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ABSTRACT

A large literature has documented links between harmful early life exposures and later life health and socioeconomic deficits. These studies, however, are typically unable to examine the possibility that these shocks are transmitted to the next generation. Our study traces the impacts of in utero exposure to the 1918 influenza pandemic on the outcomes of the children and grandchildren of those affected using representative survey data from the US. We find evidence of multigenerational effects on educational, economic, and health outcomes.

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A data appendix is available at http://www.nber.org/data-appendix/w25377

Introduction

Understanding persistent poverty and poor life outcomes has posed significant challenges for social science theories as well as crafting policy responses. Of particular interest is the so called "long arm" of childhood circumstances, where events and conditions early in an individual's life (even in utero) may set in motion an accumulation of disadvantage. The Developmental Origins of Health and Disease theory (often referred to as the Barker hypothesis) suggests that in utero insults program the fetus in ways that lead to maladaptive responses to the environment that persist throughout the life course and explain poor long term outcomes (Barker 2007). However, since the likelihood of experiencing an early insult is related to parental circumstances, there is an empirical challenge to causally examining the impacts of early insults. This issue ties into a key question of whether intergenerational persistence in poor outcomes may stem in part from genetic mechanisms that may be more resistant to policy efforts. In order to separate potential channels, some research has focused on exogenous (i.e. unrelated to genetics) health shocks during early life to explore long term outcomes.

Indeed, a strong causal link between harmful early life / in utero shocks and lowered later life health and economic outcomes has been found in a number of clearly identified empirical studies. In addition to separating genetic and non-genetic mechanisms, many of these investigations are also able to uncover biological (i.e. fetal programming) explanations by studying specific insults, including the effects of prenatal nutritional status and exposure to infectious disease. Without the use of experimental (i.e. animal model) research designs, these studies use so-called quasi-experimental designs by leveraging striking demographic events such as the Dutch famine and the 1918 flu pandemic (Almond 2006; Lindeboom et al. 2010). The

results generally suggest large, lifelong impacts of in utero insults on economic and health outcomes.

With these results established, a next question is whether they might persist into the outcomes of future generations. We explore the question of intergenerational persistence of fetal insults by extending previous work that uses the 1918 influenza pandemic as a natural experiment (Almond 2006) by adding multigenerational data. We document that in utero exposure to the pandemic can be seen in the educational attainments of multiple generations. More specifically, we find a reduction of approximately $1/10^{th}$ a year of schooling (statistically insignificant) for the first generation, $1/5^{th}$ a year of schooling for the second generation, and $1/6^{th}$ of a year of schooling for the third generation.[1] We also show large effects on economic as well as health outcomes for the second generation. Our findings suggest the importance of in utero health insults that persist across multiple generations and allow a shift in our analytical frame from the "long arm of childhood circumstances" to the "long arm of previous generation's circumstances," or, alternatively, "the long reach of history" (Kuzawa and Eisenberg 2014). Our results can also be interpreted in the context of assessing benefits of policy and environmental conditions that reduce the likelihood of in utero health insults, where the full benefits may unfold over multiple generations.

Background Literature

Our research intersects several related literatures. There is a large literature that documents associations between early life insults and later life outcomes, following the original

[1] As mentioned in greater detail in 3.1, the first generation is comprised of the parents from the Wisconsin Longitudinal Study (WLS). This is the generation that is treated, or exposed in utero to the 1918 flu epidemic. The second generation is the WLS graduates and siblings, who are the focus of the WLS, and the third generation is comprised of the offspring of the WLS graduates and siblings.

Barker Hypothesis. In addition, we draw on literature that explores multigenerational linkages of environmental exposures on life course outcomes. These literatures are rarely unified in a single analysis, largely due to data constraints. We overcome these limitations by leveraging a three-generational dataset that coincides with a key demographic event from the early 20th century—the 1918/1919 influenza epidemic.

The seminal work of the economist Douglas Almond (2006) was one of the first papers to exploit the 1918 flu pandemic as a natural experiment and to test whether in utero exposure to infectious disease influences later life outcomes. Using US census microdata to situate respondent births in both time (birth quarter) and place (exploiting geographic variation in the severity of the flu), Almond (2006) finds evidence of lower educational attainment, higher rates of physical disability, as well as lower income and socioeconomic status for cohorts exposed to the 1918/1919 flu while in utero (estimated using birth quarter, age, and timing of the flu) as compared to unexposed adjacent cohorts. In a related paper, Almond and Mazumder (2005), use a similar approach with the Survey of Income and Program Participation and find negative health effects for adults who were in utero (based on birth quarter) during the outbreak. Subsequent work has built on these outcomes, finding excess cardiovascular disease (Mazumder et al. 2010); increased likelihood of kidney disease, diabetes, circulatory, and respiratory problems in old age (Lin and Liu 2014); and increased old-age mortality in non-cancer related causes (Myrskylä et al. 2013).[2] More broadly, the negative effect from the flu dovetails with other negative in utero shocks; see for example, Catalano et al. (2011), Roseboom et al. (2001), Schulz (2010), and Torche (2011).

[2] Though Cohen (2010) and Fletcher (2014) found no effects on overall mortality.

