Center for Demography and Ecology

University of Wisconsin-Madison

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Alberto Palloni

Carolina Milesi

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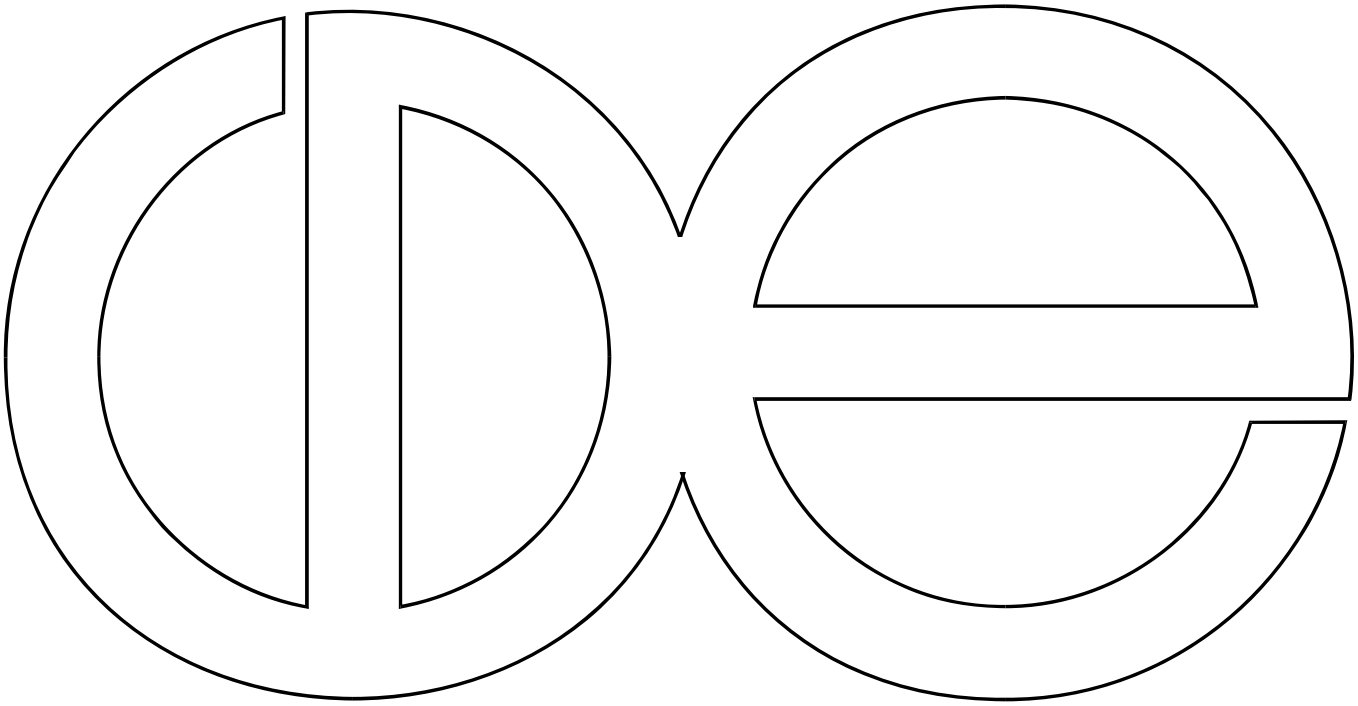

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THE ROLE OF EARLY HEALTH STATUS IN SOCIAL STRATIFICATION

A PRELIMINARY INVESTIGATION

ALBERTO PALLONI

AND

CAROLINA MILESI

CENTER FOR DEMOGRAPHY AND ECOLOGY
UNIVERSITY OF WISCONSIN-MADISON

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ABSTRACT

The persistence of social class inequalities is a salient regularity in modern societies. In this paper we argue that there are mechanisms that link early health status and late socioeconomic achievement. Our discussion is based on well-established results in the social stratification literature, on recent investigations about determinants of wages and income in labor economics, and on life-course theories of adult health status. We suggest that early health status may be a non-trivial mechanism for the intergenerational transmission of social and economic inequalities. If so, it is also responsible for a process of health selection that may partially account for observed health and mortality differentials during adulthood. Our empirical work consists of providing new estimates from NLSY-C and ECLS-K of the effects of early health status on market and non-market traits related to wages. We find that the effects are non trivial and, in many cases, are large and significant. Although this does not prove that early health status exerts a strong influence on social and economic inequalities nor that it is a contributor to the heritability of social class positions, it does provide support for the idea that it constitutes a mechanism that deserves more attention than what it has been given to it in the past.

INTRODUCTION: HEALTH INEQUALITIES AND THE PRODUCTION OF SOCIAL CLASSES

There are two 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. The second is the persistence of earnings and income inequalities in social and economic contexts where they are increasingly resistant to explanations invoking differential endowments of market-valued skills or of rents extracted from inherited assets–the standard explanations suggested by neoclassic labor economics.

One objective of this paper is to formulate a framework resting on a central proposition, namely, health inequalities during early childhood is a non-trivial contributor to the persistence of economic inequalities among adults. If this is so, two consequences follow, one relevant for social stratification theories and the other for theories of health and mortality. The first consequence is that an important mechanism reproducing social classes is the differential allocation of superior health status that occurs early in life. Thus, by influencing market and non-market related endowments, early health status contributes to the reproduction of economic inequalities. The second consequence is that further advances in the study of health and mortality disparities require that we recognize health selection processes whereby different health endowments result not only in different exposure to ill-health and mortality but also in differential ability to access social class positions throughout the life of individuals.

A second objective of the paper is to offer minimal empirical evidence, and to suggest means of acquiring additional proof, for the importance of health status as a determinant of earning capacities at adult ages. This turns out to be a rather formidable task not only because of the amount of information required to do so, but also because of important methodological pitfalls that are difficult to avoid in one single swoop.

The paper is in four sections. In the first section we lay out the essentials of our task and establish the most important conditions for making the undertaking meaningful and possible. In the second section we review findings regarding intergenerational transmission of inequalities and identify weaknesses in the conventional toolkits deployed to explain the process of interest. We emphasize recent developments regarding the contribution of market and non-market related factors to individuals' earning and asset accumulation capacity. We argue that the most influential market-valued factors (educational attainment) and several non-market factors (ability

to negotiate, perseverance, social contacts; motivation, effort invested) can be plausibly connected to health conditions early in life. In the third section we identify mechanisms through which health status may affect earnings and formulate a simple model to account for the role of health as a determinant of adult economic inequalities. In this section we highlight theories establishing a connection between health conditions late in life and health status during early childhood and adolescence. Finally, in the fourth section we provide an empirical illustration of the effects of early childhood conditions on early cognitive ability.

IS HEALTH STATUS IMPORTANT FOR SOCIAL STRATIFICATION?

A simplified notion of social class

We begin by summarily disposing of a conceptual difficulty. To simplify terminology, we will speak of social classes to refer to social positions which individuals may access and occupy for finite periods of time. Individuals belonging to a social class will be distinguished by one key attribute, namely, the appropriation or right of collection of rewards in the form of occupational prestige (a socially constructed quantity), earnings from labor, and rents from assets which, in combination, constitute the sources of personal and family income. Both occupational prestige and earnings are tightly, though not perfectly, associated with education, a proxy for the presence of rent-extracting skills. These two reward systems, prestige and factor payments (or pertinent proxies such as education), 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. By the same token we eschew discussion regarding other properties of a social class system that are superimposed on the reward system. Thus, we ignore dimensions such as legal property of means of production, rights of administration and allocation of resources in the production process, and matters of control and authority within and outside the job place. These dimensions may conflict or be inconsistent with the class reward system as defined here. If so, they should be included in a more complete discussion about the relation between health and social stratification. For the purposes of this paper though, ignoring these added complexities of class systems makes the problem more tractable. An important and immediate advantage stemming from this simplification is that we

can delimit the term social class to refer to individuals grouped by attributes such as income, education, scores in a standard prestige or socioeconomic scale.

Social stratification and race and ethnic groups

Despite the fact that there is always some overlap between social classes and ethnicity or race groupings, the relations between these two dimensions of stratification are seldom simple. Thus, income and wealth disparities across ethnic or race groups may persist even though the distribution of individuals by social class within each group is accounted for. This indicates that some or all mechanisms of reward allocation are partially dissociated from the contribution of labor or other factors entering the production process. If so, the accounting we suggest below needs to be augmented to either include a complete rendition of those mechanisms, or to identify precisely the social and economic contexts within which deviations from normal reward allocation occurs. The strategy we suggest can initially be applied without regard for race and ethnicity. But if the component processes are believed to be different across racial or ethnic groups, a more complex accounting --whereby each ethnic group is treated separately-- is called for.

Conditions of possibility

Our ultimate goal is to integrate findings in the literature on adult health and mortality disparities with the social stratification literature by building a case for the importance of early health status as a non- trivial determinant of social class and social mobility. Such a case, and the feasibility of a model that includes health as a determinant of social stratification, must be made by showing the plausibility of three conditions. Before we establish the first condition, we must identify the most important determinants of class differentials using a relevant metric of social stratification (reward system) such as earnings (wages, rents), occupational prestige or scores of socioeconomic achievement. Some of these are associated with concrete, well measured market-related factors such as cognitive skills and educational attainment, whereas a few are associated with more vaguely identified traits such as personality characteristics or social connections which, as a rule, are not recognized as renting-producing factors in a standard production function. The **first condition**, then, is *that there must a relation between health status early in the life of individuals, before they complete their training careers, and the acquisition of market*

and non-market related traits that entitles them to rewards in the occupational structure. This is a necessary condition.

The **second condition** is that *allocation of health status early in life does not occur at random but is itself a function of the social class position individuals occupy by virtue of their membership in a social class of origin.* This simply involves showing that membership in a particular social class, and the environment entailed by it, contributes significantly to early health status. It is a necessary condition for if health status early in life were randomly allocated with respect to social class of origin, it could not possibly contribute to the intergenerational transmission of inequality.

The **third condition** is that *adult health status and mortality is either directly determined by early child health conditions and/or by attributes associated with the social class to which the adult individual belongs.*

Given the scope of this paper, only the first two conditions seem relevant. Indeed, if the first two conditions are satisfied, social stratification is partially influenced by attributes directly related to early health. In this case, theories about social stratification should incorporate a health dimension.

The third condition is relevant only if one's objective is to understand how selection into social classes through early health conditions affects cross sectional health and mortality differentials by social class. If all three conditions are satisfied then not only is the social stratification system partially dependent on health attributes, as we will try to illustrate here, but also adult differentials in health and mortality depend on attributes associated with social classes as well as on the impact that health status has on accession to social positions. When this occurs, social class attributes may determine health and mortality but also, and more importantly, health status also determines social class (see Figure 1) .This is the situation the literature on health and mortality differentials associates with "health selection" a mechanism invoked to explain health and mortality differentials across social classes. Rather than being a surreptitious reintroduction of social Darwinism, it may well be an important dimension to understand social hierarchies, and more than a plausible accounting of adult health and mortality disparities.

