From Infant to Mother: Early Disease Environment and Future Maternal Health

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Economists are increasingly interested in understanding the way that early life affects the future accumulation of human capital. This work has been spurred by research demonstrating that conditions in early life can have persistent and profound impacts. For example, Currie and Thomas (2001) use data from the 1958 British Birth Cohort study and find that 4 to 5% of the variation in employment at age 33, and as much as 20% of the variation in wages can be predicted using test scores at age 7. Cunha et al. (2010) estimate structural models in which initial endowments and investments feed through to later outcomes; they arrive at estimates that are of a similar order of magnitude to those of Currie and Thomas. To put these results in context, labor economists generally feel that they are doing well if they can explain 30% of the variation in wages in a human capital earnings function.

The literature on early life influences can be broken into several strands. One strand examines prenatal, or "fetal origins." This research has recently been surveyed by Almond and Currie (2011b), and Currie, (2011). A second strand focuses on health conditions in childhood. This literature is surveyed in Elo and Preston (1992), and more recently, in Almond and Currie (2011a). Both strands suggest that health in early life has long term consequences on education, earnings, and on future health. An interesting issue highlighted by Smith (2009) is whether the effects on employment and earnings work mainly through effects on health, or whether there might be direct effects on, for example, education. A third strand of the literature asks whether poor health in the mother can be "transmitted" intergenerationally to the child (c.f. Currie and Moretti, 2007).

All of this research has been limited by the lack of comprehensive data that links the early environment of the mother to her future outcomes and those of her child. This paper exploits a previously little tapped source of such information: U.S. vital statistics natality and

mortality records. In addition to providing information on infant health, U.S. birth records have information on a number of outcomes for mothers, such as the presence of health conditions like diabetes and hypertension, educational attainment and marital status, and behaviors such as smoking and weight gain during pregnancy. The birth records also contain mothers' state of birth and age, so women can be linked back to measures of the disease environment in their state and year of birth in order to explore the impact of the early life disease environment on future outcomes.

We use these data to compare the effects of the disease environment at three different points in time: the year before the mother's birth, the mother's birth year, and the year after the mother's birth. Our data allow us to focus on a particularly important population—future mothers—and to examine the impact of maternal exposure on maternal health, maternal education and behaviors, and on child outcomes.

Our results suggest that exposure to disease in early childhood significantly increases the incidence of diabetes in the population of future mothers. The exposed mothers are less likely to be married, have fewer years of education, and are more likely to gain over 60 pounds or smoke while pregnant. Not surprisingly then, exposure increases the probability of low birth weight in the next generation, at least among whites. Among whites, this effect remains when we control for maternal behaviors as well as disease exposure. Among blacks, we find that maternal exposure reduces the incidence of low birth weight. The difference between whites and blacks may reflect a "scarring" vs. selection story similar to Bozzoli et al. (2009); whites who go on to have children are negatively impacted, while blacks who go on to have children are positively selected having survived a higher early childhood mortality rate. The rest of the paper is laid out as follows. Section II provides a brief overview of some relevant background. Section III

discusses our data and Section IV provides an overview of our methods. Section V presents the results, and Section VI concludes.

II: Background

In this section, we provide a brief overview of some of the literature on fetal origins, the effects of the disease environment in early childhood, and evidence regarding intergenerational transmission of maternal health shocks.

a) Fetal Origins

The fetal origins hypothesis holds that conditions *in utero* "program" the developing fetus in ways that can be manifested in future disease. For example, Barker (1998) argues that inadequate nutrition *in utero* leads to metabolic changes that in turn are associated with future heart disease. From the point of view of economists, an important implication of this literature is that even conditions that are present at birth reflect "nurture" and its interactions, rather than "nature" alone.[1]

Other research focuses on the possible effects of *in utero* exposure to diseases such as influenza. Almond (2006), Nelson (2010), Neelson and Stratmann (2010), and Lin (2011) study the 1918 flu epidemic and identify the effects using breaks from trend in the affected cohorts relative to cohorts born just before and after the outbreak. Almond (2006), Neelson and Stratmann (2010), and Lin (2011) further incorporate geographic variation in the intensity of prenatal influenza exposure. They use acute respiratory disease mortality (ARD) or maternal mortality in the location of birth as proxies for the intensity of infection. Identification is aided by the fact that the flu epidemic was an intense shock of relatively short duration.

[1] For a recent, engaging account of the limitation of the nature versus nurture distinction and, in particular, its limited policy relevance, see Manski 2011.

Almond (2006) and Nelson (2010) show that *in utero* exposure to influenza has long-term implications for education and labor market outcomes. The 1918 US cohort had 0.125 fewer years of schooling, earned 5% lower wages, and were 20% more likely to be disabled. The 1918 Brazilian cohort that was most exposed to the pandemic *in utero* finished 0.2 fewer years of schooling, had 20% lower wages, and were 20% less likely to be employed. Comparing Almond (2006), Neelson and Stratmann (2010), and Lin (2011) shows that, at least for years of schooling, the effects of the 1918 influenza epidemic were larger in Taiwan and the US than in Switzerland. On a base high school completion rate of 50.9%, influenza-exposed individuals were 5.89% less likely to complete high school (with larger estimated effects when national time-series variation is used).

Kelly (2011) investigates the effects of the 1957 Asian flu pandemic on British children's test scores at ages 7 and 11. Using regional variation in the number of pneumonia cases, she finds that for each 1/1,000 increase in epidemic notifications, test scores decreased 0.15 and 0.10 standard deviations for children ages 7 and 11, respectively. While this paper shows that influenza exposure *in utero* affected children's test scores, it is less clear how these effects map into adult outcomes. Moreover, 0.10 of a standard deviation, while comparable in magnitude to many conventional educational interventions (Dobbie and Fryer, 2011), is quite a small change. It is possible that the effects of the 1957 flu were smaller than those of the earlier flu pandemic because of differences across flu strains, improvements in medical care, or changes in the underlying health of those affected.

Other work by economists focuses on measures of health at birth, typically birth weight, and examines implications for future outcomes. For example, Black et al. (2007) use administrative data from Norway, where it is possible to link birth data to information about

adult education, earnings, IQ, and height (the latter two outcomes being available only for men). They find that a 10% increase in birth weight would increase future earnings, the probability of finishing secondary school, and IQ by approximately 1%, with smaller effects on height.[2]

Health at birth appears to be quite malleable. For example, economists have also been active in quantifying the negative effects of maternal smoking and drinking on the fetus (Currie and Gruber, 1996; Evans and Ringel, 1999; Noonan et al., 2007; Currie and Neidell, 2005; Currie et al. 2009; Fertig and Watson, 2009; Ludwig and Currie, 2010). Currie et al. (2009) use confidential data from birth certificates on 1.5 million births in New Jersey between 1989 and 2003 to identify siblings born to the same mother. . They compare sibling pairs in which the mother smoked during one pregnancy but not during the other. These sibling fixed effects estimates of negative effects of smoking on birth weight are smaller than ordinary least squares (OLS) estimates, but are still substantial: At the mean number of cigarettes smoked per day (ten), they estimate that smoking increases the probability of low birth weight (defined as birth weight less than 2,500 grams) by .018 percentage points on a baseline of .089 (compared to an OLS estimate of .067 percentage point increase in low birth weight).

