

VERY PRELIMINARY – Comments Welcome

Healthy, Wealthy, and Wise: Is there a Causal Relationship Between Child Health and Human Capital Development?

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Abstract

There are many possible pathways between parental education, income, and health, and child health and education, but only some of them have been explored in the literature. This essay focuses on links between parental socioeconomic status (as measured by education, income, occupation, or in some cases area of residence) and child health, and between child health and child education. Specifically, I ask two questions: What is the evidence regarding whether parental socioeconomic status affects child health? And, what is the evidence relating child health to future outcomes? I show that there is now strong evidence of these links, but that it is difficult to say how large the overall effect of health might be or whether it explains a significant amount of the intergenerational transmission of socioeconomic status.

When economists think of “human capital,” they often mean education. A large body of literature has established that investments in education pay off in the form of higher future earnings, and that on average, differences in education can explain a significant fraction of the variation in wages and incomes among adults, as well as variation in many other positive outcomes. But what determines a child’s educational success? Most studies point to family background as the number one factor. However, this raises a “chicken and egg” problem. We know that more education leads to higher income, and that children in higher income families are likely to get more education than other children. But why does income matter? Clearly we need to open the “black box” of the family in order to determine why it is that better backgrounds promote success in life.

While there are clearly many things about background that might matter, research increasingly implicates health as a potentially major factor. It may be that low parental education and income is linked to poorer child health, which in turn is linked to poorer educational attainments. In this case, health might be an important pathway for the well-documented intergenerational correlations in income and socio-economic status (Solon, 1999). While the literature on intergenerational correlations in economic status has made important strides in measurement, less is known about the mechanisms underlying the transfer of economic status between generations. Given the importance of “health capital” for education and earnings (Grossman, 1972; Case, Fertig, and Paxson, 2005; Currie and Madrian, 1999), it is possible that poor health in childhood is an important mechanism for intergenerational transmission of economic status.

There are many possible pathways between parental education, income, and health, on the one hand, and child health and education on the other, but only some of them have been explored in

the literature. This essay focuses on links between parental socioeconomic status (as measured by education, income, occupation, or in some cases area of residence) and child health, and between child health and child education. Specifically, I ask two questions: What is the evidence regarding whether parental socioeconomic status affects child health? And, what is the evidence relating child health to future outcomes? The focus is primarily on children from developed countries, because it is perhaps more obvious why the common and severe health problems of children in many developing countries might impede human capital development.

The remainder of this essay is structured as follows: Section 1 discusses evidence regarding correlations between child health and future outcomes. Section 2 begins the discussion of whether the correlations that have been documented in the literature imply causality. Section 3 examines this question with specific reference to many studies of the effects of low birth weight. Section 4 moves on to other health measures. Section 5 considers the issue of how large the effects seem to be and whether they are likely to explain much of the variance in outcomes, or intergenerational transmission. Section 6 concludes.

I show that there is strong evidence of links between parental socioeconomic status and child health and between child health and future outcomes, but that it is difficult to say on the basis of the available evidence how large the overall effect of health might be or whether it explains a significant amount of intergenerational transmission. Further progress may await more complete data linking health in childhood to eventual attainments.

1. Correlations Between Parents' Socioeconomic Status and Child Health

A good deal of evidence links parent's socioeconomic status (SES), as measured by income, education, occupation, or even area of residence, to child health. Differences in the health of high and low SES children are apparent at birth. The best evidence of this is the differential incidence of low birth weight (birth weight less than 2,500 grams). These gaps are surprisingly persistent over time and even across countries. For instance, using data from the 1958 British birth cohort, which followed all the children born in one week in March, Currie and Hyson (1999) show that among children whose father's were in the highest prestige occupations, 5 percent were low birth weight compared to 6.4 percent of children whose father's were in the lowest prestige occupations (or whose father information was missing). In California, birth records indicate that in the 1970s, 6 percent of children born into the highest income quartile of zip codes were low birth weight compared to 7 percent of children born into the lowest income quartile. By the 1990s, the incidence of low birth weight had decreased slightly for both groups, but the gap had widened: The comparable numbers were 5.5 percent and 6.8 percent.

Maternal reports about child health (in a standard question, mothers are asked if the child's health is excellent, very good, good, fair, or poor) also suggest that poor children are in worse health than richer children. In an important paper, Case, Lubotsky, and Paxson showed that in the U.S., these gaps in health status also tended to grow as children age. Table 1 shows estimates from ordered probits exploring the relationship between family income and child health for various age groups. Panel 1 reproduces estimates from Case, Lubotsky and Paxson, while panels 2 and 3 show estimates for similar analyses using Canadian and British data.

All of the estimates are negative and significant, showing that reported health status increases with income, even in early childhood. The analyses using Canadian and British data are notable because they show that even in countries with universal health insurance, poor children are in worse health than richer children. However, for each age group, the estimates are smaller in absolute value in Canada and the U.K. Moreover, the relationship between income and health flattens off in the U.K., and even declines as children age, in contrast to the monotonically increasing pattern seen in Canada and the U.S. These patterns suggest that even within countries with universal health insurance, child health can evolve in different ways, perhaps because of differences in other public policies that affect child health, such as mother's allowances, or public housing.

1. b) Correlations Between Parent's Status and Insults to Health

Currie and Stabile (2003) suggest that variations in the incidence of health insults may be of particular importance in explaining the gap in health status between rich and poor. In their Canadian panel data, poor children receive many more health insults (such as new diagnoses of chronic conditions, and hospitalizations) than richer children. The poor children recover more slowly, but after four years have recovered to the same extent as the richer children--the real difference is in the incidence of the negative shocks to health.

Many authors have documented the fact that poor children suffer more insults to their health than richer ones. For example, Newacheck (1994), Newacheck and Halfon (1998), and Case, Lubotsky and Paxson (2002) all show that poor children are more likely to have many chronic conditions. In the U.K., 11 percent of 0 to 3 year old children have chronic conditions in

families with income over 50,000 pounds, compared to 23 percent of 0 to 3 year old children in families with incomes less than 10,000 pounds per year (Currie, Price, and Shields, 2004). In the U.K. this result is driven by a higher incidence of asthma and mental health problems, the two most common classes of chronic conditions. In the U.S., a wider array of chronic conditions, such as arthritis and heart problems, are sensitive to income.

Currie and Lin (2007) update these analyses for the U.S. using data from the U.S. National Health Interview Survey for 2001-2005, and by examining mental health conditions. Mental health conditions are very prevalent, may have large impacts on educational attainments, and differentially affect poor children. But they that have been largely ignored in previous studies. Table 2 shows a sharp difference in maternal assessments of health by poverty status. Only 70 percent of poor children are reported to be in excellent or very good health compared to 86.9 percent of higher income children. The gap grows from 15.5 percentage points among 2 to 3 year olds to 19.2 percentage points among 13 to 17 year olds.

Asthma is the leading chronic condition among children and is also known to be one of the leading causes of pediatric emergency room utilization, hospitalization, and school absence (U.S. Environmental Protection Agency, 2006). Table 2 shows that approximately 1.3 million poor children, and 5.8 million non-poor children have asthma by this measure. Mental health conditions are the second most prevalent set of conditions, with Attention Deficit Hyperactivity Disorder (ADHD) being the largest single diagnosis within this category: parents of 7.1 percent of poor children and 6.0 percent of non-poor children have been told that their children have ADHD.¹ Hearing, vision, and speech problems, such as stuttering or stammering, are together

¹ ADHD is characterized by an inability to pay attention (inattention) and/or hyperactivity.

the third most common category of chronic conditions. Taking all of the listed chronic conditions together, a staggering 32.4 percent of poor children and 26.5 percent of other children have at least one of these conditions.