Figure 1 about here

In the next section we attempt to establish the first condition of possibility. We highlight findings and theories of social stratification and labor economics that connect determinants of wages and income with early health status.

TRANSMISSION OF ECONOMIC INEQUALITIES

Three features characterize the US and possibly most of the modern industrialized countries. First, economic inequalities are as persistent a feature as it is the regular health and mortality differentials by social class reviewed before. In the US, for example, the income gap between 1978-1980 and 1994-96 widened as the inflation-adjusted incomes of the wealthiest 20 percent of families with children increased by 30 percent while those of the poorest 20 percent decreased by 21 percent (Center for Budget and Policy Priorities, 1997). Although inferences are sensitive to the choice of units of analysis and outcome studied, other research confirms the persistence and possible augmentation of inequalities in the US in the last two decades of the twentieth century (Mayer and Jencks, 1993; Freeman, 1997; Morris and Western, 1999).

The second feature is that the magnitude of intergenerational transmission of economic status is considerably greater than what it was once thought to be. The third feature is that current income inequalities cannot be accounted for by standard earning equations, those including measures of market-valued skills such as cognitive abilities, educational attainment and job experience. It appears as if total income, but specially earnings from labor, are strongly dependent on family related conditions that shape individuals' environments early during their life. These early conditions confer individuals a number of traits that increase their value in the market place, but none of them is well-captured by traditionally measured skills and abilities.

On intergenerational transmission of inequality

In a compelling and enlightening review of the state of research in this area, Bowles and Gintis (2000) show that while intergenerational transmission of inequality has increased, our understanding of it has remained nebulous. Their work rests on a classic treatment of inheritance of traits with the modification that the outcome measures are representing rewards in the social stratification system. Because a variant of this framework will be deployed in later sections of the paper, we review it briefly.

The social class system is conceived of as a continuous gradation of positions. This differs somewhat from treatments of social mobility where social classes constitute discrete entities, but is similar to the tradition in sociology that uses occupational prestige, SEI scores, or occupational education as a metric for rewards (Featherman and Hauser, 1978; Blau and Duncan, 1967; Hauser and Warren, 1997). In this framework inheritability or intergenerational persistence of a characteristic is defined as the correlation between the natural log of measures of that characteristic. In a standard treatment for income we will have[1]

$$\ln Yo = \alpha + \beta \ln(Yp) + \epsilon \qquad (1)$$

where ln Yo and ln Yp are the natural logs of income in the generation of offspring and parents respectively, α is constrained to be (1-β) Mean(ln(Yp)), β is the corresponding elasticity and ϵ is (0,1) normally distributed error term. The correlation between log of income for parents and offspring is

$$\rho = \beta * (\sigma p / \sigma o) \qquad (2)$$

where σp and σo are the standard deviations of income distributions. These are also unit-free measures of inequality in the distribution of parents and offspring income respectively. When income inequality does not change across generations, $\rho = \beta$ and the elasticity of offspring income relative to parental income is a robust measure of persistence and "stickiness" of income classification across generations. If inequality is increasing, the intergenerational correlation will decrease as long as the effect of parental income on offspring' income remains invariant.

An analogous treatment can be used with any other economic-related characteristic such as wealth, earnings, consumption, education, etc. (Solon, 1992). In the sociological tradition, the reward or outcome of interest has been discrete occupational categories in the "mobility table" approach, and occupational prestige, and SEI scores in the "regression" approach (Warren and

[1] This model assumes that generations other than parental have negligible influence in income determination. This is consistent with empirical research (Warren and Hauser, 1997).

Hauser 1997). Measures of intergenerational transmission and 'stickiness' of occupational prestige analogous to those suggested above, or generalizations of it based on matrix invariance can also be derived (Featherman and Hauser, 1978). In the remainder of the paper we follow the economic tradition and use income as our relevant metric for rewards. However, in general, conclusions regarding trajectories of intergenerational mobility and our knowledge of determinants of rewards apply irrespective of the outcome of interest. [2].

Income is the sum total of labor earnings and returns to assets. In turn, labor earnings are a function of conditions that enhance productivity, namely, learned skills (educational attainment and training), genetically inherited abilities partially embodied in cognition scores (such as early cognition scores, AFTQ), and environmental enhancing conditions (normally associated with parental education, type of family of origin, and parental investments in children). Total returns to assets are a function of the size of bequests and market rents to assets. However, because few individuals receive any income from assets and fewer still are endowed with inherited property, this factor can be ignored as an important contributor to intergenerational persistence of income even though, among the very rich at least, inheritability of wealth is substantial (Bowles and Gintis, 2000).

Having thus significantly simplified the problem, we now focus only on earnings determination. To explain inheritability of earnings we need to examine three mechanisms: cognition, educational attainment and training, and environmental enhancing factors. Cognition, educational attainment, and training are the traditional factors associated with "human capital." They are partly a function of inheritance of (genetically transmitted) abilities and partly a function of skills produced through learning and socialization. Environmental enhancing conditions refer to non-market related factors that may receive market rents. Because it will be useful in a later section we represent the potential relations in a path diagram displayed in Figure 2. This is a modified version of Bowles and Gintis path diagram (Bowles and Gintis, 2000, p. 9) that explicitly includes parental education side by side with parental cognition as a determinant.

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[2] This is not entirely accurate as the previously cited work by Featherman and Hauser (1978), recent work by Hout (1984), and by Grusky and DiPrete (1990) and Diprete and Grusky (1990) suggests an attenuation and dilution of the intergenerational link.

As in Bowles and Gintis' model, parental characteristics carry the subscript p and offspring' characteristics carry the subscript o. The Greek letters represent path regression coefficients -- regression coefficients of suitably standardized variables with mean zero and unit standard deviations. Alternatively, they could be associated with the partial derivatives in the structural equation representation of each dependent variable. The β's connect parental characteristics, the α's connect offspring characteristics, and the γ's link cross-generational characteristics. The variables C are for early cognitive levels, S for levels of educational attainment, Y for total earnings, and G for genetic endowments[3].

Following standard rules of path analyses we can derive measures of total effects and correlations linking any two variables and decompose these into contributing parts. Figure 2 only displays the contribution of a subset of all possible paths. Of great interest is the one associated with genetic inheritance of cognitive skills. As it turns out, offspring's cognitive skill exert a powerful influence on their earnings (α3~.15). In addition, the evidence suggests that the inheritable component of cognitive skills is non-trivial (the h-r-h path connecting Co and Cp in the figure) .Yet Bowles and Gintis estimate that the overall path through genetic endowment alone cannot contribute more than .05 to the correlation between parental and offsprings' income. Since our path diagram includes parental education as well, our own estimate for the contribution of this path should not be more than .10, twice the magnitude of the estimate obtained by Bowles and Gintis, but still a rather modest quantity.

If not through familial wealth or cognition, can individuals' own educational attainment be the conduit for inheritability of earnings? The answer is that even controlling for cognition, school quality and education attainment, the estimated **direct** effects of parental earnings on offspring' earnings (β2) is quite strong as it accounts for between two fifths and one half of the

[3] We refer to early cognitive levels although most of the literature uses cognitive levels assessed during late teens. These two are only imperfectly correlated and a better choice would have been to include them both. However, to simplify presentation we use only one of them, those less affected by schooling and school environments. The price we pay is that we must make the rather questionable assumption that the linkage between cognitive skill and educational attainment, on the one hand, and earnings, on the other, is identical to the one empirically established between these two outcomes and cognitive skills assessed at later older ages.

gross correlation between parental and offsprings' measures of economic success (the sum total of all paths linking Yo and Yp via education and cognition outcomes).

Bowles and Gintis conclude their review with a rather bleak assessment of standard models: "understanding the process of intergenerational status transmission is hampered by two habits of mind. One is thinking of the [...] process as a literal handing-down of such things as good genes and material wealth. The other is limiting the benefits handed down to skills" (Bowles and Gintis, 2000, p. 17).

It is possible that what is passed down directly to descendants is something less obvious, such as behavioral influences or social and cultural environments within which offspring are socialized from early ages (Heckman et al 1999; Lubotsky, 2001). Moreover, there might be traits that persist across generations that have powerful effects on earnings which are not even minimally reflected by conventional human capital variables. Health conditions are among these. Their effects are partially captured in the direct path from parental to offspring's earnings ($\gamma 1$ in Figure 2) but other paths are, of course, possible.

Figure 2 about here

Unexplained earning differentials: market and non-market factors

In the foregoing we established that labor market success, as reflected in income and earnings, appears to be "transmitted" from parents to children, and that this transmission is not confined to benefits of educational attainment, bequests, or factors such as cognitive ability. In this section we review recent findings regarding factors that affect earnings.

As indicated before, the direct path from parental to offspring' earning is robust to model specification, and remains important even after controlling for a host of market-relevant offspring' traits (Mulligan, 1997; Bowles and Nelson, 1974; Bowles et al., 2000). Accumulated research findings on earnings determinants does not suggest that standard human capital variables are unimportant but rather that after fully accounting for them there is substantial unexplained variance in earnings. Part of the residual variance vanishes after controlling for race, but even within-race inequalities remain largely unexplained by human capital endowments. Furthermore, the variance explained by race remains a "black box", variously attributed to

discrimination, access to social networks and other constraints with effects that vary across labor markets.

The effects of standard labor market characteristics

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 skills. Traditionally, the 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 (a) 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) and (b) their effects are not easily explained with standard interpretations, namely, some of their effects may be due to high correlation with other, non-cognitive traits (Bowles and Gintis, 1976). We address each of these two failures in turn.

Incompleteness: Inclusion of parental earning and other parental characteristics, as well as race and ethnicity, serves the purpose of reducing unexplained variance after accounting for measures of skills and abilities This success in reducing unexplained variance of earnings is clouded by two features. First, the effects of race and parental income do not help to clarify the process as the precise mediating mechanisms remain obscure. It is unclear what is it about parental income that determines offsprings' earnings and also what is it about race that may place some individuals in a disadvantageous (advantageous) position. Second, the residual variance is associated with characteristics that are "seemingly irrelevant" (Bowles et al, 2000) to the production process and do not enter or have no precise role in standard accountings of economic productivity and factor payments. Among these characteristics one finds adult obesity, obesity at age 16, adult height, height at age 14-16, physical attractiveness, cleanliness, and other conditions of child rearing environments.

