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The Influence of Early-Life Events on Human Capital, Health Status, and Labor Market Outcomes Over the Life Course*

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Abstract

Using national data from the U.S., we find that poor health at birth and limited parental resources (including low income, lack of health insurance, and unwanted pregnancy) interfere with cognitive development and health capital in childhood, reduce educational attainment, and lead to worse labor market and health outcomes in adulthood. These effects are substantial and robust to the inclusion of sibling fixed effects and an extensive set of controls. The results reveal that low birth weight ages people in their 30s and 40s by 12 years, increases the probability of dropping out of high school by one-third, lowers labor force participation by 5 percentage points, and reduces labor market earnings by roughly 15 percent. While poor birth outcomes reduce human capital accumulation, they explain only 10 percent of the total effect of low birth weight on labor market earnings. Taken together, the evidence is consistent with a negative reinforcing intergenerational transmission of disadvantage within the family; parental economic status influences birth outcomes, birth outcomes have long reaching effects on health and economic status in adulthood, which in turn leads to poor birth outcomes for one's own children.

Keywords

birth weight; childhood SES; adult health; intergenerational mobility

I. Introduction

Gaps in cognitive and non-cognitive skills emerge before children enter school, appear to widen over the life cycle, have been linked to family resources and environment at early ages, and have long-run consequences for socioeconomic success in adulthood (Carneiro and Heckman, 2003; Fryer and Levitt, 2004). The early literature on the effects of pre-market factors on labor market outcomes emphasized cognitive skills in childhood (see e.g., Neal and Johnson, 1996). It is also likely that health status at birth and in childhood contribute to socioeconomic dimensions of inequality and health status over the life course. The focal point in the epidemiology literature has been the fetal origins hypothesis developed by David Barker and colleagues. The "Barker Hypothesis" proposes that when

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nutritional intake of a fetus is limited, the body's physiology and metabolism are changed fundamentally, and some of the consequences of these changes would become visible much later in life. A voluminous empirical literature in epidemiology supports Barker's theory, drawing largely on data from the United Kingdom. (See Barker, 1998, for a review.) Recent evidence in the developmental origins of adult disease and neuroscience literatures emphasize the critical period of development from conception to age three as one that is extremely sensitive to stressful environmental conditions due to the fact that the speed of growth is more rapid than any other stage of the life course and the nutritional needs are greatest (e.g., see Lynch and Smith, 2005, for a review; Heckman, 2007, and references therein).

In this study we investigate the linkages between health and economic status in the initial stages of life, and health, human capital, and labor market outcomes in adulthood using nationally representative longitudinal data covering a 35-year period in the U.S.; this is the first such study of the full U.S. population. The data set, the Panel Study of Income Dynamics (PSID), has the additional unique feature of allowing analyses of siblings throughout much of their life course. Many prior studies of the connection between early life health and economic status and adult health have relied on health surveys that have very limited economic data. The PSID is one of the premier income surveys in the world, and it contains significant detail on health.

This study traces the effects of birth outcomes and socioeconomic factors near the time of birth on health, human capital, and labor market outcomes over the life course. In childhood, we examine the effects on health status, cognitive development, and completed schooling, and in adulthood we examine health status and labor market outcomes. The analyses take advantage of the unique genealogical design of the PSID that allows comparisons among siblings as well as across generations within the same family.

Our results highlight that events early in life have implications for human capital development and adult health and socioeconomic attainments. This work provides a broader view of the mechanisms by which infant health affects long-run outcomes. By examining life course effects of birth weight across a broad range of subsequent outcomes, we attempt to shed light on the mechanisms through which differences in health at birth translate into differences in adult outcomes. Taken all together, these findings point toward the important interconnection between health and socioeconomic status as a mechanism for the transmission of well-being across generations within the family. Specifically, poor socioeconomic status of parents at the time of pregnancy leads to worse birth outcomes for their children. In turn, these negative birth outcomes have harmful effects on the children's cognitive development, health, and human capital accumulation, and also health and economic status in adulthood. These effects then get passed on to the subsequent generation when the children become adults and have their own children.

II. Conceptual Framework

A simple two-period overlapping generations model of the transmission of health and economic status from parents to children provides the framework for the empirical analyses that follow. The model adopts a simplified version of the basic framework from Becker and Tomes (1986). Some children have an advantage because they are born into families with favorable genetic attributes, which we refer to as the endowment component. Assume endowments are only partially inherited and parents cannot control endowment transmission, but can influence the human capital of their children through investments in their health, learning, and motivation. For example, while the child is in utero, the mother can invest in prenatal care or refrain from smoking. In the model, the central role parents

play in determining the well-being of their children is to guide the level and allocation of investment in the child until the child can make her own decisions. Assume parents are altruistic toward their children in that their children's lifetime utility is a branch of the parents' utility function.

Individuals possess three types of capital in adulthood: health, education, and financial—with health and education comprising two forms of human capital. Since much research demonstrates that investments during childhood are crucial to later development, we assume that the amount of education and health human capital in adulthood are proportional to the amount accumulated and preserved during childhood.

Assume children are born to one of two types of parents—rich or poor. Assume poor parents face credit constraints that prevent them from making worthwhile investments in their children's human capital. Given resource constraints and imperfect capital markets that do not allow parents to borrow against the future potential of their children, low-income parents may suboptimally invest in the human capital of their children at critical stages of development. Moreover, models with liquidity constraints predict that differences in the average level and timing of parental income during childhood across siblings may contribute to sibling differences in later-life success, even when parents care equally for their children.

Assume two periods of life—childhood and adulthood—and that children are born with an initial health stock, H_0. The change in health stock over time is determined by participation in health-promoting activities and the influence of these activities on health, as well as the use of health stock. Following Case and Deaton (2005), the health evolution equation can be specified as:

$$H_{t+1}=\theta m_t+(1-\delta_t)H_t \quad (1)$$

where m_t is the quantity of medical care or other health promoting activities, θ is the efficiency with which these purchases or activities create health, and δ_t is the rate at which health deteriorates at age t.

The rate at which health capital depreciates with age in childhood is partly a biological process which people do not control, but it is also affected by parental investments (e.g., medical care, nutritional diet, exercise equipment) which contribute to health in adulthood. Negative shocks to early-life health may alter the health production function in such a way that reduces the efficiency of health investment and increases the rate at which health deteriorates over time.

The rate of depreciation of the health stock increases with age and with the nature and intensity of use. This depreciation rate is determined in part by biological processes, but it is also affected by the extent to which health capital is used in consumption and in work (Case and Deaton, 2005). Although all components of capital possessed by individuals—health, education, financial—are unequally distributed, there may be fewer early-life consequences of inherited health because of its distinctive genetic component that may cause the consequences of health inequality to manifest later in life (Muurinen and Le Grand, 1985). At the same time, poor health endowment may impair cognitive development. As a result, the proportional share of health capital in total available human capital is greater for individuals who are born into poorer families. Furthermore, because these components of human capital are to some extent substitutable, health capital will constitute a more important source for producing income and enjoying leisure (Muurinen and Le Grand, 1985).

The degree of persistence in educational attainment and earnings across generations are determinants of the life course trajectory of health capital depreciation because they affect individuals' opportunity sets with respect to living and working conditions in adulthood. For example, in an economically segregated environment with low intergenerational economic mobility, individuals born to poor, less-educated parents residing in low-income neighborhoods are more likely to reach adulthood with insufficient levels of accumulated human capital to qualify for high-skilled jobs that are well paid and do not require manual labor. Thus, they will work disproportionately in physically demanding blue-collar occupations, which will increase the rate of decay of their health capital due to the greater intensity of use (Muurinen and Le Grand 1985; Case and Deaton, 2005). Stress-related life events that result from these living and working conditions may be further exacerbated by an increased need to engage in consumption activities such as smoking and binge drinking that, while hazardous in the long-run, relieve stress in the short-run.

III. Empirical Framework & Econometric Modeling

The conceptual framework emphasizes the fact that the aging process begins at conception and evolves over the life course in response to health shocks, biological deterioration, and investments in and uses of health capital. Health insults *in utero* may lead to greater physiological deterioration of metabolic and immune systems. The neuroscience literature has characterized the critical period of development from conception to age three as one that is extremely sensitive to stressful environmental conditions due to the fact that the speed of growth is more rapid than any other stage of the life course and the nutritional needs are greatest. This conceptual framework fits in with the human capital literature, with early-life health conceived of as a capacity that may influence a broad range of future productivity capacities over the life cycle. For example, adverse intrauterine conditions may lead to cognitive impairments and negatively impact a child's ability to learn. The fetal origins hypothesis provides an explanation of why there may be important interactions between parental health status and parental economic status in their children's human capital accumulation and subsequent adult attainments. This study examines the long-run consequences of poor birth outcomes and parental resources on cognitive development, educational attainment, health status, and labor market outcomes in adulthood. We investigate to what extent and how childhood SES interacts with low birth weight in the production of attainment outcomes over the life course.

The remainder of this section discusses our framework for quantifying the consequences of poor infant health. We define the parameter of interest, describe our primary identification strategy, and discuss the potential determinants of health at birth along with measurement issues that arise in estimating the long-term consequences of adverse conditions *in utero* and poor fetal health, using low birth weight as a marker. While sibling models have been used widely within economics for decades, they have not been frequently used to study the lasting impact of early life events because sibling data have not been available.

To motivate the conditions under which sibling models lead to improved estimates of these effects, consider the following model that embodies the ideas of the sibling approach. Let

$$\begin{aligned} A_{ij} &= f_i + g_{ij} \\ Pre_{ij} &= e_i + w_{ij} \\ BW_{ij} &= \alpha Pre_{ij} + \eta A_{ij} \\ Y_{ijt} &= \beta Pre_{ij} + Post'_{ijt}\delta + X'_{ijt}\phi + \gamma A_{ij} + \varepsilon_{ijt} \end{aligned} \tag{2}$$

where A is a vector of all genetic factors that affects both infant health and adult outcomes (Y) directly. Pre is a vector of prenatal parental investments and environmental factors that affect Y only indirectly through their effects on infant health. We distinguish between the family (i) and individual (j) components of these variables, and both A and Pre have a family (f_i, e_i) and individual (g_{ij}, w_{ij}) components-of-variance structure.[1] Neither the intrauterine nutrient inputs and prenatal environmental conditions (Pre) nor the genetic endowments (A) are observed in the equations above, only birth weights (BW). The birth weight equation is partitioned into the endogenous component A_{ij}, which also appears in the error term of Y, and the exogenous (policy-relevant) component Pr e_{ij}.[2] Y_{ijt} is the observed outcome later in life (e.g., childhood cognitive development, educational attainment, adult health and labor market outcomes) at age t for person j from family i. $Post_{ijt}$ is a vector of post-natal parental investments during childhood, X_{ijt} is a vector of other time-varying individual and family characteristics during childhood, and ε_{ijt} is a random transitory error term.

A key parameter of interest is β, which represents the causal impact of prenatal environmental conditions on children's subsequent outcomes. Birth weight is an indicator of the prenatal cause, not the cause itself. The prenatal environmental influences we have in mind include intrauterine nutrition, maternal stress, maternal smoking and alcohol consumption during pregnancy, the quantity and quality of prenatal care, and neighborhood conditions (e.g., exposure to environmental toxins).[3] We assume that birth weight is a valid policy marker of overall infant health and quality of intrauterine environmental conditions. The validity and usefulness of birth weight as a proxy for infant health would be ideal if the following conditions were met:

1. environmental factors that contribute to low birth weight also reduce infant health;
2. environmental factors that are harmful to infant health increase the risk of low birth weight;
3. the larger the impact of some change in prenatal environmental conditions on low birth weight, the larger the impact on infant health (i.e., all environmental factors affecting birth weight have a proportionate impact on infant health).

While low birth weight is an imperfect measure of poor health at birth, it has long been established as a leading indicator of poor infant health, and is the most commonly used measure in the literature (though there may be better measures, see Almond, Chay, and Lee, 2005). We acknowledge, however, that birth weight as a standard measure of the health of newborn babies does not fully capture the negative effects adverse conditions *in utero* may have on fetal health. While the aforementioned ideal conditions may not hold perfectly in practice[4], assume they hold approximately (for the purposes of illustration).

[1]For ease of exposition, we will first assume there are no interactive effects of family background and genetic factors with birth weight and other observable characteristics.

[2]Below we also consider an extension that allows Pre to contain an endogenous component and discuss how parameter estimates can be interpreted in that case.

[3]Prior evidence has demonstrated how environmental conditions experienced *in utero* influence birth weight. Maternal smoking during pregnancy is the leading risk factor for low birth weight in the US. There is evidence that elevated maternal stress during the prenatal period reduces birth weight and increases the probability of prematurity (e.g., see recent studies of Eskenazi et al. (2007) and Camacho (2008) using research designs that capitalize on natural experiments). On the related question of whether socioeconomic conditions in utero impact infant health, we find supportive evidence that maternal SES early in the child's life matters. In supplementary analyses (not shown but available upon request), we find that there is an interactive effect between income during pregnancy (poverty) and whether the mother was born low weight, and the probability the mother has a low birth weight child. In particular, in sibling fixed effect models also estimated using the PSID, we find that increases in income increase birth weight by much more if the mother was low birth weight herself. Previous research has identified the following key risk factors of poor health at birth: poor nutrition, maternal health (including stress); maternal low SES (poverty; health insurance coverage); maternal smoking; lack of or delayed timing of prenatal care (first trimester); genetic traits or hereditary risk factors (Institute of Medicine, 2001).

More formally, we assume that $\beta = \theta\alpha$ where θ is a scalar, $\frac{\partial Y_{ijt}}{\partial Pre_{ij}} = \left(\frac{\partial Y_{ijt}}{\partial BW_{ij}}\right) * \left(\frac{\partial BW_{ij}}{\partial Pre_{ij}}\right)$, and that prenatal environmental factors that increase birth weight (infant health) improve infant health (birth weight), and that larger impacts on birth weight imply larger impacts on infant health. Thus, substituting this equality into equation (2), the equation can be equivalently re-expressed as:

$$Y_{ijt} = \theta BW_{ij} + Post'_{ijt}\delta + X'_{ijt}\phi + (\gamma - \theta\eta)A_{ij} + \varepsilon_{ijt} \quad (3)$$

Under these (provisional) assumptions, the parameter θ represents the causal effect of birth weight (due to prenatal environmental factors) on subsequent attainment outcomes.

