NBER WORKING PAPER SERIES

CAUSES AND CONSEQUENCES OF EARLY LIFE HEALTH

Anne Case Christina Paxson

Working Paper 15637 http://www.nber.org/papers/w15637

NATIONAL BUREAU OF ECONOMIC RESEARCH 1050 Massachusetts Avenue Cambridge, MA 02138 January 2010

Material in this paper was presented at the RAND Summer Institute on July 8-9, 2009. We thank Kimberly Bryan for expert research assistance and seminar participants for many useful suggestions. We acknowledge funding from the Demography of Aging Center at Princeton University, funded under the National Institute of Aging grant P30 AG024361. We thank the Whitehall II Study team for providing us with access to physical and cognitive test information on the Whitehall II cohort. Continuing data collection for the Whitehall II study is funded by the Medical Research Council, the National Institute on Aging (AG13196), National Heart Lung and Blood Institute (HL36310) and the British Heart Foundation. The views expressed herein are those of the authors and do not necessarily reflect the views of the National Bureau of Economic Research.

NBER working papers are circulated for discussion and comment purposes. They have not been peerreviewed or been subject to the review by the NBER Board of Directors that accompanies official NBER publications.

© 2010 by Anne Case and Christina Paxson. All rights reserved. Short sections of text, not to exceed two paragraphs, may be quoted without explicit permission provided that full credit, including © notice, is given to the source.

Causes and Consequences of Early Life Health Anne Case and Christina Paxson NBER Working Paper No. 15637 January 2010 JEL No. D1,I12,J13

ABSTRACT

We examine the consequences of childhood health for economic and health outcomes in adulthood, using height as a marker of health in childhood. After reviewing previous evidence, we present a conceptual framework that highlights data limitations and methodological problems associated with the study of this topic. We present estimates of the associations between height and a range of outcomes, including schooling, employment, earnings, health and cognitive ability, using data collected from early to late adulthood on cohort members in five longitudinal data sets. We find height is uniformly associated with better economic, health and cognitive outcomes – a result only partially explained by the higher average educational attainment of taller individuals. We then turn to the NLSY79 Children and Young Adult Survey to better understand what specific aspects of early childhood are captured by height. We find, even among maternal siblings, taller siblings score better on cognitive tests and progress through school more quickly. Part of the differences found between siblings arises from differences in their birth weights and lengths attributable to mother's behaviors while pregnant. Taken together, these results support the hypothesis that childhood health influences health and economic status throughout the life course.

Anne Case 367 Wallace Hall Princeton University Princeton, NJ 08544 and NBER accase@princeton.edu

Christina Paxson 424 Robertson Hall Princeton University Princeton, NJ 08544-1022 and NBER cpaxson@princeton.edu Numerous studies have examined the association between economic status (as measured by income, occupation or education) and health. Despite occasional anomalies, three general patterns emerge from the data. The first is that health is strongly related to economic status: wealthier people live longer and have lower morbidity. This is true whether one looks across countries, within countries at any point in time, or over time with economic growth. The second is that the association between health and wealth—the "gradient"—is evident throughout the income distribution. The gradient does not merely reflect the association between poor health and absolute poverty, but is evident in wealthy countries, and among wealthier individuals within countries. The third is that the association between health and wealth and wealth is found across a wide range of ages. Differences in health across economic groups are apparent at birth: newborns from less well-off families or with less well-educated mothers have higher rates of mortality and lower birth weights than more advantaged newborns. The association between health and wealth becomes more pronounced until early old age but then eventually declines, possibly the result of selective mortality.

These patterns, although uncontroversial, have been subject to many interpretations. One hypothesis is that poor health is the product of low socioeconomic status. This may be due to the effects of material deprivation—less nutritious food, lower-quality medical care, and lower-quality housing—on health. Or, those with lower income or less education may live in more stressful environments, which could lead to greater physiological "wear and tear" on metabolic and immune systems and to a greater reliance on unhealthy behaviors such as smoking (Adler et al. 1994.) An alternative view is that poor health reduces economic status. Children in poor health may have lower educational attainment, which reduces their adult earnings potential. Adults with health problems may work less or may be less productive on their jobs, leading to

lower earnings, and they may be subject to great out-of-pocket medical costs, reducing asset accumulation.

These two views need not be mutually exclusive. Indeed, it is likely that there is a lifelong interplay between health and wealth, with different factors coming into prominence at different stages of the life cycle. Furthermore, the relationships between health and socioeconomic status are certainly dynamic—health at one stage of life has implications for future socioeconomic status, and socioeconomic status influences future health. In this paper, we focus on a specific aspect of the health-wealth gradient: the association between childhood health, on one hand, and economic and health outcomes later in life, on the other.

Educational attainment is an especially important adult outcome to consider. Schooling is obtained throughout childhood, and has a large influence on earnings, which may in turn affect health. Education may also have a direct effect on health and health behaviors later in life (Cutler and Lleras-Muney, 2008). Researchers have documented many dimensions of the association between childhood health and educational attainment.¹ In many large, cross-sectional studies, researchers have shown that cohorts affected by health insults *in utero* have lower educational attainment, on average, than do preceding and following birth cohorts that did not face the same health insult. Using a variety of data sets, researchers have also documented the association between low birth weight and educational attainment. In addition, several birth cohort studies allow researchers to trace the impact of childhood health (measured using birth weight, mothers' assessments of children's health, chronic conditions in childhood, days spent in bed, and hospitalization episodes) on progress through school and educational attainment.

¹ Currie (2009) provides a thorough review of the relationships between health in childhood and a range of outcomes, including education.

Estimates of the importance of health *in utero* and childhood on educational attainment vary, but are often found to be large and significant. Almond (2006), for example, estimates that children born to women infected with influenza during the 1918 pandemic received on average 5 months less education than children born before or conceived after this event. Case, Fertig and Paxson (2005) find, using data from the 1958 British birth cohort study, that children born at low birth weight passed 0.5 fewer O-level exams (qualifying exams taken in British secondary schools prior to 1988) than other children, on average. This effect is larger than that predicted for moving a cohort member's household from the 90th to the 10th percentile of family income at age 16.²

Less has been written on the impact of early life health on transitions to the labor force, employment and earnings in young adulthood. This is in part because studies following individuals from birth through middle age are still relatively scarce. Much of the evidence that we have comes again from being able to identify the impact of a large health shock on a birth cohort (for example, the 1918 influenza pandemic), or from a handful of panel studies, including the Panel Study of Income Dynamics for the US, and the British birth cohorts of 1958 and 1970, called the National Child Development Study (NCDS) and the British Cohort Study (BCS), respectively. Case, Fertig and Paxson (2005) find that, for men followed in the NCDS, markers

²Not all studies find a significant effect of childhood health on educational attainment. Smith (2009), for example, using retrospective reports on childhood health in the PSID finds a large and significant association between health reports and educational attainment in OLS regressions, but not in regressions that include maternal fixed effects. It is possible that the signal to noise ratio is too low in the latter, where identification is derived from differences in retrospective health reports among siblings.

of early life health make a large incremental contribution to the pseudo- R^2 of being employed at age 42. Specifically, with controls for family background, including mother and father school leaving ages, and father's and grandfathers' social classes, they find that chronic conditions in childhood and height at age 16 contribute 0.25 to the pseudo- R^2 in a probit regression of employment at age 42.

To date, life course models have had to rely on data collected from birth to middle age (as in the 1958 and 1970 British cohort studies), or from middle age to old age and death (as in the US Health and Retirement Study and in the UK Whitehall II Study). Longitudinal studies that follow cohort members from cradle to grave are, at this point, rare. An exception is the Helsinki Birth Cohort Study, which David Barker, Johann Eriksson and colleagues have used to examine the fetal origins hypothesis. Beyond this, estimates of the extent to which early life health affects socioeconomic status and health in old age are based primarily on retrospective questions asked in surveys currently being conducted on older adults.

With a lack of information on health in childhood, researchers have begun to use height as a marker of early life health and nutrition, which serves as a bridge from health in childhood to health and socioeconomic status at older ages. Our paper follows in this relatively new line of research. In the next section, we present a conceptual framework that captures the dynamic associations between health and economic status over the life course. The conceptual framework is used to highlight the methodological issues and data demands that complicate research on the long run effects of childhood health, and to motivate the use of height as a bridge from childhood into adulthood. We then present evidence on the associations between height and a range of outcomes in adulthood, including educational attainment, employment, earnings, health and cognitive function. Finally, we turn to consider the question of what aspects of the early life

environment height captures. Using data from the NLSY79—Children and Young Adults study, we show that height is related to a variety of children's health and developmental outcomes, including health at birth and cognitive development within childhood.

