Multigenerational effects of the 1918-19 influenza pandemic in Sweden

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Abstract

We use the 1918-19 influenza pandemic in Sweden as a natural experiment to estimate multigenerational effects of in utero health shocks. Potential exposure is constructed using historical records of regional influenza incidence matched to birth information available in Swedish register data. Our estimates suggest that potential in utero exposure to the Spanish flu has persistent effects on subsequent generations, and that these effects are predominantly driven by direct biological mechanisms. Potential maternal in utero exposure to the Spanish flu lowers educational attainment by 2-2.5 months (1.5-1.8%) of schooling and decreases the probability of college attendance by about 10-12% for female offspring.

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1 Motivation

According to the fetal origins hypothesis, differences in the fetal environment are causally linked to differences in socioeconomic outcomes in later life. Economists and epidemiologists have tested this hypothesis using a wide range of natural experiments, exploiting plausibly exogenous shocks to fetal health, and the fetal origins hypothesis is now generally accepted. See Almond and Currie (2011) for a comprehensive review.

This paper extends the fetal origins literature and considers multigenerational effects, i.e. effects on the children of those who experienced a fetal insult. We follow the seminal work of Almond (2006) and use maternal exposure to the 1918 influenza virus, or Spanish flu, as an exogenous health shock to estimate the effect on socioeconomic outcomes of the children of the fetally insulted. A series of papers has previously used the pandemic as a natural experiment, and its detrimental effects on later life outcomes of those affected in the womb has been established in countries such as the US (Almond, 2006), Brazil (Nelson, 2010), Switzerland (Neelsen and Stratmann, 2012), and Taiwan (Liu and Lin, 2013). In Sweden, regional influenza morbidity rates on the county (*län*) level are known during that period, which enables us to use potential exposure to identify intent-to-treat effects. By linking a fetal insult to the offspring of those in the womb, this is the first paper in the economic literature to estimate the causal effect of a fetal health shock on subsequent generations.

Such multigenerational effects could be driven by direct or indirect mechanisms. Effects of the Spanish flu on socioeconomic outcomes of the fetally exposed are well established in the literature. The Spanish flu will thus affect subsequent generations via its effect on social and economic outcomes of the fetally insulted parents, which we refer to as indirect effects. Direct or biological effects are conceivable as well. This is because the primoridal germ cells, i.e. predecessors of the ovaries in women or the sperm cells in men, already develop at the fetal stage, which makes them susceptible to the same shocks the fetus experiences. Hence, fetal exposure to a health shock also exposes the genetic material that it will transmit to its own children, who thus need to be considered "exposed". Such a mechanism is documented at least in the literature on environmental toxins, which is reviewed in e.g. Altshuler et al. (2003) and Franklin and Mansuy (2010).

We find strong multigenerational effects of the Spanish flu in Sweden. Potential maternal exposure in the second trimester leads to decreased educational attainment for female offspring (2-3 months), and decreased long-run earnings for male offspring (6.8-7.4%). These intent-to-treat estimates suggest potentially large effects on the infected. Moreover, our estimates change only marginally when controlling for parental socioeconomic status, which we interpret as evidence for direct rather than indirect effects.

This paper is organized as follows: in section 2, we discuss previous literature on this topic and in section 2, 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.

2 Related Literature

Economists have studied the importance of the fetal environment using a wide range of natural experiments. For instance, Chen and Zhou (2007), Scholte et al. (2012), Neelsen and Stratmann (2011), Almond and Mazumder (2011), van Ewijk (2011) and Almond et al. (2011) use nutritional deprivation due to famines or religious rituals and find effects of these events on either early or later life outcomes.[1] Similar results are obtained when evidence for stress exposure is used, e.g. terror attacks (Camacho, 2008), war (Lee, 2011), violent civil conflict (Valente, 2011), the al-Aqsa Intifada (Mansour and Rees, 2011) and natural disasters (Simeonova (2009), Currie and Rossin-Slater (2012), among others). In utero exposure to pollution has similarly detrimental effects, see inter alia, Almond et al. (2009), Nilsson (2009) and Currie et al. (2013).

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), US census data are used to identify departures from trend for individuals who were in utero during the pandemic. Large reductions in educational attainment, wages, socioeconomic status indices and health measures are found. Almond and Mazumder (2005) use data for the US from the Survey

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

of Income and Program Participation to show negative health effects for those in utero during the pandemic, where a similar departure-from-cohort-trend approach is used. Furthermore, Mazumder et al. (2010) use the US National Health Interview Surveys and show that the in utero shock lead to a higher prevalence of cardiovascular disease.

