How do Early Life Health Experiences Affect Equality of Opportunity for Future Generations?

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Abstract

A large literature documents that early life health environments can have long lasting impacts on individuals' well-being. Existing literatures in biology, epidemiology, psychology, child development and economics also predict that the effects of early environments should persist beyond the exposed generation. This article considers what is known about the extent to which "first generation" effects persist to later generations, with a focus on studies that use randomized experiments and "natural experiment" research designs that can help isolate causal effects from correlations. In addition to documenting persistent effects of early life environments from one generation to the next, we argue that the presence and magnitude of multi-generational linkages has important implications for the evaluation of public policies intended to promote equality of opportunity. The emerging evidence on positive interventions' multi-generational impacts suggests that existing cost-benefit analyses typically underestimate programs' true value.

It is well known that there are significant health differences between high and low-income children, and that health disparities across income groups are present even very early in life (e.g. Case, Lubotsky and Paxon 2002). Over the past decade, social scientists have come to appreciate that these health differences may also be important contributors to the persistence of income disparities across generations. A rapidly expanding literature harnessing "natural experiment" research designs has established causal relationships between a variety of measures of early life health and later life health and economic success (Almond and Currie 2011a, 2011b; Almond, Currie and Duque 2017). Importantly, while the majority of studies are based on negative "shocks" to children's health environments (such as disease outbreaks or famines) studies that focus on positive early life health interventions also find that they can lead to substantive improvements in adult health, education and earnings.

By extension, existing literatures in biology, psychology, epidemiology and economics, all predict that the effects of early life environments should echo beyond the exposed generation. Indeed, Almond et al. (2017) note that multigenerational studies are "a particularly exciting direction for future work, given that thus far, there is not a great deal of research on intergenerational effects of *in utero* shocks in humans, although they are known to exist in animal models." This article considers what is known about the persistence of multi-generational effects to date, with a focus on studies that use natural experiment research designs to isolate causal effects from correlations. In addition to documenting that the effects of early life environments spill over from one generation to the next, we argue that the presence and magnitude of multi-generational linkages have important implications for the evaluation of public policies intended to promote equality of opportunity. The emerging evidence on positive

interventions' multi-generational impacts suggests that typical cost-benefit analyses, which focus on measuring more immediate benefits, frequently underestimate programs' true value.

We begin by briefly describing what is known about the long-term effects of early life health environments on treated cohorts' well-being. In doing so, we discuss the usefulness of natural experiment research designs, and common issues regarding the interpretation of estimates. Next, we describe why it is that we might expect the effects of early life health environments to persist to later generations. We provide a brief overview of the evidence on multi-generational processes based on animal experiments that take place in a laboratory setting. We then describe recent studies that have extended the natural experiment methodology to consider similar multi-generational processes in humans. In addition to highlighting this work, our goal is to draw connections and provide insights that extend our thinking about the importance of early life interventions.

I. Early Life Health Environments and the First Generation's Well-being in Later Life

I. A. Evidence from Animal Experiments

Studies proposing that the antecedents of adult health might originate in the fetal and early life periods existed as early as the 1930s, but interest became more widespread following the work of epidemiologist David Barker (1990). The basic idea behind Barker's "fetal origins hypothesis" is that while *in utero,* a growing organism absorbs molecular information from the mother, treats this information as a signal about the postnatal environment, and programs itself in response to those signals. A key feature of the fetal origins hypothesis is that the observed health effects of the *in utero* environment can remain latent for many years.

Although there is ongoing debate about causal pathways, an abundance of animal experiments have generated substantive scientific support for the fetal origins hypothesis (e.g. Gluckman et al. 2008). One heavily studied manipulation of the prenatal environment is nutritional deprivation, where researchers have consistently found that pregnant rats that are intentionally malnourished produce offspring that are more prone to metabolic disease (conditions such as obesity, diabetes, hypertension and heart disease) and other chronic health conditions in later life, even if the offspring receive sufficient nutrition after birth (e.g. Gluckman et al. 2008). Similarly, rats that are malnourished before or during pregnancy produce offspring with smaller brains and reduced cognition (e.g. Hunter and Sadler 1987).

Animal experiments have also generated substantive evidence that *in utero* exposure to infectious disease has long term impacts on well-being. Like studies of nutritional deprivation, the effects extend beyond physical health. For example, several studies have documented that the offspring of pregnant rats who have been injected with the influenza virus are more likely to grow up to exhibit behavioral abnormalities similar to those of schizophrenia patients (Brown 2012). Importantly, some studies suggest that, rather than being due to the pathogen itself, this outcome is due to the mother's excessive immune function (Canetta and Brown 2012), which has wider implications for child development, as immune system activation has been linked to poverty and stress. Animal experiments have also documented direct links between *in utero* stress and later life measures of physical health, mental health and cognition (Weinstock 2017), with additional evidence that the impacts of *in utero* stress can be reversed through interventions, (e.g. Wakshlak and Weinstock 1990). As well, there is substantive evidence that the developing fetus is affected by exposure to smoke and other pollutants (Shea and Steiner 2008 U.S. EPA 2009).

I.B. Evidence from Natural Experiments

Isolating causal effects of the early life environment in humans is challenging because unlike the physical and biological sciences, where experiments and clinical trials are common, social scientists' opportunities to manipulate early life conditions in truly experimental settings are more constrained. It may be tempting to interpret correlations between the early childhood environment and later generations' outcomes as causal, but these associations may reflect the effects of other family and parental characteristics. For example, among adults who were nutritionally deprived *in utero* there is a higher incidence of economic disadvantage, so differences between their health, and the health of adults with adequate nutrition *in utero*, could actually be due to differences in childhood economic circumstances (which are also known correlates of later life health). Distinguishing causal effects from correlations is important for understanding the full range of processes that contribute to child development, however, and imperative to the design of effective interventions.

To overcome this challenge, researchers have looked for naturally occurring variation that generates "quasi" treatment and control groups. These "natural experiment" research designs try to emulate real experimental settings by comparing outcomes across individuals who are very similar, but are differentially exposed to potentially important environmental conditions that vary across space and/or time. The likely randomness of the variation in the health environment is often most believable when it is generated by an unanticipated health shock, such as a widespread famine, or disease epidemic.[1]

[1] We do not attempt to review every paper in this extensive literature on first generation effects. For excellent reviews of the first generation literature see Almond and Currie (2011a, 2011b) and Almond, Currie and Duque (2017).

As one example of a natural experiment, a series of studies investigate the long term effects of early life exposure to the Dutch Hunger Winter, which occurred during the winter of 1944-1945, after the German occupying force placed an embargo on food transports to the western Netherlands. Prior to October of 1944, caloric intake in the Netherlands was considered adequate, but in the wake of the embargo, official rations fell to 1,000 calories per day, and then fell further to 500 calories per day in April 1945 (see Figure 1). Many "first-generation" studies have analyzed the long-term health consequences of *in-utero* malnutrition by using variation in exposure to the Dutch Hunger Winter based on the individual's date and place of birth relative to distributed food rations. The abrupt beginning and ending of the Dutch Hunger Winter ensures a necessary assumption for any valid natural experiment research design: that in the absence of the famine, the health environments experienced by the "treatment" and "control" cohorts would have been similar. Researchers analyzing the Dutch Hunger Winter and have found evidence that *in utero* exposure to malnutrition affects later life risk of obesity, high blood pressure, cardiovascular disease, schizophrenia, and hospitalization, and has a negative impact on employment outcomes (e.g. Painter et al. 2005, Lumey et al. 2011, Scholte, van den Berg and Lindeboom, 2015). Importantly, all of these outcomes are known predictors of the next generation's health.

