Intergenerational Effects on Health - *In Utero* and Early Life

Heather Royer
University of California-Santa Barbara, NBER
royer@econ.ucsb.edu

Allison Witman
University of California-Santa Barbara
bauer@econ.ucsb.edu

April 18, 2013

Synopsis

An emerging economics literature emphasizes the importance of *in utero* and intergenerational factors in the production of health. From a theoretical perspective this is not surprising as models of human and health capital increasingly emphasize the value of early childhood investments. Complementary to this, there is growing biological evidence via the fetal origins hypothesis considering how early life influences such as those *in utero* can have consequences for adult health. This chapter discusses theoretically the role of *in utero* and intergenerational factors in the health production function and then reviews the relevant empirical literature.

Glossary

APGAR score: A measure of infant health using a five component assessment completed immediately after birth.

cross-sectional data: Data observed for many subjects at one particular point in time.

dynamic complementarity: The notion that skills produced in one period increase the productivity of investment in later periods.

ectopic pregnancy: Implantation of the embryo outside of the uterus, usually resulting in a non-viable pregnancy and great health risk to the mother.

elasticity of substitution: A summary of how well one factor can be substituted for another in production.

fixed effect model: A type of statistical model intended to deal with omitted variables bias by using a dataset collected on multiple entities (e.g., individuals) over multiple time periods. To isolate the effect of some factor(s), the statistical model uses variation within entities over time.

omitted variables bias: Over- or under-estimation of the impact of one variable on another that is caused by leaving out one or more important variables from the estimated model.

quasi-experiment or natural experiment: A study where some entities (e.g., individuals) are exposed to an intervention outside of the researcher's control.

randomized-controlled trial: A scientific experiment conducted to test the effect of an intervention by randomly assigning participants to a treatment and control group. Differences between the

treatment and control group participants are interpreted as the causal effect of the intervention.

regression discontinuity design: A statistical technique used to address issues of omitted variables bias where assignment to treatment changes discretely based on some characteristic or set of characteristics. To estimate the effect of the treatment, groups of treated and untreated individuals with similar characteristics are compared. For example, college scholarships are frequently based on grade point averages. To estimate the effect of a scholarship, outcomes for individuals who had grade point averages that made them just eligible for the scholarship are then contrasted with the outcomes for individuals who had grade point averages that made them just ineligible for the scholarship.

self-productivity: The notion that skills acquired in one period persist into other periods and that higher levels of skills in one period can increase the amount of skills acquired in later periods.

Keywords

birth weight, child development, child health production, childhood investments, fetal environment, fetal origins hypothesis, *in utero*, income shock, infant mortality, intergenerational transmission of health, maternal education, maternal drinking, maternal nutrition, maternal smoking, prenatal care

Cross-Reference

This chapter is complementary to chapters in the Encyclopedia of Health Economics that investigate the determinants of health in adulthood by explaining the transmission of health across generations, both directly and indirectly. The relevant chapters include Chapter 1.9 "Education and health in developing countries", Chapter 3.5 "Health Insurance", Chapter 3.6 "Environment/Pollution", Chapter 3.7 "Macroeconomy and Health", Chapter 3.9 "Education", Chapter 3.12 "Alcohol", Chapter 3.13 "Smoking", Chapter 3.15 "Nutrition/Food Insecurity, Food Stamps, School Lunch", Chapter 4.17 "Fetal Origins of Lifetime Health", and Chapter 8.13 "Education and Health".

Relevant Websites

`http://www.thebarkertheory.org/`

I Introduction

Today an understanding of health is not complete without considering the role of *in utero* and intergenerational influences. The recent popularity of the fetal origins hypothesis, asserting that early life influences through the fetal environment (e.g., nutritional deprivation) have latent long-run effects on health, has nudged economists to think about the production of health beginning much earlier in life. This hypothesis, complemented with economic theories of parental investment characterizes how important early-life factors can be in explaining adult health.

The goal of this chapter is to give the reader a framework and an understanding of the strength of *in utero* and intergenerational influences. To provide a concrete structure to interpret these effects, we first outline a multi-period investment model akin to the work of James Heckman that translates early life circumstances to health in adulthood. With such a mathematical model, we can be very specific about the roles of the *in utero* and intergenerational environments. Before discussing the various inputs into the health production function, we consider possible measures of health *in utero* and at birth. The bulk of the chapter then discusses how various inputs (e.g., maternal nutrition, sickness, maternal age, maternal education, family income, employment, maternal health behaviors, and environmental exposure) impact health using the Heckman investment model as a guide.