Disparities between rich and poor in the extent to which children are limited by their conditions are much greater than disparities in the prevalence of conditions between poor and non-poor children: 11.4 percent of poor children say that they are limited by their chronic conditions compared to 7.0 percent of higher income children. Poor children are 1.2 times more likely to have ever been told that they have asthma than other children, but they are 3.2 times more likely to be limited by asthma. The fraction of children with a limitation due to a chronic condition rises with age, and rises more sharply for poor children than for others. By the teenage years, poor children have almost double the probability of being limited by their chronic condition: 14.1 percent compared to 7.8 percent of other children.

Table 2 also suggests that there is a discrepancy between the conditions that are most prevalent, and the conditions that cause the most activity limitations. While asthma is the most prevalent condition, it is not the most prevalent limiting condition—that distinction belongs to mental health problems such as ADHD. Only 1.9 percent of poor children (158,451) are reported to be limited by asthma, compared to 6.2 percent (517,049) who are limited by a mental health problem (of these, 2.3 percent or 191,809 poor children are limited by ADHD).

The main diagnostic criteria for ADHD are laid out in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (American Psychiatric Association, 1994). They are: that six or more symptoms of inattention, or six or more symptoms of hyperactivity have persisted for at least six months “to a degree that is maladaptive and inconsistent with developmental level” (AAP, 2000, Table 1); that some of the symptoms were present before seven years of age; and that impairment from the symptom is present in two or more settings (such as home and school). This last criterion means that teachers are often important for the diagnosis of ADHD.

Turning to illnesses, many children suffer from asthma attacks and 3.2 percent of all poor children end up in emergency rooms due to an attack each year, compared to only 1.6 percent of other children. It is anomalous then that respiratory allergies are reported to be more common among other children than among poor children. It is likely that the same symptoms are more likely to be diagnosed as allergies (rather than as, for example, a large number of acute illnesses) among higher income children. Finally, a surprisingly large fraction of children have frequent diarrhea and/or three or more ear infections in a year, indicating a high burden of infectious disease.

The contrast between the relatively small numbers of school days missed, and the relatively large numbers of illnesses further suggests that many children go to school sick. This observation reinforces the potential role of schools in improving public health by monitoring and even treating minor childhood illnesses.

While Table 2 may appear to cover an exhaustive list of possible health shocks, Currie and Lin (2007) find that controlling for this lengthy list reduces but does not eliminate the effect of income on health status. One might suspect that this was due to differences in the way that rich and poor parents perceive the effects of illness. However, Currie and Lin report that the effect of a particular condition on health status is generally the same for rich and poor children. This suggests that the differences in health status are not merely a reflection of reporting biases.

An alternative hypothesis is that poor children are particularly burdened by conditions such as acute illnesses, which are not well covered by the NHIS. That is, the fundamental reason that income matters for overall health status may be that poor children suffer more insults to their

health than non-poor children, but it is difficult to tell because we do not observe many of these insults in the NHIS.

For example, hospitalizations for “ambulatory care sensitive conditions” (hospitalizations that could be prevented by appropriate outpatient care) are higher for poor children than for richer ones (see Begley et al., 1994; Billings et al., 1993). Casanova and Starfield (1995) and Gadoński et al. (1998) develop lists of pediatric hospitalizations for avoidable conditions. Between 1983 and 1996, Dafny and Gruber (2005) calculate that 26 percent of pediatric hospitalizations were avoidable by this definition. The most common causes of such hospitalizations were pneumonia; gastroenteritis; ear, nose and throat infections, dehydration, and kidney/urinary tract infections. They also show that such hospitalizations declined with expansions in the insurance coverage to low income children. But Currie and Stabile (2003) show that in Canada, with universal health insurance, poor children are still more likely to be hospitalized than richer ones.

As a second example, although injuries rather than illnesses are the leading cause of death among children in developed countries (Bonnie et al., 1999), NHIS data on the burden of injury is inadequate. The NHIS asks only about injuries that “required” medical attention, and reports of such injuries go up with income, which is clearly at odds with the available data on child accidental death rates. In the U.K., 6 children out of every 100,000 died annually due to injuries from 1991 to 1995. But the death rate due to injuries rose to 20 out of 100,000 among children of manual workers. The injury rate fell over a ten year period, but it fell more quickly among children of high-status fathers than among children of manual workers, so that the gap between the two groups actually rose over time (Unicef, 2000).

In the United States, deaths associated with accidental deaths (injuries associated with auto accidents and other accidents) among children 0 to 19 have declined very rapidly, falling by 47 percent between 1980 and 1998 (Currie and Hotz, 2004). This decline represents a remarkable public health triumph which is not well understood. It has been argued that improvements in the safety of products that families use and/or to which children are exposed has played an important role in this decline. For example, cars have become much safer over the past 30 years, and innovations such as childproof caps on medicines, fall bars on windows, and fencing around swimming pools are credited with saving many lives. Many safety advocates argue that these improvements are the result of increased consumer safety regulation and that the declines in accidental deaths to children demonstrate their effectiveness.

Glied (2001) discusses many important regulations affecting product safety, including the the Poison Prevention Packaging Act of 1970, and laws mandating the use of infant and child safety seats in cars, which were first introduced in 1977 and had been adopted by all states by 1984. She argues, however, that these regulations cannot explain declining trends in accident rates. It is certainly clear that the measures she discusses are unlikely to account for declining death rates in recent years.

It is extremely difficult to directly test the hypothesis that regulation is responsible for declines in accident rates, given the myriad regulations at federal, state, and even local levels. Glied's test is based on the argument that younger children should benefit most from new product regulation, or conversely, that children with older siblings may be more at risk from dangerous older products. However, the Vital Statistics mortality data does not identify whether or not children have siblings, so her test cannot be directly implemented.

In addition to regulation, efforts by epidemiologists, public health agencies and safety advocacy groups have increased the supply of information about the possible hazards confronting children in everyday life, and about strategies for preventing illness and accidents. These developments are disseminated to the public through public service announcements, product labeling (which is generally regulated, so that there is an overlap between regulation and information dissemination), school safety education programs, and parenting books. These efforts may account for some of the decline in accidental deaths to children.

One might expect improvements in products, regulations, and information to benefit everyone eventually. However, groups that are better educated may be better informed, and groups that are richer may have access to superior products sooner. Thus, it is possible that rapid improvements in products and information could benefit white children sooner than black children leading to temporary expansions of gaps in black/white death rates.

Although in the U.S. discussions of injury focus on deaths, because the death data is available, it is important to keep in mind that deaths are only the tip of the iceberg (and not relevant when we consider the possible effects of injury on educational attainment, except in so far as deaths change the distribution of children). In the Netherlands, for each injury-related death among children 0 to 14, there are 160 hospital admissions and 2,000 emergency room visits (Unicef, 2000). Hence, injuries are an important burden on children, but it is impossible to assess the size of the burden given existing data.

2. Do Correlations Imply Causality?

The literature surveyed above convincingly demonstrates that there are gaps in health between rich and poor children. But it is one thing to document gaps in health, and another to

demonstrate that these gaps have causal effects on educational attainment or other child outcomes. Some descriptive research has shown that poor birth outcomes are related to future outcomes. For example Linnet et al. (2006) use Danish registry data to show that children who were premature, or low birthweight and/or whose mothers smoked in pregnancy, had a much higher risk of ADHD. Currie and Hyson (1999) show that low birth weight children from the 1958 British birth cohort have lower test scores, educational attainments, wages, and probabilities of being employed as of age 33, even conditional on many measures of family background and circumstances. Case, Fertig, and Paxson (2005) extend this research by showing that the same is true at age 44, and for adults who suffered chronic conditions as children.