CONCEPTUAL FRAMEWORK

The framework we discuss here, which follows that presented in Case, Fertig and Paxson (2005), elucidates mechanisms through which early life experiences may influence later life outcomes. For simplicity, we assume there are three stages in life: childhood, young adulthood, and older adulthood. We also collapse "economics status" and "health status" into single variables, although each could be thought of as containing several components. For example, "economic status" could include earnings, asset income and transfer income; "health" could include chronic conditions, acute conditions, and cognitive function. Distinguishing between these different components may be important for empirical work, but not for laying out the framework.

We express economic status in older adulthood (E_A) and a measure of health in older adulthood (H_A) as linear functions of indicators of economic status and health in young adulthood (E_Y and H_Y respectively), educational attainment (S) and vectors of measures of economic status and health in childhood (E_C and H_C). Health in childhood can include prenatal health, nutrition and illnesses in childhood, and cognitive development.

(1)
$$E_A = \beta_0 + H_Y \beta_{HY} + E_Y \beta_{EY} + S\beta_S + H_C \beta_{HC} + E_C \beta_{EC} + \varepsilon_A^E$$

(2)
$$H_A = \alpha_0 + H_Y \alpha_{HY} + E_Y \alpha_{EY} + S\alpha_S + H_C \alpha_{HC} + E_C \alpha_{EC} + \varepsilon_A^H$$

Health, economic status and educational attainment in young adulthood are assumed to be functions of economic and health status in childhood:

(3)
$$E_Y = \gamma_0 + H_C \gamma_{HC} + E_C \gamma_{EC} + \varepsilon_Y^E$$

(4)
$$H_Y = \delta_0 + H_C \delta_{HC} + E_C \delta_{EC} + \varepsilon_Y^H$$

(5)
$$S = \theta_0 + H_C \theta_{HC} + E_C \theta_{EC} + \varepsilon^S$$

Equations (1) to (5), taken together, are general enough to be consistent with many hypotheses concerning the relationships between health and economic status at different stages of the life cycle. Much of the literature on health and economic status has been concerned with estimating equations (1) and (2), with a focus on the "cross effects" of adult health on later economic status, and of adult economic status on later-life health. One hypothesis, which is most closely associated with the work of Michael Marmot and colleagues, using data on British civil servants, is that the economic gradient in health observed among adults is largely due to the effects of economic status (defined in Whitehall by civil service grade) on disease risk ($\alpha_{EY} \neq$ 0). (See, for example, Brunner et al. 1999, Marmot et al. 2001, and Chandola et al. 2003.) An alternative hypothesis is that the gradient in adulthood reflects the effects of poor health on earnings and assets ($\beta_{HY} \neq$ 0). Although this has been an active and sometimes contentious area of research, our focus here is on the effects on childhood health on later life outcomes.

Equations (3), (4) and (5) indicate that childhood health and economic status determine the human capital that individuals carry into young adulthood, in the form of higher earnings potential, better health, and greater levels of schooling. (In (3), the effect of schooling on economic status has been subsumed in the effects of health and economic status in childhood.) The effect of childhood circumstances on later adulthood depends on the parameters in (1) and (2). These equations indicate that childhood circumstances may have direct effects, even controlling for educational attainment, economic status and health in young adulthood, and indirect effects that operate through the outcomes in young adulthood. Substitution of (3), (4) and (5) into (1) and (2) yields reduced form equations for health and economic status in later adulthood, expressed solely as functions of childhood characteristics. Estimates of these reduced form equations provide information on the combined direct and indirect effects of childhood circumstances.

The data demands for estimating equations (1) through (5) are high: the model requires data that follow individuals from early childhood through into later life. As we noted above, few such data sets exist. However, if there are no direct effects of childhood health and economic status on outcomes in older adulthood (i.e. $\beta_{HC} = \beta_{EC} = \alpha_{HC} = \alpha_{EC} = 0$), then equations (1) and (2) can be estimated using datasets that that follow individuals from younger to older adulthood: provided that accurate measures of education, health and economic status in young adulthood are available, further information on childhood is unnecessary. Likewise, equations (3), (4) and (5) can be estimated using data that follows individuals from infancy to young adulthood. This "separability" of equations (1) and (2) from equations (3), (4) and (5), if appropriate, greatly eases data demands.

A large body of research relies on the assumption of separability. For example, a discussion of results from the Whitehall II data argues that, among British civil servants, "early life circumstances are important because they influence adult social circumstances, which in turn influence disease risk" (Marmot et al. 2001, page 305). In other work, Brunner et al (1999) state that "whatever the salient features of the adult socioeconomic environment may be, it seems they are equally or more important than circumstances in childhood" in determining cardiovascular risk among British civil servants (page 762). Although it is convenient to assume that childhood circumstances operate only through their effects on outcomes in early adulthood—especially when working with data that do not contain information on childhood—another body of research makes the case that this assumption is incorrect. For example, evidence on the well-known fetal

origins hypothesis supports the idea that fetal health has a direct effect on health in later life $(\alpha_{HC} \neq 0)$. (See, for example, Barker 1992 and Barker 1995.)

Even with data sets that contain information over the lifespan, several econometric issues can complicate estimation of the effects of childhood health on adult outcomes. One issue is that, because of the well-documented associations between child health (H_c) and childhood economic circumstances (E_c), it may be difficult to separately identify the effects of these variables on health and economic status in adulthood. If good measures of economic status are unavailable, their effects are likely to load onto measures of child health, and vice versa. This issue will be important when interpreting estimates of the associations between height (as a marker for child health) and outcomes in adulthood. As we discuss below, height is influenced by a wide range of environmental factors experienced in childhood. Although a finding that height is associated with adult outcomes provides support for the hypothesis that childhood circumstances have longlasting effects, it does not provide information on which aspects of the environment in childhood are important.

A closely related problem is unobserved individual heterogeneity. For example, individuals may have unobserved personality traits or genetic characteristics that result in both poor health and low socioeconomic status throughout the life span. If so, it may appear as if childhood health (possibly measured as height) has effects on adult outcomes, when in fact the association is driven by these unobserved characteristics. There may also be unobservable differences across children in the quality of parenting they receive that is correlated with children's health, and which affects outcomes in adulthood.

To the extent that these traits or genetic characteristics are family-specific, one strategy is to use information on siblings or (in some cases) on twin pairs to control for unobserved

heterogeneity. Within-sibling or within-twin estimation techniques have been useful in identifying the impact of early life circumstance such as low birth weight on educational and health outcomes in adulthood (see, for example, Behrman and Rosenzweig 2004, and Black, Devereux and Salvanes 2007). Within-twin studies have the advantage over sibling studies, in that shared aspects of the uterine environment and (with monozygotic twins) genetic endowments can be controlled for. However, there are also disadvantages to twin studies: it may not be valid to draw inferences about singleton births from the experiences of twins. Furthermore, most nationally representative surveys contain too few twin pairs to use for analysis. In the last section of this paper, we rely in within-sibling methods to examine how children's heights are related to health and development in childhood.

HEIGHT, ECONOMIC STATUS AND HEALTH STATUS IN ADULTHOOD

This section presents estimates of the associations between height and several measures of economic and health status, estimated using data collected at different points in adulthood in five studies conducted in the United States and Britain. These results provide information on the role of environmental factors, which influence growth in childhood, in determining health and wellbeing much later in life. We begin with an overview of the determinants of growth and height, with an emphasis on how environmental conditions *in utero* and early childhood influence height.

Determinants of growth and height

Adult height reflects cumulative growth to maturity. After a period of rapid growth from ages 0 to 3, growth becomes relatively stable at approximately 6 cm a year until adolescence, at which

point an adolescent growth spurt accelerates growth to an (adolescent) peak height velocity of approximately 10 cm a year. Final adult height depends on the timing and duration of the adolescent growth spurt. (See Beard and Blaser 2002 for discussion and references.)

Age-specific growth patterns and final adult height depend on a combination of factors, including genes, environmental conditions (particularly under-nutrition and illness) and geneenvironment interactions. Although genes are key determinants of individual height, many studies suggest that differences in average height across populations are due largely to environmental factors (Steckel 1995). The increase in heights throughout the developed world during the 20th Century was too rapid to be due to selection and genetic variation (Beard and Blaser 2002).

