HEALTHY, WEALTHY, AND WISE:
ECONOMIC STATUS, POOR HEALTH IN CHILDHOOD,   AND HUMAN CAPITAL DEVELO

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Capital Development
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ABSTRACT

There are many possible pathways between parental education, income, and health, and between 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 adult education or
income.   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 educational and
labor market outcomes?   I show that there is now strong evidence of both links, suggesting that health
could play a role in the intergenerational transmission of economic status.

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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, beginning with Coleman (1966) point to family background as the number one factor. This raises, however, 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. Given the importance of "health capital" for education and earnings (Grossman, 2000; Case, Fertig, and Paxson, 2005; Currie and Madrian, 1999; Smith, 1999), it is possible that poor health in childhood is an important mechanism for intergenerational transmission of education and economic status.

While it is impossible to provide a conclusive answer to this larger question, this essay addresses two narrower related questions: What is the evidence regarding whether parental socioeconomic status affects child health? And, what is the evidence relating child health to future educational and labor market outcomes? That is, the puzzle at the heart of this review is whether low parental SES has an effect on future educational and labor market outcomes through its effects on child health. 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

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developing countries might impede human capital development.

The first two sections of this essay examine the evidence regarding the two questions posed above. In each section, I discuss theoretical reasons why one might expect an effect, correlational evidence, and studies aimed at determining whether there is a causal relationship. The last section of the paper attempts a summing up, aimed at determining whether we have enough information to say whether poor health is likely to be a major cause of intergenerational persistence in economic status.

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 existing evidence how large the overall effect of health might be or whether it explains a significant amount of intergenerational transmission.   A significant problem is that health is inherently multi-dimensional and difficult to summarize in a single index. Many studies focus on narrow measures (e.g. individual chronic conditions) which generally either have low prevalence, or relatively small effects. Further progress on this problem may await better health measures utilizing more complete data linking health in childhood to eventual attainments.

1. Does Parental Socioeconomic Status Affect Child Health?

a) Why Might Parental SES Affect Child Health?

In the standard model of child health production, parents are assumed to maximize an intertemporal utility function such as:

(1) $\Sigma_{t=1}^{T} E_t(1/1+\sigma)^t U_t + B(A_{T+1})$,

where σ is the discount rate, B is a bequest function, A denotes assets, and U_t is given by:

(2) $U_t = U(Q_t, C_t, L_t; X_t, u_1, \varepsilon_{1t})$,

where Q is the stock of child health, C is consumption of other goods, L is leisure, X is a vector of exogenous taste shifters, u is a vector of permanent individual specific taste shifters, and ε denotes a shock to preferences. Utility is maximized subject to the following set of constraints:

(3) $Q_t = Q(Q_{t-1}, G_t, V_t; Z_t, u_2, \varepsilon_{2t})$,

(4) $C_t = Y_t - P_{gt}G_t - (A_{t+1} - A_t)$,

(5) $Y_t = I_t + w_tH_t + rA_t$,

(6) $L_t + V_t + H_t = 1$,

where G and V are material and time inputs into health production, Z is a vector of exogenous productivity shifters, u_2 are permanent individual specific productivity shifters, ε_{2t} is a productivity shock, Y is total income, P represents prices, I is unearned income, w is the wage, r is the interest rate, and endowments of health and assets, Q_0 and A_0 are assumed to be given. Equation (3) can be interpreted as a "production function" for child health which describes the way that inputs are can be converted into health (see Grossman, 2000).

Health inputs are valued by consumers not for their own sake, but because they affect child health, which in turn has a direct effect on parental utility. Non-market time is an input into both health production and the production of other valued non-market goods (i.e. leisure activities). This model is dynamic in the sense that the stock of child health today depends on past investments in health, and on the rate of depreciation of health capital (which is one of the elements of u_2).

The model can be solved to yield Frisch demand functions for C_t, H_t, G_t, and V_t of the following form:

(7) C_t, H_t, G_t, and $V_t = F(\lambda_t, X_t, Z_t, w_t, P_t, M_t, r, \sigma, u_1, u_2, \varepsilon_{1t}, \varepsilon_{2t})$,

where λ_t is the marginal utility of wealth and M_t is a vector of moments of the distribution of $\{X_k,$

$Z_k, w_k, P_k, \varepsilon_{1k}, \varepsilon_{2k}\}$, and k=t+1,..., T.

Substituting the solutions for G and V into (3) yields a Frisch demand function for Q_t that is

conditional on Q_{t-1}:

(8) $Q_t = Q^*(Q_{t-1}, \lambda_t, X_t, Z_t, w_t, P_t, M_t, r, \sigma, u_1, u_2, \varepsilon_{1t}, \varepsilon_{2t})$,

Repeated substitution of lagged versions of (8) into (8), yields a Frisch demand for Q_t that is not

conditional on Q_{t-1}:

(9) $Q_t = Q^{**}(Q_0, \lambda_t, J_t, X_t, Z_t, w_t, P_t, M_t, r, \sigma, u_1, u_2, \varepsilon_{1t}, \varepsilon_{2t})$,

where $J_t = \{X_k, Z_k, w_k, P_k, \varepsilon_{1k}, \varepsilon_{2k}\}$, and k=1,...,t-1.

Finally, substituting in the determinants of λ_t yields a Marshallian demand function for Q_t:

(10) $Q_t = Q^{***}(Q_0, A_0, J_t^*, X_t, Z_t, w_t, I_t, P_t, M_t^*, r, \sigma, u_1, u_2, \varepsilon_{1t}, \varepsilon_{2t})$,

where M_t^* is a vector of moments of the distribution of $\{X_k, Z_k, w_k, I_k, P_k, \varepsilon_{1k}, \varepsilon_{2k}\}$, and k=t+1,...,

T and $J_t^* = \{X_k, Z_k, w_k, I_k, P_k, \varepsilon_{1k}, \varepsilon_{2k}\}$, and k=1,...,t-1.