But it is possible that these correlations are due to other characteristics of households that are associated both with poor child health and poorer outcomes. Grossman and Kaestner (1997) summarized the evidence linking child health to future educational attainment up to that point and concluded that while most studies had methodological weaknesses, it seemed more likely than not that there was a connection. The most recent research on this question seeks to go further by focusing on the causal question and asking not only whether there is a causal link, but why? These are obviously the key questions if one seeks to intervene to improve outcomes by improving health.

The next three sections examine the evidence regarding whether there are causal effects of child health on future outcomes. Section 2 a) focuses on a study using a general measure of health status. Section 3 covers studies that use birth weight as their health measure. Section 4 examines other specific health measures.

2. a) Causal Evidence from General Health Measures

Smith (2006) investigates the relationship between child health and future outcomes using data from the 1999 Panel Study of Income Dynamics. The adult children of Panel Study of Income Dynamics (PSID) respondents (who were then 25 to 47 years old) were asked a retrospective question about the state of their health when they were less than or equal to 16 years old: Whether it was excellent, very good, good, fair or poor? In ordinary least squares regressions, the answer to this question is highly correlated with 1999 outcomes. But Smith also exploits the structure of the PSID to estimate models that control for sibling fixed effects.

As shown in Table 3, in the fixed effects models, better health in childhood is related to higher incomes, higher wealth, more weeks worked, and a higher growth rate in income. The estimates imply that within families, a sibling who enjoyed excellent or very good health in childhood earns 24 percent more than a sibling who was not in good health. Strikingly, the effects are generally larger in the fixed effects models than in ordinary Least Squares (OLS) models.

The sole exception is for educational attainment where the OLS estimates show a statistically significant effect, while the fixed effects model does not. It is odd to find effects on all of the other measures while there are none for education (given the close link between education and income), but it is possible that there is less variation in education between siblings than in the other measures. Alternatively, perhaps health in childhood affects outcomes such as adult income through mechanisms other than educational attainment, such as the ability to work harder. Note that if health affects future wages through a mechanism other than education, then this has the interesting implication that better health could cause education to fall rather than rise (by increasing the opportunity cost of education).

While retrospective self-reports about health are obviously imperfect measures, Smith provides some assurance that the answers to such questions are not biased by subsequent health shocks, using data on a similar question from the Health and Retirement Survey. He asks whether HRS respondents who suffered a negative health shock between 1998 and 2004 were more likely to downgrade their self-reported childhood health status than other respondents, but finds no evidence of such a “coloring” effect. The HRS also provides some evidence regarding the relationship between specific diseases that were suffered in childhood, and the respondent’s overall retrospective rating of their health status in childhood. These data show sensible patterns: Adults who had more severe ailments in childhood are more likely to report that they suffered poor health status in childhood.

3. Causal Inference from Studies of Birth Weight

In contrast to the Smith study which uses a broad, but problematic measure of health, several recent studies examine long term effects of low birth weight on future outcomes. Low birth weight has long been used as the leading indicator of poor health among newborns for many years. Its advantages are that it is widely available over long periods of time, and is thought to be fairly accurately measured. Moreover, it is strongly linked to outcomes such as mortality: In 1996, the infant mortality rate for babies over 2,500 grams was 2.77 compared to 17.45 for babies between 1,500 and 2,500 grams, and 259.35 for babies less than 1,500 grams (Conley and Bennett, 2001). Some follow ups indicate that low birth weight babies also have lower scores on a variety of tests of intellectual and social development (Breslau et al. 1994, Brooks-Gunn, Klebanov, and Duncan, 1996), but most of these studies cannot distinguish between the effects of

low birth weight and those of factors that contribute to the incidence of low birth weight. The studies reviewed below seek to identify causal effects.

Because there are many studies of birth weight, they are divided into three groups below: those that use sibling/twin designs, those that develop an instrumental variables strategy to identify the effects of exogenous changes in education or income, and those that examine changes affecting entire cohorts of infants.

3 a) Causal Inference from Sibling/Twin Designs Examining Effects of Low Birth Weight

Behrman and Rosenzweig (2004) use data from the Minnesota Twins Registry to compare higher birth weight infants to their own twins of lower birth weight. They find that the higher birth weight twin is not only taller, but also goes on to get more schooling. They estimate that increasing birth weight by a pound increases schooling by a third of a year. However, their sample is quite small, and they do not investigate the question of how SES interacts with low birth weight in the production of child outcomes. Using data from the Panel Study of Income Dynamics (PSID), Conley and Bennett (2000) find that low birth weight reduces the probability of high school graduation in models that include mother fixed effects. Conley, Strully, and Bennett (2003) argue that low birth weight babies in low income families are at particularly high risk of poor outcomes, though again, their sample is quite small.

Recent studies conducted using large samples drawn from vital statistics records in Norway, Canada, the U.S. all show a link between low birth weight and lower educational attainment, even among twins. Black, Devereux, and Salvanes (2005) examine a large sample of Norwegian twins and find, using twin fixed effects models, that a 10 percent increase in birth weight leads to a one percentage point increase in the probability of graduating from high school

and a one percent increase in earnings. Moreover, these effects are surprisingly linear between about 1,500 grams and 3,500 grams, suggesting that an exclusive focus on the 2,500 gram cutoff for low birth weight is unwarranted. Oreopolous, Stabile, Wald and Roos (2006) use similar data from the Canadian province of Manitoba. They find that children in the 1,500 to 2,500 gram range are 8 percent less likely to reach grade 12 by age 17 than siblings who weighed over 3,500 grams.

It is difficult to conduct this exercise for the U.S. because of the paucity of data linking health at birth to future outcomes. Almond, Chay and Lee (2004) use linked birth certificate and infant death certificate data to examine the effect of low birth weight on the probability of infant death in a sample of U.S. twins. They find that the effect of low birth weight is much smaller than ordinary least squares estimates would suggest, though it is still large and significant.

Royer (2005) uses birth certificate data for California to examine longer term outcomes. From the birth certificate, we know the mother's education at the time of the birth. If the mother was born in California, we can go back in time and locate her birth certificate to find out her birth weight. Royer examines mothers who were twins born over the 1960 to 1982 period, and finds that each 1,000 gram increase in birth weight is associated with a gain of .16 years of education. There are of course, issues involved with using a selected sample of mothers but Royer addresses these and argues that they are unlikely to skew her results. This is a fairly small effect, but then low birth weight is only one, imperfect, indicator of poor health. It is remarkable that circumstances prior to ones birth should have effects many years later.

Currie and Moretti (2007) use data similar to Royer to examine interactions of low birth weight with maternal SES, as proxied by income in the grandmother's hospital of delivery at the

time the mother was born, or by income in the zip code of residence when the mother's own child is born. They find that low birth weight has significant effects on later socio-economic achievement. In particular, when they compare mothers who are sisters by conditioning on grandmother fixed effects, they find that low birth weight is associated with a four percent higher probability of living in a poor area at the time of the delivery of one's own child, and with the loss of about a tenth of a year of education.

Rucker and Schoeni (2007) examine the long-term effects of low birth weight using data from the PSID and sibling fixed effects models. They find that low birth weight is strongly related to poorer adult health and lower adult annual earnings (by 17.5 percent). Siblings who are low birth weight are less likely to have earnings (by 4.8 percentage points). A relatively small part of this reduction in earnings is mediated by lower educational attainment—low birth weight siblings are 4.8 percentage points more likely to drop out of school, and completed education is a tenth of a year lower, an estimate that is remarkably similar to those of Royer and Currie and Moretti (2007). But controlling for education has little effect on the estimated effect of low birth weight in their earnings models. A larger fraction of any effects on earnings may be accounted for by effects on cognition. Rucker and Schoeni find that measures such as passage comprehension and reading ability as well as math achievement are strongly affected by low birth weight. For example, passage comprehension is reduced by about 12 percent of the average test score at 3.3 pounds.