However, Brown and Thomas (2011) show that Almond's results are potentially confounded due to conscription procedures for World War I, which induce a change in parental quality that coincides with the timing of the Spanish flu. Nevertheless, Almond's results have been replicated in a number of countries not participating in World War I. Nelson (2010) uses Brazilian survey data and finds reduced educational attainment, lower wages and lower literacy levels for the corresponding cohorts. Liu and Lin (2013) conducts a similar study on data from Taiwan. They report that the potentially exposed cohort is less educated, shorter as teenagers, and in poorer health, with effect sizes being comparable or larger to Almond's. Neelsen and Stratmann (2012) estimate the effects of the pandemic for Switzerland, which also did not participate in any of the World Wars. Negative effects are found for educational measures, the likelihood to never marry and a socioeconomic status index. The fetal origins hypothesis is largely supported, even though the estimated effect sizes are much smaller than the ones reported by Almond (2006). Table 2 gives an overview of magnitudes found in these studies with respect to educational attainment measures.

Other noteworthy studies are Kelly (2009) and Parman (2012). Kelly uses cross-sectional variation in the Asian flu of 1957 in the UK and finds that fetal 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.

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 and Gale, 2007). In this context, it is useful to know that maternal influenza infection during pregnancy has been linked to several neurological conditions later in life. See, for instance, Brown and Derkits (2010) and Canetta and Brown (2012) for schizophrenia, as well as Parboosing et al. (2013) and Machon et al. (1997) for affective disorders. Brown and Derkits (2010) and Canetta and Brown (2012) discuss that

Table 1: Overview of selected influenza studies

Paper	Cohorts	Country	Effect size education
Almond 2006	1912-1922	US	-1.5 months of schooling -4/-5% high school completion
Lin & Liu 2012	1916-1926	Taiwan	-0.72 months of schooling
Nelson 2010	1912-1922	Brazil	-0.552 months schooling -13% college graduation rate
Neelsen et al. 2012	1912-1922	Switzerland	-0.5% ppts vocational degree

The four most important influenza studies are reported with estimates for their main specification. Where applicable, the intent-to-treat effect on the full sample is reported. In the US, roughly 33% of all citizen were infected, in Taiwan 25% and in Brazil (Sao Paulo) 22,32%. In Switzerland, more than 50% contracted the Spanish flu. The main specification for all these studies rely on a deviation-from-year of birth-trend, where a linear or quadratic trend in birth year is fitted, together with a dummy indicating birth in 1919. All above papers contain specifications where quarter of birth and / or regional variation is used. These specifications usually confirm prior results.

the maternal immune response (i.e. elevated maternal cytokine levels) 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, which has important implications for the external validity of our results.

The abovementioned articles consider the effects of maternal influenza exposure during pregnancy on child outcomes. They do not consider multigenerational effects though, and to the very best of our knowledge, neither the medical nor the economic literature has established or investigated multigenerational effects of virus infections during pregnancy. However, other prenatal shocks have been shown to trigger multigenerational responses. For instance, the Dutch Hunger Winter of 1944-1945 (see Roseboom et al. (2011) for a review of related studies) affected the prenatally exposed but also the subsequent generation (Painter et al., 2008), with epigenetic changes being a potential underlying mechanism (Heijmans et al., 2008).[2] In animal models, evidence for transgenerational effects

[2]Kaati et al. (2007) furthermore establish transgenerational (i.e. over three generations)

of prenatal shocks is abundant. A review of the corresponding literature would be beyond the scope of this article, but the interested reader is referred to, for example, Jablonka and Raz (2009) and Franklin and Mansuy (2010). See also Lundborg and Stenberg (2010).

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 and Morens, 2006). In Sweden, the first case was reported on the 5th of July in Malmö, and over the course of the pandemic, at least 10% of the Swedish population had been infected.[3] 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 at the time. It is thus responsible for the last pronounced mortality peak in Sweden until today (Sundin and Willner, 2007).

Second, the Spanish flu happened unexpectedly and its timing as well as its intensity was unforeseen even by medical professionals of that time. See Karlsson et al. (2012) and references therein for an excellent overview regarding the Swedish case.