This model can be considerably simplified by assuming that the elements of M_t^* are

functions of current and past realizations of the exogenous variables. In panel data, we can also

control for Q_0, A_0, r, σ, u_1, u_2 by including a child-specific fixed effect, η to yield the following

reduced form demand for health:

(11) $Q_t = Q'(J_t^*, X_t, Z_t, w_t, I_t, P_t, \eta, \varepsilon_{1t}, \varepsilon_{2t})$.

Alternatively, we could start with (8), allow the elements of M_t to be functions of current and past

realizations of the exogenous variable, and capture λ_t, r, σ, u_1, u_2, with either a child-specific or

family specific fixed effect to arrive at:

(12) $Q_t = Q''(Q_{t-1}, J_t^*, X_t, Z_t, w_t, P_t, \eta, \varepsilon_{1t}, \varepsilon_{2t})$.

This simple model yields several insights into why parental SES might matter for child health.   First, the budget constraint will be less binding in wealthier families, and these families will be able to purchase more or better quality material health inputs. Inputs include factors such as better quality medical care and food, as well as safer toys, housing, and neighborhoods. Set against this is the possibility that parents with a higher value of time in market work will invest less time in child health production, though to the extent that parents can purchase substitute care of adequate quality, this may not have any negative effect.

Lower socioeconomic status is, however, not only a matter of what inputs one can afford to buy, but also a matter of what one can do and chooses to do with the inputs one has at hand. Parents of lower SES may have different past experiences with the health care system, or health beliefs (e.g. about whether it is normal for a child to have coughing or wheezing), or preferences, which would be reflected in the taste shifters X and u.   Fuchs (1992) emphasizes the rate of time preference and a sense of self-efficacy as two things that are related to socioeconomic status but might have independent effects on the way parents choose to combine inputs in order to produce child health.   Cutler and Lleras-Muney (2006) also emphasize the effect of education on decision making about health behaviors.

Parental education is often highlighted as a productivity shifter in the health production function (3), and as we will show below, poorer children seem to be more subject than richer children to health insults, which would then change the productivity of future investments in child health.

In his model of capacity formation, Heckman (2007) focuses on the dynamic aspect of the

human capital investment model, and specifically on the way that Q_t depends on Q_{t-1}. Cunha, Heckman and Schennach (2006) focus on the development of cognitive skills, arguing that a model in which there are "dynamic complementarities" and "self-productivity" fits the available evidence well. Dynamic complementarities imply that investments in period t are more productive when there is a high level of capability in period t-1. Self-productivity implies that higher levels of capacity in one period create higher levels of capacity in future periods. Heckman (2007) hypothesizes that a similar argument might be true of child health, and that there might also be complementarities between child health and cognitive development, the question explored in section 2 below.

Finally, children of lower SES families are likely to have lower Q_0 at birth. There is a tendency among economists to assume that differences that are manifest at birth must be purely genetic. However, a good deal of evidence in epidemiology suggests that this view is simply wrong-headed. Studies by David Barker (for example, Barker, 1998) show that fetal conditions are related to adult risk of disease. Gluckman and Hanson (2005) offer a more recent summary of work on this idea which has come to be known as the "fetal origins hypothesis." This work can be seen in the broader context of research investigating the interplay between genes and environment (see for example, Rutter, 2006). The idea is that genes may predispose a person to a condition, but that it generally takes an environmental trigger to activate the expression of the gene. Moreover, genes that are associated with pathology in one set of circumstances may have protective functions in other contexts, which may explain why they persist in the genome. Thus, low SES children may have low Q_0 at birth because of the circumstances surrounding their births, rather than because their parents have "inferior" genetic endowments.

b) Correlations Between Parent's SES and Child Health

Differences in the health of high and low SES children are apparent at birth. 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 fathers were in the highest prestige occupations, 5 percent were low birth weight (birth weight less than 2,500 grams) 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.

Measures of SES vary from study to study. In addition to parent's occupation or measures based on area of residence, many studies focus on the correlation between maternal education and measures of child health. In fact, this robust relationship was one factor underlying the World Bank's drive over the past decade to promote maternal education in developing countries (World Bank, 1993). Much of the literature on maternal education and child health focuses on developing countries, emphasizes the effects of improvements in relatively low levels of education, and does not attempt to establish whether the measured effect of maternal education is causal.

This sub-section focuses on the relationship between income and health, as an illustration of the strong and consistent relationship between SES and health. 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) suggest that poor children are in worse health than richer children. In an important paper, Case, Lubotsky, and Paxson show that in the U.S., these gaps in health status tend 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 (since higher numbers indicate worse health). 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.

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), Brooks-Gunn and Duncan (1997), 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

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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. Moreover, a great deal of literature outside economics suggests that there might be a causal relationship between low socioeconomic status and mental health problems, perhaps because low SES acts a stressor. See for example, McLeod and Shanahan (1993), Kessler and Cleary (1980), Lundberg (1997) and Dohrenwend, Levav, and Shrout (1992).

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 15.9% of poor children and 13.1% of non-poor children have been told by a physician that they have asthma. Asthma that is properly controlled may not have serious consequences for human capital development, but poor

children are less likely than richer children to be properly managing their asthma. As a result 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.

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 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 been told that they have at least one of these conditions. Taking differences in the probability that a condition is diagnosed into account would likely increase the gap in incidence between rich and poor children, since poor children are less likely to receive medical attention for their health problems, and are therefore less likely to be properly diagnosed.