II Theoretical Framework

A Economic Framework for the Link between *In Utero* and Early Life Conditions and Later Health

Under traditional economic models of health, there is little room for early life and *in utero* events to impact later health. More recently-developed models, however, demonstrate the importance of investment and events in early life for health production. Models such as Heckman (2007) provide the mathematical structure to understand how early parental investment and initial endowments (e.g., health at birth) may affect adult health. These models invoke two important

features: self-productivity and dynamic complementarity. Self-productivity embodies the notion that skills acquired in one period persist into other periods, and that higher levels of skills in one period can increase the amount of skills acquired in a later period. Dynamic complementarities embrace the idea that capabilities acquired in one period augment the productivity of investment in the future.

Consider a simplified model with two periods of childhood investment and a constant elasticity of substitution production function as Heckman (2007) does:

$$h = f(b, \theta, [\gamma I_1^{\phi} + (1-\gamma) I_2^{\phi}]^{\frac{1}{\phi}}) \tag{1}$$

where h is a vector of adult capabilities in period 3 (adulthood), b are parental capabilities, θ is the initial endowment, I_t is investment in period t, $\frac{1}{1-\phi}$ is the elasticity of substitution of inputs across periods, and γ represents the net effect of I_1 on h (the "capability multiplier"). One can think of h as including health and other capabilities such as education in adulthood. Investments can include nutrition and medical care in childhood. Parental characteristics such as income and education influence the choice of inputs (i.e., I_1 and I_2), either by shifting tastes, acting as a constraint on the ability to purchase inputs, or changing θ.

Self-productivity implies that $\frac{\partial h}{\partial I_t} > 0$ for t=1,2; that is, investment made in prior periods raises adult capabilities. Vaccinations would be one such example; the polio vaccine taken as a child nearly assures that as adult, an individual will not impeded by polio. Under dynamic complementarities, the function $\frac{\partial^2 h}{\partial \theta \partial I_t} > 0$, meaning that the effect of investment is an increasing function of capabilities. As an example of dynamic complementarities, consider an early childhood investment made in period 1 (e.g., Head Start). This investment may augment childhood capabilities in period 2, which then will make formal schooling following Head Start more productive.

Under some simplifying assumptions, this general model can generate some useful insights about the possible role of early and *in utero* investments. First, the larger the capability multiplier (i.e., γ) the higher the optimal ratio of early to late investment. Second, if early and late investments are perfect substitutes, disadvantage in period 1 can always be overcome with later investment. As the degree of substitution approaches ∞, optimal investment in period 1 is equal to optimal investment in period 2. Third, there is a tradeoff between investing in period 1 and investing in period 2. Due

to discounting, investment in period 2 is cheaper than investment of the same amount in period 1. This consideration pushes investment to period 2, but the productivity of investment in period 1 (i.e., the size of γ) encourages in investment in period 1.

This model can explain why early life investments, even if they are small in magnitude, can have effects on more long-run outcomes. Moreover, while this chapter discusses the importance of early and *in utero* conditions collectively, it may be important to distinguish between these even further. Specifically, *in utero* investments, because of the extended period allowed for dynamic complementarities, may be more important than early childhood investments.

B Fetal Origins Hypothesis - An Epidemiological Explanation for the Possible Connection Between *In Utero* Conditions and Later Outcomes

The biological foundation for linking *in utero* conditions to later life outcomes is the fetal origins hypothesis. This hypothesis, championed by British physician David Barker, asserts that nutrient deprivation at the beginning of life can raise adult chronic disease risk. Looking across areas in England, Barker noted that infant mortality rates were correlated with later mortality rates of the same cohorts. The biological underpinnings of the fetal origins hypothesis suggest that nutrition during pregnancy affects fetal development. If a fetus is deprived of nutrients *in utero*, available nutrients are diverted for neurological development while the development of non-neurological systems are sacrificed. This tradeoff manifests itself later in life in the form of of higher hypertension risk and increased insulin sensitivity.

While the hypothesis has gained some acceptance, it is still highly disputed - partially because solid empirical support is difficult to come by. For one, the data demands of testing such a hypothesis require data on both early life conditions and later outcomes. This is an arduous demand given that the collection of high quality data in many countries is only a recent phenomenon. Probably the most foreboding critique of this hypothesis is that it was originally based on observational data and thus, is susceptible to typical omitted variables bias issues. However, it should be noted that animal studies (excluding humans) where the fetal environment is more easily manipulable generally show strong support of the fetal origins hypothesis.

The toolkit of economists is well-suited to addressing these two shortcomings of the public health and medical literature on this topic. Economists have used clever quasi-experimental strategies

(many of which we will discuss later in this chapter) to identify causal relationships between early life conditions and later outcomes. A subset of these natural experiments include the 1918 and 1957 flu epidemics, maternal fasting during Ramadan, variation in malaria prevalence either due to seasonal variation or eradication campaigns, and the implementation of the federal Food Stamps program.

