Does "*in utero*" Exposure to Illness Matter? The 1918 Influenza Epidemic in Taiwan as a Natural Experiment

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Abstract

This paper uses the sudden and unexpected 1918 influenza pandemic in Taiwan as a natural experiment to test whether conditions in early life (*in utero*) affect long run developmental outcomes. By combining several historical and current Taiwanese datasets, we find that exposure to influenza while *in utero* indeed has a profound adverse effect on outcomes in later life. The pandemic cohort are less educated, of shorter height as teenagers, and more likely to have various health issues including glaucoma, respiratory problems and diabetes in old age. The results are consistent with the fetal origin hypothesis.

Key Words: 1918 influenza, Fetal origins hypothesis, Height and weight, Education, Disease and mortality

JEL Code: I12, N35, I19

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The fetal origins hypothesis proposed by Barker in 1992 posits that "certain chronic conditions later in life can be traced to the course of fetal development." There is some evidence in medical literature suggesting that poor fetal conditions can increase the risk of schizophrenia (Brown et. al. 2004), cardiovascular disease and hypertension (Barker 1990, 1998; Langle- Evans 2001). However, it is not an easy task to establish a causal link between *in utero* environment and the long-term outcome. For example, there could be unobserved characteristics of mothers or families associated with the poor *in utero* environment, and these unobserved characteristics can have an impact on one's long-term outcomes as well. To tackle this identification issue, Almond (2006) investigates the long-term impact of the 1918 influenza pandemic in the US. The sudden, unexpected and brief nature of the 1918 influenza pandemic sets up a natural experiment to test the fetal origin hypothesis. He compares the affected cohort, i.e., those who experience the influenza *in utero* (born in 1919), to adjacent cohorts: those who were born just before or just after the 1918 influenza. He shows that cohorts *in utero* during the peak of the influenza pandemic exhibited lower educational attainment, lower income, lower socioeconomic status, a higher physical disability rate, and higher welfare income compared with those born just before or after.[1] He also uses maternal mortality rates as a proxy for the severity of pandemic and finds that those who were born in more highly affected areas are less educated.

Building on Almond's work, this paper employs a similar empirical strategy but with a Taiwanese dataset. It was estimated that nearly 770,000 people (1/5 of the total population) contracted the influenza strain, and influenza had a mortality toll of about 25,000 people in 1918 in Taiwan (Ding, 2008). Taiwan was a colony of Japan between 1889 and 1945, and the abundance of detailed demographic data compiled by the Japanese colonial government and current datasets provide a rare opportunity for the researcher to link long-term individual level datasets with the condition in their birth places.

Comparing to Almond's (2006) work, we are making a few contributions to the literature in the

[1] Using the Survey of Income and Program Participation (SIPP), Almond and Mazumder (2005) also found that the cohort exposed to influenza *in utero* had poor general health as well as trouble hearing, speaking, lifting, and walking, and higher rates of diabetes and stroke.

following way:

1. The main challenge to Almond's work has been recently raised by Brown and Thomas (2010), regarding the US involvement in World War I (July 1914-Nov 1918). Using the 1920 and 1930 US censuses, Brown and Thomas (2010) find that parents of the 1919 birth cohort are less educated, of lower socioeconomic status and older than the parents of surrounding cohorts. In Almond's work, since parents' characteristics were not controlled for, the time series estimates of coefficient on 1919 could overstate the true effect of the pandemic. In contrast, as a Japanese colony, Taiwan has no involvement in WWI.[2] Given the abundance of the dataset, we can further check whether parental characteristics of pandemic birth cohorts are any different from other cohorts, and we do not find any statistical difference in father's education and occupation between the pandemic birth cohorts and their surrounding cohorts.

2. Taiwan has better data on pandemic severity than the US. According to Almond (2006), the US did not have reliable data on regional differences in pandemic morbidity. In Taiwan, annual influenza related deaths were reported at the regional level. Monthly mortality data is also available at the national level. It allows us to identify the exact duration of pandemics, as well as employ regional and time-variation of the severity of influenza to have more specific cohort analysis.

3. Better identification of treated cohorts. Almond (2006) uses the US census, which did not report birth year, and he has to use one's reported age to impute one's birth year. Age-heaping is a known problem in the US census, i.e. those born in 1919 could report age 40 rather than age 41 in the 1960 census, leaving birth year incorrectly identified (Lleras-Muney 2005). This issue of age heaping could possibly bias the results downward. In the Taiwanese dataset, birth year is reported.

4. To the best of our knowledge, among the papers examining fetal origins hypothesis, this is the first paper to control for region-specific time trends. In our analysis, we show that it is important to control for region-specific time trends, as the main coefficients of interest reduce by half compared with the coefficients when one only controls for region dummies.

[2] In his book on military mobilization in Taiwan during the colonial period, Chou (1995), an expert on Taiwanese colonial history writes that the Japanese colonial government only began to recruit Taiwanese to serve in the Japanese military in 1937.

Apart from the advantages of using the Taiwanese dataset, there are a few noteworthy differences between Taiwan and the US. Taiwan has higher infant mortality rates than the US prior to the pandemic (16% versus 9.4%), and Taiwan also has higher infant mortality rates than the US during the pandemic (18.5% versus 10.1%). The environment in Taiwan is much less salubrious than the US, with the presence of other infectious diseases such as plague, cholera and malaria. Given the high mortality rates during the pandemic,[3] the surviving pandemic cohorts in Taiwan could be healthier than non-pandemic cohorts if the selection effect dominates over the scarring effect (Bozzoli, Deaton and Quintana-Domeque, 2009).

Unlike the US, Taiwan had two waves of life-threatening influenza outbreak between 1918 and 1920 (see Figure 1a). The first wave is from the end of October to December 1918, while the second wave is from December to February 1920.[4] We discuss our empirical strategy in detail in Section 3. One might worry that since the influenza pandemic occurred in other parts of the world during the spring of 1918, could the outbreak in Taiwan have been anticipated? In this regard, we provide several pieces of evidence. First, the articles and newspapers we surveyed suggest that the severity and extent of the spread of influenza in October/November 1918 was a shock to both the colonial Japanese government and the media. In fact, Ding (2008) specifically suggests the Japanese colonial government was slow to react to the influenza and contain the flu outbreak in the crucial period because the government did not anticipate this to be a severe pandemic. Second, it is possible that some residents, such as Japanese residents or civil servants, may be better informed about the threat of influenza than the rest of the population. If better-informed parents systematically avoided pregnancy during this period, we could have an overestimate of the true impact of the pandemic. We use the birth frequencies data, which is stratified by fathers' field of employment and residency[5] and collected by Japanese Colonial government, to compare

[3] According to Tsai (2005), the mortality rate conditional on influenza infection is 3.3% in Taiwan in 1919 and it is higher than 2.5% in the US and 1.21% in Japan.

[4] There is only one scholar, Ding (2008), who suggests that influenza struck Taiwan three times: June 1918 to September 1918, late October 1918 to December 1918, and December 1919 to February 1920. However, the only news we can find related to the first wave was on July 7, 1918 by *Taiwan Daily News*. It reports that there are increasing numbers of outpatients for cold and fever.

[5] Residency is recorded as either mainlander (Japanese) or local (Taiwanese) and field of employment includes Fields of employment include agriculture, transportation, fishery, mining, industry, commercial, government, others,

whether there is any difference in birth rates and birth frequencies by father's residency and field of employment for the pandemic cohorts comparing to surrounding cohorts,[6] and we do not find any differences. If one still worries that the second wave of influenza could be expected, we further exclude 1920 birth cohort from our analysis in the robustness check and the results remain robust.

Another concern is that the spread of the pandemic could be correlated with other factors, e.g. poor sanitation and poor economic development in an area, which could be predictive of later life health and human capital outcomes. The spread of influenza in Taiwan was believed to start in a northern port city and spread along the railroad in less than two weeks. Thus, regions with high maternal mortality are in fact those regions with higher income, higher spending on education/sanitation infrastructure both in 1918 as well as in later years.[7] This positive relationship could bias our estimates downward. In the robustness check section, we control for health and education spending[8] at the regional level and find consistent results.