Disparities between rich and poor children in the extent to which they are limited by their conditions are much greater than disparities in the reported prevalence of conditions between poor and non-poor children: 11.4 percent of poor children say that they are limited by chronic conditions compared to 7.0 percent of higher income children. The fraction of children with a

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[1] ADHD is characterized by an inability to pay attention (inattention) and/or hyperactivity. The main diagnostic criteria for ADHD are laid out in the <u>Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition</u> (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).

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.

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. 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, especially among poor children. Other types of acute health problems are also more likely to strike poor children.[2] 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. Untreated dental disease can lead to problems eating, speaking, and learning.

A significant limitation with parental reports about child health is that there may be systematic differences in the way that parents of different backgrounds perceive and report illness. For example, if a health condition such as asthma is very common in a particular group, parents may not perceive that the health problem limits their children. However, in this case, the true gap between rich and poor children in the extent to which they are limited by chronic conditions would be even larger than that reported in Table 2.

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

This last criterion means that teachers are often important for the diagnosis of ADHD.
2 This observation is consistent with a large body of previous work including Starfield et al (1984).

very limited. 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 (Unicef, 2000). One possible explanation is that rich and poor parents have different views about what sort of injuries "require" medical attention.

In the U.S., Case, Lubotsky and Paxson (2003) show that parental reports of children's health status are highly correlated with physician reports in the National Health and Nutrition Examination Survey (NHANES), a national survey which has both types of reports. Unfortunately, most questions about chronic conditions in the NHANES are asked to parents rather than based on a determination by the physician (typically, parents are asked whether a doctor has ever told them that the child has a particular condition).   More objective measures of the extent of correlation between measures of SES and health await better health data, perhaps from "registry" data sets that include all patient encounters with the medical system.

The NHANES does have objective information about some health measures, most notably body size and nutritional status based on blood tests. Hedley et al. (2004) 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). Using data from the 3rd NHANES, Bhattacharya and Currie (2001) show that 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.

Other potential health measures are not well measured in any survey. For example, there is a good deal of evidence that low income children are more likely to be exposed to toxic chemicals, some of which are thought to have effects on cognitive development. This issue is explored in the "environmental justice" literature. For example, Rauth et al. (2006) examine a group of children who were prenatally exposed to pesticide (cholorpyrifos) 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.

One might be interested in whether the temporal pattern of family income made a difference to child health. The theory sketched above suggests that persistent poverty is likely to have worse effects on health than transitory poverty, since health is a stock that will be affected by past investments, and children with low "capacities" may be less able to parlay new investments in their health capital into good outcomes. Cross-sectional surveys such as the NHIS and the NHANES cannot be used to address this question.

Several authors have used data from the National Longitudinal Survey of Youth to address this issue, although its health measures are rather limited. Korenman and Miller (1997) find that children in persistently poor families have lower height-for-age. McLeod and Shanahan (1993, 1996) and Strohschein (2005) find that child mental health, and particularly aggressive behavior, may be affected more by persistent poverty than by current poverty alone. Guo and Harris (2000) focus on a measure of the number of years since birth that the child's household has been

in poverty. They estimate a structural model and show some of the effect of poverty on child cognitive outcomes is mediated through the effects of poverty on child health at birth. They also find that later child health is significantly related to cognitive outcomes, where child health is measured using number of illnesses in the past year, whether the child had medical attention, and whether the child is reported to be limited in his or her physical activities.

c) Do Correlations Imply Causality?

The literature discussed above conclusively demonstrates that children of poor or less educated parents are in worse health on average than other children, even in a rich country like the United States. But this does not necessarily imply that low SES causes poor child health. It is possible, for example, that a third factor causes both poverty and poor child health. Since parents who are in poor health are likely to have lower earnings, and may have achieved less education, perhaps parents' poor health, rather than parents' low earnings or education, is causally related to poor child health.

Alternatively, poor child health may reduce parental earnings. Several papers have examined the relationship between maternal employment and child health. For example, Powers (2001) investigates this relationship using whether a child has specific conditions as an instrument for the child's health status. The results are sensitive to the inclusion of maternal health status, and do not establish that family income is significantly affected. Earlier work by Wolfe and Hill (1995) shows that single mothers with disabled children are less likely to be employed. A question raised by their results is whether child disability has effects on family income by encouraging marital dissolution.

It is important to identify causal effects because if parental SES does not affect child health (at least when SES is above some minimum level) then interventions to increase parental SES will not necessarily improve child health. However, the literature attempting to identify causal impacts of parental SES on child health in a developed country context is small. It is difficult to find interventions that affect parental SES, but that are not also likely to have a direct effect on children's health.

In the developing country context, Desai and Alva (1998) look at Demographic and Health Survey data for 22 countries, and show that the strong correlation between maternal education and three measures of child health becomes much weaker when controls for husband's education and area of residence are included. Their point is not that SES does not matter, but rather that it is difficult to know if maternal education is the most important aspect of SES.

Thomas, Strauss and Henriques (1991) use data from Brazil and find a strong positive effect of mother's education on child height. They find evidence that much of the correlation can be explained by measures of mother's access to information, such as reading the newspaper and listening to radio, and argue that this supports the idea that the effect is causal. Duflo (2000) makes use of a pension reform in South Africa that brought black pensioners up to parity with white pensioners. She finds that the reform had an effect on height-for-age of girls born after the reform. Chou et al. (2007) find that in Taiwan increases in compulsory education from six to nine years, coupled with an aggressive a school building program led to increases in parental schooling that were associated with an 11 percent decline in the infant mortality rate.