Following Rosenzweig and Wolpin (1995), assume generations are linked via the transmission of genetic endowments according to the following process: any child born to the same parents has a component $A^m/2$ from the mother and $A^d/2$ from the father that are the same for all siblings and a unique idiosyncratic component g. A part of the common component is transmitted across generations,

$$\rho\left(\left(A^m{}_{0i} + A^d{}_{0i}\right)/2\right) \equiv f_{1i}. \text{ Thus, for the son of parents } i,$$
$$A_{1ij} = \rho\left(\left(A^m{}_{0i} + A^d{}_{0i}\right)/2\right) + g_{1ij}, \text{ and for child } k \text{ of son } j,$$
$$A_{2ijk} = \rho\left(\left(A^m{}_{1ij} + A^d{}_{1ij}\right)/2\right) + g_{2ijk}.$$

The parent-child endowment covariance is $\operatorname{cov}\left(A_{1ij}, A_{2ijk}\right) = \frac{\rho}{2}\left(\sigma^2_A + \sigma_{AA'}\right)$, where $\sigma_{AA'}$ is the covariance between endowments of parents that is determined by the degree of assortative mating.[5]

Therefore, the regression of Y on BW produces the following coefficient:

$$\begin{aligned}&\frac{cov(Y_{ijt}, BW_{ij})}{var(BW_{ij})} \\ &= \theta\left[\frac{var\left(Pre'_{ij}\alpha\right)}{var(BW_{ij})}\right] \\ &+ \frac{cov\left(A'_{ij}\eta, A'_{ij}\gamma\right) + cov\left(A'_{ij}\gamma, Pre'_{ij}\alpha\right) + \theta cov\left(A'_{ij}\eta, Pre'_{ij}\alpha\right)}{var\left(BW_{ij}\right)} \\ &+ \frac{cov\left(Post'_{ijt}\delta, A'_{ij}\eta\right) + cov\left(Post'_{ijt}\delta, Pre'_{ij}\alpha\right)}{var\left(BW_{ij}\right)}.\end{aligned} \quad (4a)$$

Or, equivalently,

[4]For example, the estimated long-term impact of birth weight may differ by the source of birth weight differences used to identify the effect. As will be discussed later in the paper, these issues may be important to consider in interpreting results across studies with different research designs and sample populations.

[5]A similar set-up is also used by Becker (1981) and implies regression to the mean by the factor ρ in each generation.

$$\begin{aligned}\frac{cov(Y_{ijt},BW_{ij})}{var(BW_{ij})} &= \theta\left[\frac{var\left((e_i+w_{ij})'\alpha\right)}{var\left((e_i+w_{ij})'\alpha+(f_i+g_{ij})'\eta\right)}\right] \\ &+\frac{cov\left((f_i+g_{ij})'\eta,(f_i+g_{ij})'\gamma\right)+cov\left((f_i+g_{ij})'\gamma,(e_i+w_{ij})'\alpha\right)}{var\left((e_i+w_{ij})'\alpha+(f_i+g_{ij})'\eta\right)} \\ &+\frac{\theta cov\left((f_i+g_{ij})'\eta,(e_i+w_{ij})'\alpha\right)+cov\left(Post'_{ijt}\delta,(f_i+g_{ij})'\eta\right)}{var\left((e_i+w_{ij})'\alpha+(f_i+g_{ij})'\eta\right)} \\ &+\frac{cov\left(Post'_{ijt}\delta,(e_i+w_{ij})'\alpha\right)}{var\left((e_i+w_{ij})'\alpha+(f_i+g_{ij})'\eta\right)}.\end{aligned} \quad (4b)$$

There are four primary reasons we might observe a positive association between birth weight and adult outcomes (Y) in an OLS model. It may reflect: (1) the causal effect of prenatal inputs and fetal environmental conditions (first term of (4a)); (2) a positive correlation between genetic determinants of birth weight and adult outcomes (i.e., second term in numerator of (4a)); (3) a positive correlation between prenatal inputs and genetic factors (i.e., third and fourth terms in numerator of (4a)); (4) a positive correlation between determinants of birth weight and post-natal parental investments during childhood (i.e., fifth and sixth terms of (4a)). Thus, the association between birth weight and adult outcomes may overstate the true causal parameter θ due to being confounded by parental SES and genetic factors. However, this bias may be offset by downward bias induced by measurement error in the policy-relevant variation (Pre), where we observe differences in intrauterine environmental conditions with classical error $A'_{ij}\eta$. The resulting attenuation bias, $\left[\frac{var\left(Pre'_{ij}\alpha\right)}{var(BW_{ij})}\right]$, is decreasing in the signal-to-noise ratio (i.e., birth weight differences due to environmental influences versus genetic endowment).[6]

Unobservable differences across children in parental investments, quality of parenting received, parental resources, abilities, and/or personality traits or genetic characteristics that are correlated with early life health and which influence outcomes in adulthood are potential sources of bias in traditional OLS models. To the extent these characteristics are family-specific, our sibling design will enable us to control for these sources of unobserved heterogeneity. Our research design attempts to control for unobserved family background characteristics that might be associated with both low income and low birth weight by estimating models with sibling fixed effects. The research design eliminates confounding from shared unobserved family background characteristics, and attempts to restrict the identifying variation to temporary shocks experienced during the prenatal period, which induced low birth weight, that do not appear to persist into the post-natal environment.

The typical variation in birth weight between a pair of siblings is about 55 percent of the typical variation between any randomly chosen infant pair. The within-family standard deviation of birth weight is still 314 grams between siblings (see e.g., Oreopoulos et al., 2008; similar estimates obtained from PSID data). To put these magnitudes in perspective, bear in mind that maternal smoking during pregnancy is the most significant modifiable cause of low birth weight incidence, and the average difference in birth weight between a newborn with a mother who smokes and one with a mother who does not is 285 grams (Almond, Chay, and Lee, 2005).

The regression of sibling differences in attainment outcomes ($\Delta Y_{i\cdot t}$) on sibling differences in birth weight (ΔBW_i) yields:

[6]The signal-to-noise ratio is a function of the extent birth weight differences are due to prenatal environmental influences versus genetic endowment differences.

$$\begin{aligned}\frac{cov(Y_{i\cdot t},\Delta BW_{i\cdot})}{var(\Delta BW_{i\cdot})} &= \theta\left[\frac{var((\Delta w_{i\cdot})'\alpha)}{var((\Delta w_{i\cdot})'\alpha+(\Delta g_{i\cdot})'\eta)}\right] \\ &+\frac{cov((\Delta g_{i\cdot})'\eta,(\Delta g_{i\cdot})'\gamma)+cov((\Delta g_{i\cdot})'\gamma,(\Delta w_{i\cdot})'\alpha)+\theta cov((\Delta g_{i\cdot})'\eta,(\Delta w_{i\cdot})'\alpha)}{var((\Delta w_{i\cdot})'\alpha+(\Delta g_{i\cdot})'\eta)} \\ &+\frac{cov((\Delta Post_{i\cdot t})'\delta,(\Delta g_{i\cdot})'\eta)+cov((\Delta Post_{i\cdot t})',(\Delta w_{i\cdot})'\alpha)}{var((\Delta w_{i\cdot})'\alpha+(\Delta g_{i\cdot})'\eta)}.\end{aligned} \quad (5)$$

The bias in the within-family estimator is smaller than the bias in the OLS estimator under the following condition: the common family component (i.e., family-level determinants) accounts for a larger fraction of unobservables correlated with both birth weight and the adult outcome than the corresponding fraction the common family component accounts of parental pre-natal investment and fetal environmental factors. The inconsistency of the between-siblings estimator is less than that of the conventional OLS estimator if endogenous variation comprises a smaller share of the between-sibling variation in birth weight than it does of the between-families variation (Griliches, 1979; Bound and Solon, 1999).

There are a number of reasons why we expect this to be the case. First, sibling differences in prenatal investments are likely to be unrelated to their genetic endowment differences because parents typically do not know differences in child-specific endowments until after birth (the third and fourth terms of above equation), especially during the period which our adult sample was born, 1951 to 1975. Second, it is likely that the family component comprises the dominant share of the correlation between unobservable pre- and post-natal parental investments in childhood. In adopting a within-family estimator, we are eliminating approximately half of full sample variation in birth weight. Therefore, in order for bias resulting from postnatal investments that are correlated with prenatal inputs and conditions to be reduced, it is necessarily the case that the family component of this bias is larger. To the extent that prenatal parental inputs are important vis à vis prenatal environmental conditions, it is plausible that these are weakly correlated with postnatal investments, which is more likely with the inclusion of our extensive set of observable controls.

If we assume the family fixed effect fully captures all unobserved parental inputs during childhood, and we also assume parents have child-neutral preferences whereby siblings receive the same amounts of these inputs, then the sibling fixed effect estimator produces the following coefficient:

$$\frac{cov(\Delta Y_{i\cdot t},\Delta BW_{i\cdot})}{var(\Delta BW_{i\cdot})}=\theta\left[\frac{var((\Delta w_{i\cdot})'\alpha)}{var((\Delta w_{i\cdot})'\alpha+(\Delta g_{i\cdot})'\eta)}\right]+\frac{cov((\Delta g_{i\cdot})'\eta,(\Delta g_{i\cdot})'\gamma)}{var((\Delta w_{i\cdot})'\alpha+(\Delta g_{i\cdot})'\eta)}. \quad (6)$$

Full biological siblings share on average 50 percent of their genetic make-up, so genetic differences between non-identical siblings remain and can be a source of bias (as reflected in the numerator of the second term of (6)). We expect genetic endowment to be positively correlated with birth weight and indicators of well-being in adulthood, so this may lead us to overstate the consequences of poor infant health. However, Black *et al.* (2007) report long-run effects of birth weight that are similar for monozygotic and dizygotic twins, suggestive that sibling birth weight differences due to genetic endowment differences may not represent a significant source of bias empirically. Moreover, this bias may be offset by downward bias induced by measurement error in the policy-relevant variation (*Pre*), where we observe sibling differences in intrauterine environmental conditions with classical error $A_{ij}'\eta - A_{ik}'\eta$. The resulting attenuation bias, $\left[\frac{var((\Delta w_{i\cdot})'\alpha)}{var((\Delta w_{i\cdot})'\alpha+(\Delta g_{i\cdot})'\eta)}\right]$, is decreasing in the signal-to-noise ratio (i.e.,

sibling birth weight differences due to environmental influences versus genetic endowment). For similar reasons as discussed above, sibling differences in birth weight may provide a more useful signal of the quality of the intrauterine environment than between-family differences in birth weight (additional reasons discussed below).

A key question in whether OLS models versus sibling fixed effect models yield estimates that are closer to the true underlying parameter of interest (i.e., θ and ultimately, β; recall $\beta = \theta a$) is which method is able to uncover a greater proportion of identifying variation in birth weight that is due to prenatal environmental factors. Let $\rho_A \equiv \frac{\sigma_f^2}{\sigma_A^2}$ represent the proportion of total variation in genetic factors shared between siblings; and let $\rho_{Pre} \equiv \frac{\sigma_e^2}{\sigma_{Pre}^2}$ represent the proportion of total variation in prenatal environmental factors due to common family background characteristics. Based on the assumptions of the model set forth above, it can be shown that the bias in the sibling estimator will be smaller than the OLS estimator only if $\rho_A > \rho_{Pre}$ (i.e., if the family components account for a larger proportion of variance in genetic (A) than prenatal environmental factors (Pre)). Recent evidence from studies that have attempted to separate different components of familial associations in birth weight (i.e., parental genetic factors (f); fetal-specific genetic factors (g); shared sibling environmental factors (e); sibling-specific environmental factors(w)) report strong supportive evidence that this is indeed the case. In particular, Lunde *et al.*'s (2007) estimates of these components imply that ρ_A is in the range of 0.42–0.5, while ρ_{Pre} is roughly 0.3.[7]

Potential threats to identification for our sibling fixed effect models

Between-family comparisons and sibling comparisons that examine the association between birth weight and adult outcomes generally cannot make strong causal statements about birth weight effects since the underlying causes of low birth weight may also be causally implicated in later outcomes. Sibling differences in prenatal environmental conditions (using low birth weight status as a marker) that are positively correlated with sibling differences in post-natal parental investments received is one such potential source of bias.

In the discussion above, we assumed Pre_{ij} represent exogenous factors that only impact children's subsequent outcomes indirectly through their impacts on infant health. One additional consideration would be to relax this assumption by allowing for the possibility of time-varying sibling-specific events that occur during the prenatal period (of sibling j) that: 1) are correlated with birth weight (of sibling j); 2) persist into the early post-natal period; and 3) differentially impact that sibling j more than sibling k. (i.e., this would allow for the possibility of endogeneity between "Pre_{ij}" and the error term (ε_{ijt}) of the outcome equation (2)). For example, suppose that there is a source of maternal stress that affects the pregnancy of one sibling (e.g., stress induced by the loss of a job by the wife or husband)) but not the other, and that has a sibling-specific effect (perhaps because the early postnatal period is also important, so the younger sibling continues to be affected by the shock but an older sibling is unaffected). In this case, low birth weight may only reflect this alternative event.[8]

[7]Lunde *et al.* (2007) use medical birth registry data of over 100,000 families and fit a specific causal model that included genetic heritability (reflecting the genes in the child), a separate term for variation controlled by maternal genes, a common sibship environment (with separate terms for full and maternal half siblings), and separate components for full and half siblings. Maximum likelihood methods were used to estimate path coefficients under their causal model.

[8]On the other hand, transitory shocks to parental SES may be an important causal source of birth weight differences. For example, Lindo (2010) use detailed work and fertility histories from the PSID to estimate the impact of parents' job displacements on children's birth weights. He compares the outcomes of children born after a displacement to the outcomes of their siblings born before using mother fixed effects. He finds that husbands' job losses have significant negative effects on infant health, with impacts concentrated on the lower half of the birth weight distribution.

More generally, prenatal environmental factors that influence birth weight may also be causally implicated in children's subsequent outcomes. We minimize this potential source of bias with the inclusion of sibling-specific controls for early-life factors such as mother's marital status at birth, wantedness of pregnancy, childhood stage-specific parental income measures (as will be discussed further in Section IV).