As another example, following Almond's (2006) pioneering work on the 1918 flu pandemic, a number of researchers have investigated the long-term effects of early childhood exposure to infectious disease by making use of the abrupt spike in flu related deaths that occurred in 1918. Figure 2 shows that, like the Dutch Hunger Winter, the short nature of the epidemic's abrupt shock to the local disease environment has enabled outcome comparisons between cohorts who were prenatally exposed and cohorts who were born right before, or soon after, the outbreak,

with limited concerns about other time-varying contaminators. As with studies of the Dutch Hunger Winter, analyses of the 1918 pandemic and other flu outbreaks have found substantive reductions in later life educational attainment, earnings, and health (e.g. Almond and Mazumder 2005, Mazumder et al. 2010, Neelsen and Stratmann 2012, Liu and Lin 2013, Nelson 2010, Parman 2012, and Schwandt 2018).

Using similar research strategies, researchers have also investigated the long-term effects of *in utero* exposure to other diseases, nutritional deprivation resulting from seasonal variation in the observance of Ramadan, radiation and other pollutants, and maternal stress. Importantly, the set of considered outcomes in humans has included measures of later life well-being that extend beyond health, particularly measures of educational success and earnings. Recent surveys of this extensive literature are available in Almond and Currie (2011a), Almond and Currie (2011b) and Almond, Currie and Duque (2017), and they point to substantive effects of early life health "shocks" on later life health, educational attainment, and economic outcomes.

A smaller literature, also summarized in the above survey articles, has begun to harness natural experiment research designs to investigate the impact of positive, and generally less dramatic, early life health interventions. These studies are important, because in addition to contributing to our knowledge about the long-term impacts of childhood environments, they also provide evidence on the potential for policy to reduce disparities. Treatment and control groups are formed by using variation in policies that generate differential access to health inputs among otherwise nearly identical individuals. Examples include differential changes in Medicaid[2] eligibility rules across states and over time, and county-by-county variation in the original

[2] Medicaid is the primary federal program that provides health insurance to American children living in low income families.

adoption of the Food Stamp and Head Start programs.[3] By using this type of policy variation to identify program effects, researchers can disentangle the effects of family characteristics that affect take-up of health services from the effects of the program itself. This is critical, as, even conditional on observable characteristics such as income and education, families who choose to participate in programs are generally less advantaged (and, therefore, have worse outcomes) than those who do not.

Similar to studies that exploit negative shocks to the childhood environment, studies focusing on the effects of positive health interventions find that they improve later life health and economic success. *In utero* and childhood access to the Medicaid program is associated with improvements in adolescent health status (Currie et al. 2008), reductions in black adolescent's mortality rates (Wherry and Meyer 2015), reductions in metabolic syndrome related illnesses and hospitalizations (Miller and Wherry 2018), and adult mortality (Brown et al. 2017). Looking beyond health outcomes, childhood exposure to Medicaid has also been found to increase test scores (Levine and Schanzenbach 2009), educational attainment (Brown et al. 2017; Cohodes et al. 2016; Miller and Wherry 2018) and earnings (Brown et al. 2017). Similarly, several studies of the Head Start preschool program, which provides both educational and health services, have documented that, like Medicaid, Head Start also yields important long-term health and economic benefits (Carneiro and Ginja 2014; Deming 2009, Garces et al. 2002, Ludwig and Miller 2007). In the same vein, Butikofer, Loken and Salvanes (2017) examine the adoption of mother and child health centers in Norway; Glied and Neidell (2010) investigate the impacts of water fluoridation; Bhalotra and Venkataramani (2015) look at the long term effects of antibiotic

[3] The Food Stamp Program (currently called the Supplemental Nutrition Assistance Program) is a federal program that provides food purchasing assistance to low income American families. The Head Start program is the largest early childhood education program in the United States. It also targets low income children.

therapies; Bharadwaj, Loken and Nielson (2013) examine surfactant and related treatments; Fitzsimons and Vera-Hernandez (2013) look at the effects of breast feeding encouragement programs; and Hoynes, Schanzenbach and Almond (2016) examine the effects of the Food Stamp Program. All of these natural experiments provide strong evidence that early life health and nutrition interventions generate long term benefits on treated generations.

II. Evidence of Multi-generational Effects

II.A. Potential Mechanisms

Taken as a whole, a wealth of biological and social science research generates two broad conclusions. First, early life health environments have long-term impacts on the health and economic outcomes of those who experience them. Second, widespread public health interventions targeted at children have scope to reduce later life disparities. It is easy to imagine how these effects might persist to later generations, as there is substantial evidence that healthier, more educated, and higher income parents raise children who do better on a variety of measures than children whose parents are unhealthy, have low levels of education, or low incomes. This may be because families who benefit from a positive intervention have more resources to transfer onto their children, because improvements in health and socioeconomic status are accompanied by changes in behavior (such as reduced smoking, or differences in parenting practices), or reductions in parental stress (Becker and Tomes 1979, Heckman 2007, Conger and Conger 2007).

Direct biological mechanisms may also play a role. For example, several studies discussed above have found that there is an association between the early life health environment and the incidence of metabolic-syndrome in adulthood. In turn, maternal metabolic syndrome conditions are associated with increased risk of gestational diabetes, pregnancy complications related to

high blood pressure, preterm birth, and low birth weight (Catalano and Ehrenberg, 2006), which are predictive of offspring's future health and economic trajectories (Black et al 2007, Hsin 2012, Royer 2009). Moreover, an abundance of animal experiments find that prenatal health shocks have persistent effects beyond the first generation, although exact biological processes underlying this transmission are not yet fully understood.[4] Many researchers believe that epigenetic processes, which change the way information in genes is transcribed, underlie these long run effects. Unlike genes, epigenomes adjust much more rapidly to environmental insults. Epigenetic processes are also consistent with the observation that, among treated cohorts, resulting health outcomes are often not apparent until many years after birth. At the molecular level, the particular ways in which epigenetic reprogramming occurs is through the silencing of certain genes through impaired inheritance of genes, or addition of chemical compounds to the DNA, and through spatial reorganization of genes within the chromosomes (Jirtle and Skinner 2007; Hochberg et al. 2011).

In addition to these mechanisms, environmental effects on the treated generation's survival may lead to differences in future generations' observed outcomes. Among the treated generation, any additional (fewer) deaths resulting from a decline (increase) in the quality of the health environment are most likely to occur among those who are least healthy. This will lead to a mechanical increase (decrease) in average health among survivors, which is often referred to as the selection, or "culling" effect, and which will work in the opposite direction from the direct health effect--often called the "scarring" effect (e.g. Elo and Preston 1992). Importantly, if culling occurs among treated cohorts, then we would expect a mechanical spillover onto the next

[4] Examples include Zamenhof, Marthens and Grauel (1971), Crowley and Griesel (1966), Aerts and Van Assche (2006), Dunn and Bale (2009), Jimenez-Chillaron et al. (2009), Martinez et al. (2014), Recent reviews of the literature on transgenerational epigenetic inheritance include Daxinger and Whitelaw (2010), Daxinger and Whitelaw (2012), Grossniklaus et al. (2013), and Heard and Martienssen (2014).

generation's observed health as well. As in the treated generation, the effects of culling on the second generation will work in the opposite direction from the direct health effects.

