

**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 explained with standard theoretical frameworks.

In the domain of health and mortality, it is still uncertain if and to what an extent adult health and mortality inequalities across the socioeconomic ladder are entirely the product of attributes of the positions themselves and/or the partial result of health conditions established earlier in life that may influence **both** adult health and economic success.

In the domain of social stratification, the persistence across generations of inequalities in educational attainment, wages, income, and wealth is still resistant to satisfactory explanations. Although the literature on social stratification is by and large notoriously silent about the role played by individuals' early health status in shaping their social and economic opportunities as adults, the new research on human capital formation contains plenty of hints suggesting that this is a serious error of omission.

In this paper we suggest a framework that connects these disparate literatures and suggests a model and a set of procedures to estimate (a) the degree to which social mobility or lack thereof is influenced by early health conditions and (b) the contribution of early health status to observed adult health differentials. We utilize evidence from the 1958 British Cohort Study (National Child Development Study, NCDS) to illustrate that there are important mechanisms bridging these seemingly unconnected domains of study. We formulate an integrated framework incorporating early conditions as this enriches current social stratification theory to explain social and economic inequalities, on the one hand, and strengthens theories explaining adult health and mortality differentials, on the other.

INTRODUCTION

The main goal of this paper is to formulate an integrated theoretical framework to explain the possible existence of health selection effects as a mechanism producing the so-called socioeconomic gradient in health and mortality. We do so integrating literatures and findings in separate and unconnected fields, including labor economics, health and mortality and the more recent research on the influence of early health on adult health status and economic success. Using data for a cohort followed from birth to age 40, we estimate a path model that incorporates relations between variables at different stages of the life cycle of individuals. We use these estimates to simulate the life cycle of individuals and to assess if and to what an extent early child health can indeed account for at least part of the observed socioeconomic differentials in adult health.

The paper is divided into five sections. In this, the first section, we provide a quick overview of the problem, suggest the need to articulate separate literatures, and clarify key concepts. In the second section we briefly review our knowledge about adult health and mortality differentials by socioeconomic status and introduce the possible role of health selection. In the third section we 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 conventional path model, summarize the advantages and disadvantages of the data set we use, estimate parameters, and utilize simple simulations to evaluate the effects of early health status on adult socioeconomic achievement, and to measure the degree to which the association between adult health and socioeconomic status is due to the direct and/or indirect influence of early health status. Because we use a data set which captures the experience of a unique cohort in a country with distinct health and social mobility regimes, the conclusions we draw cannot be generalized. We argue, however, that conditions in other societies and for other cohorts may be either similar or involve a stronger role played by early health status. We conclude in the fifth and last section of the paper.

A. Health and economic inequalities: empirical regularities

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. Indeed, in the US at least, progress in intergenerational social mobility has slowed to a halt (Hauser, 2007; Hout, 2008). This regularity is resistant to explanations invoking differential endowments of conventional market-valued skills or of rents extracted from assets—the standard explanations suggested by neoclassic labor economics. The second regularity is the ubiquitous persistence of health and mortality differentials across socioeconomic strata which, far from having disappeared, have acquired renewed salience. Although there is agreement that some of these 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 still lingering doubts about the possibility that observed adult health and mortality differentials are non-trivially related to selection mechanisms whereby individuals who are more likely to be in poor health early on in life are prevented from experiencing upward social mobility or more likely to experience downward mobility (Power and Fox, 1991; Power et al., 1996; West, 1995). This paper focuses on this conjecture. In order to evaluate it, however, we need to first verify a central proposition, namely, that health inequalities during early childhood are a non-trivial contributor to the persistence of economic inequalities among adults. This is a theoretically relevant possibility, and remains to be seen whether it has any empirical significance. If it does, two consequences follow immediately, one relevant for social stratification theories and the other for theories of health and mortality. The first consequence is that an important mechanism reproducing economic inequalities is the differential allocation of health status occurring early in life. By influencing cognitive and non-cognitive traits, early health status will contribute to the reproduction of economic inequalities.

The second consequence, and the one of most interest to us, is that further advances in our understanding of health and mortality disparities across socioeconomic groups will be attained if and when we explicitly model health selection processes. Through these processes 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 throughout adulthood.

The focus of this paper is on the explanation of socioeconomic adult health status differentials and the goal is to examine the contribution to it made by health selection processes. Although the goal of the paper is quite modest, we note that we must at least attempt to offer a

unified explanation of the two empirical regularities identified at the outset, persistence of social inequalities and of health gradients. This requires the establishment of two conditions. The **first condition** is that *allocation of health status early in life does not occur at random but is itself a function of the socioeconomic position individuals occupy at birth*. This implies the need to show that the environment and background associated with a socioeconomic position of origin and/or health status of parents themselves contributes significantly to early child health status. This is a necessary condition for if health status early in life is randomly allocated in every generation with respect to status of origin, it could not possibly contribute to the intergenerational transmission of inequality. The **second condition** is that *there is a relation between health status early in the life of individuals, before they complete their schooling and the acquisition of cognitive and non-cognitive traits that entitles them to economic rewards in the labor market*.

Section II of the paper examines the feasibility and potential conduits of health selection processes. Section III evaluates the first and second conditions. We summarize evidence about key determinants of economic inequalities and highlight findings in social stratification and labor economics connecting determinants of wages, earnings and income with early health status. We identify a subset of determinants that are likely to be affected by early child health status.

B. Conceptual fine-tuning: social class, childhood health, and adult health status

To avoid misunderstandings, we clarify at the outset two of the central concepts we will use throughout, social class and health status.

Social Class. First, to simplify terminology, we will define socioeconomic stratification as a hierarchy of positions which individuals may access and occupy for finite periods of time. Individuals belonging to a socioeconomic position will be distinguished by a key attribute, namely, the appropriation or right of collection of rewards in the form of occupational prestige (a socially constructed quantity), earnings from labor (wages), and rents from assets which, in combination, constitute sources of personal and family income. Some of these rewards, such as occupational prestige or earnings are tightly, though not perfectly, associated with education, a proxy for the presence of rent-extracting skills. These two reward systems may not always be positively correlated and may not even be directly connected to each other. In such cases, we

will refer to only one of them rather than creating a more complex, multidimensional construct encompassing both.

Social sciences assign different importance and meaning to various dimensions of social stratification. In economics the object of study has traditionally been factor payments and, in the case of labor, wages and other types of earnings. Economists also investigate the determinants of wealth and assets as well as the factors influencing income flows derived from assets. But, for the most part, the major controversies center around wages and earnings to which economic sciences assign centrality as a determinant of the economic positions occupied by individuals within a stratified society (see Carneiro and Heckman, 2003). In contrast, classical sociological stratification theory pays considerable attention to occupations— in terms of occupational prestige as well as socioeconomic standing gauged by combinations of attributes such as occupational education and earnings (Hodge, Siegel and Rossi 1966; Duncan 1961; Warren, Sheridan, and Hauser, 1998). That is, sociology is concerned not just with the distribution of material rewards perceived by individuals but also with the allocation of more symbolic markers, some of which may be functions of collective evaluation. More complex conceptualizations, such as those associated with the notion of social class, involve the introduction of other dimensions of occupation and labor, such as control over material and human resources within the job place, degree of authority over subordinates, and location in a hierarchy of decision-making within firms (Wright, 2003).

For the most part this paper focuses on material rewards embodied in wages and earnings and on symbolic rewards such as occupational prestige, and refers only superficially to other dimensions along which individuals may be positioned within a social hierarchy. An immediate advantage stemming from this simplification is that one can confine the term socioeconomic position to refer to individuals grouped by easily measurable attributes such as wages, income, assets or occupational prestige ranks.

Finally, we completely eschew the relevant but somewhat distracting issue of race and ethnic stratification. While allocation of individuals into social classes may dominate the profile of a stratification system, the existence of race adds a layer of complication for two reasons. For one, discrimination or other forms of race-based criteria may partially determine allocation into social classes, independently of individuals' market contributions. Also, and more importantly for us, the processes through which early health status affect adult social status may vary by race.

But the evidence on this score is scant or inexistent (Palloni and Milesi, 2006). While it is clear that race-ethnic disparities in social stratification, mobility and health raise important complications (Palloni and Milesi, 2006), we will focus only on issues that can be assessed along the social class axis, however this is defined.