The mechanisms underlying these broad effects from in utero influenza exposure are not fully understood in humans. However, in studies of monkeys (Kobasa et al. 2007) and mice (Kash et al. 2006), it has been shown that the reconstructed 1918 flu virus triggers an exceptionally intense and prolonged innate immune response. More specifically, gene-expression analysis shows that proteins involved in the innate immune system have a higher and sustained expression when triggered by the 1918 reconstructed virus than when triggered by the contemporary flu of the same H1N1-type (Kobasa et al. 2007). While the specific 1918 flu strain cannot be studied in humans through purposeful exposure for ethical reasons, both animal and human studies have linked the maternal immune response during severe flu infection in pregnancy to offspring brain development as well as impaired adult behavior and cognitive outcomes (Brown and Derkits 2010; Canetta and Brown 2012; Fatemi et al. 2002; Li et al. 2014; Miller et al. 2013). These results align with research in psychology that links inflammatory processes in mothers—including stress and infection, as well as stress-induced immunosuppression—to poor birth outcomes (for a review, see Schetter 2011). Recent evidence suggests that in utero exposure to the 1918 flu may not only affect adult outcomes, but also the outcomes of subsequent generations. Richter and Robling (2015) were the first to identify an effect of first generation prenatal exposure (using birth trimester) to the 1918 flu pandemic on the outcomes of the second generation (the children of those exposed to the 1918 flu in utero). The authors use historical influenza morbidity data matched to birth information to identify potential exposure to the 1918 flu and find that first generation maternal in utero exposure in the second trimester lowers educational attainment for female children by 2-2.5 months, or by 1.5-1.8 percent, but find no such effect for male children. An analogous result is identified for first generation paternal exposure and male outcomes; that is, first generation exposure in the second

trimester lowers educational attainment for male children by 2.4 to 3 months, or by 1.8-2.2 percent, but paternal exposure showed no such effect for females. Taken together, first generation exposure to the 1918 flu while in utero results in 2 to 3 months less schooling for the second generation.

Several pathways exist for the intergenerational transmission of early life health shocks. Through socioeconomic channels, intergenerational persistence in poor outcomes could occur when a fetally insulted parent, marred by poorer health and socioeconomic outcomes, raises a child in a low-resource environment. Biologically, phenotype-to-phenotype transmission and epigenetic inheritance are hypothesized to be key mechanisms for intergenerational transmission (Kuzawa and Eisenberg 2014). In cases of in utero or very early life shocks, phenotype-to-phenotype transmission impacts the outcomes of the next generation through changes in parental biological systems that lead to altered gestational and/or lactation environments for offspring (e.g. pre-pregnancy hypertension is linked to low birthweight). Importantly, socioeconomic and biological channels are not mutually exclusive and can also interact with one another; that is, the effects of adult phenotype on offspring extend beyond physiology and metabolism to include parental behavior/environmental response as a potential source of phenotypic transmission and potentially even cumulative intergenerational phenotypic change (Benyshek 2013). For example, stress experienced by a mother prenatally may alter stress regulation in offspring, which may in turn increase risk for the same adult phenotype in the offspring as well as in subsequent generations. Epigenetic inheritance, in comparison, occurs when parental experiences alter gene expression that is subsequently transmitted to offspring and future generations through the germ line. Here, again, there is an opportunity for socioeconomic circumstances to interact with biology—e.g. where poverty, through the experience of stress, alters gene expression for

generations. Our analysis of second generation outcomes will explore some of these channels but not attempt to prioritize one over another.

Work extending effects of in utero exposure to the 1918 flu to the third generation (the grandchildren of those originally exposed in utero) in humans, to the best of the authors' knowledge, does not exist. The strongest evidence for a potential biological channel across multiple generations comes from studies of historical data from Överkalix region in Northern Sweden that exploit variance in first generation grandparental food supply during childhood. Bygren et al. (2001), for example, find that an excess of food during the period just before adolescence, a time labeled the "slow growth period" (SGP), shortens the grandson's longevity. A later study using the Överkalix data replicates the results of Bygren et al. (2001) in a second cohort, and further extends the results to include an association between first generation paternal grandmother's food supply and granddaughter's mortality risk (Pembrey et al. 2006).[3]

Our work builds off of this evidence to conduct novel examinations of multigenerational effects of in utero exposures in human populations. The current research leverages a unique survey to measure the multigenerational impacts of in utero exposure to the 1918 flu pandemic. Our hypothesis is that the previously documented direct effects of such shocks extend into the outcomes of the second generation. Going further, we also estimate whether these effects continue into the third generation. In other words, we ask whether the singular in utero shock has a multigenerational effect--on the adult outcomes of those exposed in utero, on their children,

[3] externally validated the potential for transmission across three generations by analyzing the impact of the German famine of 1916-1918 on the mental health outcomes (an index from survey questions accounting for 'general mental health', 'emotional functioning', 'social functioning' and 'vitality') of the children and grandchildren of those exposed to the famine during their SGP. The authors find that paternal (maternal) grandfather (grandmothers) exposure during their SGP is associated with better mental health in grandsons (granddaughters).

and on their grandchildren.

Data and Empirical Methodology

Data

In order to examine multigenerational effects of an in utero exposure, we require multigenerational data. Very few datasets in the US have a multigenerational component and fit the relevant time period for our exposure (i.e. birth cohorts around 1918). Our data come from the Wisconsin Longitudinal Survey (WLS), which is a random one-third survey of graduating high school seniors in Wisconsin in 1957. The majority of these respondents were born in 1939 and form our second generation. Thus, the parents of the WLS graduates form our first generation, with birth years overlapping the 1918/1919 in utero exposure period. This allows for the creation of our primary measure for in utero exposure to the 1918 flu epidemic: an indicator for either first generation parent being born during the 1918-1919 range.[4,5] Additional data are

[4] Parent year of birth is recorded from the WLS graduates. This self-reported measure is used to create the indicator of flu exposure: those parents born in 1918 or 1919. To maximize sample size, we use differing waves of the WLS and reported birth years from biological siblings to supplement the reported year of birth from WLS graduates. Parent birth years are first collected for the 1992/93 graduate wave. Missing observations are then sequentially added from graduate reports in later waves—2003 and 2011. After using all graduate reported year of parent birth variables, observations are then filled in from identically reported measures from a selected biological sibling.

