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Early childhood health, reproduction of economic inequalities and the persistence of health and mortality differentials

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Abstract

The persistence of adult health and mortality socioeconomic inequalities and the equally stubborn reproduction of social class inequalities are salient features in modern societies that puzzle researchers in seemingly unconnected research fields. Neither can be satisfactorily explained with standard theoretical frameworks. In the domain of health and mortality, it is unclear if and to what an extent adult health and mortality disparities across socioeconomic status (SES) are the product of attributes of the positions themselves, the partial result of health conditions established earlier in life that influence both adult health and economic success, or the outcome of the reverse impact of health status on SES. In the domain of social stratification, the transmission of inequalities across generations has been remarkably resistant to satisfactory explanations. Although the literature on social stratification is by and large silent about the role played by early health status in shaping adult socioeconomic opportunities, new research on human capital formation suggests this is a serious error of omission. In this paper we propose to investigate the connections between these two domains. We use data from male respondents of the 1958 British Cohort to estimate (a) the influence of early health conditions on adult SES and (b) the contribution of early health status to observed adult health differentials. The model incorporates early conditions as determinants of traits that enhance (inhibit) social mobility and also conventional and unconventional factors that affect adult health and socioeconomic status. Our findings reveal that early childhood health plays a small, but non-trivial role as a determinant of adult SES and the adult socioeconomic gradient in health. These findings enrich current explanations of SES inequalities and of adult health and mortality disparities.

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Keywords

UK; health selection; social inequality in health status; childhood health; life course; mortality; socioeconomic status (SES)

Introduction

There are two apparently unconnected regularities that stand out in modern open societies. The first is that intergenerational transmission of earnings and income inequalities, far from having been eroded, is today as strong, if not stronger, than it was in the past twenty years or so. In the US, progress in intergenerational social mobility has slowed down considerably (Hauser 1998; Hout 2005). By the same token, the intergenerational correlation of income (and wealth) continues to be strong. Indeed, the empirically estimated elasticity of offspring income relative to parental income has remained within the range of .3-.6 for well over twenty years. The bulk of this association is unexplained after resorting to standard explanations from neoclassic labor economics (Heckman & Rubinstein 2001; Bowles, Gintis & Osborne Groves 2005)

The second regularity is the ubiquitous presence of health and mortality differentials across socioeconomic strata. Far from having disappeared, these gradients have acquired renewed salience. There is little doubt that health and mortality gradients by socioeconomic status have persisted and, in some cases, even increased. The evidence is particularly striking in the US but it applies much more broadly to Western European countries and, to the extent that we know, to countries in Asia and Latin America. These gradients are evident irrespective of the metric one uses to assess individual SES: education, income, permanent income, assets and wealth, poverty thresholds, and occupational categories (for a review, see Palloni et al. 2008).

Although the gradients in health and mortality are large, persistent and somewhat insensitive to the metric for SES, their underlying mechanisms remain unclear. Of all possible mediating processes, the one that interests us in this paper is health selection. The main goal of this paper is to formulate an integrated framework to explain the potential role of health selection effects in the production of the observed adult socioeconomic gradient in health. The paper is divided into six sections. In the second and third sections we examine the nature and potential role of health selection and identify mechanisms through which early childhood health can exert an impact on both adult health status and economic success. In section four we formulate a simple model that includes the main relations and translate it into a conventional structural equation model (SEM). Section five then describes the analyses of effects of early childhood health status on adult SES attainment and on the role played by early health status in the explanation of the adult SES gradient. Since the estimated parameters from SEM are insufficient to answer our main questions we resort to Monte Carlo simulations to assess the degree to which the observed association between adult health and SES is due to the direct and/or indirect influence of early health status. Section six summarizes our findings.

What Explains the SES Gradient? the Potential Role of Health Selection

Although there is agreement that at least some of the observed differentials are explained by attributes associated with social class positions (from access to information and health care to life styles and behavioral management and control), there are lingering doubts about the relative power of these factors to account for observed SES gradients (Deaton 2002). Even before the publication of the Black report (Black et al. 1988) but particularly during the debate that followed, it was argued that at least in part the observed SES gradients are the outcome of selection mechanisms whereby individuals in poor health early in life are prevented from or are less likely to experience upward social mobility, more likely to experience downward

mobility, and that through downward or lack of upward mobility, poor early experiences also influence adult health (Manor, Matthews & Power 2003; Smith 1999; West 1991; Stern 1983; Illsley 1955; Power, Matthews & Manor 1998; Fox, Goldblatt & Adelstein 1982; Blane, Harding & Rosato, 1999; Blane, Davey Smith & Hart, 1999; Bartley & Plewis 1997).