The environmental factors thought to influence adult height include intrauterine conditions and conditions in early childhood. Both low birth weight and maternal smoking during pregnancy are significant predictors of lower stature in adulthood (Saenger et al. 2007, Li, Manor and Power, 2004). Adult height is also sensitive to environmental conditions experienced in childhood. Health and nutrition in the period from birth to age three are important determinants of adult height. The speed of growth is more rapid during this period than at any other during the life course, and nutritional needs are greatest at this point. Infections (especially gastrointestinal and respiratory) can be frequent and severe in early childhood, and such infections and inflammatory responses to them can impair growth (Crimmins and Finch 2006). In addition, children are at special risk from poor care-giving at youngest ages; once children are more autonomous, they may be better equipped to protect themselves. Research on institutionalized children who are adopted internationally shows that children experience rapid catch-up growth after adoption, although the extent of catch-up declines with the age of adoption

(Rutter et al. 1998). These results provide striking evidence of the importance of children's nutritional and psychosocial environments on growth.

Environmental factors that influence height may also affect health throughout life. Undernutrition, chronic infections and psychosocial stress acting at critical periods of development can induce permanent changes in body organs and systems. These changes, initiated and "programmed" early in life, may make individuals more susceptible to adverse health outcomes later in life when body reserves decline. Adverse influences in early life may alter gene expression and cell maturation, or change the structure of affected organs. The effects of programming can also be mediated through changes in the homeostatic set-points of hormonal and metabolic systems.³ While research has focused primarily on fetal programming (Barker 1998), it has become increasingly clear that postnatal influences are also of utmost importance.

The factors that influence height may also influence cognitive development. A large literature indicates that height is correlated with intelligence. Although a portion of this correlation is driven by genes, environmental factors are also important (Lynn, 1989). Sundet et al. (2005) use differences in cross-trait (height and intelligence), cross-twin correlations between monozygotic (MZ) and dizygotic (DZ) twin pairs to identify the roles played by shared environments and shared genes. They conclude that the environment plays a large role and is <u>3</u> References include Barker (1998), Bateson et al. 2004, Bertram and Hanson (2001), Dahlgren J. et al (2001), Dickerson and Kemeny (2004), Drake and Walker (2004), Holmang (2001), Holness, Langdown and Sugden (2000), Jackson (2000), Kajantie (2003), Kajantie, Dunkel, Turpeinen et al. (2003), Kajantie, Eriksson, Barker et al. (2003), Kajantie et al. (2004), Langley-Evans (2000), McMillen and Robinson (2005), Nilsson et al. (1998), Ozanne (2001), Phillips (2001), Samuelsson et al. (2004).

responsible for 65 percent of the height-intelligence correlation, with genes responsible for 35 percent of the observed correlation. These authors report that their results are very similar to those on cross-trait cross-twin correlations found much earlier by Husén (1959) in a large study of MZ and DZ twin pairs. Higher-birth-weight members of twin pairs are taller on average and have higher IQs than the lighter twins (Black et al. 2007). Other evidence using American twins finds that, within monozygotic twin pairs, greater fetal growth is associated with greater stature and higher educational attainment (Behrman and Rosenzweig, 2004).

The height-intelligence correlation may also be influenced by environmental conditions experienced in early childhood. Several randomized experiments conducted in low-income environments find that nutrition may be one of these environmental factors. In one study, nutritional supplements given to growth-retarded children improved their cognitive tests scores, although these gains dissipated after the supplementation ended (Grantham-McGregor 2002; Walker et al. 2005). Research from Guatemala found that children who had been provided with nutrition supplements during the first three years of life were taller, had higher scores on some cognitive tests, and higher educational attainment than children who did not receive supplements (Martorell et al. 2005). Observational studies also highlight the role of early childhood. For example, Raikkonnen et al (2009) use the Helsinki Birth Cohort Study to show that, conditional on health at birth, slower growth in height between birth and two years of age is associated with poorer performance on a range of intelligence tests in young adulthood.

Associations between height and adult outcomes over the life course

The evidence discussed above indicates that height reflects a variety of environmental factors experienced *in utero* and in early childhood, and that the environmental factors that influence

height may also affect health and cognitive development. Given this, it is not surprising that height is associated with a wide range of economic and health outcomes in adulthood. Previous research indicates that height is associated with educational status, earnings, and occupational grade (Persico, Postlewaite and Silverman, 2004; Case and Paxson, 2008a; Case, Paxson and Islam, 2008). Height is also associated with health and cognitive function at older ages (Case and Paxson, 2008b). In this section, we summarize these associations using data from five American and British surveys that capture individuals at different points in the life course, and that contain roughly comparable measures of adult outcomes.

The National Child Development Study (NCDS) tracks a cohort of all individuals born in Britain in a single week in 1958. The British Cohort Study (BCS) tracks a cohort of children born in a single week in 1970. For the NCDS, we use data from the surveys conducted when cohort members were 33 and 42 years of age, and for the BCS we use data from the survey conducted when the cohort members were 30 years old. The NCDS and BCS were designed to collect the same measures of health and economic status, providing comparable data on individuals in young to middle adulthood.

We also use data from the Panel Study of Income Dynamics (PSID), a longitudinal study of families, from which we draw information on household heads and their spouses between the ages of 25 and 55 who were interviewed between 1988 and 1997. Over this time period, the PSID collected detailed information on economic outcomes, but relatively little on health.

In addition, we draw on the Whitehall II study, a longitudinal study of British civil servants. We use information on individuals between the ages of 34 and 71, collected from 1985 to 2001. Because individuals had to be employed in the British civil service when the study began, the sample is not representative of the population in Britain. (See Case and Paxson,

2009b, for details.) However, it contains a rich set of measures of health and cognitive functioning in middle age to early old age. Our final data source is the Health and Retirement Study (HRS), which is a panel study of Americans who were aged 51 to 61 years in 1992. We use information on individuals ages 55 and older, interviewed between 1992 and 2004. The HRS contains information on employment and earnings (among those who are still employed) as well as health and cognitive function.

For each of these surveys, we select measures of economic and health status that are roughly comparable across the surveys, although not all measures were collected in all surveys. For the PSID and HRS, schooling is measured as reported years of education. For the NCDS and the BCS, individuals are asked to report the age at which they left full-time schooling, and we measure years of education as the school-leaving age minus 5. Using this method, some individuals have implausibly high values for years of education, possibly because they resumed full-time schooling in adulthood after taking time off. For this reason, we truncate years of schooling at 20 for these two surveys.⁴ We also analyze employment and the logarithm of average hourly earnings (conditional on employment). Our health measures include self-reported health status, activity limitations and disability, problems with activities of daily living, depression, and cognitive function. More details on these measures are provided below. We

⁴ The NCDS, BCS and Whitehall II studies collected detailed information on highest academic qualification attained by cohort members. These are better measures of educational achievement, and ordered probits reveal a significant association between highest degree and height in all three studies. However, it is difficult to map degrees onto years of education, or onto degrees conferred in the United States. For these reasons, we use the school leaving age for comparison with US cohorts.

regress these measures of economic and health outcomes on height in inches plus, where appropriate, indicators for age, ethnicity, sex and survey wave. In cases where there are multiple observations for an individual at different time periods, we use clustered standard errors.

Table 1 reports results for the economic outcomes. Results for men and women are reported separately because of their different labor force participation rates. We do not include results from Whitehall in this table. Although the data has a rich set of health measures, it does not include measures of hourly earnings, and there are numerous missing values for the measure of the school-leaving age. Furthermore selection into the sample was, by construction, contingent on being employed at the start of the study, so that the association between height and employment is of less interest. For the HRS, we use only individuals ages 55 to 74, since labor force participation is very low at older ages.

The results in Table 1 indicate that height is associated with economic outcomes in all of the samples. Each inch of height is associated with an increase in schooling of between 0.05 years (for 30-year-old women in the BCS) and 0.16 years (for men in the HRS). All coefficients on height in the schooling regressions are statistically different from zero. Although we do not present results from the Whitehall study here, in other work we show that height is significantly associated with the highest level of educational qualification received (Case and Paxson, 2009b). Employment is also associated with height for most groups. For men age 55 and under, each inch of height is associated with an increase in the probability of working of between 0.2 and 0.6 percentage points. Women have similar coefficients, although these are less precisely estimated among women in the NCDS. All groups show a substantial height premium in average hourly earnings, ranging from 0.012 to 0.028 for men, and 0.007 to 0.027 for women. The magnitudes of these coefficients are economically significant. For example, a height premium of 0.02 implies

that a 4-inch increase in height (a move from the 25th to 75th percentile in height) is associated with an 8 percent increase in hourly earnings.