Interpretation: Effects of schooling in particular are difficult to account for and remain strong even after controlling for cognitive abilities, school quality, and curricular content (Bowles and

Gintis, 1976; Bowles et al., 2000). It appears that benefits conferred by additional schooling are associated with skills other than those traditionally identified as enhancing productivity in the labor market. Further, educational attainment itself, the most important of conventional human capital indicators, exerts effects that are hard to interpret using standard explanations invoking credit constraints (Heckman and Rubisntein, 2001; Heckman, 2000). The same applies to measures of cognitive ability. It is likely that these effects owe more to environments that shape ability, transform outlooks, and modify motivation than to school curricular contents or actual innate cognitive skills (Heckman, 2000; Guo and Stearns, 2002)

Why should unconventional traits matter as predictors of earnings? What exactly are they, what are they useful for, and how are they acquired?

The effects of non- labor market endowments

Although microeconomic models that include unconventional traits are not standard fare, they do exist and do shed light on conditions under which their demand may grow thus fostering mechanisms to reward those who possess them.

The most important insight comes from literature on microeconomic markets with asymmetric information indicating that the employment relationship is contractually incomplete and that there are important costs associated with enforcement of contracts (Bowles et al., 2000). To lower enforcement costs in such contractual relations, employers may choose to reward "incentive enhancing preferences" (IEP), namely, 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 efficiency, perception of being in control, low fatalism, ability to function in groups and establish social networks. Many of these traits are learned or acquired outside school and other formal settings. Others, as suggested recently by Farkas (Farkas, 2003), may be part of a set of habits with which individuals from different social classes are endowed. These traits are the result of prolonged learning in the sense that they are shaped by processes that start early in life and follow variable trajectories thereafter. They are not result of a limited stay in high school or of transient participation in job training settings (Heckman, 2000).

The evidence suggesting that IEP are important contributors to human capital, and can command payments as other production factors do, is somewhat recent and does not yet amount

to a well established literature. However, the findings appear to be so ubiquitous that they are leading to important reexamination of standard human capital theories and to revisions of the foundations of public policy. In a sweeping account of conditions that could improve 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).

The evidence for this "shadow" reward system comes from very different traditions. For example, Jencks and colleagues' broad survey of determinants of economic success (Jencks, 1979) identifies traits such as industriousness, perseverance, leadership, and study habits as having important effects on earnings and occupational status independently of parental background, cognitive test scores and educational attainment. Duncan and Dunifon (1998) show the importance of traits measured (and acquired) years before observation for individuals' current earnings. They show that these traits, and not cognitive abilities, account for a significant part of the effects of measured educational attainment. Osborne (2000) also shows similar results and, in particular, illustrates that personality characteristics (fatalism, outward orientation) have significant effects on earnings. Heckman and colleagues have completed numerous studies demonstrating that market earnings are associated not so much with cognitive skills or educational attainment but with markers of behavioral and personality problems that prove to be wage-reducing (enhancing) (Heckman, Hsee and Rubinstein, 1999; Cameron and Heckman, 1993). Lubotski (2001) argues that there is strong evidence indicating that a cascading process prevails whereby early failures to rank well in cognition scores leads to higher subsequent disadvantages, a confirmation in reverse of Heckman's idea that "skills beget skills." Finally, note that Neal and Johnson (1996) suggest that much of the Black-White adult wage gap disappears for women and is sharply reduced for males when one accounts for factors shaped early on in life, the likely outcomes of family background and early school contexts.

In summary, there is an emerging body of literature suggesting not so much that classic labor economics is altogether wrong but rather incomplete. The theoretical incompleteness is

this: *there are social and economic contexts under which payment to factors of production will not follow patterns expected by classic microeconomic models, which rest on strong assumptions about equilibrium, namely, perfect information, exogenous efforts, and no search or enforcement costs. Given conditions defined by legislation, tax systems, geographic variability, or barriers to mobility of labor, one or more of the equilibrium conditions may be violated. When this occurs, workers' traits not conventionally associated with skills will get rewarded, as these correct and adjust for the consequences resulting from deviations from equilibrium and/or from asymmetric information.* Furthermore, it is conceivable that even within scenarios that do not deviate significantly from conventionally assumed ones, the acquisition and use of market skills may be contingent on exposure to conditions that enhance them and that exert their own direct influence on earnings. These conditions are characteristic of environments within which individuals are socialized during their formative years and their influences are neither offset nor greatly modified by formal education and job training later in life.

ENDURING EFFECTS OF EARLY CHILDHOOD CONDITIONS

We argue that early childhood health status is a relevant, albeit one among many others, determinant of adult earnings and social mobility. Extant evidence suggests that it may also be an important factor exerting direct influence on adult health and mortality. For our purposes it suffices to show that early health status exerts demonstrable effects on social and economic accession. Most unlikely, some of these influences are direct. More likely, the effects are indirect and belonging to one of two possible types. First, early health status may determine individuals' health status during formative years, adolescence and adulthood, and through this could have an impact on social and economic status. Alternatively, early health status exerts a direct influence in acquisition of skills and abilities relevant in labor markets that are in part a function of earning capacity. If, in addition to the presence of any of these two mechanisms of influence, early childhood status is affected by social class of origin (see below), we have in place all the elements for a process whereby accession to social classes and opportunities for mobility are passed on from one generation to the next.

In the section that follows we briefly review the evidence for the operation of the direct and first type of indirect effect. In the subsequent section we review evidence for what we

consider the most important path of influence, namely, through the acquisition of skills and abilities.

Paths of influence: direct and indirect effects of early childhood health status

Direct effects of early health on adult socioeconomic status

Does early childhood health status exert a direct influence on adult socioeconomic achievement? Some findings in the literature do suggest a positive answer. For example, results from the 1946 British cohort study reported by Wadsworth (Wadsworth, 1986; 1991; 1999) suggest 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; Rahkonnen et al., 1997; Behrman and Rosenzweig, 2002). With few exceptions these findings are somewhat fragile as are those that relate health status in early adolescence to mature educational attainment (Koivusilta, et al., 1995; 1998). In all likelihood direct (net) effects appear to exist as our ability to identify and measure all mediating paths is limited.

Indirect effects: how early health affects shapes adolescent and late health

In standard treatments, adult health (and mortality) is largely interpreted as the result of living conditions experienced during adulthood. But over the last half century, and more rapidly during the last decade, a flurry of research has also pointed to the possibly important contribution of differences in characteristics acquired farther in the past, including experiences that may have occurred during infancy and early childhood. The record of empirical evidence for these protracted effects is scattered and uneven, some of it is fairly robust and convincing, but most of it is largely suggestive and requires considerably stronger theorization (Schaffer, 2000).

A first set of mechanisms that link early exposure to later health operates when early events or experiences alter the normal physiology and functioning of vital organs in a permanent way thereby significantly changing health and mortality risks throughout the life of individuals or, alternatively, conferring disadvantages that will be manifested only later in life.

The evidence available to validate this conjecture is fragmented. What we have are partial results for adult populations in the US, the UK and Scandinavia. For example, there is evidence for the existence of mechanisms whereby early-life environments, including conditions *in utero*,

exerts influences on cellular growth, tissue development and ultimate physiological status and functioning of key organs, such as heart, lungs and kidneys. The crucial point is that such effects may not be seen for a long time after the causative processes are completed. Thus, impaired placental growth development, poor maternal nutrition and extreme low birthweight, are believed to be implicated in the onset of coronary heart, non-insulin dependent diabetes, lung disease, chronic bronchitis and immunocompetence (Ben-Shlomo and Kuh, 2002; Barker, 1998; Lucas, 1991; 1994; Thurlbeck, 1992; Fall et al., 1998). On the other hand, injuries and disabilities caused by the early onset of some illness, complications generated by rheumatoid fever, injuries due to violence, and impairments caused by substance abuse also lead to lifelong shifts in individuals' health (and mortality) risks.

Exposure to unfavorable conditions *in utero*, and also during the first year of life, modifies the growth of brain tissue and through it alters the functioning of one or more neuro-physiological centers of hormonal balance and activation. These, in turn, will affect behaviors, motivation, individual choices, resilience, frailty, and immune status (see the collection of essays in Keating and Hertzman, 1999). These mechanisms too may induce a wholesale shift in lifelong health (and mortality risks) of individuals exposed to them. But in this case the connection between the causative agent and the outcome is more remote and considerably more mediated than the processes suggested by Barker (1998).

Furthermore, research on the effects of "allostatic load" offers an alternative conduit through which early exposures may be felt later in life. Indeed, although most of the recent evidence regarding the nature and consequences of allostatic load (McEwen and Seeman, 1999) focuses on adults, it is possible that losses of plasticity of physiological response (due to non-optimal allostatic load) that increase the risks of chronic conditions in adult ages are already visible early in life and related to conditions associated with early environments (Hertzman, 2002).

Second, early events or experiences may exert an influence on the likelihood that individuals choose and/or pursue one type of life course or pathway rather than another. If these life courses or pathways are indeed endowed with different conditions affecting health and mortality, the occurrence of earlier events through which individuals are selected into those life courses or pathways can then be thought of as triggers that largely determine subsequent health

experiences (Hertzman, 1999). An example of this is when early health problems influence educational attainment and thus foreclose a number of occupational and career paths. This mechanism is very general and is indeed a prime example of how class of origin can determine class of destination via health status (Wadsworth, 1999).

An important dimension of early environments is socioeconomic status of the family of origin which, via numerous mediating mechanisms, may shape basic characteristics of the early life of individuals. There has been important research focusing on the "direct" effects of socioeconomic status and deprivation in early childhood on late adult health and mortality (Warner and Hayward, 2002; Hayward and Gorman, 2002, Forsdahl, 1978; 2002; Kuh and Ben-Shlomo, 1997; Barker, 1997; Kannisto et al., 1997; Doblhammer, 2002; Moore et al, 1997). One of the mediating influences between socioeconomic conditions in childhood and adult mortality and morbidity is variability in the access to and use of health care. Differences in health care between White and non-White children and their mothers, for example, are well documented (Smedley et al 2002). The relevance of these for later life health outcomes is obvious in the case of prenatal care, for this affects fetal growth and development as well as birthweight. But the importance of other factors--lower rate of use of prescribed medications, less visits to physicians, less compliance with vaccination schedules–remains to be confirmed.

