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 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 had less education, shorter height and lower weight as teenagers, a higher percentage of various health issues including hearing, kidney, circulatory, thyroid, and respiratory problems in old age, and they died earlier. The result is consistent with the fetal origin hypothesis.

Key Words: 1918 influenza, Fetal origin hypothesis, Disease and mortality

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I Introduction

It is now well documented that the effect of conditions in one's early life can last much longer than previously imagined. By investigating data at the individual level, Case et al. (2002, 2005) find that children who experience poor health have significantly lower educational attainment, worse health, and inferior social status as adults. Low birth weight (LBW) is frequently used as an indicator for the health of an infant during the neonatal period. LBW has been associated with adverse short-run impacts such as higher hospital costs and higher infant mortality (Almond et al 2005, Black et al 2007, Oreopoulos et al 2008) as well as long-run effects such as lower test scores, IQ, education, wages, or even shorter height (Conley and Bennett 2000; Hack et al., 2002; Boardman et al., 2002, Black et al 2007, Lin, Liu, and Chou 2007, Oreopoulos et al 2008, Lin and Liu 2009).[1]

Since low birth weight can also be seen as a proxy for fetal nutrition intake, it is natural that researchers have pushed the causal effects of early life conditions even further from the neonatal to the fetal period. This is known as the "fetal origin hypothesis" (Barker 1992), which posits that "certain chronic conditions later in life can be traced to the course of fetal development." There is epidemic evidence showing that fetal conditions increase the risk of schizophrenia (Brown et. al. 2004), cardiovascular disease and hypertension (Barker 1990, 1998, Langley Evans 2001). Couzin (2002) puts it nicely: "A fertilized egg has hoop after hoop to jump during its upcoming nine months."

However, it is not easy to track individuals from birth to adulthood due to data limitations, to say nothing of tracking someone from womb to adulthood. Hence some researches use macro level economic conditions at birth as proxy variables for health. A recession during early

[1] Some of these researches use OLS methods, while the others relied on twins data or instrumental variables method to solve the endogeneity problem caused by omitted variables.

childhood (Van Den Berg, Lindeboom, and Portrait 2006) or childhood exposure to diseases such as malaria (Bleakley 2006), hookworm (Bleakley 2007), blight (Banerjee, Duflo, Vinay and Watts 2010), or famine (Meng and Qian 2006) can all have a significant adverse effect on future education and mortality.

In particular, Almond (2006) investigates the long term impact of the 1918 Influenza Pandemic using 1960-1980 Decennial U.S. Census data. 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 a few months before or after. Using the Survey of Income and Program Participation (SIPP) Almond and Mazumder (2005) also find that the cohort exposed to influenza *in utero* had poor general health as well as trouble hearing, speaking, lifting, walking and higher rates of diabetes and stroke.

This research intends to contribute to the literature by investigating the long run education, health and mortality effect of the 1918 influenza outbreak on the Taiwanese population. The sudden, unexpected and brief nature of the 1918 influenza pandemic sets up a great natural experiment to test the fetal origin hypothesis. The abundance of detailed demographic data compiled by the Japanese colonial government from 1905 to 1945 and current datasets provide a rare opportunity for the researcher to link long-term individual level data. We evaluate Barker's fetal origin hypothesis by investigating whether exposure to influenza while *in utero* indeed has a profound adverse effect in education, height and weight as teenagers, and whether it causes various health problems, in particular early death. In addition to education and health results similar to Almond (2006), we also provide evidence of the pandemic's effect on height and weight during teenage years, its mortality effect, and most importantly, evidence that is consistent with the medical literature finding that *in utero* influenza can increase the risk of

kidney (Konje et al 1996, Langley-Evans 2001) and hearing (Heider 1934) problems. In addition, our result should be less jeopardized by the immigration and war effects inherent in US data. Given the scarcity of long-run data linkage between *in utero* individuals and their subsequent developmental outcomes many years later, our results should shed new light on the validity of the fetal origin hypothesis.

The paper proceeds as follows: Section II is the literature review. Section III describes the impact of 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

Effect of Economic Conditions during Early Life on Later Outcomes: Results from Individual and Aggregate Level Data

Research has shown that social, economic and health conditions during one's early life have not only contemporaneous effects, but have influence that lasts much longer into adulthood—even to the end of one's life. For example, Case et al (2005) find that children who have poor health, low birth weight and prenatal smoking mothers, have significantly lower educational attainment, poor health, and lower social status as adults. Case et al (2002) also find that the effects of poor health, especially chronic health conditions, are more profound for children from lower income households.

Low birth weight, which is often used as an indicator for health at neonatal stages, has recently received much attention. The short-run impacts of LBW include 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, and Oreopoulos et al 2008, Royer 2009, Lin and Liu 2009), lower wages, and even shorter height (Behrman and M. Rosenzweig 2004, Black et al 2007, Johnson and Schoeni 2007).[2]

All the research mentioned above uses individual level data to track individuals from childhood to adulthood. However, such datasets are not easy to find (Case et al 2005). Another stream of research overcomes this difficulty by matching one's educational, health, labor market experience, and even mortality outcomes later in life with the aggregate economic or social condition at the time of one's birth if birth dates and places are available. For example, Van Den Berg, Lindeboom, and Portrait (2006) merge a historical sample of the Netherlands (HSN), which covers approximately 14,000 individuals born from 1812 to 1912 and continues up to year 2000, with historical time series data on macroeconomic variables. The authors observe that "the most striking result is that the cyclical indicator in the birth year has a negative effect on mortality rate later in life." Their estimates suggest individuals born in a recession live a few years less than individuals born in a boom. They conclude that at the individual level, "economic conditions at birth have long lasting effects on mortality". Costa and Lahey (2005) find that the US death pattern experiences a seasonal difference due to the "poor nutrition take and maternal infection from respiratory disease during the winter months".

Childhood disease exposure is another possible cause of poor developmental outcomes in later life. For example, using as natural experiment the DDT-based anti-malaria campaigns in the southern U.S and Brazil in the 1920's and in Colombia and Mexico around 1955, Bleakley (2006) find that cohorts born after eradication campaigns had higher income and literacy as adults than the preceding generation. Moreover, the timing also supports the hypothesis that the observed changes coincide with childhood exposure to the campaigns rather than the pre-existing trend

[2] Because of the potential bias caused by omitted variables, such as unobserved mother's ability, early life experience, or genetic endowment, would cause both low birth weight and future developmental outcome, these researches relied on twins data or instrumental variables method to solve the endogeneity problem..

across cohorts. Bleakley (2007) further studies the de-worming campaign of 1900 and finds that those who were born in the areas that benefited from anti-hookworm campaigns saw large increases in income. Bleakley (2009) concludes that the impact of a tropical disease eradication campaign, "particularly via the effect of being infected in the early life", explains a non-trivial amount of income difference. Using interaction with hot and rainy weather as instrumental variables, Barreca (2007) finds exposure to malaria had a negative economic effect over the life cycle.

Blight matters, too. Between 1863 and 1890, phylloxera spread slowly from the southern coast of France to the rest of the country, attacking the roots of grape vines and destroying 40% of French vineyards. The shock caused major income losses among wine growing families, and the outbreaks affected different regions in different years. By exploiting the regional variation in the timing of the shock to identify its effects, Banerjee, Duflo, Vinay and Watts (2010) conclude that the shock decreased long run height by 0.6 to 0.9 centimeters.

Famine is another early life factor that has drawn recent attention. Van Den Berg, Lindeboom and Portrait et al. (2007) further explored HSN using food price and macroeconomic data from the 19th century to examine whether the 1846-1847 Dutch Potato Famine had a long-run longevity effect. Among the individuals in the sample, 398 out of 1740 people were exposed to the Potato Famine before age 3. The authors' results suggest that men exposed to severe famine for at least four months before birth and immediately afterward have a significantly lower residual life expectancy at age 50 than others, but the effect does not appear at earlier ages. Meng and Qian (2006) explore a similar issue using China's Great Famine in the 1960s as a natural experiment. They show that childhood exposure to famine had significant adverse effects on adult health and work capacity, such as height, weight, and labor supply. The magnitude of the effect is negatively correlated with age at the onset of the famine.

Finally, public health conditions such as air pollution, infant mortality and community situation are also important. Chay and Greenstone (2003) find that air pollution increases infant mortality. Ferrie, Rolf, and Troesken (2006) use Social Security record data in the United States to determine the effect of early life circumstances at the community and household level on mid-life and late-life outcomes. Chay, Guryan and Mazumder (2008) find that most of the racial convergence in the 1980's in US can be explained by the convergence in measures of black and white infant health for those cohorts. Almond, Edlund, and Palme (2009) also find that students born in a region of Sweden with higher fallout due to the Chernobyl incident performed worse in secondary school compared with other students, and in particular on math. These results also suggest that the achievement of a person is related to external conditions in the place where he was born as well as the place where he was raised.

The Womb May be More Important than the Home? The Fetal Origin Hypothesis

Although commonly used as an indicator for health in early life, birth weight is completely determined by conditions during the period in which a fetus is inside the womb. On the other hand, adverse or favorable macro conditions such as an economic boom, recession, blight, or disease do not exert their effect only at the exact date or month of one's birth, either. These conditions are more likely to be continuous, and it is likely that a boom or a recession that affects a baby has already been affecting the fetus. This leads to a reasonable deduction: conditions during pregnancy matter too.

Barker (1992) formalizes this conjecture by proposing the "fetal origin hypothesis", which argues that nutrition *in utero* and infancy affect health and mortality at old ages, and certain chronic health condition can be traced back to fetal environment. He finds that **low birth weight, as an indicator of retarded fetal growth**, is associated with high blood pressure and risk of

hypertension at age 50 (Barker 1990). He then further hypothesized that initial slow fetus and infant growth and later accelerated weight gain is the underlying mechanism that damages development later in life. Ozanne and Hales (2004) support this hypothesis by providing an experimental result on mice.[3] 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).