There is little work exploring the relationship between maternal education and child health in developed countries. Currie and Moretti (2003) use the great expansion in the number of

colleges that occurred in the U.S. in the 1960s and 1970s as an instrument for college going among American women. They show that the opening of a new college in the woman's county of birth 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 (2006) 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 those born after the cut-off enter school as the oldest children in the class, while those born before the cut-off enter school as the youngest. Being consistently the oldest child in ones class may have effects independent of the impact on school leaving (see for example Bedard and Dhuey, 2007).3 Chevalier and O'Sullivan (2007) use a change in minimum school leaving age in Great Britain to examine the effect of maternal education on birth weight. They find that the law had differential effects by socioeconomic status, and that the most impacted groups experienced the largest changes in infant birth weight.

Carneiro, Meghir and Parey (2007) examine the effect of maternal education using data from the NLSY. They instrument maternal education using local labor market conditions, and the

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3 Dobkin and Ferreira (2007) argue that school entry laws are not good instruments for

presence of a four year college and college tuition at age 17 in the county where the mother

resided when she was 14 years old. Like the college openings used by Currie and Moretti

(2003), these instruments mainly identify the effect of college education rather than, for example,

an additional year of high school. They find strong initial effects on measures of cognitive

outcomes, and stronger medium terms on a measure of behavior problems, which, as discussed

further below, might be regarded as a proxy for or correlate of mental health conditions. They

also find strong effects of maternal education on the home environment, measured using variables

such as whether the child is read to, and whether the child has special lessons. They conclude

that the effects of increases in maternal education are large relative to the effects of other

interventions designed to affect child outcomes.

A few papers attempt to look at the effects of exogenous changes in household income.

The problem here is to find exogenous sources of variation in household income that do not also

affect other conditions in the household. For example, some welfare-to-work experiments

increased household income, but these interventions also encouraged maternal employment, so

they cannot be regarded as purely income interventions. The National Research Council and

Institute of Medicine (2003) summarize this literature and find that over all, welfare-to-work

interventions had surprisingly little impact on children, positive or negative.

Berger, Paxson, 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

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educational attainment in wage equations because they affect wages by many causal pathways.

unmarried couples. 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. This suggests that income affects child mental health via its effects on observable health inputs. 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 the gaps in child outcomes that they observe. In other words, though the effect of income is statistically significant, it is small.

Burgess, Propper, and Riggs (2006) use a similar strategy and examine a cohort of children born in the U.K. in the early 1990s. They also conclude that income has little direct effect on child health, although the mother's own health, and events in the mother's early life matter more. Doyle, Harmon, and Walker (2007) use data from the 1997-2002 Health Survey for England, and instrument education and income using smoking of the grandparents, and a change in compulsory schooling laws which increased the years of compulsory schooling for some parents from 15 to 16 years. They find larger effects of income than previous studies using English data, and also find that effects are larger in poorer households. But the assumption that grandparent's smoking (an indicator of grandparent social class) affects grandchildren's outcomes only through parental income and education is a strong one.

There is 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 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).

Costello et al. (2003) discuss the Great Smoky Mountains Study, a natural experiment involving the opening of a casino on an Indian reservation. As a result of the opening, every family on the reservation received a cash transfer.   Before the opening, the poor children on the reservation had higher levels of psychiatric symptoms.   After the opening, children in families who were raised above poverty had lower numbers of conduct and oppositional disorders (though there was no effect on anxiety and depression). However, these families were also less likely to be single headed, and reported that they were better able to supervise their children after the change, suggesting that there may have been other factors in addition to income at work.

Several papers attempt to control for unobserved family background characteristics that might be associated with both low income and low birth weight by estimating models with sibling fixed effects. Conley and Bennett (2000) use the Panel Study of Income Dynamics 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) 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 in models that include mother fixed effects. 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. Similarly, Johnson and Schoeni (2007) show in sibling fixed effects models also estimated using

the PSID that increases in income increase birth weight by much more if the mother was low birth weight herself. Results based on the PSID cannot, however, be regarded as definitive given the very small sample sizes available. The Conley and Bennett 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.

Currie and Moretti (2007) examine intergenerational transmission of low birth weight using a much larger data set based on birth records from California. The data base is constructed by starting with women who gave birth after 1989. If the mother was born in California, it is possible to go back in time and locate the mother's own birth certificate to find out her birth weight. Moreover, given the mother's mother's name (i.e. the grandmother's name), it is possible to identify mothers who are sisters in the data. They define the mother's birth socioeconomic status by examining income in the zip code of the hospital where the mother was born. They find that mothers who were born in poor areas were both more likely to be low birth weight, and about 6 percent more likely to eventually deliver a low birth weight baby themselves, even in models with grandmother fixed effects.

Van den Berg, Lindeboom, and Portrait (2005) 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, who were 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.

In summary, while there is a strong and exceedingly robust correlation between various measures of parental background and child health, it is difficult to prove that the relationships are causal. The literature attempting to do so is small and still has many holes. There is however evidence in support of the contention that maternal SES early in the child's life matters, and that child mental health may be particularly susceptible. I turn now to a discussion of whether differences in child health are likely to affect future socioeconomic outcomes.

2. Effects of Child Health on Future Outcomes

a) Why Might Child Health Affect Future Outcomes?

The idea that health is a "capacity" that affects production of a wide range of future capacities fits very naturally into the framework proposed by Heckman (2007). Poor child health is likely to affect future health, which in turn can affect labor supply and productivity. There is a great deal of evidence that socioeconomic status is related to health more generally (see Adler and Ostrove (1999), Marmot and Wilkinson (1999), and Cutler and Lleras-Muney (2006) for reviews) and that low socioeconomic status in childhood is related to poorer future adult health even in

adults who are no longer of lower socioeconomic status (see for example, Power and Peckham (1990), Davey Smith, et al. (1998), Poulton et al. (2002) and Marmot et al. (2001)). Elo and Preston (1992) summarize some of this literature, and show that cohorts who suffered high death rates in childhood also tend to show high death rates in adulthood, in part because of the direct effects of childhood health conditions on future morbidity.4

Bozzoli, Deaton, and Quintana-Domeque (2007) examine the relationship between the disease environment in childhood and adult height, using cross-country data for birth cohorts. They find that in rich countries, cohorts that suffered a higher disease burden in childhood have higher adult death rates. In poor countries, however, selection causes the relationship to be the reverse—in high disease environments only relatively strong people survive to become adults, and they tend to live longer (see also Blackwell, Hayward, and Crimmins, 2001).