The introduction of social programs such as the Supplemental Feeding Program for Women, Infants, and Children (WIC) and Food Stamps in the 1970s (Hoynes et al. 2009; Almond et al., 2011) have also been shown to affect birth weight, presumably by improving nutrition, but perhaps also by reducing susceptibility to disease.[3]

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[2] Behrman and Rosenzweig (2004) find that a 1 pound increase in birth weight leads to 7% higher wages and 0.66 additional years of schooling. These estimates are based on within-twin fixed effect estimates from a sample of twins from the Minnesota Twin Registry from 1983-1990.

[3] Additionally, Currie and Moretti (2003) show that increases in the availability of college education improved birth weight. Subsequent studies using laws affecting the compulsory schooling of high school educated mothers have not shown positive impacts on birth weight (Lindeboom et al., 2009; McCrary and Royer, 2011). Conti et al., (forthcoming) reconcile these findings using data from the 1970 British Cohort

b) Childhood Health and Future Outcomes

One measure of child health that is consistently recorded around the world is infant mortality.[4] While infant mortality is obviously important in its own right, it is also important to consider because it affects selection into the sample of surviving infants (Almond, 2006; Bozzoli et al., 2009). Selection of survivors affects the estimated return to health at birth because children in poor health at birth are both more likely to die in early childhood and to be in poorer health and earn lower wages as adults. Since children who die are excluded from the sample, the estimated effect of health at birth on earnings is likely to be biased downward, with the magnitude of the bias increasing in the baseline infant mortality rate.[5] As infant mortality decreases, the estimated return to health at birth increases, and average outcomes in the population may actually decline as more unhealthy children live to adulthood.

Infant mortality, and specifically post-neonatal mortality, is also used as a marker for the early disease environment of survivors. The idea is that cohorts that had a lot of infant deaths from disease, also presumably had a lot of survivors who were exposed to disease. The reason for focusing on post-neonatal mortality, which is mortality between one month and twelve months of age, is that a large share of mortality among infants under one month is due to causes which are present at birth and thus reflect many aspects of the prenatal environment (UNICEF: Child Health Epidemiology Reference Group). In contrast, infectious diseases have historically accounted for a large proportion of infant deaths after the first month (as they continue to do in developing countries today). For example, in 1970, 63% of U.S. infant deaths were accounted

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Study. They show that the women most likely to select into higher education have higher returns to education in terms of both wages and smoking behavior.

[4] It will, however, be sensitive to the designation of live birth versus stillbirth, see Almond and Currie, 2011a.

[5] This is true when the focus is on mean shifts in the unobserved distribution of health, but not when mortality changes are due to movements in the infant survival threshold (Almond, 2006).

for by causes that would have been present at or around the time of birth such as prematurity, congenital anomalies, birth asphyxiation, and other causes. Of the remaining 37%, 22% of deaths were due to pneumonia and influenza (Singh and Dyck, 2010). Pneumonia in turn is a common side effect of diseases such as measles. By 2007, only 1% of a much smaller number of deaths were due to either pneumonia or influenza.

Crimmons and Finch (2006) show that childhood mortality is positively (negatively) associated with adult mortality (adult height) in four European countries for cohorts born in the 19th Century. They posit that the relationship is driven by the prevalence of infections and inflammations in childhood. Bozzoli et al. (2009) find that post-neonatal mortality in the year of birth accounts for 30 to 60% of the cross-cohort and cross-country variation in adult height over the period 1950-1980 in 11 European countries and the U.S. Height, like birth weight, is strongly associated with adult outcomes (Case and Paxson 2008, 2010; Schick and Steckel 2010; Strauss and Thomas 1998).

Case and Paxson (2009) consider improvements in infant mortality rates from 1900 to 1950 by U.S. state and find that higher infant mortality rates experienced in early childhood were associated with cognitive decline among the elderly. This was especially true of early childhood mortality from typhoid, influenza and diarrhea. Chay et al. (2009) consider declines in the black-white test score gap, which they link to improvements in black access to hospitals and reductions in black post-neonatal mortality during late 1960s and early 1970s.

c) Intergenerational Transmission of Maternal Health Shocks

While the papers discussed above emphasize the importance of early life health for future adult outcomes, health shocks in the early life of women may also affect the health of their future children. For example, Costa (1998) argues that much of the inequality in birth weight observed

over the course of the 20th Century may have been due to differences in mothers' early health endowments.

It has been known for some time from animal studies that environmentally induced changes in the epigenome can be transmitted from parents to offspring. For instance, Stewart, et al. (1980) starved pregnant rats and found that it took several generations for the descendants of the starved rats to return to the size of the control, non-starved rats, even when all descendants shared the same diet. Still, people are not rats, so it is necessary to investigate possible intergenerational linkages using data on human populations.

Currie and Moretti (2007) look for interactions between maternal conditions and infant health at birth using a large sample of sisters drawn from California birth certificates from the 1960s to the 1990s. For mothers who were born in California during that interval, it was possible to go back and find their birth certificates, and to use information on it to identify mothers who were sisters. Thus, there is some information about both an infant's birth weight and the mother's birth weight, as well as information about maternal circumstances at the time of her infant's birth, and at the time of her own birth.

Sister comparisons using this data set show that a sister born in poor circumstances (proxied by being born in a poor zip code) was more likely than a sister born in a higher income zip codeto be low birth weight. They also show that women who were low birth weight were more likely than their sisters to have low birth weight babies and that a woman who was low birth weight was more likely to "transmit" low birth weight to her own child if she was residing in a high poverty zip code at the time of the child's birth. What these results indicate is that like height, low birth weight is transmissible for reasons that are not purely genetic; low adult

socioeconomic status appears to compound the negative impact of maternal low birth weight, and makes it more likely that the child will also be low birth weight.

Almond and Chay (2006) build on previous work showing that the Civil Rights movement had a large effect on the health of black infants in some southern states, especially Mississippi, due to the desegregation of hospitals and increased access to medical care (Almond et al., 2006). 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) regardless of their state of residence as adults, and to compare the outcomes of their infants to the outcomes of infants born to black women in the 1961 to 1963 birth cohorts. The birth outcomes of white women in the same cohorts are examined as a control. Almond and Chay (2006) conclude that the infants of black women who had healthier infancies as a result of the Civil Rights movement and War on Poverty 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.

Our study builds on the preceding literature by examining a national sample of women born between 1961 and 1990; by examining the impact of early disease environment on the mothers' adult health, socioeconomic status, and behaviors, at the time they give birth themselves; and by examining the effects of early maternal disease environment on the health of the infants born to affected women.

III. Data

Our primary data on outcomes come from the 1989-2006 Vital Statistics natality microdata, which are derived from the birth certificates collected by the states and filed with the National Center for Health Statistics. These data provide detailed information on maternal health, demographics, and birth outcomes for the universe of births occurring each year in the United States, approximately 3.5 to 4 million births per year.