In light of these considerations, the role of birth weight remains difficult to interpret, except as a proxy for events in intrauterine life that are reflected in birth weight. It will remain unclear whether it is birth weight that really matters or whether it is other, pregnancy-specific conditions associated with birth weight that ultimately matter. Our estimates may be picking up a range of potential early life influences that we can only proxy using general birth outcomes. The most convincing identification strategy requires variation in early health conditions that is not confounded by other factors, such as parental SES and family background, that might also affect adult health. While the sibling design represents an improvement for identification purposes over traditional OLS models, this is a limitation that cannot be easily solved using the sibling difference approach. This underscores the difficulty one confronts to identify *why* early-life conditions matter for long-term outcomes.[9] Despite these limitations, using sibling differences in birth weight to gain insight into the composite impacts of infant health and its prenatal inputs—prenatal environment (e.g., exposure to stress in-utero, exposure to toxins, poverty, access to health insurance coverage, or other social stressors) and maternal behavior during pregnancy (e.g., smoking, drinking, diet/nutrition, timing and quality of prenatal care)—on long-term outcomes provide an important set of new evidence and contribution to the literature.[10]

Analysis of identical twins can more fully account for the unobserved genetic factors and therefore have greater internal validity than sibling models. This approach has been used in recent papers (e.g., Black *et al.*, 2007; Royer, 2009; Oreopoulos, Stabile, Walld, and Roos, 2008). While twin studies have some advantages over sibling designs (as used in our study) in that shared aspects of the uterine environment and (with monozygotic twins) genetic endowments are controlled for, a disadvantage is that twin births, which account for only 1–2 percent of all births, are not representative and therefore may suffer from low external validity. For example, most twins do not go the full 40 weeks gestation that is the norm for singleton births (and many do not make the 37 week cut-off for "maturity" status). Twins are born significantly lighter (e.g., the median twin birth is less than 5.5 pounds), and they are much more likely to be born premature with birth complications. Furthermore, while within-twin differences in birth weight reflect only differences in fetal nutrition, twin studies do not permit the identification of the effects of low birth weight induced by prenatal exposure to stress or maternal smoking for example. While in developing country contexts, prenatal nutrition may be the most relevant focal point. However, in the US context, stress may be a more relevant risk factor than nutrition in terms of prevalence among a greater number of pregnant women, especially low-income women. This may allow greater

[9]For example, maternal pre-pregnancy hypertension and pregnancy-induced hypertension both restrict fetal growth. If the mother's tendency to be hypertensive is passed on to her child (either genetically or by means of vascular programming due to the hormonal or metabolic environmental conditions of pregnancy), her child will be more likely to develop subsequent hypertension, but the underlying cause of the hypertension would be the mother's hypertension, not the associated reduced fetal growth. It is difficult to control for these types of potential sources of confounding.

[10]As with other associations, additional insight about causality can be gained by examining the results of instrumental variable approaches and "natural experiments". Instrumental variables approaches, in theory, can produce unbiased estimates of the long-term effects of infant health and prenatal inputs. In practice, however, such methods are difficult to implement empirically because of the difficulty of identifying valid instruments. "Natural experiments", when they arise, provide a useful way of isolating causal effects, but they rarely allow for the estimation of multiple inputs and may yield estimates that are not generalizable. As a result, standard regression techniques remain an important and necessary component of a multi-pronged estimation strategy to identify the long-term effects of infant health and its prenatal inputs. Therefore, it is important to understand the biases and limitations of such methods as well as how those estimates can be improved.

generalizability of our results. The source of birth weight differences may be important to consider in interpreting results across studies with different research designs and sample populations.

Additionally, the identifying variation in twin studies comes exclusively from differences in intrauterine growth rates, while differences in gestation length account for roughly 60 percent of birth weight differences among all births (Almond, Chay, and Lee, 2005). Sibling correlations in birth weight are roughly 0.5 and have been shown to persist after adjustment for gestation (Robson 1978; Tanner et al., 1972). In contrast, estimates of sibling correlations in the duration of gestation are much lower (Rosenweig and Wolpin, 1995; Wang et al, 1995). Moreover, it has been shown that maternal birth weight is more strongly associated with the infant's intrauterine growth than with gestation (Klebanoff and Yip, 1987; Melve and Skjaerven, 2002). Taken together, this suggests rates of intrauterine growth have a stronger family component than gestation length. Thus, while sources of birth weight variation in twin studies are identified solely from differences in intrauterine growth rates, identifying variation in sibling models of singletons arise from sibling differences in both intrauterine growth rates and gestation. These differences in the sources of identifying variation have implications for comparability of estimates across different empirical research designs if the consequences of low birth weight that result from prematurity are not symmetric to those that emanate from intrauterine growth retardation.

Moreover, the set of childhood and adult outcomes that can be studied using twin data in the US is substantially limited. The impact of early life factors may be much broader than these outcomes and include cognition, adult health, and labor market outcomes. Therefore, the twins' estimates from prior studies and the estimates presented in this study using national data are *complements*, the combination of which allows a much richer understanding of the impact of poor birth outcomes across all births, not just twins, and across a broader set of developmental, economic, and health outcomes across the life course.

In extensions of our analyses, we considered additional issues and their likely impacts on the sibling fixed effect estimates. First, parents may respond to poor birth outcomes by directing more – or fewer – resources to the disadvantaged child within the family (Rosenzweig and Wolpin, 1995). If parental investment is compensatory to children's endowments, then the consequences of the poor birth outcome will be mitigated. Alternatively, if the investment is reinforcing, then the effects will be magnified. While the empirical evidence is inconclusive, the evidence in Behrman, Pollack, and Taubman (1982) and Behrman and Rosenzweig (2004) is consistent with parental preferences that promote child-equality. Second, if poor birth outcomes or unintended births affect the entire family - as they would if they reduced resources available to all children - then estimates obtained from sibling differences may be biased downwards. That is, estimates obtained from sibling differences measure the impact of a poor birth outcome above and beyond the impact felt by all children in the family. Third, parents may respond to poor birth outcomes of prior children by choosing not to have additional children or postponing subsequent childbearing. The subset of parents who choose to have additional children after experiencing a poor birth outcome may therefore be a positively selected sample who expects favorable birth outcomes.

Each of these three cases is a form of within-family heterogeneity in parental investment, endogenous fertility, or inter-sibling effects. A series of analyses were conducted to investigate these effects. First, we examined how predictive having a prior low birth weight child is on subsequent fertility behavior, accounting for an extensive set of family background controls. Second, we compared sibling fixed effect estimates among siblings who are four or more years apart with those who are less than four years apart. Third, we tested whether sibling fixed effect estimates in families in which the first-born was low birth

weight differed from those estimated in families wherein a later-born sibling was low birth weight (controlling for observable differences in these two types of families).[11] Finally, to attempt to gauge the relative importance of the heritable component of birth weight, we compared estimated effects of low birth weight for full biological siblings with models that include step-siblings who grew up together. Data limitations and small sample sizes precluded definitive evidence on each of these three aspects. However, the analyses that were undertaken indicated that these factors do not significantly bias the sibling estimates.

IV. Data

The PSID began interviewing a national probability sample of families in 1968, with an oversample of low-income African-Americans. These families were re-interviewed each year through 1997, when interviewing became biennial. All persons in PSID families in 1968 have the PSID "gene," which means that they are followed in subsequent waves. In addition, anyone born to or adopted by PSID sample members acquires the PSID "gene" themselves and therefore is followed. When children with the "gene" become adults and leave their parents' homes, they become their own PSID "family unit" and are interviewed in each wave. This sample of "split offs" has been found to be representative (Fitzgerald, Gottschalk and Moffitt, 1998a). Moreover, the genealogical design implies that the PSID sample today includes numerous adult sibling groupings and parent-child groupings who have been members of PSID-interviewed families for nearly four decades.

Two PSID samples – the adult sample and the child sample -- are the focus of the study. (A detailed discussion of the two samples is available in the appendix.) What we call the adult sample consists of PSID sample members who were children when the study began and who have been followed into adulthood. Specifically, we choose PSID sample members born between 1951 and 1975, which consists of children 0–16 years old in the first wave of interviewing in 1968, plus children born into the PSID sample between 1968 and 1975. We then obtain all available information on these individuals for each wave, 1968 to 2003. Therefore, by 2003 the oldest person in the adult sample is 52 and the youngest is 28.

While a rich array of adult outcomes - completed education, adult health status, labor market earnings, and wages - can be assessed for the adult sample, relatively limited information is available about childhood outcomes. A much richer set of childhood information is available for a second sample, which we call the child sample. In 1997 children 0–12 years old in PSID families and their caregivers were administered a series of instruments as part of the Child Development Supplement (CDS). (See Mainieri, 2005, and Mainieri and Grodsky, 2006, for a detailed description of the CDS.) Up to two children within each PSID family were interviewed in person, and these children were then interviewed in person again in 2002/2003. We utilize information about birth outcomes (including birth weight, gestation, placement in neonatal intensive care unit), cognitive ability (including the Woodcock-Johnson), and health status (including general health status as reported by the parent) for this child sample from both waves of the CDS.

The key birth outcome variable examined in both the adult and child samples is birth weight. For the adult sample, mothers reported in 1985 whether their child (i.e., the adult) was born low birth weight, defined as less than 5.5 pounds.[12] For the child sample, exact birth weight is reported by their mothers during the interview following the birth. For example, the parent

[11]We estimated a generalized correlated random effects model to employ the specification test of the fixed effect model first proposed by Chamberlain (1982).

[12]Although the PSID low birth weight information for the adult sample is based on retrospective maternal reports, previous validation studies have demonstrated that comparisons of maternal birth weight reports and those from vital records show high rates of agreement (Baker et al., 1993; Klebanoff and Graubard, 1986).

of a child 10 years old in 1997, and therefore born in 1987, was asked in 1988 the weight of the newborn. Information collected in the PSID on the age of onset of a variety of specific health conditions suggests that very few of the low birth weight individuals in our sample experienced birth defects. Thus, it is unlikely that the presence of birth defects drives the underlying relationships between low birth weight and child/adult outcomes analyzed in this paper. We also found no evidence that estimates from the adult sample suffer significant bias from health-related attrition due to selective mortality among individuals born at low weight; any potential bias suggests that early mortality will tend to reduce the estimated effect of birth weight on later outcomes.

Mother's pregnancy intentions for each child are available for the adult sample. These retrospective reports provided in 1985, which have been shown to be valid (Joyce, Kaestner, and Korenman 2002), elicit whether the mother wanted the specific child at the time of pregnancy and, if so, whether the pregnancy was at the right time, too soon, too late, or they had no timing preference.[13] In contrast to most previous research, we differentiate between unwanted and mistimed births in our empirical analysis.

The key childhood and adulthood health outcome examined is general health status (GHS), which is available in both waves of the CDS and in the core adult survey of the PSID from 1984 through 2003. The general health status question is: "Would you say your health in general is excellent, very good, good, fair, or poor?" GHS is highly predictive of morbidity measured in clinical surveys, and it is one of the most powerful predictors of mortality, even when controlling for physician-assessed health status and health-related behaviors. (For reviews of this extensive literature, see Idler and Benyamini, 1997, and Benyamini and Idler, 1999.) GHS is also frequently used as a global measure of health status and allows us to compare findings with those from related studies such as Case, Fertig, and Paxson (2005) and Currie and Stabile (2003).

In order to scale the GHS categories (i.e., excellent, very good, good, fair, poor), we use the health utility-based scale that was developed in the construction of the Health and Activity Limitation index (HALex). (A discussion of the various options for treatment of the GHS variable is described in the appendix.) The HALex scores associated with GHS categories are based on the U.S. National Health Interview Survey, which contains a fuller health instrument than utilized in the PSID. A multiplicative, multiattribute health utility model was used to assign scores and quantify the distance between the different GHS categories. The technical details of the scaling procedures are discussed at length elsewhere (Erickson, Wilson, and Shannon, 1995; Erickson, 1998). Thus, using a 100-point scale where 100 equals perfect health and zero is equivalent to death, the interval health values associated with GHS used in this paper are: [95, 100] for excellent, [85, 95) for very good, [70,85) for good, [30,70) for fair, and [1,30) for poor health. Consistent with previous research, the skewness and nonlinearity of this scaling is reflected in the fact that the "distances" between excellent health, very good health, and good health are smaller than between fair and poor health. This scaling is currently used by the National Center for Health Statistics to estimate health-related quality of life measures and years of healthy life (Erickson, Wilson, and Shannon, 1995). We then estimate all of the regression models using the interval regression method. While the HALex approach with interval regressions is superior to alternatives, as described in the appendix, we also estimated models using the same explanatory factors but employing two commonly used alternative models: the distinction between fair/poor health

[13]Researchers have questioned the validity of information about pregnancy intention because of concern that parents may engage in "*ex post* rationalization:" After a child is born, they may disproportionately report the pregnancy is intended. Using data containing information on pregnancy intention collected both during pregnancy and after birth, Joyce, Kaestner, and Korenman (2002) find no evidence that the retrospective assessment of pregnancy intention produces misleading estimates of either the number or consequences of unintended births.

and good/very good/excellent health in a linear probability model, and an ordered probit model. The substantive conclusions are unchanged, and we report some estimates in the tables based on these models.[14]

Income is the total for the family in which the child lives, and it is measured at various points in childhood (noted in each table). Adult earnings are total labor market earnings during the previous calendar year. All dollar values are expressed in 1997 dollars using the CPI-U. Cognitive ability is measured by the Woodcock-Johnson standardized test (Woodcock and Mather, 1990), which is widely used by developmental psychologists. Details of the tests and their scoring are described in the appendix. Low birth weight is defined as a birth weight less than 5.5 pounds.

We focus on men because of the differences in health status, health behavior, and labor market outcomes for men and women, and the complexity of health status changes for women during the childbearing years. However, to increase sample sizes, females are included for analyses where the dependent variables are birth outcomes, childhood health, and completed years of education; we found no gender differences in the effects of early life factors on childhood health and completed years of education.

In addition to sibling fixed effects, an extensive set of child-specific controls are included in the models (except where indicated) to minimize potential omitted variable bias. These controls include race, birth order, maternal age at birth, birth cohort dummies, pregnancy intentions, and an indicator for whether the child was born into a two-parent family. In results not shown, the inclusion of changes in family structure during various stages of childhood yielded similar coefficient estimates for our key explanatory variables of interest. For each dependent variable, we examined alternative functional forms of the key explanatory variables to best fit the data. As a result, the functional forms vary to some degree across the models. In all regression models, standard errors are clustered at the person level.