Less tangible properties of early environments, such as exposure to discrimination or stigmatization, physical and social isolation, conflict and violence are also postulated to have important influences of their own. Some research suggests that stressful events, more commonly experienced by individuals in deprived social environments, lead to poor maternal health and increase the risks of fetal underdevelopment and low birthweight. Evidence from animal studies and humans also shows that infants subjected to abuse, lack of maternal or paternal care and, more generally, exposed to higher levels of violence and hostility experience actual physiological changes in their hormonal regulatory mechanisms, changes that are precursors of and predispose to certain chronic conditions (Hertzman, 2002; Coe, 1999; Bornstein and Cote, 2001; Francis and Diorio, 1999; Williams and Collins, 1995). More generally, experiences within certain environments “mark” individuals– perhaps through a number of physiological imprinting mechanisms-- in ways that affect subsequent health directly, e.g. increasing the risk of

morbidity associated with certain conditions and, indirectly, by shutting off or opening up life course or pathways that the individuals may eventually follows.

To summarize, two broad categories of processes may provide paths linking early childhood conditions and late adult health status. Those in the first class establish a direct link between early and adult health. The processes may involve organ dysfunction and impaired development, but with effects and outcomes being latent for long periods of time before they are manifested as morbid conditions in adult life. Those in the second class require the operation of mediators pertaining to the system of rewards and social class positions: early environments impact early health status which, in turn, influences the set of opportunities individuals can access to transit in the social hierarchy. If poverty early in life is associated with worse health both in early childhood and in adulthood, and if early health status affects transitions into and out of poverty--via cognitive ability, educational attainment, or through acquisition of non-market related skills-- adult social membership and social class health and mortality disparities will be partly shaped by early child conditions.

We end this section with an important caveat echoing Schaffer (2000) concerns about the traps of mechanically attributing too much to early experience. Whether one invokes direct or indirect effects of early childhood conditions, it is important to note that the actual mechanisms through which such effects are produced are complex. They involve processes of "imprinting" (Barker, 1997; Hertzman, 1999), "sculpting" (Cynader and Frost, 1999), "path-dependent development" (Schaffer, 2000), "sequencing" (Schaffer, 2000), "turning points" (Rutter, 1996) none of which has been completely conceptualized and, least of all, identified empirically, e.g. verified from predictable patterns in observable data after excluding competing explanations. Some of arguments discussed below, depend on the actual presence of a handful of these mechanisms. Since what is available to us are empirical estimates of gross correlations only, rather than of the mediating paths, our inferences will rest on a somewhat unstable terrain.

Effects of early childhood health status on adult economic success: two mechanisms

The strength of the case for early childhood health status as a determinant of social class position rests more on the existence of indirect than on direct effects. Two bodies of research in particular are relevant for our argument. One seeks to gather evidence to confirm the existence of a strong relation between early childhood health status and mediating mechanisms of economic

success, namely, early and late cognition and educational attainment. The other seeks evidence to support the idea that there are early health status effects on "seemingly irrelevant factors" (for labor market), to use Bowles and Gintis terminology, such as height and obesity. When considered jointly, both strands of research provide a means to bridge literatures on adult health and mortality differentials by social class and the formation of a class system via unequal distribution of health endowments.

Direct effects of early health on mediating factors: There are multiple studies showing the existence of associations between early health status --reflected in birthweight, illness during infancy, postnatal growth rates, and experience of prolonged illness-- on mediating factors. Most of the evidence shows that, however measured, early health status has a direct impact on early cognition, school performance, and late cognition and educational attainment (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., 2002). Although in one recent study some of these findings have been downgraded with a suggestion that early child health status has at best a weak relation to early cognition (Kaestner and Cornman, 1995), in the next section we show that effects are very strong. And this occurs not just in one, but in two nationally representative samples in the US.

Effects of early health status on "seemingly irrelevant factors": there is also a large body of research documenting the existence of a relation between adult height, a marker of cumulative effects of nutritional status early in life, and adult mortality and morbidity (Fogel and Costa, 1997; Fogel 1994; Floud et al., 1990; Scrimshaw, 1997). This is complemented by a growing amount of evidence suggesting the existence of a relation between income and wages, on one hand, and adult height, 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). The latter evidence by itself provides a means to link early health status with adult earnings and economic inequality.

With regard to the "seemingly irrelevant factors" the most important question for us has to do with the mechanisms through which adult height, a partial outcome of early health status conditions, confers advantages in the labor market. Persico and colleagues (Persico et al., 2001) consider a number of candidates. The most interesting ones are those that regard adult height as a

characteristic correlated with traits highly valued by employers, but acquired prior to entrance into the labor market. These traits may include cognitive and non-cognitive skills. In their attempt to test various models and to identify the nature of the mechanism that mediates the effects of adult height on earnings, they find that it is not current (achieved) height that matters at all but height during formative years (14 to 16 years). In fact, the original unstandardized effect of adult height on log of earnings (while controlling for parental but not offspring characteristics and for school quality measures) is within the range .020-.023, meaning that a one inch difference results in an increase in wages between 2.0 and 2.3 %. It alone accounts for between one third and one half of the explained variance of log earnings. However, when controlling for teen height these effects vanish, whereas those of teen height are statistically significant and fluctuate between .020 and .025. This finding rules out the possibility that adult height alters earnings or wages via employers' tastes, since these are not privy to information on teen height. By the same token, because family background and school quality measures are controlled for, it cannot reflect the influences of family or school resources embodied in teen height. Finally, the result cannot be explained by the fact that late physical maturation may be accompanied by late mental maturation. What remains is an intriguing possibility: that height during formative years facilitates (impedes) the acquisition of social skills, leadership qualities, self-esteem, and motivation, all traits that are valued in labor markets. In fact, controlling explicitly for distant measures of some of these traits attenuates the youth-height wage premium. However, there are still substantial effects left.

Altogether the findings reviewed in this section are supportive of the following three conjectures: (i) early child health status may exert direct influences on adult socioeconomic status and on adult health status, but it is likely that these effects are weak; (ii) early health status has a direct and strong impact on market-relevant skills such as early and late cognition as well as educational attainment, (iii) early childhood health influences choice of and engagement in social activities, and social networks as well as the acquisition of a number of IEP traits, such as interest and motivation, self-directedness, loyalty, industriousness, etc..., that are also rewarded in labor markets. The existence of evidence supporting (ii) and (iii) suggests that the first condition of possibility stated in section I is, indeed, met. We now summarize the state of the evidence regarding the second condition of possibility.

The dependence of early health status on parental characteristics

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 socioeconomic position of the family of origin or the mother. Among these are maternal and paternal education, income, poverty levels, parental occupation, receipts of economic assistance etc. 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 the 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 NLSY Youth Panel I, 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 minorities. Although both these studies do not 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 us birth order, length of previous and following interval, age of mother, 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 the 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, must be much larger than the direct effects.

Second, as Cramer recognizes, measures of dimensions of social class or strata used in these studies are conventionally quite poor, as they leave out many factors that may 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”. This conclusion is in keeping with results obtained for the US from the National Maternal and Child Health Survey (Palloni and Partin, 1994).

Thus, although past studies reveal weak direct effects of family socioeconomic background on indicators of early child health status, in most cases they do support the existence of gross effects, mostly operating through known mediating mechanisms. For our purposes it suffices to prove the existence of gross, not direct effects.

The tenor of the discussion in very recent research in the US and elsewhere confirms the importance of family socioeconomic background and of both, direct and indirect effects. This research has exploited large and rich data sets and they uniformly confirm that social and economic characteristics of family of origins, 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 their findings in the US-based NLSY 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 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 reproduced 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 heath status may be "inherited" from parents at birth. Thus, the apparent relation of birthweight in parents and offspring suggests the possibility of genetic inheritance, though it can also be attributed to shared environments. In a series of recent papers, Conley and Bennet (2000, 2001a, 2001b) document strong intergenerational correlation of birthweight for both Blacks and Whites in the US. They find that inheritance of parental birthweight "dramatically reduces the Black-White gap in

birthweight" (Conley and Bennett, 2001b). The authors interpret these findings as evidence of both inheritability of a low birthweight propensity as well as of the influence of shared environments by parents and offspring (Conley and Bennett, 2001b).

In summary, the bulk of the evidence indicates that, either because of conditions encountered in early environments --directly associated with parental social, economic and cultural endowments-- or through inheritance or 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 second condition of possibility identified in the first section of the paper is at least mildly confirmed.

AN EMPIRICAL EXERCISE: THE LONG REACH OF EARLY HEALTH STATUS

In this section we formulate and estimate a simple model to represent the relations between early child health status and two important factors that strongly affect the process through which individuals attain different levels of earnings and income. The goal of this section is to show that the effects of indicators of early child health status on two mediating factors, early cognition and early adolescent height, are influential and far from trivial.

A simple model

The discussion in the preceding sections suggests that the main relations are as in Figure 3. This figure represents a simple pathway linking early child health status and adult earnings. This figure spells out the linkages needed to empirically estimate the inheritability of class position through health status.

Figure 3 is clearly a simplification for not all the elements (cells) that belong in the diagram are included, and also because not all the relations (arrows) that pertain to the included cells are represented. We assume that two types of parental characteristics are relevant: parental endowments and parental investments. Parental endowments refer to genetic characteristics, including those related to health and cognition, as well as educational attainment, wealth and income. Parental investments include investments in three types of capital, human, social, and cultural (Farkas, 2003). Inputs to child health are part of human capital investments; time spent teaching and demonstrating the utilization of certain habits (such as discipline) is part of cultural capital; reinforcement of children social networks and skills to relate to others is an investment in

social capital. Both, parental endowments and parental investments affect early mental and physical health of the child (paths $\alpha 1$ and $\alpha 2$).

Early health status appears as a determinant of early cognition as well as of capacities to acquire IEP-related traits (paths $\gamma 1$ and $\gamma 2$), but it is quite unlikely to be the only one. Height at relatively early ages is a proxy for a subset of these IEP factors (Persico et al., 2001). Early cognition, an ability partly inherited from and partly conferred by parents (path $\beta 1$), is influenced by early health status as well (path $\gamma 1$). Similarly, IEP or key personality traits, could be inherited or learned (path $\beta 2$ plus an omitted path), and they too are influenced by early health status (path $\gamma 2$). Note that, for simplicity, we omit the direct paths linking parental endowments and acquisition of IEP, on the one hand, and parental investments and early cognition, on the other. Furthermore, we lump all cognition into a single measure of early cognition. This ignores the role of late cognition or ability, the kind largely influenced by non-inheritable abilities that is given more salience in research on determinants of income and wages.

Finally, adult earnings appear as a function of educational attainment (path ϵ), cognition (path $\delta 3$), and IEP (path $\delta 2$). This reflects well recent thinking about earning determination.

Figure 3: about here

Although Figure 3 simplifies what is known in the research traditions where the pertinent relations are studied, it summarizes quite well the very dense relations that our previous discussion suggests. The figure distills the most important determinants of access to social class positions (here represented by levels of earnings) and, because not all possible paths out of early health status are represented, it may well lead to underplay, rather than exaggerate, the role that early health plays in the process of social stratification.