Table 2 presents evidence on the association between height and a variety of health and cognitive outcomes in adulthood. All surveys ask respondents to rate their health. For the NCDS and BCS, self-reported health is measured on a four-point scale (1=excellent, 2=good, 3=fair and 4=poor). The other three studies use five-point scales (1=excellent, 2=very good, 3=good, 4=fair and 5=poor). The NCDS and BCS ask questions about whether the individual has a longstanding illness, or is registered as disabled; comparable questions do not exist in the other surveys. Whitehall II and HRS, both of which surveyed older individuals, contain information on problems with activities of daily living (ADLs). For Whitehall II, our measure of ADLs is the number of problems the individual has with seven items, including problems with vigorous activity, bathing or dressing, moderate activity, lifting, bending, walking several miles, and climbing several flights of stairs. For the HRS, we used six activities that reflect serious limitations: problems with dressing, walking across the room, bathing, eating, getting in and out of bed, and using the toilet. Both the HRS and Whitehall II surveys contain a measure of depression. Finally, both Whitehall II (in waves 3 and 5) and the HRS (in waves 3 through 6) conducted cognitive tests. For Whitehall II, we show results for tests of word recall and ability in arithmetic. For the HRS, we use a test of delayed word recall and an indicator of whether the individual could count backwards from 10.

In all samples, taller individuals report themselves to be in better health. In the NCDS and BCS, taller individuals are also less likely to report having a long-standing illness or to be registered as disabled. The magnitudes of these coefficients are large, in light of the fact that relatively few of these younger adults have serious illness or are disabled. For example, in the

BCS, 23.2 percent of individuals report a long-standing illness, and 1.5 are registered as disabled. The results imply that a 4-inch increase in height would reduce the probability of a long-standing illness from 23.2 percent to 21.4 percent, and reduce the probability of disability from 1.5 percent to 0.9 percent.

The association between height and health persists into older adulthood. Results from Whitehall II and the HRS indicate that taller individuals have fewer problems with activities of daily living, and are less likely to be depressed. Although the two surveys use different measures of depression, the results for both imply that a 4-inch increase in height is associated with a decline in the depression score of 4 percent of one standard deviation of the depression measure. Taller individuals also perform significantly better on cognitive tests, even among the (younger) Whitehall II sample.

Interpretation of results

The finding that height is associated with a wide range of economic and health outcomes throughout adulthood provides support for the hypothesis that early childhood circumstances have long-lasting effects. However, as discussed in the conceptual framework, there are several routes through which childhood circumstances could influence adult outcomes. One possibility is that height operates solely through educational attainment and economic status in early adulthood: children who are healthier and are raised in more advantaged circumstances achieve higher levels of education and get better first jobs, which in turn improves economic opportunities and health trajectories over adulthood. If so, then the "separability" discussed above would hold, and information on early adulthood would be sufficient to capture the effects of early life circumstances. Another possibility is that, although childhood circumstances may

influence education and economic status in early adulthood, they may also exert independent effects on long-run health and economic outcomes.

A number of studies have examined this issue by estimating reduced form models similar to those presented above (with additional controls for child health and economic circumstances, if available), and then examining whether the addition of controls for characteristics in young adulthood account for the association between childhood circumstances and later life outcomes. In some cases, the evidence suggests that childhood health influences adult outcomes, even controlling for outcomes in young adulthood. For example, Case, Fertig and Paxson (2005) show that, controlling for measures of socioeconomic status in childhood, chronic conditions in childhood and height at age 16 predict socioeconomic status of 42 year-old men from the National Child Development Study. This predictive power of chronic conditions and height at age 16 for socioeconomic status at age 42 continues even after controlling for education, health and socioeconomic status at ages 23 and 33. The same is true when examining self-reported health status at age 42.

Case and Paxson (2008b) find different results when using the Health and Retirement Study to examine the effects of height on a set of cognitive and health measures among older adults. In this case, although the reduced form estimates show that height predicts cognition and health, the coefficients on height become much smaller and are typically not significantly different from zero when controls for education are added. Older individuals who are more highly educated have better cognitive function and health status. It is unknown whether the "protective" effects of education are causal, or whether greater education reflects better cognitive ability and health in childhood.

To extend this research, we re-estimated the models shown in Tables 1 and 2, including controls for schooling, which are available for all of the data sets we use. (For Whitehall II, for which we do not have years of education, we included indicator variables for the highest degree attained.) In all cases, schooling is significantly associated with economic and health outcomes in adulthood. However, although the coefficients on height decline when controls for schooling are added, height continues to be significantly associated with adult outcomes in most cases. For the BCS, which assesses outcomes at the earliest stage of adulthood, the addition of schooling causes the coefficients on height to decline between 5 percent (for whether disabled) to 28 percent (for hourly earnings for women). The median decline in the coefficient on height is 30.3 percent for the NCDS; 54.9 percent for the PSID; and 7.5 percent for Whitehall II. Consistent with Case and Paxson (2008b), the associations between height and outcomes in old age in the HRS become much smaller once controls for years of schooling are included (the median decline is 79.2 percent.) The smallest declines among the HRS results are for average hourly earnings and the two cognitive tests, which remain significant even after schooling is included.

These results indicate that height, as a marker of early childhood circumstances, is associated with health, cognitive and economic outcomes throughout adulthood. This association appears to be partially but not fully mediated by schooling. Although these results underscore the importance of early childhood environment, they provide little information about what specific aspects of early childhood are being captured by height. We turn to this issue in the following section, where we use data from the NLSY79 Children and Young Adult Survey⁵ to document the associations between height, health and cognitive development among children.

HEIGHT AND EARLY LIFE HEALTH: EVIDENCE FROM THE NLSY

Beginning in 1986, data have been collected on the children of original female NLSY79 cohort members, as part of the NLSY Child and Young Adults Survey. These data provide information on a rich set of outcomes, including prenatal care and health at birth, children's cognitive function, schooling, and self perceptions of scholastic competence and self worth. In addition, all children born to an original NLSY79 woman can be identified as maternal siblings, which allows us to look at the associations between heights and developmental outcomes while controlling for maternal fixed effects. We use data on children who are 14.5 years of age and younger, collected every other year from 1986 to 2006.⁶

Table 3 provides descriptive statistics for these children. Eighteen percent of them were born to teen mothers, while 8 percent were born to mothers older than age 35. Approximately 8 percent were born at low birth weight (2500 grams). One in three was born to a mother who smoked during pregnancy, and one in five was born to a mother who did not seek prenatal care in the first trimester. We have information on children's heights and cognitive test score results,

⁵ The NLSY79 is a nationally representative longitudinal study of approximately 12,000 young men and women who were ages 14 to 22 when the study began in 1979. For a complete description of these data, go to <u>http://www.bls.gov/nls/nlsy79ch.htm</u>.

⁶Older children are included in the survey. However, because the NLSY79 consists of women born between 1958 and 1965, the older children in the sample were born to an increasingly selected sample of younger mothers.

both of which were collected in multiple waves of the survey. Information on mothers include her educational attainment, whether she lived with both parents at age 14, whether at age 14 her family received magazines and newspapers, whether her family had a library card, the educational attainment of her parents, her height, and her ethnicity. We will include information on mother's characteristics in some regression specifications, and mother fixed effects in others, to investigate whether there are unobservable mother-specific characteristics that affect children's heights and their development.

Table 4 presents evidence on the association between height and cognitive ability in childhood. The first panel presents results on PIAT-math and PIAT-word recognition scores for children of ages 5 and 6. The second panel presents results for children at ages 10 and 11. In addition to the earlier tests, older children are tested on PIAT-reading comprehension, digit span, and the Peabody Picture Vocabulary Test (PPVT). The bottom panel includes test score results for children at all ages. Each coefficient presented in Table 4 is the coefficient on the child's height-for-age z-score, in regressions of cognitive tests that include indicators for the child's age at testing (rounded to the nearest 6 months), an indicator for the child's birth order, and a set of race and survey year indicators.