The application of the fetal origins hypothesis in the economics literature is broad. For instance, it is the most common explanation for the association between birth weight, a measure of *in utero* nutrition, and educational attainment, adult economic outcomes, and adult health outcomes. Some might argue that this is an incorrect interpretation of the fetal origins hypothesis because the hypothesis is specifically about how *in utero* circumstances have a latent impact which is only expressed in late adulthood, not in early adulthood.

It should be noted that while the fetal origins hypothesis provides a biological basis for the relationship between the *in utero* environment and subsequent outcomes, estimates of the relationship between early life circumstances and later outcomes will combine both the biological effect and the effects of any ensuing investment decisions. Several studies have been interested in whether investment responses are compensatory or reinforcing, but due to the difficulty measuring intermediate inputs the literature has not reached a consensus regarding which type of investment behavior is more predominant.

C Measuring *In Utero* Health and Later Health

Of critical importance in the *in utero* and early life health literature is the measurement of health. Measurement of *in utero* health without intervention is nearly impossible. However, via blood samples, measurements of the maternal environment can be made (e.g., cortisol levels indicating stress). But such data are not part of standard datasets commonly used by economists.

As an alternative measure of *in utero* health, researchers frequently use measures of health at the time of birth. These include birth weight, APGAR score, length of gestation, and infant mortality. Most of these measures are likely a reflection of the effects of the *in utero* environment rather than the circumstances after birth. While shifts in many of these outcomes (e.g., birth weight) may not be so meaningful, economists frequently are interested in the tails of the distribution of these outcomes. Low birth weight (< 2500 grams), very low birth weight (< 1500 grams), and premature

birth (< 37 weeks of gestation) are focal outcomes.

In the last 10 years, health economists have debated whether birth weight is an adequate measure of *in utero* health. While the measurement of birth weight is easy, by itself, birth weight is not necessarily reflective of any health issues. Historically, interest in this measure by researchers is mainly predicated on the strong birth weight and infant mortality correlation. But such correlation does not imply causation. As an innovative approach to control for possibly confounding factors, researchers have compared birth weight differences between twins and have related those differences to within-twin-pair differences in infant mortality. A weaker birth weight and infant mortality relationship emerges from this approach. Nevertheless, the importance of birth weight as a leading health indicator has been reaffirmed with the many recent studies mapping a connection between birth weight and longer run outcomes such as educational attainment, wages, and rates of disability as adults.

Measuring early childhood health is equally difficult as measuring *in utero* health. Easily-obtained health measures such as childhood mortality are rare, making it challenging to find effects of interventions on mortality. The most common chronic conditions in childhood are asthma, hay fever, and bronchitis, but they inflict less than 15% of children in a particular year. Aggregating these conditions to derive a single index measure of health is challenging because it is unclear how to combine these outcomes sensibly. For example, an outcome of the number of chronic conditions a child has would give equal weight to epilepsy as it does to bronchitis.

III The Intergenerational Transmission of Health

The model outlined by equation (1) allows for an intergenerational transmission of health via several different mechanisms. First, parental attributes can affect a child's health directly through changes in b, parental capabilities. Second, intergenerational relationships can arise because of genetics, θ in the model. Third, parental capabilities will likely affect investments represented by I_1 and I_2. Distinguishing between these three types of mechanisms is not possible empirically.

Arguably the best measures of the intergenerational correlation in health are those relating to birth weight. The correlation in birth weight across generations is typically smaller in the U.S. than the intergenerational correlation in wages. In a study using matched children-mother

data from California, the likelihood that a child is low birth weight increases by 50 percent if her mother was low birth weight. These intergenerational relationships are slightly stronger among low socioeconomic status mothers.

Data on sibling mothers can help to understand how much of the intergenerational transmission in birth weight is genetic versus behavioral. Traditionally this is done by assuming a data-generating process where a child's birth weight is assumed to be an additively separable linear function of mother's birth weight and a mother's family fixed effect. The fixed effect is intended to capture genetic factors that mothers who are siblings share in common, but it also captures anything else the sibling mothers share. This assumed relationship is rather restrictive as it does not allow for a gene and environment interaction. Interestingly, based on non-twin mother sibling comparisons, family background characteristics do not explain the intergenerational correlation in birth weight. But some argue that these siblings are not nearly enough alike. Thus, other studies focus on twin sibling comparisons. Unlike in the case of sibling mothers, some of the intergenerational birth weight relation is explained by family background. The effect of mother's birth weight on child's birth weight in models that control for time-invariant features of the mother's family is about half the size of that from models that do not, suggesting a strong possible role for genetics.

