Multigenerational effects of the 1918-19 influenza pandemic on educational attainment: Evidence from Sweden

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Abstract

We use the 1918-19 influenza pandemic in Sweden as a natural experiment to estimate the effects of a fetal health shock on the children of those who experienced the pandemic as a fetal insult. Potential exposure is constructed using time of birth information available in Swedish register data. For women, educational attainment decreases by 3-4 months of schooling and the probability of college attendance drops by 3-5 percentage points if their mothers potentially experienced the Spanish flu as a fetal insult. For men, educational attainment decreases by 4-7 months of schooling, and the probability of college attendance drops by 7-11 percentage points if their fathers were potentially prenatally exposed. We find no mother to son nor father to daughter transmission of the health shock.

JEL classification: I12, J13, J24, N34

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1 Motivation

Animal experiments have documented that health shocks during critical developmental periods can be transmitted to several subsequent offspring generations, suggesting that health outcomes in any given generation may have biological roots in events that occurred some generations earlier (cf. Drake & Walker (2004) and Drake & Liu (2010)). In human populations, the effect of prenatal health shocks on adult outcomes has been extensively documented by economists and epidemiologists alike, and due to the evidence accumulated so far, it is now generally accepted that prenatal health causally affects both short and long run outcomes. See Almond & Currie (2011), Black et al. (2007), Currie (2009) and Currie (2011) and Aizer & Currie (2014). It is also known that prenatal health measures are correlated with the socioeconomic background of the parent, supporting the view that prenatal health differences could drive intergenerational correlations. Direct evidence for such effects of prenatal health on subsequent generations in human populations is scarce, though, and the importance of any such effects remains unclear.

This paper provides evidence for effects of prenatal health shocks on economically relevant outcomes for children of those who suffered a prenatal insult. We follow the fetal origins literature and exploit an exogenous shock to prenatal health that lies "outside the control of the mother" (Currie, 2009) to avoid confounding with maternal or family characteristics. That is, we identify reduced form effects of a prenatal insult on the grandchildren of those who suffered a health shock while they were pregnant with one of the parents. We follow the seminal work of Almond (2006) and use potential maternal in utero exposure to the 1918 influenza virus, or Spanish flu, as an exogenous health shock to estimate the causal effect on socioeconomic outcomes of children of prenatally insulted parents. In Sweden, regional influenza morbidity rates on the county (*län*) level are known during that period, and Swedish registers allow us to link individuals to their potentially exposed biological parents. This enables us to identify intent-to-treat effects of the Spanish flu on the second generation.

Most research into the effects of prenatal insults over multiple generations is done with animal experiments. Evidence in humans remains scarce (cf. Drake & Liu (2010)). In economics, we are only aware of three other studies that document multigenerational effects of prenatal insults based on quasi-experimental settings. Almond & Chay (2006) investigate the effects of improvements in broadly defined early life conditions of black mothers due to the civil rights movement on their children. Almond et al. (2010) as well as Kim et al. (2014) exploit the Chinese famine and document effects on the sex ratio and birth weight of babies born to mothers who were prenatally exposed to the Chinese famine, as well as a decrease in junior secondary school entrance. Our study contributes to this literature in three ways: first, we rely on a very well-defined prenatal insult as opposed to more broadly defined early life conditions. Second, with educational attainment and long-run earnings we observe economically in-

teresting outcomes of the children of prenatally insulted parents, and third, using regression controls we are able to shed some light on potential mechanisms driving our results.

Multigenerational effects could be direct or indirect. Effects of the Spanish flu on socioeconomic outcomes of the prenatally exposed are well-established in the literature. The Spanish flu will thus affect subsequent generations via its effect on behavioral, social and economic outcomes of the prenatally insulted parents, which we refer to as indirect effects.

Following the biomedical literature, direct or biological effects need to be considered as well. Since precursors of the ovaries in women and the sperm cells in men already develop at the prenatal stage, prenatal exposure to a health shock also exposes the germ cells that will eventually produce the children.[1] Therefore, we need to consider the offspring of prenatally insulted parents directly "exposed" (cf. Skinner (2008)). While the exact molecular mechanisms are still debated, the existence of such biological effects has been well-established through animal experiments (cf. Drake & Liu (2010)), and some evidence for human populations exists as well (Painter et al., 2008; Heijmans et al., 2008). For related overviews, see Drake & Walker (2004), Drake & Liu (2010), Franklin & Mansuy (2010), Harper (2005), Jablonka & Raz (2009) and Lundborg & Stenberg (2010).

We find strong multigenerational effects of the Spanish flu on educational attainment in Sweden. For women (men), educational attainment decreases by 3-4 (4-7) months of schooling and the probability of college attendance drops by 3-5 (7-11) percentage points if their mothers (fathers) potentially experienced the Spanish flu as a prenatal insult. These intent-to-treat estimates suggest potentially large effects on the infected (e.g. up to 12-16 months less schooling for women and 16-28 months for men). Moreover, using regression controls accounting for indirect mechanisms, the main effects are attenuated by about one standard error of the main estimate.

Our results indicate that intergenerational consequences of prenatal health (cf. Currie (2011)) are much larger than previously imagined, and that from a policy perspective investments in maternal health generate payoffs that might accrue over several generations. Moreover, these results contribute to our understanding of the intergenerational transmission of economic status and supports the view that maternal health shocks trigger intergenerational consequences (cf. Case et al. (2005) and Currie & Stabile (2003)).

This paper is organized as follows: in section 2, we discuss previous literature on this topic and in section 3, we present the historical context of the Spanish flu in Sweden. Our data is discussed in section 4 and our empirical strategy is laid out in section 5. We present our results and a range of robustness checks in sections 6 and 7. We conclude with a discussion in section 8.

[1] The oocytes in women already fully develop at the prenatal stage, whereas spermatocytes in men continue development around the time of puberty.

2 Related Literature

2.1 First generation effects

Lasting effects of prenatal health have been widely documented. See, for instance, Behrman & Rosenzweig (2004), Black et al. (2007), and Currie & Hyson (1999) and Oreopoulos et al. (2008) who use birth weight as a summary measure of prenatal health and document detrimental effects of being born with low birth weight on several later life outcomes. Apart from observational studies, economists have used a wide range of natural experiments to exploit exogenous variation in prenatal health. For instance, Chen & Zhou (2007), Meng & Qian (2009), Scholte et al. (2012), Neelsen & Stratmann (2011), Almond & Mazumder (2011), Ewijk (2011) and Almond et al. (2014) use nutritional deprivation due to famines or Ramadan exposure and find effects of these events on either early or later life outcomes.[2] Similar results are obtained when circumstantial evidence for stress exposure is used, e.g. exposure to civil conflict or war (Camacho, 2008; Lee, 2014; Valente, 2011; Mansour & Rees, 2012), death of a relative (Black et al., 2014) and natural disasters (Simeonova (2009), Currie & Rossin-Slater (2013), among others). In utero exposure to environmental pollution has similarly detrimental effects, see e.g. Almond et al. (2009), Nilsson (2009), Black et al. (2013) and Currie et al. (2014).

The Spanish Flu Literature

In a series of papers, Douglas Almond and coauthors were the first to exploit the 1918 influenza pandemic in the US to test the fetal origins hypothesis. In Almond (2006), Almond & Mazumder (2005) and Mazumder et al. (2010), large reductions in educational attainment, wages, socioeconomic status indices and several health measures are found for the cohorts that were prenatally exposed to the Spanish flu. Brown & Thomas (2011) show however that these results are potentially confounded by a change in parental quality due to conscription procedures for World War I. Evidence from a number of non-participating countries such as Brazil (Nelson, 2010), Taiwan (Lin & Liu, 2014) and Switzerland (Neelsen & Stratmann, 2012) has confirmed Almond's earlier results, though.[3]

While not much is known about the particular virus strain of the Spanish flu, it is believed that it exerted its impact via a so-called *cytokine storm*, i.e. by triggering an overreaction of the immune response (Loo & Gale, 2007). In this context, it is noteworthy that maternal influenza infection during pregnancy has

[2] The impact of nutritional deprivation at later developmental stages has also been studied. See, for instance, Kaati et al. (2007) and Berg et al. (2012).

[3] Other noteworthy studies are Kelly (2011), Parman (2012) and Karlsson et al. (2014). Kelly uses cross-sectional variation in the Asian flu of 1957 in the UK and finds that prenatal exposure to the flu has negative effects on cognitive test score measures. Parman uses the US influenza pandemic in 1918 to identify how a health shock to a child affects the outcomes for its siblings via parental investments. Karlsson et al. (2014) uses Spanish flu mortality in Sweden as a labor supply shock to test empirical predictions of macroeconomic growth models.

been linked to several neurological conditions later in life, in particular when the maternal influenza infection occurs around the second trimester. See, for instance, Brown & Derkits (2010) and Canetta & Brown (2012) for schizophrenia, as well as Parboosing et al. (2013) and Machon et al. (1997) for affective disorders. Brown & Derkits (2010) and Canetta & Brown (2012) discuss that the maternal immune response (i.e. an elevated maternal cytokine level) is the most likely mediator for the association between schizophrenia and maternal influenza infection. The mechanisms through which the Spanish flu and regular influenza strains affect fetuses are thus potentially very similar. This has important implications for the external validity of our results as they might capture the importance of maternal health in general.