A possible threat to identification in these studies that rely on twin or sibling differences, is that parents may differentially invest in one sibling or the other (Becker, 1991). If parents invest to compensate disadvantaged children, then sibling fixed effects will tend to understate the

true birth weight effect, while if they favor the stronger child, effects will tend to be over-estimated. Rosenzweig and Zhang (2006) argue that in China, parents favor the stronger child. This might be because many Chinese still expect to be supported by their children in old age. In the U.S., on the other hand, much of the available evidence suggests that investments are often compensatory (Behrman, Pollak, and Taubman, 1982, 1989; Ashenfelter and Rouse, 1998; Ermish and Francesconi, 2000, McGarry 1999; McGarry and Schoeni 1995, 1997) so that the sibling comparisons discussed above are likely to yield underestimates of the true effects of birth weight. It is however, remarkable that significant effects are found across so many different time periods and countries.

The studies discussed above suggest that low birth weight is a marker for future outcomes. One can also ask a related question: Are improvements in parental education and income associated with higher birth weights? A handful of studies look at intergenerational correlations in birth weight and ask whether the mother's socioeconomic status at the time of the child's birth can explain the observed correlations. Conley and Bennett (2000) use the PSID and examine 1,654 singleton births to sample mothers between 1986 and 1992. They find that income during pregnancy has no effect on the risk of low birth weight when the mother's birth weight is controlled, or when family fixed effects are included in the model. However, Conley and Bennett (2001) also estimate models with mother fixed effects and find that if the mother was low birth weight, then income at the time of the birth has a significant impact on the probability that the child is low birth weight. Hence, they suggest that there is an interaction between poverty at the time of the child's birth and maternal low birth weight in the production of child low birth weight. But their results cannot be regarded as definitive given the very small sample sizes in the

PSID. The models for children of low birth weight parents include only 179 children, and only a subset of these would have been born to mothers who experienced a sizeable change in income between births.

Using the linked data from California birth certificates that was described above, Currie and Moretti (2007) find that the intergenerational transmission of low birth weight is stronger for mothers who live in high poverty zip codes. Low SES also has an independent effect on the probability of low birth weight, increasing it about six percent relative to the baseline. Low SES is defined using the median income in the mother's place and time of birth, hence, these results indicate that mothers who grew up in poverty are more likely to give birth to low birth weight children, other things being equal. Similarly, Rucker and Schoeni (2007) show in their sibling fixed effects models that increases in income increase birth weight by much more if the mother was low birth weight herself.

3. b) Instrumental Variables Strategies

These findings are complementary to Currie and Moretti (2003), who find that increases in college going among American women, which were themselves driven by increases in the local availability of colleges, improve infant health outcomes. They show that the opening of a new college in the woman's county in her 17th year is a significant predictor of her educational attainment. In turn, instrumental variables models show that higher rates of college going improve infant health, as measured by birth weight and gestational age. It also increases the probability that a new mother is married, reduces parity, increases use of prenatal care, and substantially reduces smoking, suggesting that these may be important pathways for the ultimate effect on health.

On the other hand, McCrary and Royer use the law governing school entry ages in Texas as an instrument for maternal education, and do not find an effect. Given a particular age cutoff, children a few days younger than the cutoff must wait until the next year to start school. These children will then be in a lower grade when they reach minimum school leaving age, and McCrary and Royer show that they tend to get less education on average. A potential criticism of their identification strategy is that being consistently the oldest child in ones class may have effects independent of the impact on school leaving.

Black, Devereux, and Salvanes (2005) find that while changes in compulsory schooling laws in Norway increased educational attainments among people at the low end of the educational distribution, there did not seem to be an overall intergenerational effect on the education of the affected cohort's children (they do not ask whether there was an intermediate effect on health). A nice feature of their paper is that the reform of the education system was implemented at different times in different localities so that it can be distinguished from any underlying secular trend. They do find however, a significant effect of maternal education on the education of sons.

3. c) Causal Inference from Cohort Studies of Birth Weight

Several studies examine the impact of conditions at the time of birth on subsequent health. Much of this research has been inspired by the Barker “fetal origins” hypothesis (see Barker, 1998) that conditions in utero “program” the developing fetus in ways that will affect future health. For example, fetuses that are starved in utero may develop more efficient metabolisms, which then place them at higher risk for future obesity, heart disease, and diabetes. Evidence from the “Dutch Hunger Winter” of Nov. 1944 to April 1945 suggests that adults who were in

utero during the time of the famine are more likely than the surrounding cohorts to suffer various health impairments including disorders of the central nervous system, heart disease, and antisocial personality disorders (Stein et al. 1975; Roseboom et al. 2000; Neugebauer, Hoek, and Susser, 1999).

Maternal disease may play a similar role. Almond and Mazumder (2005) use data from the U.S. Survey of Income and Program Participation to follow cohorts who were affected by the influenza epidemic of 1918. The epidemic struck suddenly in the fall of 1918 and was largely over by January 1919. It is estimated that approximately a third of women of child bearing age were infected. They show that compared to cohorts in utero either just before or just after the epidemic, the affected cohorts were more likely to suffer from schizophrenia, diabetes, and stroke, as adults. Doblhammer (2004) summarizes evidence that health shocks in early life due to wars, famines, and other crises can have lasting effects on health.

Almond (2006) examines the direct effects of the influenza epidemic on education and labor market outcomes. He finds that children of infected mothers were 15 percent less likely to graduate from high school, and that the wages of affected men were lowered by 5 to 9 percent. Moreover, affected individuals were more likely to be poor and to be receiving transfer payments (in part because they were more likely to be too disabled to work). Thus, this natural experiment provides compelling evidence that negative shocks to health in utero can have very significant effects on future economic outcomes.

Almond and Chay (2003) extend this line of research by asking whether women whose health was better as infants go on to deliver healthier infants as adults. They build on previous work showing that the Civil Rights movement had a large effect on the health of black infants in

certain southern states, especially Mississippi, due to increased access to medical care (Almond, Chay, and Greenstone, 2003). Because birth records include the mother's state of birth, it is possible to identify black women who benefited from these changes (the 1967 to 1969 cohorts), and to compare the outcomes of their infants to infants born to black women in the 1961-1963 birth cohorts. The birth outcomes of white women in the same cohorts are examined as a control. They conclude that the infants of black women who had healthier infancies as a result of the Civil Rights movement show large gains relative to the infants of black women born just a few years earlier, and that these gains are largest for women from Mississippi – the most affected state. The estimates indicate that the black-white gap in the incidence of very low birth weight was 40 percent lower in the 1967-1969 cohort than in the earlier cohort.

This study differs from some of the previous ones in being less focused on health in utero. It is possible that some changes that benefited the 1967-1969 cohort affected the mother's health in utero. But other factors, such as desegregation of the hospitals, likely affected health at the time of the birth and afterwards. Almond, Chay, and Greenstone (2003) show, for example, that there was a large decline in the incidence of infant deaths due to infectious disease and diarrhea in these cohorts. Thus, the study provides some suggestive evidence that conditions after birth matter too.

Costa (1998) shows that infants born in New York in the early part of the 20th century were of similar birth weight to modern infants, but that early infant feeding practices resulted in lower weights by the time they were 10 days old. These infants in turn had higher mortality rates and lower adult height. Again, this study suggests that conditions after birth, but early in life may set the pattern for future outcomes.