Barker's hypothesis indicates that, in the context of aggregate data, individuals who experienced adverse shocks while they were still in the womb would have worse developmental outcomes. Previous aggregate research such as Van Den Berg, Lindeboom and Portrait et al. (2006), Van Den Berg, Lindeboom and Portrait et al. (2007), and Banerjee, Duflo, Vinay and Watts (2010) indeed provides indirect evidence for this hypothesis since when a baby was born in a recession, famine or blight, it is highly likely that he was affected by these adverse condition as a fetus as well. However, if an individual were exposed to an adverse environment continuously from the time she was a fetus to the time she was an infant, it would be hard to identify whether the effect is from the fetal period or the infant period. This concern leads to a potential disadvantage if we want to test the fetus origin hypothesis vs. the infant health hypothesis, although one might argue that from the perspective of public health policy, it does not matter much since we could just concentrate our resources on both pregnant women and infants.

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

[3] They find that 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.

In order to test the fetal origin hypothesis and trace out its potential effect on later developmental outcomes, we need an adverse shock that has to two properties: first, it needs to be severe enough to generate meaningful variation among the population, and most importantly, it needs to strike the population for only a very short time so that there won't be enough time for a reaction from the population that may cause an endogeneity problem.[4] The 1918 influenza pandemic is a very good candidate. First, its infection rate was high, and the number of global mortality estimations range from 21.5 million in the 1920's, to 25-40 million in 1991, and to more than 50 million in 2002 recalculation(Johnson and Mueller 2002). Garrett (2008) reports that in the United States, 675, 000 people (0.8% of the 1910 population) died due to influenza. Importantly, the outbreak arrived unexpectedly in the fall of 1918 and had largely subsided by January 1919, "generating sharp prediction for long term effect (Almond 2006)." When the cohorts who were still in their mother's womb, i.e., those who were born in the first half of 1919, exhibit lower development outcomes relative to the cohorts born just earlier and later, outside of the fetal origin hypothesis it is hard to image any other scenario or alternative hypothesis which would generate the same effect.

In pioneer research, Almond (2006) investigates the long-term impact of the 1918 Influenza Pandemic using 1960-1980 Decennial U.S. Census data. He shows that cohorts *in utero* during the peak of the Influenza Pandemic suffered from lower educational attainment, lower income, lower socioeconomic status, higher physical disability rate, and higher welfare income as adults. In addition, using maternal mortality rate as a measure of severity, 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. For men born in the United

[4] For example, rich people might migrate to an area where the pandemic is less serious, hence we will overestimate the effect of the pandemic.

States around 1918, a 1% increase in maternal mortality rate (classified by the state of birth and the year preceding the birth year) would result in 0.756 to 0.818 year decrease in years of education and 10.1% to 10.9% decrease in high school graduation rate. Almond and Mazumder (2005) also investigate the Survey of Income and Program Participation (SIPP) and find that cohorts *in utero* during the pandemic relative to cohorts born a few months earlier or later exhibit poor health, trouble hearing, speaking, lifting, walking, diabetes, and stroke. This is evidence that *in utero* exposure to sickness reduced future health. Garthwaite(2009) finds that 1918 flu pandemic cohorts have a higher probability of developing coronary heart disease, diabetes, kidney disorders, or reporting poor health. Finally, Nelson (2008) finds similar adverse effects for the 1918 exposure cohort in Brazil.

Overall, the literature has shown that early life conditions indeed affect a great variety of later outcomes. Barker's hypothesis, which focuses on the environment faced by the fetus, provides an interesting perspective, but is also hard to be separated from the infant health effect. The 1918 influenza epidemic provides a clean natural experiment for testing the fetal origin hypothesis because of its sharp and serious impact. However, so far in the literature only Almond (2006) and Almond and Mazumder (2005) have directly used 1918 data. In the following section, we will use Taiwan's historical and census data to investigate the link between the influenza pandemic and developmental outcomes many years later.

III The 1918 Influenza Epidemic in Taiwan and the Data Sets

Influenza and the Related Death

The 1918 influenza, a.k.a. Spanish Flu, was a novel H1N1 influenza that belongs to the subgroup of strains that infect humans and swine, not the avian subgroup (Taubenberger et al 1997). It hit

Taiwan most seriously in the fall and early winter of 1918.[5] Influenza entered Taiwan from Keelung, the largest port in northern Taiwan, then spread along the railway from north to south in the heavily populated western plain (Taipei, Hsin-Chu, Taichung, Kaohsiung). Cities along railways had higher death rates. On the contrary, sparsely populated eastern Taiwan (Hualien and Taitung) was affected much less seriously by the pandemic because regional transportation at that time was still poorly developed.

The influenza-related data is from *Dynamic Census of the Taiwanese Population* (台灣人口動態統計) compiled by the Japanese Colonial Government that ruled Taiwan at that time. This series of monthly periodicals records detailed vital statistics for the whole Taiwanese population. Figure 1-1 depicts the influenza related death toll from January 1918 to Oct 1919. The figure shows that the number of influenza deaths increased from around 20 to 40 during the first nine months of 1918 to 54 in October, 3,325 in November, and 2,703 in December. The death tolls the decreased sharply to 134 and 42 in January and February 1919, then back to the normal level for that period. This generates an even sharper variation than Almond (2006)'s US data, in which the abnormal death tolls were spread from October 1918 to April or May 1919. If our purpose is to compare the influenza affected group and its surrounding cohorts, the shorter span of the epidemic, the better its character as a "shock" because people have less time to respond.

[Figure 1-1 inserts here]

Influenza can also cause complications for those who already have other (chronic) respiratory diseases. For example, Noymer and Garenne (2000) find that males were more likely to die in the influenza pandemic because of their higher tuberculosis morbidity. Furthermore, due to the state of medical science in 1918, it is highly likely that influenza was often misdiagnosed

[5] Between 1918 and 1920, influenza struck Taiwan three times: June 1918 to September 1918, late October 1918 to December 1918, and December 1919 to February 1920. We focus on the second wave since it is the most serious.

as another respiratory disease. For this reason, Johnson and Mueller (2002) argue that influenza, pneumonia, and all other excess death should be used to calculate total influenza-related death. Hence we also plot influenza-related death tolls, including tuberculosis, acute bronchitis, chronic bronchitis, pneumonia, and other respiratory diseases (such as pertussis and diphtheria) along with influenza death in figure 1-1 as well. The influenza-related death toll increases from an average of 3,000 for the first ten months of 1918 to 13,575 and 10,527 in November and December, and then drops back to its pre-epidemic average after January 1919. Pneumonia, which is the most common complication and most likely to be misdiagnosed from influenza, consists of the largest proportion and increase during the October-November epidemic. Its death toll increased from a pre-epidemic average of 1,200 to 6,155 and 4,749 in October and November of 1918 respectively.

Maternal Mortality Rate and Infant Mortality Rate

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 (see section IV-2 for detail discussion). Using the *Dynamic Census of the Taiwanese Population*, we calculate the monthly maternal mortality rate from January 1918 to December 1919 for the whole of Taiwan, and the yearly rates over six regions (Taipei, Hsinchu, Taichung, Kaoshuing, Hualien, and Taitung)[6]. Figure 1-2 shows that during November and December of 1918, the probability of women dying from birth rose to

[6] Taipei (tp) region: Northern Taiwan. Currently include Taipei City, Keelung City, Taipei County and Ilan County. Hsinchu (hc) region: North-western Taiwan. Currently include Hsinchu City, Taoyuan County and Hsinchu County. Taichung region (tc): Central-western Taiwan. Currently include Taichung City, Taichung County, Changhua County and Nantou County. Kaohsiung region(kh): Southern-western Taiwan. Currently include Kaohsiung City, Tainan City, Chia-i City, Yunlin County, Chia-i County, Tainan County, Kaohsiung County, Pingtung County and Penghu County. Hualien region(hl): Central-eastern Taiwan. Currently include Hualien County. Taitung region (tt): South-eastern Taiwan. Currently include Taitung County.

1.4% and 0.95% respectively, as compared to fluctuations between 0.4% and 0.7% during other periods. Figure 1-3 describes the maternal mortality rate by region and years. The rates again show increases in 1918 as compared to 1917 and 1919, especially for Taipei, Hsinchu, and Kaoshuing regionsWe do not see an increase in the eastern Taitung and Hwalian regions, however. This is consistent with our earlier hypothesis that the eastern regions were less affected due to transportation difficulty. We also calculate infant mortality rate for each of the regions and years. The average yearly maternal and infant mortality rates were 0.54% and 17% respectively, with the maximum of 0.87% and 25% occurring in 1918.

[Figure 1-2 inserts here]

[Figure 1-3 inserts here]

Educational Attainment in the 1980 Census

The 1980 Population and Housing Census of Taiwan records the educational attainment, sex, birth place, birth year and month 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 her subsequent educational attainment. We drop all the samples who were not born in Taiwan since there was a large migration in 1949 from China. Table 1 shows the by gender summary statistics of educational attainment taken from the 1980 census for those who were born between 1917 and 1921. On average, this cohort had 2.785 years of schooling, and males (4.12 years) completed significantly more years of schooling than females (1.5 years) did. The literacy rate, percentage completion of elementary school, junior high school, senior high school, and college were respectively 45.37%, 39.94%, 7.66%, 3.92%, and 1.28%. Again, male's completion and literacy rates were always on average several times higher than those of females.

[Table 1 inserts here]

Other Health Related Datasets: the 1989 Elderly Survey and the 1927 Health Statistics for School Students

The 1989 Survey of Health and Living Status of the Elderly in Taiwan records the heath conditions for those who were above 65 at that time. It contains detailed information on sex, birth year, birth county, and most important of all, whether sample individuals had health problem from a list of disease categories common in the elderly. We can hence investigate whether the pandemic exposure cohort were more likely to be sick. In addition, we also collect data from *Health Statistics for School Students*, *1927* (昭和二年學校生徒及兒童身體檢查統計書), which records height and weight of all Taiwanese students by region and age (hence birth year) in 1927. We can use this data to see whether the exposure cohort had lower height and weight.