2.2 Second generation effects

Evidence for multigenerational effects of prenatal health remains scarce. An intergenerational transmission of birth weight has been documented in observational studies (e.g. Currie & Moretti (2007) and Royer (2009), among others), but twin studies suggest that the genetic component here is likely to be strong (cf. Royer & Witman (2014)). Quasi-experimental evidence linking parental prenatal health to children's outcomes is limited to only a few studies. Almond et al. (2010) use the Chinese famine from 1959 to 1961 as a natural experiment and compare mothers who were in utero during the time of the famine to mothers of adjacent birth cohorts. They find that children were more likely to be girls and to have low birth weight if their mother was prenatally exposed to the famine. Kim et al. (2014) furthermore shows that junior secondary school attendance of individuals born to mothers prenatally exposed to the Chinese famine was reduced by 5-7 percentage points. Almond & Chay (2006) use the civil rights era as a natural experiment and exploit that black women born in the late 1960s experienced better prenatal and infant health than black women born in the early 1960s. They show that children of mothers who were themselves born in the late 1960s had better birth outcomes than children of mothers born in the early 1960s.[4]

Evidence for multigenerational responses of health shocks is more abundant in the biomedical and epidemiological literature. See Drake & Walker (2004) and Drake & Liu (2010) for comprehensive reviews. In particular, animal experiments have produced a substantial body of evidence for multigenerational responses in health outcomes. In a recent systematic review of this literature, Aiken & Ozanne (2014) finds that out of 48 published animal experiments looking at the second generation, 44 found effects while only 4 failed to do so. In these experiments, pregnant animals are exposed to some form of stress (e.g. under- or malnutrition, or excessive exercise) and multiple generations of offspring are observed, who are then compared to a corresponding control group.

[4] Nilsson (2009) uses prenatal alcohol exposure induced by a policy experiment in Sweden and finds no effects on birth outcomes of the children of prenatally exposed parents.

Early examples are Stewart et al. (1975) and Stewart et al. (1980), who follow rats over up to 12 generations and document that adverse health effects of in utero malnourishment perpetuate over three subsequent generations even after the reintroduction of a normal diet. Similarly, Pinto & Shetty (1995) expose pregnant rats to exercise stress and show that not only their offspring but also the second generation offspring are growth-retarded despite sedentary conditions during their pregnancy. In human populations, multigenerational effects of the Dutch Hunger Winter of 1944-1945 (see Roseboom et al. (2011) for a review of related studies) have been documented by Painter et al. (2008), who show that children of mothers that were in utero during the Dutch Hunger Winter were more likely to suffer from atypical conditions. Epigenetic changes have furthermore been identified (Heijmans et al., 2008; Tobi et al., 2009).[5]

Compositional effects

prenatal health shocks may have compositional effects on the second generation through fertility responses of the parents or the prenatally exposed children, but the existing evidence for such effects is mixed. Black et al. (2013) investigate the effect of prenatal exposure to radiation on the probability of having younger siblings, and find no such effect. Nilsson (2009) does not find evidence for fertility responses due to prenatal alcohol exposure either. On the other hand, Neelsen & Stratmann (2012) finds that individuals prenatally exposed to the Spanish flu were less likely to be married, a finding similar to Almond et al. (2010) for the Chinese famine.

3 Historical context

3.1 The Spanish flu as a natural experiment

The pandemic had certain characteristics that facilitate the use as a natural experiment: First, it was a severe health shock. On a global level, it is generally agreed that the 1918 influenza pandemic was one of deadliest epidemics in human history. About 500 million were affected by the Spanish flu, and around 50 million deaths are attributable to it (Taubenberger & Morens, 2006). In Sweden, the first case was reported on the 5th of July in Malmö, and the peak occurred during October to December 1918. Over the course of the pan-

[5] A set of related studies looked at food supply during the slow growth period, i.e. the time around the age of 8-12, which is another critical period in human development. These studies suggest that health outcomes of individuals might be influenced by the food supply during their parents' and grandparents' slow growth period. See Kaati et al. (2002), Bygren et al. (2001), Kaati et al. (2007) and Pembrey et al. (2006), as well as Pembrey (2002) and Pembrey (2010) for overviews. While these studies can be criticized on statistical grounds (e.g. Senn (2002)), Berg & Pinger (2013) found mental health effects of the food supply during the ancestors' slow growth period in Germany, using exposure to the German famine for identification.

demic, at least 10% of the Swedish population had been infected.[6] The flu killed between 35000 (Åhman, 1990) and 38500 Swedes (Ansart et al., 2009) which corresponds to 0.61 - 0.67% of the Swedish population. It is thus responsible for the last pronounced mortality peak in Sweden until today (Sundin & Willner, 2007).

Second, the Spanish flu happened unexpectedly and its timing as well as its intensity was unforeseen by authorities and medical professionals of that time, see e.g. Barry (2005). In Sweden, the medical community started to raise concerns as late as August 1918, but these were largely ignored by the authorities who did not believe the Spanish flu to be a substantial threat (Åhman, 1990). See also Karlsson et al. (2014) and references therein for an excellent overview regarding the Swedish case.

Third, the pandemic ended after just a couple of months, which lends credibility to a birth cohort design. This can be seen in figure 1, where we plot the aggregate influenza morbidity per month. As in other countries, we see a distinct and relatively sudden spike in influenza morbidity in the last quarter of 1918, which exhibits an equally sudden drop to almost normal levels in the beginning of 1919.[7] We see two smaller waves of influenza infections, though. One shortly after the peak in 1918, and one in the beginning of 1920.

Fourth, it is widely believed that the Spanish flu was a socially neutral disease and infected people essentially at random. This is important insofar as a social gradient would imply that resulting estimates are confounded by the social origin of those who got infected. In this respect, note that while regular influenza strains mainly affect vulnerable populations (e.g. the very young, the elderly, and immunocompromised individuals), the Spanish flu affected primarily healthy adults. For this reason, contemporary scientists even argued whether the Spanish flu was a flu at all (Barry, 2005). Animal experiments suggest that the Spanish flu has had such unique consequences by triggering an overreaction of the immune response, thus implying that having a strong immune system might have been a liability.[8]

Nevertheless, the existence of a social gradient may still be a concern. Mamelund (2006) documents the existence of a social gradient in mortality for the Spanish flu in Oslo, Norway.[9] While contraction of the disease is arguably random,

[6] While the official records by Medicinalstyrelsen (*National Board of Health*) indicate that roughly 10% of the Swedish population had been infected, it also reports that this is likely a lower bound (Medicinalstyrelsen, 1920).

[7] Figure 12 presents influenza mortality with a similar spike.

[8] In light of this research, a social gradient in flu infection might not be our main concern, but the possibility that only poor and sick people survived. In this respect, note that only 0.6% of the Swedish population died, so that this concern does not seem quantitatively important.

[9] Using apartment size as a measure for individual wealth in two selected parts of Oslo, Mamelund reports that individuals who were able to rent apartments with two, three or four rooms had 34, 41 or 56% lower mortality rates than individuals in one-room apartments, respectively. While these estimates are not robust to the inclusion of region fixed effects, they hint to a social gradient in mortality.

a social gradient in mortality implies that the survivors might be positively selected, which renders the resulting estimates conservative. [10]

The Spanish flu is also particularly suited for testing the effects of a prenatal insult due to the age profile of the infected. As shown in figure 2, the Spanish flu predominantly affected individuals in the range of 20 to 30, i.e. individuals in the childbearing age (Taubenberger & Morens, 2006). While regular influenza strains predominantly affect the young and the old, these groups experienced only a slight elevation of their mortality rates compared to individuals in childbearing age.[11]

3.2 World War I and parental quality

The Spanish flu episode is an attractive historical event for the study of prenatal insults, but its validity relies on the assumption that the timing of the flu does not coincide with any other historical event which could potentially confound the effects. Since the flu episode occurred during the end of 1918 and lasted until the beginning of 1919, it is quite natural to ask if the effects of the Spanish flu might be confounded by the end of World War I in November 1918. Even though Sweden remained neutral during the war, the repercussions of the hostilities in Europe certainly affected the country. Most importantly, maritime warfare and trade blockades interfered with imports to Sweden, which led to a general scarcity of certain goods, and in combination with poor harvests in 1917 also to a food shortage in that year (Montgomery, 1955). Moreover, Sweden and Norway as non-participating countries were surrounded by opposing war participants, and it seems likely that concerns about Sweden's safety emerged in the population. Contemporary political events support this conjecture. For instance, in February 1914, when political tensions between the participating countries grew and war was widely anticipated, plans of the Swedish government to reduce the defense budget were an important factor contributing to the "Courtyard Crisis" (*Borggårdskrisen*), in which 32000 farmers gathered in Stockholm to protest against the government's plans, demanding higher defense spending instead.

It is likely that fear of war involvement and the economic hardship due to WWI has affected parents' fertility decisions. An indication for this is the evolution of the cohort size, which is depicted in figure 3. The important aspect to note is the sharp increase approximately one year after the influenza peak, whose

[10] In fact, we find a negative albeit insignificant correlation between poverty rates in 1917 and pre-, peak as well as percentage-change in morbidity levels using county level data. The same exercise for mortality shows a positive but equally insignificant correlation.

[11] See figure 13. In this context, note that selective mortality could imply that our control group is positively selected. Note though that the number of people who died relative to the number of people who contracted the virus is quite low. For instance, during the peak of the influenza in November 1918, roughly 3000 individuals died, whereas about 120000 individuals were reported to have contracted the flu. The scope for problems due to selective mortality therefore seems limited.

timing coincides with the end of World War I (plus 9 months) as indicated by the shaded area.[12] Taking the evolution of the cohort size at face value, we suspect that the dramatic increase in fertility starting 9 months after the armistice reflects deferred fertility.[13] Most importantly, individuals conceived in late November onwards experienced the Spanish flu as a prenatal insult at an early stage during pregnancy, but are potentially born to parents who decided to wait because of the war.

If there was a social gradient in deferral behavior, then Brown & Thomas (2011)'s argument that the Spanish flu coincides with a change in the quality composition of parents also applies in Sweden. Unfortunately, we have no individual level data on the parents of the cohort born then, but historical population statistics contain the mother's marital status per month. The fraction of in-wedlock births increased for births conceived during the WWI period from about 83% to 88%, as shown in figure 4. Taking marital status as an indicator for parental quality, this suggests that, if anything, parental quality has *increased* rather than decreased during the war. Hence, individuals conceived during the war are potentially born to parents of better quality. The drop after the end of WWI can either be due to a normalization of fertility behavior, or it could reflect family disruption due to the flu, i.e. pregnancies of initially married couples where the husband died due to the flu. Either case implies that individuals conceived after the armistice were "worse off" compared to individuals conceived before.[14]

It seems safe to say that a comparison involving war and post-war times is difficult to make, but the fact that individuals conceived in November and December 1918 might have experienced the flu as a prenatal insult requires us to find a compromise. In our analysis, we will therefore focus on the period from 1916 up to the third quarter of 1919. We thus capture everyone conceived during WWI or the Spanish flu episode while excluding people conceived both after the peak and after the armistice.