We find that exposure to influenza while *in utero* indeed has a profound adverse effect later in life. The impact on education is similar in magnitude compared to Almond (2006). In Taiwan, males who were born in 1919 receive 0.06 years (1.2%) less education than the surrounding cohorts; whereas Almond find that male who were born in 1919 in the US receives 1.4% less education than the surrounding cohorts. We provide evidence of the pandemic's effect on height during teenage years. Most importantly, our evidence is consistent with the medical literature's finding that *in utero* influenza can increase the risk of diabetes, circulatory disease, respiratory diseases, and glaucoma (Konje et al. 1996, Langley-Evans 2001), similar to what Almond and Mazumder (2005) and Mazumder et al (2010) find. Given the scarcity of long-run data linkage between *in utero* individuals and their subsequent developmental outcomes many years later, our results shed new light on the validity of the fetal origin hypothesis.

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unemployed and housework.

[6] Results presented in the Web Appendix.

[7] This phenomenon is also known as urban penalty. See Haines (2001) for a discussion of the existence of urban penalty in the US. For brevity, we report the correlation matrix between health/education expenditure and pandemic severity in the Web Appendix posted on the authors' homepage.

[8] The education expenditure we control for is lagged by 7 years since one enters primary school at age 7.

The paper proceeds as follows: Section II is the literature review. Section III describes the impact of the 1918 influenza on Taiwan and the data sets used in the paper. Section IV provides the empirical results, and Section V concludes.

II Literature Review

Research has shown that early life shocks could have long lasting effect on various developmental outcomes into adulthood (Case et al. 2002, 2005; Almond and Currie 2011a).[9] For a detailed introduction to this literature, please see Almond and Currie (2011a, 2011b).

Epidemiologists used to believe that the placenta is a perfect filter that can stop all the harmful substances outside and just let in good ones, so smoking, drinking, and gaining very little weight for pregnant women are fine (Almond and Currie 2011b). Barker (1992) first formalizes this conjecture by proposing the fetal origins hypothesis, which argues that inadequate *in utero* nutrition could "program" a fetus to prioritize brain growth at the expense of other organs and tissues. As a consequence, certain chronic health conditions such as diabetes, heart problems at old ages can be traced back to the fetal environment. Ozanne and Hales (2004) support this hypothesis by providing an experimental result on mice.[10] Barker et al. (2002) also find that this "thrifty phenotype", i.e. slow fetal growth followed by fast catch up later, predicts large differences in the cumulative cases of coronary heart disease, type 2 diabetes, and hypertension. There is also epidemic evidence showing that poor early fetal conditions increase the risk of schizophrenia (Brown et al. 2004), kidney disease and high blood pressure (Langley-Evans 2001).

1918 Influenza as a Natural Experiment to Test the Fetal Origins Hypothesis

Almond (2006) and Almond and Mazumder (2005) were one of the first to test the fetal origins

[9] These researchers find profound short-run impacts of low birth weight (LBW) such as higher hospital costs and higher infant mortality (Almond et al. 2005, Black et al. 2007, Oreopoulos et al. 2008), and the long-run effects of LBW include lower test scores, below average IQ and education (Conley and Bennett, 2000; Hack et al., 2002; Boardman et al., 2002, Black et al. 2007, Johnson and Schoeni 2007, Oreopoulos et al. 2008, Royer 2009, Lin and Liu 2009). Other effects include lower wages and even shorter height (Behrman and Rosenzweig 2004, Black et al. 2007, Johnson and Schoeni 2007). Twins data or instrumental variables method are used to solve the potential endogeneity problem caused by omitted bias.

[10] They found that the lifespan of experimental mice is considerably shortened if the postnatal period of growth is accelerated to make up for reduced growth *in utero*. In addition, these mice are susceptible to the adverse effects on longevity of an obesity-inducing diet after weaning.

hypothesis using a natural experiment. The 1918 influenza pandemic was a widespread negative shock that spanned 4 months in the US. Almond (2006) and Almond and Mazumder (2005) compare the outcomes of the cohorts *in utero* during the peak of the influenza pandemic (born in 1919) to the surrounding cohorts. They find that 1919 birth cohort suffered from lower educational attainment, income, and socioeconomic status, as well as higher physical disability rates and higher welfare income as adults, relative to the cohorts born just earlier and later. Almond finds that cohorts born in states with more severe exposure to the influenza pandemic experienced significantly worse outcomes than those born in states with less severe exposure.

Using National Health Interview Survey (NHIS) in the US, Garthwaite (2009) finds that 1918 flu pandemic cohorts, defined by those born in the 4^{th} quarter of 1918, and the 1^{st} and 2^{nd} quarters of 1919, have a higher probability of developing coronary heart disease, diabetes, kidney disorders, or reporting poor health. Nelson (2010) finds similar adverse effects in education and wage for those who were born in the 1919 exposure cohort in Brazil. Neelsen and Stratmann (2010) find the same long-term effect in Switzerland. Finally, using another influenza pandemic, i.e., the 1957 Asian flu in Great Britian, Kelly (2010) finds the epidemic density has a negative effect on test scores, birth weight and height for those who were *in utero* during the influenza.

Overall, the literature uses large and sharp pandemics (such as the 1918 and 1957 influenza) as a natural experiment to test the fetal origins hypothesis. The identification strategies are either to compare the outcome difference between exposure and its surrounding cohorts (Almond 2006, Garthwaite 2009, Nelson 2010), or to find negative associations between cross sectional variation of influenza severity (Kelly 2011), maternal mortality (Almond 2006) on later outcome.

III The 1918 Influenza Epidemic in Taiwan and the Datasets

Spread of Influenza

The 1918 influenza, as known as Spanish Flu, first entered Taiwan through a port city in northern Taiwan in late October 1918, then spread along the railway from north to south in the heavily populated

western plain within two week. The sparsely populated eastern Taiwan was affected much less by the pandemic since regional transportation at that time was still poorly developed (Ding, 2008). The most severely affected regions are areas with better economic development and areas with higher public expenditure. The more developed places in 1918 are also places with better development in economic and health infrastructure even today. Therefore, it is possible that our estimates of impact of influenza could be underestimated due to the inverse relationship between government investment and pandemic severity.

Taiwan was a Japanese colony from 1895 to 1945. Japanese colonial government that ruled Taiwan during that time collected vital statistics for the whole Taiwanese population including the cause of deaths, and published aggregated-monthly and regional-annual records in *Dynamic Census of the Taiwanese Population*. We present the aggregate level of influenza-related death tolls from 1916 to 1926 by quarter in Figure 1a.[11,12] While the fluctuation in the number of influenza related deaths seems to be seasonal with more deaths in the winter, it is clear that the fourth quarter of 1918 and the first quarter of 1920 are the worst periods of the influenza pandemic. In Figure 1b, we further present monthly influenza death tolls and one can see that the two spikes are driven entirely by the increase in November/December 1918 and January/February 1920.

[Figure 1a & Figure 1b inserts here]

As Almond (2006) has argued, maternal mortality rate is the best proxy available for measuring the severity of the effect of influenza on a fetus. First, the age distribution of influenza victims varies across

[11] Since it is highly likely that influenza could be misdiagnosed as other respiratory diseases in 1918, following Johnson and Mueller's methodology (2002), we also code other causes of death such as tuberculosis, acute bronchitis, chronic bronchitis, pneumonia, pertussis and diphtheria along with influenza as influenza-related death. The trend for Influenza-related death moves closely with the death by influenza.

[12] Almond examines cohorts born between 1912 and 1922. Our regression analysis include cohort from 1917 to 1926 rather than 1912 since there is a drastic increase in government expenditure around 1917. Scholars who specialize in colonial Taiwanese history generally divide the 45-year Japanese colonization into three periods: 1895-1915, 1916-1936 and 1937-1945. They all suggest that 1915 is an important year, because it marked the end of the 20-year period of armed resistance (Rubenstein, 1999, p. 212). We also noticed that there was a malaria outbreak in 1916 (see Figure 2a for a spike in maternal mortality rates in 1916), and therefore we exclude the 1916 cohorts in the analysis.

regions and time.[13] In order to assess fetal origins effects, only measures of pandemic intensity among those of childbearing age are relevant. We do not have further breakdown of the pandemic intensity by age group, thus maternal mortality could capture this aspect of pandemic intensity. Second, Noymer and Garenne (2000) note that males were more likely to die in the influenza pandemic, hence data for the mortality of young women is needed so the estimation will not be confounded by the excess male death. Third, puerperal death would not have been misdiagnosed, as it did not resemble other causes of death as did other major pathological illnesses during the pandemic. Finally, maternal mortality has previously been used as a proxy for the *in utero* health environment. For example, Barker and Osmond (1987) found a strong relationship between local maternal mortality rate and the stroke mortality of offspring.