Given that the notion of health selection is used with frequently changing meaning (Palloni & Ewbank 2004; West 1991; Palloni 2006), we describe in detail our conception of health selection. Suppose individuals are allocated to various health statuses at birth and suppose also that this allocation does not occur at random but depends on parental health and/or parental social class. Suppose further that individuals located at the lower end of the health distribution are also less likely to develop basic skills, perform well in school, participate in social activities and, more generally, less likely to acquire traits that are both relevant for social accession and conducive to a healthier lifestyle. Membership in a lower social class may gradually strengthen preferences that make more likely the adoption of unhealthy behaviors (such as drinking and smoking). This occurs in addition to the fact that an individual who accedes a low social class is more likely to have been drawn from the lower end of the health status distribution. These relations can proceed with feedback effects and cumulative disadvantages whereby lack of health begets fewer resources and diminished resources begets unhealthy statuses. This kind of process was identified in the Black report and a number of variants have been the focus of a large literature both substantive and empirical (Kuh & Ben-Shlomo 2004; Blane 1999; Blane, Harding & Rosato 1999; Blane, Davey Smith & Hart 1999; West 1991; Stern 1983; Illsley 1986; Power & Matthews 1997; Power, Manor & Fox 1991; Mulatu & Schooler 2002; Case, Fertig & Paxson 2005).

Undoubtedly the existence of direct effects of early health conditions on adult health could reinforce health selection, but they are neither necessary nor sufficient for health selection to operate efficaciously. If, for example, the conjecture about a critical period is borne out by the facts and does indeed lead to higher risks of heart disease or diabetes II (Barker 1998; Gluckman and Hansen 2006) this, by itself, does not automatically result in health selection. By the same token, health selection does not require the existence of health effects associated with a critical developmental period to be an effective producer of SES gradients. Ongoing efforts to distinguish whether associations between early and later life health reflect causal relations or unobserved characteristics related to socio-economic status (reviewed in Huxley, Neil & Collins, 2002) do not address the potential magnitude of health selection. Rather, the only necessary ingredient for health selection is that, at some point in the life course of individuals access to social classes depends on antecedent health status.

Most of the empirical studies that evaluate the role of health selection come to the conclusion that it is of muted importance and that the bulk of the observed SES health gradients are accounted for by mechanisms linking attributes of social class to an individual's health status and mortality. However, a rigorous test of the existence of health selection has not yet been carried out for lack of (a) an appropriate theoretical model linking early health status and adult social stratification and (b) adequate data and/or inferential procedures to identify health selection from observable relations.

This paper focuses on the health selection conjecture. In order to evaluate its relevance we first need to confirm that health inequalities during early childhood are a non-trivial contributor to the persistence of economic inequalities among adults. This can only occur if early health influences cognitive and non-cognitive traits rewarded in the labor market, effectively becoming a contributor to the reproduction of economic inequalities across generations. If this is the case, two consequences follow. The first is that an important mechanism reproducing economic inequalities is the differential allocation of health status that occurs early in life. To understand the reproduction of SES inequalities a properly specified model must explicitly

account for the initial allocation and the influence of early health status during subsequent stages in the individuals' life cycle. The second consequence is that to understand health and mortality disparities it is also necessary to explicitly model health selection processes. It is through these processes that health endowments and attributes acquired early in life could result both in different lifetime exposure to ill-health and in differential ability to harvest economic rewards during adulthood and these, in turn, could affect adult health and mortality.

Although frequently lumped together, reverse causality and health selection ought to be distinguished from each other. Reverse causality refers to processes that produce a SES gradient, due to the direct effect of adult health status on SES. A fairly typical example of this is when a health shock leads to dilution of assets, partial or total withdrawal from the labor force, losses of income and, as a consequence, to a degradation of one's SES (Smith 2004; Adams et al. 2003; Fox et al. 1985; Blane, Davey Smith & Hart, 1999). As conceived here, health selection does involve the influence of health on attributes that are instrumental to accede social positions and may also directly affect adult health status. But this is a process that unfolds over the life cycle of individuals rather than occurring in a relatively short time span or being confined to a narrow band of events, as is usual in the reverse causality argument

Early Health Status, Transmission of Economic Inequalities and Adult Health

Previous literature on early health conditions supports two ideas. First, early health status is not randomly allocated but rather seemingly closely tied to both parental social class and parental health status. Second, early health status exerts a non-trivial influence on adult socioeconomic attainment. These two conditions are sufficient to (a) sustain the intergenerational transmission of inequalities and (b) promote the operation of health selection effects which could account, albeit partially, for SES gradients in adult health and mortality.

An emerging empirical regularity is the association between child health and parental socioeconomic background as assessed by maternal and paternal education, income, poverty level, parental occupation, and receipt of economic assistance. These SES factors appear to have anywhere from weak to strong associations with characteristics such as birth weight, prematurity, growth retardation, stunting, and children's experiences with illnesses. This regularity is not confined to developing countries only but is also found in high income countries. In a recent paper, Case, Lubotsky and Paxson (2002) review three major U.S. surveys with a summary that aptly describes findings from similar research in the area: “We have shown that the relationship between income and health status observed for adults has antecedents in childhood. A family's long-run average income is a powerful determinant of children's health status, one that works in part to protect children's health upon the arrival of chronic conditions.” More importantly: “The health of children from families with lower incomes erodes faster with age, and these children enter adulthood with both lower socioeconomic status and poorer health” (p. 1330).