IV Empirical Results

Education Attainment-Comparison with the Surrounding Cohort

Figure 1-1 shows that the 1918 influenza epidemic affected Taiwan only in November and December, and had generated five times influenza related death tolls than other months. The cohort born in early 1919 would be affected most seriously since they were all inside their mothers' wombs during the epidemic. Comparing the average education levels for the early 1919 cohort in relation to other surrounding cohorts gives us the effect by using time series variations. Figure 2-1 depicts percentage college completion by half-year birth cohorts covering the period between 1916 and 1924 with data taken from the 1980 Census. Percentage college completion drops from 1.19% for the second half of 1918 to 1.07% of the first half of 1919, then bounces back to 1.22% in the second half of 1919. During the same period, the average completion rates for senior high school and junior high school (figure 2-2 and 2-3) were 3.59%, 3.49%, 3.72%,

and 7%, 7%, 7.6% respectively. We also draw a fitted line to control for the long-term upward trend in education level. Note that the first half-year of 1919 exhibits the largest departure from the trend. To check this numerically, we regress the three educational completion variables on 20 birth quarters, quarter dummies, and a linear quarter trend (not reported here). The regression results indicate that the coefficients for the first and second birth quarters of 1919 are the most negative. We also draw the same pictures for males born in the western regions only, since western regions were the most seriously affected, and female education level is very low (less than 2 years) over this period.[7] Figures 2-4 to 2-6 show the same sharp decrease for the 1919 first half-year cohort. This time series variation provides a clean natural experiment for testing the fetal origin hypothesis because of its sharp effect and large impact. It is very hard to think of an alternative to the fetal origin hypothesis that would generate the same pattern.[8]

[Figure 2-1 to 2-6 insert here]

Educational Attainment-Maternal Mortality as a Proxy for in utero Pandemic Exposure

Another way to gauge the effect of influenza on developmental outcome is to explore the regional and time variations of influenza exposure. Almond (2006) proposed four reasons that maternal mortality rate is a good proxy for damage to fetal health caused by the 1918 Influenza Pandemic, and also why maternal mortality by year and state constitutes the best available measure of how geographic differences in the pandemic affected fetal health. First, the age distribution of influenza victims varies across regions and time. In order to assess fetal origin effects, it is measures of pandemic intensity among those of childbearing age that are relevant. Maternal mortality directly captures 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

[7] The effect of health human capital on education would not be important if only a very small percentage of the population can go to school.

[8] For example, a sudden large decrease in health or public spending, in early 1919. We do not find such a case.

the mortality of young women is needed so the estimation won't 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) find a strong relationship between local maternal mortality rate and the stroke mortality of offspring.

We hence estimate the following regression:

$$\text{Education Attainment}_{ijt} = \alpha + \beta_1 * \text{Maternal Mortality Rate}_{jt} + \beta_2 * \text{Infant Mortality Rate}_{jt} + \beta_3 * \text{Female}_i + \boldsymbol{\beta_4} * \text{Regional Dummies} + \boldsymbol{\beta_5} * \text{Linear Trend (overall or by regions)} \quad (1)$$

Where i is the individual, j is the birth region, and t is the time period. Here the key independent variable is maternal mortality rate, which approximates the health environment faced by fetus. The corresponding maternal mortality rate has a half-year lag. That is, for those who were born between January to June 1919, the corresponding mortality rate was from July to December 1918. This timing best captures the spirit of fetal origin hypothesis. We also tried using contemporary and a one year lag in maternal mortality rate (not reported here), and they yield similar (but smaller) results. Female is used to capture the gender difference in educational attainment. Infant mortality rate is used to capture the environmental condition during the individual's infant period, since this would also affect their later developmental outcomes. Six regional dummies were use to capture the unobserved characteristics for each region. Furthermore, linear trends were also used to capture the tendency of the dependent variable to rise over time. In addition to the overall trend, in which we consider only Taiwan as a whole, we also allow that each region to posses its own unique linear pattern by using regional specific linear trends. These dummies help to capture the effect of variables such as regional government spending on

education, health, sanitation, or medical resources. Finally, since individuals born in the same year and region would share the same mortality rate, their error terms would be correlated. We consider this property by clustering our observations by year and region, which increases the standard errors, but does not change the estimates of our coefficients.

Table 2 reports the effect of maternal mortality rate on education years while gradually adding in control variables. Column (1) shows that a one percentage point increase of maternal mortality rate would reduce education by 1.96 years when it is the only regressor. When female, infant mortality and regional dummies are gradually added, the coefficient is still around -1.7. But adding in an overall linear trend reduces the coefficient to -0.17. However, when regional specific linear trends instead of an overall trend are used, the coefficient is -0.34. Finally, although clustering by year and region increases the standard error of the estimates for maternal mortality rate from 0.08 to 0.11 in column (6), the coefficient is still significant at the 5% level.

[Table 2 inserts here]

Table 3 reports the same step by step regressions, but with five other educational attainment measures: literacy, elementary school completion, junior high school completion, senior high school completion, and college completion. Similar to table 1, after regional specific dummies are added, the coefficient on maternal mortality rates fall considerably. However, they are all significant for all five educational categories across all five model specifications. When the full model specification is used and the data is clustered by year and region, a one percentage point increase in the maternal mortality rate would decrease the probability of an individual being literate, completing elementary school, junior high school, senior high school, and college by respectively 2.9%, 3.6%, 2.1%, 1.6% and 0.6% for OLS, and 3.3%, 4%, 1.8%, 1.3% and 0.4%

for probit estimates. As we can see, probit and OLS results are similar.[9] In addition, female reduces the average educational years by 2.6 years, indicating that discrimination against girls was rampant in Taiwan during the early 20th century. Finally, a one percentage point increase in the infant mortality rate also reduces educational attainment by 0.85 years, indicating that the environment immediately after birth matters too.

[Table 3 inserts here]

Table 4-1 and 4-2 report coefficient estimates for several different set ups for sensitivity analyses, using full model specifications. We report both results with and without clustering. The first row investigates the effect of maternal mortality on all six educational categories using only those born during 1918 to 1920. This is the sample period chosen by Almond (2006). The coefficient estimates for education years, literacy, elementary school, junior higher school, senior high school, and college are -0.32, -2.5%, -3.2%, -2.2%, -1.3%, and -0.9%, which is very similar to previous results. Row 2 reports the results excluding Hualien and Taitung, the two eastern regions with sparse population that were least affected by the 1918 influenza due to difficulties in transportation. The coefficient estimates, in the same order as above, are -0.41, -3.6%, -4.3%, -2.7%, -1.8%, and -0.7%--slightly larger than the results using the whole Taiwanese population. Row 3 and Row 4 report coefficient results for males and females separately. For males, the coefficients are significant for education year, junior high, senior high and college, but for females, only years of education, literacy, and elementary school completion are significant. The gender difference may due to the fact that males and females have different levels of educational attainment in our sample. On the average, males have 4.12 years of education, are 65.8% literate and 59.01% complete elementary school. If many males can at least finish elementary school,

[9] Hence from now on we will only report OLS result for simplicity. However, the corresponding probit estimations all yield similar results.

then the variables that affect education may not be important at the elementary school level. On the other hand, if only very few females can achieve education above junior high, then the influenza may not have any effect at higher levels either. Finally, comparing table 4-1 and table 4-2, clustering by year and region does not change the significant patterns. In summary, control variables may not have significant explanatory power if either many people or only a few people are achieving a particular level of education.

[Table 4-1 and 4-2 insert here]

How large is influenzas effect on educational attainment? The average maternal mortality rate during the first half-year of 1918 and 1919 was 0.505%, while the rate for the second half of 1918 was 0.838%. Taking the coefficients estimated in table 4 at face value, and comparing them with average educational attainment shown in table 1, we can calculate the effect of influenza. First, we know the maternal mortality rate increased 0.333%, and the coefficient of maternal mortality on education years is -0.343, so the 1918 influenza epidemic reduced Taiwanese education by about 0.11 (=0.333*0.343) years. This is roughly 4.1% of the average education length, 2.79 years. By performing similar calculations, we see that influenza accounts for 2%, 3%, 9%, 13.5%, and 15.5% respectively of the probability of being literate, completing elementary school, junior high school, senior higher school, and college. The results are in Table 5. Clearly, the explanatory power of influenza is increasing in educational level. This indicates that the effect of influenza mostly happens at the higher lever education which only less than 10% of the population can attain. If half of the population can graduate from elementary school, then the effect of influenza won't show up at the elementary school level.

[Table 5 inserts here]

However, if we discussed the effect by gender, the picture would be quite different. Although influenza reduces educational years a similar amount for both males and females, the

average education for males (4.2 years) is almost three times larger than the education for females (1.5 years). Thus, the 0.33% increase in maternal mortality accounts for 2.8% and 7.3% of the male and female education respectively. These results are consistent with the discrimination story that parents are less likely to invest in girls, and this tendency become stronger when they find out their daughters possess less health capital.

Health Attainment- Maternal Mortality as a Proxy for in utero Pandemic Exposure

So far we have shown that exposure to influenza *in utero* affects one's future educational outcome. Since health and education are highly correlated (the causality goes both ways), it is legitimate to conjecture that *in utero* influenza exposure would affect one's future health. In addition, it has been shown in the literature that conditions in early life affect health in old age and ultimately mortality(Almond and Mazumder 2005, Almond 2006, and Van Den Berg, Lindeboom, and Portrait 2006).