We first examine whether maternal mortality rates and flu pandemic exhibit similar patterns. Figure 2a shows an overall declining trend of maternal mortality rates with a few spikes during 1916-1926.[14] It shows that during the final quarter of 1918, the probability of women dying from childbirth rose to 1% (November 1.4% and December 0.95%) in 1918, compared to fluctuations between 0.4% and 0.8% during other periods.[15][16] During this period, the occurrence of other infectious disease

[13] For example, when the influenza first hit Taipei (first major city with reported influenza incidence), public gatherings such as religious and school activities still took place as usual, and it was noted in the news that the influenza was spread among students in several elementary schools in Taipei. By December 1918, many of public gatherings were cancelled. It is evident that infectious rates could have specific age/gender profile depending on the location and timing of the spread.

[14] Monthly maternal mortality rate is not available at the regional level. There were 12 administrative regions between 1916 and 1919, 7 administrative regions between 1920 and 1925 and 8 regions in 1926.

[15] There are several possible explanations as to why MMR in first quarter of 1920 was lower than 1919 despite the high influenza related death tolls. First, pregnant women may have become cautious since they would have had experience with the previous wave. Second, Noymer and Garenne (2000) noted that males were substantially more likely to die in the influenza pandemic because of higher tuberculosis morbidity. It could be that most of the deaths in 1920 were male. Third, it was suggested by Taiwan Medical Association Magazine that the second wave of influenza has much higher death rates (13%) compared to the first wave (3%), thus although it has marginally higher death tolls, maybe simply fewer people ever contracted influenza in the second. the incidence of influenza in 1920 may not be as widespread as in 1919.

[16] The Japanese colonial government invested much effort in collecting detail records of causes of death, including various infectious diseases. Therefore, we can identify the timing of each outbreak of disease and the severely infected areas. The spike in 1916 was a malaria outbreak which causes 11000 deaths (0.3% of total population) and the spike in summer of 1919 was a cholera outbreak, 3826 contracted cholera and 2693 died (0.07% of total population) (Sun, 2010). 1916 birth cohort would be excluded from the analysis. On the other hand, another cholera outbreak took place in between two flu pandemics. The cholera outbreak has a few characteristics: first, the mortality rate for 1919 cholera outbreak was high at 71%, meaning that there were only less than 1200 survivors in the total population of 3 million. While cholera outbreak increases maternal mortality rates, it could be difficult to detect the long term effect in the overall population, which would possibly bias our estimates downward. Second,

outbreaks such as malaria and cholera could have also caused an increase in the maternal mortality rate (MMR). Thus in Figure 2b, we present residual MMR estimated from a regression controlling for the malaria death tolls, cholera death tolls and a quadratic time trend. One thing worth noting is that despite that there were more influenza related deaths in the second wave than first wave, we find that MMR has a bigger spike in the first wave than the second wave. This pattern is interesting and we also observe similar results in Table 2 where we find that the impact of first wave of influenza is greater than the impact of second wave. It is apparent that the residual MMRs fluctuate with influenza death tolls.[17]

. [Figure 2a & Figure 2b inserts here]

Educational Attainment in the 1980 Census

The 1980 Census of Taiwan records the educational attainment, gender, birth place/ancestral home (*huji*), birth date and birth year for all the Taiwanese alive in 1980. To determine the effect of the 1918 influenza outbreak, we can link the severity of the epidemic, indicated by maternal mortality and infant mortality rates at the time and region of an individual's birth, to one's subsequent educational attainment. We drop all the samples who were not born in Taiwan since there was a massive migration in 1949 from China.[18] Table 1 shows the summary statistics of educational attainment taken from the 1980 census for those who were born between 1917 and 1926. On average, this cohort has 3.3 years of schooling, and males (4.8 years) completed significantly more years of schooling than females (1.9 years) did. Furthermore, the average maternal mortality rate and infant mortality rate are 0.47% and 16.55%, respectively. These region-year level variables, which can approximate the average economic social, educational, and sanitary conditions, will be used in the regression later.

[Table 1 inserts here]

Anthropometric Outcome during Adolescence

The Japanese colonial government published *"Health Statistics for School Students"*, which records

more than 85% of 1919 cholera cases were in Taipei, Tainan and Taidong regions, so in robustness check section, we could exclude these regions, and we still find similar results.

[17] If we regress MMR residual on influenza death tolls, the influenza tolls is significant at 1% level.

[18] The 1918 influenza also affects China, so we cannot use the migrants from China as a comparison group.

height and weight of all Taiwanese students by region and age (hence birth year) in 1927. It allows us to investigate whether the pandemic cohort were more likely to have lower height and weight during their adolescent years.

Health Outcome in 1989

We use data from the *1989 Survey of Health and Living Status of the Elderly in Taiwan*, which records the health conditions for those who were above 65. It contains information on gender, father's education and occupation, birth year, birth county (which we can then merge to the birth region), and most important of all, whether sample individuals have health problems from a list of disease categories common in the elderly.

IV Empirical Results

4.1 Educational Outcome-Time Series

In this section, we attempt to replicate Almond's (2006) approach with Taiwanese data. We present some time-series evidence examining the departure of pandemic birth cohorts from the surrounding cohorts in education outcomes.

In accordance with the fetal origins hypothesis, we would expect that those who were *in utero* during the peak of the pandemic (born between December 1918 and August 1919 and between December 1919 and September 1920) would be affected to a greater extent than those who were born just before the pandemic and 9 months after the pandemic.

Next we modify Almond's (2006) specification to accommodate the two pandemic waves and to incorporate regional characteristics, region-specific time trends as below:

$$y_{ijt} = \alpha + \beta_1 * I(YOB = 1919)_{ijt} + \beta_2 * I(YOB = 1919)_{ijt} +$$

$$\sum_j Region\ Fixed\ Effects_j + \sum_j Region_j * Time_t + \varepsilon_{ij}$$—Eq (1)

where y_{ijt} is the education outcome for individual i in region j born in year t. I(.) denotes an indicator function whether one's year of birth is in the given year. The data includes those who were born between 1917 and 1926. Since a concern may be that error terms from estimating Equation 1 could be serially

correlated and correlated within a region, we need to cluster the standard errors. Cameron, Gelbach and Miller (2008) illustrates that, with a small number of clusters (12 regions in our case), cluster standard errors can be biased. We use the procedure described by Cameron et al (2008), to conduct a “wild bootstrap", p-values from wild bootstrap are reported in the Table 2 in bracket.

The key here is examining whether the pandemic cohorts demonstrate any departure from a quadratic trend. Estimates of β_1 and β_2 are reported in Table 2 Panel B and Panel C for men and women. Our estimate suggests a 1.35% drop (-0.0663/4.8=0.0135) in years of education for the 1919 male birth cohort while Almond (2006) finds a 1.4% drop in years of education. Another interesting finding is that coefficients on 1920 are smaller than coefficients on 1919, despite the fact that influenza death toll is higher in 1920. There could be several reasons for this finding. First, we observe a smaller spike of maternal mortality rate in 1920 compared to 1918 in Figure 2b. It is possible that pregnant women have become more cautious due to their previous experience with influenza. Second, given the high death tolls in 1920, following Bozzoli et al. (2009), given the high mortality rates, the selection effect (culling of the weakest) could possibly dominate the scarring effect of the flu. Lastly, Taiwan Medical Association Magazine suggested that 1920 flu pandemic has a higher mortality rate conditional on infection (13%) than the 1919 flu pandemic (3%). While the influenza-related death toll in 1920 is slightly higher than that of 1919, the incidence of influenza in 1920 may not be as widespread as in 1919. We include the regression result from estimating the deviation from 1918 to 1920 birth year-quarter cohort on years of education in the Appendix.