4 Data

4.1 Measures and construction of influenza exposure

Data on influenza morbidity by month and county are available in historical records from Medicinalstyrelsen from 1911 until 1920. Medicinalstyrelsen was the central agency with overall responsibility for epidemic prevention at the

[12] The sharp drop in cohort size in May and June 1919 can be explained by family disruption and an increase in miscarriages due to the flu. For the former, see Åhman (1990). For the latter, note that in aggregate annual data on miscarriages, defined as the end of a pregnancy before the end of the second trimester, we find an increase for 1919. No increase in the number of stillbirths are found, though. See figure 7.

[13] See also Mamelund (2004) for an alternative interpretation.

[14] Note that this applies to the exposed group, but also to the comparison group.

time. To monitor the development of common epidemic diseases, all doctors in public service (*Tjänsteläkare*) were obliged to report cases of epidemic diseases to Medicinalstyrelsen. Our data is based on historical records of these reports aggregated up to county level and standardized by population size as of 1915.

The morbidity rates reveal that geographical variation in terms of the intensity and the timing of the Spanish flu is rather limited. Most variation in the intensity of the disease is driven by one outlier, Jämtland[15], which can be seen in figure 5. For the remaining part of Sweden, the variation is more limited. Most importantly, while exposure in some counties such as Kalmar and Norrbotten was *relatively* low, they were far from unaffected, which renders their use as control counties difficult. Figure 6 furthermore reveals that the Spanish flu universally gained momentum from July 1918 onwards and dropped to low levels in January 1919. While a few counties experienced an earlier onset and/or a longer duration of pandemic levels of the flu, it peaked in almost all counties in October and November 1918.

We use the unique timing of the Spanish flu to infer potential exposure. More precisely, we exploit the fact that people born in the first, second and third quarter of 1919 have likely been prenatally exposed to the flu in the third, second or first trimester, respectively. We therefore rely on the quarter of birth to identify potential exposure.

4.2 Individual level data

The individual level data are based on Statistics Sweden's Multigeneration register (SCB, 2011). This register includes all individuals born 1932 or later that have been registered as living in Sweden at some point since 1961. From the multigeneration register we use a 35% random sample of individuals born between 1932-1967. These individuals are referred to as *index persons* or *index generation* and constitute a representative sample of the Swedish population at the time the sample was collected. Information on the biological parents of the index persons is also available, given that the parent survived until 1947. This includes information of birth dates up to birth month as well as the birthplace. Our sample consists of individuals with both parents born between 1916 and September 1919.

We consider the impact of a potential parental prenatal insult on education and long-run earnings. Years of education are constructed from data in the 1970 and 1990 census and the education registers from 1999 and 2003. This source is also used to construct a college attendance dummy. Annual earnings data are taken from tax records available from 1968 to 2007. We measure income at

[15] Jämtland was particularly affected by the Spanish flu due to the age structure of its population. Excluding this county does not change our main results.

various ages as the mean of all earnings including benefits in the corresponding age range.

For the parent generation, years of education are similarly constructed from educational attainment data in the above mentioned censuses. The 1970 census is furthermore used to construct a high school completion dummy. Roughly 5% of the sample has incomplete information on parental education due to either death or emigration (of the parent) before data collection in 1970. While we cannot distinguish between these two causes of missingness, note that parental exposure in the second trimester appears to increase the likelihood of missing educational information on the parents. We therefore keep these observations in our main specification and flexibly control for parental education using indicators where missing values form their own category.[16] The income measure for the parents is constructed in the same manner as for the children but due to lower coverage of these data, we focus on the mean of all available income data, which captures the age ranges from 48 up to 64.

Descriptive statistics for the index generation by gender are shown in table 1, which reports the mean and in parentheses the standard deviation. We see that women in the index generation have slightly higher educational attainment but much lower long-run earnings than men. The variation in both education and in particular long-run earnings is higher for men. The parental characteristics are well balanced between men and women.

[16] Inclusion or exclusion of these individuals leaves all our estimates virtually unchanged (*not reported*), which we interpret as evidence that early parental death (or emigration) does not drive our results.

Selection issues

Tracing the effect of a health shock over three generations necessarily introduces selection concerns in each generation. For the generation of the grandparents, we already discussed in section 3.2 that these were most likely of better parental quality than individuals of surrounding cohorts. This positive selection is potentially exacerbated by a social gradient in mortality, killing poor and disadvantaged individuals and presumably their unborn babies more frequently. prenatal health shocks furthermore lead to intrauterine and early life mortality, implying that the surviving children are positively selected as well. This is exemplified by an increase in miscarriages and infant deaths, as shown in figures 7 and 8. These selection effects suggest that our estimates are conservative.

The data collection process furthermore introduces potential survival and fertility effects. On the one hand, observing a parent is necessarily conditional of this parent having survived until 1947 and child-bearing. If prenatal exposure to the Spanish flu implies that the exposed person does not have children (due to infertility, early death or failure on the marriage market), our sample of parents is selected and the analysis of parents' outcomes is biased.

The appropriate counterfactual for the analysis of the children would furthermore be unclear. If survival or child bearing conditional on flu exposure is correlated with other factors, then any estimated effect would be confounded. For instance, if conditional on prenatal flu exposure only descendents of high status families have children on their own whereas descendants of low status families have no children (or vice versa), then the estimated effects on the children would be confounded by the parental status. Put differently, despite flu exposure itself being random, observing the exposed family line at the point of data collection might be correlated with family characteristics.

If there is such an effect on family line survival, we would expect prenatally exposed parents to be undersampled in our data (since they either died earlier than 1947 or did have fewer children). Historical population records enable us to compare the historical cohort size with the cohort size implied by our sample, which allows us to compute the share of births that is included in our sample. This is shown in figure 9. As can be seen, we observe a general upward trend since parents are included in our sample only if they survived until 1947. The figure reveals a drop in late 1918 and early 1919. The most pronounced drop appears in January 1919, where a quadratic cohort trend would predict roughly 2% more births.[17] This drop does indeed point to a differential survival or fertility effect of the Spanish flu, although the magnitude appears small. Assuming that the children of these missing parents would have had the lowest educational outcomes implies that our estimates are furthermore conservative. In a robustness test, we bound the main effect by imputing extreme values for these missing children.

[17] A quadratic specification fits the data very well. See figure 15.

5 Empirical strategy

We estimate the effect of potential parental in utero exposure to the Spanish flu by comparing individuals with a prenatally insulted parent to individuals born in the same year whose parents have not been prenatally insulted. As discussed in section 4.1, we infer potential exposure by exploiting the timing of the Spanish flu. If a parent was born in the first, second or third quarter of 1919, it is very likely that that parent was exposed in the third, second, or first trimester, respectively. The sample is restricted so that all parents are born between January 1916 and October 1919. Cohort membership of the index generation by potential exposure status the parents are shown in figure 10. The regression equation looks as follows:

$$y_{it} = \alpha + \beta_1 \mathrm{q}^1_{1919} + \beta_2 \mathrm{q}^2_{1919} + \beta_3 \mathrm{q}^3_{1919} + \lambda_t + \eta \mathbf{x}_i^{'} + \epsilon_i \tag{1}$$

where y_{it} is the outcome of individual i born in year t, $q^j_{1919}, j \in \{1,2,3\}$, is an indicator which is 1 if the parent was born in quarter j of 1919. λ_t are birth year fixed effects. $\mathbf{x}_i^{'}$ is a vector of potential control variables, including a quadratic time trend in parental time of birth.

For the parent generation, we follow the literature and use a deviation from cohort trend design. The main equation used in the analysis is given by:

$$y_{it} = \alpha + \beta_1 \mathrm{q}^1_{1919} + \beta_2 \mathrm{q}^2_{1919} + \beta_3 \mathrm{q}^3_{1919} + \gamma_1 time + \gamma_2 time^2 + \epsilon_i \tag{2}$$

where all variables are defined as above. All estimates are obtained using ordinary least squares with robust standard errors clustered on the family level. For binary dependent variables, the linear probability model is reported.

6 Results

In the following, we present our results, starting with an analysis of the index generation. Along with our baseline model, we present specifications which control for parental outcomes to shed light on their potential role as mediators. To supplement these results, we then report our analysis for the parent generation.

6.1 Index generation

In tables 3 and 4, we present our estimation results for years of schooling and college attendance, respectively. The left panel displays the effect of maternal

exposure to the flu, and the right panel shows the effect of paternal exposure.[18] The first two columns in each panel shows results for women (i.e. daughters), and the last two columns in each panel for men (i.e. sons). Standard errors robust to clustering on the family level are reported in parentheses.

We estimate two different models. Model (1) refers to our baseline specification outlined in equation 1 without additional control variables, i.e. reduced form effects of potential influenza exposure. To shed light on potential mechanisms, note that we would expect the inclusion of an (endogenous) regression control associated with a given mechanism to affect the estimates for our exposure measures. We therefore add control variables in model (2) that reflect indirect mechanisms: first, a compromised health status of the exposed parent could lead to lower educational attainment and lower earnings, which could mechanically translate into lower educational attainment of the child. Second, a prenatal health shock is likely to decrease an individual's value on the marriage market and could thus affect the quality of the marriage partner, i.e. the quality of the second parent. In model (2), we therefore add a set of educational attainment dummies and long-run earnings for both parents. We will henceforth refer to these controls collectively as parental socioeconomic status (SES) indices.

Educational attainment: In table 3, we report the main results for years or schooling. All coefficients are scaled by 12 to indicate months. We find that the daughters of women and the sons of men who were born in the first two quarters of 1919, and who were thus potentially subject to the Spanish flu in the second and third trimester, display substantially lower educational attainment. For women, a potential maternal prenatal insult implies 3 to 4 months less schooling, and for men, a potential paternal prenatal insult implies decreased educational attainment of about 4 to 7 fewer months of schooling.