Health Attainment-Attrition Rate

Barker's hypothesis suggests that it is logical to hypothesize that the 1918 cohort experienced higher mortality compared with those born just earlier or later.. Consequently, this cohort might occupy "less than its fair share" of the sample in the 1980 census. To verify this, we run:

$$\text{Cohort Size\%}_{ijt} = \alpha + \beta_1 * \text{Maternal Mortality Rate}_{jt} + \beta_2 * \text{Infant Mortality Rate}_{jt} + \beta_3 * \text{Female}_i + \boldsymbol{\beta_4} * \text{Regional Dummies} + \boldsymbol{\beta_5} * \text{Linear Trend (overall or by regions)} \quad (2)$$

Where i denotes gender, j denotes region, t is year, and the sample years are from 1917 to 1921. That is, we first calculate the sizes of each cohort by sex, region, and year, say males born in Taipei in 1918, then divide them by the total population born between 1917 and 1921 in the 1980 census. If Barker's hypothesis is correct, a higher maternal mortality rate should be

associated with lower cohort size. Table 6 reports the results. It shows that one percentage increase of the maternal mortality rate would decrease the cohort size (compared to the overall population) by roughly 1% once the full model specification has been used. Separating the sample by sex gives the same results. Finally, the 1919 first half-year cohort occupied 8.95% of the whole 1917-1921 population, while our model predicts a 9.87% share of the whole population if the usual maternal mortality rate is used. A back of the envelop calculation indicate that the 0.33% increase of maternal mortality during the pandemic would explain 35% (=(0.33*0.988)/(9.87-8.95)) of this predicted error. Notice that since we controlled for infant mortality, here the excess mortality effect was not contributed by those who were born in 1918 and died instantly, but instead it was contributed by those who perished gradually over the course of their lifetime.

[Table 6 inserts here]

Health Attainment-Health in Old Age

The Barker hypothesis also suggests that fetus influenza exposure can affect health condition later in life. For example, Konje et al (1996) and Langley-Evans (2001) find that the "fetal kidney appears to be extremely vulnerable to the effects of growth retardation. …(and is) disproportionately affected relative to the other organs" Heider (1934) finds that "effect of influenza on hearing occurred only with children who were less than four months old at the time of the epidemic (Almond and Mazumder 2005)." Using SIPP data, Almond and Mazumder (2005) report that the pandemic-exposed cohort exhibited relatively poor health, trouble hearing, speaking, lifting and walking as well as higher rates of diabetes and stroke.[10] Table 7 uses the

[10] The effects of maternal mortality on stroke and self reported health in our data are insignificant and small, differ from the large and significant effects found by Almond and Mazumder (2005).

1989 Survey of Health and Living Status of the Elderly in Taiwan to investigate this point. Using equation 1 as the model specification, we find that an increase of one percentage point in maternal mortality exposure increases an individual's probability of having kidney disease, vertigo (dizziness), tennitus (ringing of the ears), circulatory disease, thyroid problems, and respiratory disease (including asthma, bronchitis and other breathing related disease) by 29%, 45%, 23%, 27%, 53%, and 23% respectively. These results are consistent with the medical literature mentioned above. The average percentage of the sample which has these diseases is also reported, and apparently the pandemic explains a non–trivial part of the variation.

[Table 7 insert here]

Health Attainment-Weight and Height in Childhood and Adolescents

Height and weight are also good indicators of one's health condition. For example, Forgel (1994) argued that height should be correlated with consumption hence nutrition received in childhood. If fetal hypothesis holds, we shall see those who were born in 1919 be with less height and weight, compared to the surrounding cohort. Our first evidence comes from Taipei County's Statistical Book, 1929-1938, in which it documents average height for male students from age 13 to 17 each year. This allows us to back up the average height for those who were born from 1916 to 1921, and then trace their average height from age 13 to 17.[11] Figure 3-1 reports the results. Take age 16 as an example: at this age the average height (meters) for male students born between 1917 and 1921 were 1.58, 1.59, 1.56, 1.59, and 1.59 respectively. Most of them were at the same height at the same age, except for those who were born in 1919: they were 3 centimeters shorter than their surrounding cohort at the same age. Same pattern appears except for age 13. Figure 3-2 reports the average weight of male students by age and year born, and it also shows similar dent for the 1919 cohort.

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

[Figure 3-1 and 3-2 insert here]

However, sample used in the above figures may not be representative for two reasons. First, given the fact that the average year of education at that time was 3 years, only a small fraction of population from age 13 to 17 were "students". In fact, the sample size is around only 600 each year for age 13 to 17. Secondly, the results are from Taipei County only, since Statistical Books from other counties do not report such measures. Consequently, we use data from *Health Statistics for School Students, 1927* (昭和二年學校生徒及兒童身體檢查統計書), which is a cross sectional survey that records height and weight of all Taiwanese students by region and age (hence birth year) in 1927. We regress individual height and weight using the same model specification in equations (1) and (2). Table 8-1 and 8-2 shows that a one percentage point increase in maternal mortality decreases the average weight and height of Taiwanese students by 0.5kg and 0.5cm, respectively, In addition, the effect is larger for females (0.57kg, 0.9cm) than it is for males (0.5kg, 0.3cm). However, given the fact that average height and weight are about 120 cm and 25kg, the effect is not large, especially compared with influenza's effect on education.

[Table 8-1 and 8-2 insert here]

V Discussion and Conclusion

This paper finds that prenatal exposure to the 1918 influenza pandemic affected developmental outcomes many years later. By using maternal mortality rate as a proxy variable 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 and lower weight as teenagers, a higher chance of having various health issues including hearing, kidney, circulatory, thyroid, and

respiratory problems as adults, and they died earlier. This result is consistent with the fetal origin hypothesis.

However, as Almond (2006) has mentioned, although the unexpected and short pandemic "(generates a) sharp prediction for long term effect", there are several alternative hypotheses that may bias our estimates. First, infant mortality increased sharply during the pandemic, from an average of 16% to 24%. In addition, those who were exposed to the pandemic tended to die earlier. So the adverse effect here has been estimated from a "positively selected sample." However, had the weaker subpopulation not died out and if they were observed in the later survey, the true exposure effect would be even more negative. Hence the estimation is downward-biased. If serious enough, the downward biased result could make the desired effect disappear or goes in the opposite direction. For example, Van Den Berg, Lindeboom, and Portrait (2006) find no evidence of the effect of 1918 influenza in their HSN sample. Moreover, they even find that the 1849 cholera in Utrecht and 1870 -1871 smallpox epidemic actually reduced mortality. However, in this paper, along with Almond and Mazumder (2005), and Almond (2006), the influenza effect is strong enough to obtain a negative lower bound estimate even in the presence of possible downward bias.

Second, influenza might affect later health and educational outcomes through channels other than one's initial health. For example, the sharp increase in maternal mortality during the epidemic suggests that more mothers died while giving birth. Hence the adverse developmental outcomes exhibited by the exposure cohorts may not stem from initial deteriorated health but instead because the infants become orphans. However, since maternal mortality rate increase by only 0.3% during the pandemic, even if we assume that orphans received no education at all, a back of the envelope calculation suggest this effect is negligible.

Third, the 1919 cohort was 25 years old in 1944. If this weaker cohort was less likely to be

drafted into military, then our result would also be biased since healthier men would be more likely to be drafted and die in combat. Following Almond (2006), we can also argue that since the exposure effect exists for both men and women, World War II should not jeopardize our measurement of the influenza effect. In addition, unlike the U.S. which had around 300,000 combat causalities during the war, Taiwan only had about 3,000 combat causalities because the Colonial Japanese government only began the draft of Taiwanese men in early 1945.[12] Hence our data is less exposed than US data to biases introduced by World War II. Similarly, if healthier people are more likely to emigrate from Taiwan to other countries, our measured effect would be underestimated. Nevertheless, this problem should be less serious here than Almond (2006) since there was little out migration in early 20th century Taiwan.

Finally, although we linked birth condition data and developmental outcome data from many years later, what happened in between these two data points was still not observed. It is likely that parents invest less in weaker children because they anticipate a low return on their investment. That is, health endowment at birth and health investment after birth, which depends critically on the health endowment, both enter the health and education production function. Distinguishing the two effects should be an important direction for future research.

Overall, our empirical results suggest that exposure to the influenza pandemic *in utero* had a significant long run adverse effect on an individual's education, height, weight, elderly health condition, and life expectancy. Consistent with Almond (2006), this result extends our understanding of the effects of conditions in early life one step further. While researchers had previously find that conditions immediately after birth had long-term effects, our results suggest that conditions before birth matter, too.

Moreover, identifying the relationship between early life conditions and later development

[12] In 1942, the 15-29 male population for US and Taiwan were 15 million and 700,000, respectively.

outcomes does not only serve our intellectual curiosity, but also has important policy implications. When individuals who experience poor health and hence receive lower human capital investment in the early stage of their life have children, they pass the poor health conditions on to the next generation (Currie and Moretti 2003). Currie (2009)'s detailed literature review concludes that "there is now strong evidence… (that) health could play a role in the intergenerational transmission of economic status." Policies improving early life health conditions such as the provision of food, health care, or housing to pregnant women and younger children may help break the intergenerational poverty cycle and help improve adult health. The results of this study indicate that resource reallocation toward the earlier stages of life would be an effective policy to improve adult economic and health outcomes and hence reduce inequality. The challenge, of course, remains to identify which improvements can increase fetal health and hence subsequent outcomes, as well as the cost-benefit analysis of such a policy.