This chapter continues by investigating maternal factors (e.g., income, nutrition) and other influences (e.g., environment, health care) that may explain these intergenerational correlations in health.

IV Factors Affecting *In Utero* and Later Health

A Maternal Sickness and Stress

A natural empirical test of the fetal origins hypothesis (or the effect of the *in utero* environment more generally) is to examine influences on the maternal environment during pregnancy. These influences include maternal sickness, maternal stress, and maternal nutrition. We reserve discussion of maternal nutrition until later as the literature is more expansive on that topic. In general, it is difficult to isolate the pure effect of these factors because it is nearly impossible to conceive of a quasi-experiment that only manipulates sickness or stress. For example, terrorist attacks such as September 11th have been used to understand the effect of maternal stress, but one might imagine

that these attacks could also have economic effects.

Of the maternal influences, maternal sickness is considered to be one of the most important. The 1918 flu epidemic provided a unique opportunity to examine the effect of prenatal flu exposure on long run outcomes. This flu spread rapidly and suddenly; 25 million people in the United States contracted the virus. Cohorts *in utero* at the time of the flu exhibited diminished health and economic outcomes as adults (i.e., higher disability rates, lower education attainment, and reduced wages). For the more recent Asian flu pandemic of 1957, it is possible to follow the effects of the flu across the lifecycle. In particular, unlike for the 1918 flu, one can test whether flu exposure is related to reduced birth weight, one of the underpinnings of the fetal origins hypothesis. Overall, the flu does not impact birth outcomes. However, these effects are quite heterogenous. The children born to smoking mothers or shorter mothers exhibit lower birth weights as a result of the flu. Effects on cognitive outcomes are present overall, not confined to a particular subgroup. Exposure to malaria *in utero* and during early childhood also has important consequences for long-run outcomes. While today malaria is an issue in developing countries, in the early 20th century rates of malaria in the American South were comparable to those in developing world today. Exposed cohorts have lower educational attainment and higher rates of poverty.

Relative to maternal sickness, understanding the effect of maternal stress is more challenging. Measurement of maternal stress is typically indirect because measurement of stress is difficult. As a result, studies of the maternal stress often focus on events that are presumed to affect maternal stress. Terrorist attacks such as September 11th and armed conflict in Israel are two such examples. For these events, since they are more recent, evidence on the long-run impacts is limited. However, the stress-provoking events have substantial short-run effects on the incidence of low birth weight and prematurity. As an alternative to this case study approach, some research has measured maternal stress through cortisol levels directly. Sibling comparisons - effectively comparing maternal cortisol levels across births to the same mother and relating these within-family differences to differences in long-run outcomes are used. These cortisol differences have consequences for cognitive, educational, and health outcomes.

Overall, this literature evaluates the effect of negative shocks to the maternal environment. As such, these research findings may be less interesting for policymakers who are interested in deciding which policies are best to improve the fetal environment. Indeed more research is needed on positive

shocks.

B Maternal Characteristics

Maternal attributes such as education and age can impact early life health either directly or indirectly through the choice of familial inputs or endowments. For example, a mother's education may affect her knowledge regarding the health impacts of maternal smoking. On the other hand, in the presence of assortative mating, her education may influence the education of the mate she chooses.

There is a recent growing interest in the impact of maternal education within economics. This is in part due to an expanding focus on the non-wage effects of human capital. Moreover, maternal education is one of the strongest predictors of infant health. Based on U.S. data, an extra year of schooling reduces the rate of low birth weight by 10 percent. These effects are surprisingly linear, implying that the effect of a year of high school education is roughly equal to the effect of a year of college education.

Of course, these correlations do not necessarily imply that there is a causal relationship between maternal education and infant health. Omitted variables bias is a concern, particularly because maternal education is positively related to other attributes such as family background that might improve infant health.

The recent economics literature has made great strides in identifying the causal effect of maternal education. Two of the more frequently-exploited quasi-experiments are the construction of new schools and the expansion of compulsory schooling. In the U.S., the expansion of higher education through the building of new universities and colleges between 1940 and 1990 led to reductions in the rates of prematurity and low birth weight. Outside of the U.S., the construction of new schools in areas without schools has resulted in similar improvements in infant health. When interpreting these estimates; however, one should think about these two settings as possibly identifying different effects of education in the case that there are non-linear effects of maternal education.