Third, the pandemic was over 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. Like 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.[4] We see two smaller waves of influenza infections, though.

responses to nutritional availability during the slow growth period, i.e. around the age of 8-12.

[3] 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 likeliy a lower bound (Medicinalstyrelsen, 1920).

[4] Figure A.2 presents influenza mortality with a similar spike.

One shortly after the peak in 1918, and one in the beginning of 1920.[5]

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. Here, we greatly benefit from the availability of morbidity data in Sweden. While the existing literature needs to rely predominantly on mortality due to data constraints, Mamelund (2006) documents the existence of a social gradient in mortality for the Spanish flu in Oslo, Norway.[6] There is thus some concern that inference based on mortality data might capture living in a poor neigbourhood. This is less of a problem with morbidity. Since influenza is an air-borne virus, the random component in infection rates is larger than for the case fatality rate.[7]

The Spanish flu is also particularly suited for testing the effects of a fetal insult due to the age profile of the infected. Normal influenza strains follow an "U"-pattern and infect the very young and the very old. The Spanish flu followed a "W"-age profile when it comes to mortality, killing the young, the old, and individuals in the range of 20 to 30, i.e. individuals in the childbearing age (Taubenberger and Morens, 2006).[8] Figure 2 plots the morbidity age profile in Sweden, which shows a similar pattern: Infection rates were highest among young adults and especially for females between the age of 20 to 30.[9]

3.2 World War I and parental quality

The Spanish flu episode is an attractive historical event for the study of fetal 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

[5] For the purpose of our paper, we disregard the second and third wave and only use morbidity data for the influenza spike in 1918 and 1919.

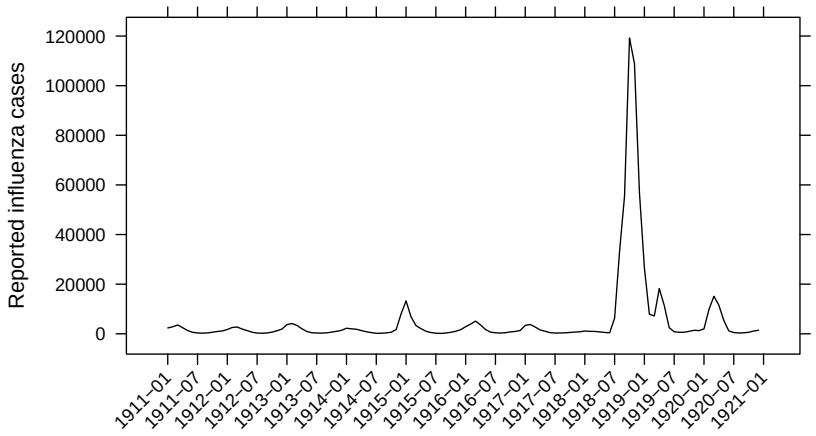
[6] Using apartment size as a measure for individual wealth in two selected parts of Oslo, Mamelund reports that individuals who were able to rent apartments with two, three or four rooms had 34, 41 or 56% lower mortality rates than individuals in one-room apartments, respectively. These estimates hint to a potentially stark social gradient in mortality.

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

[8] Taubenberger and Morens (2006, p. 20) use US data to show these patterns in mortality. Morbidity appears to follow a less pronounced "W" pattern. In fact, it looks more like an inverse-W. However, morbidity rates for people in the childbearing age are relatively high.

[9] The age profile for mortality can be seen in in figure A.1, which reveals a similar pattern but with higher mortality for men than for women in prime age.

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



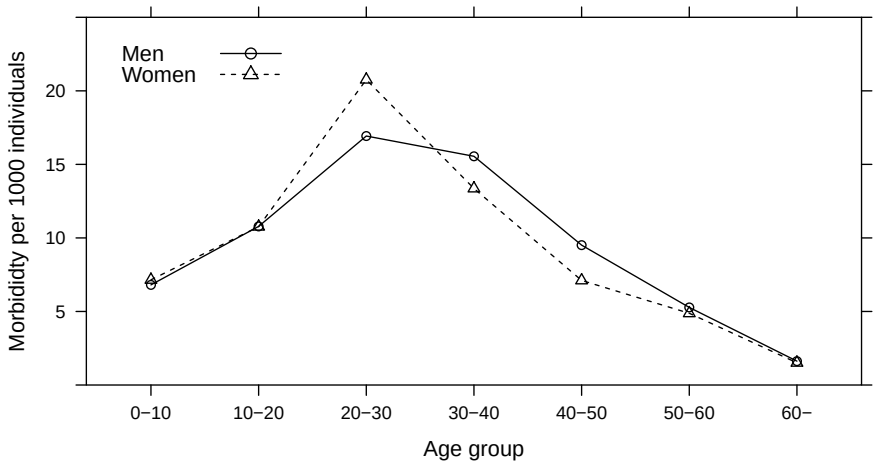
Source: Medicinalstyrelsen 1911-1920

the effects. Since the flu episode occured 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).[10]