Table 4 shows similar patterns for college attendance. For women, a potential maternal prenatal insult in the second or third trimester decreases the probability of attending college by about 4 to 5 percentage points (baseline: 34%). Men whose fathers were born in the second quarter of 1919 have a 7 to 11 percentage points decreased probability of attending college (baseline: 31%).

Earnings: In figure 11, we plot the main results for a measure of earnings including benefits at various ages. The upper, middle and lower panel displays coefficient for the first, second and third quarter of 1919, respectively. Error bars indicate standard errors, and p-values are color coded. Each bar reflects the coefficient of the baseline model for the corresponding outcome indicated on the left. We find significant earnings effects of a maternal prenatal insult in the second and third trimester on women, but no effect of exposure in the first. Having said that, all but one coefficient are negative, and the effect appears to grow as earnings are measured at later ages. Regarding men, we find a significant negative effect of a paternal prenatal insult in the third trimester when income is measured during the age 51 to 60. Most other coefficients are

[18] Note that only very few individuals have parents who have both been exposed.

negative, although insignificant.

Discussion: We consistently find effects of a maternal insult during the last two trimesters on women, and of a paternal insult during either of the last two trimesters on men. Controlling for parental SES tends to attenuate the effect by about one standard error of the baseline coefficient, which suggests that at least part of the effect may be driven by indirect mechanisms. However, it is interesting to note that there appears to be no effect of a maternal prenatal insult on sons or of a paternal prenatal insult on daughters. That is, the effects seems to be transmitted along sex-specific lines, which is difficult to reconcile with indirect effects alone.

In the following, we analyze the effect of this prenatal insult on the parents of our index generation. Apart from enabling us to compare the Swedish case to the existing literature, this exercise helps us to understand how the effect is transmitted through generations.

6.2 Parent generation

In table 6, we present estimation results for the parents of the index generation. As discussed in section 3.1, we restrict our sample to cohorts born between 1916 and September 1919 to ensure that all parents have been conceived during World War I. In the first four columns, we report results for women (mothers of the previously analyzed index generation), and the last four columns report results for men (fathers of the index generation). We report results for education as measured by years of schooling (scaled by 12 to reflect months), the probability of high school graduation, (log) lifetime earnings as well as the educational attainment of the spouse to reflect potential marriage market effects.

For women, we find neither effects on educational attainment nor on earnings. However, the spouses' educational attainment is lowered by 3 to 5 months if the person was born in any of the first three quarters of 1919, suggesting that prenatal exposure to the Spanish flu has had an effect on the marriage market success. For men, we find large negative effects of prenatal flu exposure on educational attainment, as well as the educational attainment of the spouse. Men born in any of the first three quarters of 1919 deviate by about 4 to 7 months from to the common cohort trend in educational attainment. Similarly, the corresponding spouses have about 3 to 4 months less schooling.

Discussion: These results indicate that potentially prenatally exposed men suffered large decreases in their educational attainment, whereas women did not experience any negative effect on educational outcomes but decreased success on the marriage market. It is worthwhile pointing out that women's average educational attainment in this time period (8 years) is close to the minimum that we are able to observe (7 years)[19]. Hence, it is likely that our estimates for

[19]This is due to the construction of the educational attainment information in the census.

educational outcomes are biased towards zero by the limited nature of our dependent variable, which would explain that we find effects on marriage market outcomes but not on education. The distribution of signs supports this conjecture as well. If there was indeed no effect of exposure on women, we would expect 50% of the signs to be negative, and 50% of the signs to be positive. However, all but one of the coefficients for women are negative. We take this as suggestive evidence that our estimates on educational attainment for women are probably biased towards zero, and that both men and women have been affected by the Spanish flu.[20]

6.3 Effect sizes and potential mechanisms

Using potential exposure instead of actual infection implies that our estimates capture intent-to-treat effects, i.e. they exploit the change in the overall mean of those potentially exposed instead of the change in the mean of those who actually contracted the disease. Only a fraction of individuals that we classify as "exposed" has actually been infected with the disease, and this fraction including their outcomes cannot be identified. From a policy perspective, it is interesting to convert these intent-to-treat effects to effects for those who actually contracted the virus. Assuming that those who are erroneously classified as exposed do not show any departure from the cohort (i.e. those who were correctly classified as unexposed), we can approximate the average treatment effect on the treated by scaling the estimates with the infection rates among the subpopulation of interest. See, for instance, Heckman et al. (1994) and Heckman et al. (1999) for a related discussion in the treatment effects literature.

The relevant scale factor would be the morbidity rate of pregnant women. Unfortunately, our morbidity data only refers to the entire Swedish population, but statistics provided in Medicinalstyrelsen (1920) indicate that the infection rate among women aged 20-30 was as high as 25%. Even though these numbers are associated with a great deal of uncertainty, we assume this to be the infection rate among pregnant women and obtain the effect on the infected (treatment effect on the treated) by multiplying each estimate with four (1/0.25=4). Focusing on educational attainment, this implies an effect of 12 to 16 months less schooling for daughters of prenatally insulted women, and 16 to 28 months less schooling for sons of prenatally insulted men. For the parents themselves, this implies 3 to 6 months less schooling for women (though as we argued in section 6.2, this is likely a lower bound) and 16 to 28 months less schooling for men.

Three features of these results are puzzling: first, the effects have an almost inconceivably large magnitude, in particular when we take into account that selection effects probably render them conservative. Second, there is no fading

[20] Restricting the sample to women with daughters and men with sons (not reported) does not change the main results.

out of the effect over generations. The estimated effects for prenatally insulted men and their sons are of similar magnitude, and the effects for daughters of prenatally insulted women are larger than the estimated effects on the mothers themselves, which could be an artifact of the measurement for mothers, though. Third, the sex-specificity of the transmission and the missing mother-son and father-daughter link is noteworthy.

These features beg the question about potential mechanisms. Note that indirect mechanisms are likely since both parents' education levels are affected by potential prenatal flu exposure. Intergenerational correlations in education and earnings are typically less than one, though, and tend to be rather small in Sweden (Björklund & Salvanes, 2011; Niknami, 2010; Lindahl et al., 2013). If our results were purely driven by indirect mechanisms, we would expect the following: first, a much smaller effect to begin with. Second, a fading out of the effect, i.e. the effect on the second generation should be much lower than the effect on the first, and third, we would expect to observe an effect of a maternal (paternal) prenatal insult on sons (daughters). On the other hand, if the effects were purely driven by direct biological effects, we would not expect the inclusion of parental SES controls to affect the main estimates. We do however observe an attenuation of the effect by about one standard error of the main estimate.

It seems most likely that our results are driven by an interaction of indirect and direct effects, which might explain the large magnitude. For instance, prenatal flu exposure might decrease parents' budget sets and education levels, which limits the scope for and potentially the type of investments into their children (indirect effect). An endowment difference in children's health due to parental flu exposure (direct effect) might furthermore lead to reinforcing parental investment responses (interaction), thus exacerbating the effects.[21] Data constraints do not allow us to provide more explicit evidence along these lines, though.

7 Robustness checks

7.1 Seasonality

We have conducted a series of robustness checks to test the sensitivity of our results. First, given that the Spanish flu occurred in a seasonally distinct time, there is some concern that our estimates might capture seasonal fluctuations. To investigate this possibility, we consider a placebo exposure by shifting exposure one and two year backwards as well as forwards in time. The results are reported in tables 7 to 8 for years of education and college attendance for the index

[21] For instance, Hsin (2012) finds that low educated mothers reinforce initial endowment differences, whereas highly educated mothers compensate for initial endowment differences.

generation, and in table 9 for the parents. None of these tables resemble our main estimates or point to seasonality as a potential driver of our results.

7.2 Invisible Sample Selection

As discussed in section 4, it is necessary to evaluate how important selective family survival until data collection is for our main results. To this end, we compare the cohort size implied by our sample to historical birth records and use the negative deviations from a quadratic time trend fitted to the share included to infer the number of parents that are missing. The main dip in the share included in the relevant time perod occurs in the last quarter of 1918 and the first quarter of 1919, which we consider to be potentially due to exposure to the Spanish flu. In total, 436 parents are missing, out of which 277 are missing in 1919. This dip can potentially confound our results if some family types are systematically missing, thus rendering the exposed and comparison group fundamentally different. This would be true in our context if, for instance, the missing 1918 parents consist of the lower tail of the quality distribution of the parents born in the last quarter of 1918, whereas the missing parents born in the first quarter of 1919 consist of the upper tail of the corresponding distribution. Such a selection pattern, however plausible or not, would imply that we compare the children of different distributions.

To investigate this possibility, we impute these missing parents. We assume that each would have had two children and allocate the highest possible educational outcome (20 years, equivalent to a PhD) to the children of the parents missing in the first quarter of 1919 and the lowest possible educational outcome (7 years, or compulsory schooling) to the children of the parents missing in the last quarter of 1918. While this is arguably a very extreme scenario, it serves to establish a conservative upper bound.

The results are shown in table 10. We see that being born in the first or third quarter of 1919 is now associated with positive outcomes, which reflects that we added observations with extreme levels of education in the first quarter of 1919 and observations with extremely low education levels in the last quarter of 1918. Our results for the effect of a maternal prenatal insult on women in the second and third trimester break down. Our main results for negative effects of a paternal prenatal insult on men are only slightly attenuated, tough. We now find a decrease of men's education levels by 3 to 6 months of education, implying that the proposed selection pattern can explain at most 1 to 2 months of the main effect in the second trimester. This leads us to conclude that while extreme forms of selective family line survival may explain our findings for exposure in the third trimester, the large effects of a paternal prenatal insult on men in the second trimester persist, implying that selection alone cannot explain our results.