Specifically, for mothers (gen 1 females), 7,976 observations come from graduates in the 1992/93 wave, 366 additional observations come from the 2003/05 wave, 84 observations come from 2011 wave, and 68 observations come from the selected sibling. This leaves a base sample size of 8,494, ~94% of which is from the graduate reported 1992/93 wave. For fathers (gen 1 males), 7,929 observations come from graduates in the 1992/93 wave, 361 additional observations come from the 2003/05 wave, 77 come from the 2011 wave, and 91 observations come from siblings, leaving a base sample size of 8,458 (again, ~94% is from the grad reported 1992/93 wave).

From this base, 7 observations are dropped for gen-1-female's (WLS mother's) year of birth due to reported gen 2's year of birth being 10 or less years after gen 1's year of birth; 45 additional observations are lost if this threshold is increased to 15 years. For gen-1-male's year of birth, 2 observations are removed for identical reasons; this increases to 25 additional observations for the 15 year threshold. We attribute this reduction in the sample mostly to measurement error.

[5] The 1918 flu epidemic in Wisconsin was from September 1918 through December 1918, but did not reach the severity experienced in many other states. According to historical records, Wisconsin had the fourth lowest numbers of deaths out of 25 reporting states (Burg 2000, Shors and McFadden 2009).

collected on the later life outcomes of the WLS graduates and a selected sibling, as well as a limited number of outcomes for the children (third generation) of the graduates/siblings, providing the structure for our multigenerational analysis.

Summary statistics are presented in table 1. As shown, approximately 10 percent of WLS parents are born in either 1918 or 1919. On average fathers are born in 1907 and mothers are born in 1911; consequently, a birth year of 1918 or 1919 is closer to the right-tail of the distribution of births and the rate of 10 percent is driven primarily by relatively young mother exposures.[6] Since WLS graduate's fathers tend to be on average 4 years older than mothers, this right-tail problem is larger for generation 1 males. This is seen in Figure 1. While not the primary focus of the WLS data collection, several parental (first generation) outcomes are available, including years of schooling, occupational prestige, and family socioeconomic status in 1957.[7]

[insert table 1 here]

The primary focus of the WLS data collection is high school seniors in 1957 Wisconsin, the second generation of our study. Given this focus on graduates (and their siblings), a large number of economic and health variables are available in each wave of the WLS (irregular intervals roughly 10-15 years apart: 1975/77, 1992/93/94, 2003-2005/2004-2007, and in 2011). In addition to examining years of schooling, several additional dependent variables in the second

[6] Appendix tables 7 and 8 provide evidence that our results are not driven solely by confounding between exposure to the influenza pandemic and being young mothers.

[7] The index of socioeconomic status is a factor weighted score combining data on father and mother's years of schooling, father's occupational prestige, and average parental income. Replacing this measure with average parental income (see appendix table 1) does not change the effect of the flu indicator. Job prestige measures for both mother and father are based on Duncan's Socioeconomic Index, which is a measure of job prestige based on income, education, and surveyed perceptions of general social standing for certain occupations (Duncan 1961).

generation intend to capture broad differences in economic and health well-being.[8] These include income during the peak earning years (i.e., family income collected when graduates are 53 years of age on average), net worth at initial retirement age (i.e., net worth collected when graduates are 65 years of age on average), and general indicators of health measured by body mass index and a count of doctor diagnosed illnesses (both collected in the wave when graduates are age 53 on average). Finally, the WLS data contains information that is collected from the second generation about the third generation; we focus on years of schooling as the main outcome of interest.

Empirical Methodology

Our empirical strategy follows that of Almond (2006) by examining harmful effects of being exposed in utero on later life outcomes. We then extend this analysis by estimating multigenerational impacts on both the second and third generations. In so doing, the primary estimating equation is given by the following form:

$$y_{if,g} = \beta_0 + \beta_1(YOB = 1918/19)_{f,1} + \boldsymbol{\gamma T_{f,1}} + \boldsymbol{\delta X_{if,g}} + \varepsilon_{if,g}$$

Our primary focus is on the coefficient β_1, which measures the effect of having a parent/grandparent born in 1918-1919 on a number of outcomes for i individuals in f families for generation g. Parent year of birth time trends and their square are denoted by $\boldsymbol{\gamma T_{f,1}}$; $\boldsymbol{\delta X_{if,g}}$ represents generation specific controls; and $\varepsilon_{if,g}$ is representative of a family clustered error term.

[8] We note that our three generations of individuals are drawn from three non-overlapping set of birth years; the mean birth year for generation 1 is ~1910, generation 2 is ~1940, generation 3 is ~1965. Thus, since we are performing the analysis separately by generation, we are controlling for much of the time-variation in the meaning of education. We also perform analysis stratified by sex in Appendix Tables 10-12, so that we can also control for the differential meaning of education in each generation.

For the first generation, we control for birth year and its square, capturing age-specific trends that are tied to our first-generation outcomes of interest (e.g. years of schooling). For the analysis of second and third generations, birth years and squares for both first generation parents are included as controls along with generation-specific controls for sex, age, and birth order.

Our estimation strategy follows an intent to treat design. It is likely that the actual incidence of flu differed by socioeconomic status and social standing (Mamelund 2006; Sydenstricker 1931). Our use of year-of-birth, however, avoids potential confounding by SES because all—both rich and poor—are defined as treated if they are born in one of the two years. Although avoiding confounders associated with actual disease incidence, this estimation strategy will underestimate the true effect of flu exposure, lowering the magnitude of our coefficient of interest.