Beginning in 1989, the standard birth certificate collected information on whether the mother had any medical risk factors for the pregnancy, a number of which can be interpreted as indicators of maternal health. The 1989 revision asked whether the mother suffered from any of 17 different conditions, including anemia, cardiac disease, lung disease, diabetes, genital herpes, amniotic fluid problems, blood disorders, chronic or pregnancy-associated hypertension, eclampsia, incompetent cervix, previous large or preterm deliveries, renal disease, Rh sensitization, uterine bleeding, or other medical risk factors. In 2003, the standard birth certificate was revised again, and the set of medical risk factors collected was reduced from 17 to 10 and changed; while the 2003 revision continued to ask about diabetes, chronic or pregnancy-associated hypertension, eclampsia, and previous preterm deliveries, it replaced the remainder of the conditions on the list with items on other previous poor pregnancy outcomes, previous C-sections, and infertility treatment.[6] Because the timing of the adoption of both the 1989 and 2003 revised birth certificates varied by state, information on medical risk factors are unavailable for some states during some years of our sample, although reporting of the medical risk factors

[6] Infertility treatment was counted as two separate risk factors, since it is composed of one indicator for use of fertility-enhancing drugs, artificial insemination, or intrauterine insemination, and one indicator for the use of assisted reproductive technology (e.g., in vitro fertilization (IVF), gamete intrafallopian transfer (GIFT)).

we study generally improves over time. To capture these differences in reporting over time, our specifications include child's state of birth by year of birth fixed effects. [7]

We focus on diabetes and hypertension, which previous research suggests may be caused by health insults experienced *in utero* or in early childhood. Since chronic hypertension, pregnancy-associated hypertension, and eclampsia are all characterized by high blood pressure, we combine them into a single outcome for "hypertension," which we set equal to 1 if the mother suffers from any of these conditions; these conditions are all listed under the heading "Hypertension" on the 2003 revised birth certificate and are primarily distinguished by when they develop (i.e., before or after 20 weeks gestation) and the presence of other symptoms (i.e., protein in the urine and seizures).

We also analyze infant's birth weight as an outcome, since it is highly influenced by maternal health, and has been shown to be a significant predictor of the child's long-term outcomes. The child's birth weight is our primary measure of the extent to which health insults suffered by the mother are transmitted to the child.

Along with the medical risk factors for the pregnancy, the natality data also provide information on the infant's month and year of birth, parity (birth order), and plurality, as well as the mother's race and ethnicity, age (in years), educational attainment, marital status, smoking behavior, weight gain during pregnancy, and place of birth. For mothers who were born in the U.S., the data report the U.S. state or territory of birth. Since the data do not include the mother's year of birth, we construct this variable using the mother's age and the month and year the child was born; the mother's year of birth is constructed as the child's year of birth minus the mother's age minus one if the child was born between January and June, and as the child's year

[7] These fixed effects are for the state of occurrence of the child's birth, as opposed to the state of residence of the child, since the state of occurrence determines which version of the birth certificate is used.

of birth minus the mother's age if the child was born between July and December. This construction is more likely to match the mother's actual year of birth than simply using the difference between the child's year of birth and the mother's age, because on average, mothers are their reported ages plus one-half, and over the calendar year, births occur on average at the beginning of July.[8]

We use mothers' race, place, and year of birth to link them to our measures of the *in utero* and early childhood disease environment: the post-neonatal mortality rates (number of deaths between 28 days and 1 year of age per 1,000 live births) for their race in their state and year of birth. These measures of infant mortality also come from National Center for Health Statistics (NCHS) data. We calculate the number of deaths between 28 days and 1 year for each state, year, and race using the 1960-1991 mortality microdata, and we obtain the number of live births for each state, year, and race from NCHS publications for years between 1960 and 1968, and from the natality microdata afterwards. We focus on death rates for African Americans and whites, since the "others" have not been consistently identified historically.

Figures 1 to 5 plot the post-neonatal mortality rates for all, whites, and blacks from 1960 to 1990 for five large states. These figures show that there is considerable variation both in the levels, and, more importantly for our identification strategy, in the timing of changes in the rates across states. It is also interesting to note that the peaks (deviations from trend) in the graphs

[8] To see this, consider the following example of three women born on three dates in 1960: January 1, July 1, and December 31. If they give birth between January and June 1990, when they are the ages of 30, 29, and 29, our mechanism assigns them the birth years 1959, 1960, and 1960, respectively. Using the difference between the child's birth year and maternal age assigns 1960, 1961, and 1961. If they give birth between July and December 1990, when they are 30, 30, and 29, both mechanisms assign them the birth years 1960, 1960, and 1961. This suggests that our mechanism is more likely to match the mother's birth year. For verification, we conducted simulations using all pair-wise combinations of three years of birthdays for mothers and three years of birthdays for children. The simulations suggest that our assignment mechanism matches the mother's actual year of birth 75% of the time, compared to 50% of the time for the difference between the child's birth year and maternal age. This imputation method is also used in Devereux and Hart (2010).

correspond well to known epidemics, especially of measles.[9] While the trend in measles cases was sharply downwards after a vaccine was introduced in 1963, there were peaks in the number of cases nationally in 1962, 1965, 1971, 1977, and 1989-1990 (Wood and Brunell, 1995). There were also local measles epidemics, for example, in Chicago in 1983 (Bennish et al., 2011). Epidemics of flu likely also played a role in generating the variation in our data. The tail end of the "Asian flu" pandemic played itself out in 1963, while there were national outbreaks of "Singapore flu" and "Bangkok flu" in 1979-80 and 1980-81 respectively (Glezen et al., 1987).

Since our measures of the disease environment begin in 1960, we restrict our analysis to white and black women born in the U.S. between 1961 and 1990. We restrict our analysis to first births since the number of births may be affected by the woman's health and circumstances (and we are unable to identify repeated observations on the same woman across sibling births). We also exclude from our analysis mothers who are younger than 16 or older than 40 years old, as well as women who had multiple births, which result in higher rates of complications.

After making these sample selections, we aggregate the data into cells based on the mother's state and year of birth, age, and race, and the child's state and year of birth. We aggregate the data both to reduce the size of the dataset, which is very large, and because variation in infant mortality rates occurs at the level of the mother's race, state and year of birth. Since the availability of our health outcomes varies, we conduct this aggregation separately for each outcome, and during estimation, weight our models by the number of births in each cell.