Child health Child health status does not only refer to what we normally can measure (things such as birthweight, number of chronic conditions or assessment of health status by third parties). In fact, most of what child health really is remains concealed by such feasible measures. To assess child health we should also include factors that surround gestation and birth, physical growth and development, conditions that may be chronic or acute such as asthma, transient episodes of infections many of which have rather striking long term effects, particularly if they are not timely treated. We should consider mental health, but not just severe or mild mental impairments, but also emotional stability and depression. Finally, importance should be given to general fitness and lack of frailty. Both of these features are sometimes visible to the naked eye and can be easily undermined by mild deficiencies in micronutrient intake, such as iron (Thomas, et al, 2006) or more overt poorer nutrition (Glewwe et al, 2001; Popkin et al.2003) Although we have much to learn, traits such as physical frailty or an introverted personality exert powerful effects on children's behaviors towards others and on the behaviors of others toward them. These are non trivial characteristics in a world of open competition for economic positions. The relevance of what we, as a rule, DO NOT measure when we refer to child health becomes obvious upon reviewing an impressive report on the science of early childhood development published by the National Research Council (National Research Council, 2000). There, a case is made that health and well-being of children depends closely on health and well-being of their parents. The authors report that health, mental or physical, of a child, the ability to develop a well-controlled, balanced temperament and a constructive personality and behavior management style, depends on things as trivial as sleeping habits, and on much less trivial phenomena such as how discipline is taught to them. Emphasis on the possibility that these influences sculpt the health status of children is not just a quirk of the literature on child development. As it turns out, this literature is identifying processes that can imperil, impose forbidding constraints, or enhance the development of traits valued in the job market. Maternal care, the environments children experience at home, in school and, now more than ever, in health care centers --the quality of

which is in direct proportion to family income—should be understood better. Jointly, they influence nutritional status, exposure to illnesses, mental well-being and the occurrence of experiences that could inhibit or retard the normal development of equilibrated personalities and temperaments. All these are factors that affect the likelihood of later economic success. Similar findings appear to hold in animal studies (Suomi, 1999; Meaney, 2001) where it has been known for a while that position in complicated social hierarchies of adults is shaped by early experiences as well.

Needless to say what we conventionally measure as child health and the indicators we will use in this paper are far from reaching the core of these states and traits that apparently matter so much. This is a very important reason why the estimated effects of early child health on both social stratification and, very likely, on the relation between adult health and socioeconomic status underplays the true importance of early childhood health.

Adult health status For the most part the literature on socioeconomic differentials in health and mortality has dealt with the mortality part. It is only recently that the availability of surveys that privilege health information has made possible to confirm that similar differentials are observed when using metrics other than mortality. One of them is given by information on self-rated or self reported health status. Although there is disagreement about what exactly do self rating measures and the degree to which they are comparable from group to group (let alone from country to country) there is abundant literature suggesting that they are consistent across persons, reflect well actual health status as assessed by physicians and are a very good, perhaps the best, predictor of short term individual mortality. In this paper we will use self reported health assessed at two different stages in the life cycle of individuals, at age 33 and at age 41.

II. PERSISTENCE OF ADULT HEALTH AND MORTALITY DIFFERENTIALS: THE ROLE OF HEALTH SELECTION PROCESSES

There is little doubt that health and mortality gradients by socioeconomic status have persisted and in some cases have 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. The main difficulty in the assessment of differentials has to do with the choice of a metric to classify individuals in social strata. Due to data availability the

more expeditious way turns out to be the use of educational achievement or years of schooling. Since the landmark study of Kitagawa and Hauser (1973) mortality differentials by education has persisted in the US. Repeatedly new studies using different data sources uncover a recurrent finding, mortality rates among the better educated as several times lower than those who are less educated (Feldman et al, 1989; Lauderdale, 2001, Preston and Taubman, 1994; Preston and Elo, 1995; 1996). In a recent paper it was estimated that the disparities in adult mortality (over age 20) as of 2005 are equivalent to a displacement of mortality risks equivalent to ten years: individuals with lower levels of education experience mortality risks equivalent to those that the better educated experience at ages that are ten years older (Palloni, 2006). A similar and striking disparity exists with regards to self reported health status both in the US (Palloni, 2006; Lynch, 2003; 2005; Smith and Kington, 1997a,b) the US and England and Wales (Banks et al, 2006) and even among adults in Latin America and the Caribbean (Palloni et al., 2006)

These disparities are also commonplace in the developed world where analogous information is available. As Valkonen has shown, the gradients are strikingly similar in the US, England and Wales, Finland, Hungary, Norway, and Denmark (Valkonen, 1987; Rogot, 1992).

Using other metrics to assess social class does not alter the picture. Indeed, the gradients tend to become steeper when one uses permanent or even more transient measures of income (Duleep, 1989; Pappas et al, 1993; Sorlie et al, 1995; McDonough et al, 1997; Lynch, 2003; 2005). An even stronger relation and steeper gradients obtain with measures of wealth and assets (Spittel, 2004; Mare and Palloni, 1988; Palloni and Spittel, 2005; Smith, 1999; Adams et al., 2003; Attanasio et al., 2000), or poverty (Menchick, 1993). Using occupational rank—either as prestige score or with the standard British classification- leads to similar though somewhat more attenuated disparities (Kitagawa and Hauser, 1973; Moore and Hayward, 1990; Mare, 1986; Marmot et al, 1997; Power, 1991; Fox et al., 1985). And, as mentioned above, just about all these differentials remain strong whether one uses mortality or self reported health status (Lynch, 2003; 2005)

This much is not controversial: the empirical evidence stubbornly suggests that gradients of health and mortality are large, persistent and somewhat insensitive to the use of heterogeneous metrics. What is controversial are the mechanisms producing them: what of income, wealth, education or occupation is health protective?. Of all possible mediating processes, the one of interest in this paper is the so-called *health selection mechanism*. Because this term is used in the

literature with changing and frequently inconsistent meanings and definitions, we will first define what we consider relevant health selection mechanisms (for a more detail review see West, 1995 and Palloni and Ewbank, 2006)

A. Reverse causality

This is the most frequently invoked relation whereby the gradient is generated because antecedent health status constrains the social position individuals may occupy at any given point in time. To the extent that a major illness leads to dilution or liquidation of assets, a cross sectional study (and even a limited panel study) may find a negative association between health status or mortality and assets or income one simply because the health deterioration process operated as a shock on assets and wealth (Smith, 1999; Adams et al, 2003). A similar mechanism was invoked early on to partially explain the gradient in England and Wales though here the outcome affected by antecedent health status was occupational and labor force status rather than wealth or assets (Fox et al 19xx).

B. Direct selection or drift

This type of mechanism is generally associated with simple processes that involve an antecedent limitation that precludes social accession. Serious physical or mental illnesses that impair individual's ability to perform routine tasks, for example, will necessarily limit the occupational paths that may be followed throughout the individual's life course. To the extent that such limitations are also associated with higher levels of ill health or later mortality risk, the mechanism will induce a spurious association between social class and health and mortality (West, 1991) . By definition, drift or direct selection is rare in most populations and should not account for more than a trivial fraction of the observed adult health and mortality gradients.

C. Heterogeneity

To the extent that the composition of a closed population changes over time (age) in ways that are directly influenced by individual frailty, levels of health and mortality in various social subgroups will converge as those who are more frail are selected out, leaving only individuals above a threshold frailty level in all social groups (Vaupel et al, 1979; Palloni and Ewbank, 2006; Beckett, 2000)). This mechanism could be responsible for weakening or even reversals of

gradients at older ages. To our knowledge, the magnitude of the contribution of this type of selection to the reduction of gradients has not been conclusively estimated. In any case, it is only in perverse cases (during periods following wars or population crisis that change mean frailty negatively in lower status groups and positively in higher status groups) can this type of selection induce an artificial increase of the gradient. In most empirically relevant cases heterogeneity leads to a downward bias of the adult health and mortality gradients.