Van den Berg, Lindeboom, and Portrait (2005) tackle a related question and ask whether economic conditions in utero impact the health of affected cohorts. They use an extraordinary historical data set from the Netherlands that covers about 14,000 randomly chosen people born in the Netherlands between 1812 and 1912, and followed up to 2000. The data draws together all of the administrative records of vital events (births, marriages, and deaths) for these people. They compare people who were in utero during a recession to those who were in utero just before the recession. If economic conditions show a secular trend towards improvement, then this design will tend to under-estimate the effect of the recession. They use GNP to measure economic conditions, and exclude time intervals that included epidemics, in order to focus on the effects of fluctuations in economic activity. They find that those born in recessions suffer up to 7 percent higher mortality rates after the first year of life compared to those born just prior to the recession. It is interesting to speculate on whether this difference reflects the effects of absolute or relative deprivation, but that question is not addressed. Relative deprivation might have an effect through maternal stress responses, for example.

Maccini and Yang (2006) examine the long-term effects of variations in rainfall in the year and location of birth on the adult outcomes of Indonesian's using data from the Indonesia Family Life Survey. They find that a 20 percent increase in rainfall at the time/place of birth increases women's educational attainment by .15 years, and their height by .14 centimeters. They also live in households with 5 percent higher per capita expenditures, and marry higher earning spouses.

All of these studies suffer potential biases from "fetal selection." The problem is that only surviving fetuses are recorded in most of these data sets. Hence, shocks which tended to cull

weak fetuses might lead the population of surviving infants to be stronger than it would have been otherwise. It is notable then, that there is such robust evidence of negative effects of health shocks in utero and in infancy – the fetal selection argument suggests that these estimates understate the true negative effects. Dehejia and Lleras-Muney (2004) provide some evidence that in the U.S. black women are less likely to give birth during recessions, which tends to raise mean birth weights. This might be due either to fetal selection, or to fewer planned pregnancies.

This research builds on a previous body of evidence showing that people who suffered infectious diseases in their youth, were more likely to develop chronic conditions in old age, and to die at younger ages. For example, Costa (2000) shows that in a sample of Union Army Veterans, those who enlisted in areas with high infant mortality rates, or who had serious infectious diseases while they were in the service, were more likely to have chronic respiratory conditions, heart problems, and joint and back problems at older ages. However, it is difficult to say from data of this kind whether the relationship is causal—it is possible that physically weaker people are more likely to be sick at both younger and older ages.

4. Evidence From Studies of Other Specific Health Measures

For birth weight then, there is evidence both that parental SES affects child health, and that child health affects future child outcomes. There are fewer studies examining other specific health measures, and most of these focus on the effects of particular health conditions on longer term children's outcomes. This section is organized by drawing an example from each of several specific health domains: Nutrition, mental health, chronic physical conditions, acute conditions, and toxic exposures.

4. a) Poor Nutrition

Randomized trials in developing countries indicate that poor nutrition can harm cognitive development. In one of the more famous studies, Pollitt et al. (1993) report on a randomized trial of a nutritional supplementation program in Guatemala that had large impacts on the test scores and schooling attainment of treated children. Grantham-McGregor (1991) reports on a similar successful randomized trial of nutritional supplementation combined with psychosocial stimulation among Jamaican children who were developmentally delayed.

It is less obvious that nutritional supplementation is likely to have a large effect on the cognitive achievement of children in richer countries. Several studies have found, however, that participation in the U.S. WIC (Supplemental Nutrition for Women, Infants, and Children) program prenatally is associated with higher test scores. WIC is a program that provides coupons that can be redeemed for specific foods to women, infants, and children who are deemed to be “nutritionally at risk”. Rush et al. (1988) examine a large sample of WIC participants and a control group of pregnant women who were also receiving prenatal care in clinic settings. When children were followed up at age 4 and 5, the WIC children had better outcomes on cognitive tests, even though the control women were on average higher income and better educated than the WIC women.

Kowaleski-Jones and Duncan (2002) use data from the National Longitudinal Survey of Youth and sibling fixed effects models to examine the effect of prenatal participation in WIC. They find some evidence of positive effects on temperament, though not on motor or social skills. These findings suggest that better nutrition could improve cognitive performance even in a relatively well-nourished (or at least non-deprived) population.

The pioneering work of Fogel and others has established that height is a good measure of the average health of populations, and that there is a robust relationship between height and economic well-being (c.f . Fogel, 1994; Floud, Wachter, and Gregory, 1990). Case and Paxson (2006) use data from the British cohort studies to show that the well-established relationship between adult height and earnings (as well as high-status occupations) disappears when controls for cognitive test scores in early child hood are added to a regression model. Since much of the variation in adult height is due to nutrition in childhood (particularly during the critical 0 to 3 phase), they argue that poor nutrition in childhood likely affects both cognitive performance and adult height, leading to the observed correlation between height and earnings, even in countries where physical strength is no longer closely related to earnings potential.

Today, however, we hear more about the rising epidemic of obesity, even among young children, than about nutritional deficiencies. Hedley et al. estimate that of children 6 to 19, 31 percent are overweight or at risk of overweight, while 16 percent are overweight. The poor are at higher risk of obesity than the rich, hence the growth in obesity will likely exacerbate existing differences in health between rich and poor since many diseases such as heart disease and diabetes, are related to obesity (Cutler, Glaeser and Shapiro, 2003). Moreover, those who are obese are often still badly nourished. Using data from the 3rd National Health and Nutrition Examination Survey, which included blood serum measures of nutritional status, Bhattacharya and Currie (2001) show that 10 percent of U.S. adolescents 12 to 16 were anemic and 10 percent were short of vitamins A, C, or E. Family income below 1.3 times the U.S. poverty line was a significant predictor of high blood cholesterol and high body mass index even conditional on other demographic variables.

4. b) Mental Health

The prevalence and importance of child mental health problems have been increasingly recognized. The MECA Study (Methodology for Epidemiology of Mental Disorders in Children and Adolescents) cited in the 1999 U.S. Surgeon General's Report on Mental Health finds that approximately one in five children and adolescents in the U.S. exhibit some impairment from a mental or behavioral disorders, 11 percent have significant functional impairments, and 5 percent suffer extreme functional impairment. (Shaffer et al., 1996; U.S. DHHS, 1999). These are very large numbers of children. Moreover, as Currie and Madrian (1999) discuss, mental health problems are one of the leading causes of days lost in the work place, because they strike many people of working age.

It is surprising then that there is relatively little longitudinal research documenting the long-term effects of children's mental health problems. A few studies look at the longer term consequences of behavior problems in relatively large samples. Kessler et al. (1995) uses data from the U.S. National Comorbidity Study which surveyed 8,098 respondents 15 to 54 years old from 1990 to 1992 and assessed their current psychiatric health as well as collecting information about past diagnoses of mental problems. Using retrospective questions about onset, they find that those with early onset psychiatric problems were less likely to have graduated from high school or attended college.

Farmer (1993, 1995) uses data from the 1958 British Birth Cohort Study to examine the consequences of childhood "externalizing" behavioral problems on men's outcomes at age 23. She finds that children who fell into the top decile of an aggregate behavior problems score at ages 7, 11, or 16 had lower educational attainment, earnings and probabilities of employment at

age 23. Gregg and Machin (1998) also use data from the 1958 cohort and find that behavioral problems at age 7 are related to poorer educational attainment at age 16, which in turn is associated with poor labor market outcomes at ages 23 and 33.

A similar study of a cohort of all New Zealand children born between 1971 and 1973 in Dunedin found that those with behavior problems at age 7 to 9 were more likely to be unemployed at age 15 to 21 (Caspi et al., 1998). Miech et al. (1999) examine adolescents from this cohort who met diagnostic criteria for four types of disorders (anxiety, depression, hyperactivity, and conduct disorders) when they were evaluated at age 15, and who were followed up to age 20. They find that youths with hyperactivity and conduct disorders obtained significantly less schooling, while anxiety and depression had little effect on schooling levels.