Corroborating these findings, recent studies find that there are strong income gradients of child health as measured by either reported health status in England (Currie, Shields & Wheatley Price 2007; Case, Lee & Paxson 2007) or chronic conditions in England and the US (Case, Lee & Paxson 2007); and that these gradients do indeed increase with age in countries with and without sizeable socialized medicine sectors such as Canada and the US (Currie & Stabile 2003; Case, Lubotsky & Paxson 2002). This evidence suggests that the adult socioeconomic and health and mortality gradient is formerly mirrored among children (Brooks-Gunn, Duncan & Britto 1999). However, in a recent review of extant evidence Currie (2007) concludes that although there is very strong evidence that children of poor socioeconomic background are in worse health than other children, it is not yet established beyond doubt that the association

reflects a causal link. There are number of causal mechanisms through which this gradient can emerge and it is likely that their relative importance varies with social context.

A somewhat different though complementary idea is that heath status in early childhood may be "inherited" from parents at birth. The apparent relation of parents' and offspring birth weight suggests the possibility of genetic inheritance, although it can also be due to shared environments. In a series of papers, Conley and Bennett (2000; 2001) document strong intergenerational correlation of birth weight for both blacks and whites in the U.S. They find that inheritance of parental birth weight "dramatically reduces the black-white gap in birth weight" (Conley & Bennett 2001). The authors interpret these findings as evidence of both inheritability of a propensity to low birth weight and the influence of environments shared by parents and offspring (see also Currie & Moretti 2007).

The literature concerning early health effects on adult socio-economic attainment focuses almost exclusively on cognition and educational attainment as potential conduits. For example, findings from the 1946 British cohort study reported by Wadsworth (1999; 1991; 1986) indicate that the experience of serious illness during childhood is directly and indirectly associated with decreased educational attainment and increased risks of downward social mobility. Similar evidence has surfaced in other studies (Behrman & Rosenzweig 2002; Lichtenstein et al. 1993; Power, Li & Manor 2000; Lundberg 1991) and is most recently reviewed in Victora et al. (2008). With few exceptions these findings are fragile, as are those that establish direct links between health status in early adolescence and mature educational attainment (Koivusilta, Rimpela, & Rimpela 1998).

A more distal mechanism of influence may occur if events experienced early in life determine individuals' choices and life course, locking them into a reduced set of possible paths. If these career pathways are endowed with different conditions, require the exercise of different abilities, and expose individuals to different opportunities, educational and occupational attainment will vary. Thus, the occurrence of earlier events through which individuals are selected into those life courses or pathways can be thought of as triggers that largely determine subsequent social and economic experiences (Hertzman 1999). An example of this is when early health problems influence early education and thus limit educational attainment and foreclose a number of occupational and career paths. Health shocks at critical periods can be triggers of processes such as those illustrated by Lubotsky (2001) who suggests the emergence of cascading spirals whereby early failures to rank well in cognition, for example, lead to higher subsequent disadvantages.

An additional set of mechanisms by which early health status could impact social stratification is through its influence on non-cognitive skills rewarded in schooling and labor markets (Heckman & Rubinstein 2001; Bowles, Gintis & Osborne Groves 2005). Such skills were originally conceived as personality attributes such as leadership or perseverance. Yet, they may also include physical characteristics, such as height and weight, which may further enhance (depress) the chances of acquiring valued non-cognitive skills. For instance, growing evidence of a relationship between adult height and weight, on one hand, and income and wages, on the other (Behrman & Rosenzweig 2002; Loh 1993; Persico, Postlewaite & Silverman 2004; Sargent & Blanchflower 1994; Averett & Korenman 1996) alongside the long record of ties between early health and nutrition and later life weight and height (Scrimshaw 1997; Floud, Wachter & Gregory 1990; Fogel & Costa 1997; Fogel 1994) outlines at least one conduit linking early health status with adult earnings and economic inequality. When such non-cognitive skills are considered jointly with conventional measures of ability, we find strands of research bridging literatures concerning early health status and determinants of labor market outcomes.

Modeling Socio-Economic and Health Status Attainment

We formulate a general model linking early life events to adult attainment that captures the most important relations identified above. This model builds upon theoretical work first proposed by Grossman (1972) and extended most recently by Heckman (2007). We pose two simultaneous equations representing an individual's SES and health status at different points in the life cycle as a function of past investments, and past SES and health status.

$$S_x = S_x(s_o, H_{x-1}, S_{x-1}, E_{x-1})$$
$$H_x = H_x(h_o, S_{x-1}, H_{x-1}, I_{x-1})$$

S_x and H_x represent individual socioeconomic position and health status at age x, E_{x-1} and I_{x-1} are vectors of human capital and health status inputs or investments (disinvestments) in the age interval (x-1, x), and s_o and h_o are vectors of traits that individuals receive at birth. Common elements may span the vectors E and I and the vectors s_o and h_o. Such a general formulation may include unobserved inputs and traits such as parents' time investments and genetic frailty and is readily adaptable to different datasets varying in the availability of measures of inputs, attainment, traits and proxy indicators of traits as well as in the frequencies of observations over the life cycle.