For all tests at all ages, we find a positive and significant association between test scores and heights in OLS regressions – with associations becoming larger at older ages. With two exceptions, we continue to find a positive and significant association when mother fixed effects are added to regressions. The addition of mother fixed effects diminishes the association between height and cognitive tests for most tests, but does not eliminate it. The coefficient on height, in regressions that include mother fixed effects, is identified solely off of differences in test scores

between siblings, when tested at a given age, and differences in their height-for-age z-scores at that age. Siblings could differ in their heights and their test scores because the household environment one was born into could have been healthier, an explanation we will examine later in this section. Differences in heights could also be due to differences in the genetic material inherited from parents, or the interaction between genes and environment. Without more information, it is not possible to say more than this. However, we can say that, even controlling for the genetic material siblings share, and for that part of their home environment that is constant, children who were taller outperform shorter siblings on cognitive tests throughout childhood.

We also examine whether taller children progress more rapidly through school. We use two measures of progress in school: the highest grade attained, and an indicator if a child is at the appropriate grade, given his or her age. OLS regression coefficients on height are presented in columns 1 and 3 of Table 5, and those from regressions that include mother fixed effects are presented in columns 2 and 4. Results are reported for children ages 6 to 14, with the top panel presenting information on highest grade attained, and the bottom panel on whether the child is at an appropriate grade level (that is, whether children over 7 years and 6 months have completed grade 1, those over 8 years and 6 months have completed grade 2, and so forth).

Height-for-age z-scores at age five are significant predictors of children's progress through school, with or without controls for mother fixed effects. Using information on height at age five interacted with age (columns 3 and 4), we can test whether this is because taller children start school earlier, or because they advance more quickly through grades. At age 6, taller siblings are no more or less likely to be at appropriate grade-for-age: results including mother fixed effects suggest that a taller and shorter sibling differing by one standard deviation in height-for-age z-

scores are, at age 6, equally likely to reach grade-for-age ($-0.0179 + 0.0024 \times 6 = -0.0035$). However, by age 14, the taller sibling is 1.6 percentage points more likely to be observed at grade-for-age.

There are several reasons why taller children may progress more rapidly through school. One is that they have better cognitive skills. Another is that taller children have greater self esteem, perhaps because they are treated differently by parents, teachers or peers. We can distinguish between these two possibilities using data that were collected directly from the children. At ages 12 to 14, children were asked a battery of questions about how well they perform at school, and how pleased they are with their lives. Each individual item is scored from 1 (low) to 4 (high), and the Total Scholastic Competence score and Total Self Worth score are sums of individual items. Column 2 of Table 6 presents regression results in which a scholastic or self worth score is regressed on height for age z-scores, with controls for mother and child characteristics. Results in column 3 control in addition for mothers' fixed effects.

We find that taller children—and, within sib-ships, taller siblings—are more likely to report that they are good students. On average, their Total Scholastic Competence score is significantly higher, with or without mother fixed effects. These results are consistent with evidence that taller children have better cognitive test scores and progress through school more quickly than less tall children and siblings. In contrast, taller children and taller siblings are not more likely to report being pleased with themselves, or that they are happy with how they do things. Taller children are significantly *less* likely to report that they are "happy with the way I am." Total Self Worth scores are not significantly associated with height. The associations we find between height and cognitive test scores, educational attainment, and self-perceptions of scholastic competence do not appear to spill over into children's sense of their worth.

Previous research indicates that physical growth is related to children's health. Results from the NLSY Child and Young Adult Survey are consistent with this evidence. Children's heights are significantly associated with their health in childhood. Table 7 presents evidence that taller children, and taller siblings within sib-ships, are significantly less likely to have a limiting condition. Most prominently, taller children are less likely to be reported as having an emotional or neurological limiting condition.

Finally, the Children and Young Adults data allow us to investigate why some children, and within a sib-ship why some siblings, are taller than others. Table 8 looks the association between height-for-age z-scores in childhood and children's birth weight, birth length, and mothers' prenatal health. With or without mother fixed effects, we find that children who were heavier and longer at birth are significantly taller throughout childhood. This suggests that, even within a sib-ship, it may be difficult to place smaller newborns onto the same trajectory that larger siblings are on. Controlling for birth weight and length, we find that children born to mothers who received no prenatal care have height-for-age z-scores in childhood that are two tenths of a standard deviation shorter on average. This is true for comparisons across all children in OLS regression (column 1), or in mother fixed effect regressions, where identification comes from comparing mothers' behaviors and children's heights within sib-ships (column 2).

With controls for birth length and weight, we find maternal smoking and drinking while pregnant do not appear to affect height in childhood. However, this is because their effects are largely realized in birth weight and length. Columns 5 and 6 report the associations between birth weight, birth length, and mothers' behaviors while pregnant from regressions that include mother fixed effects. Controlling for all the characteristics of mothers that do not change between pregnancies, we find birth weight is significantly and negatively associated with smoking during

pregnancy, while birth length is significantly and negatively associated with reporting no prenatal care, and with drinking alcohol while pregnant.

CONCLUSION

There are several reasons why it is important to study of the long-run consequences of health in childhood. Knowledge about the effects of childhood health problems on economic and health outcomes later in life is useful for assessing the benefits of interventions that improve child health. Furthermore, an understanding of the childhood antecedents of adult disease may be useful for making projections of future trends in adult morbidity and disability. However, because of a lack of data that follows individuals from childhood to adulthood, it is difficult to quantify the consequences of childhood health. In this paper, we have shown how height, which reflects (in part) environmental conditions in the prenatal and early childhood periods, can be used to understand the long-run consequences of childhood health.

Physical growth in childhood is related to health in childhood. Data from the NLSY— Children and Young Adult Survey demonstrate that, within families, a mother's behavior during pregnancy can result in significant differences between her children—differences that may play out over the life course. Among children in general, and more strikingly among siblings, those who are heavier and longer at birth are taller in childhood on average. Among children in general, and among siblings more specifically, children who are taller are healthier, they score significantly higher on cognitive tests and make their way more quickly through school. They are also more likely to perceive themselves to be scholastically competent. All of these characteristics send children on different trajectories into adulthood and old age—trajectories that were, in part, set in place by mothers' behaviors while pregnant.

The evidence presented here on the associations between height and outcomes throughout adulthood indicate that childhood circumstances are important. Taller individuals attain more education, earn more, and are more likely to be employed. They are also more likely to have better health and cognitive outcomes in middle age to old age. A substantial portion of the better outcomes of taller individuals appears to be due to their higher levels of educational attainment. However, education cannot explain all of the association between height and later life outcomes.

When considering these results, it is important to keep in mind that the environments in which children are being raised today are substantially different than those in which the oldest individuals we examine—from the HRS and Whitehall—experienced as children. These older individuals grew up in environments in which major causes of child deaths included diarrhea, pneumonia, and diseases such as diphtheria, measles and typhoid (Case and Paxson, 2009a). A large body of literature indicates that exposures to infectious disease can have adverse implications for cognitive development and adult health (Finch and Crimmins, 2004; Costa, 2000). It may be tempting to speculate that, with the decline in infectious disease, childhood health will have a smaller role in determining adult outcomes. However, the fact that less healthy children born in the last two decades are shorter, and that these shorter children have worse performance on cognitive tests and slower progress through school, indicates that child health is likely to remain an important determinant of health and economic status once these children reach adulthood.