[Table 2 inserts here]

As suggested by Brown and Thomas (2010), the findings in cohort comparison in Almond’s (2006) paper could be driven by the underlying differences in parental characteristics. While there was no WWI involvement in Taiwan, it could still be possible that better-informed parents systematically avoided pregnancy during this period. We do not have parental information in the 1980 Census for the relevant birth cohort. Therefore, we examine this issue in several ways. First, we use the birth rate data, which is

stratified by residency[19] and collected by Japanese Colonial government, to compare whether there is any difference in birth rates between Japanese and Taiwanese for the pandemic cohorts comparing to surrounding cohorts,[20] and we do not find any differences. Second, we use the birth frequencies data, which is stratified by fathers' fields of employment, we compare whether civil servants are more likely to avoid pregnancy during flu pandemic compared to other fields, and we find no significant difference. Lastly, in the *1989 Survey of Health and Living Status of the Elderly in Taiwan,* each respondent reports their parental education level and occupation, so we can perform the same cohort analysis and again we find no significant difference. We provide all three sets of the regression results in the Web Appendix. If one still worries that the second wave of influenza could be expected, we further exclude 1920 birth cohort from our analysis in the robustness check and the results remain robust.

4.2 By Trimester of Exposure

Since we can identify the exact months when influenza death tolls peaked—in November/December 1918 and December/January 1919--and we know the birth month, we can further examine whether influenza exposure during different trimesters could have differential impacts. The specification is as follows:

$$y_{ijt} = \alpha + \beta_1 * I(first\ trimester\ in\ Pandemic) + \beta_2 * I(second\ trimester\ in\ Pandemic) +$$

$$\beta_3 * I(third\ trimester\ in\ Pandemic) + \alpha X_{ijt} + v_j + \varepsilon_{ij} \quad \text{----- Eq (2)}$$

I(.) is an indicator function whether individual i has experienced Pandemic in first, second or third trimester. We could code the dummy variable equals to one if two out of the three months in the trimester was during the peak of Pandemic (November-December 1918 or January-February 1920), the dummy would equal to zero if none of the three months in the trimester was during the peak.[21] We find that no

[19] Residency is recorded as either mainlander (Japanese) or local (Taiwanese) and field of employment includes Fields of employment include agriculture, transportation, fishery, mining, industry, commercial, government, others, unemployed and housework.

[20] Results presented in the Web Appendix.

[21] For those who experience only 1 out of 3 months of the trimester during the peak, we would code them as missing. The control group consists of those who never has in utero exposure to influenza.

matter which trimester one experience the peak of the flu pandemic *in utero*, this experience will reduce his/her education by about 0.068-0.85 years. There is no obvious pattern as to which trimester is most important in order for the *in utero* environment to have a long term impact.

[Table 3 Insert Here]

4.3 Regional Variation in Maternal Mortality Rate

Another way to gauge the effect of influenza on developmental outcome is to explore the regional-time variations in influenza severity.[22]

We estimate the following regression:

$$y_{ijt} = \alpha_{ijt} + \beta_1 * WeightedMMR_{ijt} + \beta_2 * Infant\ Mortality\ Rates_{jt}$$

$$+\beta_3 * Female_i + v_j + \varepsilon_{ij}\text{—Eq (3)}$$

where i is the individual, j is the birth region, and t is the birth year-month.[23] Infant mortality rate is used to capture the environment during the individual's infancy since this could also affect their later developmental outcomes. The main coefficient of interest is β_1 on MMR, which approximates the health environment faced by the fetus. The MMR measures are available either at annual regional level or monthly aggregate level. There are various ways to construct the appropriate MMR for individual i, which we would explore in the robustness check section. First, we estimate Equation 3 with regional-annual MMR and calculate the weighted MMR, where weight is determined based on months of *in utero* exposure in a given year. For example, if one is born in June 1919, then the weighted MMR would be the weighted average between two thirds of 1919 MMR and one third times 1918 MMR. The results are reported in Table 4 Row 1. We find that a 1 percentage point increase in MMR results in a reduction in the years of education by 1.768 years. However, one should be cautious in interpreting this

[22] This specification again is modeled after Almond's working paper version (2005). We also try to replicate Almond's (2006) in imputing maternal infection rates using 1917-, 1918- and 1919- maternal mortality rate. According to the formula Almond uses: maternal infection rate$_{1918}$ = (MMR$_{1918}$ - MMR$_{1917}$)/(maternal morbidity rate - MMR$_{1917}$). Even though the monthly mortality rate spiked in the winter of 1918, with a strong declining trend and a shorter duration of pandemic, several of the regional MMR$_{1918}$ are still less than MMR$_{1917}$, leaving this exercise less informative.

[23] In the Taiwan census, everyone reports the birth county. We then match it to the region level as defined in 1919.

coefficient. This is a period during which Taiwan had experienced an overall improvement in education attainment, so it is essential to control for a linear time trend. Moreover, this improvement in education can vary across regions, so a set of region specific time trends are included in the specifications. Next, we include regional time trends and the results are reported in Row 2. This would be our preferred specification throughout the remainder of this paper. Once we control for the time trend, we find that the coefficient reduce by almost half. Every 1 percentage point increase in MMR would reduce the years of education by 0.866 years (0.866/3.2 = 27%). Compared to Almond's (2005) finding of 0.947 year (0.947/10.7 = 8.8%), while similar in absolute scale, the relative impact is much greater in Taiwan than in the US. In Row 3, we use a lag regional-annual MMR from the previous year. In Row 4, we include sanitation expenditure, educational spending 7 years later (when the cohort reaches the age to enter elementary school) and agriculture GDP per capita at the region-year level. This could capture some additional regional variations that cannot be eliminated by the region-specific time trend and region fixed effect. In Row 5, we use the mean of aggregate-monthly MMRs of the 9 months prior to the birth. e.g. if one is born in September 1918, the average monthly MMR is the mean from monthly MMR from January-September 1918. Throughout various specifications, the estimates appear to be quite stable except the residual MMR specification.[24]

[Table 4 Inserts Here]

[Table 5 inserts here]

Table 5 reports coefficient estimates for several different set ups for sensitivity analyses, using Equation 3 with regional-specific time trends. The first two rows report results for males and females separately. Overall, the negative effect of maternal mortality sustains in both genders. In addition, the coefficients for the male subsample are slightly larger than their female counterparts. The gender difference may be due to the fact that, compared to males, very few females (less than 3%) have achieved an educational level higher than junior high school. Row 3 reports the results excluding Hualien and

[24] We only report linear probability result for simplicity. In the Web Appendix, we report the regression results with probit estimates.

Taitung, the two eastern regions with sparse populations that were less affected by the 1918 influenza due to difficulties in transportation. In footnote 12, we mention that Taipei, Tainan and Taidong saw exposures to cholera in 1919 which resulted in mortality rates but very few survivors. We exclude these regions from our analysis in Row 4. Some may worry that the second wave of influenza may be expected, thus those parents who give births during that time could be the below average parents. In Row 5, we present the same specification as Table 4 Row 2, but excluding 1920 birth cohort.[25] Overall, all five measures from this table obtain negative and significant result, which are similar to our baseline results in Table 4 Row 2.