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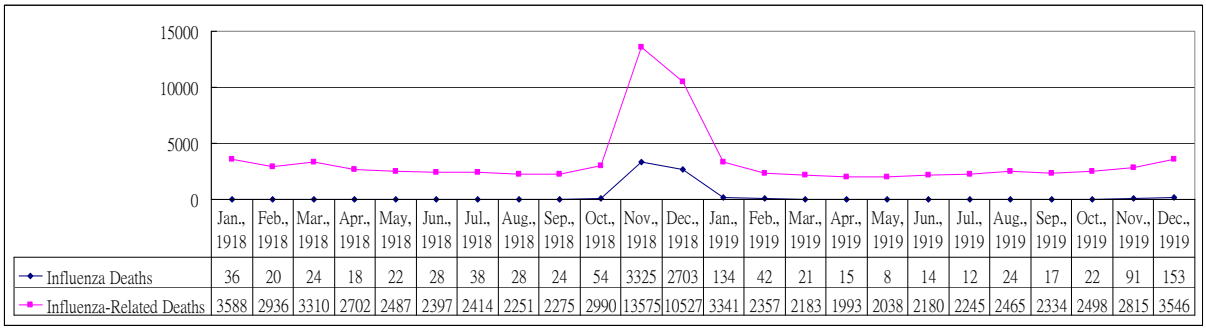

	Jan., 1918	Feb., 1918	Mar., 1918	Apr., 1918	May, 1918	Jun., 1918	Jul., 1918	Aug., 1918	Sep., 1918	Oct., 1918	Nov., 1918	Dec., 1918	Jan., 1919	Feb., 1919	Mar., 1919	Apr., 1919	May, 1919	Jun., 1919	Jul., 1919	Aug., 1919	Sep., 1919	Oct., 1919	Nov., 1919	Dec., 1919
Influenza Deaths	36	20	24	18	22	28	38	28	24	54	3325	2703	134	42	21	15	8	14	12	24	17	22	91	153
Influenza-Related Deaths	3588	2936	3310	2702	2487	2397	2414	2251	2275	2990	13575	10527	3341	2357	2183	1993	2038	2180	2245	2465	2334	2498	2815	3546

Figure 1-1: Monthly Influenza-Related Deaths, 1918-1919

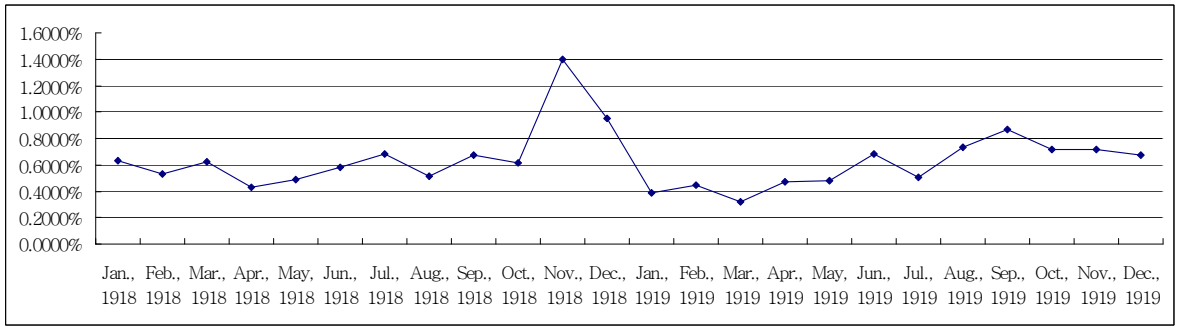

Figure 1-2: Monthly Maternal Mortality Rate, 1918-1919

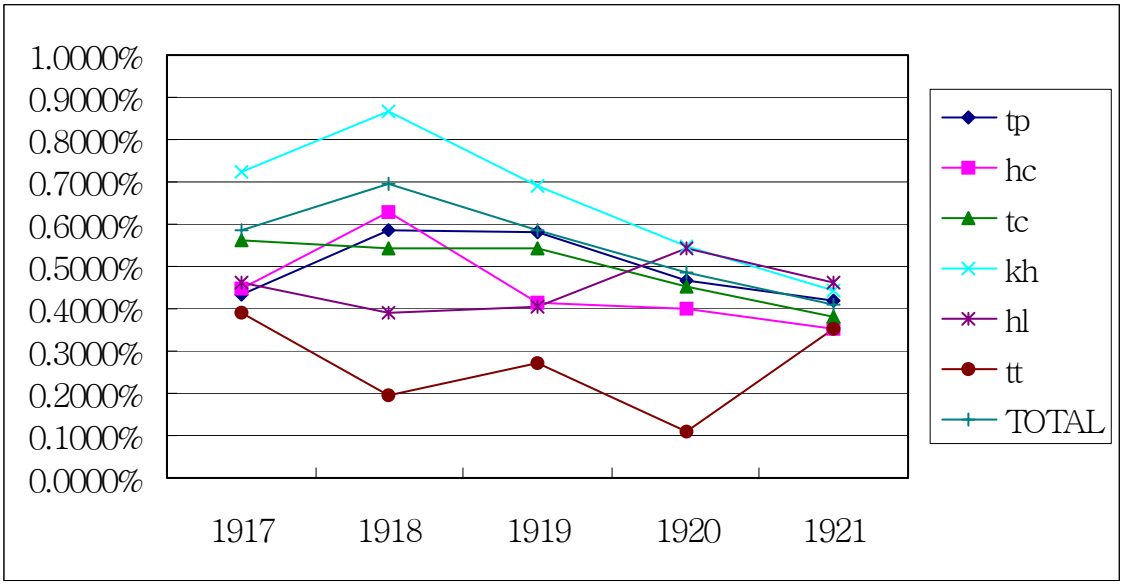

Figure 1-3: Maternal Mortality Rate, 1917-1921

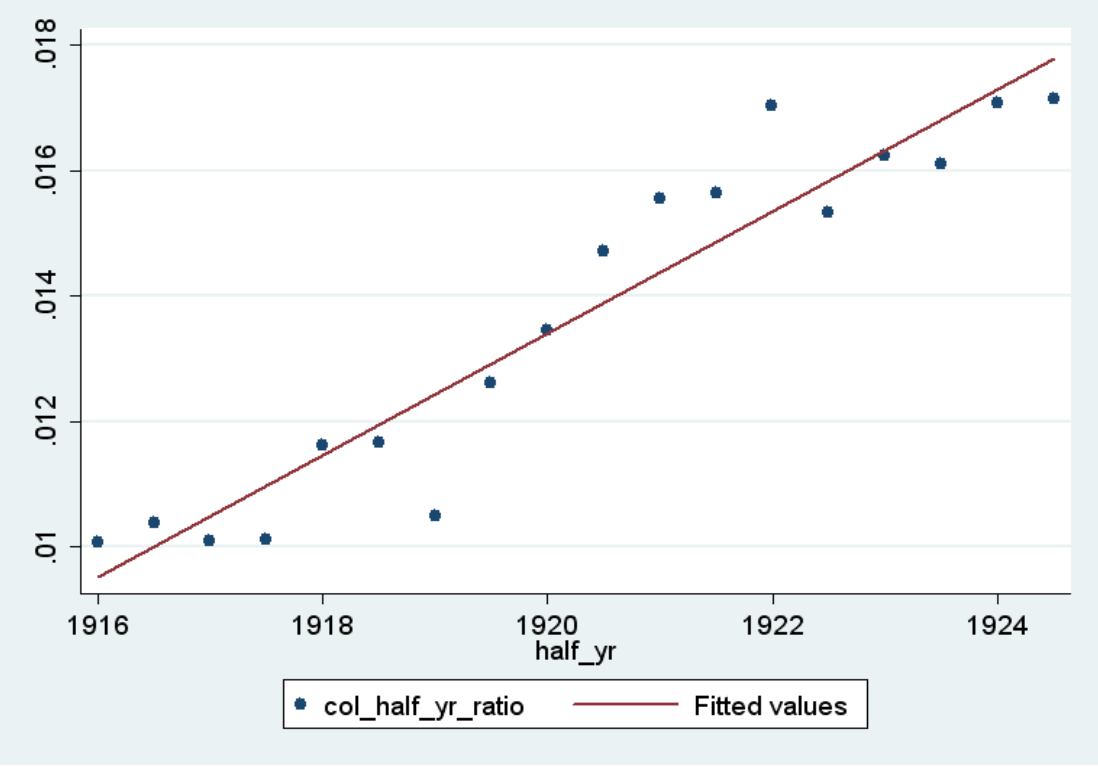

Figure 2-1: Percentage College Education Completion by half-year birth cohorts: Complete Sample from 1980 Census

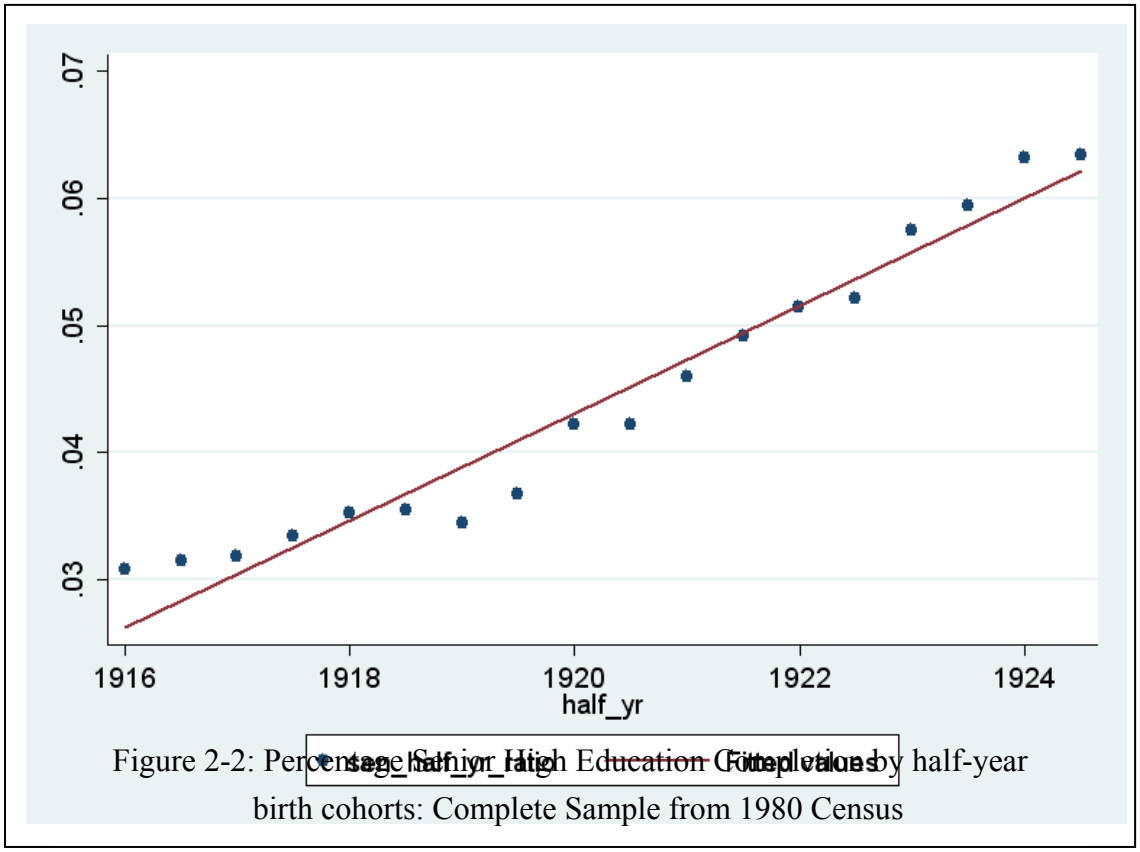

Figure 2-2: Percentage Senior High Education Completed by half-year birth cohorts: Complete Sample from 1980 Census