V. Estimates

Simple Within-Sibling Comparisons for All Outcomes

Using multivariate models, subsequent sections describe estimates of the impact of birth weight and other early life factors on a wide array of outcomes. These models conclude that being born low weight has a negative impact on various outcomes over the life course, even after controlling for sibling fixed effects and an extensive set of factors. The conclusions drawn from these models are consistent with the findings from a simple comparison of outcomes among siblings in which at least one sibling was born low birth weight and at least one was not, i.e., discordinant sibling groups. Table 1 reports the outcomes for the discordinant sibling groups, with outcomes for the low birth weight sibling in column [1] and all other siblings reported in column [2]; the difference is reported in the final column. We use the additional information on birth outcomes that is available in the child sample to define poor birth outcome as being born low weight or prior to 37 weeks (i.e., premature) instead of just being born low weight; therefore, just in Table 1, and only for the outcomes in childhood, the contrast is with poor birth outcomes defined in this manner.

The simple differences in childhood outcomes, which are measured at age nine on average, show that the low birth weight siblings are 8.7 percentage points more likely to be in poor/fair/good health (versus in excellent/very good health) and score 4–7 points lower (which is

[14]Ordered probit models produced qualitatively similar patterns of results.

also 4–7 percent lower relative to the average test score) on Woodcock-Johnson achievement tests, depending on the subject matter.

High school dropout rates are 3.1 percentage points higher for the low birth weight siblings (where the pooled high school dropout rate in the sample is roughly 15 percent), although this difference is not statistically significant. In adulthood (i.e., all ages 18–52), the low birth weight siblings are 6.4 percentage points more likely to be in poor or fair health, 4.8 percentage points more likely not to have positive earnings, have 17.5 percent lower annual earnings (4863/27727) among those with labor market earnings, work 7.4 percent fewer hours during the year (107/1438), and have 12.7 percent lower hourly wages (1.74/13.66). These are large and statistically significant estimates across a variety of outcomes that imply lasting effects of birth weight across the life course. Moreover, the estimated effects on most labor market outcomes imply stronger effects later in the life course. For example, the effects on hourly wages are 10 percent during ages 18–26 (0.97/10.02), increasing to 22 percent during ages 37–52 (4.13/19.10).

Childhood Health

Using the child sample, we see that being born low birth weight is strongly associated with health in childhood (Table 2). Because birth weight – not just whether the child was born low weight – is measured in the child sample, we examined the non-linearities in the effects of birth weight. Several less parametric specifications were estimated, and it was concluded that the largest effect of birth weight was at low birth weight levels and that additional weight for normal-weight babies had no effect. To summarize these findings, we chose to report a spline with the notch at 5.5 pounds and birth weight centered at 3.3 pounds, which is the average weight among low birth weight babies. This model implies being born low birth weight (evaluated at 3.3 pounds) reduces the health index by 4.86 points (column 1). Accounting for sibling fixed effects reduces the estimated impact by more than half, but the remaining estimate of 1.67 is still large and statistically significant (column 2). The OLS estimates imply that among low birth weight babies, a 1-pound increase in weight is associated with a 1.85 point increase in the health index, although no significant relationship is observed in the sibling models. The estimates from the sibling fixed effect models also imply that additional weight beyond 5.5 pounds has no effect on childhood health. Moreover, the effect of low birth weight is well represented by an indicator for being low birth weight. This finding suggests that the models of the effects of birth weight in the adult sample are most likely capturing the important differentials despite the fact that continuous birth weight is not available for that sample.

Columns 3 and 4 examine gestation where the spline has a notch at prematurity (i.e., pregnancy of 37 weeks), and gestation is centered on the average gestation among premature births (i.e., 34 weeks). The estimates imply that longer gestation has beneficial effects for childhood health, but the benefit is only for premature births. This finding is true in the models that account for sibling fixed effects, although including sibling effects reduces the estimate by over half from 0.7552 to 0.2820. Combining gestation and birth weight leads to somewhat similar conclusions; the effects of each of the two individual factors are reduced, but still significant (columns 5 and 6). An exception is for birth weight in the sibling models, where the coefficient is still negative but insignificant. A third birth outcome measure examined is placement in a Neonatal Intensive Care Unit (NICU). We specify this effect with an indicator for placement in an NICU and the number of weeks in a NICU minus one. Childhood health is lower among those who were placed in a NICU when they were born, and this holds between siblings (column 7).

Socioeconomic factors at pregnancy influence childhood health. Having private health insurance coverage during pregnancy improves the child health index by 1.02 points in the

mother fixed effect models (column 9). Increases in income for families with $15,000–$50,000 improve childhood health, with a $10,000 increase translating into a 0.53 percentage point increase for these families (column 11, which include mother fixed effects). There is no effect among the highest income families, and an unexpected negative effect among the lowest income families in the mother fixed effects models. Family income in the year of pregnancy is partly determined by the mother's labor supply. The fact that women may be staying home with an older child (and this might be related to child health) might explain some of the anomalous income results in the child health models. Including health insurance, participation in government transfer programs, and family income in the mother fixed effect models simultaneously, lead to the same substantive conclusions.

Childhood Cognitive Achievement

Table 3 reports estimates of the effects of early life events on cognitive achievement using the child sample, where all models include sibling fixed effects. Measures of both reading and math achievement are strongly influenced by birth weight. Passage comprehension is 10.5 points lower for those at 3.3 pounds (column 1), which is 12 percent of the average test score and equal in size to black-white differences (not reported in table). The spline in birth weight indicates that the majority of the beneficial effect of being born heavier is concentrated among low birth weight children. Additional weight helps babies born low weight, with babies born at 5.5 pounds experiencing no harmful effects on cognitive achievement in childhood. Additional weight above 5.5 pounds has no effect. Placement in a NICU, as an alternative indicator for a poor birth outcome, is also strongly associated with lower achievement in childhood. The sibling fixed effect estimates indicate negative effects of placement in NICU of 3.3 points for a one week stay; each additional week is associated with a reduction of 0.8 points for passage comprehension (column 2).

Family income at pregnancy has a positive effect on childhood achievement among poorer families (i.e., families with income less than $15,000) but not non-poor families (column 3). A $10,000 increase in income among poor families translates into improvements in passage comprehension by 4.8 points, or 6 percent of the average. Income's effect is only partially explained by low birth weight; once birth weight is controlled for, the income effect among the poor declines by about 5 percent depending on the level of income (column 4). At the same time, the effects of birth weight declines but remains large at 4.3 points, or 5 percent of the average score.

Qualitatively, the estimates of the effect on math achievement (Table 3, columns 6–10) parallel the estimates of the effect on reading. That is, there are substantial negative effects of being born low weight, and the effect is concentrated among babies born less than 5.5 pounds. Specifically, being born low weight (i.e., 3.3 pounds vs the weight of an average normal weight baby) is associated with a 7.8 point lower score, which is eight percent of the average score among all children. An additional pound among low weight babies improves scores by 3.8 points; additional weight does not improve applied problem scores among children who were born normal weight. Family income improves math achievement among low-income families but not among richer families (column 8). When birth weight and family income are included simultaneously, income effects are unchanged while the birth weight effects are reduced somewhat, but again remain quite large and statistically significant (column 9).

We also tested whether controlling for general health status in childhood eliminates the effect of low birth weight; we find that it does not. As shown in column (10) of Table 3, estimated effects of low birth weight and low parental income on math achievement remain significant and magnitudes unaffected; the point estimates of low birth weight on reading

achievement are largely unchanged, but become imprecisely estimated with the inclusion of general health status in childhood (column 5).

Completed Education

At the low end of the educational distribution, being low birth weight has substantial effects (Table 4). The estimates from the linear probability model that includes sibling fixed effects implies that low birth weight children are 4.79 percentage points more likely to drop out of high school, or roughly one-third more likely relative to the average dropout rate of 15 percent (column 3). This effect is fairly similar to the estimate without sibling fixed effects (6.7 percentage points in column 2). The estimate is robust to direct controls for family income in childhood (columns 4 and 5). Despite the fact that low birth weight increases the probability of dropping out of high school, the effect on total years of schooling is modest – a reduction of just one-tenth of a year – and imprecisely estimated (column 6).

Adult Health

A series of models that examine the relationship between birth weight and health in adulthood are reported in Tables 5–7. We begin by presenting a model that does not include sibling fixed effects and find that low birth weight is associated with worse health outcomes in adulthood (column 1, Table 5). The magnitude of the relationship is substantial. A useful way to interpret the estimate is in relationship to the size of the effect of age on health, with the effect of low birth weight on adult health equivalent to being 8.7 years older. That is, GHS is 2.369 points lower for adults who were born low weight, which is equal to 8.7 years evaluated at an effect of age of −0.2714.[15]

Several variables among the demographic factors are interesting in their own right. First, there are large racial differences in adult health, which have been widely documented (Anderson, Bulatao, and Cohen, 2004). The gap between whites and blacks is 3.3 points, which is about one point larger than the effects of low birth weight.[16] Second, first births are on average lighter, but controlling for birth weight those who are later in birth order have worse health in adulthood, although the effect size is modest. Third, being born into a single parent family and having an older mother at birth are both insignificant.

Fixed-effects models rely on a sub-sample of families with two or more children. Therefore we check that non-fixed effect model estimates for this sub-sample are similar to those for the overall sample to ensure that any differences in results between non-fixed effects and fixed-effects analyses are due to different statistical procedures rather than different samples. Column 2 restricts the sample to men with brothers in the sample but does not include brother fixed effects, while column 3 includes the fixed effects. The effect of low birth weight increases from −2.88 to −3.77 when the fixed effects are included. While the fixed effect estimate is not statistically significantly larger than the non-fixed effect estimate, the pattern of a larger coefficient is consistent with Smith (2009), who argues that measurement error in childhood health biases the effects on adult outcomes downwards. Additionally, the negative effect of birth order is eliminated when the fixed effects are included (column 3).

As an alternative to the interval regression model of the health index, estimates in column 4 are from a linear probability model of being in poor/fair health vs. in good/very good/

[15]We did not find significant nonlinear effects of age on health in our sample, and the linear specification of age eases interpretation of the birth weight effect in relationship to the age effect.

[16]The black-white gap is of similar magnitude in our data irrespective of whether sample weights are included in the estimation.

excellent health. The conclusions are qualitatively the same; being born low birth weight increases the probability of being in fair or poor health as an adult by 7.03 percentage points.

It has been estimated that more than half of pregnancies in the U.S. each year are unintended, either mistimed or unwanted at conception (Forrest, 1994; Joyce, Kaestner, and Korenman, 2000). Pregnancy intentions collected for the PSID adult sample reveals similarly alarming rates of unintended pregnancies (Table A2). The consequences of unintended pregnancy are of important policy interest in their own right, but we are also interested in whether our estimates of the effects of low birth weight are robust to the inclusion of parental fertility timing preferences. Previous research has found that unintended pregnancy has an adverse effect on maternal behaviors and use of prenatal care. Relatively few U.S. studies have examined the association between pregnancy intention and adverse health and developmental consequences for children beyond infancy. Using the PSID measures of maternal pregnancy intentions, we find significant variation in parental pregnancy intention within the same family. More importantly, adult health is 2.55 points lower for adults whose mother did not want them, relative to adults whose parents wanted them and had them at the preferred time (column 5, Table 5).[17] However, these factors account for none of the birth weight effect; the effect of low birth weight is −3.7 regardless of whether these controls are included.[18]

Effect of birth weight over the life course—We find that the harmful effect of low birth weight increases with age (column 1, Table 6). During ages greater than 36, the low birth weight effect is 5.96 points, while it is smaller (3.06 points), but still substantial, for adults 18–36. The differential effects by age are even greater in relative terms. That is, as implied by the age coefficients in column 1, the health status measure is substantially higher for people younger than 37 than for people 37 or older. This implies that equal sized absolute effects represent larger relative effects at older ages.

Birth weight, childhood health, and adult health—The PSID in 1999 and 2001 asked adults to recall their health in childhood (i.e., ages less than 17) and rate it as excellent, very good, good, fair, or poor. Using this more restrictive sample we investigate the effects of childhood health on adult health, and we examine the extent to which birth weight influences adult health through its effects on health in childhood. Column 2 of Table 6 shows that for this more restrictive sample and accounting for sibling effects, the effect of low birth weight is −6.78. This effect is larger than the effect in column 3 of Table 6 because this sample is much older, and as we saw in column 1 of Table 6, the effects of low birth weight increase with age. Adding retrospective childhood health accounts for a substantial share of the low birth weight effect, lowering it to −4.97 or by about one-quarter. In addition, retrospectively reported childhood health has substantial effects on adult health. Therefore, a substantial share of the effect of low birth weight on adult health works through observable (to the respondent) differences in childhood health. Moreover, if childhood health were more perfectly measured, the effects of low birth weight may have been reduced even further.

Parental income, health insurance, and birth weight—A family's resources in childhood may have a lasting impact on a child's wellbeing, which we investigate in Table 7. The fixed effects models identify the effect of family income from differences in family income between siblings at the same life stage, i.e., ages 13–16. It is important to note that if

[17]Adults whose mother wanted them but had no timing preferences were in worse health as adults, but only 2 percent of births fall into this category.

[18]These estimates differ from the early-life consequences analyzed in Joyce, Kaestner, and Korenman (2000) who do not find significant differences in maternal behaviors or child outcomes among siblings who were mistimed versus wanted pregnancies.

parents' permanent income matters most for their children's adult status attainments, this sibling fixed effect specification represents a very stringent (and perhaps inappropriate) test of the importance of family income because the identification relies on transitory changes in family income to generate between-sibling differences at the same stage in childhood. The family's income-to-needs ratio is used as our measure of parental income (where a value of 1 is equivalent to family income equal to the poverty line).

Because we observe the greatest number of children when they are in their teen years, we focus on the effects of family income when the child was 13–16 years old to boost sample size, although several models were estimated that test whether income received at different stages during childhood have differential effects (0–4; 5–8; 9–12; 13–16 years old).[19] It has been found that income received in the infant and toddler years has a greater effect on educational attainment than income received at other points in childhood (Levy and Duncan, 2000). Case, Lubotsky, and Paxson (2002) find no evidence that stage-specific income matters for health status; rather, it is permanent income that is most important. We find some evidence consistent with the hypothesis that income received at younger ages has greater benefits: income at the youngest ages has the largest effect when income at each stage is included simultaneously (results not shown, available from authors upon request). However, stage-specific income is highly correlated across stages, and the point estimates were not statistically significantly different from each other.