How large is the influenzas effect on educational attainment? The average maternal mortality rate during the first half of 1918 was 0.55%, while the rate for the second half of 1918 was 0.81%. Taking the coefficients estimated in Table 4 at face value, and comparing them with the average educational attainment shown in Table 1, we can calculate the effect of influenza. Given that the maternal mortality rate increased 0.27% during the 1918 pandemic, and the coefficient of maternal mortality on education years is -0.866, it reduces years of education by about 7.0% (0.27*0.866/3.32). Compared with Almond (2006), which obtained only a 2-3% decrease in years of education, the 1918 influenza asserts a larger impact on Taiwan.[26] Besides the fact that MMR is a proxy for in utero environment, maternal deaths can also have a direct effect on the surviving child. We do a back-of-envelope calculation estimating the direct effect and it suggests that the direct effect would only lower the years of education by 0.01 year (0.26%).[27]

4.4 Health Attainment- Height in Childhood and Adolescents

Height for age is widely considered as a long-run measure of nutritional status (Thomas, 1994). If fetal origins hypothesis holds, we shall see those who were born in 1919 are shorter compared to the

[25] In the Web Appendix, we provide regression results for replicate Table 4 excluding 1920 birth cohort.
[26] Our point estimate is very similar to Almond's (2006). The only difference is that at the baseline education level are much lower in Taiwan than the US. Thus, it appears to have bigger impact.
[27] Assuming that all children who lost their mother (during births) receive zero year of education, we should find the direct impact of increasing MMR by 0.26 percentage point would lower years of education by only 0.01 year (0.26%) (0.26*0+99.74*3.32=3.31)

surrounding cohorts. Our first evidence comes from Taipei County's Statistical Book, 1929-1938, which documents average height for male students from age 13 to 17 in each year. This allows us to back out the average height for those who were born from 1916 to 1921, then trace their average height from age 13 to 17.[28] Figure 3 reports the results. Take age 16 for example, we find that the average height for male students born between 1917 and 1921 were 1.58, 1.59, 1.56, 1.59, and 1.59 meters respectively. Almost all of them were the same height at age 16, except the 1919 cohort were 3.5 centimeters (1.38 inch) shorter than their surrounding cohorts. Another interesting pattern is that the 1919 birth cohort experience adolescent growth spurt later than the rest of surrounding birth cohorts at age 17, so by age 17, the difference in height between 1919 and 1920 birth cohort is 1 cm (0.39 inch). This pattern can be due to two reasons: first, as we have shown earlier, the average education during this period is only 3.3 years, therefore, it is a selected population who were still in school at age 17. It can be that the selection effect dominates the scarring effect by age 17 for the 1919 birth cohort. Another reason is related to what Case and Paxson (2008) find--- children growing up with better nutritional/disease environment experience an earlier adolescent growth spurt.[29]

[Figure 3 insert here]

Next, we use data from *Health Statistics for School Students, 1927*, which is a cross sectional survey that records the height of all Taiwanese students by region and age (hence birth year) in 1927. We regress individual height using the same model specification as in Equations 3 with region-specific time trend. Table 6 shows that one percentage point increase in maternal mortality decreases the average height of Taiwanese students by 0.5cm, In addition, the effect is slightly larger for females than it is for males.[30]

[Table 6 insert here]

4.5 Health Attainment-Health in Old Age

The Barker hypothesis also suggests that fetal influenza exposure can affect health conditions later in life.

[28] For example, a person who is age 14 in the 1931 Statistical book should be born in 1917.

[29] Given that only a small fraction of the population are still enrolled in school at age 13 during this period, the above figure does not reflect the average treatment effect of the pandemic. Yet it is still interesting that, conditional on attending schools, the 1919 cohort are always shorter at all ages.

[30] Mazumder et al (2010) find that men born in 1919 are about 0.05 inch (0.127cm).

We investigate this claim by using the 1989 Survey of Health and Living Status of the Elderly in Taiwan and present the results in Table 7. The model specification is the same as for Equation 3 with region-specific time trends. Although we know from this dataset whether an individual is born in the first or the second half of the year, we do not have the individual birth month, and so we cannot calculate the weighted MMR per the earlier specification.[31] For those who were born between January and June of any given year, we assign to them the maternal mortality rates from the previous year for their region. For those who were born between July to December, we assign them the maternal mortality rates from that year.

As the results in Table 7 indicate, we find that an increase in maternal mortality rate increases an individual's probability of having respiratory disease (including asthma, bronchitis and other breathing related disease), diabetes, and glaucoma. These findings are consistent with the findings from Mazumder et al (2010).

[Table 7 insert here]

V Discussion and Conclusion

Using maternal mortality rate as a proxy measuring the degree of exposure and combining several historical and current datasets in Taiwan, we find that exposure to influenza while *in utero* indeed has a profound adverse effect on later outcomes. The pandemic cohort had less education, shorter height as teenagers and a higher chance of having various health issues including glaucoma, respiratory problems and diabetes. This result is consistent with the fetal origins hypothesis.

[31] Birth month was collected in the survey, but it was not available in the publically accessible dataset. We are working on obtaining birth month information from the statistical agency that collected the data. The finest level of birth date information that is currently available to us is birth year and whether one is born in the first half or the second half of the year.

Comparing the magnitude of the 1918 influenza impact between different countries may shed light on the "thrifty phenotype" claim of fetal origins hypothesis, which argues that cohorts who encounter *in utero* shock might do worse when they encounter postnatal abundance. In other words, given the same pandemic severity, we would observe a larger negative effect for those pandemic cohorts who reside in places with a better environment. Compared to the US, Taiwan had a much lower education level, a higher infant mortality rate, and shorter life expectancy at birth during this time period, so we might expect the pandemic to have a smaller impact in Taiwan. However, comparing our findings with Almond's (2006) finding, we find that pandemic has a bigger negative impact in Taiwan than in the US. We do want to point out that it is difficult to test the thrifty phenotype claim in this particular setting since it is possible that the severity of pandemic could differ, or simply, the production function of education may not be comparable across these two countries. This could be an interesting direction for future research related to the fetal origins hypothesis.

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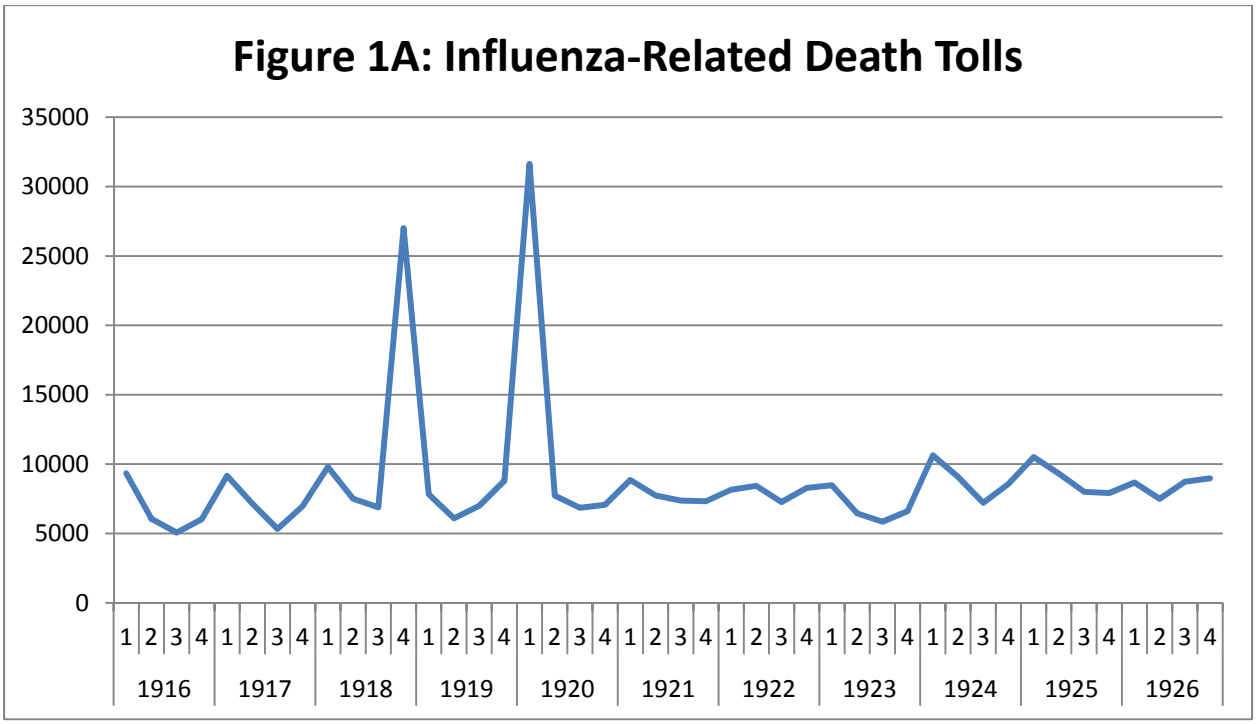
Figure 1A: Influenza-Related Death Tolls
35000
30000
25000
20000
15000
10000
5000
0
1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4
1916
1917
1918
1919
1920
1921
1922
1923
1924
1925
1926