More recently, McLeod and Kaiser (2004) use data from the National Longitudinal Survey of Youth (NLSY) to show that children who had behavior problems at ages 6 to 8 are less likely to graduate from high school or to attend college, even after conditioning on maternal characteristics. Like Miech et al. they find that in models that included both “internalizing” and “externalizing” behavior problems, only the latter were significant predictors of future outcomes. One limitation of this study is that it focuses on a relatively small number of children who, given the design of the NLSY, were born primarily to young mothers.

Several studies focus on particular “externalizing” mental health conditions. Mannuzza and Klein (2000) review three studies of the long-term outcomes of children with ADHD. In one study, children diagnosed with ADHD were matched to controls from the same school who had never exhibited any behavior problems and had never failed a grade; in a second study, controls were recruited at the 9-year follow up from non-psychiatric patients in the same medical center

who had never had behavior problems; and in a third study, ADHD children sampled from a range of San Francisco schools were compared to non-ADHD children from the same group of schools.

These comparisons consistently show that the ADHD children had worse outcomes in adolescence and young adulthood than control children. For example, they had completed less schooling and were more likely to have continuing mental health problems. However, by excluding children with any behavior problems from the control groups, the studies might overstate the effects of ADHD. Also, the studies do not address the possibility that the negative outcomes might be caused by other factors related to a diagnosis of ADHD, such as poverty, the presence of other learning disabilities, or the fact that many people diagnosed with ADHD end up in special education.

Currie and Stabile (2006) address these problems by examining the effects of ADHD in sibling fixed effects models using data from the NLSY and the (similar) Canadian NLSCY. They focus on children who were 4 to 11 years old in 1994 and show that as of the end of the sample period, children with high scores on an ADHD screener had lower cognitive test scores than other children for math and reading (by approximately 1/3 of a standard deviation), and much higher probabilities of being in special education or having repeated a grade.

Currie and Stabile (2007) extend these results and show that ADHD appears to have larger effects on academic outcomes than childhood depression, conduct disorders, or other mental problems. Moreover, the effects are quite similar in the U.S. and in Canada, lending support to the idea that they represent real impairments. The estimates are also robust to controlling for other learning disabilities, and it appears that poor mental health at the beginning of the sample

period has an effect independent of the fact that it predicts poorer mental health as of the end of the sample period. Finally, the effects of ADHD are large relative to those of physical chronic conditions.

Perhaps the most widely known studies of the long-term effects of aggression or conduct disorders are associated with Richard Tremblay who tracked a group of 1037 boys from Kindergarten to age 15 in Montreal, Canada. He found that boys who were highly aggressive in Kindergarten were much more likely to be persistently aggressive, and that this was most true of children of young or less educated mothers (c.f. Nagin and Tremblay, 1999). Campbell et al. (2006) use data from the NICHD Study of Early Child Care and Youth Development to track children from 24 months to 12 years of age, and find that children who persist in moderate or high levels of physical aggression past Kindergarten have higher levels of externalizing problems as pre-teens.

A third strand of related research examines the importance of “non-cognitive skills”. For example, Blanden, Gregg, and Macmillan (2006) ask whether rising returns to non-cognitive skills can explain growing income inequality. In their analysis of the 1958 and 1970 British birth cohort data sets, they include characteristics such as “hyper” and “anxious” as well as measures such as “self esteem” and “extrovert” as measures of non-cognitive skills and find that rising returns to positive mental characteristics does indeed account for some of the increase in inequality between the two cohorts.

However, Heckman, Stixrud, and Urzua (2006) conceptualize non-cognitive skills as innate traits (similar to native ability) and measure them using the Rotter Locus of Control Scale and the Rosenberg Self Esteem Scale. They conclude that such non-cognitive skills are important

determinants of academic and economic success. It seems clear that these measures of non-cognitive skills are likely to capture some aspects of mental health as well as innate character traits.

In addition to the evidence showing negative effects of childhood mental health conditions on future outcomes, there is at least some evidence that changes in family circumstances can affect childhood mental health conditions. In particular, the Moving to Opportunity experiment, which randomly assigned some public housing residents to receive assistance to move to low poverty neighborhoods, found improvements in the mental health of girls. Specifically, the experimental group experienced reductions in generalized anxiety disorders and psychological distress. Curiously, there was no such positive effect for boys (Orr et al., 2003).

Berger, Paxon, and Waldfogel (2006) explore the relationship between family income, home environments, and child mental health outcomes (and cognitive test scores) at age three in the “Fragile Families and Child Wellbeing Study”. This study is following a cohort of 5,000 children born in several large U.S. cities between 1998 and 2000, and over-samples births to unmarried couples. Presumably we think income matters because it affects something about the home environment. Berger et al. show that all of the measures they examine (which include measures of parenting skills as well as physical aspects of the home) are highly related to income. Moreover, controlling for these measures reduces the effects of income on outcomes considerably. However, they present simulations based on their estimates (estimates which are likely to over-state the effect of income given the likely direction of omitted variables bias) that imply that even cash subsidies that brought every family up to the poverty line would not eliminate gaps in child outcomes.

Dahl and Lochner (2005) provide the most recent and compelling evidence that cash may matter—they use expansions of the Earned Income Tax Credit as instruments for household income and find that each thousand dollars of income improves childrens' test scores by 2 to 4 percent of a standard deviation. An attractive feature of the changes in the EITC is that households may well have regarded them as permanent, so this experiment may approximate the effects of changes in permanent, rather than transitory income. Their result implies however, that it would take on the order of a \$10,000 transfer to having an educationally meaningful effect on test scores, so that the result is not inconsistent with that of Berger et al.

4. c) Chronic Physical Conditions: Asthma

We saw above that asthma was the most prevalent chronic condition. The literature regarding longer term consequences of asthma illustrates some of the many problems involved in making links between childhood conditions and future outcomes. Asthma is more common among the poor than among the non-poor, and the poor are much more likely to be limited by their asthma. Still, the evidence linking asthma to child outcomes is inconclusive. Part of the problem is that if the asthma is well controlled, then it should have little impact on the child. Yet many of the studies examining the effect of asthma on cognitive outcomes or schooling attainment compare children whose asthma is well controlled to those without asthma. For example, Lindgren et al. (1992) compare siblings without asthma to siblings with asthma who were receiving daily maintenance medication, and find no differences in achievement test scores. The purpose of these studies was generally to see if the medication children took to control their asthma affected their cognitive functioning.

Several studies do indicate that children with asthma are more likely than other children to have behavior problems, even when the asthma is well controlled. For example, Annett et al. (2000) found that asthmatic children scored between 2/3 to one standard deviation below the norm on a test of impulse control. Calam et al. (2003) conducted a prospective birth cohort study of 663 children and found that children who had three or more attacks of wheezing by age three had more behavior problems. Butz et al. (1995) studied 392 inner-city (predominantly African-American) children with asthma and found that those with a high level of symptoms were twice as likely to experience behavior problems as those with low levels of symptoms. Bussing et al. (1995) use data from the 1988 NHIS, and find that after controlling for confounders, asthma doubled the odds of having behavior problems. These changes in behavior may reflect relatively subtle effects of childhood illness on parenting and family functioning.

One large population based study does find an effect of asthma on school absences, the probability of having learning disabilities, and grade repetition. Fowler et al. (1992) uses data from the 1988 NHIS for children from grades one to twelve, and find that the asthmatic children averaged 7.6 days absent compared to 2.5 days for well children. Nine percent of the asthmatic children had learning disabilities, compared to 5 percent of the well children, and 18 percent had repeated a grade, compared to 15 percent of the well children.