Finally, the same (or related) biological processes that generate improvements in the treated generation's later life health may directly affect the treated generation's fecundity. Moreover, effects of the early life environment on treated cohorts' human capital may be accompanied by deliberate changes in the timing of childbearing or completed family size.

II.B. Research Challenges

While an ever-expanding number of animal experiments indicate that the effects of prenatal and childhood environments can be transmitted to later generations,[5] human studies are nearly non-existent. Social scientists have documented that health and economic status persist across multiple generations (Solon 2015; Clark 2014), but, as with first generation studies, isolating causal mechanisms in humans is challenging because social scientists' opportunities to manipulate early life conditions are more constrained. A second challenge is that data availability is limited. Multi-generational studies require information spanning many years, and containing detail on both individuals' outcomes and their parents' *childhood* circumstances. There are currently few such datasets in the United States, and most of them are small, so given the sources of variation that are typically used in natural experiment estimation strategies, it is difficult to obtain statistically precise estimates. Some researchers are beginning to link administrative data --mostly outside of the United States-- but to date, the opportunities for multi-generational data analyses within a causal research framework have been limited.

II.C. Evidence

[5] Useful reviews of this literature include Daxinger and Whitelaw (2010; 2012), Heard and Martienssen (2014), Hochberg et. al. (2011), Nadeau (2009).

In this section, we describe current natural experiment research on the extent to which the effects of early life environments persist to later generations. We include all multi-generational studies we are aware of that employ the types of research designs described above, but we note that due to the data challenges associated with analyzing multi-generational effects, the extent to which researchers are able to employ the full range of robustness tests that prevail in the best first generation studies varies-- this is very much an emerging literature. We summarize the empirical evidence in Table I, and briefly describe the studies below. Our discussion focuses on how the emerging literature speaks to the potential linkages laid out in Section II.A, and highlights issues that are relevant to obtaining a better understanding of generational persistence.

II.C.i. Famine

A few studies have been able to extend the use of historical famines to examine how such nutritional "shocks" to the childhood environment affected the next generation. Using a research design similar to that described in Section I.B., Painter et al. (2008) investigate the multi-generational impacts of the Dutch Hunger Winter. They find no evidence that *in utero* exposure to the famine affected mothers' reports of the next generation's birth weight or incidence of prematurity, but they do find that mothers reported reductions in the second generation's birth length, and increases in predictors of the second generation's later life obesity, such as neonatal adiposity. Mothers who were exposed in the first generation also reported that their offspring experienced poor health in adulthood at nearly twice the rate as the children whose mothers were not exposed. These estimates should be interpreted with a degree of caution because they are based on a small sample, and parents' recollections of their children's health. Nevertheless, patterns in the estimates may provide clues to the underlying mechanisms: the effects are largest among first generation mothers who were exposed to the famine near the beginning of gestation,

and the authors find no evidence that fathers' exposure to famine affected later offspring. Interestingly, the differences in poor health are *not* clearly due to conditions related to metabolic syndrome or psychiatric conditions, which have been generationally linked to nutritional deprivation in animal studies.

Van den Berg and Pinger (2016) invoke a related identification strategy based on the German famine of 1916-1918 to investigate generationally persistent effects resulting from food restrictions experienced during the period just before adolescence (often called the Slow Growth Period).[6] Notably this is one of only a few papers to examine the effect of a negative childhood shock outside of the first few years of life: the authors' focus on this later developmental period stems from a series of small sample [7] studies of data from the Overkalix region of northern Sweden suggesting that the slow growth period may be a period of increased sensitivity for epigenetic imprinting (Pembrey 2002, Cooney 2006). Also motivated by the Overkalix studies' findings, and previous research suggesting that some heritable epigenetic modifications may be sex dependent (e.g. Hochberg 2011, Pembrey et al. 2006), van den Berg and Pinger explore gendered linkages between low maternal grandmothers' (paternal grandfathers') food access during the years right before adolescence and granddaughters' (grandsons') outcomes. They hypothesize that because the period immediately prior to adolescence is a sensitive period for the methylation of male sperm (but not for the development of female eggs), only male slow growth period famine exposure should affect later generations.

[6] Like the Dutch Hunger Winter, the German famine was severe and sharply delineated in time, making it a useful natural experiment.

[7] Approximately 300 offspring and grandchildren of parents and grandparents born in 1890, 1905, and 1920.

The authors compare second and third generation's adult height, mental health,[8] and educational outcomes, based on the age of famine exposure among the first generation. Importantly, their analyses rely on the assumption that there were no differences in survival between those who were exposed during their slow growth period and those who were exposed at earlier or later stages of development. In contrast to Painter et al. (2008), van den Berg and Pinger's second generation analyses are more mixed, with no evidence that maternal exposure affects later offspring outcomes, and inconsistent patterns associated with paternal exposure. In addition, the authors do not find statistically significant effects on the third generation's height or education. However, they do find evidence of the types of gendered relationships described above when they focus on mental health. Specifically, paternal grandfather exposure to the famine during his slow growth period is *positively* associated with grandsons' mental health, while maternal grandmother exposure is *positively* associated with the mental health of granddaughters. Controlling for family economic circumstances does not reduce the magnitude of the coefficient estimates, suggesting that the effects are due to direct biological processes, which, because of the gender-specific patterns, the authors interpret as evidence of epigenetic mechanisms.

Several studies of the Chinese famine find evidence of multi-generational effects. These studies all use a similar approach of comparing the offspring of cohorts conceived before, during, and after the 1959-1961 Chinese famine and utilizing geographic variation in the famine's intensity. Almond et. al. (2010) find that treated generation's fetal exposure to malnutrition increases the likelihood of female births in the second generation, which is consistent with the Trivers and Willard (1973) prediction that poor fetal conditions should favor daughters.

[8] Mental health is measured by the Mental Component Summary Scale, which is based on a factor analysis of inputs measuring general mental health, emotional functioning, social functioning and vitality.

Extending the research design to consider later life outcomes, Kim et al. (2014) find that the second generation is less likely to attend junior high school, and Fung and Ha (2010) find that the second generation has lower height-for-age and weight-for-age. Given that mortality was high in the famine period, Kim et al. also consider selective survival as a potential mechanism, and determine this would bias against their findings, since the evidence suggests that mortality was higher among the less advantaged, whose baseline health is generally lower than other groups.

II.C.ii. Disease Exposure

Several researchers have extended the first generation literature on the long-term effects of early life exposure to infectious disease to consider effects on later generations. Using generationally linked Swedish administrative data, Richter and Robling (2013) analyze outcomes among the children of those who were differentially exposed to the 1918-1919 influenza pandemic *in utero.* The sheer number of observations (a 35% random sample of all individuals born in Sweden between 1932-1967) gives the researchers the statistical power they need to identify the effects of exposure at different stages of fetal development. Like van den Berg and Pinger (2016), they find evidence of gendered linkages across generations. Specifically, they find that mothers' (fathers') *in utero* exposure to the Spanish flu reduced educational attainment among female (male) offspring by 1.8% (2.1%), and reduced the probability of college attendance by 12% (12%). They find no evidence that these impacts translated into lower earnings, however, which could be due to the fact that Sweden has a compressed earnings distribution. The effect seems to be driven by the first generation's exposure during the second trimester.