Results

First Generation

Our initial analysis explores the direct effects of being born during the 1918 Influenza Epidemic. While health data are sparse for the WLS graduate's parents (first generation), a number of economic outcomes are available, especially during the initial sample year of 1957. Table 2 explores the relationship between these economic variables and an indicator for birth during 1918/1919. Columns (1)-(3) of table 2 show the relationship between a WLS graduate's father being born during 1918/1919 and the father's years of schooling, the father's occupational prestige, and the family's index of socioeconomic status in 1957 (i.e. when the first generation members are approximately 40 years old). While statistically insignificant, a negative association

is observed between years of schooling and being born in 1918 or 1919.[9] This is carried over into father's job prestige in column (2), from which the indicator of in utero exposure to the 1918 flu is associated with an approximate 0.1 standard deviation decline in the index of occupational prestige. These effects culminate in column (3), which shows a statistically significant negative effect of in utero exposure to the flu and later life economic well-being: a 1.3 decline in the SES index, which corresponds to a decline of roughly 10 percent of a standard deviation. The findings of table 2 corroborate past studies that show in utero exposure to the 1918 flu led to poorer economic outcomes later in life (Almond 2006).[10]

[insert table 2 here]

Mirroring the results for males in the first generation, a negative but statistically insignificant association is observed between an indicator of birth in 1918/1919 and schooling for first generation females. This association, however, becomes statistically significant at the 1 percent level for the index of family SES, the coefficient being nearly identical to that of a male's in utero exposure to the flu, though it is unclear whether these effects flow from labor market and/or marriage market sources. The findings of table 2 are extended in table 3, which explores potential marriage market effects of early life exposure to the flu epidemic.[11]

Column (1) of table 3 regresses the indicator for male in utero exposure on an identical measure for spouse's exposure. Females who were born in 1918/1919 were 5 percentage points more likely to marry men who were also born during the same period. Furthermore, as shown in

[9] First generation years of schooling are reported by the second generation WLS graduates. Measurement error is likely, which may result in the insignificant coefficients of table 2.
[10] Richter and Robling (2013; Table 12) find a similar effect for in utero female exposure to flu in the first trimester.
[11] Mothers and fathers of WLS graduates are assumed to be married.

columns (2) and (3), these women were more likely to marry men with fewer years of school and lower job prestige. These effects are significant at the 1 percent level. Similar effects are seen in columns (4)-(6) for 1918/1919 born males, who are 14 percentage points more likely to marry flu exposed females (p=0.00) and marry females with 0.26 fewer years of schooling (p=0.101). Given the findings of table 3, however, we cannot rule out that this is a marriage market effect.[12]

[insert table 3 here]

Second Generation

Our hypothesis is that the direct effects observed in tables 2 and 3 extend into future generations. To address this hypothesis, we regress a number of economic and health outcomes of the WLS graduates and siblings, the offspring of the first generation examined in tables 2 and 3. These estimations are performed in tables 4 and 5. Column (1) in both tables focus on an indicator for *either* parent being born in 1918/1919; the primary regressor of column (2) is an indicator for *father's* (gen. 1 males) in utero exposure to the 1918 flu; column (3) considers *mother's* (gen. 1 females) exposure; and column (4) includes separate indicators for both mother and father's exposure. Controls included in all columns include father's year of birth and its square, mother's year of birth and its square, an indicator for sex in the second generation, a measure of birth order in the second generation, and second-generation year of birth and its square.

Table 4 focuses on economic outcomes of the WLS graduates. To reiterate, our hypothesis is that in utero exposure to the 1918 influenza pandemic has effects that persist for multiple generations. Table 2 shows the direct, first generation effects, while table 4 begins to show the indirect effects that are transmitted to offspring. Panel A regresses years of schooling in

[12] Appendix table 2 repeats the estimation of table 3 while also controlling for an indicator of own-flu exposure and own year of birth measures.

the second generation on indicators of first generation exposure to the 1918 flu.[13] From column (1), either parent being born in 1918/1919 is associated with a statistically significant decline of 0.21 years of schooling in the second generation. This estimate is likely understated because the sampling design of the WLS is a focus on high school seniors; thus, individuals with fewer than twelve years of schooling are underrepresented in the data.[14] Columns (2)-(4) disambiguate this effect into the maternal and paternal lines. From which, the effect of column (1) seems to be driven by mother's in utero exposure to the flu.[15]

The findings of Panel A are extended into Panel B, which replaces years of schooling as the dependent variable with the natural log of family income for the 1992 wave, a time when the second generation respondents are 53 years of age on average and represents a time of peak earning in the life course. As with years of schooling, a negative association is seen throughout the specifications of Panel B. Having either parent born in 1918/1919 is associated with a 22 percent decline in family income. Once again, this effect seems to be driven by mother's, not father's, exposure. Panel C replaces income with a measure of net worth for the 2004 wave. This measure of net worth is when the second generation respondents are on average 65 years of age and is representative of earnings throughout the life course. The dual indicator of column (1) is negative but statistically insignificant at conventional levels (p=0.23). When looking at the effect of mother exposure in column (3), however, the effect becomes statistically significant at the

[13] Appendix Table 6 includes a number of alternative measures for years of schooling in place of the simple count used in Table 4.
[14] Table 3 includes selected siblings of the WLS graduates. These siblings do not have to be high school graduates.
[15] This finding differs from that of Richter and Robling (2013), who find maternal exposure tied to daughter outcomes and paternal exposure tied to son outcomes.

10% level (p=0.069), implying those with mothers born during 1918/1919 have approximately $36,000 less in net worth by 2004.