References

- Abbott, R.D., L.R. White, G.W. Ross, H. Petrovitch, K.H. Masaki, D.A. Snowdon, and J.D. Curb. 1998. "Height as a Marker of Childhood Development and Late-life Cognitive Function: The Honolulu-Asia Aging Study." *Pediatrics* 102(3): 602-9.
- Adler, N.E., T. Boyce, M.A. Chesney, S. Cohen, S. Folkman, R.L. Kahn, and S. L.Syme. 1994. "Socioeconomic Status and Health: The Challenge of the Gradient," *American Psychologist*, 15-24.
- Almond, D. 2006. "Is the 1918 Influenza Pandemic Over? Long-term Effects of *in utero* Influenza Exposure in the Post-1940 U.S. Population." Journal of Political Economy 114(4): 672-712.
- Barker, D.J.P. 1992. "The Fetal Origins of Diseases of Old Age," *European Journal of Clinical Nutrition* 46 (Supplement 3): S3-9.
- Barker, D.J.P. 1995. "Fetal Origins of Coronary Heart Disease," *British Medical Journal* 311(6998): 171-74.
- Barker, D.J.P. 1998. "Programming the Baby," Pp. 13-41 in *Mothers, Babies and Health in Later Life*, 2nd ed., Churchill Livingstone, Edinburgh.
- Barker, D.J.P. 2004. "The Developmental Origins of Well-being." *Philosophical Transactions of the Royal Society B* 359: 1359-66.
- Bateson, P., D.J.P. Barker, T. Clutton-Brock, D. Deb, B. D'Udine, R.A. Foley, P. Gluckman, K. Godfrey, T. Kirkwood, M.M. Lahr, J. McNamara, N.B. Metcalfe, P. Monaghan, H.G. Spencer, and S.E. Sultan. 2004. "Developmental Plasticity and Human Health." *Nature* 430:419-21.
- Beard, A. S. and M. J. Blaser. 2002. "The Ecology of Height: The Effect of Microbial Transmission on Human Height." *Perspectives in Biology and Medicine* 45 (Autumn): 475-99.
- Behrman, J. R. and M. R. Rosenzweig. 2004. "Returns to Birthweight." *Review of Economics* and Statistics 86 (May): 586-601.
- Bertram, C.E., and M.A. Hanson. 2001. "Animal Models and Programming of the Metabolic Syndrome." *Br Med Bull*. 60:103-21.
- Black, S.E., P.J. Devereux, and K.G. Salvanes. 2007. "From the Cradle to the Labor Market? The Effect of Birth Weight on Adult Outcomes." *Quarterly Journal of Economics* 122 (February): 409-439.
- Brunner, E., M. J. Shipley, D. Blane, G. Davey Smith, and M. G. Marmot. 1999. "When Does Cardiovascular Risk Start? Past and Present Socioeconomic Circumstances and Risk Factors in Adulthood," *Journal of Epidemiology and Community Health* 53: 757-64.
- Case, A. and C. Paxson, 2008a. "Stature and Status: Height, Ability and Labor Market Outcomes." *Journal of Political Economy* 116(3): 499-532.
- Case, A. and C. Paxson. 2008b. "Height, Health and Cognitive Function at Older Ages." *American Economic Review Papers and Proceedings* 98(2): 463-467.
- Case, A. and C. Paxson. 2009a. "Early Life Health and Cognitive Function in Old Age," *American Economic Review Papers and Proceedings* 99(2): 104-109.
- Case, A. and C. Paxson. 2009b. "The Long Reach of Childhood Health and Circumstance: Evidence from the Whitehall II Study," mimeo, Center for Health and Wellbeing, Princeton University.

- Case, A., A. Fertig and C. Paxson. 2005. "The Lasting Impact of Childhood Health and Circumstance." *Journal of Health Economics* 24: 365-389.
- Case, A., C. Paxson and M. Islam. 2008. "Making Sense of the Labor Market Height Premium: Evidence from the British Household Panel Study." *Economic Letters* 102(3): 174-6.
- Chandola, T., M. Bartley, A. Sacker, C. Jenkinson, and M. Marmot. 2003. "Health Selection in the Whitehall II Study, UK" *Social Science and Medicine* 56: 2059-72.
- Costa, D. "Causes of Improving Health and Longevity at Older Ages: A Review of the Explanations," *Genus* LXI(1): 21-38.
- Crimmins, E. M., and C.E. Finch. 2006. "Infection, Inflammation, Height and Longevity." *Proceedings of the National Academy of Sciences* 103 (January 10): 498-503.
- Currie, J. 2009. "Healthy, Wealthy and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development." *Journal of Economic Literature* 47(1): 87-122.
- Cutler, D. and A. Lleras-Muney. 2008. "Education and Health: Evaluating Theories and Evidence," Chapter 2 of Schoeni, R.F., J. House, G.A. Kaplan and H. Pollack, eds. *Making American Healthier*, New York: Russell Sage Foundation.
- Dahlgren, J., C. Nilsson, E. Jennische, H.P. Ho, E. Eriksson, A. Niklasson, P. Bjorntorp, K. Albertsson Wikland, and A. Holmang. 2001. "Prenatal Cytokine Exposure Results in Obesity and Gender-Specific Programming." *Am J Physiol Endocrinol Metab.* 281:E326-34.
- Dickerson, S.S. and M.E. Kemeny. 2004. "Acute Stressors and Cortisol Responses: A Theoretical Integration and Synthesis of Laboratory Research." *Psychological Bulletin* 130:355-91.
- Drake, A.J. and B.R. Walker. 2004. "The Intergenerational Effects of Fetal Programming: Non-genomic Mechanisms for the Inheritance of Low Birth Weight and Cardiovascular Risk." *J Endocrinol*. 180:1-16.
- Finch, C.E. and E.M. Crimmins. 2004. "Inflammatory Exposures and Historical Changes in Human Life Spans," *Science* 305(5691): 1736-1739.
- Grantham-McGregor, S. 2002. "Linear Growth Retardation and Cognition." *Lancet* 359 (February 16): 542.
- Holmang, A. 2001. "Perinatal Origin of Adult Disease." Scand Cardiovasc J. 35:178-85.
- Holness, M.J., M.L. Langdown and M.C. Sugden. 2000. "Early-life Programming of Susceptibility to Dysregulation of Glucose Metabolism and the Development of Type 2 diabetes mellitus." *Biochem J*. 1:349 Pt 3:657-65.
- Husén, T. 1959. Psychological Twin Research. New York: Free Press.
- Jackson, A.A. 2000. "Nutrients, Growth, and the Development of Programmed Metabolic Function." *Adv Exp Med Biol.* 478:41-55.
- Kajantie E. 2003. *Mechanisms of Growth in Small Preterm Infants and Early Life Origins of Adult Cardiovascular Disease*. Thesis. University of Helsinki, Hospital for Children and Adolescents 24.1.2003. Available at http://ethesis.helsinki.fi .
- Kajantie, E, L. Dunkel, U. Turpeinen, U.H. Stenman, P.J. Wood, M. Nuutila and S. Andersson. 2003. "Placental 11β-hydroxysteroid Dehydrogenase-2 and Fetal Cortisol / Cortisone Shuttle in Small Preterm Infants." *J Clin Endocrinol Metab* 88:493-500.
- Kajantie, E., J. Eriksson, D.J.P. Barker, T. Forsén, C. Osmond, P.J. Wood, S. Andersson, L. Dunkel, and D.I.W. Phillips. 2003. "Birthsize, Gestational Age and Adrenal

Function in Adult Life: Studies of Dexamethasone Suppression and ACTH₁₋₂₄ Stimulation." *Eur J Endocrinol* 149:569-75.