8 Discussion and conclusion

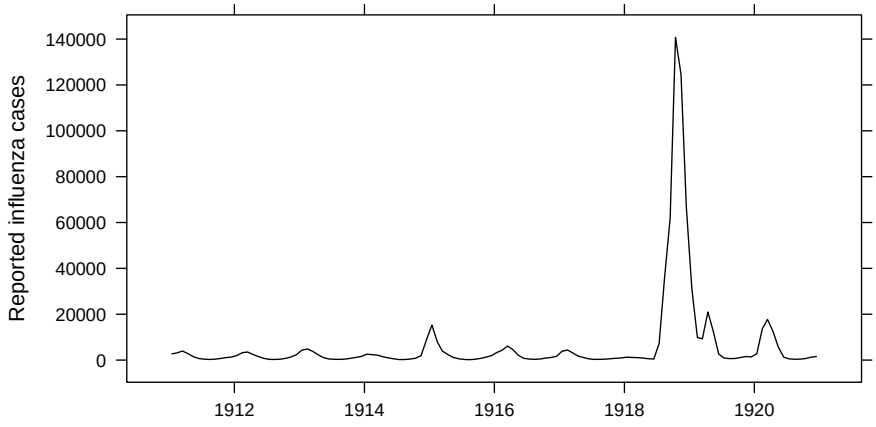
We use the 1918-19 influenza pandemic in Sweden as a natural experiment to estimate the effects of a prenatal health shock on the children of those who experienced the prenatal insult. Using birth information available in Swedish register data, we are able to infer potential prenatal exposure to the Spanish Flu, enabling us to trace the effect over two generations. Our results indicate that there are strong multigenerational effects of the Spanish Flu in Sweden. Potential maternal in utero exposure in the second trimester leads to decreased educational attainment for women (2-4 months). For paternal in utero exposure in the second trimester, we find similar albeit larger effects on men (4-7 months). These intent-to-treat estimates indicate potentially large effects on the treated.

Could confounding factors drive the results? We have already discussed in section 4 that it is unlikely that our estimates capture a social gradient in morbidity, and concerns about selective mortality render our estimates conservative. We have furthermore shown that selection based on fertility alone cannot explain our results either. A possibility is that our results are driven by other historical events affecting either the cohort exposed to the Spanish flu or their offspring. Our survey of the historical literature did not yield any clear alternative explanation, though, and as can be seen in figure 10, the birth-years of the children of the prenatally insulted parents are spread out fairly evenly over time, which makes alternative historical events affecting the offspring of the prenatally exposed less likely.

It is interesting to note that the gender pattern is strikingly robust. Prenatal exposure of mothers matters for daughters, while prenatal exposure of fathers matters for sons. While it is beyond the scope of this paper to speculate on the reasons for the missing mother-son and father-daughter links, note that this pattern is consistent with gender-specific transgenerational epigenetic inheritance (cf. Pembrey et al. (2006) and Berg & Pinger (2013)).

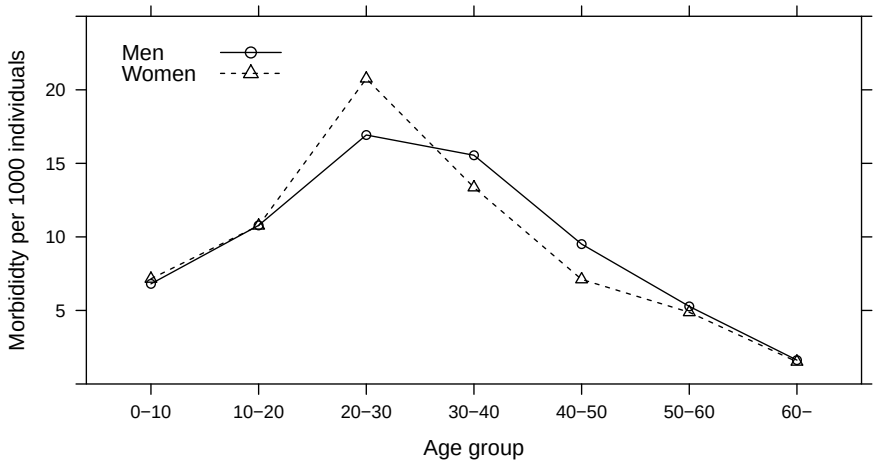
We therefore conclude that the Spanish flu had gender-specific multigenerational effects. Maternal (paternal) in utero exposure to the flu decreased womens' (mens') educational attainment, and these effects appear not to be driven by seasonal effects nor fertility selection.

Figure 1: Influenza morbidity by month in Sweden, 1911-1920



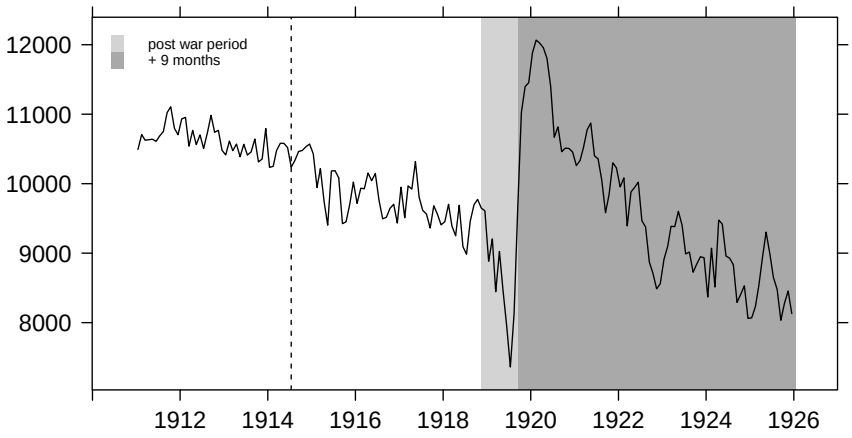
Source: Medicinalstyrelsen 1911-1920

Figure 2: Age and gender profile of influenza morbidity in Sweden.



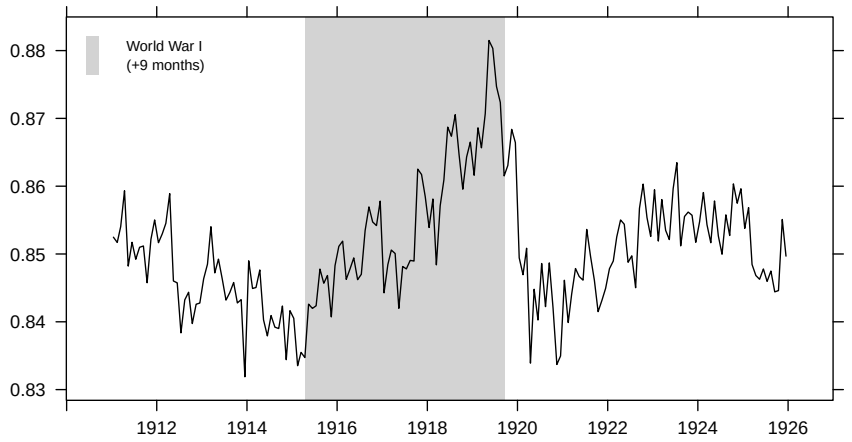
Note: Coverage of the patient's age in the morbidity reports is limited to a subset of the data, and the fraction of the population this data covers is not known. The *level* of the above curve corresponds to the case of complete coverage, and needs to be interpreted as a lower bound. *Source:* Medicinalstyrelsen 1920.

Figure 3: Cohort size of newborns in Sweden, 1911-1925



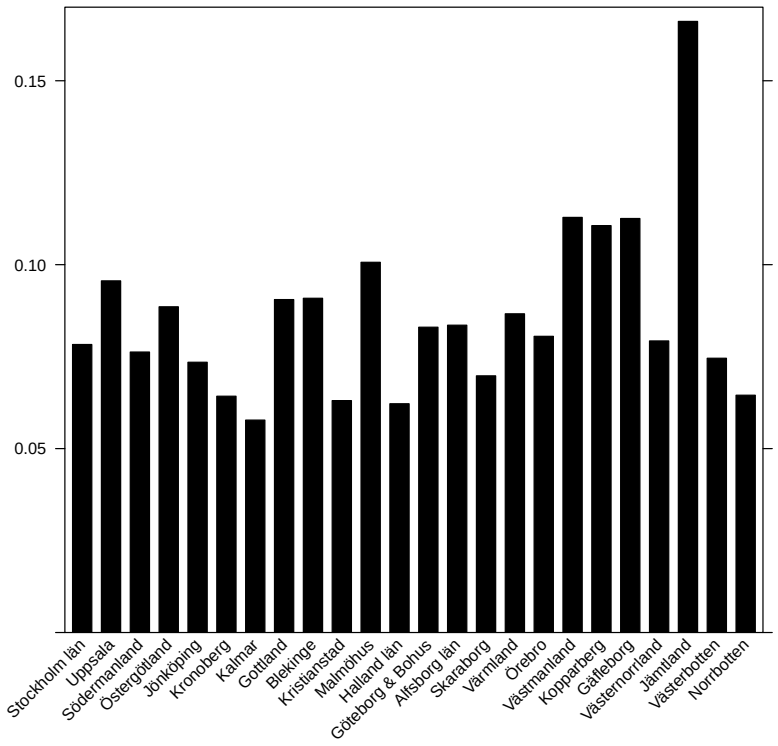
Note: Adjusted for (pre-1918) seasonal patterns. The dashed line indicates the beginning of World War I. *Source:* Statistics Sweden

Figure 4: Births in wedlock (in %) 1911-1925



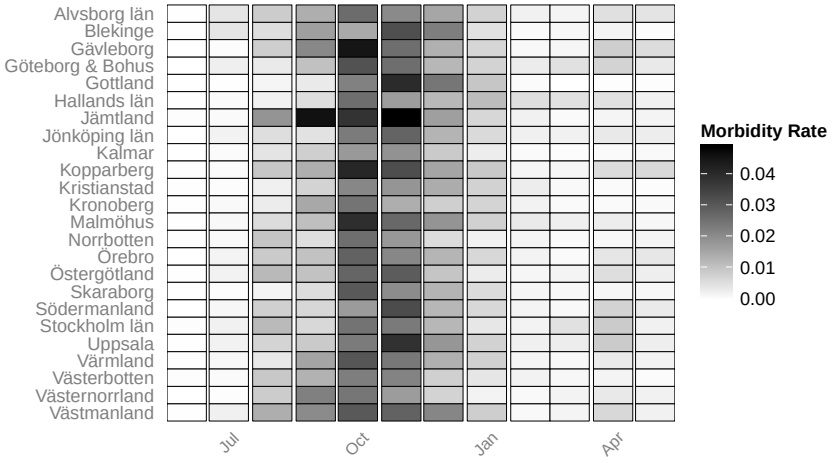
Source: Medicinalstyrelsen 1920. Adjusted for seasonal effects.