The first two columns of Table 7 examine the direct effect of family income after accounting for sibling fixed effects, with growing up in poverty leading to worse health in adulthood. Relative to adults whose parents had income-to-needs ratios of 1.0 to 2.0, adults who grew up in poverty had 2.13 percentage points lower health, which is equivalent to being 6.7 years older (−2.1252/−0.3171). This effect is unchanged when birth weight is controlled (column 2).

It is also the case that a family's ability to respond to a health shock, such as low birth weight, may mitigate the lasting effect of the shock. We investigate this hypothesis by interacting the low birth indicator with health insurance coverage in childhood, within the fixed effect specification (column 3). Having health insurance in childhood mitigates the effects of low birth weight: the harmful effects of low birth weight are 2.7 times larger for those who were uninsured in childhood. Moreover, this effect persists with controls for childhood poverty status.

Labor Market Outcomes

A series of labor market outcomes are examined: whether the person has positive annual earnings, and then among those with positive earnings, log annual earnings and log wages. For comparison, Tobit models are estimated that include the men with zero earnings. Annual hours were also examined and it was found that all of the effects were on earnings/no earnings and not on hours conditional on working.

We find that low birth weight is strongly associated with future labor market outcomes. The Tobit estimates imply that children born of low weight have $4,583 lower earnings in adulthood (column 7, Table 8). This effect is reduced to $2,966 when sibling fixed effects are accounted for, but is still quite substantial. There are strong effects of birth weight on having any earnings during the year (4.35 percentage points in column 3) even within the sibling models. While the effect on log annual earnings is not statistically significant at the 0.10 level, it is quite substantial at 10.7% (column 6).

[19]We also estimated models that included parental income during pregnancy and birth weight in the same model for the small sub-sample with valid measures of both, but small sample sizes led to imprecisely estimated coefficients.

Effect of low birth weight over the life course—The simple differences between siblings reported in Table 1 implied that the effect of birth weight on labor market outcomes increased with age. This finding is shown in multivariate models in Table 9 where age is interacted with birth weight. Specifically, the effect of low birth weight on having positive earnings increases from 3.77 percentage points at age 25 to 6.27 percentage points at age 35. In addition, the effect of birth weight on log earnings becomes statistically significant once the nonlinear effects are included (column 2 in Table 9). This model implies that the earnings penalty for being born low weight increases from 10.22 percent at age 25 to 15.62 percent at age 35.

Parental income, health insurance, and birth weight—The literature on intergenerational transmission of economic status implies a substantial father-son correlation in income (Solon, 1992; Zimmerman, 1992). Not surprisingly, we find when looking within families by including sibling fixed effects, family income in childhood is closely related to subsequent labor market earnings (columns 5 and 9 in Table 10). Controlling for family income reduces the association between low birth weight and adult labor market earnings, but not substantially (columns 3, 6, and 9 in Table 8 vs columns 2, 6, and 10 in Table 10). For example, the effect of low birth weight on annual earnings (from the Tobit) is reduced from $2,966 (column 9, Table 8) to $2,899 (column 10, Table 10) after childhood family income is included in the model.

Interactions of low birth weight with childhood health insurance are significant and substantial, which parallels the findings with adult health status as the outcome. The harmful effects of low birth weight were felt almost exclusively among children whose parents did not have health insurance (columns 3, 7, 11, and 15 in Table 10). For example, the effects of being born low weight increases the probability of not working (annual earnings) by 7.1 percentage points ($6,222) among adults who did not have health insurance in childhood, while there is no effect among adults who were insured in childhood.

Low birth weight, education, and labor market outcomes—Low birth weight may influence labor market outcomes through its effect on education. We tested this hypothesis by controlling for completed education and found that the effect of low birth weight changed very little. (Compare columns 4, 8, and 12 in Table 10 vs columns 3, 6, and 9 in Table 8.) This finding is consistent with the conclusion that low birth weight did not affect total years of education and implies that the effect of birth weight on adult labor market earnings does not work primarily through completed years of schooling.

Robustness & Interpretation

We have shown that low birth weight is a strong predictor of educational and adult labor market outcomes and health status over the life cycle, and remains so even for sibling comparisons. The role of birth weight remains difficult to interpret, except as a proxy for events in intrauterine life that are reflected in birth weight. There are two plausible interpretations of these findings. One interpretation is that the results provide strong support for a causal role of poor infant health (that interacts with parental SES) on later-life outcomes. A more cautious interpretation of the evidence is that we have documented strong correlations between low birth weight and children's subsequent attainments, some of which may reflect other influences that vary with early health conditions. As discussed in Section III, sibling differences in prenatal environmental conditions (using low birth weight status as a marker) that are positively correlated with sibling differences in post-natal parental investments received are a potential source of bias. In light of this concern, we performed a series of additional robustness checks, which provided evidence inconsistent with this claim.

First, if it were the case that these results were driven by sibling differences in post-natal parental resources/investments, we would expect that as important observed child/sibling-specific characteristics are included in the models such as wantedness of pregnancy, childhood stage-specific parental income, maternal work hours, marital status measures (e.g., during adolescent years), (which are correlated with low birth weight and children's subsequent outcomes) the estimated impact of low birth weight on outcomes would decrease. This does not occur—the estimates remained largely unaffected. Since the inclusion of important observed time-varying factors do not change the estimated effects, it seems unlikely that unobserved factors would. While not conclusive, these findings provide further support we are identifying the impact of low birth weight, not differences in post-natal investments in the child, on adult attainment outcomes.

As corroborating evidence, we tested whether a negative relationship exists between low birth weight and parental investments in the child during the post-natal period within the sibling fixed effect model. For example, we examine among siblings with discordant birth weights, whether the low birth weight child had a differential likelihood of being sent to preschool, private school during K-12 years, and maternal work hours. We find no evidence of such relationships.

As a whole, this evidence lends support to the interpretation of the results that the relationship we uncover between low birth weight and children's subsequent adult attainments over the life cycle do not reflect a lack of parental investment in the child during the post-natal childhood period that is either caused by or correlated with low birth weight.

There are two possible explanations why the sibling fixed effect estimates of impacts of low birth weight are generally larger than the OLS estimates. First, the extensive set of family background factors—parental income, child health insurance coverage, parental smoking, maternal age, mother's marital status at birth, wantedness of pregnancy, etc..—in the OLS models lead to estimates that are biased downwards because low birth weight may be caused by low income and many of these other factors. Thus, including low birth weight and an array of family background characteristics amounts to "over-controlling." This is tantamount to adjustment for a factor that lies on the causal path between infant health and adult health status. Measurement error in birth weight is a second reason to explain the differential effects estimated in the OLS and fixed effect models. Classical random measurement error in birth weight can lead to attenuation bias of estimated coefficients towards zero; recall information is collected from retrospective maternal reports of child birth weight. Non-random measurement error in maternal reports is also possible. For example, the inclusion of mother fixed effects may increase the estimated coefficients by removing the effects of the mother-specific measurement error. Smith (2009), using sibling models to examine the effects of a global measure of childhood health status (from retrospective self-reports) on adult SES outcomes, finds a similar pattern where the fixed effect estimates were generally larger than those from OLS models. Smith argues that there is likely non-random measurement error in the child health measure. If siblings report similarly, then the difference in child health between siblings will be more accurately measured than the difference between random individuals. Thus, in this case, controlling for measurement error by inclusion of mother fixed effects will increase the magnitude (in absolute value) of the estimated coefficients.

Finally, we tested for various sources of selective attrition by low birth weight. We performed a series of tests and the results imply that selective attrition is not a significant source of bias. A detailed discussion is contained in the Data Appendix.

VI. Discussion

This study provides the first evidence on the relationship between early life health outcomes and cognition, human capital accumulation, labor market outcomes, and health status in adulthood for a nationally representative sample of the U.S. population. We find that poor health at birth and limited parental resources (including low income, lack of health insurance, and unwanted pregnancy) interfere with cognitive development and health capital in childhood, reduce educational attainment, and lead to worse labor market and health outcomes in adulthood. These effects are substantial, and they are robust to the inclusion of sibling fixed effects and an extensive set of controls. The results reveal that being born low weight ages people in their 30s and 40s by 12 years, increases the probability of dropping out of high school by one-third, lowers labor force participation by 5 percentage points, and reduces labor market earnings by roughly 15 percent.

Previous studies have demonstrated that birth outcomes themselves are determined to a large degree by social and economic conditions (e.g., Conley and Bennett, 2000; Dehejia and Lleras-Muney, 2004). Additionally, we find that socioeconomic factors influence the lasting impacts of poor infant health when it occurs. In particular, we find that the negative long-run consequences of low birth weight are smaller among children whose families had health insurance, which is consistent with Currie and Hyson (1999) who find that socioeconomic status reduces the harmful effects of low birth weight among British women. In addition, consistent with Case, Fertig, and Paxson's (2005, Figure 1) analysis of Brits born the week of March 3, 1958, the relationship between low birth weight and adult health increases as adults age.

While poor birth outcomes reduce human capital accumulation, this consequence explains only a fraction of the total effect of low birth weight on labor market earnings. This finding is consistent with a number of studies, including Persico, Postlewaite, and Silverman (2004) who find that the benefits to adult labor market earnings for being taller at age 16 is reduced by only 20 percent once completed years of schooling is accounted for; Luo and Waite (2005) who find that two-thirds of the effect of childhood health on adult income remains after controlling for education; and with Smith (2009) who examines the relationship between labor market earnings and retrospective reports of self-assessed health in childhood collected in the PSID. Other pathways through which early life health affects adult labor market outcomes should be examined. Other such channels may be childhood cognitive and non-cognitive skills; we find the former to be strongly linked to birth outcomes and others have shown it to be a powerful predictor of labor market outcomes (Case and Paxson, 2008).

Adult health is positively associated with childhood family income, especially for improvements in income at the very bottom of the income distribution. The average effects over the entire income distribution are smaller, implying that gains in income are likely to translate into substantial improvements in health for a small, although typically more vulnerable, population.

Longitudinal data that include information on initial health conditions and later-life health are rare, which has limited efforts to estimate long-term effects of conditions in utero and during early infancy. Few studies have produced direct evidence over the life course that demonstrate linkages in the intergenerational transmission of disadvantage whereby parental SES during pregnancy influences the risk of poor infant health, which have far-reaching impacts on child health, cognitive function and subsequent effects on children's adult SES and health attainments. Few studies follow individuals from birth through middle age. This study makes contributions to the literature along these lines. Our work contributes to a burgeoning literature on long-run impacts of early life nutritional and psychosocial

environments (including stress). The results shed light on early life antecedents of adult disease and underscore the important role of the early years in human capital formation. Our evidence is also consistent with emerging neuroscience research in animal studies that demonstrate that exogenous in-utero exposure to stress impairs the developing brain of the fetus (Uno et al., 1990; Welberg, Seckl, and Holmes, 2001; Schneider et al., 1992). Prior work has found relatively small short-run effects but larger long-run effects; this may be due to the possibility that some of these effects remain latent during childhood and manifest in adulthood.

By combining results from prior twin studies with the results from our sibling models of cognition, childhood health, education, and health, earnings, and wages in adulthood we obtain a much more compelling understanding of the long-run effects of early life events. Indeed, our findings on the effects of early-life events using the national PSID sample are remarkably consistent with a small but growing set of very recent studies by economists (Case, Fertig, and Paxson, 2005; Van den Berg, Lindeboom, and Portrait, 2006; Black, Devereux, and Salvanes, 2007; Oreopoulos et al., 2008; Almond, 2006; Almond and Mazumder, 2005; Almond and Chay, 2006; Behrman and Rosenzweig, 2004; Berndt et al., 2000; Royer, 2009; Currie and Moretti, 2007; Aizer et al., 2010). These recent studies have examined different countries and settings with divergent health care systems and populations: Brits born the week of March 3, 1958; Dutch born between 1812 and 1912; Norwegians born 1967 to 1997; Canadians in Manitoba born 1978 to 1985 (excluding 1983); Dutch and Minnesotan twins; U.S. women born in the 1960s; children born around 1918 in the U.S.; and births in California 1960–1982 and 1989–2001. The designs of these studies are varied, but all have included some attractive features, and their findings are qualitatively consistent with an extensive epidemiological literature (e.g., Moster et al, 2008; Swamy et al, 2008; Hovi et al, 2007; Heck et al, 2002; and earlier studies summarized in Barker, 1998) and a burgeoning sociological literature (e.g., Blackwell, Hayward, and Crimmins, 2001; Conley and Bennett, 2000; Conley and Bennett, 2001; Elo and Preston, 1992; Luo and Waite, 2005).

Moreover, while there are too few twins in the PSID to support twin analyses, recent evidence from two distinct settings - Canadians born in Manitoba 1978–1982/1984–85 (Oreopoulos et al., 2008) and Norwegians born 1967 to 1997 (Black, Devereux, and Salvanes, 2007) - have concluded that estimates of the effects of early life events on adult outcomes are quite similar in sibling models and twin models. Taken together with these recent diverse studies, we believe the estimates reported here provide compelling evidence for lasting impacts of early life events, and birth weight in particular.

The relationships estimated in our study imply a mechanism for the transmission of well-being across generations within the family. Specifically, poor economic status of parents at the time of pregnancy leads to worse birth outcomes for their children. In turn, these negative birth outcomes have harmful effects on the children’s cognitive development, health, and human capital accumulation, and also health and economic status in adulthood. These effects then get passed on to the subsequent generation when the children, who are now adults, have their own children.

The experiences of the PSID adult sample, which were born 1952 to 1975, do not necessarily represent the experiences of earlier or subsequent cohorts. Medical technology and practices and infant survival for a given weight have changed over time—those with very low birth weights are much more likely to survive than they were in the 1970s (Almond, Chay, and Lee, 2005). At the same time, we do find substantial harmful effects of negative birth outcomes on childhood health and cognitive development for the more recent cohorts, born 1985 to 1997, as represented by the child sample. As the PSID continues to

follow these children researchers will be able to assess the longer-run effects on health and economic status for this cohort.

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Appendix

Samples

Child Sample

The child sample consists of all children interviewed in CDS-I or CDS-II, with descriptive statistics reported in Table A1. The CDS-I sample included all PSID sample members 0–12 living in PSID families as of 1997. Up to two children within the same family were interviewed resulting in a sample of 3,540 children in 2,348 different families in 1997, and 1,132 families had two children interviewed. Interviews for these children were completed again in 2002/2003 when they were 5–18 years old. In total there are 6,447 child-year observations. See Mainieri (2005) and Mainieri and Grodsky (2006) for details about the CDS.