We estimate a modified version of this model using the National Child Development Study (NCDS) from Great Britain. The NCDS is a prospective longitudinal study of nearly all (98%) children born in the week of March 3-9, 1958 in England, Scotland, and Wales. Medical, social, demographic and economic data were collected on these individuals in interviews with parents, teachers, doctors and cohort members at birth and ages 7, 11, 16, 23, 33 and 41/42. (Ferri (1993) includes a detailed description of the study.) The general model is modified according to availability of measures in the NCDS. We adopt self-reported social class at age 33 and self-reported health status at ages 41/42 as measures of adult attainment. Figure 1 illustrates a path diagram of the main relations that we hypothesize link early child health to these adult outcomes and which may be estimated with the NCDS. Measures of mother's health status and family social class at birth are included and may be interpreted as proxy measures for either inherited traits and/or unobserved parents' behaviors which may affect child health and schooling. Early health status in turn affects cognitive and non-cognitive skills which, jointly with educational attainment, are the main inputs for the production of adult social class. Adult health status then depends not only on prior period socio-economic status, but also prior lags of health status, health at birth and parents' health at the cohort member's birth. While there are many additional relations which may be included, we emphasize the indirect effects of early health on both adult socio-economic and health status through cognitive and non-cognitive skills, as well as through educational attainment.

Four important aspects of this framework merit further explanation. First, we rely on an indicator of low birth weight as a measure of health at birth. Birth weight is determined by both intrauterine growth and gestation length, distinct clinical concepts with different underlying causes which have been associated with different health consequences in later life (reviewed in Huxley, Neil & Collins, 2002; Victora et al., 2008). Our analysis is then limited to the aggregate effects of intrauterine growth and gestation as they are reflected in the available measures of socio-economic attainment and adult health in the NCDS. Despite this general measure of early health risks mixing differential effects of different underlying causes, we discuss below how our findings are robust to alternative definitions of early child health using different combinations of birth weight and gestation. Second, the observations of adult attainment are limited to measures in middle adulthood. The increasing risk for adverse health events with age implies smaller estimates of adult health gradients during this period of

adulthood. However, truncating the life course at age 41/42 also minimizes the potential bias due to the likely increase in effects of health on socio-economic status and heterogeneity during late adulthood. Third, we do not model dynamic parent behaviors that may reflect efforts to compensate the negative effects of early child health status. A review of recent findings (Currie, 2007) reveals that at least in the West, children with disadvantages tend to trigger compensating parental investment. Our estimates then approximate lower bounds of early health effects which take account of such parental behaviors.

Fourth, attrition (permanent or temporal) and item non-response reduces the available sample to a potentially non-random subset of individuals. The number of valid cases decreases approximately 38 percent from the first to the last wave, from 17,415 in 1958 to 10,828 in 2000. Combined with item non-response, the final number of cases with available information is 3,677; 1,858 of whom are males. There have been few efforts to take account of the potentially non-random nature of attrition and item non-response in over twenty years of research with the NCDS. Given reports of the selective nature of wave non-response in the NCDS by education and employment characteristics (Ferri, 1993; Hawkes & Plewis, 2006), we analyze in a separate study models of attrition using Bayesian techniques to adjust for item non-response. However, to the extent that attrition may truncate the observations of individuals from disadvantaged social backgrounds, the present analysis will under-estimate the contribution of health selection.

Our formulation departs in two important manners from related studies of early life health effects which use the NCDS (Case, Fertig & Paxson 2005; Manor, Matthews & Power 2003; Power et al.1990, Currie & Hyson 1999) or other data sets (Mulatu & Schooler 2002; van de Mheen et al. 1997; 1998a; 1998b; Van den Berg, Lendeboom & Portrait 2006; Mackenbach, van de Mheen & Stronk 1994). First, we analyze the importance of cognitive and non-cognitive pathways for the effects of early health conditions on adult health status and social class. Second, we extend the analysis beyond interpreting estimates of early health effects by quantifying the ultimate impact of early health with a simulation method that simultaneously takes account of both the estimated effects and the distribution of the population by type of life trajectory.

Results

The analyses are carried out in two stages. First we describe results that pertain to intergenerational transmission of social class in order to address how much of the observed relation between paternal and offspring social class is attributable to mechanisms involving early health status as measured by birth weight and number of chronic conditions at ages 7 and 16. In the second stage of analysis we test the health selection conjuncture itself, analyzing the social class health gradient at age 41/42 as a function of lagged social class at age 33. Here we ask the following question: *what is the contribution of early health status to the association between lagged social class and health status?* We restrict the sample to males for all analyses due to men's higher rates of full labor force participation and the preponderance of classifications of women's adult social class according to their partner's social class. (All models were estimated using Stata10 and M-PLUS, version 5, 2008).

Stage 1: Early childhood health and the intergenerational transmission of social class

Table 2 displays the estimated ordered probit estimates of social class of destination as a function of social class of origin, parental health status, and childhood health conditions. There are three important results. First, parental class is positively correlated with offspring's social class and robust to the intermediate outcomes reflected in the covariates added across Models I through V. Second, the only indicator of non-cognitive skills that we introduce in the model (a score of early behavioral maladjustment) does not have a direct effect on adult social class.