In the only study to explicitly examine school readiness, Halterman et al. (2001) examine 1058 children entering Kindergarten in urban Rochester and find that asthmatic children had lower scores on a test of school readiness skills, and that their parents were three times more likely to report that they needed extra help with learning. There were no differences, however, on tests of language, motor, and socio-emotional skills. It is interesting to note that the negative

effects of asthma were concentrated among children whose asthma caused activity limitations (suggesting that it was not adequately controlled) and that boys were more likely to be in this group than girls.

A difficulty with all of these studies is that since asthma is more prevalent among poor and minority children than among other children, the apparent connection between asthma and outcomes could reflect omitted third factors. However the fact that several of the studies use very homogeneous groups of children and still find differences in behavior (c.f. Annett et al. (2000), Butz et al., (1995), Halterman et al. (2001)), suggests that asthma probably does have a causal effect at least on behavior problems and perhaps on school readiness if it is not properly controlled.

4. d) Acute Conditions

It is likely that the large gaps in maternally reported overall health reflect more than the presence or absence of specific chronic conditions. Poor children often suffer from multiple health problems and suffer more “minor” acute illnesses such as dental caries, allergies, and ear infections than other children. For example, According to the U.S. Centers for Disease Control (2004), poor children have almost 12 times more restricted activity days because of dental problems than higher-income children, and untreated dental disease can lead to problems eating, speaking, and learning. It is, however, difficult to get estimates of the magnitude of these effects.

As discussed above, allergies are also extremely common, but the reported incidence is higher for richer children than for poorer children, even though poorer children are more likely to suffer from complications due to allergies. This suggests that reporting problems make it difficult to accurately assess the differential burden of allergies.

Ear infections (otitis media) affect most young children at one time or another and are the most common reason children visit a doctor. Like dental caries, they can be extremely painful, though more than 80 percent of infections resolve themselves within three days if untreated. Among children who have had acute otitis media, almost half have persistent effusion after one month, a condition that can cause hearing loss. Researchers estimate that at any given time roughly 5 percent of two- to four-year-old children have hearing loss because of middle ear effusion lasting three months or longer (O'Neill, 2003). And hearing loss can delay language development. Again, though, it is difficult to tell how important these effects might be in explaining disparities in outcomes.

Taken together, these common acute problems represent a significant drain on children's resources and may have a significant impact on their chances of success in life.

4. e) Toxic Exposures

Some scourges of childhood such as lead poisoning, have seen huge improvement with the adoption of public health measures such as banning lead in paint and gasoline. Lead has been proven to decrease IQ by 2 to five points for each 10 to 20 microg/DL above the current standard (Pocock et al., 1994), and the majority of affected children are low income. Indeed, the government tracks lead poisoning by looking for areas with a combination of older housing stock and low income households.

Before the regulation of lead, children were exposed to leaded paints, lead in drinking water (from lead solder in pipes), lead in gasoline, and lead in canned food. Evidence from the NHANES surveys showed that 88.2 percent of children aged one to five had lead levels above 10 microg/dl in 1976 to 1980, 8.6 percent had lead levels above the threshold in 1988-1991, and only

2.2 percent had levels this high in 1999-2000. These figures imply that the number of children with unsafe lead levels declined from 13.5 million to less than one half million over this period (U.S. Centers for Disease Control, 2003).

However, there is a good deal of evidence that low income children are more likely to be exposed to other toxic chemicals, some of which are thought to have effects on cognitive development. For example, Rauth et al. (2006) examine a group of children who were prenatally exposed to pesticide (chlorpyrifos) and find that children in a high exposure group were five times more likely to be developmentally delayed than those in a lower exposure group. This is not an experimental study, but the results suggest that differential exposure to toxic substances might be a significant problem for poor children.

The literature examining “environmental justice,” the question of whether minorities and low income people are disproportionately exposed to pollutants, suffers from several methodological weaknesses that leave room for doubt about the findings. First, many studies examine data at the county level, which may simply be too high a level of aggregation to yield meaningful results. Most counties contain a broad range of people. For example, it is not uncommon to find that counties with facilities that use toxic chemicals (and may emit them) are both more heavily African-American and higher income, a finding which may just reflect the fact that they are more urban. A second, and related problem is that many studies include few controls for factors besides race and income that might be correlated with pollution levels, such as levels of industrial activity.

Third, most of these studies have geographical areas, rather than people, as their units of observation, so that there is no natural control group: Areas with industrial activity are likely to

be quite different than those without such activity. Fourth, many studies focus only on a particular town or area, so that it is difficult to generalize or to see regional patterns.

There is little research on the question of whether exposure to toxic releases at the level that generally occurs in the population has negative health effects. Data on possible human health effects generally comes either from animal studies, or from disastrous releases. Woodruff et al. (1998) run 1990 data from the U.S. Environmental Protection Agency's Toxic Release Inventory through a dispersion model and calculate that 90 percent of Census tracts have concentrations of several chemicals greater than cancer benchmarks. This suggests that American children (and others) may be at risk from toxic releases, but does not establish any direct relationship between releases and health effects.

5. Can Health Account for Differences in Child Outcomes or Intergenerational Effects?

Table 4 summarizes the evidence linking several different domains of child health to outcomes. In order for the health problem to lead to disparities, it must be the case either that the problem is more prevalent among the poor or that the problem has a larger negative effect on the poor, and it must also be the case that the health condition is associated with negative child outcomes. The first row of Table 4 repeats the information regarding effects of ADHD. It is almost twice as prevalent among the poor compared to the non-poor, and does have large negative effects. Still, the overall incidence is low enough that if ADHD were the only health problem that differentially afflicted low income children, it could not by itself explain much of the disparity in outcomes.

To see this, consider a typical standardized test which might have a mean score of 50 and a standard deviation of 15 in the non-ADHD population. If ADHD lowers scores by a third of a standard deviation, and 4 percent of non-poor children have ADHD while 6 percent of poor children have it, then the mean score among the non-poor will be 49.8 ($.96*50 + .04*45$) while the mean score among the poor will be 49.7 ($.94*50 + .06*45$).

The same is true for many of the other specific conditions, while for some of the other large categories, such as injuries and environmental exposures, we do not have accurate evidence regarding the likely long-term effects, or the extent of the disparity in exposures. This suggests that disparities in health in childhood might account for differences in adult outcomes, but that it is too soon to say how large the magnitude may be. We would need to sum over the major conditions and account for the possible non-linear effects of multiple health problems.

As discussed above, several studies have demonstrated that there are intergenerational correlations in health status, and that there may be interactions between parental health status and parental economic status in the production of child health. But thus far, few researchers have attempted to directly assess the extent to which poor child health mediates the effects of parental income on children's income.

Eriksson, Bratsberg and Raaum (2005) use a data set from Denmark that began with 14 year olds in 1968, and followed them up until 2001. In 2001, they had measures of the original cohort's current health conditions and their own earnings. They also had retrospective reports about the parent's health conditions as well as the parental report of income in 1968. They show that children of poor parents are more likely to have serious health problems as adults, and that adding measures of the adult child's chronic conditions (e.g. heart disease, hypertension, cancer

etc.) to a typical Solon model of intergenerational correlations in earnings reduces the estimated transmission of earnings by about a quarter. This finding is provocative, though one might expect the addition of contemporaneous variables to reduce the estimated effect of past ones such as parental income. Perhaps in future, with better data, it will be possible to directly test the idea that parental income affects child income in part because of its effects on child health.

6. Conclusions

This essay surveys literature focusing on two questions: Do parental circumstances affect child health at early ages? And does child health matter for future attainments? The answer to both questions appears to be “yes” though it is too early to tell how important these feedbacks between health and more conventional measures of human capital may be.