Adult Sample

Given our goal of assessing impacts of early life events as far into adulthood as possible, we chose for the adult sample boys born between 1951 and 1975, which originally consisted of 4,441 boys with the PSID gene. These boys included children 0–16 years old in the first wave of PSID interviewing in 1968 (N=3,502), plus children born into the PSID sample between 1968 and 1975 (N=939). 103 boys have died by 2003. These boys are included in the analyses for the years they are observed alive. We estimated mortality models, but there were too few deaths to precisely estimate any relationships.

Of these 4,441 boys, 2,652 had at least one valid report of labor market outcome in adulthood, i.e., as a PSID head or wife/"wife". 2,745 had at least one valid report of general health status (GHS) in adulthood. Adult GHS is based on reports for PSID heads and wives/"wives" as well as all family members in 1986.

While the decline in the initial sample of 40 percent (38 percent for samples used in the models of GHS) is substantial, it is low given the long period over which these children and their families are followed. For example, among the 17,287 newborns participating in the 1970 British birth cohort sample, 6,454 (37 percent) were not interviewed (i.e., were not in the "observed sample") in 1999/2000 when they were 30 years old. Moreover, studies have concluded that the PSID sample of heads and wives remains representative of the national sample of adults (Fitzgerald, Gottschalk, and Moffitt, 1998b; Becketti et al, 1988), and that

the sample of "split offs" is representative (Fitzgerald, Gottschalk and Moffitt, 1998a). The 95–98% wave-to-wave response rate of the PSID makes this possible. In addition, we have also examined whether birth weight predicts whether the child has an observed adult health or labor market measure, and we find that birth weight is not predictive of this outcome, implying no selective attrition with respect to birth weight.

We have also examined whether birth weight predicts survival until 1968, which is the initial interview year. That is, some children may have been born to PSID families prior to 1968 but not survived until the first interview. Moreover, the children not surviving may have been disproportionately low birth weight. We can examine this issue because PSID mothers report complete birth histories. We modeled the probability that siblings of our adult sample died before 1968 and found that birth weight was not predictive, and the number of such deaths was small.

The ability to conduct analyses comparing siblings is a unique feature of our study. The 2,745 (2,652) boys who reported GHS (labor market outcomes) in adulthood came from 1,444 (1,448) different PSID families; 1,187 families had at least 2 boys. Data are combined across all waves for each person, and in total there are 26,407 (31,610) person-year observations, or an average of 9.6 (11.9) observations per person, for the analyses of adult health (adult labor market outcomes).

Table A2 reports descriptive statistics for the samples used in the models of adult health status, both for the full sample and the sample of boys who have at least one brother reporting GHS. The two samples are quite similar in almost all respects. Low birth weight, which is reported by the mother of the child in questions added to the PSID in 1985, is measured by an indicator taking the value 1 if the newborn was less than 5.5 pounds, 0 otherwise. Exact weight was only collected for births after 1986, and these cohorts are not yet old enough to examine their adult outcomes. Less than 1% of the sample had missing data for birth weight, and these cases were dropped from the analyses. 6.47 percent of the sample had low birth weight. Gestation is not available in the adult sample. Income is the total for the family in which the child lives, and it is measured at various points in the childhood. Earnings are total labor market earnings during the previous calendar year. Drinking and smoking of parents are indicated by whether the family spent any money on these goods. All dollar values are expressed in 1997 prices.

Health Index

A number of previous studies using surveys have demonstrated that a change in GHS from fair to poor represents a much larger degree of health deterioration than a change from excellent to very good or very good to good (e.g., Van Doorslaer and Jones, 2003; Humphries and Van Doorslaer, 2000). More generally, this research has shown that health differences between GHS categories are larger at lower levels of GHS. Thus, assuming a linear scaling would not be appropriate.

To analyze health disparities in the presence of a multiple-category health indicator, three alternative approaches have been used, each with its own set of advantages and disadvantages. The most common and simplest approach is to dichotomize GHS by setting a cut-off point above which individuals are said to be in good health (e.g., excellent/very good/good vs. fair/poor). The disadvantage of this approach is that it does not utilize all of the information on health. Additionally, it uses a somewhat arbitrary cut-off for the determination of healthy/not-healthy, and the measurement of inequality over time can be sensitive to the choice of cut-off (Wagstaff and Van Doorslaer, 1994).

A second approach is to estimate an ordered logit or ordered probit regression using the GHS categories as the dependent variable, and rescale the predicted underlying latent variable of this model to compute "quality weights" for health between 0 and 1 (Cutler and Richardson, 1997; Groot, 2000). The key shortcoming of this approach is the probit and logit link functions are inadequate to model health due to the significant degree of skewness in the health distribution (i.e., the majority of a general population sample report themselves to be in good to excellent health). Van Doorslaer and Jones (2003) assess the validity of using ordered probit regressions to impose cardinality on the ordinal responses comparing it with a gold standard of using the McMaster 'Health Utility Index Mark III' (HUI).[20] They conclude "…the ordered probit regression does not allow for any sensible approximation of the true degree of inequality."

The third approach, adopted first by Wagstaff and Van Doorslaer (1994), assumes that underlying the categorical empirical distribution of the responses to the GHS question is a latent, continuous but unobservable health variable with a standard lognormal distribution. This assumption allows "scoring" of the GHS categories using the mid-points of the intervals corresponding to the standard lognormal distribution. The lognormal distribution allows for skewness in the underlying distribution of health. The health inequality results obtained using this scaling procedure have been shown to be comparable to those obtained using truly continuous generic measures like the SF36 (Gerdtham et al., 1999) or the Health Utility Index Mark III (Humphries and van Doorslaer, 2000) in Canada, but has not been validated as an appropriate scaling procedure using U.S. data. The disadvantage of this approach is it inappropriately uses OLS on what remains essentially a categorical variable and does not exploit the within-category variation in health. This is particularly problematic for the analysis of health dynamics over a relatively short time horizon. Ignoring within-category variation in health will cause health deterioration estimates to be biased and induce (health) state dependence because within-category variation increases when going down from excellent to poor health.

Several surveys have been undertaken that contain both the GHS question and questions underlying a health utility index. In this paper, we adopt a latent variable approach that combines the advantages of approaches two and three above, but avoids their respective pitfalls. Specifically, utilizing external U.S. data that contain both GHS and health utility index measures, we use the distribution of health utility-based scores across the GHS categories to scale the categorical responses and subject our indicators to the transformation that best predicts quality of life. This scaling thus translates our measures into the metric that reflects the underlying level of health. Specifically, using a 100-point scale where 100 equals perfect health and zero is equivalent to death, the interval health values associated with GHS are: [95, 100] for excellent, [85, 95) for very good, [70,85) for good, [30,70) for fair, and [1,30) for poor health.

Woodcock-Johnson Achievement Tests

Woodcock-Johnson Psycho-Educational Battery-Revised (WJ-R) is a well-established and respected measure of intellectual ability, including current developmental status, degree of mastery in reading and mathematics, and group standing. In the CDS-I and CDS-II, three subtests were administered to measure reading and math achievement: the letter-word, the passage comprehension, and the applied problems tests. These scales can be used individually or, in the case of the reading tests, can be combined to create scores for Broad

[20]The McMaster Health Utility Index can be considered a more objective health measure because the respondents are only asked to classify themselves into eight health dimensions: vision, hearing, speech, ambulation, dexterity, emotion, cognition, and pain. The Health Utility Index Mark III is capable of describing 972,000 unique health states (Humphries and van Doorslaer, 2000).

Reading. When applicable, the Spanish version of the WJ-R (Batería-R, Form A) was used for children whose primary language was Spanish.

The WJ-R has standardized administrative protocols. For respondents under 6 years, the interviewer administered two subtests: Letter-Word Identification and Applied Problems. For respondents 6 years and older, the interviewer additionally administered the Passage Comprehension subtest. The Woodcock-Johnson (WJ-R) Test of Achievement is an 'easel' test where a response book sits in front of the respondent. The interviewers placed the easel at an angle so that they and the respondents could both see the stimuli (pictures) simultaneously.

Since the WJ-R can be used for respondents from ages 2 to 90 years, items in the WJ-R were arranged by difficulty for all persons between those ages. The easiest questions were presented first and the items became increasingly difficult as the respondent proceeded through the test. The interviewer started testing at the appropriate starting point based on education level of the child or youth as the general guideline. At the beginning of every subtest, usually on the first page, there was a chart organized by grade in school that informed the interviewer at what item they should start administering the test.

Raw scores were calculated for the WJ-R using basal and ceiling. The basal and ceiling criteria were created to limit the amount of time any one person spends on each subtest. When the respondent got six or more consecutive items correct, then they established their basal. The interviewer continued testing until the respondent established ceiling, which was six or more consecutive items incorrect and the end of the testing page has been reached.

WJ-R has standardized scoring protocols. The tests are designed to provide a normative score that shows the CDS target child's reading and math abilities in comparison to national average for the child's age. The standardized scores are constructed based on the target child's raw score on the test (essentially the number of correct items completed) and the child's age to the nearest month. Raw scores are charted on normative tables based on the child's age and the percentile into which the child falls. For more information about standardized scoring and interpretation see Woodcock and Mather (1989, 1990).

Table 1

Differences in Outcomes Between Brothers, by Birth Weight Sample: Boys with At least One Brother Who Does Not Have the Same Birth Weight Classification

	Low birth weight~		Not low birth weight		Difference
	[1]		[2]		[1] minus [2]
Outcomes in childhood: child sample					
Proportion good/fair/poor health^	0.291	[285]	0.204	[362]	0.087 ***
Woodcock-Johnson achievement measures:					
Broad Reading standardized score	89.57	[37]	96.22	[46]	−6.65 **
Letter/Word standardized score	88.28	[47]	93.00	[60]	−4.72
Passage Comprehension standardized score	93.00	[37]	98.93	[59]	−5.93 *
Applied problem standardized score	94.53	[47]	98.80	[59]	−4.26
Outcomes in adulthood: adult sample					
Proportion high school dropout^	0.189	[265]	0.158	[514]	0.031
Proportion attended college^	0.374	[265]	0.374	[514]	0.000
Proportion fair/poor adult health					
All ages	0.146	[957]	0.082	[2200]	0.064 ***
18–26	0.080	[199]	0.064	[470]	0.017
27–36	0.136	[536]	0.073	[1223]	0.063 ***
37–52	0.230	[222]	0.120	[507]	0.109 ***
Proportion with no earnings					
All ages	0.108	[1100]	0.061	[2542]	0.048 ***
18–26	0.053	[318]	0.033	[798]	0.021 *
27–36	0.126	[565]	0.054	[1243]	0.072 ***
37–52	0.143	[217]	0.122	[501]	0.021
Annual earnings, including 0s					
All ages	$20,390	[1100]	$26,047	[2542]	−$5,657 ***
18–26	$15,773	[318]	$17,541	[798]	−$1,768 **
27–36	$20,164	[565]	$26,886	[1243]	−$6,722 ***

	Low birth weight~		Not low birth weight		Difference
	[1]		[2]		[1] minus [2]
37–52	$27,743	[217]	$37,511	[501]	−$9,768 **
Annual earnings, excluding 0s					
All ages	$22,863	[981]	$27,727	[2388]	−$4,863 ***
18–26	$16,664	[301]	$18,132	[772]	−$1,468 *
27–36	$23,062	[494]	$28,418	[1176]	−$5,356 ***
37–52	$32,367	[186]	$42,712	[440]	−$10,345 *
Annual hours, including 0s					
All ages	1,331	[1803]	1,438	[4132]	−107 ***
18–26	991	[904]	1,042	[2112]	−52
27–36	1,623	[677]	1,812	[1509]	−188 ***
37–50	1,824	[222]	1,972	[511]	−148 **
Wages					
All ages	$11.92	[990]	$13.66	[2409]	−$1.74 ***
18–26	$9.05	[297]	$10.02	[755]	−$0.97 *
27–36	$12.46	[502]	$13.85	[1189]	−$1.39 **
37–52	$14.97	[191]	$19.10	[465]	−$4.13 *

Number of observations reported in brackets.

*significant at 10%;

**significant at 5%;

***significant at 1%.

^Girls are included for the analysis of child health and educational attainment to increase sample size.

~For the child outcomes, the number of cases were too small to consider only low birth weight. So for these outcomes the comparison is between siblings who did and did not have poor birth outcomes, where poor outcome is defined as being born less than 5.5 pounds or before 37 weeks (i.e., premature).