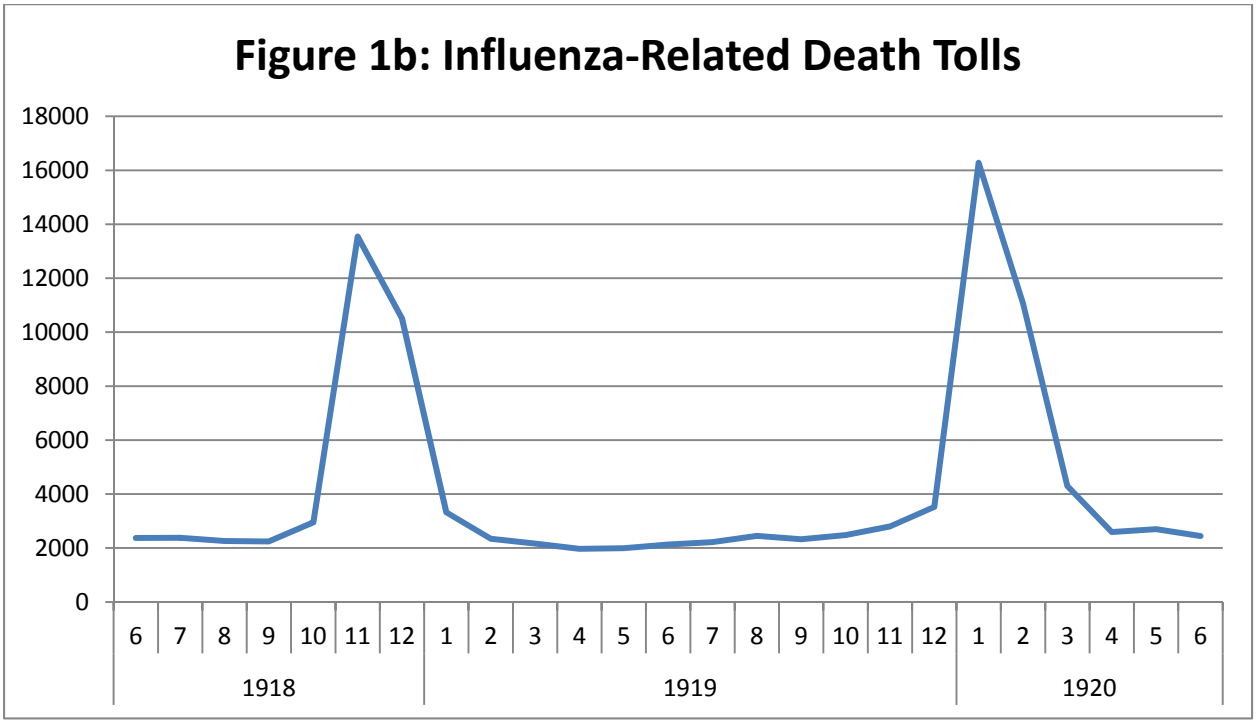
Figure 1b: Influenza-Related Death Tolls
18000
16000
14000
12000
10000
8000
6000
4000
2000
0
6 7 8 9 10 11 12 1 2 3 4 5 6 7 8 9 10 11 12 1 2 3 4 5 6
1918
1919
1920

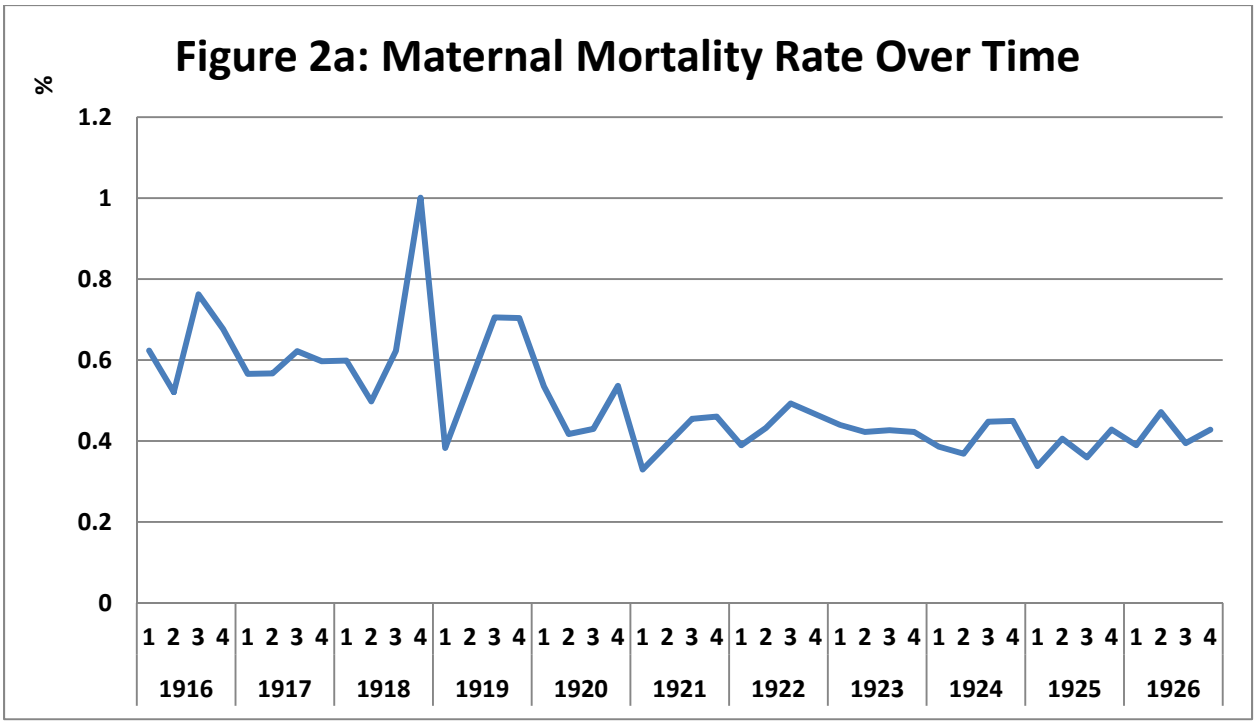
Figure 2a: Maternal Mortality Rate Over Time
%
1.2
1
0.8
0.6
0.4
0.2
0
1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4
1916 1917 1918 1919 1920 1921 1922 1923 1924 1925 1926

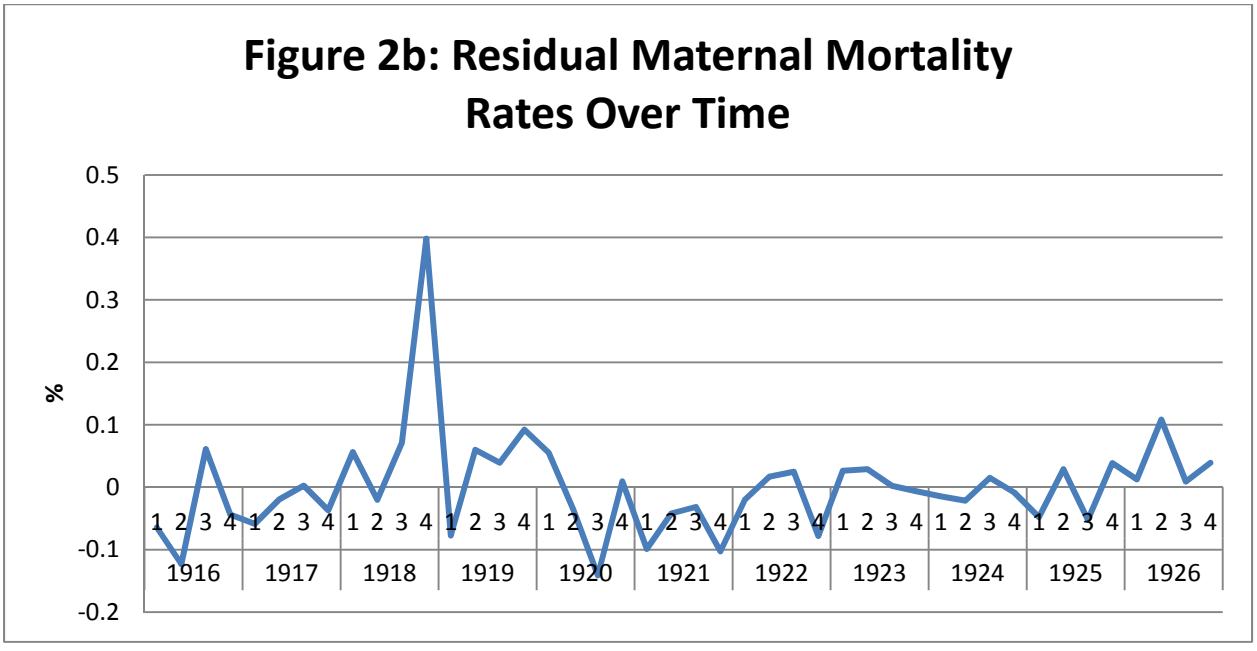
Figure 2b: Residual Maternal Mortality Rates Over Time
%
0.5
0.4
0.3
0.2
0.1
0
-0.1
-0.2
1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4 1 2 3 4
1916 1917 1918 1919 1920 1921 1922 1923 1924 1925 1926