Following the broader health focus of the Barker hypothesis, the economic effects of table 4 are replaced with health measures in table 5. We consider three broad measures of general health and well-being later in life: self-reported health in later life (~53 years old), height, and BMI (again at ~53). The use of self-reported health is intended to capture general well-being later in life; this is tested in Panel A of table 5. Mazumder et al. (2010) provide evidence that in utero exposure to the 1918 flu is associated with a reduction in height and an increase cardiovascular disease, so it is possible that these negative health outcomes could be transmitted to offspring; this is tested in Panel B of table 5.[16] And given prior work linking early life exposure to the Dutch famine to BMI (Stein et al. 2007) and metabolic function (Lumey et al. 2009) in adulthood, as well as the fact that both the Dutch famine and the 1918 flu impacted maternal stress and nutritional status,[17] we examine BMI as another broad indicator of health for the second generation; this is tested in Panel C of table 5. A consistent pattern emerges across all panels. Like SES outcomes in table 4, gen. 1 female exposure to the flu is shown to have negative, statistically significant association with all health outcomes in the second generation.

From Panel A, parental—or gen. 1—flu exposure reduces self-reported health by roughly half a point ($p<0.05$) on a Likert scale from 1-5; 1 being very poor health and 5 being excellent

[16] Appendix Table 14 tests indicators for a number self-reported health conditions. As shown, a weak positive association is shown between gen. 1 female flu exposure and an indicator of cardiovascular disease during the 1992/93 wave of the WLS (Appendix Table 15A). This association, however, reduced in magnitude and statistical significance for later-life waves of the WLS (Appendix Table 15B and 15C).
[17] The Dutch famine influenced maternal nutritional status directly through caloric restriction and the 1918 flu through symptoms such as appetite loss, vomiting, and/or diarrhea.

health. As shown and like prior estimates, this effect is driven by gen. 1 female exposure. For adult height, gen. 1 female exposure is associated with a reduction of about 0.2 inches ($p<0.10$), which is larger than the direct effect documented by Mazumder et al. (2010). of 0.05 inches; however, Mazumder et al.'s (2010) estimated effect falls within the 95% confidence interval of our estimated coefficient. Panel C considers the effect on second generation BMI. From column (1), having either parent being born in 1918/1919 is associated with a statistically significant increase in the offspring's BMI of 0.41 points. As with the economic effects of table 4, this increase in BMI appears to be driven by mother's exposure. This is seen in columns (2)-(4), which estimate a statistically significant positive coefficient for mother's exposure but a coefficient that is statistically indistinguishable from zero for father's exposure.

A persistent effect of in utero exposure to the 1918 Flu is seen in tables 4 and 5. First generation exposure consistently has a statistically significant and economically meaningful effect on second generation health and economic outcomes. And this effect appears to be driven solely by mother's exposure; although, due to marriage market associations, we cannot determine definitively that mother's exposure produces this multigenerational effect through biological mechanisms.

Appendix Tables 10A-11G re-estimate Tables 4 and 5, splitting the sample by generation 2's sex. In short, the negative effects of first generation exposure appear to be more prominent in second generation males; such that, the harmful multi-generational effects are most prominent in second generation men whose mothers were exposed to the 1918 flu.[18] Potential reasons for

[18] When omitting those with no family income during the 1992 wave of the WLS, negative effects from gen. 1 female flu exposure are primarily seen in second generation males. This indicates, however, that there is a greater frequency of no income amongst second generation females from gen. 1 flu exposure. Additionally, when looking at net worth in the 2011 wave of the WLS, we see no difference by gen. 2's sex.

this sex disparity could be tied to general trends in the differences between sons and daughters in intergenerational mobility (Chadwick and Solon 2002). In other words, sons are more likely to inherit their families SES standing than daughters. This implies that the negative gen. 1 shock from in utero exposure is more likely to affect sons through the proposed SES channel than daughters, resulting in a larger estimated effect for gen. 2 males. Furthermore, there appears to be a growing gap between in the intergenerational elastiticy between sons and daughters during the time period in question (Olivetti and Paserman 2015). Another potential reason for the sex difference could be the larger standard deviation in gen. 2 male outcomes; this is shown in Appendix Table 9. Finally, the findings of Appendix Tables 10A-11G differ from Richter and Robling (2015), who show homogeneous sex effects from in utero flu exposure for education: gen. 1 female exposure led to reduction in gen. 2 female education, and gen. 1 male exposure is associated with gen. 2 male education. Given this disparity between the two studies, our under-representation for gen. 1 male exposure, and the mixed findings in Tables 10A-11G, we view our general finding of gen. 1 female exposure working through gen. 2 male outcomes as preliminary and suggestive. Indeed, as we test in Appendix Tables 12A-12G, the sex differences in gen. 2 are often not statistically significant.

Third Generation

As mentioned previously, the focus of the WLS data collection is high school graduates in 1957, but additional samples have been collected for a number of variables on the children of these graduates. Table 6 explores the effects of the WLS parents on the years of schooling for WLS children, estimating the effect of flu exposure across three generations.[19]

[19]Analysis of the WLS grad/sibling children (i.e. the third generation) restricts the sample to those children that are biological children and that are 35 years of age and older by the 2003/2004 wave of the WLS.

Consistent with the second-generation estimations, a persistent effect of the in utero flu exposure is observed, and this effect seems to be driven by grandmother exposure (gen. 1 female). The joint indicator for either parent is negative and close to statistical significance at the 10 percent level (p=0.106), suggesting that exposure in the first generation is associated with 0.12 years of schooling less in the third generation. The effect of grandfather exposure is insignificantly different from zero, while the coefficient of grandmother exposure is similar in magnitude to the joint estimate of column (1) while being statistically significant at the 10% level (p=0.066). Furthermore, the decline in schooling seen in the third generation is similar, but slightly smaller in magnitude compared to the second generation effects seen in Panel A of table 4.[20] This again indicates a persistent effect that may be attenuating over time. Importantly, while the estimate for grandfather exposure is insignificant, it is consistent with intergenerational inheritance (Kuzawa and Eisenberg 2014). To say the same for grandmother exposure would require effects to persist into the fourth generation (due to the differences in gametic development), and this data is not available in the WLS.