D. Indirect or background health processes

The most interesting of all selection processes, and also the most likely to have a pervasive effects, is referred to as indirect selection. Suppose individuals are allocated to various health statuses at birth and supposed also that 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 perform well in school, to participate in social activities and, more generally, to acquire traits that are both relevant for social accession in the social stratification system and conducive to an unhealthier life style. It may well be that membership in a lower social class strengthens the adoption of unhealthy behaviors (such as drinking and smoking), but this occurs in addition to the fact that an individual who acceded this social class is also more likely to have been drawn from the lower end of the health status distribution. The relations can proceed with feedback effects and cumulative damage and disadvantages whereby lack of health begets less resources and diminished resources begets unhealthy states. This kind of selection process is the one that was prominently noted in the Black report. It has also been the focus of a large literature both substantive and empirical (Power and Fox, 1991; Power et al., 1996; Stern, 1983; West, 1988; Chandola et al, 2003; Blane, 1985; Blane et al., 1993; 1999; Manor et al., 2003; Illsey, 1955; Power and Matthews, 1997; Kuh and Shlomo, 1997; Palloni and Ewbank, 2006).

Two remarks are important. The first is that the effective operation of health selection does not require that early health status be allocated via a specific mechanism. It can occur and account for the health gradient even if early health is allocated randomly. However, the *intergenerational transmission of health status and social class via health status* can only operate if early health status is allocated non randomly. That is, health selection can be a

pervasive process influencing health gradients if and only if antecedent health status is distributed non randomly.

Secondly, while the existence of mechanisms whereby early conditions influence adult health could reinforce health selection they are neither necessary nor sufficient for it to happen. If, for example, the critical period conjecture is borne out by the facts and does indeed lead to higher risks of congestive heart disease or diabetes II, this by itself will not automatically result in health selection. By the same token, health selection does not require the existence of critical period type of effects to be an effective producer of social class gradients. All that is needed is that at some point in the life course of individuals (or at multiple stages in it) accession to social classes be a function of antecedent health status.

It is fair to say that most of the empirical studies that evaluate indirect selection mechanisms lead to the conclusion that they are of rather muted importance, and that the bulk of the gradients must be attributable to mechanisms linking attributes of the social class to the health status and mortality of individuals. However, we believe that an accurate 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 procedures to identify health selection from feasible observable relations.

III. EARLY HEALTH STATUS, TRANSMISSION OF ECONOMIC INEQUALITIES AND ADULT HEALTH

This section develops arguments to support two ideas. First, that early health status is not randomly allocated. Instead, both parental social class and health status have a lot to do with it. Second, that early health status is a non trivial determinant affecting the life chances of individuals in the social stratification system. These two conditions are sufficient to generate an environment of relations that (a) can lead to intergenerational transmission of inequalities and (b) promote the operation of health selection effects that could account at least partially for the social class gradient in adult health and mortality.

A. Non-random allocation of early health status

Perhaps one of the most recurrent findings in the literature on child health is that early childhood health conditions are related to a number of indicators that reflect the socioeconomic position of the family of origin. Among these are maternal and paternal education, income,

poverty levels, parental occupation, and receipts of economic assistance. These factors appear to have anywhere from weak to strong associations with characteristics such as birth weight, prematurity, growth retardation, stunting, children's experiences with illnesses, and other indicators of child health status. The relations are by no means uncontroversial. Indeed, close scrutiny of various studies available to us does not eliminate ambiguities about either the magnitude or the type of processes involved. Thus, for example, in a meta analysis of 895 studies carried out between 1970 and 1984, Kramer (1987) found that several of the above mentioned socioeconomic variables had weak **direct** effects on prematurity, low birthweight and intrauterine growth retardation (IUGR). These findings applied both to developing and developed countries. On the other hand, analyzing the first wave of the Children of the National Longitudinal Survey of Youth (NLSY-C), Cramer (1995) found that while income from earnings and from family and public assistance is indeed associated with birthweight, the effects are small and often not significant at conventional statistical levels. Yet he also finds that low income accounts for much of the excess incidence of low birthweight among blacks and other racial and ethnic minorities.

Although none of these studies find strong associations between early childhood health indicators and parental income, or other indicators of parental social class position, it is important to remember two issues. First, these studies refer to **direct effects** of social class, that is, those that remain after controlling for a number of intermediate factors, such as birth order, length of previous and following interval, mother's age, mother's marital status, timing of prenatal care and the like. Our argument is that social class affects birthweight (or alternative indicators of child health status) *regardless of the nature of mediating mechanisms*. Thus the evidence of a weak **direct** relation is immaterial for our purposes. In most studies included in Kramer's review and in Cramer's work, the gross effects of income and maternal education, for example, are much larger than the direct effects.

Second, as Cramer (1995) recognized, measures of dimensions of social class or strata used in these studies are conventionally quite poor, as they leave out many factors that have significant effects. Thus, "if these other dimensions were included in the model, surely the effects of economic status would be impressive, both in predicting birth outcome and in explaining ethnic differences in birth outcomes" (Cramer, 1995: 244). This conclusion is in

keeping with results obtained for the U.S. from the National Maternal and Child Health Survey (Palloni and Partin, 1994).

The tenor of the discussion in very recent research in the U.S. and elsewhere confirms the importance of family socioeconomic background for both its direct and indirect effects. This research has exploited large and rich data sets, uniformly confirming that social and economic characteristics of the family of origin, including but not limited to maternal education, have strong effects on child health and mortality. In a recent paper, Case and colleagues (Case et al., 2001) complete a review of 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 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 eroded faster with age, and these children enter adulthood with both lower socioeconomic status and poorer health" (Case et al, 2001 p. 29). Thus, the adult socioeconomic and health and mortality gradient is formerly mirrored among children (Brooks-Gunn et al., 1999). There are number of mechanisms through which this gradient can emerge and it is likely that their relative importance will vary with social context.

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

Children from disadvantaged families are more likely to suffer from poor health. "Wealthier parents may be better able to purchase medical care, nutritious foods, and safer environments for their children and, in these and many other ways, income may have a causal effect on children's health" (Case, Lubotsky and Paxson 2000, p. 1039). Not only are disadvantaged children at a higher risk of poor health, research suggests their families are less

likely to be able to deal with the consequences of poor childhood health than advantaged families. In their research on the impact of early childhood health (measured by low birth weight) on educational attainment Currie and Hyson (1999) find that low birth weight has long-term effects on children from advantaged as well as disadvantaged families. However, the authors note that disadvantaged children “suffer from double jeopardy in that they are more likely to suffer both from the effects of low socioeconomic status and from low birth weight” (Currie and Hyson 1999, p. 250). The authors argue that while children from all social backgrounds are at risk of developing health problems, advantaged families are able to assuage the disadvantage of poor health. These same mechanism may be at the root of recent findings suggesting gaps in health status of children by social class widens as they age both in countries with and without sizeable socialized medicine sectors such as Canada and the US (Currie and Stabile, 2003)

In summary, the bulk of the evidence indicates that, either due to early environments -- conditions directly associated with parental social, economic and cultural endowments-- or through inheritance and predispositions, there is a correlation between parental health status and parental economic status, on one hand, and offspring’s health conditions during early childhood, on the other. If so, the third condition of possibility identified in the first section of the paper is confirmed.

B. Factors on social stratification: what matters for crafting inequality

In theory at least, payments to labor in the form of wages and salaries (earnings) ought to be a function of productive (market-related) skills, namely, traits (acquired or inherited) that contribute to the production process and, as such, are explicitly entered in a production function. Standard economic theory concedes that the importance of these factors may vary depending on market conditions but, by and large, is unconcerned with the possibility that markets may reward other traits.

Conventional wage enhancing traits Traditionally, relevant skills have been equated with educational attainment, experience, on the job training, and cognitive abilities. These variables have been shown consistently to exert important effects on earnings but, equally consistently, they fail to account for more than a fraction of earning variance within the same generation

(Bowles and Gintis, 1976; Heckman and Rubinstein, 2001; Jencks, 1979; Jencks and Phillips, 1998). Indeed, according to a recent survey of the empirical literature between 67 and 80 percent of the variance of (log) earnings remains unexplained after accounting for a person's age, years of schooling, years of labor market experience, and parental characteristics reflecting socioeconomic background (Bowles et al., 2000). Furthermore, there is a persistent direct effect of parental earnings (or background) on offspring's earnings which is robust to model specification, and *remains so even after controlling for a host of market-relevant offspring's traits, including years of schooling, cognitive ability and labor market experience* (Mulligan, 1997; Bowles and Nelson, 1974; Bowles et al., 2000).

Additionally, the effects of standard labor market characteristics are not easily explained with standard interpretations. In particular, while the effects of years of schooling on earnings are strong and always statistically significant, the mechanism through which they operate remains obscure. One conduit or mediating mechanism, cognitive abilities is, as one would expect, quite important but it only accounts for a small fraction of the effects of education.¹ Thus, some of the effects of education on earnings may be due to high correlations between education and other, non-cognitive traits (Bowles and Gintis, 1976; Carneiro and Heckman, 2003).