Experimentation with other measures of non-cognitive traits (early height and teacher's evaluation of individual attractiveness) yields similar outcomes: non-cognitive skills matter but their role pales relative to that of parental social class, cognitive skills and education. Third, measures of early health status have only weak *direct* influence. Indeed, the only variable of some importance is the number of chronic conditions at age 7. In Models III through V neither birth weight nor number of chronic conditions at age 16 matter very much. Including gestation length (results not shown) offers a more precise estimate of the effect of intrauterine growth restriction, but does not change the significance of either a continuous measure of birth weight or an indicator of low birth weight. Alternative specifications with a single variable combining a continuous or dichotomous measure of birth weight with gestation or a set of indicators characterizing four possible combinations of low/normal birth weight and pre-term/normal status are also not significant.

However, it should be noted that birth weight has significant *indirect* effects on adult social class. Table 3 reports significant effects of birth weight on chronic conditions at age 7 and cognitive skills. Chronic conditions at age 7 in turn are significantly associated with both cognitive skills and behavioral maladjustment. The significant effects of both cognitive skills and behavioral maladjustment on education and the importance of both cognitive skills and education for adult social class outline the importance of an indirect pathway through health at age 7, cognitive skills and to a lesser extent, behavioral maladjustment. Elsewhere (personal citation) we have shown that these estimated relations imply that between 7 and 11 percent of the heritability of parental social class works through early health. All of these results are robust to alternate specifications in which adult social class is treated as continuous and estimated with ordinary least squares regression.

Stage 2: Early childhood health and the SES gradient in adult health

Table 4 displays the results of estimating a simple logistic model with poor/fair self-reported health status at age 41-42 as a dependent variable with lagged social class at age 33 as a predictor. Unsurprisingly, the bivariate relation between lagged social class and current health is highly significant: moving from the lowest to the highest social class, on average reduces the probability of reporting poor or fair health by 50 percent (Model I). Perhaps the most startling features of these estimates are the strong influence of low birth weight and parental social class (Models III and IV). While adding gestation length to estimate the effect of intrauterine growth restriction slightly increases the effect of low birth weight (results not shown), the alternate specifications of child health using combinations of birth weight and gestation discussed above do not demonstrate any differential effects. Under the onslaught of large effects of cognitive skills, that virtually sweep all the effects of educational attainment, the influence of lagged social class at age 33 and parental social class disappear altogether. This confirms other findings in the literature that suggest that a large fraction of social class differentials in health are education-related. It also points to cognitive skills as an indirect pathway linking early health to adult health. However, the mechanisms through which low birth weight and parental social class continue to exert effects on offspring self-rated health at adult ages remain to be explained.

The next step in the analysis involves simulation using the estimates from the structural equation model (SEM) characterized by the full set of equations in Tables 2, 3 and 4. We start with the individual members of the cohort for whom we have full information and simulate what would have been their class of destination and their health status at age 41 assuming that they are exposed to the regime of transitions and attainment embedded in the full set of reported estimates in Tables 2, 3 and 4. This simulation includes the role of chance and takes account for uncertainty in model estimates via a resampling approach. (The details of the simulation are available on request and are more fully described in (citation omitted to avoid author

identification)). To avoid cluttering and without any serious loss of information we reclassified social classes into three groups: Low (1 and 2), Middle (3 and 4) and High (5 and 6). The final product of the simulation is a six by six mobility matrix cross-classifying individuals according to their class of origin and their predicted class of destination. Once the matrix is estimated we add assumptions about fertility differentials by social class and examine the long term behavior of an initial and arbitrary vector of individuals by social class. Repeatedly applying the matrix to an initial vector of population classified by social class and health status, we obtain the vector of population we would observe after convergence to a steady state. Each steady state matrix reflects only the characteristics embedded in the structural equation parameters and is independent of the initial vector of population distribution by social class and health status. Matrix analysis helps us to simultaneously consider the influence of the magnitude of effects in the structural equation model and the distribution of the population by all observed characteristics included in the model. The resulting algebra is analogous to that of Leslie matrices in stable populations (e.g., Preston & Campbell 1993;Lam 1986;Keyfitz 1977). In all the exercises with matrices we assume complete homogamy in the marriage market. This ensures that we maximize the effects of both parental social class and original health status. Introducing heterogamy would lead to downplay some of the effects we uncover.

Simulation results allow us to answer a number of questions of interest. First, what is the steady state distribution of social class and health status implied by the estimated SEM? Second, what proportion of the population remains in their class of origin and their initial health status? Third, what is the steady state correlation between lagged social class at age 33 and health status at age 41? We answer these questions by manipulating the estimates of the structural equations to characterize three alternative scenarios: (a) one where early health effects are as estimated by our structural equation model, (b) one where early health effects are set to zero and, finally, (c) one where the effects of early health are multiplied by five. This strategy enables us to assess exactly the size of direct and indirect effects of the covariates of interest, something that cannot be done with our estimates pursuing conventional path analysis decomposition procedures.

Table 5 (panel a) displays the most important results. First, in the scenario with high fertility differentials we see that the proportion of the population born into the lowest social class and poor health status who will remain there when they reach adulthood changes from .272 to .123 when effects of early childhood health are set to zero (value of P1). This is a large change (55 percent). The same (but in the reverse direction) occurs when we compare P2, the proportion of high class individuals in good health who will remain there. When the effects of early childhood health are exaggerated (scenario of Maximum Early Health Effects) the changes in P1 are very large as its value increases from .272 to .967; and the changes in P2 are somewhat more attenuated, from .418 to .563. The results for the two fertility regimes are very similar.