Compulsory schooling reforms in the 20th century led to cohorts born close to one another to have different educational requirements. These compulsory schooling laws dictate when individuals can legally drop out of school. In countries where many individuals drop out at the minimum schooling age and the compulsory schooling laws are enforced, increases in the compulsory schooling

age are useful instruments for maternal education. In the U.S., the size of the population affected by compulsory schooling reforms is rather small. In contrast, in Britain, at least historically, most individuals drop out of school at the minimum schooling age. Thus, one can use regression discontinuity techniques where contrasts are made between individuals proximate in date of birth who might be otherwise identical except for their level of schooling. The British compulsory reforms generally point to no effects of maternal education on infant health.

The discussed quasi-experiments increase education by extending the end of schooling. Alternatively, an increase in educational attainment could be achieved by reducing the age at school entry. Increases in schooling via augmenting either the beginning or end of schooling could potentially estimate different effects of education. As for the latter, there could be a mechanical effect of extra schooling. Being in school longer may act as an incarceration effect, reducing rates of sexual activity and thus, result in delayed fertility.

This conceptual difference may be an explanation for the difference between the conclusions reached from using school entry policies and other studies. School entry policies impact the start of schooling. Despite their differences in acquired schooling, comparisons of individuals born before and after school entry dates (i.e., the date by which a child must have reached age 5 to enter school) show no evidence of effects of maternal education on infant health.

One difficulty often neglected in this literature is that an instrument for maternal education may affect both fertility and infant health. In the case that there are fertility effects of education, the measured effect of maternal education on infant health suffers from a selection problem.

Similar to that of maternal education, the effects of maternal age could be direct or indirect. Women at either end of the childbearing age spectrum experience worse infant health outcomes. Support of the biological effects of maternal age has been confirmed with animal studies, but maternal age may also influence the choice of prenatal and postnatal inputs. Women who give birth at earlier ages may not have the income or access to adequate medical care that older mothers do. Thus, the fact that maternal inputs vary with maternal age obfuscates the causal effect of maternal age. Specifically, women who give birth at younger ages are of lower socioeconomic status than women who give birth at older ages. Thus, the adverse impacts of giving birth at a younger age may be overstated in the cross-section while the opposite is true for older ages.

The main empirical evidence of the effects of maternal age comes from sibling-based compar-

isons. That is, one can compare the outcomes of children born to the same mother. Such an approach effectively controls for fixed differences (e.g., socioeconomic status which may be fixed) across mothers. However, to the extent that maternal age is correlated with other attributes that vary across a woman's lifecycle, these sibling contrasts will not capture solely the effect of maternal age. The sibling estimates do confirm the expected direction of biases - the effects of young maternal age are not as adverse as one would expect from correlations and the effects of advanced maternal age are worse than the cross-sectional correlations imply.

C Income

There is a well-documented, positive correlation between income and child health. Income is not a direct input into health production, thus the impact of parental income on child health must operate through either budgetary constraints or by shifting parental preferences. Higher-income parents can afford to purchase more food, health care, and safer environments for their children. Parental tastes for child health inputs may also vary by income, as evidenced by income gradients in smoking, drinking, and prenatal care.

The effect of income can operate through many channels and economists have distinguished between the effects of transitory and permanent income because each type may have a distinct impact on health outcomes. A temporary income shock (e.g., drought, famine, variation in rainfall) can have an immediate, one-time effect that lasts into adulthood, particularly if the shock occurs during gestation or just after birth. Permanent family income has a direct correlation with child health, with the impact of permanent income on health growing as children age into adulthood.

Disparities in health across socioeconomic groups are evident at birth. Low income children have a higher incidence of low birth weight, poorer reported health status, and higher rates of chronic conditions in childhood; however, there is little evidence that the impact of being low birth weight varies by SES. Researchers have documented an income-health gradient that steepens over time, indicating that the disparities in health between high and low income children grow with age. The hypothesized mechanism behind the steepening of the gradient is the prevalence of shocks experienced by low income children. Although a health shock does not differentially impact low income children, the higher frequency of shocks experienced by low income children causes the gap in health status to widen with age.

Temporary income shocks near the time of birth produce detectable effects on health in only some studies. Negative income shocks, such as the phylloxera infestation that destroyed forty percent of French vineyards between 1863 and 1890 and the Dust Bowl phenomenon in the American Midwest during the 1930's have been found to have minimal effects on health in adulthood. Individuals born in a phylloxera-affected region were shorter than their unaffected peers; however, other measures of population health were unchanged. Health in old age was also unaffected for individuals born in the Dust Bowl era. Positive income shocks as measured by rainfall improved the adult health, height, and completed education of females in Indonesia who were less than one year old during the increase in rainfall. No results were found for men or for rainfall shocks while the child was *in utero*, suggesting that improved outcomes for women during high rainfall years may be related to gender bias in nutritional intake during infancy.