Table 1 provides an overview of our data. Overall, 2.2% of women have diabetes, and 6.5% have hypertension, making these fairly common risk factors for pregnant women. In terms of behaviors, 7.8% of women gained too little weight (less than 16 pounds), while 2.9% gained

too much weight (more than 60 pounds). A little over 12% of women smoked during the pregnancy, 58.6% of mothers were married at the time of the birth, and the incidence of low birth weight in the infants of the sample mothers was 6.8%. The post-neonatal mortality rate was 4.8 per 1,000, with a standard deviation of 2.3. Our identification is based on deviations from trend, and once we control for maternal state of birth dummies and state-specific trends, this standard deviation falls to 0.7, a 70% reduction. The next two columns of Table 1 show means separately for women with above the median post-neonatal mortality rates, and for cells with below the median post neo-natal mortality rates at the time of their own birth. Women born into areas with below the median post-neonatal mortality rates have a lower incidence of diabetes and hypertension, and their infants have better birth outcomes. The women are also less likely to gain less than 16 pounds during the pregnancy, but more likely to gain over 60 pounds. Given the construction of our sample, these mothers are younger on average, and are less educated, more likely to smoke, and less likely to be married at the time of the birth. Thus, they achieve better health outcomes despite these health behaviors.

Table 1 shows that there are surprisingly small differences in the incidence of diabetes and hypertension by race; this may be accounted for by the fact that black mothers are almost three years younger on average and much less likely to smoke. However, black mothers are more than twice as likely as white mothers to gain too little weight during the pregnancy, and their infants are twice as likely to be low birth weight. Black mothers were also exposed to consistently worse post-neonatal environments: The mean post-neonatal mortality rate for black mothers was 8.7 per 1000.

While provocative, these means suggest that it is important to control adequately for the strong trends in the data that are related to maternal age.

IV: Methods

To examine the relationship between the fetal and early childhood disease environment and adult health, we estimate the following linear probability model:

$$Y_{rstcba} = \alpha + PNMR_{rst}\beta + X_{rstcba}\gamma + \delta_s + \pi_{cb} + \theta_a + \delta_s * t + \theta_a * t + \varepsilon_{rstcba}$$

where Y_{rstcba} is the average outcome for mothers of race *r* who were born in state *s* in year *t* and who gave birth in year *c* in state *b* at age *a*; $PNMR_{rst}$ is a vector of the post neonatal mortality rates for infants of race *r* in the mother's state and year of birth, and X_{rstcba} is a vector of control variables (i.e., fraction Hispanic, fraction Hispanic missing, an indicator for black). The model also includes mother's state of birth fixed effects (δ_s), child's year of birth by child's state fixed effects (π_{cb}), maternal single year age fixed effects (θ_a), mother's state of birth linear time trends (δ_s**t*), linear time trends in the maternal age fixed effects (θ_a**t*), and a random error term (ε_{rstcba}).

While there are many possible ways one could specify the vector of post neonatal mortality rates, we specify it as the race-specific post-neonatal mortality rate for the state in the year before, year of, and year after the mother's birth to capture the disease environment *in utero*, during the birth year, and in the following year. We estimate models with all three of these variables, as well as models with one post-neonatal mortality rate at a time. Our focus on the disease environment prior to the age of 1 follows previous literature on early life health in that it implicitly assumes that the disease environment has larger effects on fetuses and infants than on toddlers. This would be the case if, for instance, infants who survived a disease acquired some immunity to the disease.

The inclusion of mother's state of birth fixed effects helps to account for any persistent differences in outcomes between mothers born in different states, and the child's state by year of

birth fixed effects control for contemporaneous factors in the state that affect all births (such as the quality of medical care), as well as for reporting differences caused by the staggered adoption of the revised birth certificates. We include maternal age fixed effects to control for the well-known relationship between maternal age and health, and include maternal age linear time trends to allow for this relationship to change over time. We also include mother's state of birth linear time trends, so that all effects of the post-neonatal mortality rates, which are falling over time, are identified from departures from trend, which we think of as corresponding to disease outbreaks.[10] We cluster standard errors by mother's state of birth.[11]

We also estimate similar models with child outcomes rather than maternal outcomes. These models are estimated with and without controls for maternal characteristics and behaviors including smoking, educational attainment, marital status, and weight gain during pregnancy. These characteristics and behaviors may themselves have been impacted by the mother's early life circumstances. When we include them in the model, we are asking whether the mother's early life circumstances seem to affect the child through these pathways.[12]

IV: Results

[10] While we cannot be sure that all of the breaks in trend for PNMR are driven by disease, the correspondence between the patterns in Figures 1 through 5 and well-known disease trends is encouraging. Concern that these breaks might be driven by changes in composition, rather than by disease, also motivates our decision to include controls for maternal age and behaviors in our specifications.

[11] We cluster standard errors by mother's state of birth to deal with within-state serial correlation. This yields more conservative standard errors than clustering by mother's state and year of birth. For computational ease, we estimate this model in two stages: First, we obtain residuals from the regressions of the outcome or the post-neonatal mortality rates on all other control variables except for the mother's state of birth fixed effects. Second, we regress the residuals for the outcome on the residuals for the post-neonatal mortality rate and include mother's state of birth fixed effects and cluster the standard errors at the level of the mother's state of birth. We have verified that this approach obtains the same result as estimating these regressions in one stage.

[12] However, we recognize that we may also have introduced some bias due to endogenous controls, see Angrist and Pischke, 2009.

Table 2 shows estimates of the effects of the PNMR on the incidence of diabetes and hypertension among women with first births. In panel 1, the variables for $PNMR_{t-1}$, $PNMR_t$, and $PNMR_{t+1}$ are entered together; panel 2 shows estimates from three separate models, each of which controlled for one of the three PNMR variables, in each column. Table 1 suggests that a higher PNMR in the year after birth is associated with a significantly elevated risk of diabetes overall, and in both white and black women. The estimated coefficient is more than twice as large for blacks as for whites. When the three PNMR variables are entered separately, panel 2 shows that the estimated effects are still largest for PNMR in the year after birth, suggesting that despite the fetal origins literature, the fetal environment offers some protection against the effects of disease relative to the post-birth environment.

Relative to the mean prevalence of the incidence of diabetes, these estimates suggest that an additional post-neonatal death per 1,000 in the year after the mother's birth is associated with a 1.8% higher probability of having diabetes during her own first pregnancy at the mean age of 23.7. The elevation in risk is actually much greater for black mothers, who experience a 3.5% increase in the probability of diabetes during pregnancy for a one unit increase in the $PNMR_{t+1}$, compared to white mothers who experience an increase of 1.4% for a one unit increase. Moreover, Table 1 shows that there is a large difference between the mean $PNMR_{t+1}$ for whites (4.0) and the mean $PNMR_{t+1}$ for blacks (8.4) which suggests that the early life disease environment increased the probability of diabetes during pregnancy by almost 30% for blacks compared to 5.6% for whites (to the extent that overall PNMR rate captures a similar treatment effect as deviations from state-specific linear trends).

Table 3 examines the effects of the early disease environment on measures of the mother's socioeconomic status and behaviors. This table is striking in that a worse disease

environment in early childhood predicts uniformly worse outcomes. Moreover, unlike Table 2 in which only exposure in the year after birth seemed to matter, Table 3 suggests that exposure in the year before birth and the year of the birth also matter, at least for some outcomes.