Conclusion

This paper presents novel evidence of multigenerational effects of in utero health insults. We use the sudden and unexpected influenza pandemic in 1918/1919 to trace out the effects of in utero exposure to infectious disease on own outcomes for the first generation, children's outcomes, and grandchildren's outcomes. We find that this exposure reduces educational attainment and related economic outcomes across three generations. For example, individuals in

[20] For gen. 1 female exposure in column (4), the p-value for a difference in coefficients between Panel A of Table 4 and Table 6 is 0.411, implying the effect in the third generation is statistically indistinguishable from that in the second generation.

our second generation, the WLS graduates and their siblings who have mothers who were exposed to the 1918 flu in utero, complete 1/5 fewer years of schooling (or 2.4 months less schooling). The size of our estimated effect aligns well with those of Richter and Robling (2015) who find that first generation in utero exposure to the 1918 flu results in 1/6 fewer years of schooling (or 2 months to 2.5 months less schooling) for the maternal line. We then extend results to the third generation for the first time in the literature and find that individuals in the third generation, who have grandmothers who were exposed to the pandemic in utero, complete 1/7 fewer years of schooling (or 1.7 months less schooling) than individuals without affected grandmothers.

There are at several potential mechanisms for the persistence of poor outcomes across three generations we find in our data. As previously outlined, the intergenerational persistence in poor outcomes due to early life health shocks could occur through socioeconomic channels, where a fetally insulted person with resulting low educational attainment raises a child in a low-resource environment that reduces opportunities for high educational attainment of the child. In order to see multigenerational effects, the reduced opportunities for high educational attainment of the child must then reduce opportunities for their own children. Alternatively, intergenerational transmission may occur through epigenetic inheritance through the germ line, a distinct possibility based on findings of the Överkalix studies, or phenotype-to-phenotype transmission and cumulative intergenerational phenotypic change.

Our analysis is unable to fully distinguish between these proposed intergenerational channels, the socioeconomic and biological, but it is important to note that the channels are by no means mutually exclusive. Further analysis (appendix tables 3-5) that is intended to partially account for the socioeconomic mechanism leads to mixed results in which the coefficient of first

generation flu exposure is attenuated to insignificance for some outcomes but not others. This provides evidence for a socioeconomic channel but does not eliminate the possibility of epigenetic mechanisms or other biological channel through which socioeconomic status "gets under the skin" to influence outcomes of subsequent generations (e.g. the socioeconomic circumstances of one generation may "get under the skin" of the next and subsequently be passed via biology and/or socioeconomics to other generations). Likewise, the estimates of grandfather effects on grandchildren are imprecise but consistent with transgenerational inheritance (Kuzawa and Eisenberg 2014). Additionally, while the WLS provides a unique framework to analyze multiple generations, the measurement of flu exposure is somewhat crude (from self-reported parents' year of birth) and is in the right-tail of the distribution of parent birth years, suggesting the possibility of confounding from resource-poor younger mothers.[21] That is, exposure to influenza in the first generation is mechanically tied to mother's ages of 20 and 21 because the WLS sample is drawn based on having a child who was born in 1939 (and thus graduating high school in 1957). While we cannot definitively separate these two effects, our ability to compare outcomes of slightly older mothers (ages 22 and 23, born in 1917 or 1916) provide evidence more consistent with in utero exposure to influenza than impacts of having a mother who is 20/21 years old.

From a policy perspective, our evidence may suggest a novel source of multigenerational persistence in poverty through biosocial factors and suggest a need to consider evidence of transgenerational social and/or biological mechanisms. We document the extent to which

[21] We specifically explore the confounding effects of younger mothers in appendix tables 7 and 8. These tables show that younger mothers are indeed initially disadvantaged (i.e. less years of schooling); however, this young-mother disadvantage does not persist in subsequent generations. Rather, only those mothers born in the 1918-1919 range have significant negative effects on later generations.

harmful early life environments cascade through generations, promoting a disadvantaged start for those whose grandparents exposed to a hazardous early life environment.

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Tables and Figures

Table 1. Summary Statistics

Variable	N	Mean	Std. Deviation
First Generartion			
Flu Indicators			
Ind. for First Gen. Flu Exposure (Either Sex)	8368	0.112	0.315
Ind. for First Gen. Flu Exposure (Male)	8456	0.030	0.170
Ind. for First Gen. Flu Exposure (Female)	8487	0.093	0.290
Controls			
First Generation Male's Year of Birth	8456	1907.435	7.021
First Generation Female's Year of Birth	8427	1911.189	6.164
Dependent Variables			
First Generation Male's Years of Schooling	8456	9.778	3.392
First Generation Female's Years of Schooing	8487	10.499	2.804
First Generation Male's Job Prestige	8385	330.662	214.361
First Generation Female's Job Prestige	6683	12.814	19.934
Family SES	8487	16.231	11.143
Second Generation			
Controls			
Female Indicator	12459	0.530	0.499
Birth Year	12459	1939.406	4.289
Birth Order	12459	2.529	1.828
Dependent Variables			
Years of Schooling	12459	13.678	2.361
Family Income, 1992 (in $1000s)	12112	68.027	177.105
Net Worth, 2004 (in $1000s)	10498	913.046	2558.927
Self-reported health, 1992	9888	4.149	0.668
Height (inches)	9783	67.406	3.851
BMI, 1992	9671	26.742	4.657
Third Generation			
Controls			
Birth Year	19913	1963.901	3.636
Female Indicator	19913	0.497	0.500
Birth Order	19913	2.196	1.263
Dependent Variable			
Years of Schooling	19913	14.294	2.309

Summary & Notes: This table provides summary statistics for all variables used. The samples above may not be representative for all estimations. For example, the inclusion of siblings in the second generation increase the sample of parental year of birth and the corresponding flu indicator. This increase in sample does not significantly change the sample statistics above. Similar sample changes occur for the second generation controls above. For the third generation, the sample is restricted to biological children over 35 years of age.