The fetal origins literature discussed above strongly suggests that conditions in utero affect not only birth weight but features such as basic metabolism, which in turn affect future health outcomes. For example, Barker (1998) maintains that fetuses starved in utero may develop more efficient metabolisms, which then place them at higher risk for future obesity, heart disease, and diabetes. Since adult health is strongly linked to adult economic well-being, this suggests a relationship between health in utero and future outcomes.5

Poor fetal and child health can also have direct effects on the acquisition of skills. For

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4 Elford, Whincup, and Shaper (1991) provide a critical review of the literature linking early SES and adult heart disease and argue that most studies do not establish a causal relationship because the results are not very specific (childhood SES affects other outcomes besides heart disease), they are not consistent (either within or across studies), many studies do not adequately control for confounders, that there is little evidence of a dose-response relationship, and that the estimated effects are often weak.
5 Currie and Madrian (1999) summarize the evidence linking health and labor market outcomes among working age adults. The relationship between SES and outcomes may be weaker for older adults. For instance, Adams et al. (2003) find no relationship between SES and mortality or sudden onset conditions among the elderly, though they do find an association of SES with the incidence of gradual onset conditions.

example, maternal alcohol consumption can lead to permanent brain damage, as can trauma during the birth itself. O'Conner et al. (2000) study Romanian orphans and find that those who spent the longest time in deprived conditions had the worst cognitive outcomes. Thus, we know that severe insults in utero or in early childhood can cause permanent cognitive impairments. The question is really how sensitive these "sensitive periods" are and whether damage due to less extreme deprivation is noteworthy or widespread.

Among older children, some commentators have focused on school absences as a mechanism for health to affect education (Grossman and Kaestner, 1997), though the mean number of days absent (shown in Table 2) is quite small for both poor and non-poor children. Hence, if poor health among school aged children has an effect on the acquisition of skills it is more likely to come through impairing children's ability to learn while they are in school. Conditions such as anemia and lead poisoning (discussed below) would have this effect, though they are now relatively rare in developed countries. Conditions such as dental caries and ear infections are much more common so they might have a greater overall impact.

As stressed above, mental health conditions may be a particularly important mechanism because they are common, and because they may have particularly deleterious effects. For example, Currie and Stabile (2006, 2007) argue that some mental health conditions have worse effects on schooling attainment than most physical chronic conditions. Many of the "non-cognitive" skills that Heckman (2007) emphasizes may in fact be interpreted as mental health conditions.

b) Correlations Between Child Health and Future Outcomes

There is a good deal of evidence from developing countries that children who are in poor health also tend to have lower educational attainments. Behrman (1996) summarizes this literature, but expresses some skepticism about whether the relationship can be said to be causal.6

In developed countries, there has been relatively little examination of the question of whether poor health in childhood is linked to lower future socioeconomic status (other than through effects on future health status). One exception is a recent literature linking low birth weight to negative future outcomes. Low birth weight has long been used as the leading indicator of poor health among newborns. 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 short tem outcomes such as infant 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). Many follow ups indicate that low birth weight babies have lower scores on average on a variety of tests of intellectual and social development (see for example, Breslau et al. 1994, Brooks-Gunn, Klebanov, and Duncan, 1996).

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,

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6 In one of the strongest papers on this topic, Kremer and Miguel (2004) use an experimental design and show that treatment for parasitic worm infections can increase schooling attendance dramatically (though they do not find effects on test scores in their Kenyan sample). Bleakley (2007) shows that the eradication of hookworm in the American south also had a significant effect on school attendance, and that in addition, it affected literacy.

Fertig, and Paxson (2005) extend this research by showing that the same is true at age 42. They also show that adults who suffered chronic conditions as children have worse educational and labor market outcomes.

In their study of the Health and Retirement Survey, a national survey of older adults, Luo and Waite (2005) find that the effect of a retrospective measure of childhood SES on future health, education, and income is attenuated by the inclusion of child health measures, suggesting that child health may explain some of the impact of low childhood SES on future outcomes.

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. In a 1997 paper, Grossman and Kaestner summarized the existing evidence linking child health to future educational attainment and concluded that while most studies had methodological weaknesses, it seemed more likely than not that there was a connection. In the past 10 years there has been an explosion of research on this topic, much of it paying careful attention to the causal question. Causality is obviously key if one seeks to intervene to improve cognitive outcomes by improving health, or to determine how much of the societal payoff to improving health might come from improving cognitive functioning and future productivity. The discussion below is arranged by type of health measure.

c) Causal Effects of Child Health on Future Outcomes

i) Fetal Origins

The most compelling examinations of the fetal origins hypothesis look for sharp

exogenous shocks in fetal health that are caused by conditions outside the control of the mother. Doblhammer (2004) summarizes evidence that health shocks in early life due to wars, famines, and other crises can have lasting effects on health. For example, researchers have focused on the "Dutch Hunger Winter" of November 1944 to April 1945, when Dutch citizens were reduced to starvation by the Nazi occupation. Adults in utero during the time of the famine are more likely than those in 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 can 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 diabetes, stroke, activity limitations, cancer, hypertension, and heart problems and reported poorer general health status as adults.