Table 2

Effects of Birth Outcomes, Family Income, and Health Insurance Coverage During Pregnancy on Child Health: Child Sample, Including Girls

	Dependent variable: General health status in childhood. Interval Regression Model: 100pt-scale, 100=perfect health												
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
Spline for birth weight (lbs) effects:													
Low birth weight	−4.8589*** (1.2281)	−1.6659* (1.0405)			−3.7647** (1.5821)	−0.2802 (1.2598)						−2.3731*** (0.6883)	−1.5580*** (0.5814)
(Birth weight-3.3)*Low birth weight	1.8540*** (0.6800)	−0.0710 (0.4942)			1.2004 (0.8789)	−0.7682 (0.6052)							
(Birth weight-3.3)*NOT Low birth weight	−0.0524 (0.1458)	−0.0213 (0.1705)			−0.0914 (0.1580)	−0.0433 (0.1762)							
Spline for gestation length (weeks) effects:													
Premature			−0.9606 (0.9309)	−0.8546 (0.8765)	0.2242 (1.0609)	−0.7567 (0.9440)							
(Gestation length-34)*Premature			0.7552*** (0.2759)	0.2820* (0.1711)	0.4313 (0.3510)	0.3540* (0.2030)							
(Gestation length-34)*NOT premature			0.1240 (0.1070)	0.0487 (0.1221)	0.1128 (0.1181)	0.0304 (0.1257)							
NICU													
Placement in NICU							−1.5447*** (0.4520)						
Duration in NICU (in weeks) - 1							−0.1476 (0.1470)						
Health insurance coverage during pregnancy:													
None (reference group)													
Private								1.7802*** (0.6169)	1.0205* (0.6971)			1.5663** (0.6098)	1.0244* (0.7016)
Medicaid								−0.0343 (0.7295)	−0.1801 (0.7564)			0.0536 (0.7257)	0.0602 (0.7573)
Gov't program participation during pregnancy:													
WIC								−1.1388*** (0.3849)	0.0967 (0.5315)			−0.8759** (0.3968)	0.1857 (0.5309)
Food Stamps								0.0696 (0.5995)	0.0106 (0.6673)			0.0956 (0.6028)	0.0085 (0.6678)
AFDC								0.0574 (0.6364)	0.0536 (0.6959)			−0.0269 (0.6378)	−0.0345 (0.6949)
Spline for family income (in $10,000s) in year of pregnancy:													
Family income*($0–15,000)										−0.3972 (0.6531)	−2.0028*** (0.6220)	−0.5462 (0.6518)	−2.2060*** (0.6256)
Family income*($15–50,000)										0.8337*** (0.1818)	0.5311** (0.2201)	0.5279*** (0.1944)	0.4818** (0.2224)
Family income*($50–100,000)										0.0170 (0.1199)	−0.0504 (0.1965)	0.0399 (0.1201)	−0.0316 (0.1971)

	Dependent variable: General health status in childhood. Interval Regression Model: 100pt-scale, 100=perfect health												
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**	**(9)**	**(10)**	**(11)**	**(12)**	**(13)**
Constant	92.2224*** (0.8964)		91.1806*** (0.9645)		91.6847*** (1.0371)			91.4434*** (0.9005)		91.2922*** (0.9454)		91.6030*** (1.1355)	
Full or sibling sample?	Sibling	Sibling	Sibling	Sibling	Sibling	Sibling	Sibling	Sibling	Sibling	Sibling	Sibling	Sibling	Sibling
Mother fixed effects?	No	Yes	No	Yes	No	Yes	Yes	No	Yes	No	Yes	No	Yes
# of Mothers	1,129	1,129	1,129	1,129	1,129	1,129	1,129	1,129	1,129	1,129	1,129	1,129	1,129
# of Children	2,226	2,226	2,226	2,226	2,226	2,226	2,226	2,226	2,226	2,226	2,226	2,226	2,226
# of Child-year observations	4,108	4,108	4,108	4,108	4,108	4,108	4,108	4,108	4,108	4,108	4,108	4,108	4,108

Robust standard errors in parentheses.

* significant at 10%;

** significant at 5%;

*** significant at 1%.

All regression models also include controls for gender, child age, race, birth order, mother's age at birth, whether born to two-parent family, and birth year cohort dummy indicators (5-yr intervals). All models also include a spline in income above $100K. Less than 5% of sample has parental income of more than $100K, and effects in this range are driven by outliers

Premature is defined as a pregnancy of less than or equal to 37 weeks; Low birth weight is defined as less than 5.5 lbs.

Table 3

Effects of Birth Outcomes and Family Income During Pregnancy on Child Achievement in Reading & Math: Child Sample

	Dependent variables--Woodcock-Johnson child achievement measures:									
	Reading Std Score: Passage Comprehension					**Math Std Score: Applied Problems**				
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**	**(9)**	**(10)**
Spline for birth weight (lbs) effects:										
Low birth weight	−10.5456* (5.4789)			−4.3320* (3.3829)	−3.7343 (3.3917)	−7.8442* (4.9497)			−4.9129* (3.0053)	−5.0170* (3.0364)
(Birth weight-3.3)* Low birth weight	5.0252* (2.7886)					3.7562* (2.3737)				
(Birth weight-3.3)* NOT Low birth weight	−0.3200 (0.9551)					0.2439 (0.8714)				
NICU										
Placement in NICU		−3.2893* (2.4511)					−0.5511 (2.3235)			
Duration in NICU (in weeks) - 1		−0.8148* (0.5371)					−0.8914 (0.5429)			
Spline for family income (in $10,000s) in year of pregnancy:										
Family income*($0–15,000)			4.8132* (3.3053)	4.6069* (3.3100)	5.0895+ (3.3222)			5.2170* (2.9897)	5.2219* (2.9818)	5.1224* (3.0024)
Family income*($15–50,000)			1.0255 (1.2023)	1.0261 (1.2023)	1.1559 (1.2009)			0.5780 (1.1089)	0.6086 (1.1061)	0.5306 (1.1105)
Family income*($50–100,000)			−0.3827 (1.0887)	−0.3366 (1.0903)	−0.4060 (1.0879)			−1.0968 (0.9768)	−1.2314 (0.9767)	−1.2184 (0.9803)
Childhood health status:										
Excellent (reference group)										
Very Good					−4.3598** (1.7028)					−0.2705 (1.5551)
Good					−1.9356 (2.1867)					0.1220 (1.9923)
Fair					−3.1631 (4.5734)					2.5288 (4.0785)
Constant	105.8150*** (4.9758)	105.1225*** (3.2139)	99.1089*** (4.8582)	98.8293*** (4.8625)	100.3815*** (4.8960)	102.5998*** (4.5665)	104.7925*** (3.0022)	98.6143*** (4.4168)	97.8822*** (4.4153)	98.2537*** (4.4865)
Mother fixed effects?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
# of Mothers	239	239	239	239	239	240	240	240	240	240
# of Boys	456	456	456	456	456	478	478	478	478	478
# of Child-year Observations	609	609	609	609	609	730	730	730	730	730

Robust standard errors in parentheses.

* significant at 10% (one-tailed test);

** significant at 5%;

*** significant at 1%.

All regression models also include controls for child age, race, birth order, mother's age at birth, whether born to two-parent family, birth year cohort dummy indicators (5-yr intervals), and a spline in income above $100k. Less than 5% of the sample has family income of more than $100k, and effects in this range are driven by outliers.

Table 4

Birth Weight, Childhood Family Income, and Educational Attainment

	Dependent variable:					
	High school dropout (Estimation: Linear probability model)					Years of education
	(1)	(2)	(3)	(4)	(5)	(6)
Low birth weight	0.0570*** (0.0202)	0.0671*** (0.0215)	0.0479* (0.0259)	0.0629*** (0.0211)	0.0472* (0.0259)	−0.1073 (0.1221)
Family income-to needs ratio at ages 13–16, spline:						
Income-to-needs ratio * ratio is <1				−0.1582*** (0.0389)	−0.0339 (0.0609)	0.2399 (0.2870)
Income-to-needs ratio * ratio is 1 to 2				−0.1058*** (0.0200)	−0.0097 (0.0317)	−0.2222 (0.1492)
Income-to-needs ratio * ratio >2 to 3				−0.0755*** (0.0189)	0.0090 (0.0299)	0.2399* (0.1410)
Income-to-needs ratio * ratio is >3				−0.0037 (0.0048)	0.0052 (0.0112)	−0.0277 (0.0526)
Constant	0.0867*** (0.0232)	0.0824*** (0.0253)	0.1427*** (0.0331)	0.3368*** (0.0402)	0.1749*** (0.0618)	12.8611*** (0.2914)
Full or Sibling Sample?	Full	Sibling	Sibling	Sibling	Sibling	Sibling
Sibling fixed effects?	No	No	Yes	No	Yes	Yes
Number of families	2,094	1,655	1,655	1,655	1,655	1,655
Number of individuals	5,817	5,160	5,160	5,160	5,160	5,160

Sample includes men and women. Standard errors in parentheses.

* significant at 10%;

** significant at 5%;

*** significant at 1%.

All models also include controls for gender, race, birth order, mother's age at birth, whether born into a two-parent family, birth year cohort dummy indicators (5-yr intervals), and the set of parental fertility timing preference variables.

Table 5

Birth Weight and Adult Health

	Dependent variable: general health status in adulthood				
	Interval Regression Model: 100pt-scale, 100=perfect health			P(Fair/Poor Hlth) Linear Probability Model	Interval Regression Model: 100pt-scale, 100=perfect hlth
	(1)	(2)	(3)	(4)	(5)
Low birth weight	−2.3690** (1.1570)	−2.8854* (1.5500)	−3.7659*** (1.3658)	0.0703*** (0.0261)	−3.7290*** (1.3437)
Age - 25	−0.2714*** (0.0255)	−0.2719*** (0.0311)	−0.3137*** (0.0270)	0.0037*** (0.0006)	−0.3173*** (0.0271)
Parental fertility timing preferences:					
Wanted child & pregnant at right time (ref. group)					
Did not want child					−2.5542*** (0.7753)
Wanted child & pregnancy too soon					0.8360 (0.9365)
Wanted child & pregnancy delayed					−1.4197 (1.0509)
Wanted child & no timing preferences					−6.8602*** (2.4986)
Birth order	−0.4112*** (0.1535)	−0.4029** (0.1800)	−0.1832 (0.2837)	0.0006 (0.0057)	−0.0527 (0.2907)
Born into two-parent family	0.7715 (0.8207)	0.8985 (0.9926)	−0.3752 (1.2435)	−0.0164 (0.0281)	−0.1539 (1.2528)
Mother's age at birth:					
13–19	0.1204 (0.7290)	0.9310 (0.8091)	−0.3042 (0.7811)	0.0022 (0.0156)	−0.7938 (0.7975)
20–25 (reference group)					
26–30	−0.2052 (0.5525)	0.0495 (0.6444)	−0.2899 (0.6900)	0.0076 (0.0149)	−0.4062 (0.7001)
31–34	−0.6013 (0.7082)	−0.7986 (0.8219)	0.3468 (1.2333)	−0.0032 (0.0257)	0.4256 (1.2420)
>=35	1.1593 (0.7676)	0.5374 (0.9961)	2.6438 (1.6151)	−0.0247 (0.0360)	2.6781 (1.6393)
Race/Ethnicity:					
Non-Hispanic black	−3.2677*** (0.4988)	−3.4821*** (0.5803)			
Non-Hispanic white (reference group)					
Constant	88.8994*** (1.0830)	88.8961*** (1.3421)	94.4143*** (1.6704)	−0.0113 (0.0482)	95.3859*** (2.1864)
Full or brother sample?	Full	Brother	Brother	Brother	Brother
Brother fixed effects?	No	No	Yes	Yes	Yes
Number of families	1,444	723	723	723	723
Number of individuals	2,745	1,888	1,888	1,888	1,888

	Dependent variable: general health status in adulthood				
	Interval Regression Model: 100pt-scale, 100=perfect health			P(Fair/Poor Hlth) Linear Probability Model	Interval Regression Model: 100pt-scale, 100=perfect hlth
	(1)	(2)	(3)	(4)	(5)
Person-year observations	26,407	18,252	18,252	18,252	18,252

Robust standard errors (clustered on individual) in parentheses;

* significant at 10%;

** significant at 5%;

*** significant at 1%

All models also include birth year cohort dummy indicators (5-yr intervals), and indicators for Hispanic and “other” non-Hispanic race. Column (5) includes dummy indicators for missing parental fertility timing preferences.

Table 6

Birth Weight, Childhood Health, and Adult Health Over the Life Course
(Interval regression. Dependent variable: general health status in adulthood: 100pt-scale, 100=perfect health)

	(1)	(2)	(3)
Low birth weight * age <=36	−3.0606** (1.2139)		
Low birth weight * age>36	−5.9642** (2.5660)		
Spline for age effects:			
(Age - 25) * age<=36	−0.3059*** (0.0319)		
(Age - 25) * age>36	−0.3093*** (0.0277)		
Low birth weight		−6.7814*** (2.4231)	−4.9796** (1.9744)
Age - 25		−0.2687*** (0.0332)	−0.2731*** (0.0330)
Childhood health status:			
Excellent (reference group)			
Very Good			−2.4865*** (0.6752)
Good			−6.7623*** (1.1738)
Fair			−9.6623** (4.5800)
Poor			−23.5482*** (6.3610)
Full or brother sample?	Brother	Brother	Brother
Sub-sample w/childhood health information?	No	Yes	Yes
Brother fixed effects?	Yes	Yes	Yes
Number of families	723	273	273
Number of individuals	1,888	653	653
Person-year observations	18,252	8,966	8,966

Robust standard errors (clustered on individual) in parentheses;

* significant at 10%;

** significant at 5%;

*** significant at 1%.

All models also include controls for birth order, mother's age at birth, whether born into a two-parent family, birth year cohort dummy indicators (5-yr intervals), and the set of parental fertility timing preference variables.

Table 7

Childhood Family Income, Health Insurance, Birth Weight, and Adult Health
(Interval regression. Dependent variable: general health status in adulthood: 100pt-scale, 100=perfect health)

	(1)	(2)	(3)
Parental income-to-needs ratio at ages 13–16:			
<1 (in poverty)	−2.1252 ** (0.9838)	−2.1252 ** (0.9826)	−2.1100 ** (0.9798)
1 to 2 (reference group in columns 1 and 2)			
>2 to 3	−0.4154 (0.8775)	−0.3424 (0.8589)	
>3	−0.4401 (1.0659)	−0.3549 (1.0488)	
Low birth weight		−3.6605 *** (1.3480)	
Low birth weight * had health insurance (in all yrs 1968 to 1972)			−1.9148 (1.8702)
Low birth weight * no health insurance (at some point 1968 to 1972)			−5.1029 *** (1.8726)
Age - 25	−0.3171 *** (0.0272)	−0.3166 *** (0.0271)	−0.3167 *** (0.0271)
Brother fixed effects?	Yes	Yes	Yes
Number of families	723	723	723
Number of individuals	1,888	1,888	1,888
Person-year observations	18,252	18,252	18,252

Standard errors (clustered on individual) in parentheses.

* significant at 10%;

** significant at 5%;

*** significant at 1%.

All models also include controls for birth order, mother's age at birth, whether born into a two-parent family, birth year cohort dummy indicators (5-yr intervals), and the set of parental fertility timing preference variables.