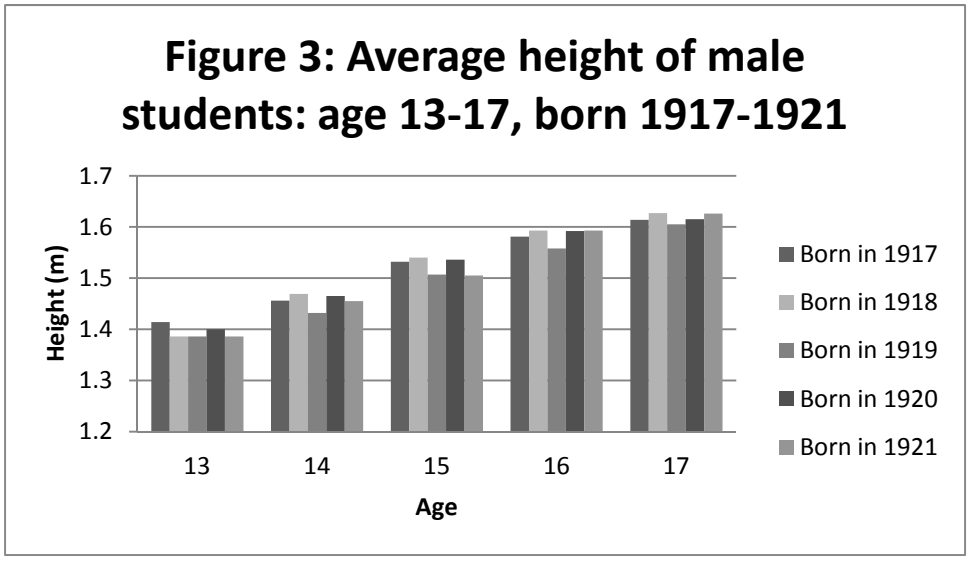
Figure 3: Average height of male students: age 13-17, born 1917-1921
1.7
1.6
1.5
1.4
1.3
1.2
Height (m)
13
14
15
16
17
Age
Born in 1917
Born in 1918
Born in 1919
Born in 1920
Born in 1921

Table 1A: Individual Level Summary Statistics from
1980 Census for 1917-1926 Birth Cohorts

	all	male	female
Years of Education	3.32	4.807	1.932
	(3.864)	(3.964)	(3.196)
Literacy Rate (%)	52.22	72.63	33.17
	(0.500)	(0.446)	(0.471)
Elementary School Completion (%)	46.98	66.48	28.77
	(0.470)	(0.472)	(0.453)
Middle School Complete (%)	9.92	15.89	4.34
	(0.299)	(0.366)	(0.204)
High School Completion (%)	5.23	8.46	2.21
	(0.223)	(0.278)	(0.147)
College Completion (%)	1.53	2.79	0.34
	(0.123)	(0.165)	(0.583)
Female (%)	51.72		
	(0.500)		
Number of Observations	809721	390910	418811

Table 1B: Regional-Year Level Summary Statistics

	Mean	Min	Max
Maternal mortality rate (%)	0.47	0.11	1.00
Infant mortality rate (%)	16.55	11.4	24.74
7-year Lagged Education Expenditure (per capita)[1]	1.188	0.131	3.584
Government Sanitary Expenditure (per capita)	0.11	0.006	0.635
Agriculture GDP per capita	41.51	18.58	71.64

1. Usually one enters school when he/she turns 7. Thus the reported education expenditure is lagged 7 years. It is imputed using total government education expenditure for a given year/region divided by total population in a given year/region.
Note: Standard errors are reported in parentheses.

Table 2: Departure in Education Outcomes from 1917-1926 Birth Cohorts

VARIABLES	(1) Years of Schooling	(2) Elementary School	(3) Middle School	(4) High School
	Panel A: All (Male and Female)			
1919 Birth Cohort	-0.0618***	-0.0047**	-0.0052***	-0.0051***
p-value from cluster	[0.011]	[0.066]	[0.001]	[0.000]
p-value from wild bootstrap	[0.000]	[0.004]	[0.000]	[0.000]
1920 Birth Cohort	-0.0412	-0.004	-0.004***	-0.0028***
p-value from cluster	[0.098]	[0.260]	[0.005]	[0.000]
p-value from wild bootstrap	[0.136]	[0.248]	[0.000]	[0.004]
Observation	870468	870468	870468	870468
	Panel B: Male Only			
1919 Birth Cohort	-0.0663***	-0.0025	-0.0073***	-0.0084***
p-value from cluster	[0.005]	[0.229]	[0.007]	[0.000]
p-value from wild bootstrap	[0.004]	[0.108]	[0.000]	[0.000]
1920 Birth Cohort	-0.0371	-0.0029	-0.0037*	-0.0044**
p-value from cluster	[0.138]	[0.297]	[0.146]	[0.001]
p-value from wild bootstrap	[0.128]	[0.328]	[0.096]	[0.016]
Observation	419554	419554	419554	419554
	Panel C: Female Only			
1919 Birth Cohort	-0.0562**	-0.0065**	-0.0031**	-0.0022***
p-value from cluster	[0.042]	[0.120]	[0.000]	[0.006]
p-value from wild bootstrap	[0.032]	[0.044]	[0.016]	[0.000]
1920 Birth Cohort	-0.0424	-0.0046	-0.0042***	-0.0013*
p-value from cluster	[0.272]	[0.419]	[0.002]	[0.153]
p-value from wild bootstrap	[0.320]	[0.456]	[0.000]	[0.076]
Observation	450914	450914	450914	450914

Note: Wild bootstrap p-values with 500 repetitions and p-values from clustering at regional level are in brackets. All models allows for correlation among observations within the same region. Infant mortality rate, gender, region dummies and region-specific time trends are included.

Table 3: Differential Impact of In Utero Exposure to Influenza Outbreaks by Trimesters 1917-1922

	Dependent variable			
	Years of Schooling (1)	Elementary School (2)	Middle School (3)	High School (4)
First Trimester	-0.0684*	-0.0057	-0.0064***	-0.0039**
p-value from clustering	[0.023]	[0.124]	[0.002]	[0.015]
p-value from wild bootstrap	[0.052]	[0.112]	[0.000]	[0.040]
Second Trimester	-0.068**	-0.0062*	-0.0054**	-0.0024*
p-value from clustering	[0.025]	[0.077]	[0.015]	[0.007]
p-value from wild bootstrap	[0.048]	[0.096]	[0.020]	[0.072]
Third Trimester	-0.085***	-0.0104***	-0.0033*	-0.0029
p-value from clustering	[0.004]	[0.004]	[0.108]	[0.056]
p-value from wild bootstrap	[0.000]	[0.000]	[0.092]	[0.136]

Note: N=399,818. Each column represent results from a regression. Wild bootstrap p-values with 500 repetitions and p-values from clustering at regional level are in brackets. All models allows for correlation among observations within the same region. We code each of the trimester as 1 if one experience 2 out of 3 months of trimester during the peak of pandemic (defined as Nov/Dec 1918 or Jan/Feb 1920), 0 if zero month out of 3 months of trimester during the peak of pandemic Those who experience only 1 month of trimester during the peak of pandemic would be coded as missing. Infant mortality rate, gender, region dummies and region-specific time trends are also included in all regressions.