Table 2. First Generation: Economic Effects

	Male			Female		
Dependent Variable:	Years of Sch.	Std. Job Prestige	Family SES	Years of Sch.	Std. Job Prestige	Family SES
	(1)	(2)	(3)	(4)	(5)	(6)
Ind. for First Gen. Flu Exposure (Male)	-0.139	-0.116*	-1.290**			
	(0.200)	(0.060)	(0.599)			
Ind. for First Gen. Flu Exposure (Female)				-0.133	-0.066	-1.361***
				(0.095)	(0.043)	(0.376)
Birth Year Time Trends	Y	Y	Y	Y	Y	Y
Observations	8456	8385	8456	8487	6683	8487
R Sqr.	0.032	0.008	0.023	0.013	0.004	0.013

Summary & Notes: This table provides direct effects of in utero and early life exposure to the 1918 Flu Epidemic. Birth year time trends inlcude year of birth and its square and inclusion in the estimation is parent specific. All estimation is performed with OLS with robust standard errors reported in parenthesis. Statistical significance is denoted by *, **, and ***, representing significance at the 10, 5, and 1% levels, respectively.

Table 3. First Generation: Marriage Market Effects

	Male			Female		
Dependent Variable:	Flu Birth Year	Years of Sch.	Std. Job Prestige	Flu Birth Year	Years of Sch.	Std. Job Prestige
	(1)	(2)	(3)	(4)	(5)	(6)
Ind. for First Gen. Flu Exposure (Male)				0.140***	-0.259	-0.039
				(0.034)	(0.158)	(0.072)
Ind. for First Gen. Flu Exposure (Female)	0.056***	-0.340***	-0.118***			
	(0.014)	(0.117)	(0.036)			
Birth Year Time Trends	Y	Y	Y	Y	Y	Y
Observations	8348	8487	8412	8348	8456	6660
R Sqr.	0.090	0.011	0.008	0.120	0.021	0.001

Summary & Notes: This table provides evidence of marriage market selection in the first generation. Birth year time trends include year of birth and its square and inclusion in the estimation is parent specific. All estimation is performed with OLS with robust standard errors reported in parenthesis. Statistical significance is denoted by *, **, and ***, representing significance at the 10, 5, and 1% levels, respectively.

Table 4. Second Generation Economic Effects

Dependent Variable: By Panel	(1)	(2)	(3)	(4)
Panel A. Years of Schooling				
Ind. for First Gen. Flu Exposure (Either Sex)	-0.209***			
	(0.073)			
Ind. for First Gen. Flu Exposure (Male)		-0.084		-0.059
		(0.129)		(0.129)
Ind. for First Gen. Flu Exposure (Female)			-0.211***	-0.209***
			(0.075)	(0.075)
First Gen. Birth Year Time Trends	Y	Y	Y	Y
Second Gen. Controls	Y	Y	Y	Y
Observations	12459	12459	12459	12459
R Sqr.	0.085	0.084	0.085	0.085
Panel B. ln Family Income, 1992				
Ind. for First Gen. Flu Exposure (Either Sex)	-0.221**			
	(0.111)			
Ind. for First Gen. Flu Exposure (Male)		-0.098		-0.066
		(0.186)		(0.186)
Ind. for First Gen. Flu Exposure (Female)			-0.262**	-0.259**
			(0.116)	(0.116)
Base Controls	Y	Y	Y	Y
Observations	12112	12112	12112	12112
R Sqr.	0.021	0.021	0.021	0.021
Panel C. Std. Net Worth, 2004				
Ind. for First Gen. Flu Exposure (Either Sex)	-0.026			
	(0.022)			
Ind. for First Gen. Flu Exposure (Male)		0.033		0.038
		(0.047)		(0.047)
Ind. for First Gen. Flu Exposure (Female)			-0.038*	-0.040*
			(0.021)	(0.021)
First Gen. Birth Year Time Trends	Y	Y	Y	Y
Second Gen. Controls	Y	Y	Y	Y
Observations	10498	10498	10498	10498
R Sqr.	0.007	0.007	0.008	0.008

Summary & Notes: This table provides evidence of signficant harmful effects of flu exposure in the first generation on second generation economic outcomes. First generation time trends are given by year of birth and its square for both WLS parents (i.e., gen.1 males and females). Second generation controls inlcude an indicator for sex, second generation year of birth and its square, and birth order. All estimation is performed with OLS with family clustered standard errors reported in parenthesis. Statistical significance is denoted by *, **, and ***, representing significance at the 10, 5, and 1% levels, respectively.