The immediate years after the armistice in November 1918 developed ambiguously. On the one hand, the scarcity of goods was alleviated, and political reforms such as the introduction of the eight hour working day and district nurses arguably led to improvements in the living conditions of people, and to maternal and infant health in particular (Sundin and Willner, 2007). On the other hand, business uncertainty prevailed, and many of the previously booming industries faced difficulties in transitioning from the war environment to peacetime production (Montgomery, 1955).

[10]However, the Swedish export sector faced a high demand during the war, particularly from Germany.

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



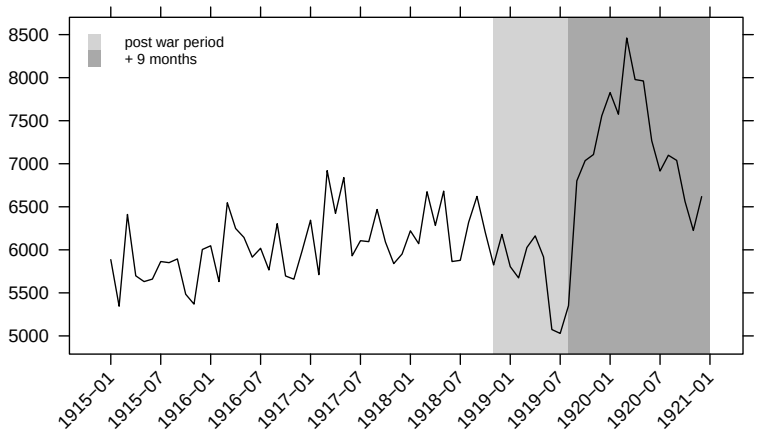
Source: Medicinalstyrelsen 1920

Notes: This graph is based on a subset of the morbidity data in Sweden where the age of the patient was registered, thus the number on the vertical axis is not informative of the prevalence of influenza in the population at large.

The overlap of World War I with the Spanish flu episode begs the question if the pool of potential parents (i.e. the grandparents to individuals in our sample) could be affected. As Brown and Thomas (2011) has pointed out, drafting procedures are likely to affect parental quality in the US, but drafting procedures do not apply to our case. Nevertheless, 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 Februrary 1914, when political tensions between the participating countries grew and war was widely anticipated, plans of the Swedish government to reduce the defence budget was 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 defence spending instead.

This raises the question if parents might have deferred their fertility decisions in anticipation of war involvement. If certain types of parents postpone their fertility decisions and other types do not, then Brown and Thomas (2011)'s ar-

Figure 3: Cohort size of newborns in Sweden, 1915-1920



gument that the composition of parental quality changes over time also applies in Sweden. Unfortunately, we have no observable information on the parents of the cohort born during that time. However, if it is true that people defer their fertility decisions, we would expect an increase in the cohort size of newborns after the war, which we plot in figure 3. The most important aspect of figure 3 is the sharp increase approximately one year after the influenza peak, whose timing coincides with the end of World War I (plus 9 months) as indicated by the shaded area. 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 and thus a potential change in parental quality.

In light of the above discussion, it seems safe to say that even though Sweden did not participate in World War I, a comparison involving war and post-war times is difficult to make. Economic environments are very different, and the cohort size pattern suggests that parental quality might have shifted. Most importantly, some of the individuals who experienced the Spanish flu as a fetal insult are potentially born to parents of higher quality, which might cushion the effect of the flu. For these reasons, the subsequent analysis will be carried out with and without excluding individuals conceived after the end of World War I.