On average, an increase of one death per 1,000 in the year of birth or the year after reduces educational attainment by about 0.1%; reduces the probability that the mother is married at the time of the birth by 0.6% and increases the probability that the mother smoked during pregnancy by 2%. We interpret the lower education, lower probability of marriage, and the higher probability of smoking as indicators of lower socioeconomic status, suggesting that exposure to disease in infancy reduces adult socioeconomic status.

An extra death per 1,000 in the year after birth is also associated with a 1.7% higher probability of weight gain over 60 pounds during the pregnancy. It is possible that this reflects a biological mechanism of the sort that Barker discusses, though it may also reflect behavior. Note that weight gain during pregnancy tends to be negatively correlated with pre-pregnancy weight, so that women who are overweight to begin with often put on less weight (Lin, 2009). Since women of lower socioeconomic status are more likely to be overweight, the increased probability of high weight gain does not suggest a simple relationship between early disease environment and lower future socioeconomic status. Weight gain over 60 pounds is associated with a higher risk of diabetes and hypertension during pregnancy, as well as with a higher probability of complications of labor and delivery.

As in Table 2, the percentage changes implied by the Table 3 coefficients are larger for blacks than for whites: An extra post-neonatal death in the year after birth reduces education by 0.1% for whites compared to 0.2% for blacks. Comparable figures for other outcomes are: 0.9% for whites compared to 1.3% for blacks for marriage; 1.7% for whites compared to 2.2% for

blacks for weight gain over 60 pounds; and 1.3% for whites compared to 3.4% for blacks for smoking during pregnancy.

Table 4 shows the estimated effects of the mother's early life environment on the probability that her child is low birth weight. In these regressions, the coefficients on $PNMR_{t-1}$, $PNMR_t$, and $PNMR_{t+1}$ are seldom individually statistically significant. However, F-statistics indicate that the three variables are jointly significant. Moreover, the coefficients on the three variables are similar to each other, suggesting that exposure to disease at any point in the mother's early life is equally likely to lead to negative consequences for her child.[13]

Overall, an extra post-neonatal death in the year after birth (for example) is estimated to increase the probability that the offspring is low birth weight by 0.6%. For whites, the corresponding estimate is 0.5% while for blacks it is actually -0.1%. Viewed on its own, this pattern of positive effects on low birth weight for whites and negative effects on low birth weight for blacks might suggest that whites were "scarred" by exposure, but that the most impacted blacks were selected out of the sample of future mothers altogether. This story would be similar to the Bozzoli et al. scenario in which adults from developed countries who were exposed to high post-neonatal mortality rates as children are short because they are scarred by the experience, while adults from less developed countries who were exposed to disease are tall because they are the survivors. This could be true even though, as we have seen, those black women who do go on to give birth appear to be more negatively impacted than white women, as scarring and selection effects need not be uniform across different dimensions of health.

[13] For a child who was born in January of year t, the disease environment in t is likely to be a better measure of the disease environment in early life than the disease environment in year t+1, whereas the opposite is true for a baby born in December. Hence, the results regarding the timing of the health insults are subject to error and must be interpreted with caution.

Panel B of Table 4 show the same models controlling for the maternal characteristics and behaviors examined in Table 3. The estimated coefficients on the PNMR variables remain jointly significant and positive for whites, though the magnitude is slightly reduced. For blacks, the estimated effect is still negative, and is larger in absolute value. One interpretation of this pattern is that even within groups defined by education, marital status, and so on, exposure to high post-neonatal mortality rates in early life reduces the probability that the black women who would be most at risk of having low birth weight babies ever have children.

VI. Discussion and Conclusions

We investigate the connection between early life health, future socioeconomic status, future health, and the health of future offspring. Although post-neonatal mortality rates around the time of the birth are crude measures of early life health, our estimates tell a consistent story. We find that women who were exposed to higher post-neonatal mortality rates in early life are more likely to have diabetes when they give birth to their own children. They are also less educated, less likely to be married, more likely to smoke while pregnant, and more likely to gain over 60 pounds during the pregnancy. These effects are present for both white and black women, but are larger for black women. To the extent that some women are sufficiently negatively impacted that they never give birth, these estimates likely understate the true negative impacts.

We also find some evidence consistent with intergenerational transmission of health shocks. Among whites, mothers who were exposed to high post-neonatal mortality rates in early life have infants of lower birth weight. Among blacks, women from cohorts with higher exposure are less slightly likely to have low birth weight infants, which might reflect selection effects.

Overall, our estimates suggest that the disease environment in early life, as proxied by departures from state- and race-specific post-neonatal mortality rate trends, has long lasting impacts which are felt in multiple aspects of a woman's life. This suggests that life trajectories are malleable but perhaps in subtle ways: There can be lag of twenty years or more between improvement in the early childhood health environment and its manifestation in maternal health, and indeed on the initial health of the next generation.

References

Almond D. “Is the 1918 Influenza Pandemic Over? Long-Term Effects of *In utero* Influenza Exposure in the Post-1940 U.S. Population.” Journal of Political Economy 2006; 114; 672-712.

Almond D and Chay KY. “The Long-Run and Intergenerational Impact of Poor Infant Health: Evidence from Cohorts Born During the Civil Rights Era.” Department of Economics, Columbia University Working Paper. 2006.

Almond D, Chay KY, and Greenstone M. “Civil Rights, the War on Poverty, and Black-White Convergence in Infant Mortality in Mississippi,” *MIT Department of Economics Working Paper No. 07-04.* 2006.

Almond D and Currie J. 2011a. “Human Capital Development Before Age Five,” in Orley Ashenfleter and David Card (Eds.), The Handbook of Labor Economics, 4b. Amsterdam: Elsevier Science B.V.

Almond D and Currie J. 2011b. “Killing Me Softly: The Fetal Origins Hypothesis.” *Journal of Economic Perspectives,* Vol. 25(3): 153-172.

Almond D, Hoynes HW, and Schanzenbach DW. “Inside the War on Poverty: The Impact of Food Stamps on Birth Outcomes.” *Review of Economics and Statistics;* 2011; 93(2): 387-403.

Angrist JD, and Pischke JS. 2009. *Mostly Harmless Econometrics: An Empiricist’s Companion.* Princeton University Press, Princeton NJ.

Barker DJP. 1998. *Mothers, Babies and Health in Later Life.* 2d ed. Edinburgh, UK: Churchill Livingston.

Behrman JR and Rosenzweig M. “Returns to Birth weight.” *The Review of Economics and Statistics*; 2004; 86(2): 586-601.

Bennish M, Arnow P, Beem M, Doveikis S. “Epidemic Measles in Chicago in 1983,” *American Journal of Diseases of Childhood* April 1986; 140; 341-344.

Black SE, Devereux PJ, and Salvanes KG. “From the Cradle to the Labor Market? The Effect of Birth Weight on Adult Outcomes.” *Quarterly Journal of Economics* 2007; 122(1); 409-439.

Bozzoli C, Deaton A, and Quintana-Domeque C. “Adult Height and Childhood Disease.” Demography 2009; 46(4); 647-669.

Case A and Paxson C. "Stature and Status: Height, Ability, and Labor Market Outcomes." Journal of Political Economy 2008; 116(3); 499-532.