Figure 5: Morbidity rates by county, Jul 1918 to Feb 1919



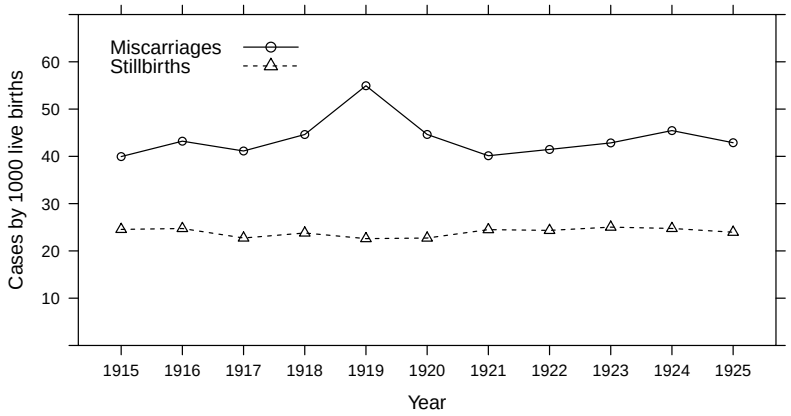
Source: Medicinalstyrelsen 1920.

Figure 6: Timing of the Spanish flu by county



Source: Medicinalstyrelsen 1911-1920

Figure 7: Stillbirths and miscarriages in Sweden, 1915-1925



Source: Medicinalstyrelsen and SCB Befolkningsrörelsen 1915-1925.

Table 1: Descriptive sample statistics

	Women		Men	
	unexposed	exposed	unexposed	exposed
Years of schooling	11.55	11.58	11.39	11.41
	(2.67)	(2.70)	(2.83)	(2.87)
College	0.33	0.34	0.29	0.30
	(0.47)	(0.48)	(0.45)	(0.46)
Earnings	175394	175720	260443	260296
	(78913)	(83079)	(142580)	(138989)
Birthyear	1949	1949	1949	1949
	(6)	(5)	(6)	(5)
Years of schooling mother	8.26	8.31	8.27	8.31
	(2.13)	(2.20)	(2.14)	(2.20)
Years of schooling father	8.97	9.08	8.99	9.10
	(2.81)	(2.90)	(2.83)	(2.92)
High school mother	0.20	0.21	0.20	0.20
	(0.4)	(0.4)	(0.4)	(0.4)
High school father	0.33	0.34	0.33	0.34
	(0.47)	(0.47)	(0.47)	(0.48)
Age at birth mother	29.07	29.24	29.16	29.18
	(5.50)	(5.42)	(5.49)	(5.40)
Age at birth father	30.41	30.20	30.52	30.15
	(5.51)	(5.38)	(5.51)	(5.37)
n	74036	17358	76689	18001

The index generation consists of all individuals in the sample with parents born between 1916 and October 1919. The figures above represent the mean and standard deviation (in parentheses).

	Generation	How does selection occur?
F0	Grandparents	Selective fertility during WWI? Selective survival of health shock?
F1	Parents	Selective mortality • Miscarriages • Stillbirths • Infant deaths • Survival to 1947
F2	Children	Fertility of F1

Table 2: Selection in three generations

Figure 8: Infant Deaths

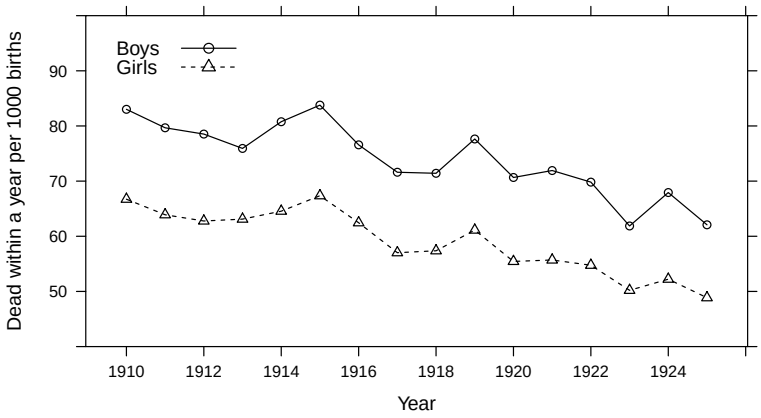
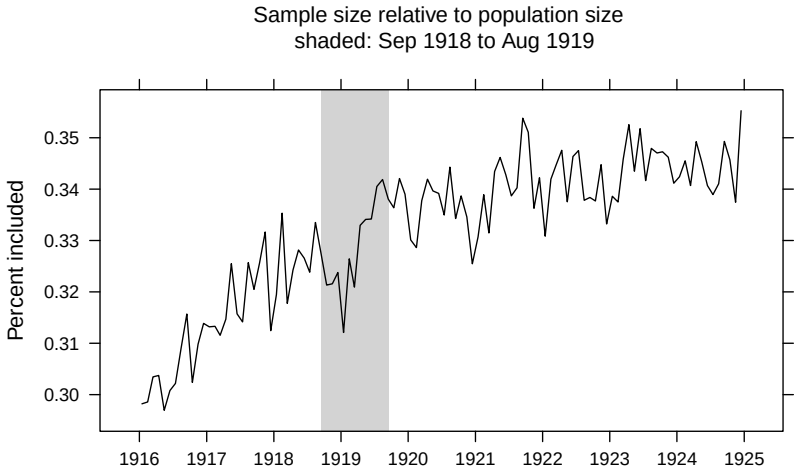
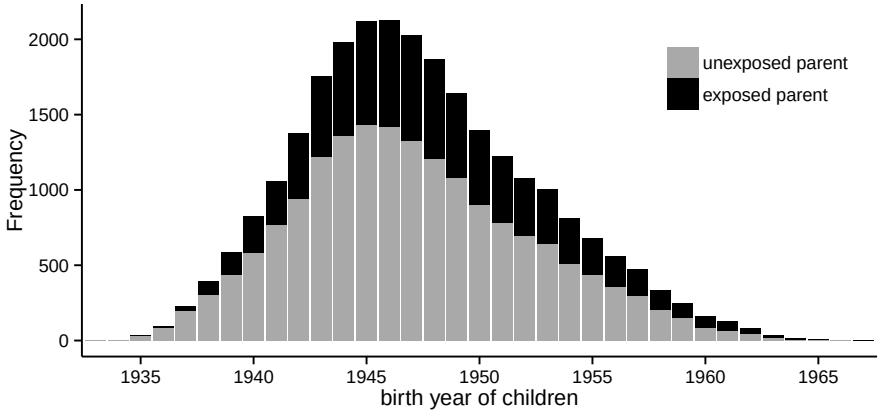


Figure 9: Evolution of share of births included.



This graph depicts the cohort size implied by our sample as a fraction of the historical cohort size. The upward trend arises due to a survival cutoff in 1947. *Source:* Medicinalstyrelsen 1920 and SCB.

Figure 10: Distribution of birth-years of index generation



Source: Multigeneration register, Statistics Sweden

Table 3: Regression results for Years of Education

	Maternal exposure:				Paternal exposure:			
	Women		Men		Women		Men	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
janfebmar19	**−3.45***	**−2.74***	−0.50	−0.08	−1.54	0.53	**−4.22***	−3.34
	(1.49)	(1.37)	(1.61)	(1.41)	(1.83)	(1.67)	(2.03)	(1.81)
aprmayjun19	**−3.69***	−2.25	0.00	1.51	−3.28	−2.10	**−7.24****	**−4.36***
	(1.84)	(1.65)	(1.89)	(1.69)	(2.17)	(1.97)	(2.32)	(2.08)
julaugsep19	−0.54	0.12	2.11	2.91	−0.68	2.66	−3.19	−1.44
	(2.26)	(2.05)	(2.36)	(2.11)	(2.64)	(2.43)	(2.90)	(2.56)
All quarters	**−3.90****	**−2.92***	−0.63	0.15	−2.09	−0.28	**−5.24****	**−3.65***
	(1.43)	(1.30)	(1.52)	(1.34)	(1.70)	(1.55)	(1.87)	(1.66)
MeanDep	138.66	138.66	137.66	137.66	138.66	138.66	137.66	137.66
Min	84	84	84	84	84	84	84	84
Max	240	240	240	240	240	240	240	240
Adj. R^2	0.07	0.21	0.04	0.22	0.07	0.21	0.04	0.22
Num. obs.	12732	12732	13257	13257	12732	12732	13257	13257
Parental SES:	no	yes	no	yes	no	yes	no	yes

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

(1) refers to baseline model, (2) controls for socioeconomic status indicators of both parents. Robust standard errors clustered on the family level are reported in parentheses. Control variables include birthyear fixed effects and a quadratic time trend for parental time of birth.

Table 4: Regression results for college

	Maternal exposure:				Paternal exposure:			
	Women		Men		Women		Men	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
janfebmar19	**−0.05***	**−0.04***	0.00	0.01	−0.03	0.00	−0.05	−0.04
	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)	(0.03)
aprmayjun19	**−0.05***	−0.03	0.01	0.02	−0.04	−0.02	**−0.11*****	**−0.07***
	(0.03)	(0.02)	(0.03)	(0.02)	(0.03)	(0.03)	(0.03)	(0.03)
julaugsep19	−0.01	0.00	0.03	0.04	−0.01	0.04	−0.06	−0.04
	(0.03)	(0.03)	(0.03)	(0.03)	(0.04)	(0.04)	(0.04)	(0.04)
All quarters	**−0.06****	**−0.05***	0.00	0.01	−0.04	−0.01	**−0.07****	**−0.05***
	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)	(0.02)	(0.03)	(0.02)
MeanDep	0.34	0.34	0.31	0.31	0.34	0.34	0.31	0.31
Min	0	0	0	0	0	0	0	0
Max	1	1	1	1	1	1	1	1
Adj. R^2	0.03	0.15	0.02	0.15	0.03	0.15	0.02	0.15
Num. obs.	12318	12318	12775	12775	12318	12318	12775	12775
Parental SES:	no	yes	no	yes	no	yes	no	yes

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

(1) refers to baseline model, (2) controls for socioeconomic status indicators of both parents. Robust standard errors clustered on the family level are reported in parentheses. Control variables include birthyear fixed effects and a quadratic time trend for parental time of birth.