Table 4: Effect of Maternal Mortality Rate on Educational Attainment 1917-1922

	Dependent variable			
	Years of Schooling	Elementary School	Middle School	High School
(1): Estimation of Equation 3				
weighted maternal mortality rates	-1.768***	-0.230***	-0.072***	-0.044***
p-value from cluster	[0.000]	[0.000]	[0.000]	[0.000]
p-value from wild bootstrap	[0.000]	[0.000]	[0.004]	[0.000]
(2): (1) + Region-specific time trends				
weighted maternal mortality rates	-0.866**	-0.098*	-0.043**	-0.035*
p-value from cluster	[0.129]	[0.153]	[0.110]	[0.081]
p-value from wild bootstrap	[0.028]	[0.100]	[0.036]	[0.076]
(3): (2) + MMR(t-1)				
MMR (t-1)	-0.694*	-0.084	-0.033**	-0.024**
p-value from cluster	[0.139]	[0.179]	[0.061]	[0.041]
p-value from wild bootstrap	[0.056]	[0.180]	[0.016]	[0.016]
(4): (2) + Government Sanitation Expenditure, Education Expenditure and regional GDP per capita				
weighted maternal mortality rates	-0.824**	-0.097	-0.038**	-0.030
p-value from cluster	[0.143]	[0.155]	[0.146]	[0.132]
p-value from wild bootstrap	[0.048]	[0.136]	[0.040]	[0.116]
(5): (2) + 9-Month Average MMR				
9-month moving average MMR	-1.224*	-0.141	-0.063**	-0.045**
p-value from cluster	[0.160]	[0.194]	[0.108]	[0.083]
p-value from wild bootstrap	[0.072]	[0.348]	[0.028]	[0.040]

Note: N=445,987. Each coefficient represent a result from a regression. Wild bootstrap p-values with 500 repetitions and p-values from clustering at regional level are in brackets. All models allow for correlation among observations within the same region. Infant mortality rate, gender and region dummies are also included in all regressions.

Table 5: Robustness Check of Maternal Mortality Rate on Educational Attainment 1917-1922

	Dependent Variables			
	Years of Schooling	Elementary School	Middle School	High School
	(1)	(2)	(3)	(4)
(1): only male				
weighted maternal mortality rates	-1.067*	-0.100*	-0.069*	-0.060**
p-value from cluster	[0.097]	[0.120]	[0.104]	[0.074]
p-value from wild bootstrap	[0.052]	[0.100]	[0.100]	[0.044]
(2): only female				
weighted maternal mortality rates	-0.672*	-0.096	-0.019	-0.012
p-value from cluster	[0.192]	[0.205]	[0.181]	[0.181]
p-value from wild bootstrap	[0.092]	[0.112]	[0.228]	[0.240]
(3): Excluding Eastern Regions				
weighted maternal mortality rates	-0.882**	-0.095	-0.049**	-0.039**
p-value from cluster	[0.140]	[0.181]	[0.087]	[0.074]
p-value from wild bootstrap	[0.048]	[0.148]	[0.012]	[0.048]
(4): Excluding Taipei, Tainan and Taidong in 1919/1920				
weighted maternal mortality rates	-0.885*	-0.102	-0.045*	-0.032
p-value from cluster	[0.153]	[0.180]	[0.100]	[0.122]
p-value from wild bootstrap	[0.092]	[0.148]	[0.064]	[0.180]
(5): Excluding 1920 birth cohort				
weighted maternal mortality rates	-0.886	-0.098	-0.047**	-0.038*
p-value from cluster	[0.157]	[0.194]	[0.107]	[0.085]
p-value from wild bootstrap	[0.108]	[0.204]	[0.048]	[0.084]

Note: Each coefficient represent a result from a regression. Wild bootstrap p-values with 500 repetitions and p-values from clustering at regional level are in brackets. All models allow for correlation among observations within the same region. Infant mortality rate, gender, region dummies and region-specific time trends are also included in all regressions.

Table 6: Effect of Maternal Mortality Rate on Height(meter) in the 1928 Survey

	Female	Male
maternal mortality rates $_{(t-1)}$	0.047**	0.070*
p-value from clustering	[0.011]	[0.021]
p-value from wild bootstrap	[0.032]	[0.096]
Observation	31,039	83,211

Note: ***, **, and * are significant at 1%, 5%, 10% respectively. Wild bootstrap p-values with 500 repetitions and p-values from clustering at regional level are in brackets. All models allow for correlation among observations within the same region. Infant mortality rate, region dummies and region-specific time trends are also included in all regressions.

Table 7: Effect of 1918 Maternal Mortality Rate on Disease Prevalence in the 1989 Elderly Survey

	Kidney Disease	Vertigo (Dizzy)	Circulatory disease	Respiratory disease	Glaucoma	Diabetes
	(1)	(2)	(3)	(4)	(5)	(6)
Mean	*0.047*	*0.271*	*0.163*	*0.146*	*0.0135*	*0.0512*
			Panel A: Linear Probability Model			
weighted maternal mortality rate	0.2907	0.25682	0.25434	0.2253***	0.0464**	0.11925*
p-value from clustering	[0.016]	[0.170]	[0.021]	[0.039]	[0.007]	[0.021]
p-value from wild bootstrap	[0.136]	[0.116]	[0.120]	[0.004]	[0.040]	[0.096]
			Panel B: Probit Model			
weighted maternal mortality rate	2.897***	0.807	0.965***	0.971**	1.295**	1.040***
p-value from clustering	[0.000]	[0.138]	[0.010]	[0.013]	[0.045]	[0.007]

Note: Wild bootstrap p-values with 500 repetitions and p-values from clustering at regional level are in brackets. All models allow for correlation among observations within the same region. Infant mortality rate, gender, region dummies and region-specific time trends are also included in all regressions.

Appendix Table: Departure in Education Outcomes from 1917-1926 Birth Cohorts

	Dependent variable			
	Yrs of education	Elementary School	Middle School	High School
q_19184	-0.0039	0.0014	-0.0021	-0.0020
	(0.0373)	(0.0059)	(0.0022)	(0.0018)
q_19191	-0.0630**	-0.0041	-0.0064**	-0.0052***
	(0.0261)	(0.0028)	(0.0022)	(0.0016)
q_19192	-0.1091***	-0.0093***	-0.0077***	-0.0066***
	(0.0159)	(0.0025)	(0.0014)	(0.0010)
q_19193	-0.0370	-0.0037	-0.0025	-0.0031*
	(0.0367)	(0.0043)	(0.0025)	(0.0015)
q_19194	-0.0567	-0.0038	-0.0051**	-0.0063***
	(0.0336)	(0.0039)	(0.0023)	(0.0020)
q_19201	-0.0620**	-0.0067*	-0.0043**	-0.0028**
	(0.0230)	(0.0032)	(0.0015)	(0.0010)
q_19202	-0.0756	-0.0081	-0.0072***	-0.0021
	(0.0547)	(0.0082)	(0.0022)	(0.0013)
q_19203	-0.0274	-0.0017	-0.0045	-0.0025
	(0.0447)	(0.0054)	(0.0030)	(0.0015)
Constant	-329.1337***	-45.0720***	-12.1505***	-5.8007***
	(4.6927)	(0.7693)	(0.1145)	(0.1090)
Observations	870,468	870,468	870,468	870,468
R-squared	0.1698	0.1752	0.0475	0.0274

Note: Each of the coefficient represent deviation in years of education for a given birth year-quarter cohort relative to those who were born between 1917 to 1926. All models allows for correlation among observations within the same region. Infant mortality rate, female dummy, birth quarter dummies, region dummies and region-specific time trends are included. The highlighted areas correspond to 9-months delay of the two spikes occurred in Figure 2b in 1918 4th quarters and 1919 4th quarter.