With suitable parameter estimates, the relations represented in Figure 3 are useful to answer three large questions:

(i) How much of current earnings inequality is explained by differentials in early health status?

(ii) How "heritable" must early health status be for it to play more than a trivial role in the process of intergenerational transmission of inequality?

(iii) Given current estimates of the influence of family background on early health status, how much of the heritability of earnings is attributable to early health status?

Our goal in this paper is not to answer these questions. Instead, we aim to establish a base from which the questions can be answered, e.g., we provide initial estimates of the effects of early child health status on cognition and early height, two of the mediating factors that influence directly the levels of adult earnings. Clearly, if early health status has not discernible effects on either factor, it cannot possibly represent a conduit through which earnings inequality is transmitted.

Data

We use two U.S. data sets. Both are panel studies which include measures of early health and family socioeconomic background, in addition to direct assessments of children's cognitive ability on different subject areas, and direct and self reported physical measures.

The NLSY-C

The National Longitudinal Survey of Youth (NLSY-C) is a U.S. nationally representative sample of 12,686 men and women ages 14 to 21 as of December 31, 1978. Blacks, Hispanics, and economically disadvantaged whites were oversampled. Respondents were interviewed annually between 1979 and 1994, and biennially since then. Starting in 1986, information on the children of the NLSY female respondents has been collected every two years. The number of children born to interviewed mothers has increased from 5,255 in 1986 to 8,395 in 1998 (the last wave of data collection currently available), representing a large majority of the children to be born to this cohort of women. The first waves of the NLSY-C overrepresented children born to younger, less highly educated, and racial/ethnic minority mothers (Guo and Harris 2000). Although this issue has become less important in later waves, controls for characteristics overrepresented in the sample (such as maternal age) are included in the analysis.

The ECLS-K

The Early Childhood Longitudinal Data-Kindergarten Class of 1998-1999 (ECLS-K) is a nationally representative sample of approximately 22,000 children enrolled in about 1,000 public and private, half and full-day kindergarten programs during the 1998-1999 school year. Children from different racial/ethnic and socioeconomic backgrounds are included, with an oversample of Asian children, private kindergartens, and private school kindergartners. In contrast to the

NLSY-C – which represents children born to a cohort of mothers – the ECLS-K represents a cohort of children. Although the overall majority of U.S. children attend preprimary programs such as kindergarten[4], it is possible that those who do not attend do so because of limiting health conditions. This possible selection does not take place in the NLSY-C, because the sampling criterion is not tied to school enrollment. In any case, if it is true that children excluded from the ECLS-K are less healthy than those included in the sample, current estimates of early health effects are biased downwards.

Children from Hispanic origin may be slightly better represented in ECLS-K than in NLSY-C, where the cognitive assessment was only administered in English. In ECLS-K, even though children had to demonstrate a certain level of proficiency in English to be administered the Reading cognitive assessment, those who were proficient in Spanish were administered the Math component of the assessment in Spanish (5 per cent of the sample was assessed in Spanish). Children from other non-English home language backgrounds who were not proficient in English were excluded from the assessment (approximately 2 per cent of the sample). Since children who are not proficient in either English or Spanish are excluded from cognitive assessments in both samples, the distinction between NLSY-C and ECLS-K is almost exclusively relevant to Hispanics.

Model specification

Our most immediate aim is to obtain a range of values for the parameters $\gamma 1$ and $\gamma 2$ in both data sets. In order to identify differences across race and ethnic groups, we estimate these parameters separately by race, including Non Hispanic Whites (NHW), Non Hispanic Blacks (NHB), and Hispanics. We use a single indicator of early health status –birthweight—and a single indicator of IEP factors –childhood height. There is no doubt these indicators oversimplify the constructs they attempt to measure. That said, it is also the case that low birthweight is the major determinant of infant mortality in developed countries, and it contributes substantially to the overall burden of childhood handicap (Paneth, 1995). Height on the other hand, is one of the

[4] In the U.S., in 1996, 37 percent of 3-year-olds, 58 percent of 4-year-olds, and 90 percent of 5-year-olds were enrolled in center-based programs or kindergarten (National Center for Education Statistics 1999).

few non-market related factors for which effects on earnings are estimated at the same time that effects of early health on height are.

Specification of outcomes

In both data sets cognition is measured as children's performance on reading and math tests. In NLSY-C, the outcome of interest in the Peabody Individual Achievement Test (PIAT), taken by all children age 5 or older (Baker et. al 1993). Specifically, we use the first available assessment across the seven waves of data collection carried out every two years between 1986 and 1998[5]. In ECLS-K, early cognition corresponds to the fall of kindergarten Item Response Theory (IRT) scores children obtained through an un-timed one-on-one computer-assisted personal interview (West, Denton, and Germino-Hausken 2000).

In the case of height, we find no difficulties measuring it in ECLS-K, since we can retrieve *direct* anthropometric measures obtained in the fall of kindergarten. Quite clearly, height at kindergarten age may not be a strong predictor of height during early adolescence but the relations may still be informative.

In the case of NLSY-C we face additional difficulties. First, not all children in NLSY-C have a direct assessment of height – between 20 and 50 per cent of the children (depending on the data collection wave) have been assigned a maternal report on height. To minimize measurement error, we created two data sets, one where the we average the available measures of height across the first three waves of data collection (1986, 1988, and 1990) and another where the average is calculated across the last four waves (1992,1994,1996 and 1998). Second, because we wish to obtain an assessment of height during a stage of childhood later than kindergarten, we created two additional data sets, one containing all individuals and one containing only those who where older than 53 months. Thus, for the case of NLSY-C we have a total of four estimates of effects of birthweight on height. To minimize effects of reverse causality we use the midpoint year as the reference for child's age, but the initial year of data as the reference for all other covariates.

We estimate three alternative models for height. One for height measured in inches, another for height as a percentile location of height-for-age in the standard age and sex specific

distribution elaborated by the National Center for Chronic Disease Prevention and Health Promotion (CDC, 2000), and a final one for height as a z-score of the height-for-age measure in the same standard distribution. Since all three of these yield identical inferences, we only show the results corresponding to the first definition of height.

Specification of key independent variables

We formulate two different models for each outcome variable –early cognition and height--, one where birthweight is a dichotomous variable (low birthweight versus normal birthweight)[6], and one where birthweight is a continuous measure. In terms of covariates, a first set of models includes only measures of early health associated with birthweight: in ECLS-K, an indicator for prematurity and single birth; in NLSY-C, birth order, and indicators for prematurity, delivery by cesarean section, child's hospitalization after delivery longer than the mother, and child's admission to the hospital during the first year of life. A second set of models adds the family background and individual level controls, namely, gender, children and mother's age, socioeconomic conditions of the family (mother's education, income, poverty status, and family structure). Descriptive information of all the variables used in both samples is included in Appendices 1 and 2.

Specification of model type

For all estimates we use OLS specification despite the fact that in most cases our dependent variable is continuous but positive-valued. Although different specifications (using z-scores or power transformations) should be estimated to ensure robustness of estimates, our experience with these samples suggests that results are insensitive to functional forms.

Results

The most important results are in Tables 1a, 1b, 2a, and 2b. Tables 1a and 1b display estimates of effects on cognitive ability, whereas Tables 2a and 2b include estimates of effects on height for various sample specifications. While estimates in Tables 1a and 2a come from models

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[5] To avoid the possibility of confounding period effects, a dummy variable indicating the year in which the child was assessed is included in the regression analysis, with 1986 as the reference category.

[6] Abiding to conventional definitions, low birthweight is defined as less than 2,500 grams or 5.5 pounds. Normal birthweight refers to the interval between 5.5 and 8.82 pounds. More than 4,000 grams or 8.82 pounds is considered high birthweight.

where birthweight is specified as a discrete variable (low birthweight vs normal birthweight), estimates in Tables 1b and 2b use birthweight as a continuous variable.

Results for cognition

In the ECLS-K sample the effects of birthweight on cognition are relevant irrespective of how the variable is defined. Effects are significant for all groups except Hispanics. In Table 1a, estimates are significant with a fairly conservative specification of p values ($p<.001$) for NHW, whereas for NHB a more liberal value of p ($<.01$) is required to assign statistical significance. Effects on math ability are consistently high among NHW and NHB whereas those for reading ability are important only for NHB, at significance levels of .01 or less. Since the mean value of tests scores differ for math and reading, and also across racial/ethnic groups, the meaning of the estimated regression coefficients varies. In the case of math scores, a change from low to normal birthweight implies an average increase of about 8.5 percent for NHW (1.82/21.2) and about 6.7 percent (1.12/16.6) for NHB. In the case of reading scores the average increases are more modest for NHW and somewhat larger for NHB, 3.7 percent (.86/23.5) and 6.3 percent (1.25/19.9) respectively.

The fact that the effect of birthweight remains important and significant even when we introduce the variable in its continuous version (see Table 1b) suggests that the disadvantages associated with lower birthweight are not concentrated at the lower end of the distribution, among the fraction of births who are classified as “Low Birthweight” using what is, after all, an arbitrary cutting point.

Results in the NLSY-C are less decisive since the effects of birthweight are important only among NHW and only for math scores.

In summary, these results suggest that early health status as measured by birthweight has some effects on early cognitive ability. The results are consistent for one ethnic group, namely Non Hispanic Whites, less so for Non Hispanic Blacks, and not relevant at all for Hispanics.

It is important to note that the estimated effects are most likely **lower-bound estimates** of the gross effects of early health status for two reasons. First, birthweight is **only one** of the indicators of early health among many others about which we have no information and whose relation to birthweight might be only tenuous. Second, because the effects of low birthweight are very likely mediated by conditions that develop after birth, some of which we are being

controlled for in our equations, the effects we retrieve are **net** effects of birthweight not the **gross effects of health status**.

Results for height

Tables 2a and 2b display estimated effects of birthweight, in its discrete and continuous specification, on measures of height in inches[7]. In the case of ECLS-K the results of the various models are remarkably similar and lead to virtually the same conclusions. The first is that effects of birthweight are strong and statistically significant. Second, these effects are robust to the specification of birthweight -- they are equally strong and statistically significant whether one uses it as a discrete or continuous variable. Third, the effects are not trivial and very similar across ethnic groups: since average height among children in the ECLS-K is about 44.7, 45.1 and 44.3 for NHW, NHB and Hispanics respectively, the estimated effects imply that a change from low to normal birthweight would increase height by about 1.8 percent in all three race groups. Working with approximations from the estimated equations where birthweight is a continuous variable leads to the inference that the approximate elasticity of height (in inches) relative to birthweight (lbs) is of the order of 6 percent for all three groups. That is, a 10 percent change in birthweight leads to a .6 percent in stature.