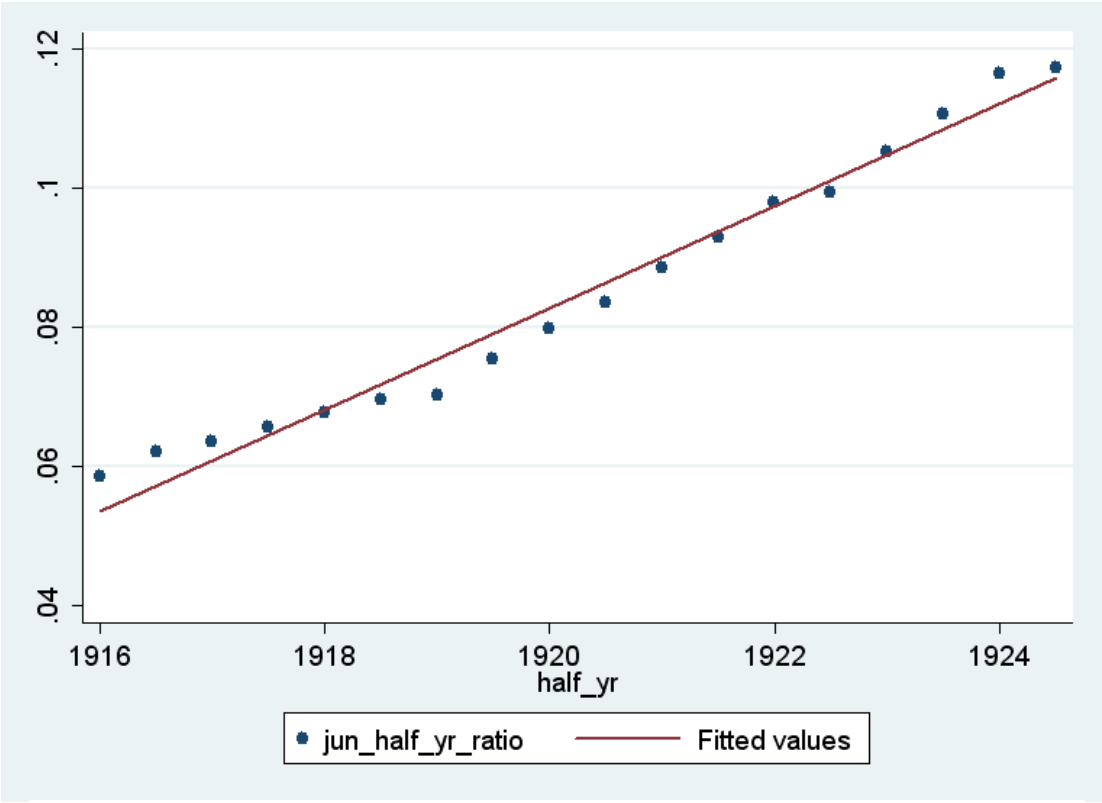

Figure 2-3: Percentage Junior High Education Completion by half-year birth cohorts: Complete Sample from 1980 Census

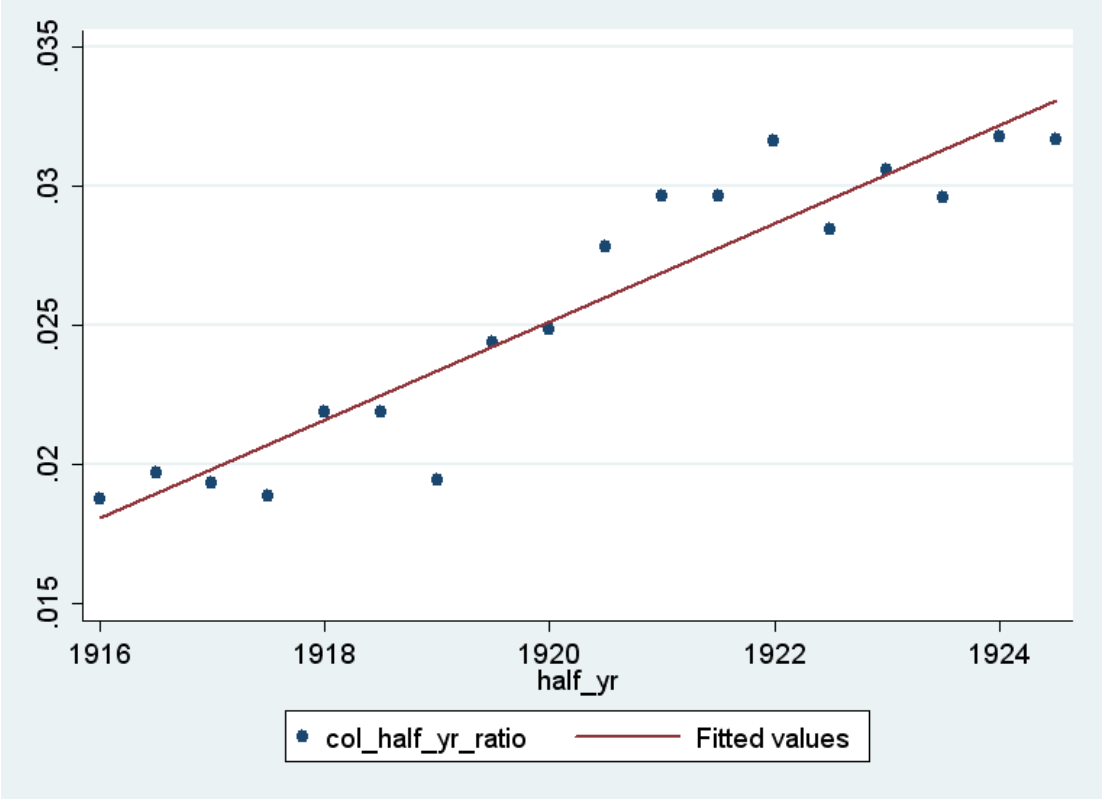

Figure 2-4: Percentage College Education Completion by half-year birth cohorts: Males born in the Western Region from 1980 Census

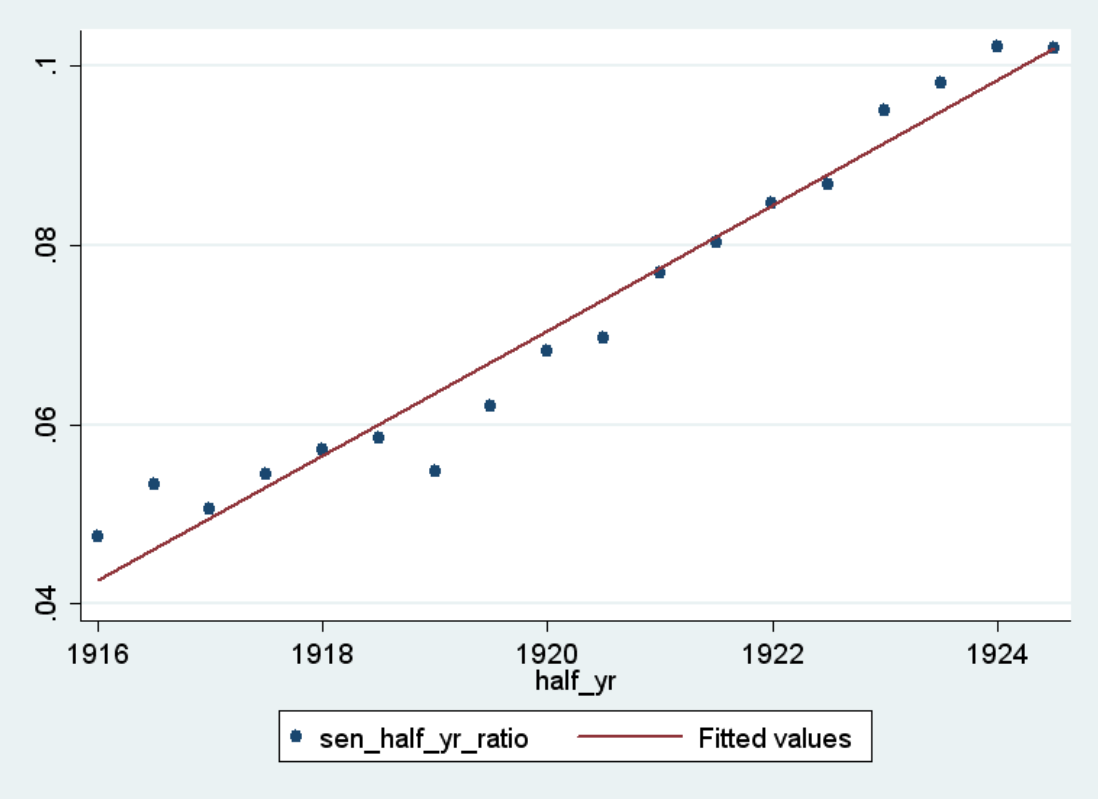

Figure 2-5: Percentage Senior High Education Completion by half-year birth cohorts: Males born in the Western Region from 1980 Census