4 Data

4.1 Measures and construction of influenza exposure

As discussed in section 3.1, we rely on influenza morbidity data, standardized by population as of 1915, for our analysis.[11] Morbidity data 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 time and in order to monitor the development of common epidemic diseases, all doctors in public service (*Tjänsteläkare*) were obligated to report cases of epidemic diseases to Medicinalstyrelsen. Our data on influenza morbidity is based on these reports and presented in table 2 for the county level and just before and during the peak of the Spanish flu. We see that while there is some geographical variation in the intensity of the disease, infection rates during the peak vary only from roughly 7 to 18%.

While the use of morbidity avoids capturing a social gradient, some problems due to measurement errors emerge. First, it is likely that the reported infection rates understate the true rates (Åhman, 1990) since not every infected person went to the doctor and doctors with private practices did not report to Medicinalstyrelsen. This measurement error furthermore potentially increases with the level of influenza cases. This is because doctors facing an epidemic might reallocate their time to treating their patients instead of carefully reporting influenza cases to the authorities. For these reasons, we use the morbidity data to infer the timing of the influenza. While this approach ignores level differences, it still enables us to exploit some geographical variation in the onset and the timing of the influenza. Moreover, it makes our estimates more comparable to the existing literature.

Second, morbidity for the city of Stockholm and Gothenburg are only reported for the poor and in the case of Malmö, reporting behavior is inconsistent over time. As a consequence, we indirectly infer the timing of the influenza in these

[11] Some authors also combine influenza mortality with other causes of death, such as pneumonia. The reason is that doctors might misclassify influenza mortality as pneumonia mortality. While this might be a problem in our case as well, it does not appear to be a problem of practical importance. Looking at the development of pneumonia deaths over time in figure A.2, we see a small spike in 1919, but compared to the reported cases of influenza deaths, this spike seems negligible. A comparison of incidence rather than fatalities yields a similar picture, which can be seen in figure A.3 in the appendix.

Table 2: Influenza morbidity by county

County	Pop	Influenza morbidity			
		prior	%	*peak*	%
Stockholms stad	392427	105	0.03	5604	1.43
Stockholm (län)	228230	390	0.17	19730	8.64
Uppsala	132400	124	0.09	14253	10.77
Södermanland	183839	148	0.08	15751	8.57
Östergötland	300165	183	0.06	28546	9.51
Jönköping	219895	0	0.00	18492	8.41
Kronoberg	156596	175	0.11	11630	7.43
Kalmar	227622	154	0.07	14119	6.20
Gottland	55451	37	0.07	5585	10.07
Blekinge	150055	121	0.08	15125	10.08
Kristianstad	234994	157	0.07	17342	7.38
Malmöhus	475893	769	0.16	57682	12.12
Halland	147296	201	0.14	11709	7.95
Göteborg o. Bohus	406112	560	0.14	37678	9.28
Alvsborg	293577	354	0.12	28645	9.76
Skaraborg	241026	73	0.03	18700	7.76
Värmland	260447	421	0.16	25298	9.71
Örebro	212113	331	0.16	19224	9.06
Västmanland	162774	127	0.08	20254	12.44
Kopparberg	242349	308	0.13	29512	12.18
Gävleborg	260586	340	0.13	31478	12.08
Västernorrland	259826	220	0.08	21413	8.24
Jämtland	124541	305	0.24	21885	17.57
Västerbotten	170299	345	0.20	13862	8.14
Norrbotten	174227	222	0.13	12077	6.93

Notes: Population in 1915 and influenza morbidity prior, i.e. January to June 1918, and during the Spanish flu epidemic, i.e. from July 1918 to February 1919. Reported cases for the city of Stockholm are based on poor people only. Note that neither the figures for Gothenburg and Bohus nor Malmöhus include the cities of Gothenburg or Malmö.

cities by the morbidity rates in the surrounding counties, i.e. if the Spanish flu hits a county that surrounds a given city, we take this as evidence that the flu has reached that city. In a robustness check, we will exclude these cities to evaluate the impact of this approach.

Third, there will be measurement errors due to misclassification. Since our main focus lies on fetal insults, we match individuals to regional influenza morbidity

rates during their parents' in utero months. However, since we do not observe the date of conception, we need to infer conception by the time of birth. In the absence of information on the gestation length, we assume that each pregnancy lasted for 38 weeks, which is the duration of a normal pregnancy. This is somewhat error-prone as it is known that health complications such as the ones considered here shorten the gestation period. Additionally, we only observe the birthmonth, and not the exact birthday, which aggravates the uncertainty as to the timing of the influenza exposure.[12] We deal with this uncertainty in two ways: First, we match ten months of morbidity rates to each parent, being aware of the fact that for individuals born late in a given month, this assignment rule captures one month pre-conception. Likewise, for the individual born in the beginning of a month, this matching includes about a month after birth. Note though that it is ruled out that exposed individuals are in our comparison group. Second, we aggregate influenza exposure to trimesters, which helps to alleviate problems due to misclassification.