Means-tested government transfer programs provide an exogenous, measurable income shock to eligible families and have been shown to improve child health. Mexico's randomized-controlled experiment of PROGRESA provides cash transfers to households that comply with required behaviors including prenatal care, medical checkups, meeting nutritional guidelines, and attending educational meetings. Although it is not possible to separate the impact of the income transfer from the other features of the program, children born into the program have lower rates of illness than control families, are less likely to be anemic, and are slightly taller than control children. Furthermore, the impact of the program increased the longer the family received PROGRESA transfers. In the United States, it is unclear whether cash transfers to families participating in the Aid to Families with Dependent Children Program increased infant birth weight, while maternal participation in the Food Stamp Program (comparable to an income shock) increased the birth weight of infants at the low end of the birth weight distribution.

Macroeconomic conditions at the time of birth are related to both health at birth and long-run health and the relationship appears to have changed over time. Research using data on individuals born in the Netherlands between 1812 and 1912 finds that babies born in boom years have lower mortality rates later in life and live longer than babies born in recession years. More recent data suggests that the relationship between macroeconomic conditions and child health may have reversed. In the United States, a higher unemployment rate is associated with improvements in birth outcomes such as incidence of low birth weight and post-neonatal mortality. During times of high

unemployment, maternal health behaviors (smoking and drinking) improve and different types of women select into motherhood, which may explain the improved birth outcomes. Although aggregate birth outcomes improve during times of high unemployment, the impact of a job displacement for an individual family negatively impacts infant health. Comparing children in the same family, children born just after a parental job loss have lower birth weight than siblings born prior to the job loss.

D Health Care

Prenatal care can improve infant health by identifying conditions that can harm health such as low weight gain and by providing health and nutrition information to the mother. While it is well documented by researchers that policy levers can improve rates of prenatal care utilization, it is still unclear whether increased prenatal care translates to better infant health. Examinations of Medicaid expansions yield mixed results, but other policy changes that increased care have resulted in improvements in birth outcomes. Access to prenatal care appears to improve birth outcomes for those most at risk for poor birth outcomes such as low-income women and minority women who would have otherwise had minimal or low-quality prenatal care. A primary mechanism through which prenatal care improves birth outcomes is to reduce maternal smoking, which is the leading cause of growth retardation for fetuses. Health care at the time of birth is associated with a decline in the neonatal mortality rate, likely a result of access to life-saving technology.

Public health insurance programs such as Medicaid in the United States and National Health Insurance (NHI) in Canada provide prenatal and delivery care with the goal of improving both infant and maternal health. Introduction of universal health insurance in Canada during the 1960's and 1970's reduced infant mortality by 4 percent and reduced low birth weight classification on average, with single mothers experiencing a substantial reduction in the incidence of low birth weight. In the 1980's and 1990's, Medicaid significantly expanded its eligibility threshold to include a larger share of low-income, pregnant women. The program expansion initiated cost-saving measures, changing the insurance structure from fee-for-service to managed care for some enrollees. Evaluations of the changes consistently show impacts on prenatal care utilization but yield differing results on birth outcomes, with some researchers concluding that the changes improved birth outcomes and others finding no effect. Physician incentives to provide care are influenced by the type of payment

structure Medicaid uses. Of particular interest is the relative incentives of Caesarian versus vaginal deliveries. Reduced incentives to provide care have been shown to increase the probability of low birth weight, prematurity, and neonatal mortality; however, studies that examine increased incentives to provide care find no effect on infant health.

The 1964 Civil Rights Act mandated desegregation of hospitals and greatly improved the quality of prenatal care available to blacks, particularly in the southern United States where hospitals for non-whites were of poor quality. Desegregation reduced post-neonatal mortality rates with gains driven by reductions in preventable deaths from pneumonia and gastroenteritis. The health of infants at birth also improved, as evidenced by reduced incidence of low birth weight and improved APGAR scores for the cohort born after desegregation. The narrowing of the black-white test score gap in the 1980's can be traced back to improved health of black cohorts born after desegregation, indicating that access to care that improved birth outcomes translated to increased human capital development later in life.

Another way to identify whether increased care translates to better outcomes is to examine infants on either side of the 1,500 gram very low birth weight classification. Infants below 1,500 grams receive more intense care than infants just above the threshold, resulting in lower mortality rates for infants classified as very low birth weight. In line with the findings that improved care after desegregation increased the test scores of black children, very low birth weight infants just below 1500 grams who received additional care outperform their peers with birth weights exceeding 1500 grams.

