(0.047)		(0.125)		(0.132)			
16 Years	0.451	**	-0.060		0.145		0.653	***	_	
	(0.145)		(0.058)		(0.154)		(0.162)			
17 + Years	0.813	***	-0.189	*	0.635	**	1.235	***	_	
	(0.221)		(0.088)		(0.235)		(0.248)			
Father's OccSES imputed	0.976	***	0.087		0.012		-0.310	*	_	
	(0.121)		(0.049)		(0.131)		(0.137)			
R's Early OccSES Imputed	_		_		-0.058		_		-0.027	
					(0.162)				(0.064)	
Demographic										
Age	0.098	**	-0.004		-0.006		0.088	*	-0.019	
	(0.031)		(0.012)		(0.033)		(0.035)		(0.013)	
Age^2	-0.108	**	0.002		0.039		-0.086	*	0.043	**
	(0.038)		(0.015)		(0.040)		(0.043)		(0.016)	
Black	-0.437	***	0.078	***	-0.193	**	0.055		0.180	***
	(0.056)		(0.022)		(0.062)		(0.066)		(0.024)	
Latino	-0.203		0.169	*	1.014	***	-2.850	***	0.375	***
	(0.187)		(0.074)		(0.204)		(0.210)		(0.082)	
Asian	0.910	**	0.047		0.696	*	0.708	*	0.294	*
	(0.306)		(0.122)		(0.327)		(0.344)		(0.132)	
Other	-0.182		0.191	*	-0.465		0.369		0.101	
	(0.227)		(0.090)		(0.241)		(0.255)		(0.095)	
Married	_		_		0.261	**	0.284	***	-0.087	**
					(0.082)		(0.087)		(0.033)	