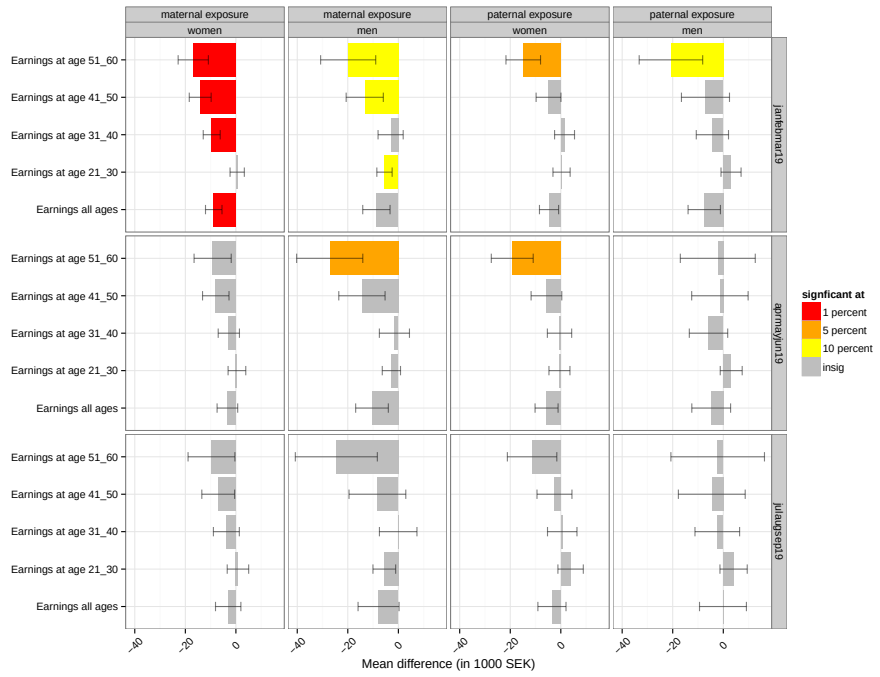


Figure 11: Coefficients and standard errors for earnings over the life cycle. Earnings are measured as mean earnings over the corresponding period. Error bars indicate one standard error, with p-values color coded. Controls include birthyear fixed effects for the index generation, and quadratic time trends for parental time of births.

Table 5: Mean income over the lifecycle (in SEK)

	women	men
Earnings all ages	160580	240089
Earnings at age 21_30	99872	171387
Earnings at age 31_40	133139	229595
Earnings at age 41_50	191422	278535
Earnings at age 51_60	233783	318401

Table 6: Regression results for the parent generation

	Women				Men			
	yoe	hs	earn	yoe spouse	yoe	hs	earn	yoe spouse
janfebmar19	−0.99	−0.01	−0.03	−2.80*	−4.35**	−0.05*	0.00	−3.36**
	(0.98)	(0.02)	(0.05)	(1.32)	(1.64)	(0.02)	(0.04)	(1.23)
aprmayjun19	−1.65	−0.02	−0.02	−4.56**	−7.24***	−0.09***	−0.06	−3.10*
	(1.16)	(0.02)	(0.05)	(1.56)	(1.90)	(0.03)	(0.06)	(1.46)
julaugsep19	−1.19	−0.02	0.01	−4.01*	−7.31**	−0.08*	−0.05	−3.89*
	(1.45)	(0.02)	(0.07)	(1.93)	(2.27)	(0.03)	(0.07)	(1.75)
All quarters	−1.21	−0.01	−0.03	−3.30**	−5.37***	−0.07**	−0.02	−3.28**
	(0.93)	(0.01)	(0.04)	(1.25)	(1.51)	(0.02)	(0.04)	(1.15)
MeanDep	99.27	0.21	11.13	109.17	109.06	0.35	12.02	99.16
Min	84	0	0	84	84	0	0	84
Max	240	1	13.5	240	240	1	15.54	240
Adj. R^2	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Num. obs.	18238	18238	18238	18238	18367	18367	18367	18367

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Robust standard errors reported in parentheses. Control variables include quadratic time trend.

Table 7: Placebo Regressions for Years of Education

	Maternal exposure:				Paternal exposure:			
	Women		Men		Women		Men	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
janfebmar17	−1.20	−0.88	−2.15	−1.41	−1.17	−0.87	−1.71	−1.21
	(1.30)	(1.19)	(1.39)	(1.23)	(1.16)	(1.07)	(1.21)	(1.09)
aprmayjun17	−0.61	0.09	−0.25	0.96	−1.82	−0.63	−0.81	−0.21
	(1.25)	(1.16)	(1.40)	(1.27)	(1.20)	(1.10)	(1.27)	(1.13)
julaugsep17	−0.68	−0.55	−1.81	−0.58	−1.07	−0.14	−0.26	−0.53
	(1.35)	(1.24)	(1.41)	(1.24)	(1.20)	(1.12)	(1.36)	(1.20)
janfebmar18	0.77	−0.02	−2.17	−2.07	0.95	0.74	0.55	−0.06
	(1.22)	(1.11)	(1.30)	(1.16)	(1.32)	(1.18)	(1.36)	(1.23)
aprmayjun18	0.43	−0.72	2.30	1.01	0.84	−0.37	1.74	1.01
	(1.20)	(1.10)	(1.30)	(1.16)	(1.31)	(1.19)	(1.39)	(1.18)
julaugsep18	−0.55	−0.55	1.38	−0.09	2.12	1.26	0.13	−0.61
	(1.15)	(1.06)	(1.30)	(1.14)	(1.24)	(1.14)	(1.43)	(1.27)
janfebmar20	−0.47	−0.26	0.78	0.78	−1.00	−1.94	0.12	0.59
	(1.07)	(0.99)	(1.13)	(1.01)	(1.42)	(1.28)	(1.54)	(1.38)
aprmayjun20	0.54	1.23	1.47	0.94	−0.86	0.19	−0.32	1.15
	(1.23)	(1.12)	(1.30)	(1.15)	(1.57)	(1.45)	(1.74)	(1.52)
julaugsep20	0.02	0.09	2.01	2.13	0.95	1.03	−2.00	−0.60
	(1.43)	(1.30)	(1.53)	(1.35)	(1.84)	(1.71)	(1.98)	(1.74)
janfebmar21	1.55	1.06	0.42	0.53	1.72	1.23	−0.46	0.21
	(0.91)	(0.85)	(0.96)	(0.85)	(1.32)	(1.22)	(1.42)	(1.25)
aprmayjun21	−1.11	−1.46	0.31	−0.23	0.33	0.34	−1.98	−2.35
	(0.99)	(0.91)	(1.05)	(0.93)	(1.35)	(1.24)	(1.47)	(1.30)
julaugsep21	−0.65	−0.92	−1.18	−1.17	3.12	2.83	−2.27	−1.62
	(1.12)	(1.03)	(1.19)	(1.06)	(1.60)	(1.50)	(1.68)	(1.47)
Parental SES:	no	yes	no	yes	no	yes	no	yes

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

(1) refers to baseline model, (2) controls for socioeconomic status indicators of both parents. Robust standard errors clustered on the family level are reported in parentheses. Control variables include birthyear fixed effects and a quadratic time trend for parental time of birth.

Table 8: Placebo Regressions for College

	Maternal exposure:				Paternal exposure:			
	Women		Men		Women		Men	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
janfebmar17	−0.01	−0.01	−0.03	−0.02	−0.02	−0.01	**−0.03***	−0.03
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.01)
aprmayjun17	−0.01	0.00	0.01	0.03	−0.02	0.00	−0.01	0.00
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
julaugsep17	−0.01	−0.01	−0.02	−0.01	−0.02	−0.01	−0.01	−0.02
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
janfebmar18	0.00	−0.01	−0.03	−0.03	0.02	0.01	0.02	0.01
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
aprmayjun18	−0.01	−0.02	0.02	0.00	0.03	0.01	0.02	0.02
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
julaugsep18	−0.02	−0.02	0.02	0.00	0.02	0.00	0.01	0.01
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
janfebmar20	0.00	0.01	0.00	0.00	−0.04	**−0.05***	0.00	0.00
	(0.02)	(0.01)	(0.02)	(0.01)	(0.02)	(0.02)	(0.02)	(0.02)
aprmayjun20	0.02	**0.03***	0.01	0.00	−0.03	−0.02	−0.02	0.00
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
julaugsep20	0.01	0.01	0.00	0.01	0.00	0.00	−0.02	0.00
	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)	(0.02)
janfebmar21	0.02	0.02	0.00	0.00	0.02	0.01	−0.01	0.00
	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)	(0.02)	(0.02)	(0.02)
aprmayjun21	−0.01	−0.01	0.01	0.00	0.02	0.01	−0.02	−0.02
	(0.01)	(0.01)	(0.01)	(0.01)	(0.02)	(0.02)	(0.02)	(0.02)
julaugsep21	−0.02	−0.02	−0.02	−0.02	0.05	0.04	−0.02	−0.01
	(0.02)	(0.02)	(0.02)	(0.01)	(0.02)	(0.02)	(0.02)	(0.02)
Parental SES:	no	yes	no	yes	no	yes	no	yes

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

(1) refers to baseline model, (2) controls for socioeconomic status indicators of both parents. Robust standard errors clustered on the family level are reported in parentheses. Control variables include birthyear fixed effects and a quadratic time trend for parental time of birth.