E Maternal Behaviors

Negative correlations between income and behaviors such as smoking, drinking, and drug use suggest that these habits may be a possible mechanism for transmission of health to infants. The decision to drink or smoke may be related to other maternal behaviors or characteristics that could affect infant health; therefore, an extensive set of control variables or a natural experiment that changes smoking behavior independent of maternal characteristics is necessary to isolate the impact of these behaviors outcomes such as birth weight and infant mortality. Numerous studies have linked maternal drinking and smoking with reduced infant health and long-term human capital outcomes.

i Alcohol

In a survey of Danish mothers who had recently given birth, women who reported drinking four or more drinks per week while pregnant were more likely to have a preterm delivery than women who reported drinking no alcohol. However, this worse birth outcome could be caused because women who choose to drink during pregnancy are negatively selected on other attributes. Accordingly, there has been a shift to the use of quasi-experimental approaches to unraveling the alcohol and child outcome relation.

Variation in the legal drinking age across states and over time has been used to identify the causal effect of maternal drinking on infant health. A lower drinking age is associated with more alcohol consumption during pregnancy, an increase in premature births, and an increase in the probability of low birth weight. The reduction in health at birth can partially be attributed to changes in the composition of births, increasing the number of births without a father listed and suggesting that more unplanned pregnancies occur when drinking laws are less stringent.

Maternal alcohol consumption can have long term effects on human capital development, as demonstrated by a policy experiment in Sweden. In 1967, grocery stores in certain regions were temporarily allowed to sell strong beer that was previously only available in government-run liquor stores. Children exposed the longest to the policy while *in utero* had lower completed education, lower earnings, and higher rates of welfare participation than children that were not exposed to the policy experiment.

ii Smoking

Smoking during pregnancy increases health risk for both the mother and infant in the form of complications such as miscarriage, membrane ruptures, ectopic pregnancy, pneumonia, and still-birth. Women who smoke during pregnancy have lower birth weight babies on average and are at a greater risk for having an infant classified as low birth weight. The seminal study of the impact of smoking on infant health is the randomized-controlled trial of Sexton and Hebel (1984), in which pregnant smokers were randomized into a treatment group receiving assistance quitting smoking and a control group receiving no intervention. Babies whose mothers were in the treatment group were on average 92 grams heavier than control group babies.

The 1964 Surgeon General Report on Smoking and Health alerted the nation to the health hazards of smoking resulting in a reduction in smoking among pregnant women that was concentrated among higher-educated mothers. A study comparing birth outcomes of children before and after the release of the Surgeon General Report reveals that higher smoking rates are associated with lower birth weight. However, no effect of smoking was found on gestation, prematurity, or the likelihood of having a low birth weight baby. These results are similar to studies that use increases in cigarette excise taxes to estimate the impact of smoking on birth weight.

F Nutrition

From famines in developing countries to supplemental nutrition programs in developed ones, studies consistently conclude that nutrition is a fundamental input into health production, impacting both short and long run health. Randomized-controlled trials that offer nutritional supplements to the treatment group have demonstrated that micronutrients play a key role in cognitive development. Assessing the direct impact of nutrition on health is difficult due to significant measurement error in the nutritional content of food items; therefore, most natural experiments examine how quantity of food relates to health outcomes. Research suggests that policies that improve the nutrition of pregnant women and infants will be effective at improving the health and human capital of the next generation.

The ideal setting for conducting research is the randomized-controlled trial, a technique that has been used in developing countries to study the impact of poor nutrition on cognitive development. In Jamaica, babies that were given nutritional supplements had higher mental development than the control group, indicating that lack of nutrition is a causal factor in stunted mental development. Children in Guatemala who received a nutritional supplement tested higher on knowledge, numeracy, reading, and vocabulary assessments than children given a placebo. The same children were followed up with as adults. Adults who were treated with the nutritional supplement as a child had higher reading comprehension, nonverbal and cognitive scores, and higher completed education (women only) than the control group.

The majority of economic research on nutrition in developing countries studies the impact of famines on health, education, and labor market outcomes. Famines are extreme events and estimating the impact of a famine can be confounded by selection because only survivors are

observed. Furthermore, the health effects of a famine may not solely operate through nutritional deprivation - famines may affect other inputs to health and human capital such as disease resistance and school attendance. The Chinese Famine of 1959 to 1961 had a significant impact on children and babies *in utero* during the event. Children exposed *in utero* were shorter, lighter, and acquired fewer years of education than children born just before and after the famine. Exposure in early childhood had a detectable, yet smaller effect on long term outcomes than *in utero* exposure. The famine also tilted the sex ratio in favor of girls, reduced the literacy rate, reduced employment, and reduced the marriage rate for children born during the time of the famine.