Almond (2006) examines the direct effects of the influenza epidemic on the education and labor market outcomes of people affected by the disease in utero. 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, Edlund, and Palme (2007) examine the effects of the Chernobyl disaster, which sent a radioactive cloud over Sweden in April 1986. The amount of fallout varied substantially across regions but was considered "low dose" everywhere. They find that children who were in utero 8 to 25 weeks at the time of exposure were 3.6 percent less likely to qualify for high school and had 5 percent lower grades. Restricting themselves to sibling comparisons increases the estimated effects to 5.6 percent and 8 percent.

Nilsson (2007) examines the effects of a temporary liberalization of alcohol policy in two Swedish counties which led to a brief upsurge in binge drinking among young women in those counties. The design allows him to compare cohorts immediately before and after the affected cohort, and to use children born in other counties (excluding neighboring counties) as controls. He finds that children who were in utero during the experiment have fewer years of schooling, lower future earnings, and higher welfare dependence as adults.

On the other hand, Cutler, Miller and Norton (2007) examine the long-term effects of the economic upheaval on members of the Health and Retirement Study who were in utero during the depression era "Dust Bowl". Their specifications control for region, year of birth, and region specific trends so that their models are identified by regional deviations from these trends. They also control for economic conditions during the first five years after birth, in order to focus on shocks in utero alone. They do not find any significant effects of negative economic shocks on future health (measured using chronic conditions, difficulties with activities of daily living, mortality, height, and body mass index). On the other hand, they do not find any effect of their measure of economic distress on infant deaths, births, or the infant mortality rate. This suggests

that these income shocks did not translate into early life health shocks for these cohorts, and that this is why there is no evidence of later life effects.

ii) Birth weight

Many studies of the effects of birth weight use sibling/twin comparisons. 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.

Several recent studies conducted using large samples drawn from vital statistics records in Scotland, Norway, Canada, the U.S. all show a link between low birth weight and lower educational attainment, even among siblings or twins. Lawlor et al. (2006) examine the birth weights of Scottish siblings born between 1950 and 1956 and find that lower birth weight siblings had lower scores on a test of intelligence at age 7. Black, Devereux, and Salvanes (2005) examine a large sample of Norwegian twins and find, using twin fixed effects, 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. Although these studies emphasize sibling or twin comparisons, the estimates obtained this way are remarkably similar to those obtained using Ordinary Least Squares (OLS).

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: For example, at the low end of the birth weight distribution, a one pound increase in birth weight from 540 to 1000 grams would reduce the probability of infant death by 0.15. At higher birth weights, increases in weight have no effect on infant death, but still reduce hospital costs. For example, moving from 2000 to 2500 grams is associated with a reduction of $2000 (year 2000 dollars) in hospital costs.

Royer (2005) uses birth certificate data for California (similar to that of Currie and Moretti, 2007, which was described above) to examine longer term outcomes. Royer examines mothers who were twins born over the 1960 to 1982 period, and finds that each 1,000 gram

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increase in birth weight is associated with a gain of 0.16 years of education. There are of course, issues involved with using a selected sample of women who have given birth, 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 one's birth should have effects many years later.

In addition to examining the effects of mother's SES on birth weight, Currie and Moretti (2007) use the California birth certificate data to examine the long term effect of maternal low birth weight. They find that low birth weight has significant effects on later socioeconomic outcomes. In particular, when they compare mothers who are sisters by conditioning on grandmother fixed effects, they find that the sister who was low birth weight is three percent more likely to live in a poor area at the time she delivers her own child, and three percent less likely to be married when she gives birth. The low birth weight sister also has about a tenth of a year less education on average.

Johnson 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 lowers 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 both Royer (2005) and Currie and Moretti (2007). But in their models of earnings, controlling for education has little effect on the estimated effect of low birth weight. It is possible that a larger fraction of the effects

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of birth weight on earnings is accounted for by effects on cognition. Johnson and Schoeni find that measures such as passage comprehension, reading ability and 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.

There are several possible threats to identification in studies that rely on twin or sibling differences. First, random measurement error in birth weight can bias estimated coefficients towards zero.   Studies based on birth certificate data will likely have some error, but not as much as studies based on retrospective maternal reports of child birth weight. One could easily imagine non-random measurement error in maternal reports as well. For example, a mother might tend to report that both children were of what she considers to be an average birth weight. In this case, controlling for mother fixed effects will increase the estimated coefficients by removing the effects of the mother-specific measurement error.

More fundamentally, fixed effects estimates implicitly assume that all of the relevant omitted variables are the same for both members of the sibling pair.   But siblings who are not identical twins have some genetic differences, and parents may treat even identical twins differently. For example, fixed effects may be biased if parents 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, whereas if parents favor the stronger child, effects will tend to be overestimated.

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., the available evidence suggests that investments are often compensatory (Behrman, Pollak,

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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 (especially given the possibility of measurement error). It is, however, remarkable that significant effects are found across so many different time periods and countries.

Almond and Chay (2005) examine the effect of a mother's health at birth (and in early childhood) on the health of her children using a different identification strategy based on the comparison of cohorts. 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 desegregation of hospitals and increased access to medical care (Almond, Chay, and Greenstone, forthcoming). For example, there was a large decline in deaths due to infectious disease and diarrhea in these cohorts. 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 the outcomes of 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. Almond and Chay conclude that the infants of black women who had healthier infancies as a result of the Civil Rights movement show large gains in birth weight 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 children of mothers from the the 1967-1969 cohort compared to the earlier cohort.

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.7

For birth weight then, there is a good deal of evidence that child health affects future child

outcomes. It is notable that the effects on education are smaller than those on future earnings. It

is also remarkable that the evidence is so consistent across countries and time periods, that the

effects appear to be roughly linear, and that the inclusion of maternal fixed effects (i.e. sibling

comparisons) produce estimates that are roughly similar to OLS estimates.

There are fewer studies examining other health measures, or measures for older children.