The puzzle is not that extant research suggests that standard human capital variables are irrelevant for earnings, but that after fully accounting for them there is still substantial unexplained variance in (log) earning and a remarkably high degree of uncertainty about the mechanisms through which standard factors realize their effects (Palloni and Milesi, 2006).

In short: current conventional accounting of labor earnings is not satisfactory. Not only do they explain a small fraction of the variance of earnings, but also variables that are important explanatory factors are not supposed to be so, and those that we expect to perform do so but only up to a point and, furthermore, we are not able to explain or interpret their effects. Why should other individual traits matter at all either as direct predictors of earnings or as mediating factors between years of schooling and earnings? What exactly are they factors, what are they useful for, how are they acquired, how is it that they become beneficial for employers?

¹ According to estimates drawn from an extensive review by Bowles et al (2000) cognitive scores account for no more than 20 percent of the effects of education on earnings.

The relevance of “soft-skills”

Although microeconomic models that include unconventional traits are not standard fare, those that have been formulated do shed light on conditions under which the demand for these traits may grow, thus fostering mechanisms to reward individuals who possess them. Perhaps the most important theoretical insight comes from the literature on microeconomic markets with asymmetric information, where employment relationships are contractually incomplete and associated with potentially significant enforcement costs (Bowles et al., 2000). Bowles and colleagues start with a conventional (Walrasian) model but proceed to do away with assumptions regarding mechanic equilibrium and invoking exogenous effort in the work place. The new situation is one of asymmetrical information and subject to incentive problems, since workers’ effort is not fixed and can vary according to the work situation. Enforcement of contractual relations is costly and employers will choose to reward employees’ characteristics or traits that minimize such enforcement costs while ensuring their full cooperation. Bowles and colleague baptize these traits "incentive enhancing preferences" (IEP). They refer to personal traits that facilitate the timely flow of quality labor inputs into the production process. Examples of such IEP are loyalty, predisposition to telling the truth, low disutility of effort, low time discount rate, "self-directedness", sense of efficacy, perception of being in control, low fatalism, and ability to function in groups and establish social networks. The degree to which these traits matter --that is, the degree of deviation from a Walrasian world--is possibly variable and will depend on local markets conditions, type of industry or establishment, technological change, and legal environments , among other factors. Thus, the degree to which earnings are responsive to IEP and/or more conventional market skills will also vary according to these conditions.

Some of these traits are undoubtedly shaped through school experience and will thus be captured by years of schooling. Some are learned or acquired outside school and other informal settings. Yet others, as suggested recently by Farkas (Farkas, 2003), may be part of a set of habits with which individuals are endowed as a result of prolonged learning and early socialization. These are traits sculpted early on in childhood, through parents’ involvement, hands-on-teaching, mentorship, participation in extracurricular activities, etc. Some could contribute to educational attainment and cognitive performance; others may be unrelated to

either. Many of these, however, are most definitely not the result of a limited stay in high school or of transient participation in job training settings (Heckman, 2000)².

The evidence that IEP are important contributors to human capital, and can facilitate rent extraction just as other production factors do, is somewhat recent and does not yet amount to a well-established body of literature. However, the findings are so ubiquitous as to encourage reexamination of standard human capital theories and to encourage revisions of the foundations of public policy. In a sweeping account of conditions that could improve the effectiveness of policies designed to enhance human capital, Heckman (2000) suggests that a broader view of skills and the process that produces them is called for. In particular, Heckman points out that exclusive preoccupation with cognition and academic performance, as assessed by test scores, educational attainment or adult-on the job training is misguided, and that models and policies must make room for conditions associated with "motivation" and "social adaptability" (Heckman, 2000). Evidence for this "shadow" reward system comes from very different traditions: one emphasizing personality traits (Jencks, 1979; Dunifon and Duncan 1998; Osborne 2000), another focusing on attitudinal and motivational traits (Heckman, Hsee and Rubinstein, 1999; Cameron and Heckman, 1993; Nollen and Gaertner 1991; McClelland and Franz 1992) and yet other honing on physical characteristics such as obesity among women (Gortmaker et al 1993, Harper 2000, Register and Williams 1990; Sargent and Blachflower, 1994; Register and Williams 1990; McLean and Moon, 1980) and height among males (Loh 1993; Judge and Cable (forthcoming); Harper, 2000; Sargent and Blachflower 1994; Persico et al., 2001). An important interpretational issue is that what appears to matter with these traits is not their value or status during adulthood but early on in the life of the individual. Thus, it is not weight or height at age 30 per se that matters but what *they reflect about height and weight during early childhood or adolescence* (Persico et al., 2001). Thus, adult height appears to have an effect only because it is related to early height. And it is the latter what actually facilitates the acquisition of traits (such as leadership, responsibility, working habits) that are demanded in the labor markets. Height and weight my simply act as signals to employers that reveal the possible existence of such desirable qualities, much more than diplomas or other formal credentials. A growing literature in labor economics attempts to identify the formal conditions that characterize markets conditions such

² If the most important among these traits were a function of parental involvement and background only, their effect would vanish once a control for the latter is introduced. We are unaware of models including good measures for either.

that traits not conventionally included in standard accounts of labor market theory, even assortative mating (Ermish et al., 2005), acquire importance in any given context (Bowles, Gintis and Osborne, 2005).

Admittedly the evidence available so far is tenuous and does not constitute a proper body of work on which the relevance of IEP's could rest. But it is still suggestive. And, more importantly for the main argument in this paper, it establishes an additional conduit through which early health status may influence social stratification. For example, it is well known that both early obesity and childhood height are strongly influenced by birthweight.

C. Enduring effects of early childhood health and other conditions

The foregoing establishes that there are a number of factors influencing adult earnings that may be plausibly related to early health status. We will now discuss if there is any evidence substantiating the claim. If so, then early health exerts an influence of later economic success, either directly or indirectly.

The bulk of the effects we refer to below are not direct but instead mediated by other factors and they can be classified into two classes: first, those that influence individuals' cognitive abilities at various ages, ultimate educational attainment, and in-the-job acquisition of skills and, second those that shape, constrain or facilitate the acquisition of IEPs or soft-skills.

Direct Effects

A typical example of a direct effect is when early health status leads to some form of physical or cognitive disability that limits educational attainment and obstructs labor market opportunities or on the job advancement. According to a recent estimate, for example, anywhere between 12 and 15 percent of all American children have some type of disability (Westat, 2000). More importantly, even among those who succeed in attaining high school diplomas, “disability and type of disability profoundly affect their immediate post-high school activities” (Wells et al., 2003), after the effects of family background have been controlled for. This evidence points to a direct connection of antecedent health (inducing disability) and labor market opportunities.

Indirect effects through conventional traits

By and large, the influence of early childhood health status is likely to be indirect. The literature documenting their existence focuses almost exclusively on cognition and educational attainment as potential conduits. Thus, findings from the 1946 British cohort study reported by Wadsworth (Wadsworth, 1986; 1991; 1999) 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 (Lichtenstein et al., 1993; Power et al., 2000; Lundberg, 1991; Rahkonen et al., 1997; Behrman and Rosenzweig, 2002). 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, et al., 1995; 1998).

Other mechanisms could alter individuals’ ability and motivation to learn, school performance and attainment and ultimately labor market success. Exposure to unfavorable conditions *in utero*, subsequent low birthweight, and illnesses and poor growth and development during the first years of life modify the growth of brain tissue and through it alter the functioning of one or more neuro-physiological centers of hormonal balance and activation. These, in turn, will affect behavior, motivation, individual choices, resilience, frailty, and immune status (see the collection of essays in Keating and Hertzman, 1999; Hack et al., 1995). These processes have the potential to induce a wholesale shift in lifelong health (and mortality risks) of individuals exposed to them and to shape and mold cognitive experiences and learning early in life.