An interesting quantity displayed in the last column of panel a Table 5 is the logistic regression coefficient of the probability of poor/fair self-reported health status on lagged social class. The value estimated from our data hovers around -.382. But when early health status effects are set to zero, the gross effects fall to -.340, an 11 percent decrease: this can be legitimately interpreted as indicating that, in our data and for this particular cohort, the pathways associated with early health that contribute to the correlation between adult health status and social class account for no more than 11 percent of the observed relation.

An alternative method for evaluating the magnitude of health selection effects calculates the SES gradient as the average (across classes) conditional probability of being in bad health given the social class. The maximum value of such an indicator is .50. As shown in Table 5, panel b, and for the high fertility scenario, the indicator has a value of .170 when we use the SEM estimates. In the scenario where early health effects are set to 0, the indicator decreases to .

123. When the effects of early health are multiplied by 5 the indicator grows to about .406. If we take as metric the degree of departure from the maximum gradient, it follows that early health effects explain about 10 percent of the observed gradient (.170/.50-.123/.50). We obtain very similar results in the low fertility scenario. The contribution of early health to the SES gradient in adult health may not be massive but it is certainly not trivial and should not be dismissed too lightly, particularly in view of the fact that we are likely to be underestimating the effect of early health.

Summary and Conclusion

The main goal of this paper is to address in a systematic way the question of whether health selection effects have much or anything to do with the observed adult socioeconomic gradient of health and mortality. To answer this question we proceed by constructing a framework connecting literatures in labor economics and social mobility with research findings from epidemiology and demography regarding extant adult gradients and the role of early health status. This preliminary framework led way to the formulation of a simplified model which was then estimated using a very unique data set that enables us to retrieve information on the life cycle of a cohort. The final estimates from structural equation models were then used as input in a Monte Carlo simulation. Estimating steady state mobility matrices and associated statistics allows us to assess the contribution of early health to the observed adult social class differentials in health status. We find that early childhood health has an important effect on adult social class positions. The results in Table 2 show that part of the relation between parental and offspring social class is accounted for by early health and by the skills that early health influences. The effects of early health are mostly indirect. At least in this model framework, the effects of early health work mostly via its impact on cognitive skills. We also find that early childhood health plays an important role as a mechanism through which adult socioeconomic health gradients are produced. In particular, in this cohort, health selection effects account for approximately 10 to 12 percent of the overall association between lagged social class and adult health status.

There are two important caveats to these findings. First, the estimated effects we obtain from the matrix exercise are likely to be exaggerated since we assume a marriage market completely dominated by homogamy. Indeed, assortative mating and departures from health or social class homogamy will lead to attenuation of the effects that are passed on from one generation to the next. However, the second caveat is that the estimated effects we retrieve are also likely to be lower bounds. This is true for three reasons: First, the sample we use experienced considerable attrition and, according to preliminary analyses, we know that neither permanent nor temporal attrition nor item non-response, occur at random. For example, those who belong to the lowest social classes and who are born low birth weight are more likely to have attrited. It is likely that discarding these cases leads to overall attenuation of the effects of interest. Second, the measures of childhood health are exceedingly coarse. Were we to be in possession of more fine-tuned measures, including physical and psychological impairments, it is likely that the relationships could be stronger than those found. Third, in countries such as the UK around 1958, conditions surrounding the lives of the most deprived in the population were in all likelihood vastly superior to the conditions of the most deprived in low income countries, where early malnutrition, stunting, limited growth and development, and insufficiency of micronutrients, are rampant. In these contexts the room for early health status to leave a deep imprint is much larger and so is the number of routes through which it can craft adult socioeconomic achievement and adult health inequalities.

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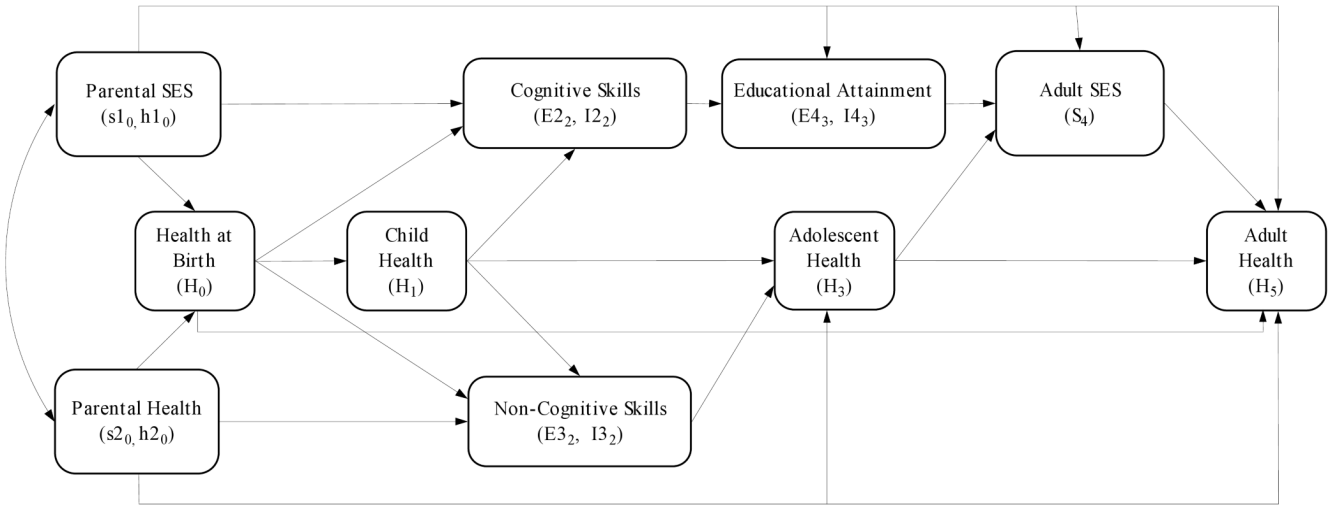