Case A and Paxson C, “Health and Wealth: Early Life Health and Cognitive Function in Old

Age.” *American Economic Review: Papers & Proceedings* 2009; 99(2); 104–109.

Case A and Paxson C. 2010. “Causes and Consequences of Early Life Health.” National Bureau of Economic Research Working Paper No. 15637, http://www.nber.org/papers/w15637 (accessed November 2010).

Chay KY, Guryan J, Mazumder B. 2009. “Birth Cohort and the Black-White Achievement Gap: The Roles of Access and Health Soon After Birth.” NBER Working Paper No. 15078.

Conti G, Heckman JJ, Lopes H, and Piatek R. “Constructing Economically Justified Aggregates: An Application of the Early Origins of Health.” *Journal of Econometrics,* forthcoming. PUBLISHED?

Costa DL. “Unequal at Birth: A Long-Term Comparison of Income and Birth Weight.” *The Journal of Economic History* 1998; 58(4); 987-1009.

Crimmons E and Finch C. “Infection, inflammation, height, and longevity.” Proceedings of the National Academy of Sciences 2006; 103(2); 498-503.

Cunha F, Heckman JJ, and Schennach SM. “Estimating the Technology of Cognitive and Noncognitive Skill Formation.” *Econometrica* 2010; 78(3); 883-931.

Currie J. “Inequality at Birth: Some Causes and Consequences.” *American Economic Review* 2011; 101(3):1-22.

Currie, J and Gruber J. 1996. "Saving Babies: The Efficacy and Cost of Recent Changes in the Medicaid Eligibility of Pregnant Women." *The Journal of Political Economy* 1996; 104(6): 1263-1296.

Currie J and Moretti E. “Mother’s Education and the Intergenerational Transmission of Human Capital: Evidence from College Openings.” *Quarterly Journal of Economics* 2003; 118(4); 1495-1532.

Currie J and Moretti E. "Biology as Destiny? Short- and Long-Run Determinants of Intergenerational Transmission of Birth Weight." Journal of Labor Economics 2007; 25(2); 231-264.

Currie, J and Neidell M. “Air Pollution and Infant Health: What Can We Learn From California’s Recent Experience?” *Quarterly Journal of Economics* 2005; 120(3): 1003-1030.

Currie J, Neidell M, and Schmeider JF. “Air Pollution and Infant Health: Lessons from New Jersey.” Journal of Health Economics 2009; 28(3); 688-703.

Currie J and Thomas D. 2001. "Early Test Scores, School Quality and SES: Long Run Effects on Wage and Employment Outcomes." In Solomon Polachek (ed.) Worker Wellbeing in a Changing Labor Market (Research in Labor Economics, Volume 20): 103-132.

Devereux, PJ and Hart RA. “Forced to be Rich? Returns to Compulsory Schooling in Britain.” *Economic Journal* 2010; 120 (549):1345-1364.

Dobbie W and Fryer RG. “Are high quality schools enough to increase achievement among the poor? Evidence from the Harlem children's zone,” *American Economic Journal: Applied Economics* 2011; 3(3):158-187.

Elo I and Preston S. “Effects of Early-Life Conditions on Adult Mortality: A Review,” *Population Index* 1992; 58(2):186-212.

Evans WN and Ringel JS. “Tobacco Taxes and Public Policy to Discourage Smoking,” *Tax Policy and the Economy* 1999; 13; 1-55

Fertig AR and Watson T. “Minimum Drinking Age Laws and Infant Health Outcomes.” *Journal of Health Economics* 2009; 28(3); 737-747.

Glezen WP, Decker M, Joseph S, and Mercready R. “Acute Respiratory Disease Associated with Influenza Epidemics in Houston, 1981-1983,” *The Journal of Infectious Diseases* 1987; 155(6): 1119-1204.

Hoynes HW, Page ME, and Stevens AH. 2009. “Is a WIC Start a Better Start? Evaluating WIC’s Impact on Infant Health Using Program Introduction.” National Bureau of Economic Research Working Paper No. 15589, http://www.nber.org/papers/w15589 (accessed November 2010).

Kelly E. “The Scourge of Asian Flu: *In utero* Exposure to Pandemic Influenza and the Development of a Cohort of British Children.” Journal of Human Resources 2011; 46(4); 669-694.

Lin, M. 2011. “Does *In utero* Exposure to Illness Matter? The 1918 Influenza Epidemic in Taiwan as a Natural Experiment.” Working Paper.

Lin W. “Why has the Health Inequality among Infants in the US Declined? Accounting for the Shrinking Gap", *Health Economics* 2009; 18(7): 823-841.

Lindeboom M, Llena-Nozal A, and van der Klaauw B. “Parental Education and Child Health: Evidence from a Schooling Reform.” *Journal of Health Economics* 2009; 28(1): 109-131.

Ludwig DS and Currie J. “The Association Between Pregnancy Weight Gain and Birthweight: A Within-Family Comparison.” *The Lancet* 2010; 376(9745): 984-990.

Manski C. “Genes, Eyeglasses, and Social Policy.” *Journal of Economic Perspectives* 2011; 25(4); 83-94.

McCrary J and Royer H. "The Effect of Female Education on Fertility and Infant Health: Evidence from School Entry Policies Using Exact Date of Birth.” *American Economic Review* 2011; 101(1):158-95.

Neelson S and Stratmann T. 2010. “Long Term Effects of Prenatal Influenza Exposure: Evidence from Switzerland.” Working Paper.

Nelson RE. “Testing the Fetal Origins Hypothesis in a Developing Country: Evidence from the 1918 Influenza Pandemic.” Health Economics 2010; 19(10); 1181-1192.

Noonan K, Reichman NE, Corman H, and Davel D. “Prenatal Drug Use and the Production of Infant Health.” *Health Economics* 2007; 16(4): 361-384.

Schick A and Steckel RH. 2010. “Height as a Proxy for Cognitive and Non-Cognitive Ability.” NBER Working Paper No. 16570.

Singh G and Dyck P. “Infant Mortality in the United States: 1935-2007: Over Seven Decades of Progress and Disparities,” Health Research Service Administration, Maternal and Child Health Bureau, Washington D.C., U.S. Dept. of Health and Human Services, 2010.

Smith JP. “The Impact of Childhood Health on Adult Labor Market Outcomes,” The Review of Economics and Statistics, 2009; 91 #3; 478-489.

Stewart, RJC, Sheppard HG, Preece RF, and Waterlow JC. “The Effect of Rehabilitation at Different Stages of Development of Rats Marginally Malnourished for Ten to Twelve Generations.” *British Journal of Nutrition* 1980; 43(3): 403-412.

Strauss J and Thomas D. “Health, Nutrition, and Economic Development.” Journal of Economic Literature 1998; 36(2); 766-817.

UNICEF: Child Health Epidemiology Reference Group. http://www.unicef.org/publications/index_24840.html (accessed August 7, 2011).

Wood D and Brunell P. “Measles Control in the United States: Problems of the Past and Challenges for the Future,” *Clinical Microbiology Reviews*, 1995; 8(2): 260-267.