Thus, for example, recent studies show that cognitive scores and educational attainment among low birthweight (less than 2,500 grams) children are significantly lower than among

normal birthweight children, even when comparisons are made between siblings (Boardman et al., 2002; Conley and Bennett, 2000a; Matte et al 2001). Other studies replicate this finding in other contexts and extend it to include influences of other birthweight intervals, such as very low birthweight children (less than 1,500 grams) or different ranges within the normal birthweight distribution (Seidman et al., 1992; Richards et al, 2001; Jefferis et al 2002; Elgen and Sommerfelt, 2002; Hack et al, 2002). Behrman and Rosenzweig (2002) analyze data on female twins from a sample of the Minnesota Twin Registry. Using within monozygotic twin estimators, they show that augmenting a child's birthweight in one pound increases schooling attainment by about a third of a year, and boosts adult earnings by over 7 percent. More generally, the evidence shows that, however measured, early health status has a direct impact on early cognition, school performance, late cognition, and educational attainment (Taubman, 1975; O'Brien, 1996; Grossman, 1972; Edwards and Grossman, 1979; Shakotko et al., 1981; Rosenzweig and Wolpin, 1994; Korenman et al., 1994; Richards et al., 2001; Ricciuti and Scarr, 1990; Scarr, 1982; Hack et al., 1995).

A more distal mechanism of influence can occur when early events or experiences determine individuals' choices and life courses, locking them to a reduced set of possible paths to follow. If these life courses or pathways are endowed with different conditions, require the exercise of different abilities, and expose individuals to different opportunities, individuals' 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. Take the case of apparently trivial events, such as early chronic otitis media. It has been shown that this may be influential in determining some dimensions of cognitive ability, particularly in the acquisition and development of verbal skills (Shriberg et al., 2000). If so, exposure to the condition during a critical period in the life of the individual will simply preclude some developmental paths. These types of health shocks at critical periods are important to trigger processes such as those illustrated by Lubotsky (2001) who argues that there is strong evidence for cascading spirals whereby early failures to rank well in cognition, for example, leads to higher subsequent disadvantages, a confirmation in reverse of Heckman's idea that "skills beget skills."

The evidence discussed above suggests alternative causal chains whereby early childhood health status acts through conventional mediating mechanisms of economic success, namely, early and late cognition and educational attainment.

Indirect effects through ‘soft skills’ Another set of mechanisms by which early health status could impact social stratification is through its influence on "soft-skills" or IEP's, perhaps by affecting physical characteristics, such as height and weight, which enhance (depress) the chances of acquiring these "soft-skills." Relevant to the effect of early health on "soft-skills" is a large body documenting the influence of early health and nutritional status on adolescent and adult weight and height (Fogel and Costa, 1997; Fogel, 1994; Floud et al., 1990; Scrimshaw, 1997; Martorell et al, 2001). This evidence provides at least one conduit linking early health status with adult earnings and economic inequality when it is complemented by growing research on the existence of a relation between income and wages, on one hand, and adult height and weight, on the other (Behrman and Rosenzweig, 2002; Martel and Biller, 1987; Loh, 1993; Sargent and Blanchflower, 1994; Persico et al., 2001; Hamermesh and Biddle, 1994; Averett and Korenman, 1996; Thomas and Strauss, 1997).¹² Through what mechanisms do adult height and weight, partial outcomes of early health status conditions, confer advantages (disadvantages) in the labor market? As discussed before, both with regard to height (Persico et al 2001) and weight (Sargent and Blanchflower, 1994; Averett and Korenman, 1996), the key does not seem to reside on adult physical characteristics, but on appearance during adolescence. This suggests that being a tall man and a non obese woman facilitates the acquisition, prior to entrance into the labor market, of traits that are highly valued by employers. Thus, these skills, not adult physical appearance per se, are what makes early height and weight relevant for labor market success.

In sum, when conventional and "soft-skills" traits are considered jointly, we find strands of research that enable us to bridge literatures on early health status, on the one hand, and on determinants of labor markets and socioeconomic status, on the other. This literature is not conclusive but provides sufficient empirical evidence on which the plausibility of the second condition of possibility mentioned above can rest.

¹² Although "soft-skills" not reflected in height or weight could also be associated with early health status, we are not aware of literatures documenting these effects.

In the following section we translate the relations identified before into a path model covering a large section of the life cycle of individuals, we use a data set to estimate its parameters and then use path analysis and simulations to generate statistics that illustrate two dimensions of the process: (a) the degree to which early health accounts for the intergenerational transmission of inequalities and (b) the degree to which selection mechanisms triggered by early health conditions account for the adult social class differentials in health status

IV. MODELS, DATA AND ESTIMATION OF EFFECTS

We formulate a parsimonious model capturing the most important relations for which we found empirical evidence in the discussion above. The model can be translated into a path diagram that applies very generally. However, since the data set we use only yields information up to about age 40, we will limit its reach and we will only focus on social class position and health status at age 40 as the final outcomes. This has an obvious disadvantage in that most of health events occur after age 40 and, thus, we are likely to underestimate the total adult health gradient. It has an important advantage, however, since by truncating the life course at age 40 we minimize problems engendered by reverse causality and heterogeneity.

A. Models

Figure 1 displays the dimensions of the model and the principal relations involved. Parental social class and parental health are on the left side of the diagram. These influence health conditions early on and this, in turn, affects cognitive and soft skills. Added to educational attainment, these are the main inputs for the production of offspring's social class (at age 40). Figure 1 is sufficient to represent relations affecting the intergenerational transmission of social class.

Figure 2 is an enhanced version of Figure 1 that includes health status at both ages 30 and 40. Although it portrays relations between contemporaneous measures of social class and health status, the primary concern for us is the relation between social class at age 30 and health status at age 40. Exclusive examination of the latter minimizes the possibility of overplaying the strength of the association due to reverse causality.

Figure 1

Figure 2

Each of these two figures translates into a series of standard set of structural equations (not involving feedbacks). The parameters of the system are estimated using maximum likelihood procedures to enable us to handle equations where the dependent variables are discrete.

B. Data set and description of variables.

The National Child Development Study (NCDS) or BCS-58 for short, is a prospective longitudinal study of nearly all (98%) children born in the week of March 3-9, 1958 in Great Britain (England, Scotland, and Wales). The study is known as the 1958 British Cohort. Medical, social, demographic and economic data has been collected on these individuals from birth through age 42, including follow-up interviews with them, their parents, teachers, doctors, or their partners at ages 7, 11, 16, 23, 33 and 42. The number of valid cases has decreased approximately 35 percent from the first to the last wave, from 17,414 in 1958 to 11,419 in 2000. The remaining respondents were found to be mostly representative of the original sample, although attrition is slightly higher among disadvantaged respondents (Ferri, 1993). For a detailed description of the study see Ferri (1993). Our analysis considers only males, and it includes data collected at all but one of the follow-up surveys. The final number of cases with available information is 1987. There is a relatively strong association between probability of attrition and social class of origins as there is between early health status (as reflected in low birthweight) and risk of attrition. This characteristic of the data set is likely to bias downward some of the association of interest to us.

We now briefly describe selected key variables used in our analyses. First, **educational attainment** is measured as a dummy variable with a value of 1 if the individual passes 5 or more O-level exams by age 16. In England, students take qualifying exams in high school to assess their achievement. The results of these exams determine their future schooling and/or employment trajectories. If a student passes five or more O-level exams, they are qualified to then take A-level exams at the end of high school. Successful performance on these exams allows them entrance into university. Clearly, a more refined measure of educational attainment, perhaps analogous to number of years of schooling would have been preferable. As an indicator

of a **non cognitive trait** we use a score of maladjustment. At age 11 cohort members were assessed by their teachers on a number of noncognitive and behavioral aspects. The maladjustment scale used in this analysis is the square root of a simple sum of twelve aspects of behavioral deviance: Unforthcomingness, withdrawal, depression, anxiety for acceptance by adults, hostility towards adults, 'writing off' of adults and adult standards, anxiety for acceptance by children, hostility towards children, restlessness, 'inconsequential' behavior, and miscellaneous symptoms, and miscellaneous nervous symptoms. The possible" maximum sum of the scales is 153, however, the maximum recorded in this sample is 70. **Parental Social Class** was measured based on the social class category of the mother's husband at the time of the cohort member's birth. If he was not present in the household, then the mother's social class before pregnancy was used. The NCDS reports parental social class from the 1951 Registrar General's Classification. This system considers social class as reflected in the type of occupation in which the parent was employed. In this analysis, parent's social class was categorized according to a reduced form of the original: unskilled manual, semi-skilled manual, skilled manual, skilled non manual, managerial & technical, or professional. Cases where the parent was unemployed, a student, dead or retired were considered missing. **Early Childhood Health** is assessed using three measures, each gathered at different points in the cohort member's childhood. The first is a dichotomous measure cohort member's weight at birth. In this analysis, a child's birth weight was considered low if the child was less than 88 ounces (2,500 grams) at birth.