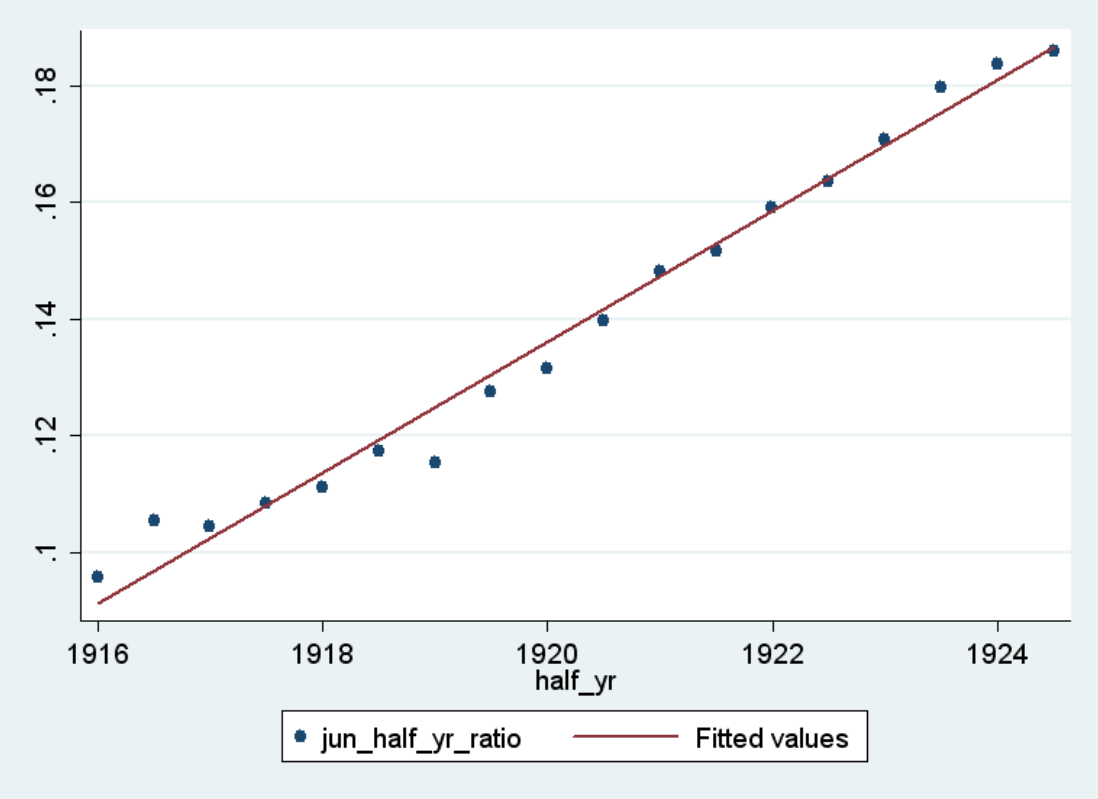

Figure 2-6: Percentage Junior High Education Completion by half-year birth cohorts: Males born in the Western Region from 1980 Census

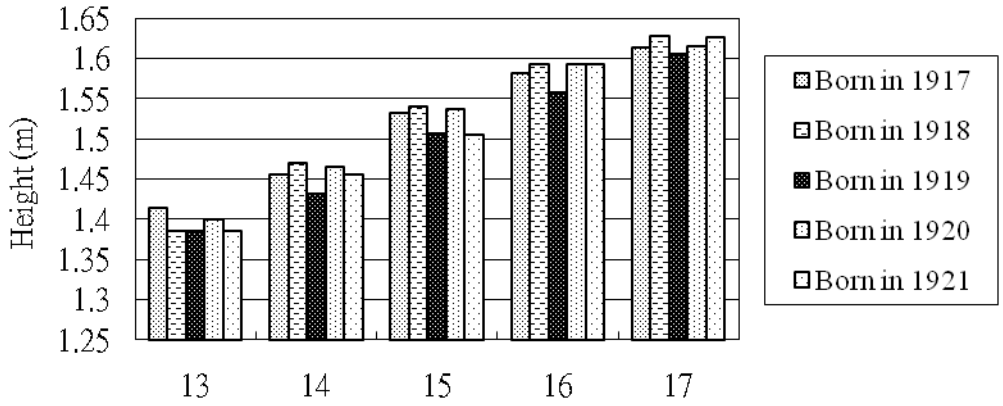

Figure 3-1: Average weight of male students: age 13-17, born 1917-1921

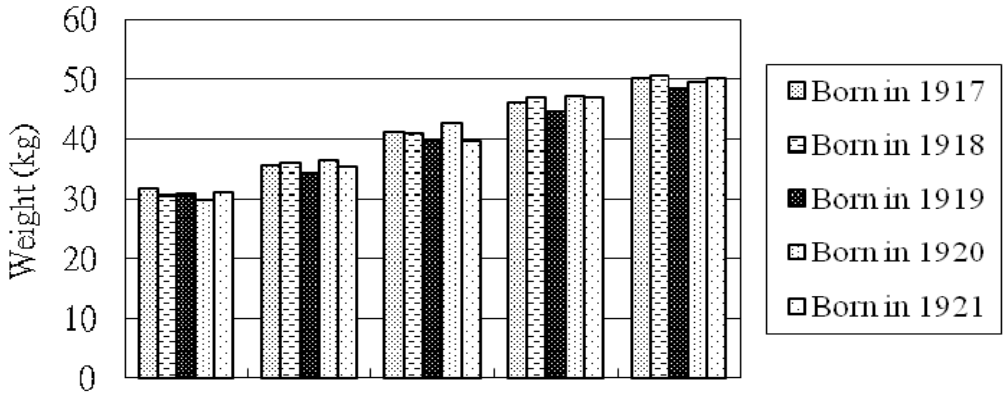

Figure 3-2: Average weight of male students: age 13-17, born 1917-1921

Table 1: Summary Statistics- Individuals Born between 1917 and 1921 in 1980 Census

Variable	Total Mean (n=361,082)	Male Mean (n=172,475)	Female Mean (n=188,625)
Educational variables			
Years of schooling	2.785	4.188	1.502
	(3.696)	(3.939)	(2.918)
Literacy (%)	45.37	65.83	26.66
	(0.498)	(0.474)	(0.442)
Elementary School	39.94	59.01	22.51
Completion (%)	(0.490)	(0.492)	(0.418)
Junior High School	7.66	12.68	3.08
Completion (%)	(0.266)	(0.333)	(0.173)
Senior High School	3.92	6.36	1.69
Completion (%)	(0.194)	(0.244)	(0.129)
College Completion	1.28	2.37	0.28
(%)	(0.112)	(0.152)	(0.053)
Female (%)	52.24		
(=1 if female)	(0.499)		

Mortality variables			
Variable	Mean	min	Max
Maternal mortality	0.54	0.11	0.87
rate (%)	(0.144)		
Infant mortality rate	16.91	13.11	24.74
(%)	(1.718)		

Table 2: Effect of Maternal Mortality Rate on Years of Education: 1917-1921

	(1)	(2)	(3)	(4)	(5)	(6)
Maternal	-1.957***	-1.832***	-1.704***	-0.172**	-0.343***	-0.343***
Mortality Rate	(0.042)	(0.044)	(0.059)	(0.076)	(0.081)	(0.110)
Female		-2.680***	-2.677***	-2.675***	-2.675***	-2.675***
		(0.012)	(0.012)	(0.012)	(0.012)	(0.034)
Infant		-0.256	0.449	-0.527	-0.849*	-0.849*
mortality rate		(0.369)	(0.440)	(0.441)	(0.446)	(0.482)
Regional	No	No	Yes	Yes	Yes	Yes
dummy						
Overall	No	No	No	0.168***	No	No
Linear Trend				(0.005)		
Regional	No	No	No	No	Yes	Yes
specific trend						
Clustered on	No	No	No	No	No	Yes
year & region						
Constant	3.893***	5.265***	5.368***	4.650***	4.745***	4.745***
	(0.025)	(0.056)	(0.081)	(0.084)	(0.090)	(0.094)
Observations	361,082	361,082	361,082	361,082	361,082	361,082

Note: ***, **, and * are significant at 1%, 5%, 10% respectively. The corresponding maternal mortality rate has a half-year lag. Using a contemporary or one year lag, maternal mortality rate yields similar results.

Table 3 Effect of Maternal Mortality Rate on Five Educational Attainment Categories 1917-1921: (1)-(5) OLS and (6) Probit Results

	Literacy	Elementary School	Junior High	Senior High	College
(1)independent variable: Maternal mortality rate (half-year lag) only					
Maternal Mortality	-0.293***	-0.280***	-0.047***	-0.035***	-0.010***
Rate	(0.006)	(0.006)	(0.003)	(0.002)	(0.001)
(2)independent variables: (1) + female + infant mortality rate					
Maternal Mortality	-0.267***	-0.267***	-0.037***	-0.031***	-0.009***
Rate	(0.006)	(0.006)	(0.003)	(0.002)	(0.001)
(3)independent variables: (2) + Regional Dummies					
Maternal Mortality	-0.219***	-0.224***	-0.066***	-0.039***	-0.015***
Rate	(0.008)	(0.008)	(0.005)	(0.003)	(0.002)
(4)independent variables: (3) + Regional Specific Trend Dummies					
Maternal Mortality	-0.029***	-0.036***	-0.021***	-0.016***	-0.006**
Rate	(0.011)	(0.011)	(0.006)	(0.004)	(0.003)
(5)independent variables: (4)+Cluster on half-year and region					
Maternal Mortality	-0.029**	-0.036***	-0.021***	-0.016***	-0.006**
Rate	(0.014)	(0.013)	(0.008)	(0.006)	(0.003)
(6)independent variables: (5), probit +Cluster on half-year and region					
Maternal Mortality	-0.033**	-0.040***	-0.018***	-0.013***	-0.004*
Rate	(0.016)	(0.014)	(0.006)	(0.005)	(0.002)

Note: Standard errors are in the parentheses. ***, **, and * are significant at 1%, 5%, 10% respectively. The corresponding maternal mortality rate has a half-year lag. N=361,082. Probit results in row (6) report marginal effects, and are also clustered by half-year and region.