The remainder of this section is organized by first discussing a paper by Smith (2007) that relies

on an overall health measure, and then drawing an example from each of several specific health

domains and discussing the evidence linking these domains with future outcomes. The chosen

domains are nutrition, mental health, chronic physical conditions, acute conditions, and toxic

exposures.

iii) Evidence from an Overall Health Measure

Smith (2007) investigates the relationship between child health and future outcomes using

data from the 1999 Panel Study of Income Dynamics. The adult children of the Panel Study of

7 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

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Income Dynamics (PSID) respondents (who were 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.

A striking result is that aside from the models for education, the estimated effects are generally larger in the fixed effects models than in OLS models, whereas one would expect that controlling for omitted variables that were correlated both with health and with outcomes would reduce the estimated effects. Smith argues that there is measurement error in the health measure and that it is unlikely to be random. If siblings report similarly, then the difference in health between siblings will be more accurately measured than the difference between random members of the population. In this case, controlling for the measurement error by adding mother fixed effects will increase the size of the estimated coefficients.

The lower estimated effect on education suggests that perhaps health in childhood affects outcomes such as adult income through mechanisms other than educational attainment, which is consistent with the results of Johnson and Schoeni discussed above. For example, sickly children may be less able to work hard as adults.   Note that if health affects future wages through a

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either to fetal selection, or to fewer planned pregnancies.

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.

iv) 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. Maluccio et al. (2007) follow the same children into adulthood and show effects on completed education. 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 prenatal participation in the U.S. WIC (Supplemental Nutrition for Women, Infants, and Children) program is associated with higher test scores. WIC is a program that provides coupons redeemable for specific foods to women, infants, and children 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 future 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.

Given the trends in obesity, it is worth noting that even the obese are often badly nourished. However, I am not aware of studies that have shown a direct causal relationship between obesity in childhood and future educational or labor market outcomes, though there could be a relationship from childhood obesity to adult obesity, and between adult obesity and wages (Cawley, 2004).

v) 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 longitudinal samples. Kessler et al. (1995) uses data from the U.S. National Comorbidity Study, which surveyed 8,098 respondents 15 to 54

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years old from 1990 to 1992, assessed their current psychiatric health, and collected 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. 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 (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. However, few psychological studies go on to look at longer term outcomes such as educational attainment.

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.

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However, by excluding children with any behavior problems from the control groups, the studies might overstate the effects of ADHD.

The longitudinal studies reviewed above establish important information about the correlations between mental health in childhood and longer term outcomes. But beyond adding available controls to regression models, they do not generally address the possibility that the negative outcomes might be caused by other factors related to a diagnosis of mental health problems, such as poverty, the presence of other learning disabilities, or the fact that many people diagnosed with mental health problems end up in special education.

Currie and Stabile (2006) address these problems in the context of ADHD using sibling fixed effects models estimated on data from the NLSY and the (similar) Canadian NLSCY. An advantage of these data sets is that all children were administered "screener questions" that can be used to form a scale for ADHD. Thus, their analysis is not dependent on the child having been taken to a doctor for a diagnosis. 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 the 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 (2008) 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.

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.

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 (which could change over time) as well as innate character traits.

Ding et al. (2006) propose an innovative instrumental variables strategy, which is to use genetic markers known to be associated with mental health conditions as instruments for these conditions in models of educational attainment. In their preferred specifications, they find that depression and inattention are associated with a one standard deviation reduction in Grade Point Average. In order for genetic markers to be valid instruments, however, it is necessary that they have no direct impact on cognitive functioning. It is not clear that we can be certain of this at this

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point. Moreover, the results of Ding et al. (2006) are sensitive to the specification of the vector of health conditions. This is a significant problem given that we do not have a good sense of what vector of measures of health conditions is needed to fully account for the effects of health.

In summary, because of their high prevalence and negative consequences, mental health conditions are among the most important health problems impacting children's outcomes.   The available evidence suggests that "externalizing conditions" such as ADHD or aggression have more significant consequences for outcomes such as completed education and earnings than internalizing conditions.

vi) Chronic Physical Conditions: The Example of Asthma

We saw above that asthma was the most prevalent childhood 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 well controlled asthma 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, rather than to see whether asthma per se affected outcomes.

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

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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 (Annett et al. (2000), Butz et al., (1995), Halterman et al. (2001)) use very homogeneous groups of children and still find differences in behavior suggests that asthma when not properly controlled probably does have a causal effect, at least on behavior problems and perhaps on school readiness.

vii) 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. In developing countries, children often suffer from conditions such as parasitic infections. Poor children in developed countries are generally much healthier, but as discussed above, are more likely to suffer from acute illnesses such as dental caries and ear infections than richer compatriots. 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). Hearing loss can delay language development, but it is difficult to tell how important these effects might be in explaining disparities in outcomes.

viii) 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 shown to decrease IQ by two 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.   Lead may also have negative effects on children's mental health, making them more prone to anti-social behavior (Needleman and Gastsonis, 1991).

Before the regulation of lead, children were exposed to lead from paints, water pipes, gasoline, and canned food. Evidence from the NHANES surveys showed that 88.2 percent of children aged one to five had lead levels above10 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).

There is little research on the question of whether exposure to toxic releases at the level that now generally occurs in the population has negative health effects. Data on possible human health effects generally comes either from animal studies, or from disastrous accidental releases.

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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.