- Kajantie, E., J. Eriksson, C. Osmond, P.J. Wood, T. Forsen, D.J.P. Barker and D.I. Phillips. 2004. "Size at Birth, the Metabolic Syndrome and 24-h Salivary Cortisol Profile." *Clin Endocrinol* (Oxf). 60:201-7.
- Langley-Evans, S.C. 2000. "Critical Differences Between Two Low Protein Diet Protocols in the Programming of Hypertension in the Rat." *Int J Food Sci Nutr.* 51: 11-17.
- Li, L., O. Manor and C. Power. 2004. "Early Environment and Child-to-Adult Growth Trajectories in the 1958 British Birth Cohort." *American Journal of Clinical Nutrition* 80: 185-92.
- Lynn, R. 1989. "A Nutrition Theory of the Secular Increases in Intelligence, Positive Correlation between Height, Head Size and IQ." *British Journal of Educational Psychology* 59: 372-77.
- Marmot, M., M. Shipley, E. Brunner, and H. Hemingway. 2001. "Relative Contribution of Early Life and Adult Socioeconomic Factors to Adult Morbidity in the Whitehall II Study." *Journal of Epidemiology and Community Health* 55: 301-7.
- McMillen, I.C. and J.S. Robinson. 2005. Developmental Origins of the Metabolic Syndrome: Prediction, Plasticity, and Programming. *Physiol Rev* 85: 571-633.
- Nilsson, C., M. Niklasson, E. Eriksson, P. Bjorntorp, and A. Holmang. 1998. "Imprinting of Female Offspring with Testosterone Results in Insulin Resistance and Changes in Body Fat Distribution at Adult Age in Rats." *J Clin Invest*. 101:74-8.
- Ozanne, S.E. 2001. "Metabolic Programming in Animals." Br Med Bull. 60:143-52.
- Persico, N., A. Postlewaite, and D. Silverman, 2004, "The Effect of Adolescent Experience on Labor Market Outcomes: The Case of Height," *Journal of Political Economy*, 112(5), 1019-53.
- Phillips, D.I. 2001. "Fetal Growth and Programming of the Hypothalamic-Pituitary-Adrenal Axis." *Clin Exp Pharmacol Physiol* 28:967-70.
- Samuelsson, A.M., I. Ohrn, J. Dahlgren, E. Eriksson, B. Angelin, B. Folkow and A. Holmang. 2004. "Prenatal Exposure to Interleukin-6 Results in Hypertension and Increased Hypothalamic-pituitary-adrenal Axis Activity in Adult Rats." *Endocrinology* 145:4897-911.
- Räikkönen, K., T. Forsén, M. Henriksson, E. Kajantie, K. Heinonen, A. Pesonen, J.T. Leskinen, I. Laaksonen, C. Osmond, D.J.P. Barker and J.G. Eriksson. 2009. "Growth Trajectories and Intellectual Abilities in Young Adulthood, The Helsinki Birth Cohort Study." *American Journal of Epidemiology* 170(4): 447-455.
- Rutter, M. and the English and Romanian Adoptees (ERA) study team. 1998. "Developmental Catch-up and Deficit Following Adoption After Severe Global Early Privation." *Journal of Child Psychology and Psychiatry* 39: 465-476.
- Saenger, P., P. Czernichow, I. Hughes and E.O. Reiter. 2007. "Small for Gestational Age: Short Stature and Beyond," Endocrine Reviews, 28(2): 219-251.
- Smith, J.P. 2009. "The Impact of Childhood Health on Adult Labor Market Outcomes," *The Review of Economics and Statistics* 91(3):478-489.
- Silventoinen, K. 2003. "Determinants of Variation in Adult Body Height." *Journal of Biosocial Science* 35 (April): 263-85.
- Steckel, R. H. 1995. "Stature and the Standard of Living." *Journal of Economic Literature* 33 (December): 1903-40.

- Sundet, J. M., K. Tambs, J. R. Harris, P. Magnus, and T. M. Torjussen. 2005. "Resolving the Genetic and Environmental Sources of the Correlation Between Height and Intelligence: A Study of Nearly 2600 Norwegian Male Twin Pairs." *Twin Research and Human Genetics* 8 (August): 307-11.
- Walker, S.P., S.M. Chang, C.A. Powell, and S. M. Grantham-McGregor. 2005. "Effects of Early Childhood Psychosocial Stimulation and Nutritional Supplementation on Cognition and Education in Growth-Stunted Jamaican Children: Prospective Cohort Study." *The Lancet* 366(9499): 1804-7.

Table 1: Regressions of education, employment and earnings on height in inches

Dependent variable:	NCDS	NCDS	BCS	PSID	HRS	
	age 33	age 42	age 30	ages 25-55	ages 55-74	
		Men				
Schooling (years)	0.0913		0.0679	0.0686	0.1571	
	(0.0114)		(0.0104)	(0.0111)	(0.0164)	
Employed (indicator)	0.0061 (0.0015)			0.0018 (0.0008)	0.0015 (0.0019)	
Log average hourly earnings, if employed	0.0262	0.0248	0.0122	0.0145	0.0277	
	(0.0037)	(0.0042)	(0.0023)	(0.0029)	(0.0051)	
Maximum observations		5,833	5,424	31,996	27,606	
		Women				
Schooling (years)	0.0940		0.0550	0.0454	0.1185	
	(0.0107)		(0.0104)	(0.0106)	(0.0119)	
Employed (indicator)	0.0036	0.0028	0.0093	0.0023	0.0029	
	(0.0025)	(0.0022)	(0.0021)	(0.0010)	(0.0017)	
Log average hourly earnings, if employed	0.0170	0.0195	0.0114	0.0067	0.0272	
	(0.0042)	(0.0048)	(0.0025)	(0.0030)	(0.0044)	
Maximum observations		5,815	5,757	31,999	35,927	

Note: Each cell contains a coefficient and standard error on height in inches, from a regression of the dependent variable on height and a set of ethnicity indicators. For results using the PSID and HRS, indicators for year of age are also included. For all outcomes except schooling, the PSID and HRS contain multiple observations per person, and so survey wave/year dummies and included and standard errors are clustered at the individual level. "Schooling" is defined as reported years of schooling for the PSID and HRS, and as the age at which the individual left full-time education minus 5 for the NCDS and BCS. In some cases, regressions contain smaller numbers of observations due to missing values of the dependent variables.

Table 2: Regressions of measures	of health and	cognition of	n height in inches
	01 11001111 01110	•••••••••••••••••••••••••••••••••••••••	

Dependent variable:	NCDS age 33	NCDS age 42	BCS age 30	PSID ages 25-55	Whitehall II ages 34-71	HRS ages 55+
Self-reported health status ¹	-0.0171 (0.0026)	-0.0150 (0.0028)	-0.0126 (0.0023)	-0.0085 (0.0027)	-0.0241 (0.0032)	-0.0118 (0.0031)
Has long-standing illness	-0.0010 (0.0014)	-0.0036 (0.0017)	-0.0044 (0.0014)			
Is registered as disabled	-0.0023 (0.0004)	-0.0016 (0.0006)	-0.0016 (0.0004)			
# ADLs ²					-0.0141 (0.0004)	-0.0024 (0.0020)
Depression score ³					-0.0758 (0.0260)	-0.0214 (0.0047)
Word recall score ⁴					0.0574 (0.0101)	0.0434 (0.0046)
Counting backwards ⁵						0.0041 (0.0007)
Numerical ability score ⁶					0.4419 (0.0278)	(0.0007)
Maximum observations	11,	648	11,181	63,995	29774	66,269

Notes: Each cell contains a coefficient and standard error on height in inches, from a regression of the dependent variable on height, an indicator for the respondent's sex, and (for all but Whitehall) a set of ethnicity indicators. For the PSID, Whitehall and HRS, indicators for year of age are included. The PSID, Whitehall and HRS contain multiple observations per person, so survey wave/year indicators are included, and standard errors are clustered at the individual level. For some outcomes, regressions contained smaller numbers of observations because data were not collected in all waves, or because of missing values of the dependent variables.

¹For NCDS and BCS: 1=Excellent, 2=Good, 3=Fair, 4=Poor; for PSID, Whitehall and HRS: 1=Excellent, 2=Very good, 3=Good, 4=Fair, 5=Poor ²For Whitehall, range=0 to 7, mean=0.28; for the HRS, range=0 to 5, mean=0.26.

³For Whitehall, range=0 to 45, mean=13.8, standard deviation=7.3; for HRS, range=0 to 8, mean=1.4, standard deviation=1.9.

⁴For Whitehall, range=0 to 18, mean=6.4, standard deviation=2.4; for HRS, range=0 to 10, mean=4.6, standard deviation=2.1;

⁵Coded as equal to 1 if the individual could accurately count backwards from 10, and 0 if not. Mean=0.88.

⁶Range=0 to 33, mean=22.4, standard deviation=6.5.

	Mean	Std. Dev.	observations						
Child characteristics (on	Child characteristics (one observation per child)								
Mother was teenager at child's birth	0.181		10,324						
Mother was older than 35 at child's birth	0.078		10.324						
Child's birth order	1.97		10,324						
Child female	0.489		10,324						
Child born at low weight	0.075		10,318						
Mother smoked during pregnancy	0.322		9,422						
Mother had no prenatal care	0.015		9,200						
Mother began prenatal care in 1 st trimester	0.799		9,200						
Mother began prenatal care in 2 nd trimester	0.144		9,200						
Mother began prenatal care in 3 rd trimester	0.039		9,200						
Child characteristics (multi	ple observati	ons per child)							
Piat—math: math test	100.35	(14.08)	31,175						
Piat—rec: reading recognition test	103.95	(14.85)	31,045						
Piat—comp: reading comprehension test	101.17	(13.96)	26,199						
Digit span test (range=1 to 19)	9.78	(3.12)	16,968						
Peabody Picture Vocabulary Test (PPVT)	90.45	(20.30)	17,341						
Height-for-age z-score	0.137	(1.36)	46,671						
Mother characteristics									
Highest grade attained	13.21		3,778						
Lived with intact family at age 14	0.745		4,433						
Family at age 14 received magazines	0.524		4,445						
Family at age 15 received newspaper	0.724		4,447						
Family at age 14 had library card	0.695		4,446						
Highest grade attained by mother's mother	10.56		4,206						
Highest grade attained by mother's father	10.59		3,770						
Mother's adult height (inches)	64.24		3,248						
Mother Hispanic	0.179		4,471						
Mother Black	0.267		4,471						