Figure 1. Path diagram representing relations between health and SES over the life cycle

Table 1

Variable Description

Variable	Description
Parental social class	Social class of mother's husband at the time of the cohort member's birth. Six-categories from 1951 Registrar General's Classification: (1) unskilled manual, (2) semi-skilled manual, (3) skilled manual, (4) skilled non-manual, (5) managerial & technical, (6) professional. If this information was missing, either because the mother was single or the father had no available information, we use the mother's own social class before pregnancy (about 3% of the cases). We only use this variable as a covariate, treating it as a continuous variable.
Maternal health	Simple sum of four conditions, all reported by the cohort member's mother: smoking while pregnant, abnormal pregnancy, retrospective reports about whether she developed a chronic conditions before the child's birth and whether she experienced any illness while pregnant. To simplify estimation and since the variable is only used as an independent variable, we treat this variable as continuous variable rather than as an ordered categorical variable.
Early childhood health	Three measures gathered at different points in the cohort member's childhood: (1) Dichotomous measure of weight at birth: low if weight is less than 88 ounces (2,500 grams) at birth; (2) Number of chronic conditions at age 7, reported by a medical practitioner who indicates whether or not the child exhibits each of the following conditions: general motor handicap, disfiguring condition, mental retardation, emotional maladjustment, head and neck abnormality, upper limb abnormality, lower limb abnormality, spine abnormality, respiratory system problem, alimentary system problem, urogenital system problem, heart condition, blood abnormality, skin condition, epilepsy, other central nervous system condition, or diabetes. In the analysis we use a variable that indicated whether the cohort member had 0, 1, 2, or 3 or more chronic conditions, and we treat it as a continuous measure. (3) Number of chronic conditions at age 16. In addition to the conditions reported at age 7, a medical practitioner indicated whether the child had any eye, hearing or speech condition.
Cognitive skills	Standardized average of four test scores, measuring four cognitive domains: Verbal, Non-Verbal, Reading Composition and Mathematics; measured when cohort members were 11 years old. Expressed as z-score and treated as a continuous variable.
Non- cognitive skills	Score of behavioral maladjustment, equivalent to the sum of twelve items representing different aspects of behavioral deviance reported by teachers when child was 11 years old; treated as continuous variable.
Educational attainment	Dummy variable with a value of 1 if the individual passes 5 or more O-level exams, and 0 otherwise. Information collected by schools when cohort members were 20 years old.
Adult social class	Classification of cohort member's own social class, measured at ages 33 and 41/42. Same 6-category classification as parental social class. When predicting adult social class we treat this variable as an ordered one and use ordered probit models to assess the effects of covariates on it.
Adult health status	Individuals' own self-reported health status at ages 33 and 41/42. To simplify analyses we grouped the four-point original scale into two categories: poor or fair (=1); and good or excellent (=0). When predicting adult health status we treat this variable as categorical and estimated logistic regressions.

Table 2

Ordered Probit of Adult Social Class at age 41/42

	Model I	Model II	Model III	Model IV	Model V
Parental Social Class	0.253***	0.249***	0.250***	0.250***	0.125***
	(0.021)	(0.021)	(0.021)	(0.021)	(0.022)
Maternal Health		-0.056	-0.057	-0.052	-0.002
		(0.037)	(0.037)	(0.037)	(0.038)
Low Birth Weight			-0.139	-0.111	0.034
			(0.140)	(0.141)	(0.143)
Number Chronic Conditions Age 7				-0.264**	-0.178+
				(0.089)	(0.091)
Number Chronic Conditions Age 16				-0.043	-0.011
				(0.053)	(0.054)
Cognitive Score Age 11					0.367***
					(0.039)
Behavioral Maladjustment Age 11					-0.001
					(0.003)
Educational Attainment Age 20					0.806***
					(0.071)
Observations	1,858	1,858	1,858	1,858	1,858
Adjusted R-Squared	0.028	0.028	0.028	0.030	0.115
Log Likelihood	-2,596	-2,595	-2,594	-2,589	-2,362