As in the case of cognition, results from NLSY-C are slightly less definitive. For example, when birthweight is specified as a discrete variable (Table 2b) the effects are strong and significant for NHW and NHB, but not so for Hispanics. Apart from this exception, the magnitude of the coefficients is remarkably similar to those obtained in ECLS-K, irrespective of model specification. By the same token, the results from models where birthweight is treated as a continuous variable show strong and highly significant effects, including Hispanics. And, here once again, the numerical results across data sets and samples are satisfyingly close to each other.

These findings only increase our confidence in the following conclusion: the impact of birthweight on both, early stature (from ECLS-K) and stature among older children (from NLSY-C) is strong and robust to data set and model specification.

[7] As indicated before, we also estimated models for height as percentile in a standard distribution of height-for- age and as z-scores in the same standard distribution. The results lead to conclusions that are no different from those we draw here using height in inches.

As was the case for cognition, and for the same reasons stated before, here too effects of early health status are likely to be underestimated by the effects of birthweight. And, as in the case of cognition, the processes seem to be very similar among NHW and NHB but may obey a different logic among Hispanics.

CONCLUSION

This paper produced two results, one associated with theory and the other with empirical regularities. The theoretical result is this: we argued that there are important reasons to support the idea that early health status plays a role in social stratification and in the heritability of positions at least when using some metrics of social class. To make the point we review accumulated research in conventional social stratification and in more recent work on labor economics. The main conclusion from this review is that wage and income determination are processes affected by a number of conditions which may be partially explained by early health status.

That this is indeed a suggestive possibility worthy of further analysis is what the second, more empirical, result of the paper demonstrates. In fact, we showed that a very crude measure of early health status, birthweight, appears to have strong **net effects** on both early cognition and early and adolescent stature, examples of market and non market factors, respectively, that explain wage and income determination. It is very likely that better measures of health status and a less narrow focus on gross (rather than net) effects, may reveal that early health status is much more important as a determinant of social stratification than what has conventionally been recognized. Also, our analyses only identified two factors on which early health could exert influences, early cognition and heights. It may well be that there are additional market and non market factors on which we should focus attention. For these and other reasons we spell out in the text, we believe we are downplaying the influence of early health in social stratification.

The results we obtain suggest that the processes involved are not identical across ethnic groups and that one should make sure that theorization and estimation of effects is done separately by race or ethnic groups. This is not a surprising finding, as there are a number of social conditions that make social stratification processes different within ethnic groups. Yet, we

have no clear way to account for our findings and we strongly suspect that further research will confirm that other relations among those in Figure 3 also are different by ethnicity.

What is it that remains to be done? The first and most important task is to ensure that the empirical effects we obtain in this paper can be blended with estimates of other relations depicted in Figure 3 to be able to calculate the contribution of early health on earnings and income determination. This is a feasible undertaking and preliminary results (Palloni and Milesi, 2002) suggests that this contribution is important, and that at least part, of the heritability of social class positions verified in recent data sets occurs as a result of the production of unequal early health status. Indeed, we estimate that one fifth of the total correlation between income across generations could be accounted for by early health status. But this estimate is primitive at best as it was obtained with less than optimal data sets and a blunt methodology.

A second pending problem is related to race and ethnic groups. This matter is urgent and daunting. We need to put forward conjectures associated with differential processes by ethnic and race groups as the various patches of theory we used to weave our final arguments do not always concern themselves with ethnic a race differences. Surely, some of these differences will vanish once we take into account factors not included in our models or better measures of factors we were able to include. But it would be truly astonishing if unexplained (ethnic related) residuals vanished. The degree to which this will happen probably has much to do with the political and institutional contexts being studied, those that provide the raw materials with which populations create ideologies about and deal with race and ethnicity

There are also important gaps in our theory, issues mentioned in passing that deserve more thorough treatment, and others we simply neglected to address altogether that need to be reintroduced. Although the skeleton of a cogent theory may exist, it needs to be propped considerably to become really useful. The lacunae are many and too glaring but we can identify some of them briefly. First, the paths through which early child health influence earning potential need to be explored better. Birthweight may be a key determinant, but it is unlikely to be the only or even the most important one. Early health status is strongly related to parental decision-making and investments in child health during infancy and subsequent years. Surely these have effects that are independent of health status at birth. Just as parental cultural, economic or social

investments are ongoing processes, so are parental health investments spread over the life of a child.

Second, to understand better the exact contribution of intergenerational transmission of income we need to model more thoroughly the dynamics of assortative mating with respect to earning-relevant characteristics. This is important when dealing with innate characteristics, such as cognitive ability, but it is also relevant when examining factors such as health status. We need to better understand couple formation and childbearing practices as a function of earning-relevant parental characteristics.

Third, we need to provide a more tightly argued theoretical rational for a procedure to derive estimates of the contribution of early health on social mobility and the distribution of individuals into social class positions. It is true that one can use first principles to calculate the degree to which a social class system in one generation mirrors the previous one and how much of it is only due to early health status (Palloni and Milesi, 2002). But there are alternative strategies and they can conceivably produce different results

In sum, the estimates provided in this paper have a number of shortcomings and are quite fragile because they are derived from a defective and incomplete theoretical framework and with rather crude methodologies. Yet, despite these limitations, we can state with confidence the one and only conclusion that seems warranted, namely, that there are good theoretical and empirical reasons to reformulate theories of social stratification assigning importance to the role played by early health status on the social careers of individuals.

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Table 1a: Coefficients from OLS Regression of Low Birth Weight on Cognitive Ability

ECLS-K. Fall Kindergarten IRT Scores.

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
Math														
White non-Hispanic	-2.00	(0.37)***	-0.06	9607	4	0.01	66.81	-1.82	(0.33)***	-0.06	9607	14	0.21	2271.09
Black non-Hispanic	-1.32	(0.40)**	-0.08	2338	4	0.01	18.82	-1.12	(0.36)**	-0.07	2338	14	0.20	513.62
Hispanic	-0.07	(0.48)	0.00	2866	4	0.01	15.74	-0.68	(0.41)	-0.03	2866	14	0.27	886.24
Reading														
White non-Hispanic	-1.05	(0.43)*	-0.03	9608	4	0.00	18.17	-0.86	(0.39)*	-0.02	9608	14	0.18	1879.04
Black non-Hispanic	-1.52	(0.50)**	-0.07	2338	4	0.01	13.30	-1.25	(0.45)**	-0.06	2338	14	0.20	535.90
Hispanic	0.13	(0.73)	0.00	2027	4	0.00	2.17	-0.54	(0.66)	-0.02	2027	14	0.20	463.43

NLSY-C. PIAT Raw Scores.

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
Math														
White non-Hispanic	-2.70	(0.87)**	-0.06	3215	13	0.14	483.50	-1.59	(0.55)**	-0.03	3215	22	0.65	3387.37
Black non-Hispanic	2.03	(0.80)*	0.06	2089	13	0.17	386.04	-0.60	(0.50)	-0.02	2089	22	0.69	2454.57
Hispanic	-0.64	(1.38)	-0.01	1319	13	0.09	119.50	0.01	(0.79)	0.00	1319	22	0.70	1601.59
Reading														
White non-Hispanic	-2.34	(0.95)*	-0.04	3211	13	0.15	504.06	-1.44	(0.60)*	-0.03	3211	22	0.66	3430.62
Black non-Hispanic	2.33	(0.84)**	0.06	2084	13	0.20	459.25	-0.63	(0.54)	-0.02	2084	22	0.67	2329.87
Hispanic	-0.50	(1.53)	-0.01	1320	13	0.10	136.47	0.38	(0.88)	0.01	1320	22	0.71	1618.26

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

Table 1b: Coefficients from OLS Regression of Continuous Birth Weight on Cognitive Ability

ECLS-K. Fall Kindergarten IRT Scores.

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
Math														
White non-Hispanic	0.62	(0.07)***	0.11	9607	3	0.01	104.98	0.46	(0.06)***	0.08	9607	13	0.21	2291.28
Black non-Hispanic	0.43	(0.09)***	0.11	2338	3	0.01	25.40	0.35	(0.09)***	0.09	2338	13	0.20	518.84
Hispanic	0.06	(0.09)	0.01	2866	3	0.01	15.43	0.18	(0.08)*	0.04	2866	13	0.27	888.17
Reading														
White non-Hispanic	0.35	(0.08)***	0.05	9608	3	0.00	31.30	0.30	(0.07)***	0.05	9608	13	0.18	1890.90
Black non-Hispanic	0.43	(0.12)***	0.09	2338	3	0.01	15.05	0.32	(0.11)**	0.07	2338	13	0.20	535.78
Hispanic	-0.08	(0.14)	-0.01	2027	3	0.00	2.37	0.09	(0.13)	0.01	2027	13	0.20	462.61

NLSY-C. PIAT Raw Scores.

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
Math														
White non-Hispanic	0.28	(0.16)	0.03	3215	12	0.14	476.80	0.29	(0.10)**	0.03	3215	21	0.65	3386.66
Black non-Hispanic	-0.44	(0.19)*	-0.05	2089	12	0.17	384.62	0.15	(0.12)	0.02	2089	21	0.69	2454.65
Hispanic	-0.25	(0.27)	-0.03	1319	12	0.09	118.82	0.10	(0.15)	0.01	1319	21	0.70	1600.78
Reading														
White non-Hispanic	0.23	(0.17)	0.02	3211	12	0.14	499.44	0.28	(0.11)*	0.03	3211	21	0.66	3431.29
Black non-Hispanic	-0.49	(0.20)*	-0.06	2084	12	0.20	456.56	0.18	(0.13)	0.02	2084	21	0.67	2329.85
Hispanic	-0.16	(0.29)	-0.02	1320	12	0.10	136.65	0.14	(0.17)	0.01	1320	21	0.71	1618.70

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

Table 2a: Coefficients from OLS Regression of Low Birth Weight on Height (inches)

ECLS-K. Height at fall of kindergarten year.