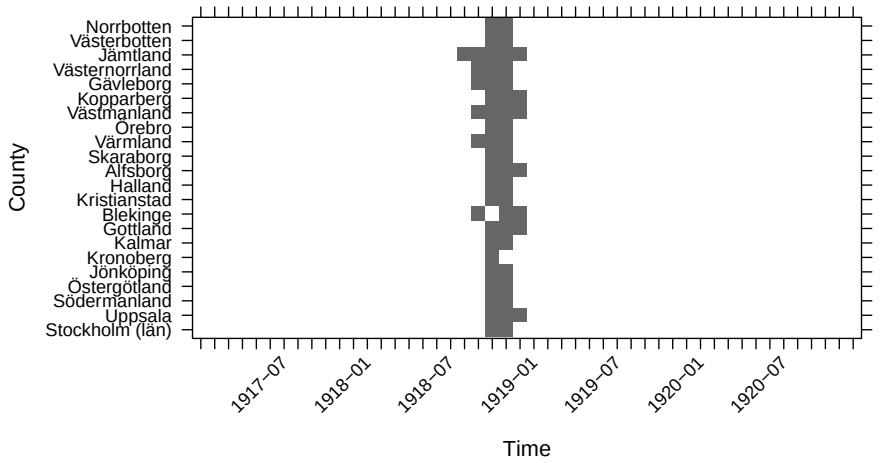
To be more explicit, our trimester exposure dummies are constructed in the following way: it takes the value 1 if the monthly morbidity rate in at least one of the months associated with a given trimester exceeds a threshold τ. In our main specification, this threshold will be 1.5% of the regional population. Figure 4 shows the exact exposure status for all counties and all months during the period considered, using a threshold of $\tau = 1.5\%$.

4.2 Individual level data

The individual level data are based on Statistics Sweden's multi-generation register (SCB, 2011) from which a 35% random sample of individuals born in Sweden between 1932-1967 is available. 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. This includes information of birthdates up to birthmonth as well as the birthplace used to match regional influenza incidence during the parents' fetal stage to the index persons.

[12] Regional mobility might pose an additional problem if a pregnant women changes counties before delivery. We do not consider this a substantial problem since most people in the 1910s lived in rural areas, worked in agriculture, and delivered babies predominantly at home, all of which limits regional mobility. The use of data on county level further reduces problems due to regional mobility, since Swedish counties are rather large geographical units and mobility would have to occur across counties to pose a problem.

Figure 4: Spanish flu exceeding 1.5 percent of population by county.



Source: Medicinalstyrelsen 1911-1920

We consider the impact of a potential parental fetal insult on education and long-run earnings. Annual earnings data are taken from tax records available from 1968 to 2007. We use all earnings information available between the age of 27 and 64. For each gender, we construct a measure of long-run earnings by taking the mean residual for each individual from a regression of annual earnings on measurement year dummies. For the parent generation, years of education are constructed from educational attainment data in the 1970 census, which is also used to construct a high school completion dummy. For the index generation, years of education is constructed from data in the 1990 census and the education registers from 1999 and 2003, depending on the year of birth. This source is also used to construct a college attendance dummy.

Our main sample consists of all individuals in the sample with both parents born between 1915 and 1920. We exclude observations with missing information on education of the index person or their parents. As mentioned in section 3.2, all our estimations are carried out including and excluding individuals conceived after the end of World War I. Descriptive statistics for the index generation by gender and exposure are shown in table 3. The first row for each variable in table 3 reports the mean and the second (in parentheses) the standard deviation.

The table shows that, unconditionally, women with exposed parents have slightly higher educational attainment and long-run earnings than women with unexposed parents. When it comes to men, those with exposed parents also have higher educational attainment but lower unconditional long-run earnings. For both men and women, the exposed parents have higher educational attainment and are younger at the time of birth of the children in the sample.