Vriheid (2000) looks at the question of whether residence near a hazardous waste site has health effects, and highlights some of the methodological weaknesses of existing studies. Residents of areas near hazardous waste sites are more likely to be poor and have lower levels of education than people in the remainder of the country (Currie and Neidell, 2005; Gallagher and Greenstone, 2005) so that their children's health outcomes are likely to differ even in the absence of negative health effects from exposure. Some studies control for some observable confounding factors, but there is still a possibility that there are unobservable characteristics of people who live close to hazardous waste sites which would tend to cause bad outcomes. An additional problem is that the number of hazardous waste sites analyzed in many of the previous studies is small, so that some "results" may actually be due to sampling variability. These problems plague much of the literature on toxic effects so that it is quite difficult to measure effects of pollution on health, let alone show that there are long-term consequences of exposures.

In two important and innovative works, Chay and Greenstone (2003a, b) used changes in regulation to identify pollution's effects on infant mortality. They argued that the 1970 and 1977 Clean Air Acts caused exogenous changes in pollution levels, and that the changes were different in different areas. These changes can be used to examine pollution's effects on housing markets and infant mortality. They found that a $1\mu g/m^3$ reduction in total suspended particulates (a

common measure of overall pollution at that time) resulted in 5-8 fewer deaths per 100,000 live births.   Reyes (2005) uses variation in prenatal lead exposure caused by the Clean Air Acts on infant health outcomes, and finds that even small amounts of lead are associated with adverse outcomes.

Currie and Neidell (2005) examine the effects of air pollution on infant deaths in the 1990s. They used individual-level data and within-zip code variation in pollution over time to identify the effects of pollution. They included zip code fixed effects to account for omitted characteristics like ground water pollution and socioeconomic status, and found that reductions in two pollutants – CO and PM_{10} – in the 1990s saved over 1,000 infant lives in California. Pollution may also have many negative health effects without causing deaths. Neidell (2004) also uses within-zip code variation in pollution levels in California to provide evidence that air pollution affects child hospitalizations for asthma.

These studies show that pollution can have causal effects on child health, but there has been little investigation of whether these negative health effects have long term consequences for children's outcomes. The National Children's Study, which was authorized by the Children's Health Act of 2000, will attempt to remedy this situation by examining the effects of environmental exposures on 100,000 children who will be followed from birth to age 21 (see http://www.nationalchildrensstudy.gov).

3. Can Health Account for Gaps in Child Outcomes?

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Table 4 summarizes the evidence linking several different domains of child health to outcomes. In order for the health problem to lead to disparities, the problem must either be more prevalent among the poor or have a larger negative effect on the poor, and must also be 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 with 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 (0.96*50 + 0.04*45) while the mean score among the poor will be 49.7 (0.94*50 + 0.06*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. As we have seen above, long-term effects of low birth weight on education are statistically significant and found in many different settings, but are relatively small (on the order of a tenth of a year of education, on average).

Still, it is important to keep in mind that health is a multi-dimensional concept, and ideally we would like to account for the effects of all of the health insults suffered by a child and the possible interactions between them. Moreover, several studies suggest that health insults may

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have large effects on future earnings and/or employment probabilities, even if they have relatively little effect on completed education. Completed education is a crude proxy (with relatively little variation) for the possibly more subtle effects of health in childhood on cognitive functioning. And childhood health may affect adult outcomes by influencing non-cognitive skills, and through effects on adult health.

In contrast to the estimates for most conditions that have been routinely observed at birth or later, the fetal injuries investigated by Almond and his collaborators appear to have very large effects on future outcomes. Recall that children of U.S. mothers infected during the flu epidemic were 15% less likely to graduate from high school, while the Swedish children exposed to low-level radiation after Chernobyl were 5.6% less likely to qualify for high school. These results raise the provocative idea that the best way to safeguard children's health may be to start with their pregnant (or pre-pregnant) mothers. They also suggest that our standard measures of child health after birth (such as birth weight) are inadequate as they do not appear to fully capture the effects of these fetal health insults.

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. The fetal origins literature provides a natural explanation of why this might be. But thus far, few researchers have attempted to directly assess the extent to which intergenerational transmission of income might operate through the effects of parental income on child health, and subsequent effects of poor child health on children's adult education and income.

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Eriksson, Bratsberg and Raaum (2005) have conducted one such study using data from Denmark that began with 14 year olds in 1968, and followed them 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.

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 educational and labor market outcomes? The answer to both questions is "yes", though it is too early to tell how important these feedbacks between health and more conventional measures of human capital may be. The available evidence suggests that fetal health may be particularly important, and hence that protecting the health of mothers may be one of the most effective ways to improve child health.

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 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 potentially valuable additions to the large epidemiological and medical literature on this subject.

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 may 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, for example, 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 is relatively little evidence that modest cash transfers have large effects.[8] 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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[8] However, see Dahl and Lochner (2005) for a recent study finding positive effects of cash transfers under the EITC program.

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.S.: Case, Lubotsky, Paxson, NHIS				
Ln(Income)	-0.183	-0.244	-0.268	-0.323
	[.008]	[.008]	[.008]	[.008]
Canada: Currie and Stabile, NLSCY				
Ln(Income)	-0.151	0.216	-0.259	-0.272
	[.026]	[.019]	[.024]	[.040]
U.K.: Case, Lee, Paxson, HSE				
Ln(Income)	-0.143	-0.212	-0.203	-0.194
	[.036]	[.026]	[.030]	[.034]

Notes: Standard errors in brackets. Regressions control for year
effects, family size, sex, mother age tat 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
Maternal Assessment of Child Health		
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
Health at Birth		
Birth weight (grams)	3221	3348
Birth weight < 2500 grams	0.112	0.078
Ever Chronic Conditions		
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
Activity Limitations		
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
Illness and Medically Attended Injury		
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 diabilities, developmental delays, mental Down's syndrome and autism.
b. Mental problem causes limit includes limits due to learning disabilities, developmental and other mental problems.

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

Ordinary Least Squares	Education	Weeks Worked	HH Income	Wealth	Ln Earnings	Growth 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 (2007). 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 houshold 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.

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