Table 3: Descriptive Statistics, NLSY Child and Young Adult Sample (ages 0 to 14.5 years)

Table 4: Heights and test scores

	PIAT-math	PIAT-rec	PIAT- comp	Digit span	PPVT		
Regressions of test	scores on height-f	for-age z-scores	s, 5 and 6 yea	r olds			
No mother fixed effects	0.637	0.395					
	(0.124)	(0.121)					
Mother fixed effects	0.402	0.267					
	(0.183)	(0.180)					
Observations	6,492	6,384					
Regressions of test set	Regressions of test scores on height-for-age z-scores, 10 and 11 year olds						
No mother fixed effects	0.734	0.559	0.548	0.133	0.753		
	(0.151)	(0.161)	(0.147)	(0.035)	(0.196)		
Mother fixed effects	0.744	0.299	0.463	0.091	0.201		
	(0.218)	(0.221)	(0.208)	(0.052)	(0.252)		
Observations	6,206	6,192	6,133	6,162	6,105		
Regressions of	test scores on hei	ght-for-age z-s	cores, all age	S			
No mother fixed effects	0.713	0.587	0.520	0.175	0.971		
	(0.087)	(0.095)	(0.087)	(0.024)	(0.136)		
No mother fixed effects, mother	0.642	0.573	0.535	0.167	0.925		
controls*	(0.108)	(0.116)	(0.108)	(0.030)	(0.167)		
Mother fixed effects	0.376	0.192	0.248	0.089	0.362		
	(0.078)	(0.083)	(0.084)	(0.025)	(0.148)		
Observations	30,421	30,296	25,606	16,579	16,853		

Notes: All models include indicators for the child's age (rounded to the nearest 6 months) at testing, an indicator for the child's sex, whether he or she was born to a teen-aged mother or an older (over 35) mother, the child's birth order, and a set of race and survey year indicators. The models with "mother controls" include the mother's highest grade; indicators for whether the mother (at age 14) lived in a family that was intact, received magazines, a newspaper and had a library card, and lived in an urban area; the highest grade attained by her mother and father, and her adult height. For the top two panels, there is one observation per child. In the bottom panel, with multiple observations per child, the standard errors are clustered at the child level. *Samples with mother controls included are smaller than those noted in table, due to missing values.

	(1)	(2)	(3)	(4)
	Highest grade at	tained		
Height-for-age z-score at age 5	0.0177 (0.004)	0.0086 (0.0051)	-0.0145 (0.0095)	-0.0287 (0.0109)
Height-for-age z-score at age 5 * age			0.0033 (0.0010)	0.0038 (0.0011)
At appropriate gra	de level given ag	e (indicator, mea	n=0.873)	
Height-for-age z-score at age 5	0.0118 (0.0023)	0.0056 (0.0029)	-0.0092 (0.0048)	-0.0179 (0.0061)
Height-for-age z-score at age 5 * age			0.0022 (0.0006)	0.0024 (0.0007)
Mother fixed effects?	no	yes	No	Yes

Table 5: Grade level in school and height, children ages 6 to 14

Notes: Observation=23783. "At appropriate grade level" is defined to equal 1 if the children over 7 years and 6 months of age have completed grade 1, if children over 8 years and 6 months have completed grade 2, and so on. All models include a set of indicators for the child's age rounded to the nearest 6 months, an indicator for the child's sex, whether he or she was born to a teen-aged mother or an older (over 35) mother, the child's birth order, and a set of race and survey year indicators. Standard errors are clustered at the child level.

	Mean of	Coefficient on heigh	t-for-age z-score
	dependent variable	No fixed effects	Mother fixed effects
	Scholastic Competence		
Good at school work	2.93	0.030	0.025
	2.95	(0.011)	(0.018)
As smart as other kids the same age	3.06	0.035	0.019
	3.00	(0.011)	(0.018)
Does schoolwork quickly	2.84	0.042	0.041
	2.84	(0.012)	(0.020)
Remembers things easily	2 78	0.034	0.039
	2.78	(0.011)	(0.018)
Does class work well	2 22	0.013	0.012
	3.22	(0.010)	(0.017)
Can figure out answers at school	2.00	0.028	0.027
C C	2.66	(0.011)	(0.019)
Total Scholastic Competence score	17.50	0.183	0.164
	17.50	(0.046)	(0.072)
	Self Worth		
Pleased with self	3.44	-0.010	0.008
	5.44	(0.009)	(0.014)
Likes the way leading life	3.41	-0.013	-0.004
	5.41	(0.010)	(0.016)
Happy with self as a person	2.60	-0.008	-0.002
	3.60	(0.008)	(0.013)
Likes who I am	2.41	0.008	0.013
	3.41	(0.009)	(0.016)
Happy with the way I am	2.42	-0.020	-0.029
	3.43	(0.009)	(0.015)
Happy with how I do things	2.20	0.004	0.010
	3.29	(0.010)	(0.017)
Total Self Worth score	20.50	-0.043	-0.011
	20.58	(0.037)	(0.057)

Table 6: Child's self-perception and height, ages 12 to 14

Note: Observations= 7,438. Each individual item is scored from 1 (low) to 4 (high), and the Total scores are sums of the individual items. All models include a set of indicators for the child's age rounded to the nearest 6 months, an indicator for the child's sex, whether he or she was born to a teen-aged mother or an older (over 35) mother, the child's birth order, and a set of race and survey year indicators. Standard errors are clustered at the child level.

		Has a limiting condition that is:				
	Has limiting condition	Emotional/neurological	Respiratory	Other		
Mean of dependent variable	0.100	0.022	0.045	0.040		
No mother fixed effects	-0.0050 (0.0014)	-0.0026 (0.0007)	0.0004 (0.0009)	-0.0031 (0.0009)		
Mother fixed effects	-0.0031 (0.0013)	-0.0017 (0.0007)	0.0007 (0.0008)	-0.0018 (0.0009)		

Table 7: Regressions of health measures on height-for-age z-scores

Notes: Observations=46,101. All models include indicators for the child's age (rounded to the nearest 6 months) at testing, an indicator for the child's sex, whether he or she was born to a teen-aged mother or an older (over 35) mother, the child's birth order, and a set of race and survey year indicators. Standard errors are clustered at the child level.

Table 8: Prenatal health and height

	Height-for-age z-score in childhood				Birth weight z-score	Birth length z-score
Birth weight z-score	0.147	0.105				
	(0.013)	(0.015)				
Birth length z-score	0.098	0.093				
	(0.010)	(0.011)				
No prenatal care	-0.213	-0.227	-0.266	-0.264	-0.028	-0.325
-	(0.101)	(0.107)	(0.106)	(0.111)	(0.127)	(0.181)
Prenatal care, 2 nd trimester	0.026	0.047	0.005	0.026	-0.130	-0.051
	(0.033)	(0.033)	(0.034)	(0.034)	(0.038)	(0.055)
Prenatal care, 3 rd trimester	0.082	0.096	0.085	0.095	-0.057	0.064
	(0.055)	(0.051)	(0.057)	(0.052)	(0.066)	(0.095)
Smoking frequency	-0.008	-0.022	-0.067	-0.040	-0.097	-0.071
	(0.018)	(0.032)	(0.019)	(0.033)	(0.035)	(0.050)
Drinking frequency	0.009	0.004	0.004	-0.003	-0.023	-0.060
	(0.010)	(0.012)	(0.010)	(0.123)	(0.014)	(0.020)
Mother fixed effects?	no	yes	no	yes	yes	yes
Observations		37,	568		8,088	8,088

Notes: Regressions for which the dependent variable is the height-for-age z-score include indicators for the child's age (rounded to the nearest 6 months), an indicator for the child's sex, whether he or she was born to a teen-aged mother or an older (over 35) mother, the child's birth order, and a set of race and survey year indicators. Standard errors are clustered at the child level. Regressions for which the dependent variable is birth weight or birth length include a cubic for the mother's age at the time of the birth, an indicator for the child's sex, whether he or she was born to a teen-aged mother or an older (over 35) mother, the child's birth order, and a set of race and survey year indicators. Standard errors are clustered at the order, and a set of race and survey year indicators. Standard errors are clustered at the child solved.