Table 3: Descriptive statistics for the index generation

	Females			*Males*		
	Exposed Parent	*Unexposed Parent*	*All*	*Exposed Parent*	*Unexposed Parent*	*All*
Years of education	11.41 (7.18)	11.33 (7.28)	11.36 (7.25)	11.26 (8.17)	11.24 (8.16)	11.25 (8.16)
College	0.31 (0.21)	0.30 (0.21)	0.30 (0.21)	0.27 (0.20)	0.26 (0.19)	0.27 (0.19)
Long-run earnings[a]	11.91 (0.93)	11.89 (0.99)	11.89 (0.97)	12.24 (1.06)	12.27 (0.88)	12.26 (0.93)
Year of birth	1947.94 (28.41)	1947.37 (29.37)	1947.54 (29.14)	1947.90 (27.86)	1947.46 (29.43)	1947.59 (28.98)
Mother's education	7.92 (3.56)	7.83 (3.14)	7.86 (3.27)	7.89 (3.25)	7.84 (3.14)	7.85 (3.17)
Father's Education	8.57 (6.30)	8.42 (5.77)	8.47 (5.94)	8.59 (6.48)	8.41 (5.76)	8.47 (5.99)
Mother's age at birth	29.35 (29.05)	29.41 (29.94)	29.39 (29.66)	29.32 (28.57)	29.51 (29.64)	29.45 (29.32)
Fathers's age at birth	30.27 (28.68)	30.53 (29.18)	30.45 (29.03)	30.24 (28.47)	30.64 (29.47)	30.51 (29.19)
n	6545	14822	21367	6909	15288	22197

Notes: The index generation consists of all individuals in the sample with parents born between 1915 and 1920. The first figure in each cell indicates the mean, and the second figure in parenthesis represents the standard deviation. [a]This represents the mean of logged earnings, whereas the mean of residual logged earnings is used in the estimations.

5 Empirical strategy

In our preferred specification, we estimate the effect of potential parental in utero exposure to the flu by comparing individuals with a fetally insulted parent to individuals in the same cohort whose parents have not been fetally insulted. Cohort membership here is defined by year of birth, and exposure status by cohort is shown in figure 5.[13] As discussed in section 4.1, we use indicators for potential parental in utero exposure for each pregnancy trimester on the county level, and all results reported in the next section are based on a threshold of 1.5% of the population. The main equation used in the analysis for the index generation is as follows:

$$y_{ic} = \alpha + \sum_{j=1}^{3} \beta_j I_c^j + \sum_k \beta_k X_{ic}^k + \delta I_c^* + \gamma_c^p + m_i + m_i^p + \omega_i + \gamma_c^p h(t^p) + \epsilon_{ic}^p \quad (1)$$

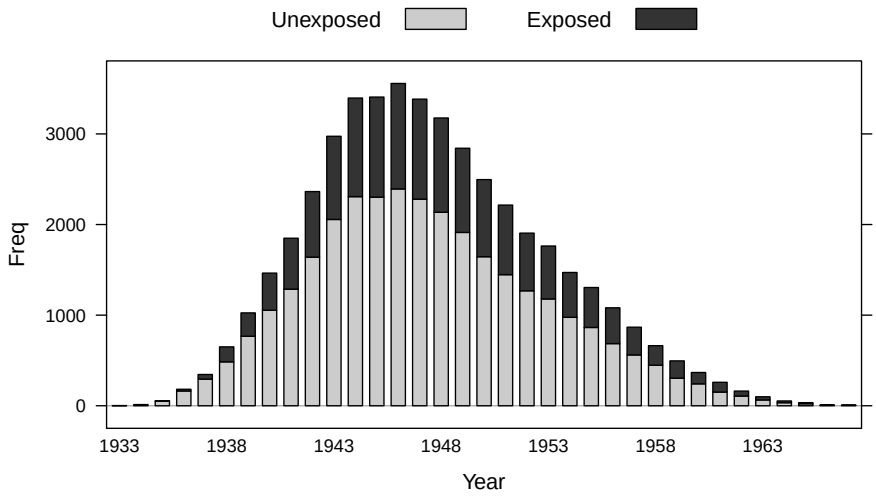
where y_{ic} is the outcome of individual i in county c, I_c^j is an indicator which is one if reported influenza incidence exceeded the threshold τ in the county of birth c of individual i's parent in at least one of the months associated with a given trimester j of that parent. X_{ic}^k are potential control variables, and I^* is an indicator for exposure in the first trimester after birth, similarly defined as I. γ_c^p are fixed effects for the parent's county of birth, m_i are calender month fixed effects (referring to the birthmonth), and m_i^p are calender month fixed effects for the fetally insulted parent. ω_i are birthyear fixed effects, and $\gamma_c^p h(t^p)$ indicates a county-specific quadratic time trend in parental time of birth.