Also like van den Berg and Pinger (2016), Richter and Robling compare multi-generational estimates based on analyses that do, and do not, control for parents' schooling levels. Their results are somewhat harder to interpret, however, as they find no evidence that mothers' education explains the persistent effect on daughters, while controlling for father's education does eliminate the estimated link between father-son pairs. Thus, a superficial interpretation of their results would be that biological processes underlie the transmission from mothers to daughters, whereas socioeconomic processes underlie the transmission from fathers to sons. Another point of interest is that when the authors find significant effects on the treated generation (only in some specifications), it is first trimester exposure that appears to be important, in contrast to the primary influence of second trimester exposure on the second generation. The authors argue that if non-biological mechanisms were driving the generationally persistent effects of in *utero* flu exposure, then the relevant trimester would be the same for both generations.

Almond, Currie and Herrmann (2012) use U.S. Vital Statistics data to investigate generationally persistent impacts of broader disease exposure. They examine how state level variation in infant mortality rates at the time of the mothers' birth relate to their offspring's likelihood of being low birthweight. Among whites, they find that higher infant mortality in the year after the mother is born is associated with an increase in the probability that her baby will be born below the low birth weight threshold. Among blacks, the estimates are opposite in sign. The authors posit that these racial differences in health effects are driven by racial differences in selective survival described in Section II.A. Specifically, the observed improvement in black infants' health is consistent with processes where the selection effect dominates the scarring effect, whereas for whites, the scarring effect dominates the selection effect. The dominating

processes may differ across racial groups because of differences in underlying health or access to health services.

II.C.iii. Radiation Exposure

Black et al. (2013) build on an existing medical literature documenting the detrimental effects of exposure to acute radiation (previously based on the survivors of the atomic bombs at Hiroshima and Nagasaki), by investigating the effects of *in utero* exposure to low-dose levels of radioactivity on later life cognitive functioning. The authors' research design is based on regional variation within Norway in *in utero* exposure to low levels of radioactivity that resulted from extensive nuclear weapons testing in Russia during the 1950s and 1960s. Norway's proximity to Russia made it particularly vulnerable to nuclear fallout, with differences in treatment intensity across locations due to differences in locations' wind, rainfall, and topography. This variation is applied to administrative data that includes IQ scores. A nice feature of the research design is that there was little public awareness of exposure, so regional differences in outcomes are unlikely to be driven by individuals' stress about the effects of radiation, or by avoidance behaviors.

Black et al.'s main analyses focus on the impacts of *in utero* exposure on the treated generation. Consistent with the medical literature, they find that *in utero* exposure during the 3rd and 4th month of gestation (but not other months) leads to a decline in first generation males' IQ scores.[9] In addition, they find an effect on the IQ of later sons that is about 60% as large as the "first generation" effect. This suggests that a substantial part of the adverse cognitive effects of *in utero* radiation exposure is passed from one generation to the next.

[9] IQ information is only available for men. However, similar effects are found for first generation women's exposure *in utero* on their son's IQ.

II.C.iv. Stress

Lee (2014) examines whether the first generation effects of stress exposure during the *in utero* period, spill over onto later generations. His natural experiment is based on the Kwangju Uprising in South Korea, which was unanticipated, short-lived (10 days), and geographically concentrated. He finds evidence that maternal exposure to stress while *in utero* has detrimental impacts on the next generation's birth outcomes, and that the effects are particularly strong when exposure occurs in the second trimester. There is no evidence that the changes are driven by changes in mothers' or fathers' socio-economic status.

Several drawbacks to Lee's data are worth noting because they highlight broader challenges in this literature. First, because there is limited information about fathers on the birth certificate, the analysis focuses on the effects of mother's exposure to the uprising. This is a common limitation encountered by analyses of administrative natality data, which aid researchers' ability to identify precise effects by providing large samples, but provide limited parental information, particularly for fathers. Second, the data do not provide information on mothers' location of birth, so it must be inferred based on the child's the location of birth. We discuss the potential impacts of data issues more below.

II.C.v. Positive Policy Interventions

An even smaller number of studies investigate multi-generational effects of positive health interventions, although these, too, find consistent evidence that the effects of early life environments persist to later generations. Analyses of positive interventions are particularly important because, along with providing additional information on the persistent effects of early life environments, they can provide important insights towards the design of effective anti-poverty policies by helping to more fully quantify the range of long run program benefits.

To date, only one study has investigated the effects of an intervention that targets the prenatal period. East et al. (2018) build on the existing literature documenting that Medicaid has substantive positive effects on children's long-term health and labor market outcomes, by further investigating the health of treated cohorts' offspring at the time of their birth. Like many first generation studies of Medicaid, their natural experiment is based on changes in eligibility rules during the 1980s and 1990s that lead to dramatic increases in individuals' prenatal and early childhood coverage, and were adopted differentially across states and time. Figure 4 provides some intuition about the "treatment" and "control" groups created through this research design. The states with the darkest shading are those that expanded coverage by the largest amount, and states with the lightest shading expanded coverage the least.

East et al. link "first generation" *in utero* and childhood Medicaid eligibility to later generation's outcomes through information on mothers' date of birth and state of birth available in the U.S. Vital Statistics Natality files. This allows them to determine mothers' likely eligibility for Medicaid at each stage of childhood. They find that mothers' early life Medicaid eligibility positively impacts their children's birth weight. The estimated effects of treated cohorts' *in utero* eligibility on later offspring are about ten times as large as the estimated effects associated with one additional year of eligibility later in childhood. Strong patterns in the estimates also point towards beneficial effects on offspring's incidence of low birth weight, very low birth weight, and likelihood of being born prematurely.

Other natural experiments investigate multi-generational effects of early life health interventions targeting older children. All of these studies also find evidence of positive effects, which has the important implication that generational trajectories can be altered even after birth. Almond and Chay (2006) study the multi-generational impact of Title VI of the Civil Rights Act

of 1964, which prohibited segregation and discrimination in hospitals receiving federal funds. Prior to Title VI, southern blacks were excluded from full access to hospital resources. Earlier work by Almond et al. (2006) documents a strong association between Title VI and reductions in racial gaps in infant mortality due to diarrhea and pneumonia, which made up a large fraction of infants' hospital treatments. Their experimental variation rests on the abruptness of the change in infant mortality after 1964, the sharp decline in death from specific conditions that were treatable in hospitals, and the contrast with small changes in infant mortality among whites. This same natural experiment is employed by Almond and Chay to examine the health of the descendants of black and white women who were differentially exposed to Title VI. Applying this strategy to the outcomes of the next generation, Almond and Chay find that black women born after hospital integration were less likely to have low birthweight infants than black women born earlier: specifically, the treated generation's access to better quality health care reduced the black-white gap in very low birth weight incidence among the second generation by 30%.