European famines during World War II had long term impacts on health and human capital accumulation for individuals exposed early in life. Individuals who were *in utero* during the Dutch Famine experienced higher rates of chronic disease in adulthood. Children exposed to the Greek Famine during gestation and the first two years of life showed reduced educational attainment and literacy, with the largest impacts on children who were 0 to 12 months old during the famine. The impact of a famine can reach late into life - men exposed *in utero* to the Dutch Potato Famine of 1846 to 1847 had a lower life expectancy at age fifty than cohorts born just after the famine.

Controlled nutritional deprivation for brief periods of time is associated with reduced physical and cognitive development, as evidenced by recent research into the outcomes of children *in utero* during Ramadan. Ramadan occurs for one lunar month per year and observance includes fasting between sunlight and sunset. In a study using data from the United States, Iraq, and Uganda, the authors document reduced birth weight, reduced gestation length, a decline in male births, reductions in educational attainment, and even increased rates of mental disabilities for children of Arab mothers *in utero* during Ramadan.

Even in developed countries, nutrition interventions can positively impact the birth outcomes of at-risk children as evidenced by analyses of the United States' Supplemental Nutrition Program for Women, Infants, and Children (WIC). WIC is aimed at low-income pregnant women and women with young children with the goal of improving the nutrition and health of this group. Consistently estimating the effect of WIC participation on infant health is difficult due to non-random selection into the program - unobserved maternal characteristics that affect infant health may be systematically different for mothers that choose to enter the program than for mothers who do not. Estimates that account for selection into the program yield a positive impact of WIC participation

on birth outcomes such as incidence of low birth weight and gestation length. Infants at the low end of the socioeconomic and birth outcome distribution gained the most from WIC.

G Environment

Environmental quality can be considered a direct input into health, with infant health responding to maternal exposure to pollution while *in utero* as well as post-birth. Isolating a causal relationship between pollution and health is challenging for many reasons. First, measurement error in pollution levels attenuates coefficients and makes a relationship difficult to detect. Second, there are numerous pollutants, many of which are measured infrequently or not at all. Lastly, a number of confounding variables must be ruled out in order to interpret a relationship between environmental quality and health as causal. For example, families may sort into areas of varying pollution levels based on socioeconomic characteristics or business cycles may have an independent effect on both pollution levels and health. Furthermore, the relationship between health and pollution may be nonlinear meaning, that reductions in pollution below a given level may not improve health.

Researchers have exclusively relied on quasi-experimental designs such as policy changes or temporal variation in pollution levels to assess the impact of environment on infant health. The introduction of the Clean Air Act of 1970 reduced infant deaths in the most polluted counties. Similarly, infant mortality declined more in counties with greater reductions in total suspended particulates during the 1981 to 1982 recession. The introduction of the EZPass toll system in the Northeastern United States reduced traffic and thus pollution levels near the freeway, subsequently increasing birth weight and reducing prematurity for newborns near the freeway. The Chernobyl fallout over Sweden did not detectably affect infant health; however, students that were *in utero* during the fallout experienced deficiencies in human capital as evidenced by lower test scores and high school graduation rates.

V Conclusion

From both a theoretical and empirical perspective, there has been an increasing focus on the importance of *in utero* and early life conditions on later health and outcomes. Theoretical models emphasize the timing of investments. If investments are substitutable across periods, then dis-

advantage early in life can be overcome by later life investments. However, early investment is important if skills acquired during early periods can help beget skills in later periods. This chapter highlights several mechanisms through which transmission of health may occur - initial endowments, environmental influences, parental abilities, and investments. Researchers have relied heavily on quasi-experimental strategies such as policy changes, natural disasters, and sibling studies to identify a causal relationship between early life influences and health. We think of this as an emerging and growing literature.

Further Reading

Almond, Douglas and Janet Currie, "Human Capital Development Before Age Five," *Handbook of Labor Economics*, 2011, *4*, 1315–1486.

_ and _ , "Killing Me Softly: The Fetal Origins Hypothesis," *The Journal of Economic Perspectives*, 2011, *25* (3), 153–172.

Barker, David, "The Developmental Origins of Adult Disease," *Journal of the American College of Nutrition*, 2004, *23* (suppl 6), 588S–595S.

Currie, Janet, "Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development," *Journal of Economic Literature*, 2009, *47* (1), 87–122.

_ , "Inequality at Birth: Some Causes and Consequences," *National Bureau of Economic Research Working Paper No. 16798*, 2011.

Heckman, James, "The Technology and Neuroscience of Capacity Formation," *Proceedings of the National Academy of Sciences (PNAS)*, 2007, *104* (33), 13250–13255.

Sexton, Mary and J. Richard Hebel, "A Clinical Trial of Change in Maternal Smoking and Its Effect on Birth Weight," *The Journal of the American Medical Association*, 1984, *251* (7), 911–915.