Table 4-1: Effect of Maternal Mortality Rate on Six Educational Attainment Categories: Sensitivity Analysis Using Full Model Specification

Education	Literacy	Elementary	Junior High	Senior High	College

Year		School			
(1)1918-1920 (n=209,606)					
-0.324***	-0.0252*	-0.032**	-0.022***	-0.013**	-0.009***
(0.109)	(0.015)	(0.014)	(0.008)	(0.006)	(0.003)
(2)Male only (n=172,457)					
-0.355***	-0.011	-0.020	-0.035***	-0.030***	-0.014***
(0.134)	(0.016)	(0.017)	(0.011)	(0.008)	(0.005)
(3)Female only (n=188,625)					
-0.332***	-0.045***	-0.049***	-0.009	-0.003	0.001
(0.096)	(0.015)	(0.014)	(0.006)	(0.004)	(0.002)
(4)Exclude Hualien and Taitung (n=348,087)					
-0.413***	-0.036***	-0.043***	-0.027***	-0.018***	-0.007**
(0.087)	(0.012)	(0.011)	(0.007)	(0.005)	(0.003)

Table 4-2: Effect of Maternal Mortality Rate on Six Educational Attainment Categories: Sensitivity Analysis Using Full Model Specification with Cluster on Half-Year and Region

Education Year	Literacy	Elementary School	Junior High	Senior High	College
(1)1918-1920 (n=209,606)					
-0.324**	-0.025	-0.032*	-0.022**	-0.013**	-0.009**
(0.134)	(0.017)	(0.017)	(0.008)	(0.005)	(0.004)
(2)Male only (n=172,457)					
-0.355**	-0.011	-0.020	-0.035**	-0.030***	-0.014**
(0.163)	(0.016)	(0.015)	(0.015)	(0.011)	(0.006)
(3)Female only (n=188,625)					
-0.332***	-0.045**	-0.049***	-0.009*	-0.003	-0.001
(0.118)	(0.018)	(0.017)	(0.005)	(0.004)	(0.001)
(4)Exclude Hualien and Taitung (n=348,087)					
-0.413***	-0.036**	-0.043***	-0.027***	-0.018***	-0.007**
(0.118)	(0.015)	(0.014)	(0.008)	(0.006)	(0.003)

Note: ***, **, and * are significant at 1%, 5%, 10% respectively. The corresponding maternal mortality rate has a half-year lag. Hualien and Taitung belong to the east region of Taiwan and are less affected by the pandemic.

Table 5 How Much Educational Attainment is Explained by the 1918 Taiwanese Influenza Pandemic? A Back of the Envelope Calculation

	Education Year	Literacy	Element. School	Junior High	Senior High	College
Overall	2nd half of 1918 maternal mortality rate increased by 0.33%					
(A)Coefficient	-0.343	-0.029	-0.036	-0.021	-0.016	-0.006
(B)Reduced Education Attainment by (0.33*(A))	-0.11319	-0.00957	-0.01188	-0.00693	-0.00528	-0.00198
(C)Average Education Attainment	2.785	45.37%	39.94%	7.66%	3.92%	1.28%
(D)% Explained	-4.06	-0.02	-0.030	-0.090	-0.135	-0.155
Male	2nd half of 1918 maternal mortality rate increased by 0.33%					
(A)Coefficient	-0.355	-0.011	-0.02	-0.035	-0.03	-0.014
(B)Reduced Education Attainment by (0.33*(A))	-0.11715	-0.00363	-0.0066	-0.01155	-0.0099	-0.00462
(C)Average Education Attainment	4.188	65.83	59.01	12.68	6.36	2.37
(D)% Explained	-2.797	-0.006	-0.011	-0.091	-0.156	-0.195
Female	2nd half of 1918 maternal mortality rate increased by 0.33%					
(A)Coefficient	-0.332	-0.045	-0.049	-0.009	-0.003	0.001
(B)Reduced Education Attainment by (0.33*(A))	-0.10956	-0.01485	-0.01617	-0.00297	-0.00099	0.00033
(C)Average Education Attainment	1.502	26.66	22.51	3.08	1.69	0.28
(D)% Explained	-7.294	-0.056	-0.072	-0.096	-0.059	0.118

Table 6: Effect of Maternal Mortality Rate on Cohort Population Percentage-
Individuals born between 1917 and 1921 in 1980 Census

	Ratio	Ratio	Ratio	Ratio	Ratio-Male	Ratio-Female	Number (1000)
Maternal mortality	2.091***	-1.342***	-1.077***	-0.988***	-1.986**	-1.969**	-3.568***
	(0.371)	(0.225)	(0.268)	(0.268)	(0.774)	(0.744)	(0.967)
Female	0.154	0.142***	0.143***	0.143***			0.515***
	(0.162)	(0.033)	(0.033)	(0.033)			(0.121)
Infant Mortality Rate	-0.043	-2.265*	-2.440**	-2.217*	-5.142	-3.827	-8.006*
	(4.463)	(1.221)	(1.199)	(1.159)	(3.178)	(3.372)	(4.186)
Regional dummy	No	Yes	Yes	Yes	Yes	Yes	Yes
Regional specific trend	No	No	No	Yes	Yes	Yes	Yes
Liner trend	No	No	Yes	No	No	No	No
Constant	0.148	1.040***	0.708***	0.719***	1.985***	1.725**	2.596***
	(0.805)	(0.270)	(0.255)	(0.250)	(0.729)	(0.735)	(0.901)
Observations	120	120	120	120	60	60	120

Note: ***, **, and * are significant at 1%, 5%, 10% respectively. Regression is weighted by cohort population.

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

	Kidney Disease	Vertigo (Dizzy)	Tinnitus (ear ringing)	Circulatory disease	Thyroid problem	Respiratory disease
Maternal Mortality	0.287***	0.465**	0.226*	0.268*	0.533*	0.225
	(0.064)	(0.199)	(0.134)	(0.150)	(0.032)	(0.158)
Average % who had the disease	6.7%	28.2%	15.89%	21.26%	1.35%	19.2%

Note: Other independent variables includes: sex, infant mortality, birth county dummies, county specific trend, and year dummies. The regression is clustered by county and year. Samples are those who were born in Taiwan between 1916 and 1928, N=2,367. Respiritory diseases include asthma, bronchitis, and other breathing related disease. ***, **, and * are significant at 1%, 5%, 10% respectively

Table 8-1: Effect of Maternal Mortality Rate on Weight (kg) in the 1928 Survey

	Overall	Overall	Overall	Male	Female
Maternal mortality	-1.353***	-0.425***	-0.504***	-0.500***	-0.568***
	(0.049)	(0.103)	(0.106)	(0.124)	(0.205)
Female	-0.260***	-0.282***	-0.283***		
	(0.014)	(0.014)	(0.014)		
Infant	-0.037***	-0.037***	-0.042***	-0.046***	-0.035***
Mortality rate	(0.004)	(0.006)	(0.006)	(0.007)	(0.010)
Age	1.66***	1.66***	1.76***	1.733***	1.660***
	(0.006)	(0.006)	(0.041)	(0.065)	(0.069)
Regional dummy	No	Yes	Yes	Yes	Yes
Regional specific	No	No	Yes	Yes	Yes
trend					
Constant	7.21***	6.86***	6.15***	6.457***	6.518***
	(0.087)	(0.136)	(0.329)	(0.537)	(0.613)
Observations	130232	130232	130232	95103	35129

Table 8-2: Effect of Maternal Mortality Rate on Height (m) in the 1928 Survey

	Overall	Overall	Overall	Male	Female
Maternal mortality	-0.025***	-0.002	-0.005*	-0.003	-0.009*
	(0.001)	(0.003)	(0.003)	(0.004)	(0.005)
Female	-0.790***	-0.762***	-0.762***		
	(0.034)	(0.035)	(0.035)		
Infant	-0.084***	0.028**	0.015	0.012	0.021
Mortality rate	(0.009)	(0.013)	(0.014)	(0.018)	(0.024)
Age	4.469***	4.561***	4.534***	4.434***	4.375***
	(0.016)	(0.017)	(0.138)	(0.138)	(0.159)
Regional dummy	No	Yes	Yes	Yes	Yes
Regional specific	No	No	Yes	Yes	Yes
trend					
Constant	80.787***	77.079***	77.699***	78.548***	78.158***
	(0.223)	(0.390)	(1.236)	(1.131)	(1.336)
Observations	114250	114250	114250	83211	31039

Note: ***, **, and * are significant at 1%, 5%, 10% respectively

Table A1 Influenza Related Death by Diseases: 1918-1919 Monthly Data

	Influ -enza	Tuberc -ulosis	Acute Bronchitis	Chronic Bronchitis	Pneum -onia	Other Respiratory Disease	Total
1918/01	36	572	241	540	1350	849	3588
/02	20	507	220	401	1125	663	2936
/03	24	540	261	398	1441	646	3310
/04	18	511	201	293	1160	519	2702
/05	22	469	217	288	1036	455	2487
/06	28	437	253	213	1036	430	2397
/07	38	435	231	242	1052	416	2414
/08	28	449	226	241	831	476	2251
/09	24	387	222	259	936	447	2275
/10	54	519	274	326	1223	594	2990
/11	3325	982	607	814	6155	1692	13575
/12	2703	725	485	682	4749	1183	10527
1919/01	134	524	294	380	1370	639	3341
/02	42	384	204	328	906	493	2357
/03	21	452	182	253	854	421	2183
/04	15	417	183	215	791	372	1993
/05	8	411	184	190	826	419	2038
/06	14	448	173	225	929	391	2180
/07	12	467	220	203	932	411	2245
/08	24	534	219	275	927	486	2465
/09	17	551	221	275	798	472	2334
/10	22	633	207	276	853	507	2498
/11	91	578	265	308	1064	509	2815
/12	153	629	262	437	1453	612	3546