Two studies by Butikofer and coauthors examine the effects of public health interventions on the intergenerational persistence of educational attainment. Estimating the impact of an intervention on the *intergenerational transmission* of outcomes is a related, but different, concept from estimating the direct effect on later generations' outcomes. Specifically, the intergenerational transmission coefficient for a particular outcome can be thought of as the correlation in that outcome between fathers and sons. High correlations between fathers and sons indicate that family background is a strong predictor of the next generation's "success," whereas small correlations indicate that parents' status is not as important—in other words, there is more equality of opportunity when intergenerational correlations are low.[10] Butikofer et al.

[10] In many ways, a policy's effect on equality of opportunity is more directly measured by its effect on the intergenerational correlation than by its effect on later generation's aggregate outcomes. Estimating

(2017) consider the introduction of Norwegian mother and child health centers, exploiting the fact that the timing of center openings varied substantially across locations, while Butikofer and Salvanes (2015) study the effects of a Norwegian tuberculosis control campaign making use of the fact that the tuberculosis control campaign should have had a bigger effect in geographic areas that had higher pre-campaign tuberculosis levels. Although both studies focus on how the interventions improved treated cohorts' later life health and earnings, they also find that the interventions reduced the intergenerational persistence of educational attainment by 10-14 percent.

Two recent analyses of early life interventions that provided a combination of health and education services also find evidence of generationally persistent effects (Barr and Gibbs 2017; Rossin-Slater and Wüst 2016). These studies build off of an existing literature documenting that cohorts exposed to high-quality preschool programs (especially Head Start) experience better long-term effects (e.g. higher levels of schooling and earnings, better health, and lower likelihood of engaging in risky behaviors). Both studies create comparison groups using geographic variation in the roll-out of targeted, high-quality preschools that improved poor children's health environments by providing nutritional, dental, and other health-related services. Barr and Gibbs investigate the effects of the U.S. Head Start program, which was adopted by different counties in different years throughout the 1960s, as part of the U.S. War on Poverty. They find evidence of a significant impact of Head Start availability on a summary measure of well-being that combines the next generation's educational attainment, incidence of teen pregnancy, and interaction with the criminal justice system. Specifically, the children of women

intergenerational correlations requires data that contains information on the same outcome for both children and their parents, however. Most U.S. datasets that contain generationally linked outcomes are small, which limits the extent to which natural experiments can be applied to produce precise estimates. Several Scandinavian countries have linked administrative data across generations, which has allowed for more progress on this front.

who lived in counties with a Head Start program during their preschool years score about 0.25-0.45 standard deviations higher on the index than the children of women who did not have access to Head Start when they were young. Rossin-Slater and Wust exploit variation across municipalities in the timing of government approved preschool openings during the period between 1930 and 1960. They find that children of women who had access high quality preschool by age 3 have 0.4 percent more years of schooling and are 6 percent less likely to only have a compulsory level of education at age 25.

III. Discussion

Taken as a whole, there is emerging evidence that early childhood environments have substantive spillover effects onto later generations. This is true not only for severe negative health shocks, but also for positive policy interventions. In terms of magnitudes, studies find that the effects on second generation outcomes are either smaller or similar in magnitude than the effects on the treated generation, suggesting that the transmission coefficient is at most about one.[11]

East et al. (2018) find the effect of early life access to Medicaid on low birth weight incidence in the next generation is about 40% of the effect on the same outcome for the treated generation. This is roughly consistent with Currie and Moretti (2007), who find that the probability of being a low birth weight infant is nearly 50 percent higher among children whose mothers were themselves below the low birth weight threshold. Similarly, Black et al. (2013) find that about 60% of the effect of radiation on the first generation's IQ score is transmitted to the second generation. Rossin-Slater and Wüst (2016) find that the effects on the second

[11] Not all studies estimated the effects on the first and second generations, so we focus on those that did in this discussion as well as on studies where there is an analogous estimate on first generation outcomes in another paper.

generation are 27-107% of the magnitude of the effect on the first generation, depending on the measure of education examined. Given the wide range of these estimates it is hard to draw comparisons to estimates of the intergenerational correlation in educational outcomes, but to provide some context, in the U.S., the intergenerational correlation in parent-child schooling is about 0.46, and is similar in Denmark where Rossin-Slater and Wüst's study is conducted (Hertz et al. 2007). A number of other studies also find effects that are similar in magnitude for the first and second generation (Painter et al. 2008; Richter and Robling 2013; Barr and Gibbs, 2017). Studies that investigate the effect of public health investments on the intergenerational transmission of education find that they reduce the intergenerational *transmission coefficient* by between 0.10 and 0.14 depending on the study (Butikofer et al., 2017; Butikofer and Salvanes, 2017). One additional point of interest is that Van den Berg and Pinger (2016) find no evidence of first generation's adolescent exposure to famine on second generation's outcomes, but they do find effects on third generation outcomes, suggesting a second to third generation transmission coefficient of zero. Although again this is the only study to focus on the adolescent period of exposure.

These studies indicate that public investments in early life health may have persistent and substantive impacts beyond the treated generation. An important implication is that benefit/cost ratios based only on cohorts immediately affected by program interventions will underestimate their overall efficacy- *even when taking treated generation's long-run benefits into account*.

The dollar value ascribed to those benefits will strongly depend on how the future benefits are discounted. There is an active literature in economics that considers appropriate discount rates with respect to interventions with long-run impacts, especially regarding climate

change (e.g. Weitzman 2013), but using discount rates in the mid-range of this literature suggests that the magnitude of these previously ignored multi-generational benefits may be non-trivial.

For example, in their study of the multi-generational effects of early life access to Medicaid, East et al. (2018) use the 3% discount rate chosen by the U.S. Government Interagency Working Group on the Social Cost of Carbon, and they estimate that even when benefit calculations are restricted to the those associated with low birth weight in the first year of life, the medical cost savings generated by the second generation's improved health in *the first year of life alone* may be roughly 30% of the cost of the initial investment. This calculation ignores the additional medical cost savings beyond the first year of life that result from any health improvements that are associated with reductions in low birth weight, and they ignore increases in later life earnings and tax revenues that accompany higher birth weights (e.g. Black et al. 2007, Bharadwaj et al. 2018).

While, to our knowledge, this is the only multi-generational study to date that has explicitly considered the dollar value of second generation benefits relative to program costs, the magnitude of second generation effects estimated for some other U.S. early life interventions, also hints that there may be high returns. Barr and Gibbs (2017), for example, estimate that the first generation's participation in the Head Start program increased the second generation's probability of completing high school by roughly 25%. Tamborini et al. (2015) estimate that the difference in the average present discounted value of lifetime earnings for a female completing high school, compared to dropping out of high school, is $0.14 million (2012 dollars). Using this descriptive evidence, a very rough estimate of the expected earnings benefit to the second generation is 0.14 million*0.25 = $35,000.[12] In contrast, in 2013 federal per pupil Head Start

[12] The gain for males earning a high school degree is $0.18 million, so the benefits are similar for males. Note that these are not *causal* estimates of the effect of a high degree. Additionally, Barr and Gibbs find changes in college

expenditures were about $8,000 (U.S. Department of Health and Human Services, Administration for Children and Families 2013). Of course, because of the time lags required to measure program effects on later generations, even thoughtful benefit/cost calculations should be interpreted cautiously when applying them to changes in policy today because present day program parameters and contextual environments will be different from the treatment period.