The second and third measures of early childhood health utilize the rich data the NCDS provides of the number of chronic conditions the cohort member experiences in childhood (age 7) and early adolescence (age 16), and are either a sign of a condition present at birth or acquired in childhood. The child is assessed by a physician in a medical examination. The doctor is asked to assess whether or not the CM exhibits different conditions reports results in a summary of Abnormal Conditions. The doctor is instructed to record any abnormal conditions and the degree to which the condition would be a handicap to ordinary schooling (at age 7) or future employment (ate age 16). The variables used in the analysis indicate the whether the cohort member had 0, 1, 2, 3 or more chronic conditions at age 7 and at age 16.

The cohort members' cognitive abilities are measured when they are 11 years old. This variable is a standardized average of 4 test scores : Verbal, Non-Verbal, Reading Composition & Math.

Finally health status is measured from the self –reported health status at ages 33 and 41-42. To simplify analyses we grouped the five-score original scale into two categories: poor or fair and all others.

C. Analyses

The analyses of data will be carried out in two stages. In the first stage we describe results that pertain to intergenerational transmission of social class. The question we ask is the following: how much of the observed relation between paternal and offspring's social class is attributable to mechanisms involving early health status as measured by birthweight and number of chronic conditions at ages 7 and 11?

The second stage of analysis focuses on the social class health gradient at age 40 as a function of lagged social class at age 30. Here we ask the following question: what is the contribution of early health status to the association between lagged social class and health status?

In both stages the analysis proceeds according to the following steps: (a) we first present a summary of the main results obtained from the path model; (b) we decompose the total paths that account for the correlation between the main variables of interest and calculate the contribution of paths associated with early health status; and, finally, (c) we use the path model to simulate a population that initially has the characteristics observed in the BCS58 data and then is assumed to experience a life course governed by the path model. In the analysis of social stratification we estimate a mobility matrix that combines class of origin and class of destination and study its steady-state behavior. In the analysis of the social class health gradient we obtain a mobility matrix that combines social class and health status of origin with social class and health status of destination and, as before, we study its properties in steady state.

Does early health matter for adult social stratification?

Table 1 (panel a) displays the estimated regression coefficients predicting social class of destination as a function of parental social class of origin and parental health status. The relation

with parental social class is strong and statistically significant and suggests that maternal health (as reflected in maternal low birthweight) has no influence. As a curiosity with no strategic importance, we report also that ‘elasticity’ of offspring’s social class with respect to parents’ social class is about .30, close to the minimum of the range of estimated elasticities in the US between parental and offspring’s incomes!

Table 1 (panels a and b)

Table 1 (panel b) presents final estimates of a more complete model for social class of destination. Four features are worth highlighting. First, note that despite controls, parental class exerts a significant, albeit attenuated, effect on offspring’s social class. Second, measures of early health status have only marginal *direct* influence. Indeed, the only variable of some importance is the number of chronic conditions at age 11. Neither birthweight nor number of chronic conditions at age 7 matter very much. However, it should be noted (results not shown) that birthweight and number of chronic conditions at age 7 *do exert powerful effects on number of chronic conditions at age 11*. Thus, their influence on social class of destination is largely *indirect*. Third, educational achievement and early cognition have large and statistically significant effects on social class of destination. It should be remembered that the way we measure education (as a dummy variable reflecting whether or not an individual passes more than 5 O-level exams) is a sure way to downplay its effects. Most of the slack created by this rather coarse choice about the indicator of education is picked up by cognition. And yet, even under this very conservative scenario, educational attainment matters a great deal: an individual who manages to pass at least 5 O-level exams will rise about .60 in the rank of social classes (from 1 to 6). Fourth and finally, the only indicator of soft skills we introduce in the model (a score of early behavioral maladjustment) plays only a marginal role. Experimentation with other indicators of soft-skills (early height and a score of attractiveness) yield similar outcomes: soft-skill matter but their role pales relative to that of parental social class, cognition and education.

The second step of the analysis consists of decomposing the association between parental and offspring social class into components paths and, in particular, estimating the fraction of that association contributed by paths that involve early health. Given the set of estimated structural equation models the results we arrive at is that roughly 58 percent of the correlation between

parental and offspring social class can be accounted for paths involving early health status. This is fairly substantial but, as we show in the simulation exercise, is also misleading since it does not take into account population exposed to the events of interest: it only accounts for the magnitude of the estimated effects

The third step in the analysis consists of performing a more rigorous test using the estimated structural equations. We start with the individual members of the cohort for whom we have full information¹⁴ and simulate what would have been their class of destination assuming that they are exposed to the regime of transitions and attainment embedded in the structural equations. This simulation includes the role of chance as we utilize the residual variance for continuous variables and a simple Bernoulli process for discrete variables. The final product of the simulation is a mobility matrix cross classifying individuals by class of origin and destination. Once this matrix is estimated we add assumptions about fertility differentials by social class and can then study the long term behavior of an initial and arbitrary vector of individuals by social class. The resulting algebra is analogous to that of Leslie matrices in stable population. A full description can be found elsewhere (Keyfitz, 197x; Preston and Cambell, 1993; Lam, 1986).¹⁵

We can use the matrix results to answer a number of questions of interest. For example, given the estimated structural equation model, what would the steady state distribution by social classes be? The latter is independent of the initial distribution of individuals by social class, the one observed in 1958, and only reflects the effects captured by the structural equation model. Second, what proportion of the population remains in their class of origin? Third, what is the correlation between social class of origin and destination? Because we can manipulate the estimates of the structural equations (by eliminating selected paths) and the fertility differentials by social class (by altering the fertility gradient) the above stated questions can be answered under alternative scenarios. We choose the following: (a) a scenario where early health effects are as estimated, (b) a scenario where early health effects are set to zero, (c) a scenario where the effects of education and cognition are set to zero, (d) a scenario where the fertility gap between the highest and lowest social class is doubled.

¹⁴ See below our comment on the magnitude and impact of attrition

¹⁵ In all the exercises with matrices we assume complete homogamy. This ensures that we maximize the effects of both parental social class and original health status. Introducing homogamy would lead to downplay some of the effects we uncover.

Table 2 displays simple indicators summarizing the behavior of the mobility matrix. First, fertility differentials, at least within the range simulated, do not affect the results greatly. Second, a comparison of the scenario where effects are as estimated with one where the effects passing through early childhood health (birthweight, number of chronic conditions at ages 7 and 11) are set to zero leads to a reduction of 9 percent in the stickiness of those in the lowest class $100*(1-.147/.162)$ and a decrease of about 2 percent in the downward mobility of those at the top of the social class hierarchy. The contrast with a scenario where early child health effects are multiplied by five (Max Early Health Effects) is quite sharp: in these were the true effects the stickiness of the lowest class would be reduced by 46% $100*(1-.147/.272)$. By the same token the downward mobility of those at the top would be reduced by 11 percent or $100*(1-.450/.505)$. Examination of the effects of parental social class on offspring social class suggests that the contribution of early health status to such relation is quite small or about 2 percent ($100*(1-.298-.304)$). Were the early health effects multiplied by 5 the contribution would increase to about 9.5 percent $100*(1-.298/.326)$

In sum, early child health contributes to intergenerational transmission of social class but the contribution is small, possibly in the range 5 to 10 percent. This is not inconsistent with the rather large estimate of the contribution associated with early child health we obtained from decomposition of path coefficients: it should be remembered that the results from the matrix exercise combine the magnitude of effects from the structural equation model with the distribution of the population in various states. Therefore, strong effects may ultimately matter little if they only involve a small fraction of the population.

It is important to remember, though, that the magnitude of effects derived from the matrix exercise, regardless of how small they may seem, is comparable to that associated with other factors that have received close scrutiny in the literature. Furthermore, for reasons alluded to before and explored in more detail in the conclusion the magnitude of effects we estimate is, in all likelihood, grossly underestimated.

Does early health induce health selection affecting the adult socioeconomic gradient of health?

Table 3 (panel a) displays the results of estimating a simple logistic model with self-reported health status at age 41-42 as a dependent (discrete) variable with lagged social class at age 33 as a predictor. Unsurprisingly, the relation is strong and the estimated effect of social

class is highly significant: if one neglects cases with missing values, an increase of social class ranking leads on average to a 50% decrease in the probability of self reporting in poor/fair health. The gradient is steep and marked.