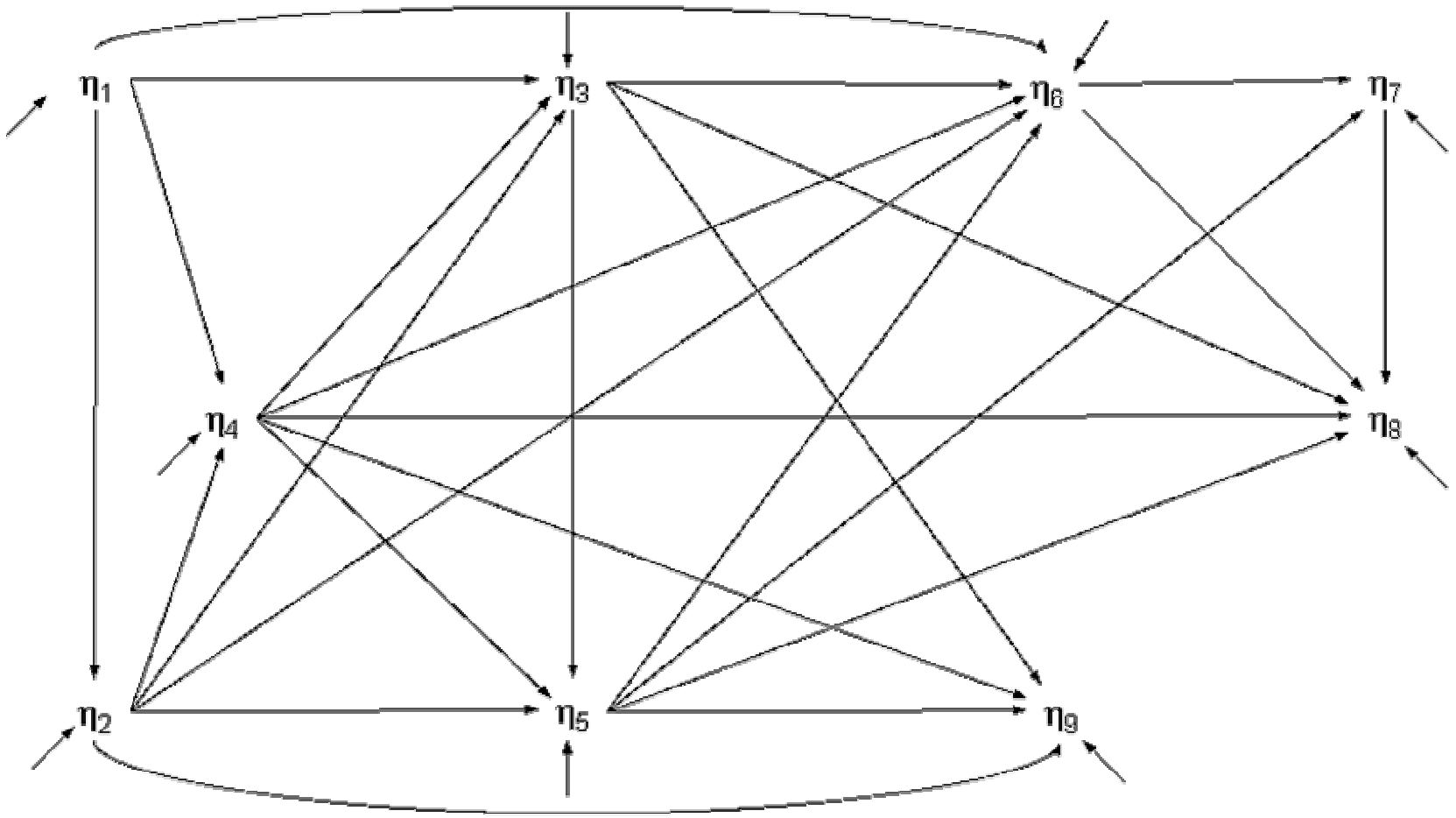
** p<.05, ** p<.01, ***p<.001*

Table 8 Gamma Coefficients for Final Model-Women

	Father's OccSES		Childhood Health		OccSES 1st Job		Educational Attainment		Health 1999	
Father's Education										
Missing	-0.058		0.022		-0.128		-0.839	***	_	
	(0.137)		(0.054)		(0.146)		(0.154)			
0-11 Years	-0.582	***	0.099	*	-0.088		-0.573	***	_	
	(0.095)		(0.038)		(0.101)		(0.107)			
13-15 Years	1.353	***	0.092		0.167		0.462	**	_	
	(0.138)		(0.056)		(0.148)		(0.157)			
16 Years	3.217	***	0.010		0.387	*	0.685	***	_	
	(0.132)		(0.057)		(0.152)		(0.161)			
17 + Years	4.048	***	0.053		0.413	*	0.987	***	_	
	(0.165)		(0.074)		(0.199)		(0.210)			
Mother's Education										
Missing	-0.187		0.117		0.123		-0.499	**	_	
	(0.170)		(0.067)		(0.181)		(0.190)			
0-11 Years	-0.232	**	0.086	*	-0.103		-0.614	***	_	
	(0.088)		(0.035)		(0.094)		(0.099)			
13-15 Years	0.222		-0.029		-0.136		0.597	***	_	
	(0.118)		(0.047)		(0.125)		(0.132)			
16 Years	0.451	**	-0.060		0.145		0.653	***	_	
	(0.145)		(0.058)		(0.154)		(0.162)			
17 + Years	0.813	***	-0.189	*	0.635	**	1.235	***	_	
	(0.221)		(0.088)		(0.235)		(0.248)			
Father's OccSES imputed	0.976	***	0.087		0.012		-0.310	*	_	
	(0.121)		(0.049)		(0.131)		(0.137)			
R's Early OccSES Imputed	_		_		-0.058		_		-0.027	
					(0.162)				(0.064)	
Demographic										
Age	0.098	**	-0.004		-0.006		0.088	*	-0.019	
	(0.031)		(0.012)		(0.033)		(0.035)		(0.013)	
Age^2	-0.108	**	0.002		0.039		-0.086	*	0.043	**
	(0.038)		(0.015)		(0.040)		(0.043)		(0.016)	
Black	-0.437	***	0.078	***	-0.193	**	0.055		0.180	***
	(0.056)		(0.022)		(0.062)		(0.066)		(0.024)	
Latino	-0.203		0.169	*	1.014	***	-2.850	***	0.375	***
	(0.187)		(0.074)		(0.204)		(0.210)		(0.082)	
Asian	0.910	**	0.047		0.696	*	0.708	*	0.294	*
	(0.306)		(0.122)		(0.327)		(0.344)		(0.132)	
Other	-0.182		0.191	*	-0.465		0.369		0.101	
	(0.227)		(0.090)		(0.241)		(0.255)		(0.095)	
Married	_		_		0.261	**	0.284	***	-0.087	**
					(0.082)		(0.087)		(0.033)	

** p<.05, ** p<.01, ***p<.001*

Figure 1 Path Diagram of Endogenous Effects in Model 1



η_1 =Father's Occ SES; η_2 =Childhood Health; η_3 =Occ SES 1st Job; η_4 =Education; η_5 =$Health_{1999}$; η_8 =Occ SES Current Job; η_7 =Average Work Hrs; η_8 =$Earnings_{2000}$; η_9 =$Health_{2001}$