Standard errors in parentheses

+ $p < 0.10$,

* $p < 0.05$,

** $p < 0.01$,

*** $p < 0.001$

Table 3

Pathways to Health and Socio-economic Attainment

	Chronic Conditions Age7	Cognitive Skills Age11	Behavioral Maladjustment Age11	Chronic Conditions Age16	Educational Attainment (Logit)
Parental Social Class	-0.001	0.202***	-0.671***	0.003	0.350***
	(0.005)	(0.015)	(0.158)	(0.009)	(0.057)
Maternal Health	0.015	-0.081**	0.218	0.009	-0.107
	(0.010)	(0.028)	(0.290)	(0.016)	(0.102)
Low Birth Weight	0.095*	-0.262*	0.292	0.005	-0.210
	(0.037)	(0.105)	(1.089)	(0.062)	(0.415)
Number Chronic Conditions Age 7		-0.270***	1.457*	0.287***	0.097
		(0.066)	(0.680)	(0.039)	(0.288)
Number Chronic Conditions Age 16					-0.011
					(0.153)
Cognitive Score Age 11					2.314***
					(0.123)
Behavioral Maladjustment Age 11					-0.044***
					(0.010)
Constant	0.052*	-0.376***	9.911***	0.139***	-2.780***
	(0.021)	(0.059)	(0.612)	(0.035)	(0.243)
Observations	1,858	1,858	1,858	1,858	1,858
R-Squared	0.005	0.106	0.013	0.029	--
Adjusted R-Squared	--	--	--	--	0.382
Log Likelihood	--	--	--	--	-715

Standard errors in parentheses

Two-tailed tests.

$^{+}p < 0.10$,

$^{*}p < 0.05$,

** $p < 0.01$,

*** $p < 0.001$

Table 4

Logit of Fair or Poor Adult Self-reported Health at Age 41-42

	Model I	Model II	Model III	Model IV	Model V
Social Class Age 33	-0.126*	-0.101+	-0.096+	-0.096+	-0.024
	(0.054)	(0.055)	(0.055)	(0.055)	(0.061)
Parental Social Class		-0.131*	-0.136*	-0.136*	-0.078
		(0.061)	(0.062)	(0.062)	(0.064)
Maternal Health		0.109	0.110	0.108	0.087
		(0.104)	(0.104)	(0.105)	(0.106)
Low Birth Weight			0.808*	0.803*	0.747*
			(0.316)	(0.318)	(0.321)
Number Chronic Conditions Age 7				-0.039	-0.141
				(0.247)	(0.256)
Number Chronic Conditions Age 16				0.187	0.134
				(0.137)	(0.139)
Cognitive Score Age 11					-0.324**
					(0.111)
Behavioral Maladjustment Age 11					0.012
					(0.008)
Educational Attainment Age 20					0.015
					(0.209)
Constant	-1.542***	-1.258***	-1.293***	-1.322***	-1.776***
	(0.178)	(0.261)	(0.262)	(0.263)	(0.293)
Observations	1,858	1,858	1,858	1,858	1,858
Adjusted R-Squared	0.004	0.009	0.013	0.014	0.026
Log Likelihood	-701	-697	-695	-694	-685
Information Criteria					
Akaike (AIC)	1,405	1,403	1,399	1,401	1,391
Bayesian (BIC)	1,416	1,425	1,427	1,440	1,446

Standard errors in parentheses

Two-tailed tests.

$^{+}p < 0.10$,

$^{*}p < 0.05$,

$^{**}p < 0.01$,

$^{***}p < 0.001$

Table 5

Selected Indicators from Simulated Social Class and Health Status Mobility Matrix [(a), (b)]

Panel a: Logit estimates

Scenario	P1	P2	Logistic Regression	
			Est.	Sth. Er.
High Fertility Differentials				
Estimated parameters	0.272	0.498	-0.382	0.178
No early health effects	0.123	0.503	-0.340	0.180
Max. early health effects	0.967	0.562	-1.386	0.170
Low Fertility Differentials				
Estimated parameters	0.272	0.418	-0.384	0.178
No early health effects	0.123	0.504	-0.340	0.180
Max. early health effects	0.967	0.563	-1.386	0.170

Panel B: Conditional probabilities of being in bad health, given adult social class

Adult Social Class	Estimated parameters		No early healtd effects		Max. early healtd effects	
	High Fertility	Low Fertility	High Fertility	Low Fertility	High Fertility	Low Fertility
Low	0.208	0.207	0.137	0.136	0.923	0.920
Middle	0.161	0.162	0.144	0.140	0.192	0.190
High	0.141	0.140	0.087	0.086	0.103	0.100
Average	0.170	0.170	0.123	0.121	0.406	0.403

P1 = Proportion born in Lowest social class and poor health status who remain in the same class and health category

P2 =Proportion born in Highest social class and poor health status who remain in the same class and health category

Logistic Regression = Logistic regression coefficient of offspring self-reported health status at age 41-42 and his social class at age 33.

(a) In the scenario with low fertility differentials we used the observed differentials; they are estimated as follows: CEB(class j)=A*exp(r*class j) with A=2.12 and r=-.041 and where CEB are number of net number of children born by age 30. This implies that the ratio of CEB in the lowest class to the highest is of the order of 1.22.

(b) In the scenario Max Early Health Effects, all effects passing through variables reflecting early health conditions are augmented by a factor of 5.