Figure 1: Post-Neonatal Mortality Rate, California

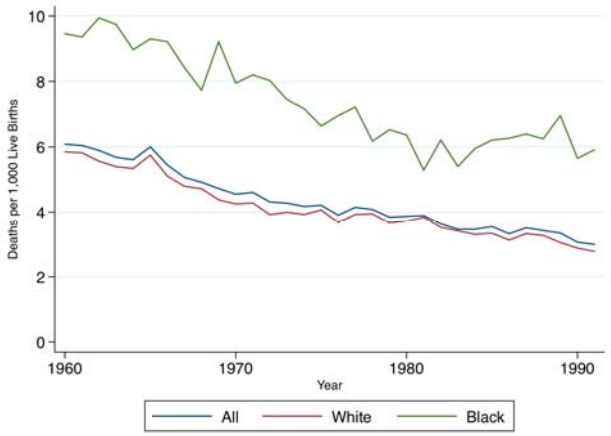

Figure 2: Post-Neonatal Mortality Rate, Georgia

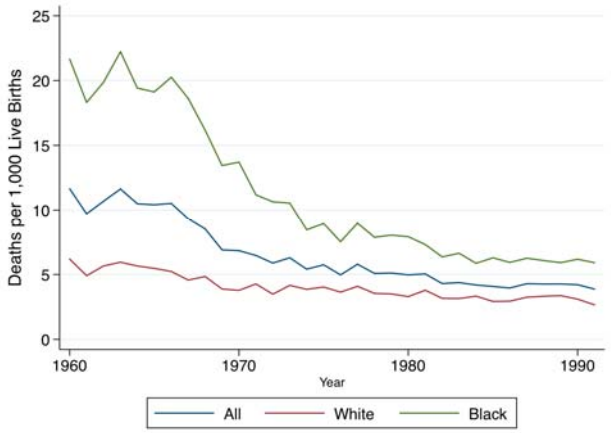

Figure 3: Post-Neonatal Mortality Rate, Illinois

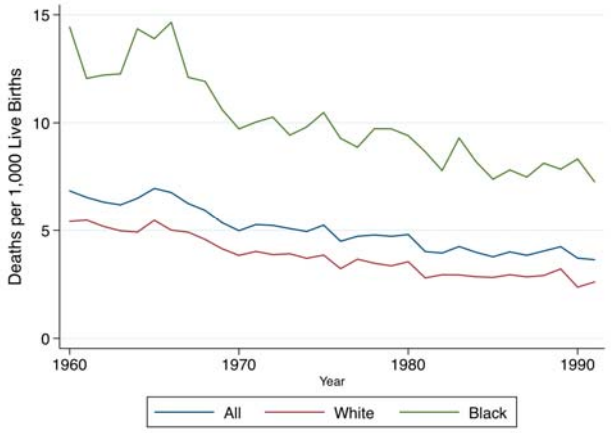

Figure 4: Post-Neonatal Mortality Rate, Michigan

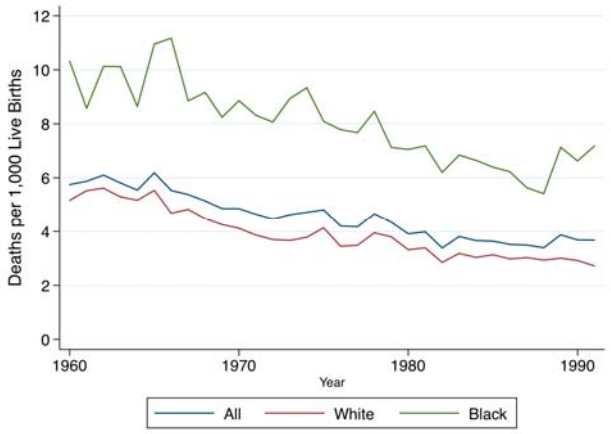

Figure 5: Post-Neonatal Mortality Rate, Pennsylvania

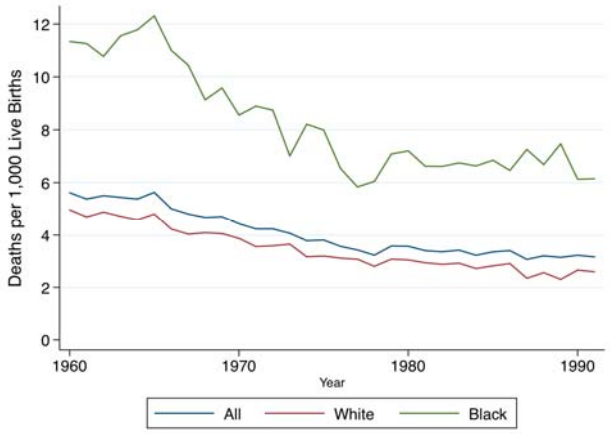

Table 1: Summary Statistics

Variable	All	Below Median PNMR	Above Median PNMR	Whites	Blacks
PNMR t	4.829	3.533	6.126	4.100	8.695
	(2.264)	(0.406)	(2.595)	(0.870)	(3.246)
PNMR t-1	4.970	3.679	6.263	4.204	9.035
	(2.394)	(0.558)	(2.796)	(0.907)	(3.482)
PNMR t+1	4.700	3.540	5.861	4.003	8.395
	(2.152)	(0.528)	(2.509)	(0.836)	(3.042)
Diabetes	0.022	0.020	0.023	0.022	0.018
	(0.044)	(0.037)	(0.050)	(0.042)	(0.053)
Hypertension	0.065	0.064	0.066	0.064	0.066
	(0.071)	(0.064)	(0.078)	(0.068)	(0.087)
Low Birthweight (<2500g)	0.068	0.062	0.075	0.059	0.119
	(0.075)	(0.060)	(0.086)	(0.063)	(0.106)
Low Weight Gain (<16 lbs)	0.078	0.070	0.086	0.068	0.134
	(0.083)	(0.068)	(0.095)	(0.070)	(0.115)
High Weight Gain (>60 lbs)	0.029	0.035	0.023	0.029	0.029
	(0.050)	(0.050)	(0.049)	(0.048)	(0.058)
Smoking	0.124	0.154	0.093	0.138	0.054
	(0.124)	(0.130)	(0.109)	(0.125)	(0.087)
Years of Education	13.17	12.95	13.40	13.32	12.38
	(1.51)	(1.52)	(1.46)	(1.51)	(1.20)
Married	0.586	0.555	0.617	0.661	0.189
	(0.335)	(0.301)	(0.362)	(0.294)	(0.244)
White	0.841	0.998	0.684	1.000	0.000
	(0.365)	(0.042)	(0.465)	(0.000)	(0.000)
Child's Birthyear	1997.7	1999.5	1995.9	1997.7	1997.5
	(5.17)	(4.71)	(4.96)	(5.15)	(5.24)
Maternal Age	23.65	22.35	24.95	24.11	21.22
	(5.20)	(4.50)	(5.53)	(5.19)	(4.53)
Mother's Birthyear	1973.5	1976.7	1970.4	1973.1	1975.8
	(6.73)	(5.21)	(6.64)	(6.68)	(6.58)
Cells	1271011	524104	746907	982523	288488
Number of Births	16780418	8395124	8385294	14118124	2662294

Notes: For each dependent variable, means and standard deviations are calculated using as weights the number of births in the cell that reported the outcome. For each independent variable, means and standard deviations are calculated using as weights the number of births in the cell.