Because the causal literature on the multi-generational effects of early life environments is still in its infancy, we are still very far from pinpointing underlying processes. As described earlier, an extensive literature has documented that a variety of environmental conditions affect treated generations' physical health, mental health, education levels, and earnings—all of which have the potential to influence the well-being of later offspring, and most of which are likely to be correlated with each other. This, together with many data constraints, have prevented thorough analyses of the candidate mechanisms.

The literature to date does provide suggestive evidence that there may be important mechanisms besides the early environment's effects on treated generations' socioeconomic status: most studies that have controlled for the first generation's education and/or earnings have found that their estimated effects on later generations' outcomes are robust to the inclusion of these variables.[13]

Some studies of multi-generational effects find suggestive evidence that changes in maternal health and health behaviors may play a role. Almond, Currie and Herrmann (2012), for example, document the infant mortality rate in early childhood is positively associated with the later incidence of diabetes, high weight gain and smoking during pregnancy, although

attendance, so the benefits may be even larger than implied by this back of the envelope calculation focusing only on changes in high school graduation.

[13] As noted earlier, an exception is Richter and Robling (2013), who find that controlling for fathers' education eliminates the persistent effects of flu exposure between fathers and sons (but not mothers and daughters).

controlling for these outcomes does not explain all of the effect of the disease environment on later generations. On the other hand, East et al. (2018) find no changes in maternal health or behaviors among mothers who had *in utero* Medicaid access. Almond and Chay (2006) find that hospital integration reduced mothers' medical risk factors, but do not investigate the extent to which this explains their second generation effects.

Another possible channel --that is relatively easy to observe-- is changes in fertility. Many of the studies described above do not perform direct tests for changes in first generation fertility, however. Among those that do, there is little evidence that early life environments change overall fertility (East et al., 2018; Rossin-Slater and Wüst, 2016), but this possible mechanism deserves further investigation.

Another channel that can often be observed is a change in the *composition* of women giving birth. Even if aggregate later life fertility is unaffected by early life health environments, the *types* of women giving birth may be affected. Many of the multi-generational studies described in this paper try to get a sense of the importance of this potential mechanism by including controls for the first generation's demographic characteristics, to see how much their inclusion changes the estimated effect on future generations. The results are wide-ranging, with some studies finding that compositional changes may drive part of, but not all of, the generationally persistent effect (Almond, Currie and Herrmann, 2012; East et al., 2018) and others' finding little supportive evidence (Van den Berg and Pinger, 2016).

Finally, as discussed above, selective survival could be another important channel influencing later generations' observed health. In particular, the extreme negative shocks that some studies have harnessed to create treatment and control groups also led to large changes in contemporaneous mortality rates. This may have had a mechanical, positive influence on the

next generation's observed health (through selection into birth). To date, however, Almond, Currie and Herrmann (2012) are the only authors to find potential evidence that this type of selection may influence second generation effects.

Thus far, data constraints have limited our ability to fully investigate early life environments' generationally persistent effects, and underlying mechanisms. Datasets that include information on both parents and their children are typically small, limiting ability to precisely detect multi-generational linkages. As the United States moves towards other countries' willingness to invest in linked administrative data, we may be able to learn more, but administrative data often lack sufficient detail to speak to mechanisms. Efforts to move forward on this front will require creativity.

As this literature continues to grow and more data becomes available, we make several recommendations for future researchers to consider when conducting multi-generational studies. First, the data available for second generation analysis is often limited, but would in most cases allow for an investigation of changes in first generation fertility and the composition of births in the second generation. This is crucial for understanding the mechanisms behind the effects observed in the second generation. Second, to the extent the data allows, researchers should investigate the direct effects on the first generation, focusing in particular on outcomes which are known to be predictive of children's outcomes, and explore the extent to which these first generation effects can "explain" the second generation effects. This may allow for the identification of intermediate outcomes, which if affected in the first generation, are very likely to have spillover effects to the second generation. Gaining an understanding of these types of "early warning indicators" would allow research to make stronger statements about the potential

effect of current, or more recent programs on future generations.[14] Finally, if direct comparisons can be made on the magnitude of the effect on first and second generations' outcomes (either within studies or across studies), researchers should do this as it will help to build a more complete picture about the intergenerational transmission of health and economic outcomes.

Although there is still much to be learned, the analyses described in this article offer an important perspective on inequalities and the potential role for government intervention. Generational persistence in the impacts of early life environments suggest that historical differences in fetal health conditions between advantaged and disadvantaged groups may undermine contemporaneous efforts to close health and economic gaps. At the same time, these early results indicate that early life health investments have payoffs that extend well beyond those that social policymakers usually consider.

[14] We thank Jens Ludwig for making this point.