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
White non-Hispanic	-0.64	(0.14)***	-0.07	9613	4	0.03	297.44	-0.78	(0.10)***	-0.08	9613	14	0.19	1969.95
Black non-Hispanic	-0.65	(0.16)***	-0.10	2337	4	0.03	59.12	-0.73	(0.15)***	-0.11	2337	14	0.17	421.50
Hispanic	-0.52	(0.16)**	-0.07	2875	4	0.01	34.75	-0.71	(0.15)***	-0.09	2875	14	0.17	535.49

NLSY-C. Average height across waves. All children

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
First three waves (1986, 1988, 1990)														
White non-Hispanic	-0.20	(0.79)	0.00	3400	7	0.11	393.75	-0.87	(0.24)***	-0.02	3400	16	0.92	8507.49
Black non-Hispanic	2.55	(0.78)**	0.08	1977	7	0.18	404.15	-1.06	(0.25)***	-0.03	1977	16	0.92	4926.15
Hispanic	-0.23	(1.20)	-0.01	1245	7	0.14	188.12	-0.66	(0.41)	-0.02	1245	16	0.90	2869.84
Last four waves (1992, 1994, 1996, 1998)														
White non-Hispanic	-1.14	(0.99)	-0.02	2954	7	0.10	314.28	-1.29	(0.30)***	-0.03	2954	16	0.92	7439.31
Black non-Hispanic	0.75	(0.80)	0.02	2029	7	0.15	340.85	-1.14	(0.32)***	-0.03	2029	16	0.87	4086.28
Hispanic	-1.59	(1.26)	-0.04	1359	7	0.14	212.42	-1.03	(0.46)*	-0.02	1359	16	0.89	2948.64

NLSY-C. Average height across waves. Exclusion of "younger" children

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
First three waves (1986, 1988, 1990). Exclusion of younger half of the sample.														
White non-Hispanic	-0.93	(0.72)	-0.04	1563	7	0.05	81.31	-0.78	(0.30)**	-0.03	1563	16	0.84	2879.73
Black non-Hispanic	1.85	(0.62)**	0.09	1186	7	0.10	123.16	-0.77	(0.26)**	-0.04	1186	16	0.85	2231.94
Hispanic	-0.93	(1.02)	-0.04	646	7	0.07	43.68	-0.63	(0.44)	-0.03	646	16	0.83	1139.63
Last four waves (1992, 1994, 1996, 1998). Exclusion of younger 25 per cent of the sample.														
White non-Hispanic	-0.45	(0.80)	-0.01	2304	7	0.07	167.75	-1.22	(0.32)***	-0.03	2304	16	0.85	4416.58
Black non-Hispanic	0.36	(0.63)	0.01	1798	7	0.14	260.70	-1.33	(0.32)***	-0.05	1798	16	0.78	2700.69
Hispanic	-0.84	(1.00)	-0.03	1144	7	0.12	146.94	-0.84	(0.49)	-0.03	1144	16	0.80	1821.16

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

Table 2b: Coefficients from OLS Regression of Continuous Birth Weight on Height (inches)

ECLS-K. Height at fall of kindergarten year.

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
White non-Hispanic	0.44	(0.02)***	0.27	9613	3	0.06	567.68	0.44	(0.02)***	0.26	9613	13	0.21	2280.60
Black non-Hispanic	0.32	(0.04)***	0.21	2337	3	0.04	93.48	0.35	(0.03)***	0.23	2337	13	0.18	473.29
Hispanic	0.26	(0.03)***	0.16	2875	3	0.03	74.13	0.29	(0.03)***	0.18	2875	13	0.18	583.67

NLSY-C. Average height across waves. All children

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
First three waves (1986, 1988, 1990)														
White non-Hispanic	0.11	(0.14)	0.01	3400	6	0.11	393.88	0.41	(0.04)***	0.05	3400	15	0.92	8555.08
Black non-Hispanic	-0.56	(0.18)**	-0.07	1977	6	0.18	401.40	0.38	(0.06)***	0.05	1977	15	0.92	4942.00
Hispanic	-0.28	(0.22)	-0.04	1245	6	0.14	189.63	0.39	(0.08)***	0.05	1245	15	0.90	2884.41
Last four waves (1992, 1994, 1996, 1998)														
White non-Hispanic	0.38	(0.17)*	0.04	2954	6	0.10	313.62	0.56	(0.05)***	0.06	2954	15	0.92	7487.58
Black non-Hispanic	-0.12	(0.18)	-0.02	2029	6	0.15	340.32	0.49	(0.07)***	0.06	2029	15	0.06	4104.79
Hispanic	0.16	(0.24)	0.02	1359	6	0.14	207.68	0.52	(0.09)***	0.06	1359	15	0.89	2957.57

NLSY-C. Average height across waves. Exclusion of "younger" children

	A. Infant Health only							B. A + Controls + Family background						
	Est.	SE	Beta	N	df	R^2	LR	Est.	SE	Beta	N	df	R^2	LR
First three waves (1986, 1988, 1990). Exclusion of younger half of the sample.														
White non-Hispanic	0.39	(0.14)**	0.08	1563	6	0.05	84.87	0.49	(0.06)***	0.10	1563	15	0.85	2922.94
Black non-Hispanic	-0.37	(0.16)*	-0.07	1186	6	0.10	119.17	0.31	(0.07)***	0.06	1186	15	0.85	2239.97
Hispanic	0.09	(0.20)	0.02	646	6	0.06	42.91	0.39	(0.09)***	0.08	646	15	0.83	1149.09
Last four waves (1992, 1994, 1996, 1998). Exclusion of younger 25 per cent of the sample.														
White non-Hispanic	0.22	(0.13)	0.04	2304	6	0.07	169.19	0.50	(0.05)***	0.08	2304	15	0.86	4450.50
Black non-Hispanic	-0.04	(0.14)	-0.01	1798	6	0.13	260.44	0.47	(0.07)***	0.08	1798	15	0.78	2718.43
Hispanic	0.06	(0.19)	0.01	1144	6	0.12	144.28	0.45	(0.09)***	0.08	1144	15	0.80	1824.94

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

Appendix 1: Unweighted Descriptive Statistics of Variables Used in Regressions of Cognitive Ability and Height on Socioeconomic and Health Conditions. Early Childhood Longitudinal Study-Kindergarten Cohort.

	White non-Hispanic			*Black non-Hispanic*			*Hispanic*		
	N	Mean	Std.Dev.	N	Mean	Std.Dev.	N	Mean	Std.Dev.
Dependent variable									
Math IRT scores	10433	21.21	7.46	2855	16.63	5.59	3388	16.02	6.05
Reading IRT scores	10433	23.50	8.60	2854	19.93	6.94	2374	19.79	7.59
Height (inches)	10453	44.74	2.16	2855	45.08	2.23	3400	44.29	2.12
Controls									
Male	11741	0.52	0.50	3210	0.50	0.50	3762	0.51	0.50
Child's age (months)	10488	68.87	4.46	2867	68.18	4.52	3426	67.82	4.38
Mother's age (years)	10046	33.86	5.95	2584	32.06	8.34	3142	31.77	6.59
Socioeconomic conditions									
Mom's ed: Less than High School	11143	0.07	0.25	2921	0.19	0.39	3509	0.35	0.48
Mom's ed: High School	11143	0.29	0.45	2921	0.37	0.48	3509	0.30	0.46
Mom's ed: Some Higher Education	11143	0.34	0.47	2921	0.34	0.47	3509	0.26	0.44
Mom's ed: Bachelor or more	11143	0.30	0.46	2921	0.10	0.31	3509	0.10	0.29
ln(household income)	11343	10.95	0.65	2966	10.27	0.67	3562	10.43	0.65
Below poverty	11343	0.09	0.29	2966	0.38	0.49	3562	0.33	0.47
Single parent family	10262	0.15	0.36	2638	0.53	0.50	3205	0.25	0.43
# Siblings in household	10262	1.38	1.02	2638	1.55	1.35	3205	1.53	1.20
Infant health									
Single birth	10234	0.97	0.16	2620	0.97	0.17	3192	0.98	0.14
Low birth weight	11220	0.06	0.24	2874	0.13	0.34	3506	0.07	0.26
High birth weight	11116	0.14	0.34	2791	0.06	0.24	3425	0.10	0.30
Birth weight (pounds)	11116	7.52	1.31	2791	6.89	1.45	3425	7.30	1.33
Premature	11176	0.17	0.37	2866	0.19	0.39	3498	0.16	0.37

Appendix 2: Unweighted Descriptive Statistics of Variables Used in Regressions of Cognitive Ability and Height on Socioeconomic and Health Conditions. Children of the National Longitudinal Survey of Youth.

	White non-Hispanic			*Black non-Hispanic*			*Hispanic*		
	N	Mean	Std.Dev.	N	Mean	Std.Dev.	N	Mean	Std.Dev.
Dependent variable									
Math IRT scores	3612	20.27	10.99	2419	18.76	11.68	1566	18.47	11.69
Reading IRT scores	3608	21.95	12.13	2413	21.99	12.34	1565	20.85	13.43
Mean height (inches) across first 3 waves	3731	40.49	9.98	2235	43.87	11.07	1441	41.86	9.94
Mean height (inches) across last 4 waves	3766	48.19	11.74	2576	52.63	11.55	1801	50.15	11.53
Controls									
Male	5804	0.51	0.50	3044	0.51	0.50	2070	0.52	0.50
Child's age (months)	3612	77.88	17.65	2419	81.79	21.90	1566	80.11	20.61
Mother's age (years)	5802	25.07	5.15	3044	23.49	5.33	2070	24.65	5.36
Socioeconomic conditions									
Mom's ed: Less than High School	3608	0.26	0.44	2419	0.32	0.47	1560	0.44	0.50
Mom's ed: High School	3608	0.43	0.49	2419	0.39	0.49	1560	0.33	0.47
Mom's ed: Some Higher Education	3608	0.18	0.39	2419	0.22	0.41	1560	0.19	0.39
Mom's ed: Bachelor or more	3608	0.14	0.34	2419	0.07	0.25	1560	0.05	0.22
ln(household income)	3584	9.93	0.75	2383	9.36	0.73	1540	9.64	0.76
Below poverty	3586	0.20	0.33	2389	0.52	0.41	1544	0.36	0.39
Single parent family	3610	0.16	0.30	2417	0.58	0.43	1565	0.26	0.37
Infant health									
Birth order	5802	1.79	0.96	3044	2.04	1.19	2070	2.09	1.23
Premature	5239	0.12	0.33	2699	0.14	0.35	1807	0.11	0.31
C-Section	5279	0.21	0.41	2729	0.20	0.40	1831	0.23	0.42
Low birth weight	5377	0.07	0.25	2801	0.13	0.34	1860	0.07	0.26
High birth weight	5377	0.11	0.32	2801	0.06	0.23	1860	0.10	0.30
Birth weight (pounds)	5377	7.41	1.30	2801	6.88	1.41	1860	7.34	1.32
Hospitalization after birth	4985	0.08	0.27	2567	0.11	0.32	1718	0.10	0.30
Hospitalization during 1st year	5804	0.07	0.26	3044	0.06	0.24	2070	0.06	0.24

Figure 1: Relation between early health status, social class attainment and adult health and Mortality (“indirect selection”)