Table 9: Placebo Regressions for the Parent Generation

	Women				Men			
	yoe	hs	earn	yoe spouse	yoe	hs	earn	yoe spouse
janfebmar17	−0.37	0.00	−0.06	−1.42	−0.66	−0.01	0.03	−0.60
	(0.81)	(0.01)	(0.05)	(1.10)	(0.98)	(0.01)	(0.03)	(0.72)
aprmayjun17	−0.94	−0.02	−0.01	−3.47**	−2.33*	−0.03	−0.01	−1.69*
	(0.80)	(0.01)	(0.03)	(1.06)	(1.00)	(0.01)	(0.03)	(0.73)
julaugsep17	−0.25	0.00	−0.04	−1.13	−1.11	0.00	−0.02	−1.17
	(0.86)	(0.01)	(0.04)	(1.14)	(1.03)	(0.01)	(0.03)	(0.78)
janfebmar18	0.84	0.01	0.06*	0.77	1.20	0.01	0.01	1.03
	(0.79)	(0.01)	(0.03)	(1.04)	(1.11)	(0.02)	(0.04)	(0.82)
aprmayjun18	1.44	0.02	0.05	2.58*	1.65	0.01	0.03	2.30**
	(0.80)	(0.01)	(0.03)	(1.06)	(1.15)	(0.02)	(0.03)	(0.88)
julaugsep18	1.35	0.02	0.03	0.69	3.28**	0.04**	−0.01	1.24
	(0.78)	(0.01)	(0.03)	(1.00)	(1.16)	(0.02)	(0.03)	(0.86)
janfebmar20	0.65	0.01	0.01	−1.04	−0.72	−0.01	−0.01	1.13
	(0.68)	(0.01)	(0.03)	(0.89)	(1.26)	(0.02)	(0.03)	(0.98)
aprmayjun20	0.24	0.00	0.01	0.35	−2.13	−0.02	−0.06	−1.79
	(0.79)	(0.01)	(0.03)	(1.05)	(1.40)	(0.02)	(0.04)	(1.05)
julaugsep20	0.27	0.01	0.00	0.75	−2.91	−0.03	−0.11*	−1.71
	(0.92)	(0.01)	(0.04)	(1.23)	(1.61)	(0.02)	(0.04)	(1.21)
janfebmar21	0.88	0.02	0.03	−0.17	−0.64	−0.02	−0.05	−0.67
	(0.60)	(0.01)	(0.03)	(0.78)	(1.14)	(0.02)	(0.03)	(0.87)
aprmayjun21	0.87	0.02	−0.02	1.29	1.57	0.02	−0.03	−0.62
	(0.66)	(0.01)	(0.03)	(0.85)	(1.22)	(0.02)	(0.03)	(0.92)
julaugsep21	0.11	0.00	0.02	−0.60	0.43	0.00	−0.11**	1.12
	(0.73)	(0.01)	(0.03)	(0.95)	(1.36)	(0.02)	(0.04)	(1.08)

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

Robust standard errors reported in parentheses. Control variables include quadratic time trend.

Table 10: Upper Bounds on the Regression Results

	Maternal exposure:				Paternal exposure:			
	Women		Men		Women		Men	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Years of Schooling								
janfebmar19	**7.84*****	**8.53*****	**11.71*****	**12.06*****	**6.56****	**8.72*****	**5.79****	**6.71*****
	(1.68)	(1.61)	(1.77)	(1.63)	(2.05)	(1.93)	(2.18)	(2.02)
aprmayjun19	−0.03	1.26	3.34	**4.81****	−1.08	0.30	**−5.75***	−2.95
	(1.86)	(1.68)	(1.90)	(1.72)	(2.19)	(2.01)	(2.32)	(2.08)
julaugsep19	4.25	**4.75***	**6.46****	**7.21*****	2.11	**5.65***	−1.34	0.29
	(2.30)	(2.09)	(2.38)	(2.15)	(2.67)	(2.48)	(2.89)	(2.56)
College								
janfebmar19	0.03	0.03	**0.09*****	**0.09*****	0.02	**0.05***	0.02	0.03
	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)	(0.02)
aprmayjun19	−0.03	−0.01	0.03	0.04	−0.03	0.00	**−0.10****	**−0.06***
	(0.03)	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)	(0.03)
julaugsep19	0.02	0.03	0.05	**0.07***	0.01	0.06	−0.04	−0.02
	(0.03)	(0.03)	(0.03)	(0.03)	(0.04)	(0.04)	(0.04)	(0.04)
Num. obs.	12465	12465	12938	12938	12382	12382	12848	12848
Parental SES:	no	yes	no	yes	no	yes	no	yes

$^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$

(1) refers to baseline model, (2) controls for socioeconomic status indicators of both parents. Robust standard errors clustered on the family level are reported in parentheses. Control variables include birthyear fixed effects and a quadratic time trend for parental time of birth.

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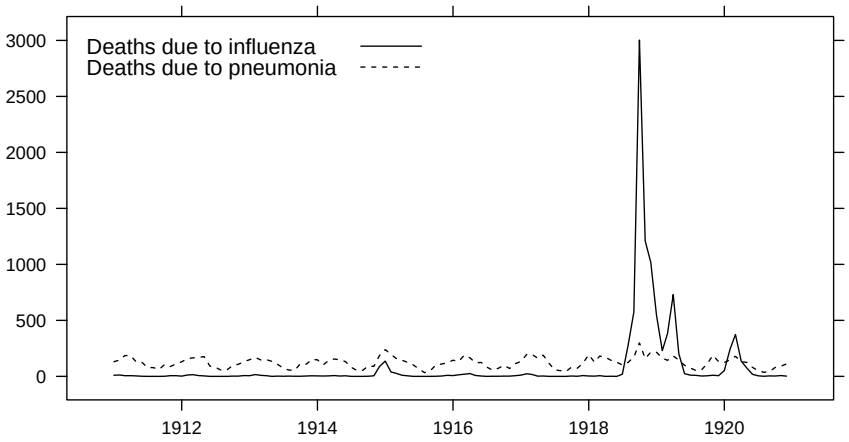
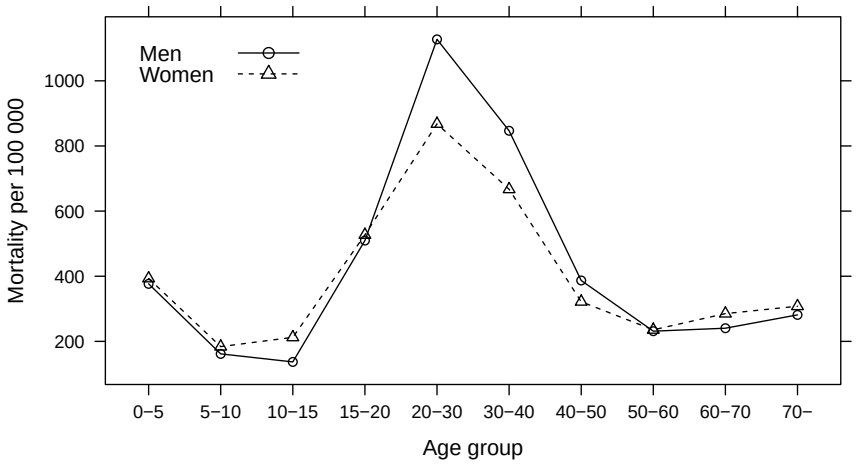
9 Appendix

Figure 12: A comparison of deaths due to influenza and pneumonia in Sweden.



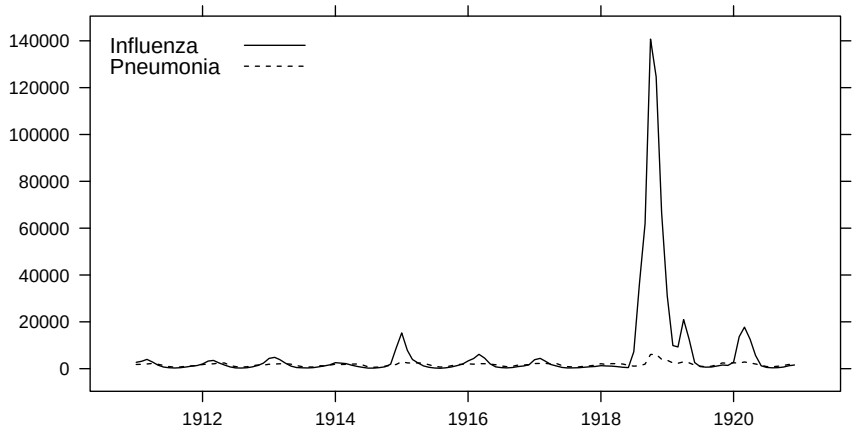
Source: Medicinalstyrelsen 1911-1920

Figure 13: Age and gender profile of influenza mortality in Sweden.



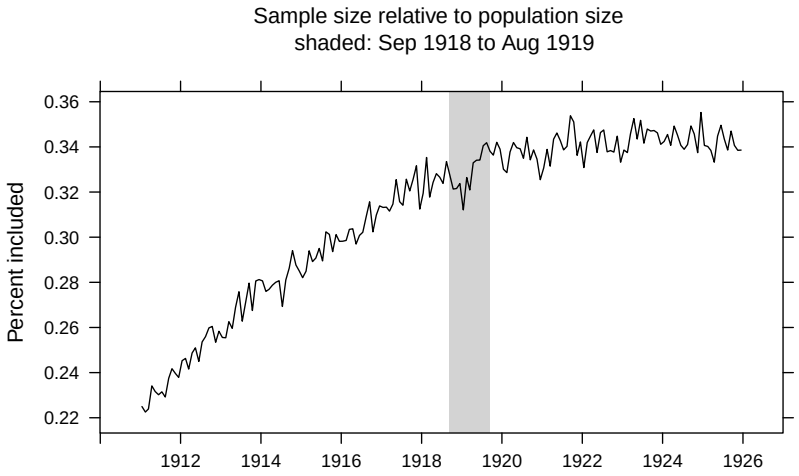
Source: Medicinalstyrelsen 1920

Figure 14: A comparison of influenza and pneumonia incidence in Sweden.



Source: Medicinalstyrelsen 1911-1920

Figure 15: Evolution of share of births included.



This graph depicts the cohort size implied by our sample as a fraction of the historical cohort size. The upward trend arises due to a survival cutoff in 1947. *Source:* Medicinalstyrelsen 1920 and SCB.