Table 8

Birth Weight and Adult Labor Market Outcomes

	Dependent variables:								
	No Earnings. Estimation: Linear Probability model			ln(earnings), (cond'l on positive earnings, 1997$).			Annual labor market earnings Estimation: Tobit model (in $000s, 1997$)		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Low birth weight	0.0153 (0.0167)	0.0311 (0.0232)	0.0435* (0.0224)	−0.2159*** (0.0779)	−0.2935*** (0.1027)	−0.1073 (0.0902)	−4.5827*** (1.7447)	−5.7488*** (2.2135)	−2.9656* (1.7086)
Age - 30	0.0041*** (0.0004)	0.0042*** (0.0005)	0.0050*** (0.0005)	0.0440*** (0.0015)	0.0426*** (0.0018)	0.0351*** (0.0018)	1.0725*** (0.0552)	1.0296*** (0.0641)	0.8859*** (0.0597)
(Age - 30) squared	0.0002*** (0.0000)	0.0002*** (0.0001)	0.0002*** (0.0001)	−0.0015*** (0.0001)	−0.0013*** (0.0002)	−0.0011*** (0.0002)	−0.0292*** (0.0055)	−0.0259*** (0.0062)	−0.0169*** (0.0060)
Constant	0.0376** (0.0162)	0.0539** (0.0212)		10.3085*** (0.0689)	10.3259*** (0.0891)		35.6322*** (2.2744)	37.3697*** (3.1193)	
Full or brother sample?	Full	Brother	Brother	Full	Brother	Brother	Full	Brother	Brother
Brother fixed effects?	No	No	Yes	No	No	Yes	No	No	Yes
Number of families	1,448	680	680	1,448	680	680	1,448	680	680
Number of individuals	2,652	1,760	1,760	2,652	1,743	1,743	2,652	1,760	1,760
Person-year observations	31,610	21,354	21,354	29,450	19,830	19,830	31,610	21,354	21,354

Robust standard errors (clustered on individual) in parentheses;

* significant at 10%;

** significant at 5%;

*** significant at 1%.

All regression models also include controls for birth order, mother's age at birth, whether born to two-parent family, parental fertility timing preference variables, race, and birth year cohort dummy indicators (5-yr intervals).

Table 9

Birth Weight and Adult Labor Market Outcomes Over the Life Course

	Dependent variables:			
	No Earnings. Estimation: Linear Probability Model	**ln(earnings), (cond'l on positive earnings, 1997$).**	**Annual labor market earnings (in $000s, 1997$) Estimation: Tobit model**	**ln(wage), (cond'l on positive earnings, 1997$).**
	(1)	**(2)**	**(3)**	**(4)**
Low birth weight	0.0627** (0.0247)	−0.1667* (0.0965)	−4.8191** (1.8959)	−0.1751* (0.0967)
(Age - 30) * Low birth weight	0.0025 (0.0026)	−0.0054 (0.0089)	−0.5310** (0.2082)	−0.0051 (0.0088)
$(\text{Age - 30})^2$ * Low birth weight	−0.0005** (0.0002)	0.0015** (0.0006)	0.0545*** (0.0162)	0.0014** (0.0006)
(Age - 30)	0.0049*** (0.0005)	0.0353*** (0.0018)	0.9089*** (0.0618)	0.0353*** (0.0018)
(Age - 30) squared	0.0002*** (0.0001)	−0.0011*** (0.0002)	−0.0195*** (0.0063)	−0.0011*** (0.0002)
Implied effect of low birth weight at age:				
25	0.0377	−0.1022	−0.8016	−0.1146
35	0.0627	−0.1562	−6.1116	−0.1656
Full or brother sample?	Brother	Brother	Brother	Brother
Brother fixed effects?	Yes	Yes	Yes	Yes
Number of families	680	680	680	672
Number of individuals	1,760	1,743	1,760	1,731
Person-year observations	21354	19830	21354	19,624

Robust standard errors (clustered on individual) in parentheses.

* significant at 10%;

** significant at 5%;

*** significant at 1%.

All regression models also include controls for birth order, maternal age at birth, whether born into 2-parent family, race, birth year cohort dummy indicators (5-yr intervals), and parental fertility timing preference variables.

Table 10

Childhood Family Income and Health Insurance, Birth Weight, Education, and Adult Labor Market Outcomes

Dependent variable	No Earnings, Estimation: Linear Probability Model				ln(earnings), (conditional on positive earnings, 1997$).				Annual labor market earnings: Estimation: Tobit model (in $000s, 1997$)				ln(wage), (one-off on positive earnings, 1997$).			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
Low birth weight		* 0.0433 (0.0226)		** 0.0458 (0.0221)		-0.1035 (0.0908)		* -0.1222 (0.0887)		* -2.0991 (1.7295)		** -3.3594 (1.6573)		* -0.0810 (0.0573)		* -0.0951 (0.0556)
Low birth weight * had health insurance (in all yrs 1968 to 1972)			0.0049 (0.0265)				** 0.2092 (0.0994)				1.5379 (2.1853)				0.0961 (0.0720)	
Low birth weight * no health insurance (at some point 1968 to 1972)			** 0.0712 (0.0331)				** -0.3402 (0.1343)				** -6.2219 (2.4675)				*** -0.2169 (0.0802)	
Parental income-to-needs ratio, ages 13–16																
<1 (in poverty)	* 0.0232 (0.0158)	* 0.0231 (0.0158)	* 0.0244 (0.0157)		* -0.1293 (0.0661)	* -0.1283 (0.0660)	* -0.1276 (0.0655)		*** -3.7335 (1.1860)	*** -3.7253 (1.1855)	*** -3.7945 (1.1836)		-0.0009 (0.0431)	-0.0060 (0.0430)	-0.0037 (0.0429)	
1 to 2 (reference group)																
>2 to 3	-0.0017 (0.0136)	-0.0020 (0.0133)			0.0202 (0.0449)	0.0224 (0.0448)			-0.5256 (1.2631)	-0.4581 (1.2570)			-0.0106 (0.0362)	-0.0087 (0.0361)		
Income-to-Needs Ratio: >3	0.0126 (0.0165)	0.0115 (0.0163)			0.0450 (0.0678)	0.0466 (0.0675)			-1.3437 (2.1482)	-1.2770 (2.1451)			0.0213 (0.0518)	0.0226 (0.0516)		
Educational Attainment																
High School dropout				*** 0.0710 (0.0140)				*** -0.2706 (0.0565)				*** -5.1539 (1.0729)				*** -0.1708 (0.0396)
High School graduate (reference group)																
Some college				0.0134 (0.0092)				0.0611 (0.0382)				1.2007 (0.9942)				** 0.0649 (0.0275)
College graduate				** -0.0210 (0.0093)				*** 0.2588 (0.0516)				*** 0.9488 (1.8126)				*** 0.2350 (0.0480)
Age - 30	*** 0.0050 (0.0005)	*** 0.0050 (0.0005)	*** 0.0050 (0.0005)	*** 0.0051 (0.0005)	*** 0.0351 (0.0010)	*** 0.0352 (0.0010)	*** 0.0351 (0.0010)	*** 0.0341 (0.0010)	*** 0.1875 (0.0596)	*** 0.1806 (0.0597)	*** 0.1872 (0.0597)	*** 0.1703 (0.0593)	*** 0.0257 (0.0013)	*** 0.0257 (0.0013)	*** 0.0257 (0.0013)	*** 0.0250 (0.0013)
(Age - 30) squared	*** 0.0002 (0.0001)	*** 0.0002 (0.0001)	*** 0.0002 (0.0001)	*** 0.0002 (0.0001)	*** -0.0011 (0.0002)	*** -0.0011 (0.0002)	*** -0.0011 (0.0002)	*** -0.0010 (0.0002)	*** -0.0171 (0.0060)	*** -0.0171 (0.0060)	*** -0.0170 (0.0060)	*** -0.0155 (0.0060)	*** -0.0007 (0.0001)	*** -0.0007 (0.0001)	*** -0.0007 (0.0001)	*** -0.0006 (0.0001)
Full or brother sample?	Brother	Brother	Brother	Brother	Brother	Brother	Brother	Brother	Brother	Brother	Brother	Brother	Brother	Brother	Brother	Brother
Brother fixed effects?	Yes	Yes	Yes	Yes	yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Number of families	680	680	680	680	680	680	680	680	680	680	680	680	672	672	672	672
Number of individuals	1,760	1,760	1,760	1,760	1,743	1,743	1,743	1,743	1,760	1,760	1,760	1,760	1,731	1,731	1,731	1,731
Person-year observations	21354	21354	21354	21354	19830	19830	19830	19830	21354	21354	21354	21354	19624	19,624	19624	19,624

Robust standard errors (clustered on individual) in parentheses.

* significant at 10%; (one-tailed test);

** significant at 5%;

*** significant at 1%.

All regression models also include controls for birth order, All regression models also include controls for birth order, mother's age at birth, whether born to two-parent family, parental fertility timing preference variables, race, and birth year cohort dummy indicators (5-yr intervals).

Table A1

Unweighted Descriptive Statistics for Child Sample

	Full sample: N=6,447		Sample restricted to children w/1 or more sibs in the sample: N=4,111	
	Mean	**Std. Dev**	**Mean**	**Std. Dev**
CHILD OUTCOMES:				
Health Status:				
Excellent	.5094		.5043	
Very Good	.3181		.3140	
Good	.1452		.1515	
Fair	.0254		.0292	
Poor	.0019		.0010	
Age (range: 1–18)	8.3	(4.3642)	8.2	(4.1537)
Year born (range: 1985–1997)	1991		1991	
Woodcock-Johnson Achievement (standardized scores):				
Passage comprehension	102.1	(16.3)	102.1	(16.2)
Letters/Words	101.5	(18.7)	101.1	(18.4)
Broad reading	102.0	(18.1)	101.9	(17.8)
Math--applied problems	103.5	(18.2)	104.3	(18.1)
EARLY-LIFE MEASURES:				
Sibling-specific birth variables:				
Birth weight (lbs)	7.2735	(1.4265)	7.2978	(1.4165)
Low birth weight (<5.5 pounds)	.0890		.0905	
Gestation length (weeks)	39.43	(2.27)	39.46	(2.22)
Prematurity (<37 weeks)	.0856		.0849	
Placement in NICU	.1244		.1161	
# of days in NICU (cond'l on placement)	13.9	(23.1)	12.2	(20.5)
Family income in year of pregnancy	39,580	(35,257)	43,522	(41,627)
Health insurance coverage during pregnancy:				
None	.0963		.0896	
Private only	.5674		.5692	
Medicaid	.3552		.3568	
Gov't program participation during pregnancy:				
WIC	.4337		.4515	
Food Stamps	.2309		.2546	
AFDC	.1689		.1816	
Parental infant health history:				
Mother low birth weight	.0850		.0850	
Father low birth weight	.0589		.0589	
Birth order:				
First born	.3967		.3998	
Second child	.3598		.3603	

	Full sample: N=6,447		Sample restricted to children w/1 or more sibs in the sample: N=4,111	
	Mean	**Std. Dev**	**Mean**	**Std. Dev**
Third or fourth child	.2198		.2171	
Fifth or higher	.0237		.0227	
Mother's age at child's birth:				
13–19	.1025		.0911	
20–25	.3083		.3255	
26–30	.3122		.3224	
31–34	.1861		.1854	
>35	.0909		.0756	
Born into two-parent family	.6723		.6750	
Race/ethnicity				
Non-Hispanic white	.5636		.5908	
Non-Hispanic black	.4190		.3900	
Other	.0174		.0192	

Table A2

Unweighted Descriptive Statistics for Adult Sample

	Full sample: N=26,407		Sample restricted to men w/1 or more brothers: N=19,219	
	Mean	**Std. Dev**	**Mean**	**Std. Dev**
ADULT OUTCOMES:				
Health Status:				
Excellent	.3089		.3095	
Very Good	.3628		.3606	
Good	.2425		.2415	
Fair	.0683		.0697	
Poor	.0175		.0186	
Age (range: 16–49)	32.3	(6.3777)	32.1	(6.2731)
Year born (range: 1951–1974)	1960		1960	
Labor Earnings:				
No earnings	.0683	(0.2523)	.0717	(.2580)
Annual earnings (1997$)	27,869	(29,269)	27,507	(30,335)
Annual earnings, cond'l on earnings>0	29,913	(29,298)	29,632	(30,469)
Education:				
Years of education	12.7327	(2.0179)	12.8612	(1.9552)
High school dropout	.1766	(0.3814)	.1544	(.3615)
EARLY-LIFE MEASURES:				
Sibling-specific variables:				
Low birth weight (<5.5 pounds)	.0647	(0.2461)	.0640	(.2449)
Cond'l on low birthweight, fraction who were first-born child	.3713		.3462	
Average family income: ages 0–4	34,419	(19,909)	34,887	(20,261)
Average family income: ages 5–8	37,966	(23,776)	38,065	(23,479)
Average family income: ages 9–12	41,638	(29,175)	41,302	(27,786)
Average family income: ages 13–16	43,911	(30,607)	44,106	(30,633)
Average income-to-needs ratio <1 during ages 13–16	.2321		.2444	
Birth order:				
First born	.3167		.2500	
Second child	.2616		.2530	
Third or fourth child	.2852		.3282	
Fifth or higher	.1366		.1687	
Mother's age at child's birth:	26.8	(6.2089)	27.0	(6.1195)
13–19	.0995		.0897	
20–25	.3107		.3208	
26–30	.2328		.2405	
31–34	.1421		.1463	
>35	.2146		.2028	
Born into two-parent family	.8597	(0.3474)	.8677	(.3389)

	Full sample: N=26,407		Sample restricted to men w/1 or more brothers: N=19,219	
	Mean	**Std. Dev**	**Mean**	**Std. Dev**
Parental fertility timing preferences:				
Wanted child & pregnant at right time	.2866		.2593	
Did not want child before pregnancy	.3153		.3451	
Wanted child & pregnancy too soon	.1350		.1259	
Wanted child & pregnancy delayed	.0643		.0569	
Wanted child & no timing preferences	.0261		.0215	
Wanted child & missing info on timing preferences	.1732		.1922	
Childhood family-specific variables:				
Average family income<=$15K (ages 13–16), for at least 1 child	.2418	(0.4283)	.2150	(.4110)
Race/ethnicity				
Non-Hispanic white	.5448		.5221	
Non-Hispanic black	.4327		.4568	
Non-Hispanic other	.0850		.0070	
Hispanic	.0140		.0141	
Parent's (head's) education:				
High school dropout	.5371		.5309	
High school graduate	.2734	(0.4458)	.2822	(.4502)
Some college	.0935	(0.2912)	.0904	(.2868)
College graduate	.0618	(.2409)	.0645	(.2456)
M.A. or higher	.0343	(.1819)	.0320	(.1760)
No parental health insurance at some point, 1968–1972	.5145	(.4999)	.5135	(.4500)
Parental health behaviors (1997 $):				
Smoked cigarettes at some point, 1968–1972	.7519	(.4320)	.7570	(.4290)
Annual cigarette expenditures (5-yr avg, 1968–1972)	489	(526)	489	(515)
Annual alcohol consumption (5-yr avg, 1968–1972)	375	(606)	393	(636)