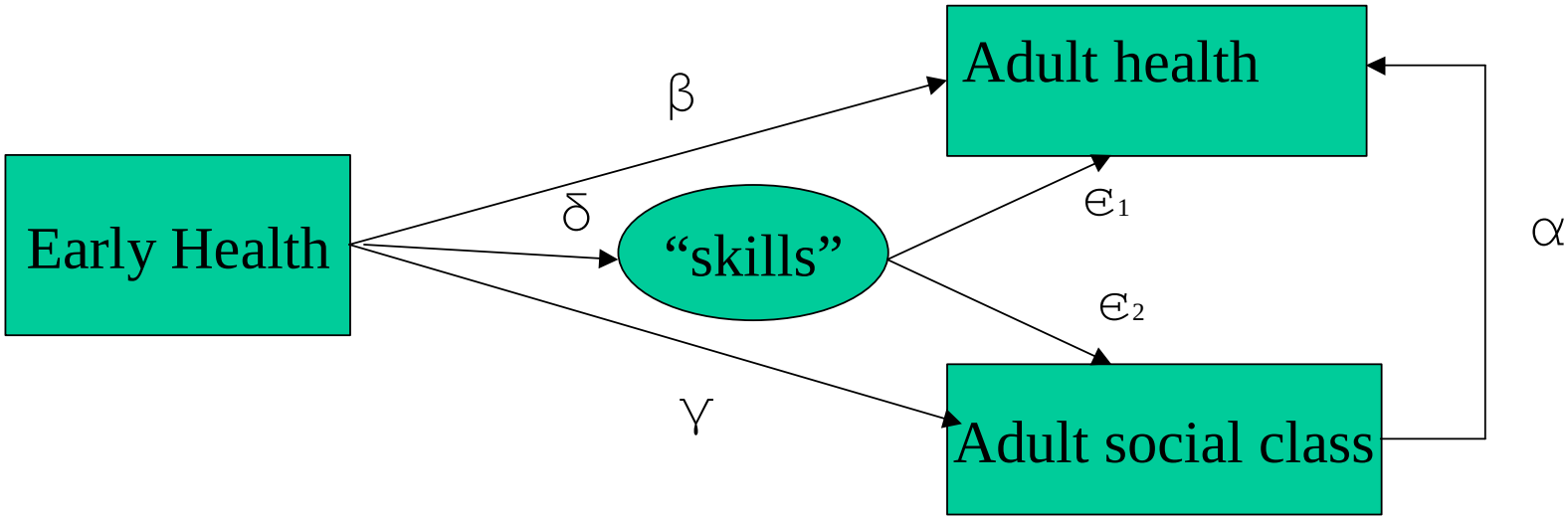


Figure 2: Path diagram representing the contribution of Genetics skills, and environments on adult earnings

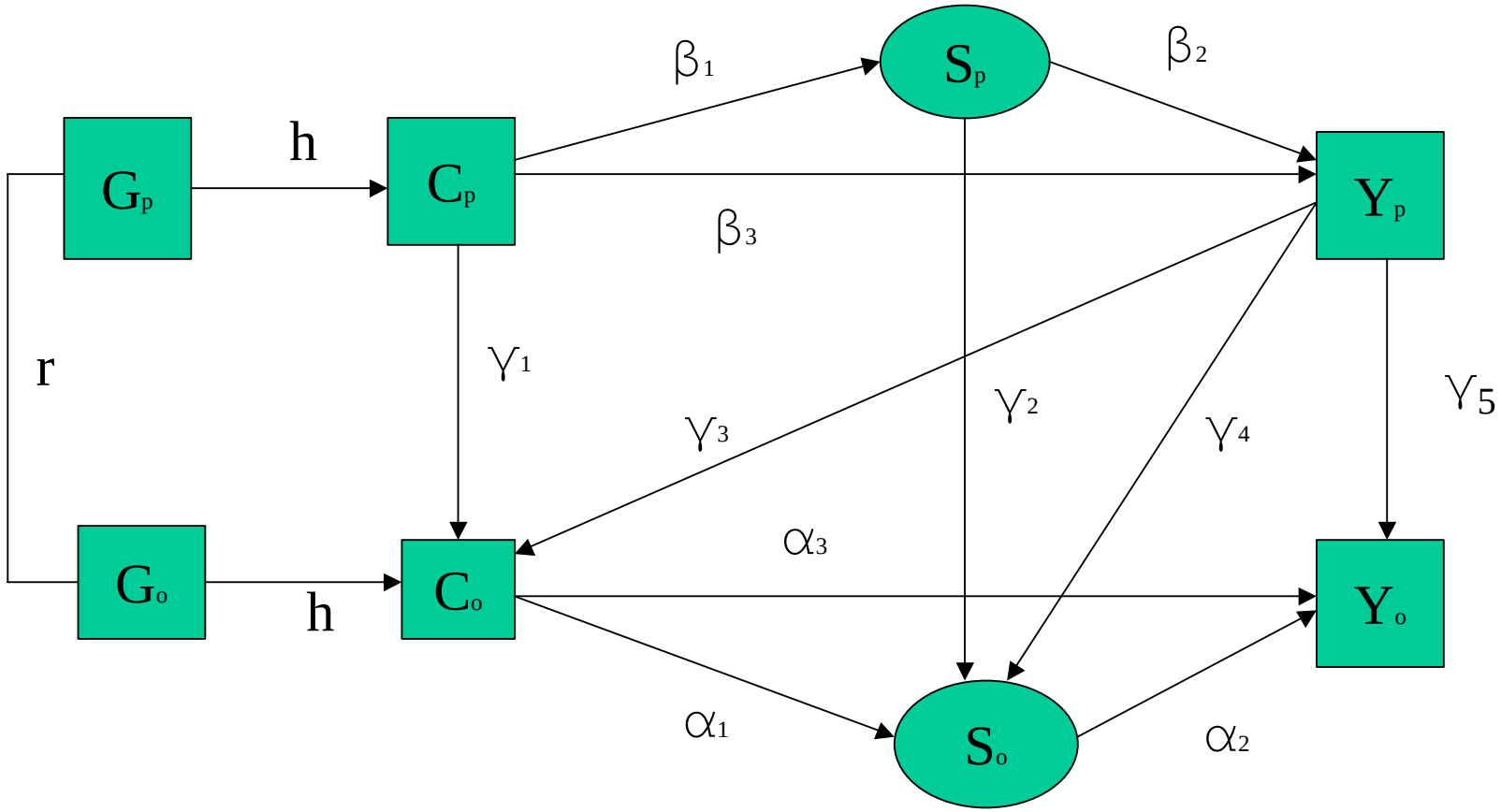
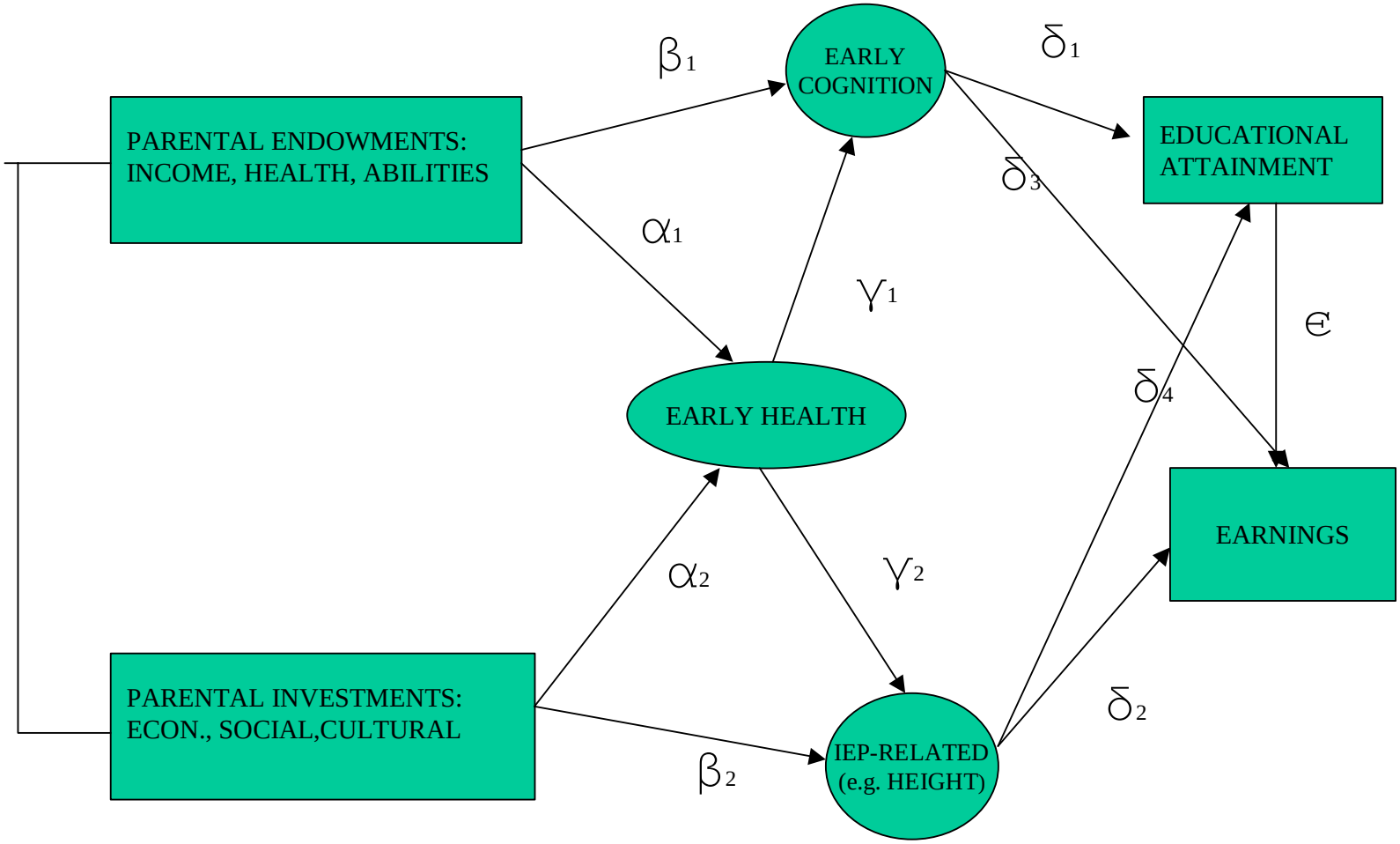


Figure 3: Simplified path diagram representing the relations between early child health conditions and adult earnings



Center for Demography and Ecology
University of Wisconsin
1180 Observatory Drive Rm. 4412
Madison, WI 53706-1393
U.S.A.
608/262-2182
FAX 608/262-8400
comments to: palloni@ssc.wisc.edu
requests to: cdepubs@ssc.wisc.edu