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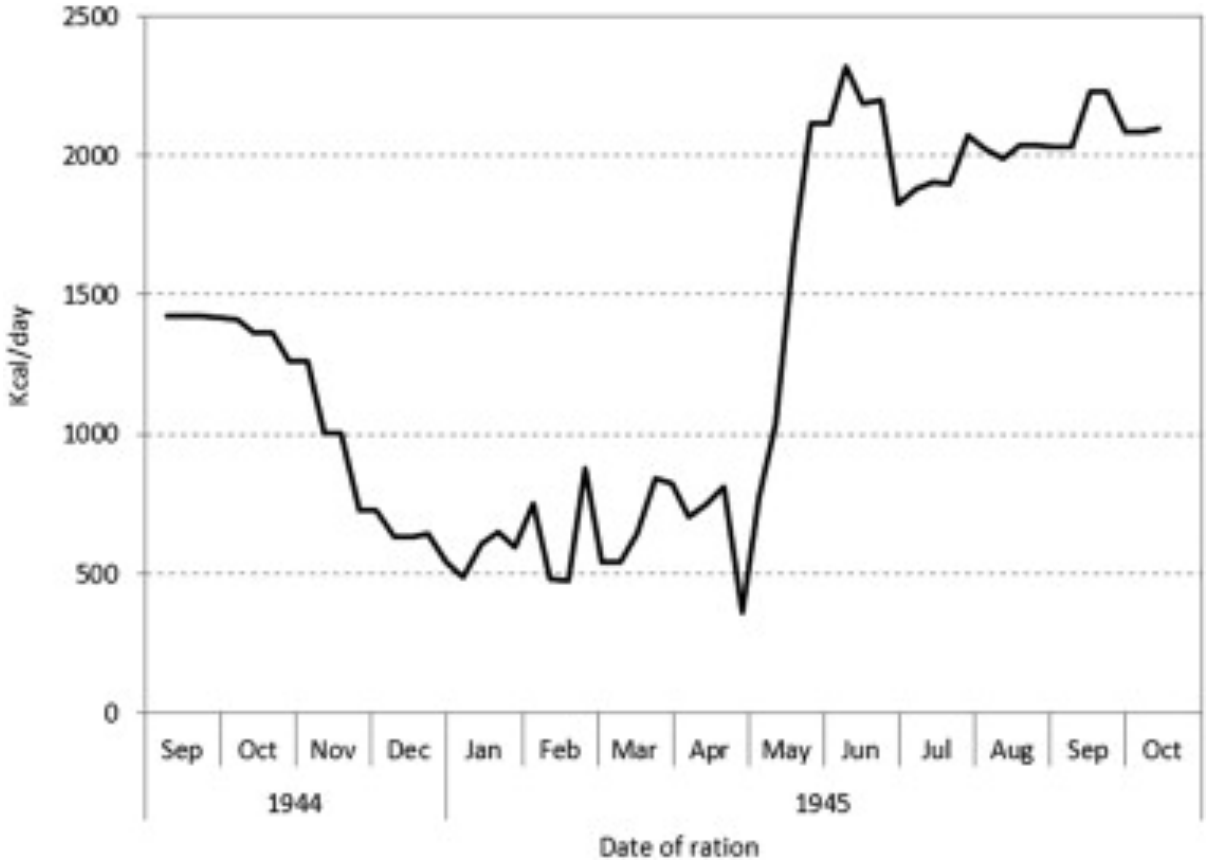
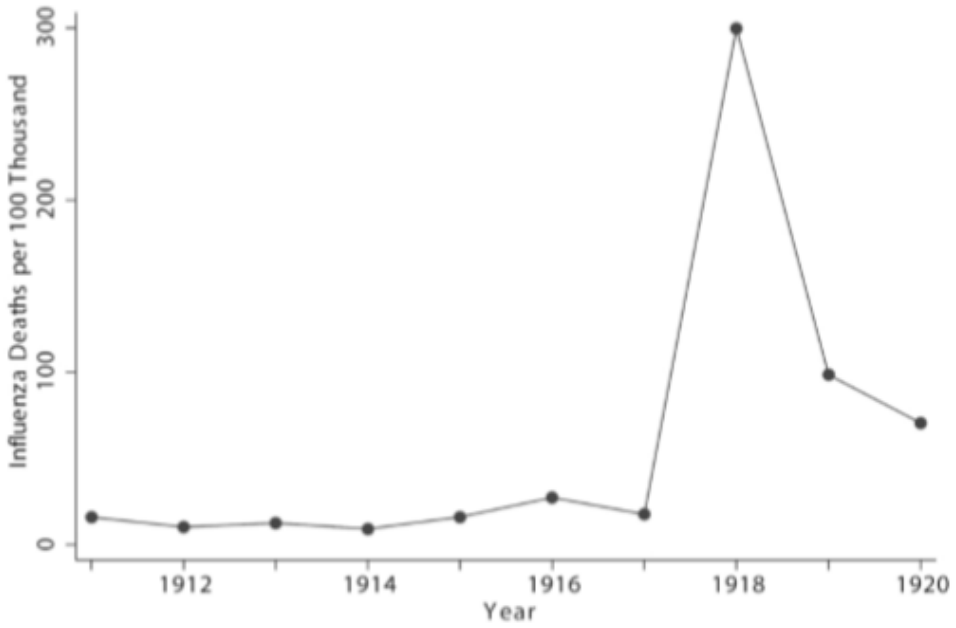
Figure 1. Official Food Rations During the Dutch Hunger Winter (in kcal)



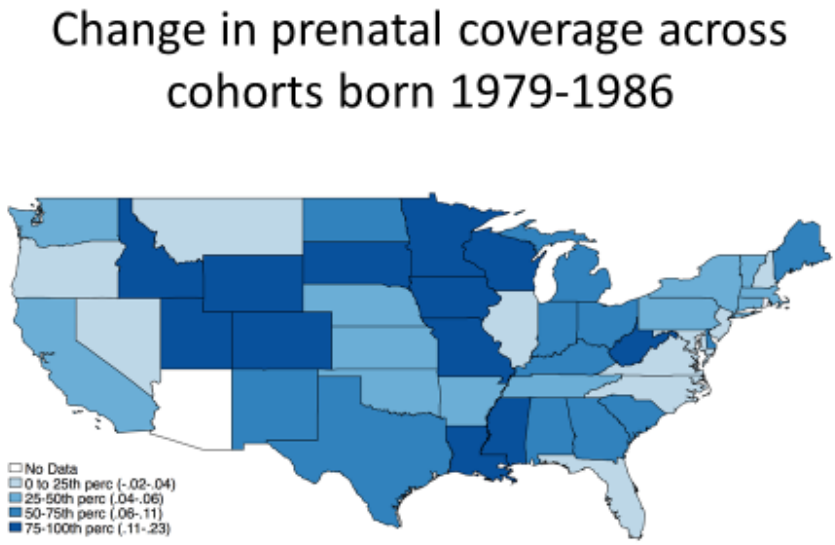
Source: Ekamper et al. (2014)

Figure 2. Timing of the 1918 Influenza Epidemic



Source: Almond (2006)

Figure 3. Change in Medicaid prenatal coverage across cohorts born 1979-1986



Source: East et al. 2018

Table 1. List of Multi-generational Papers

Study	Data	Empirical Strategy	Results	Interpretation/Mechanisms/Heterogeneity
Painter et al. (2008) Examines effects of Dutch Hunger Winter on children of individuals exposed in utero	Interviews of the Dutch Famine Birth Cohort, who are individuals born in Amsterdam between 1943-1947 First generation N=655	Difference-in-difference design using the Dutch Hunger Winter start and end dates to examine effects of parents' in utero exposure to famine on children's short and long-run outcomes. During this period, the food consumption of a previously well-nourished population was reduced by more than 75%, with rations limited to 1000 calories per day by the end of November 1944, and 500 calories per day by May 1945, when the war ended.	No effect on next generation's birth weight or incidence of prematurity. Reductions in the second generation's birth length, increased ponderal index, and neonatal adiposity, which is a predictor of later life obesity. Doubling of poor health in adulthood, although the differences in poor health are not clearly due to conditions related to metabolic syndrome or psychiatric conditions (which have been generationally linked to nutritional deprivation in animal studies).	Exposure to the famine during the period of early gestation was most important to the next generation's health in later life. No evidence of intergenerational transmission among first generation men who were exposed to the famine.
Van den Berg and Pinger (2016) Examines long-run effects of adolescent exposure to the German famine on the second and third generations' outcomes.	The German Socioeconomic Panel (SOEP), Sample is second and third generation individuals whose parents/grandparent were born in 1902-1913.	Difference-in-difference design using the German famine start and end dates to examine effects of the first generation's exposure to famine just before adolescence on their children and grandchildren's adulthood outcomes.	No statistically significant effects on the third generation's height or education. Gendered effect on mental health outcomes—famine during the paternal grandfather's (grandmother's) pre-pubescent period is	No evidence of effects on the second generation. Controlling for family economic circumstances does not reduce the magnitude of the coefficient estimates, suggesting that the effects are due to direct biological mechanisms.