Table 5. Second Generation Health Outcomes

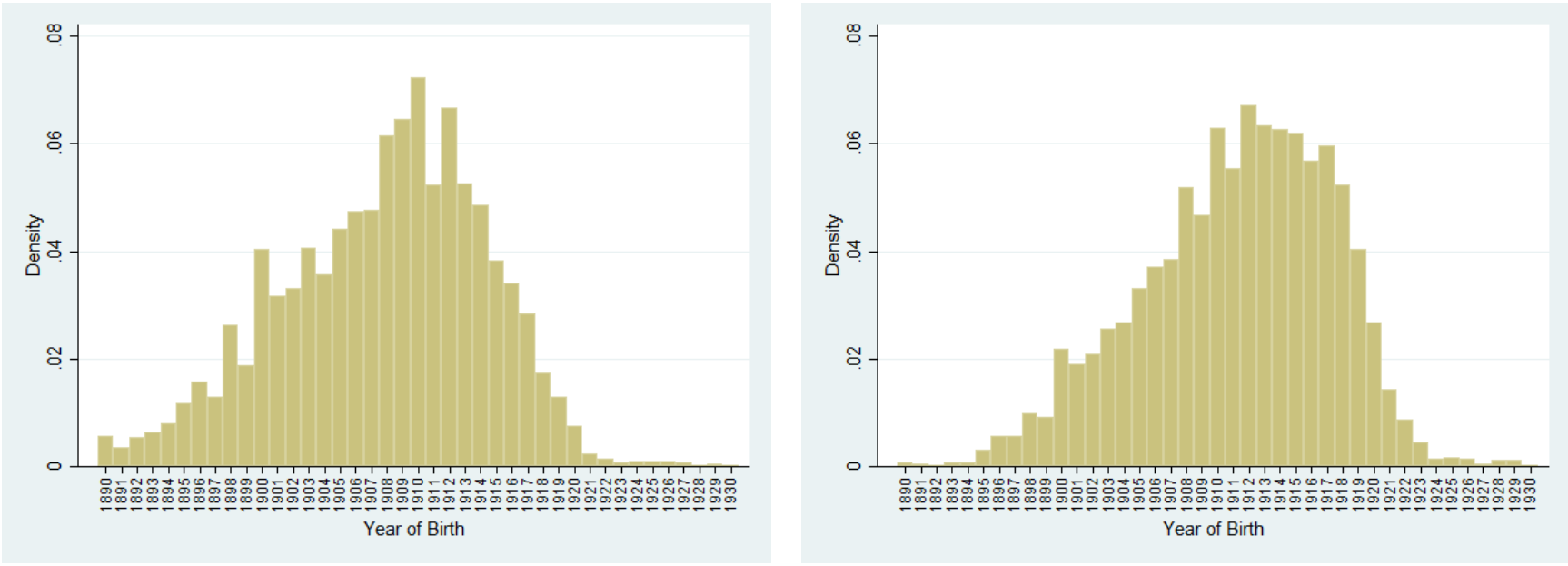
Dependent Variable: By Panel	(1)	(2)	(3)	(4)
Panel A. Self-reported health (Likert: 1=very poor; 5=excellent)				
Ind. for First Gen. Flu Exposure (Either Sex)	-0.054**			
	(0.027)			
Ind. for First Gen. Flu Exposure (Male)		-0.004		0.005
		(0.047)		(0.047)
Ind. for First Gen. Flu Exposure (Female)			-0.061**	-0.061**
			(0.028)	(0.028)
Base Controls	Y	Y	Y	Y
Observations	9888	9888	9888	9888
r2	0.009	0.009	0.009	0.009
Panel B. Height (inches)				
Ind. for First Gen. Flu Exposure (Either Sex)	-0.102			
	(0.101)			
Ind. for First Gen. Flu Exposure (Male)		0.102		0.127
		(0.168)		(0.168)
Ind. for First Gen. Flu Exposure (Female)			-0.169*	-0.175*
			(0.103)	(0.103)
Base Controls	Y	Y	Y	Y
Observations	9783	9783	9783	9783
r2	0.586	0.586	0.586	0.586
Panel C. BMI				
Ind. for First Gen. Flu Exposure (Either Sex)	0.412**			
	(0.191)			
Ind. for First Gen. Flu Exposure (Male)		0.082		0.023
		(0.344)		(0.346)
Ind. for First Gen. Flu Exposure (Female)			0.422**	0.421**
			(0.193)	(0.194)
Base Controls	Y	Y	Y	Y
Observations	9671	9671	9671	9671
r2	0.028	0.028	0.028	0.028

Summary & Notes: This table provides evidence of harmful effects of flu exposure in the first generation on second generation health outcomes. First generation time trends are given by year of birth and its square for both WLS parents (i.e., gen.1 males and females). Second generation controls inlcude an indicator for sex, second generation year of birth and its square, and birth order. All estimation is performed with OLS with family clustered standard errors reported in parenthesis. Statistical significance is denoted by *, **, and ***, representing significance at the 10, 5, and 1% levels, respectively.

Table 6. Generation Three Effects of Generation One Flu Exposure: Years of Schooling

Dependent Variable: Years of Schooling				
	(1)	(2)	(3)	(4)
Ind. for First Generation Flu Exposure (Either Sex)	-0.125			
	(0.077)			
Ind. for First Generation Flu Exposure (Male)		-0.007		0.010
		(0.136)		(0.136)
Ind. for First Generation Flu Exposure (Female)			-0.144*	-0.144*
			(0.078)	(0.078)
First Gen. Birth Year Time Trends	Y	Y	Y	Y
Second Gen. Controls	Y	Y	Y	Y
Third Gen. Controls	Y	Y	Y	Y
Observations	19913	19913	19913	19913
R Sqr.	0.079	0.078	0.079	0.079

Summary & Notes: This table shows that the generational effects of flu exposure extend into the third generation of the WLS. First generation flu exposure is shown to have a negative association with third generation years of schooling. First generation time trends are year of birth measures for each parent and their square. Second generation controls inlcude an indicator for sex, second generation year of birth and its square, and birth order. Third generation controls include a sex indicator, year of birth and its square, and birth order. Given the focus on years of schooling, the sample is restricted to those third generation individuals 35 years of age and older, or born during or before 1970 (data were collected in 2005). All estimation is performed with OLS with family (second generation) clustered standard errors reported in parenthesis. Statistical significance is denoted by *, **, and ***, representing significance at the 10, 5, and 1% levels, respectively.



(a) Father **(b)** Mother

Figure 1. First Generation Distribution of Year of Birth

Summary & Notes: This table figure plots the distribution for first generation year of birth. Flu exposure is classified as being born in either 1918 or 1919.