Table 2: Effects of Early Life Health Environment on Maternal Health

Entered Together	Diabetes All	Hypertension All	Diabetes White	Hypertension White	Diabetes Black	Hypertension Black
PNMR t-1	-0.007	-0.232	-0.068	-0.387	0.273	0.352
	(0.091)	(0.206)	(0.105)	(0.224)	(0.192)	(0.389)
PNMR t	0.150	-0.034	0.135	-0.148	0.046	0.149
	(0.106)	(0.210)	(0.106)	(0.189)	(0.305)	(0.401)
PNMR t+1	0.394*	0.157	0.298*	-0.020	0.624*	0.401
	(0.134)	(0.197)	(0.140)	(0.230)	(0.258)	(0.413)
F(3,50)	3.00	0.91	2.00	1.83	4.15	1.18
p-value	0.0390	0.4440	0.1258	0.1530	0.0106	0.3260
Number of Cells	1256790	1256790	973013	973013	283777	283777
Entered Separately						
PNMR t-1	0.184	-0.200	0.076	-0.458	0.520*	0.574
	(0.121)	(0.210)	(0.126)	(0.231)	(0.251)	(0.375)
PNMR t	0.326*	-0.075	0.232	-0.335	0.522	0.553
	(0.145)	(0.210)	(0.139)	(0.190)	(0.342)	(0.394)
PNMR t+1	0.465*	0.062	0.339*	-0.209	0.761*	0.628
	(0.167)	(0.219)	(0.165)	(0.246)	(0.328)	(0.382)
Number of Cells	1256790	1256790	973013	973013	283777	283777

Notes: All coefficients and standard errors are multiplied by 1000. These regressions were estimated using race-age-child birth year-child state of birth-mother state of birth-mother year of birth cells and are weighted by the number of births in each cell. Specifications include controls for black, Hispanic, missing Hispanic, child state of birth by child's year of birth fe, mother state of birth fe, mother's age fe, mother's age by mother's birth year linear time trends, and mother's state of birth linear time trends.Standard errors (in parentheses) are clustered by mother's state of birth. The significance level is * $p<0.05$.

Table 3: Effects of Early Life Health Environment on Maternal Behaviors

All Entered Together	Years of Education	Married	Low Weight Gain	High Weight Gain	Smoker
PNMR t-1	-7.262	-2.376*	-0.075	0.136	1.532*
	(3.635)	(0.986)	(0.247)	(0.106)	(0.419)
PNMR t	-17.887*	-2.950*	-0.454*	0.103	2.577*
	(2.348)	(0.828)	(0.213)	(0.105)	(0.383)
PNMR t+1	-18.204*	-3.600*	-0.448	0.487*	2.501*
	(3.564)	(1.138)	(0.273)	(0.132)	(0.479)
F(3,50)	23.59	4.57	2.10	5.60	17.49
p-value	0.0000	0.0066	0.1122	0.0022	0.0000
Number of Cells	1259783	1271011	1179920	1179920	1176560

White Entered Together	Years of Education	Married	Low Weight Gain	High Weight Gain	Smoker
PNMR t-1	-2.602	-3.923*	0.084	0.071	0.661
	(4.050)	(0.989)	(0.236)	(0.122)	(0.531)
PNMR t	-14.241*	-4.243*	-0.251	0.102	1.807*
	(2.593)	(0.712)	(0.224)	(0.118)	(0.395)
PNMR t+1	-13.302*	-5.628*	-0.446	0.497*	1.796*
	(3.951)	(1.132)	(0.261)	(0.134)	(0.490)
F(3,50)	13.22	13.32	1.78	5.25	9.66
p-value	0.0000	0.0000	0.1625	0.0031	0.0000
Number of Cells	974825	982523	924179	924179	915716

Black Entered Together	Years of Education	Married	Low Weight Gain	High Weight Gain	Smoker
PNMR t-1	-25.858*	4.455*	-1.397	0.517*	0.649
	(4.562)	(1.443)	(0.706)	(0.252)	(0.500)
PNMR t	-24.133*	1.041	-1.805*	0.019	2.017*
	(4.026)	(0.713)	(0.467)	(0.329)	(0.651)
PNMR t+1	-22.960*	-2.472*	-1.140	0.634*	2.030*
	(4.151)	(0.775)	(0.627)	(0.238)	(0.718)
F(3,50)	25.34	21.17	26.22	6.58	4.72
p-value	0.0000	0.0000	0.0000	0.0008	0.0056
Number of Cells	284958	288488	255741	255741	255741

Notes: All coefficients and standard errors are multiplied by 1000. These regressions were estimated using race-age-child birth year-child state of birth-mother state of birth-mother year of birth cells and are weighted by the number of births in each cell. Specifications include controls for black, Hispanic, missing Hispanic, child state of birth by child's year of birth fe, mother state of birth fe, mother's age fe, mother's age by mother's birth year linear time trends, and mother's state of birth linear time trends. Standard errors (in parentheses) are clustered by mother's state of birth. The significance level is * $p<0.05$.

Table 4: Effects of Early Life Health Environment on Infant Health

	Entered Together			Entered Together, Controlling for Maternal Behaviors		
	LBW All	LBW White	LBW Black	LBW All	LBW White	LBW Black
PNMR t-1	0.269	0.219	-0.558	0.211	0.193	-0.430
	(0.167)	(0.180)	(0.366)	(0.177)	(0.187)	(0.395)
PNMR t	0.305	0.341	-0.642	0.155	0.235	-0.650
	(0.174)	(0.185)	(0.620)	(0.175)	(0.186)	(0.669)
PNMR t+1	0.383*	0.285	-0.179	0.202	0.173	-0.385
	(0.167)	(0.166)	(0.551)	(0.164)	(0.164)	(0.536)
F(3,50)	6.16	5.99	3.91	2.75	4.54	4.54
p-value	0.0012	0.0014	0.0138	0.0522	0.0069	0.0069
Number of Cells	1271011	982523	288488	1271011	982523	288488

Notes: All coefficients and standard errors are multiplied by 1000. These regressions were estimated using race-age-child birth year-child state of birth-mother state of birth-mother year of birth cells and are weighted by the number of births in each cell. Specifications include controls for black, Hispanic, missing Hispanic, child state of birth by child's year of birth fe, mother state of birth fe, mother's age fe, mother's age by mother's birth year linear time trends, and mother's state of birth linear time trends. Regressions in the second panel control for mother's education, marital status, smoking behavior, excessive or inadequate weight gain during pregnancy, and whether any of these maternal behaviors are missing. Standard errors (in parentheses) are clustered by mother's state of birth. The significance level is * $p<0.05$.