	Third generation sample N=2670 Second generation sample N=6548		associated with worse mental health outcomes for grandsons (granddaughters). 16% of a standard deviation for males. 22% of a standard deviation for females.	
Almond et al. (2010) Studies the long-run consequences of in utero exposure to the Chinese famine including sex ratio and other bith outcomes of the second generation	China Census of Population in 2000 Sample is those born 1954-1964 N~750,000 Hong Kong Natality data Sample is those born 1957-1965 N~600,000	Difference-in-difference design using the Chinese famine. The authors' model the intensity of the famine based on mortality rates around the date of birth, as well as location-specific mortality rates matched to the individuals' place of birth for some outcomes. Among residents of Hong Kong the authors compare migrants from mainland China to non-migrants.	Famine-exposed women had offspring which were 0.4 percentage points less likely to be male Among migrants to Hong-Kong, Famine-exposed women had offspring which were 8% more likely to be low birth weight and 1.2 less likely to be male	
Kim et al. (2014) Studies the effects of in utero Chinese famine exposure on second generations'	China Census of Population in 2000 Sample is children ages 13-15 whose mothers were born 1954-1966	Difference-in-difference design using the Chinese famine. The authors' model the intensity of the famine based on parents' location and date of birth	One standard deviation increase in mothers' mortality rate exposure reduces sons junior high attendance by 1.6-6%, and reduces female attendance by 1.4-7%.	Largest effects from in utero exposure to famine. Results similar when control for parental education. Effect of fathers' mortality rate exposure not robust to 2SLS.

medium-run outcomes.	N~40,000 second generation children	using local mortality rates. Also use 2SLS and instrument for mortality rates with weather.		
Richter and Robling (2013) Examines effects of the 1918 flu epidemic on children of individuals exposed in utero	Statistics Sweden multigenerational register Sample is individuals with both parents born between 1915-1920 N~60 thousand second generation individuals	Difference-in-difference design using the 1918-1919 influenza pandemic. Variation in exposure comes from birth date relative to start and end dates of the pandemic, as well as geographic variation in influenza incidence in the parents' location of birth.	Reduced second-generation offspring's educational attainment by 1.8-2.1%, and probability of college attendance by 12%. No effect on earnings.	This effect is gendered: only maternal (paternal) exposure affects daughters' (sons') outcomes. The effects are largest for parents exposed in the second trimester while in in utero. The effects for women appear to come from biological channels, whereas for men, accounting for the changes in the first generations' educational outcomes explains most of the effects found on the second generation.
Almond, Currie and Herrmann (2012) Investigates relationship between infant mortality rate in state-year of first generations' birth and first generation	1989-2006 U.S. Vital Statistics data Number of births~16 million	State-by-year variation in infant mortality rates (IMR) with state and year fixed effects. Look at the effect of IMR in year before, during, and after birth of the first generation.	An additional post-neonatal death in the year after the mother is born is associated with a 0.6% increase in the probability that her baby will be born below the low birth weight threshold.	Effects on whites's low birth incidence is 0.5% and effects on blacks -0.1%. Authors posit that differences by race may be due to differences in the effect of IMR on selective survival of first generation across races. Find changes in mother's incidence of diabetes, as well as educational attainment,

mother's long-run outcoms as well as second generation health at birth				marital status, weight gain during pregnancy, and smoking during pregnancy. Including these in the model slightly diminishes the effect on low birth weight for whites, but increases (in absolute value) the effect for blacks.
Black et al. (2017) Studies long-run effects of radiation exposure in first and second generation	Norwegian Registry Data. Include cohorts born 1956-1966 and their children. First generation males N=19079, second generation males N=24281.	Difference-in-difference design using variation across locations and over time in exposure to radioactivity with location and time fixed effects. Variation across locations comes from wind, rainfall, and topography.	In-utero exposure during pregnancy months 3 and 4 (but not other months) leads to a decline in the exposed generation's and second generation's IQ score for men. Effects sizes are 0.04 IQ points for exposed generation and 0.025 IQ points for the second generation. This implies an intergenerational transmission coefficient of 0.625	Data on IQ score unavailable for women as only in military records.
Lee (2014) Examines the outcomes of second generation children whose mothers and fathers exposed to	Korean Vital Statistics in 2000 and 2002 Sample is mothers' who were in utero at time of uprising and age 20-22 when giving birth	Difference-in-difference design comparing outcomes of children born in city of Kwangju to those in other locations, and comparing outcomes of children based on whether their	Maternal prenatal exposure to the Kwangju Uprising reduced birth weight of the next generation by 56 grams, reduced gestation by 2 days, increased likelihood of low birth weight and preterm birth.	Adding controls for mother's and father's socioeconomic status does not change the results. Largest effects from exposure in second trimester. Larger effects for second generation boys.

stress from the Kwangju Uprising	Second generation N~1 million	mother was in utero at time of Kwangju Uprising (10 days in May 1980)		
East et al. (2018) Studies the effect of early life Medicaid on second generation infant health outcomes	1994-2015 U.S. Vital Statistics data	State-by-year variation in eligibility rules for Medicaid and State Children's Health Insurance Program with state and year fixed effects. Look at the effect of generosity in utero and at ages 1-18.	Mothers' early life Medicaid eligibility positively impacts their children's birth weight. 1 year of mother's in utero exposure increase average birth weight by 30 grams. 1 year of mother's childhood exposure increase average birth weight by 2.5 grams.	No evidence of changes in overall fertility or timing of births. Evidence that early life Medicaid increases the fraction of white births relative to minority births, and the fraction of births to high school dropout women. Accounting for changes in mother's characteristics explains about a third of the health effects.
Almond and Chay (2006) Investigates the relationship between first generation access to integrated hospitals after birth and second generation infant health	1979-2000 U.S. Vital Statistics data	Double difference estimates comparing black and white women before and after Title VI of the Civil Rights Act of 1964, and triple difference adding in a comparison with foreign born women	Black women born after hospital integration were less likely to have low birthweight infants than black women born earlier. Reduced the black-white gap in very low birth weight incidence among the second generation by 30%.	Find reductions in medical risk factors for mothers
Butikofer et al. (2017) Studies the long run effects of	Norwegian Registry Data, cohorts born 1936-1960 still alive in 1967	Use differential timing of rollout of Norwegian mother and child health centers across locations	Centers reduced the intergenerational persistence of educational attainment by 10 percent.	Effects are only statistically significant for father-son educational transmission.

Norwegian mother and child health centers	N~300,000	with location and time fixed effects.		
Butikofer and Salvanes (2017) Studies the long run effects of Norwegian Tuberculosis control campaign	Norwegian Registry Data, cohorts born 1930-1945 N~440,000	Study the Tuberculosis control campaign in Norway. Variation in exposure comes from birth date relative to start date of program, as well as geographic variation in tuberculosis incidence in the location of birth.	14% reduction in intergenerational persistence in education from fathers to sons if the son is exposed to control campaign	
Barr and Gibbs (2017) Studies the effect of Head Start on second generation adult outcomes	NLSY79 and CNLSY First generation N~2400, Second Generation N~3500	Use differential timing of rollout of Head Start across locations with location and time fixed effects.	A significant impact of Head Start availability on a summary measure of well-being that combines the next generation's educational attainment, incidence of teen pregnancy and interaction with the criminal justice system. Mother's access to Head Start increased this index by 0.25-0.45 standard deviations.	
Rossin-Slater and Wüst (2016) Examines the long run effects of preschools in Denmark	Administrative population register data in Denmark First generation N~900,000	Use differential timing of rollout of preschools across locations with location and time fixed effects.	Children of women who had access to preschool by age 3 have 0.4 percent more years of schooling (not significant in all models) and are 6 percent less likely to only have a compulsory level of education at age 25.	Little evidence of selection into fertility. Results suggest transmission coefficient of education from first to second generation of 0.27.