Much of this literature is extremely recent, suggesting that this is a new and fruitful area of research in Economics. Economists bring two new things to the table: A good sense of how large effects would have to be to be important (honed through many years of estimating human capital earnings functions), and an emphasis on credible identification of causal effects rather than a focus exclusively on description of correlations. These are valuable additions to a literature that has heretofore been almost exclusively medical and epidemiological.

The research summarized here suggests that child health is important not only for its own sake but because it affects children’s future prospects more broadly, as well as the prospects of their future children. Investments in prevention are likely to have a large payoff in terms of future human capital accumulation, but it is important to learn what types of investments are most effective. Even if we find that health problems associated with low income have large causal

effects on children's outcomes, this does not necessarily imply that a program of cash subsidies to parents is the most effective way to remediate the problem. Currie (2006) argues that while many in-kind programs have effectively attacked the consequences of poverty for children, there has been relatively little evidence that modest cash transfers have large effects. Moreover, equalizing access to health care is not sufficient to eliminate gaps in health. We need to understand more about the reasons why poor children suffer a higher incidence of negative health events, even in utero, so that we can do more to prevent them.

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**Table 1: The Steepening of the Health-Income Gradient with Child Age
A Comparison of the U.S., Canada, and the U.K.
Ordered Probits (1=excellent, 5=poor health)**

Age:	0 to 3	4 to 8	9 to 12	13 to 17 (15)
<u>U.S.: Case, Lubotsky, Paxson, NHIS</u>				
Ln(Income)	-0.183 [.008]	-0.244 [.008]	-0.268 [.008]	-0.323 [.008]
<u>Canada: Currie and Stabile, NLSCY</u>				
Ln(Income)	-0.151 [.026]	0.216 [.019]	-0.259 [.024]	-0.272 [.040]
<u>U.K.: Currie, Shields, and Price, HSE</u>				
Ln(Income)	-0.146 [.040]	-0.212 [.028]	-0.196 [.031]	-0.174 [.034]

Notes: Standard errors in brackets. Regressions control for year effects, family size, sex, mother age at birth, father present, etc.

Table 2: Health of Poor and Non-Poor Children
 NHIS 2001-2005 Sample Children Files, Children 2-17

	Poor	Non-Poor
<i>Maternal Assessment of Child Health</i>		
health is excellent/very good	0.700	0.869
AGE 2~3	0.746	0.901
AGE 4~8	0.725	0.873
AGE 9~12	0.682	0.870
AGE 13~17	0.661	0.853
<i>Health at Birth</i>		
Birth weight (grams)	3221	3348
Birth weight < 2500 grams	0.112	0.078
<i>Ever Chronic Conditions</i>		
Ever told Asthma	0.159	0.131
Ever mental problem ^a	0.119	0.079
Ever told ADHD, 2-17	0.071	0.060
Trouble hearing or seeing	0.076	0.053
Stuttering or stammering-past 12 mo.	0.026	0.012
Ever told heart problems	0.018	0.014
Ever told diabetes	0.002	0.002
Ever told had arthritis	0.002	0.001
Any of the above	0.324	0.265
<i>Activity Limitations</i>		
Limit b/c of chronic conditions	0.114	0.070
AGE 2~3	0.061	0.037
AGE 4~8	0.097	0.062
AGE 9~12	0.139	0.087
AGE 13~17	0.141	0.078
Asthma/resp. prob causes limit	0.019	0.006
Mental problem causes limit ^b	0.062	0.035
ADHD causes limits	0.023	0.014
Hearing/vision causes limit	0.008	0.005
Speech problem causes limit	0.019	0.015
<i>Illness and Medically Attended Injury</i>		
Days missed illness/injury past 12 mo.	4.471	3.531
injured/poisoned requiring med.attention last 3 mo.	0.024	0.031
asthma attack past 12 m	0.073	0.057
ER due to asthma last 12 m	0.032	0.016
resp. allergy last 12 m	0.115	0.135
frequent diarrhea last 12 m	0.018	0.012
3+ ear infection last 12 m	0.072	0.056
# Obs.	7,363	36,858
# Obs. Representing	8,339,503	44,476,130

NOTES:

a. Ever told mental problem includes learning disabilities, developmental delays, mental

b. Mental problem causes limit includes limits due to learning disabilities, developmental

**Table 3: Predicting Adult Education and Earnings Using Child Health
PSID 1999, 25-47 Year Old Children of Original Respondents**

		Weeks			Growth	
Ordinary Least Squares	Education	Worked	HH Income	Wealth	Ln Earnings	HH Income
Health in Childhood	0.353	2.33	0.13	1,847	0.123	0.052
Excellent/Very Good	[4.28]	[3.36]	[5.03]	[2.05]	[3.90]	[1.78]
Ln Parent's Income 1-16	0.779	0.801	0.227	4,283	0.215	0.195
	[10.8]	[1.30]	[9.79]	[5.18]	[7.67]	[7.30]
Sibling Fixed Effects	0.114	4.3	0.24	10,005	0.248	0.245
Health in Childhood	[1.15]	[4.05]	[4.98]	[2.29]	[3.66]	[4.07]
Excellent/Very Good	0.363	4.62	0.011	11,647	-0.023	0.336
Ln Parent's Income 1-16	[1.14]	[1.37]	[.070]	[.83]	[.011]	[1.58]

Source: Smith (2006). Models also control for mother and father education, race/ethnicity, age, age squared. T-statistics in brackets. Income is in \$10,000 units. Growth is measured as the percent change between household incomes at age 25 and 1999.

Table 4: Disease Prevalence and Effects.

	Overall Prevalence	Poor vs. Non-Poor Rate	Effect
ADHD	4.19% boys, 1.77% girls (Cuffe et al., 2004)	6.52 vs. 3.85% (Cuffe et al. 2004)	.26 SD reduction in PIAT Math, .32 SD reduction in PIAT reading in adolescent children (Currie and Stabile, 2007).
Asthma	13% diagnosed 6% attack in past 12 mo. (Bloom, 2003).	15.8 vs.12% (Bloom, 2003) 33.2 vs. 20.8% have limitations (Akinbami et al.)	Doubles odds of behavior problems (Bussing et al., 1995). 7.6 days absent vs. 2.5 for non-asthmatic children, 9% have learning disabilities vs. 5% non-asthmatic, 18% repeated grades vs. 12% non-asthmatic (Fowler et al., 1992). Are effects causal?
Lead Poisoning	2.2% have blood lead above CDC standard in 99/00 (CDC web site).	~60% of children w confirmed high lead levels are Medicaid eligible (Meyer et al.2003)	Increase from 10 to 20 microg/DL reduces IQ scores by 2-5 points (c.f. Pocock et al. 1994).
Other toxic exposures	Unknown	Unknown	Unknown
Obesity	31% at risk/overweight 16% overweight (Hedley et al., 2004)	BMI>85%tile 3.4 pp more likely on a base of 8.9%. (Bhattacharya&Currie)	Higher rates of adult disease, but exact magnitudes controversial. Effects on schooling attainment?
Anemia	9% toddlers iron deficient, 3% anemic (Looker et al. 1997).	Poor children 50% more likely to be deficient (Looker et al. 1997).	Long-term supplementation of anemic children improves cognitive functioning, but no evidence that supplementation of deficient children has effects. Given low rates of anemia, effects on disparities in school readiness may be small.
Injuries	Unintentional=16.5 per 100,000; Intentional=6.5 per 100,000 in 1998 all children 0-19. (Currie and Hotz, 2004)	Poor children 2-5X more likely to die (National Safe Kids Campaign, 1998).	Unknown.