Table 3 (panel b) displays results of a more complete model, with controls for parental background and individuals' life cycle traits. Perhaps the most startling features of these estimates are the strong influence of LBW and the stubborn influence of parental social class (not maternal health status). Under the onslaught of large effects of cognition, that virtually sweep all the effects of educational attainment, the influence of lagged social class at age 33 disappears altogether. This confirms other findings in the literature that suggest that a large fraction of social class differentials in health are education-related. In our case, however, it remains to be explained the mechanisms through which LBW and parental social class continue to exert effects on offspring self rated health during adulthood.

The second step of the analysis consists of decomposing the association between lagged social class at age 33 and offspring self-rated health status at age 41-42 into components paths and, in particular, estimating the fraction of that association contributed by paths that involve early health. Given the set of estimated structural equation models the results we arrive at is roughly 48 percent, *that is, of the overall association between lagged social class and health status at age 41-42, about one half is accounted for mechanisms associated with selection into social classes.*

In the third step we estimate steady state matrices transforming an initial vector of population classified by social class and health status into one corresponding to n-generations ahead. As before, each 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.

We use the matrix results to answer the following: given the estimated structural equation model, what would the steady state distribution by social classes and health status be?. Second, what proportion of the population remains in their class and health status of origin? Third, what is the relation between social class at age 33 and health status at age 41-42? Because we can manipulate the estimates of the structural equations (by eliminating selected paths) and the fertility differentials by social class (by altering the fertility gradient) the above stated questions can be answered under alternative scenarios. We choose the following: (a) a scenario

where early health effects are as estimated, (b) a scenario where early health effects are set to zero, (c) a scenario where the effects of education and cognition are set to zero, (d) a scenario where the fertility gap between the highest and lowest social class is doubled.

Table 4 displays the most important results. First, in the high fertility differentials scenario we see that p_1 , the proportion of the population born into the lowest social class and poor health status who are stuck there during adulthood changes from .052 to .042 when effects of early childhood health are set to zero. This is a relatively large change (.19) for a relatively small quantity. The same (but in the reverse direction) occurs when we compare p_2 , the proportion of high class individuals in poor health who remain there. When the effects of early childhood health are exaggerated (scenario of Max Early Health Effects) the changes are sharper and quite large but only for p_1 , not for p_2 .

An interesting quantity displayed in Table 4 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 -.150 but when early health status effects are set to zero, the gross effects fall to .134, an 11 percent decrease: this can be legitimately interpreted as indicating that, in our data and for this particular cohort at least, the pathways associated with early health that contribute to the correlation between adult health status and social class account for not more than 11 percent of the actual relation. This is not a large contribution, at least not large enough to debunk the idea that the bulk of the observed association between social class and health has more to do with the effects of social class traits than with health selection processes.

This estimate is entirely consistent with the rather large estimated contribution of early health selection obtained from decomposition of the path model: the latter only involves the magnitude of effects whereas the matrix simulation involves both, the magnitude of effects and the relative size of the population characterized by conditions that may lead to either bad or good health or lower or higher social classes.

Table 4

Finally, it is worth noticing how similar the estimates of the stratification analysis and the health gradient analysis are: early health plays only a modest role in explaining intergenerational inequality. And, as it should be, it also plays a proportionally small role in explaining the socioeconomic gradient of adult health status.

V. SUMMARY AND CONCLUSION

The main goal of this paper is to address in a systematic way the question of whether or not health selection effects have much or anything to do with the adult socioeconomic gradient of health (and mortality). In order to answer the 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. The framework led way to the formulation of a simplified model which was then estimated using a very unique data set. The final estimates from a structural equation models were then transformed and used as input in a Monte Carlo simulation study of social and health mobility. This was possible by estimating (steady state) mobility matrices yielding embedded statistics that can be used to address the original question posed. Our findings can be summarized thus:

- i. early child health has an important, albeit not the most important, role in the determination of adult social class positions. Of the overall relation between parental and offspring social class, early health might account for no more than 10 percent. This is not a trivial amount and is comparable to the contribution of other factors considered broadly in the literature
- ii. early childhood health plays only a small and marginal role as a mechanism through adult socioeconomic gradients are produced. In particular, health selection effects in the sample we use cannot account for more than 10 percent of the overall association between lagged social class and adult health status.

Before closing, it is worth pondering two important caveats. The first is that 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.

The second caveat leads to the opposite inference: the estimated effects we retrieve are likely to be lower bounds. This is because of three reasons:

- (a) the sample we use experienced considerable attrition and the latter did not occur randomly. Indeed, those who belonged to the lowest social classes and the poorest health status are more likely to have attrited. If, through multiple imputation procedures we are able to utilize rather than discard these cases, it is more than likely that the effects of early childhood health will increase
- (b) The measures of childhood health are exceedingly coarse. Were we in possession of more fine tuned measures, including physical and psychological impairments, it is likely that the relationships would be stronger than what we found.
- (c) in countries such as the UK around 1950, the conditions of the most deprived portion of 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, insufficiency of micronutrients, are rampant. In these contexts the room for early health status to leave a deep imprint is 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: Simplified path diagram representing the relations between early child health conditions and adult social class

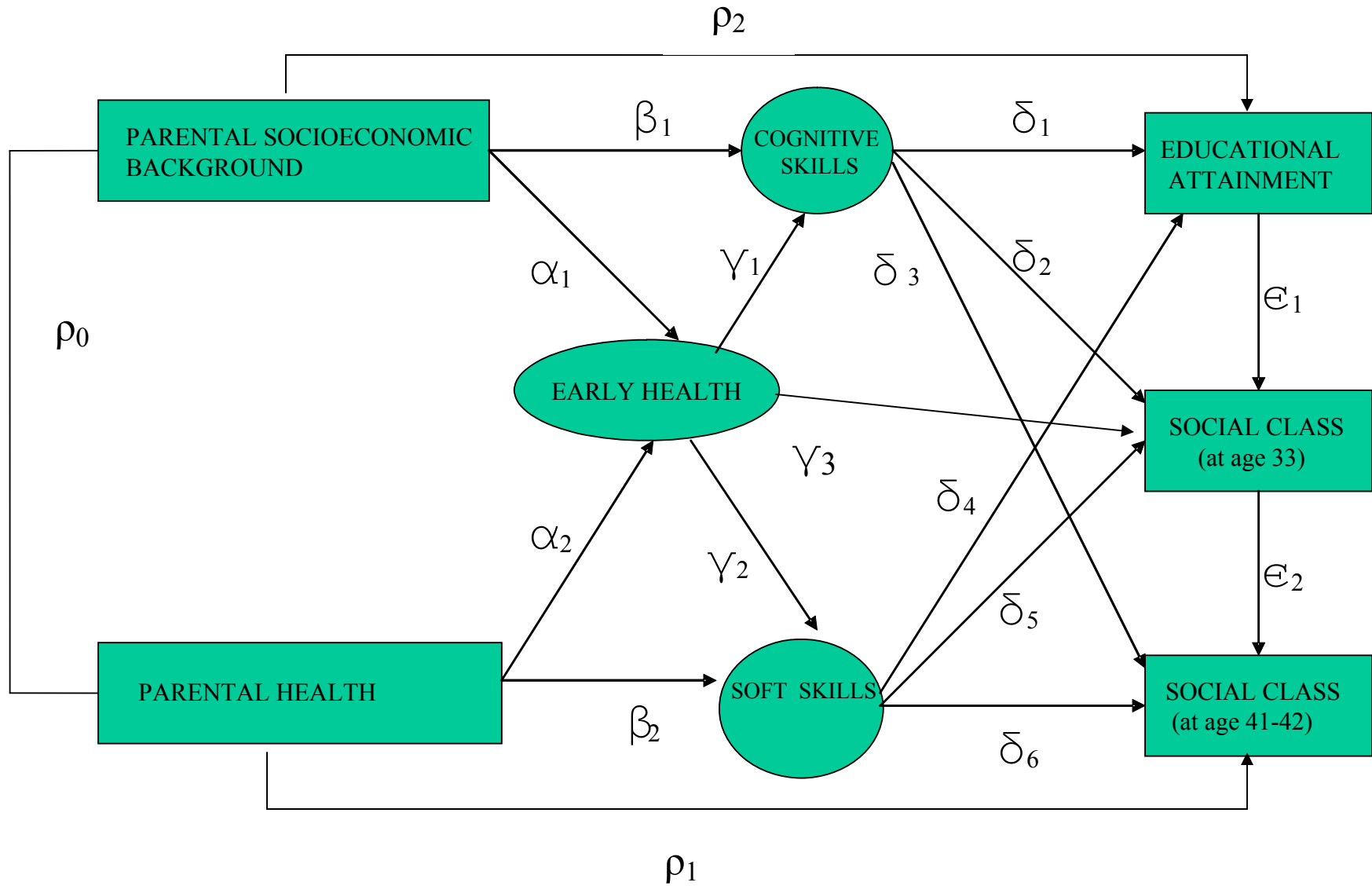


Figure 2: Simplified path diagram representing the relations between lagged social class and adult SR health states at age 41-42.

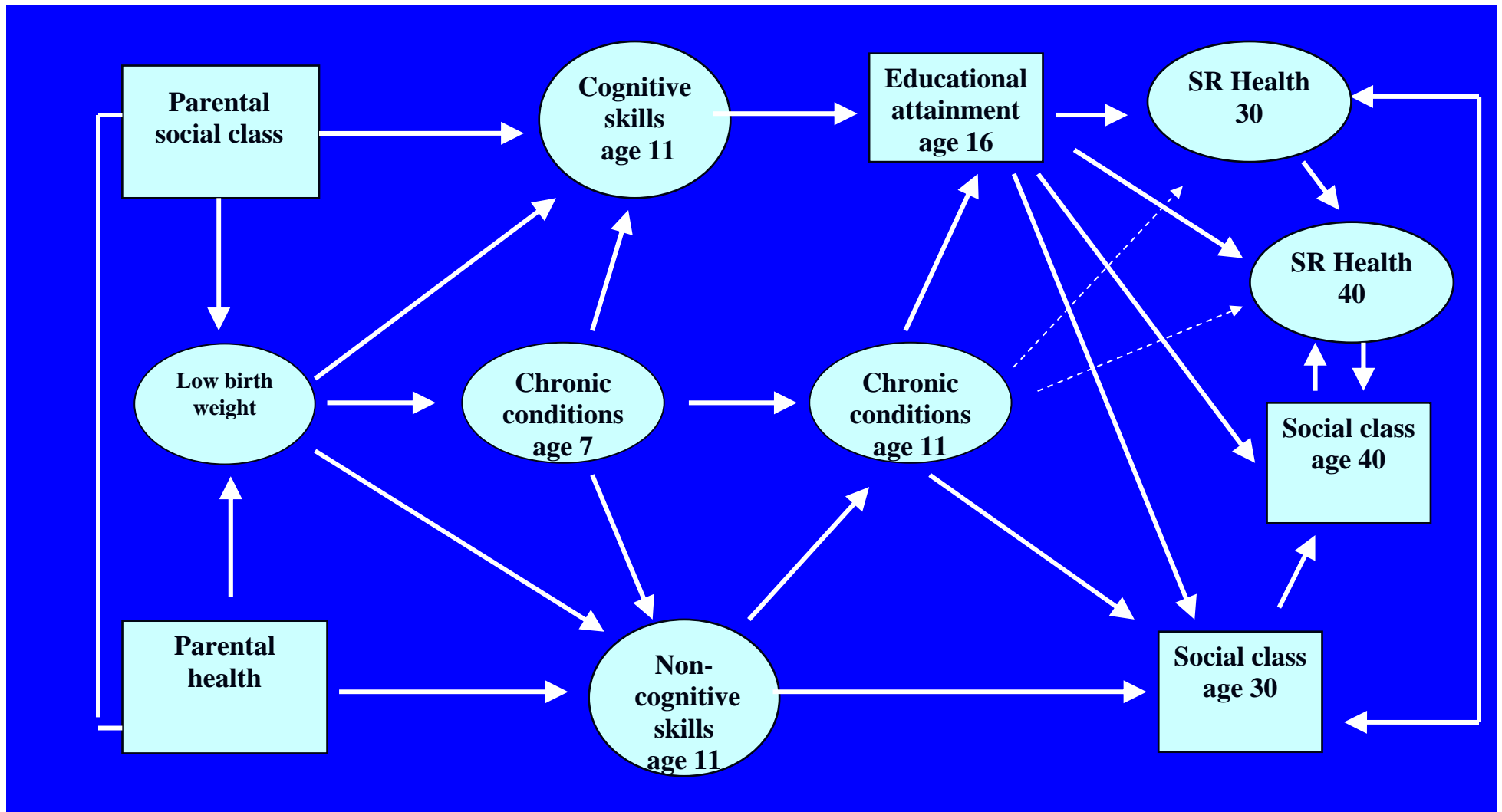


Table 1: Estimates of Effects on Offspring Social Class Position at age 41-42

Panel a: Simple Linear (OLS) Models

Variable	MODEL I		MODEL II	
	Estimate	SdEr	Estimate	SdEr
Constant	3.112		3.111	
Fathers Social Class	.284	.022***	.289	.021***
Maternal Health	-.018	.117	-	-
N= 1999				
R-Squared	.055		.054	

Panel b: A more complete (OLS) model

Variable	Estimate	SdErr
Constant	3.498	.087
Father's Social Class	.127	.022***
Maternal Health	-.012	.110
Low Birth Weight	-.003	.141
# Chr Cond Age 7	-.156	.089*
# Chr Cond Age 11	-.013	.053
Cognitive score Age 7	.459	.037***
Maladjustment Score Age 11	-.027	.017
Educational Attainment	.558	.072***
N	1999	
R-Squared	.549	

Two Tailed tests: * p<.05	**p<.001	***p<.0001

Table 2: Selected Indicators from simulated (steady state) social class mobility matrix ^{(a), (b)}

Scenario	P1	P2	BETA
High Fertility Differentials			
Estimated Parameters	.162	.495	.304
No early health effects	.147	.505	.298
Max Early Health effects	.272	.450	.326
Low Fertility Differentials			
Estimated Parameters	.162	.507	.302
No early health effects	.145	.508	.278
Max Early Health effects	.219	.038	.326

P1= Proportion born in Lowest social class who remain
P2=Proportion born in Highest social class who remain
BETA=linear regression coefficient of offspring social class at age 41-42 on parental social

(a) In the scenario with low fertility differentials we used the observed differentials; they are estimated as follows:

$$CEB(\text{class } j) = A * \exp(r * \text{class } j)$$

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 ration 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.

Table 3: Estimates of Effects on Adult Self Reported Health at Age 41-42^(a)

Panel a: Simple Logit Models

Variable	Estimate	SdEr
Constant	-1.503	.170
Social Class Age 33	-.136	.052**
N		1999
LL		-13904
Information Criteria		
Akaike (AIC)		27850
Bayesian (BIC)		27967

Panel b: A more complete logit model

Variable	Estimate	SdErr
Constant	-1.695	.275
Father's Social Class	-.107	.058**
Maternal Health	.011	.287
Low Birth Weight	.811	.282***
# Chr Cond Age 7	-.013	.202
# Chr Cond Age 11	.139	.118
Cognitive score Age 7	-.451	.097***
Maladjustment Score Age 11	.119	.044**
Educational Attainment	.111	.215***
Social Class Age 33	-.011	.055

Tabel 3 Panel b: Cont.

N	1999	
LL	-1811	
Information Criteria		
Akaike (AIC)		3646
Bayesian (BIC)		3714

Two tailed tests : * $p < .05$ ** $p < .01$ *** $p < .001$

(a) The dependent variable attains the value 1 if individual self reports health status as poor or fair

Table 4: Selected Indicators from simulated social class and health status mobility matrix ^{(a), (b)}

Scenario	p1	p2	gamma
High Fertility Differentials			
Estimated Parameters	.052	.045	-.150
No early health effects	.042	.045	-.134
Max Early Health effects	.215	.047	-.360
Low Fertility Differentials			
Estimated Parameters	.051	.048	-.152
No early health effects	.038	.043	-.138
Max Early Health effects	.219	.038	-.362

p1= Proportion born in Lowest social class and poor health status who remain
p2=Proportion born in Highest social class and poor health status who remain
gamma=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:

$$CEB(\text{class } j) = A * \exp(r * \text{class } j)$$

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 ration 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.