Our motivation for the inclusion of these control variables is as follows: We control for post-birth exposure to avoid confounding exposure in the third trimester with postnatal exposure. The calender month fixed effects capture season of birth effects, which are known to affect outcomes. We include county specific time trends for the parents to control for seculiar trends, e.g. in educational attainments. Note that by simultaneously controlling for a time trend based on the time of birth of the parents and the year of birth of the index person, we indirectly control for age at birth (partially).

For the parent generation, we follow the literature and use a deviation from

[13] Defining the cohort on a monthly basis leaves the point estimates virtually unchanged. However, some monthly defined cohorts contain only very few individuals, which is why we prefer to use yearly cohorts.

Figure 5: Distribution of birthyears of index generation



cohort trend design. The main equation used in the analysis is given by:

$$y_{ic} = \alpha + \sum_{j=1}^{3} \beta_j I_c^j + \delta I_c^* + \gamma_c + m_i + \gamma_c h(t) + \epsilon_{ic} \tag{2}$$

where y_{ic} is the outcome of individual i in county c, I_c^j is an indicator which is one if reported influenza incidence exceeded a threshold in individual i's county of birth c in at least one of the months associated with a given trimester j. I^* is an indicator for post-birth exposure. γ_c are birthcounty fixed effects, m_i are calender month fixed effects, and $\gamma_c h(t)$ indicates a county-specific quadratic time trend.

All estimates are obtained using ordinary least squares with standard errors being clustered on the county level. We apply the standard finite sample adjustment following Cameron et al. (2008) and Cameron and Miller (2010) and base all inferential statements on the t-distribution with *g-1* degrees of freedom, where g refers to the number of counties.[14]

[14]In the case of binary dependent variables, the linear probability model has been adopted for convenience, and the resulting estimates should be considered linear approximations.

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 4 to 11, we present our estimation results for years of schooling, college attendance, long-run earnings and number of children, respectively. The upper panel displays the effect of maternal exposure to the flu, and the lower panel shows the effect of paternal exposure. The left panel shows results for women, and the right panel for men. We first show regression results for the full sample, and then for the sample where parents conceived after the end of World War I are excluded. Standard errors clustered by parental county of birth are reported in parentheses.

We estimate three 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 a regression control associated with a given mechanism to affect the estimates for our exposure dummies. We therefore subsequently add control variables in models (2)-(3). To start with, exposed parents might have lower education levels due to exposure, which could mechanically translate to lower education levels of their children. In model (2), we therefore control for years of education of the fetally insulted parent by including a set of dummy variables representing different education levels. A second potential mechanism reflects assortative mating: A fetal 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 (3), we controls for both parents' education levels to investigate this possibility. Apart from capturing such parental composition effects, controlling for both parents' education levels also takes parental socioeconomic status more comprehensively into account.

Educational attainment: As can be seen in table 4, a maternal fetal insult in the second trimester has detrimental effects on the educational attainment of female offspring. Exposure lowers schooling levels by 2 to 2.5 months (1.5-1.8%), depending on the specification. We find no effects on male offspring. For paternal exposure, we find negative effects on male offspring's educational attainment of 2.4 to 3 months (1.8-2.2%), though. In all these cases, the point estimates are attenuated by at most 18% when controlling for the different mechanisms, but they are well within the range of one standard error for each estimate. When parents conceived after the end of World War I are excluded, we generally see an increase in magnitudes of these estimates. For instance, a maternal fetal insult in the second trimester is now estimated to decrease female offspring's schooling by about 2.6 to 3.3 months. We furthermore find some negative effects of exposure in the third trimester on male offspring's education level. This effect is potentially mediated by indirect mechanisms, though, as we see a large drop in magnitude (by about 40%) and a loss of statistical significance when controlling for both parental education levels. The magnitude of the effect of paternal exposure in the second trimester on male offspring's schooling has similarly increased and remains stable over specifications. However, statistical significance is lost, which could be due to the loss in sample size. It is interesting to note though that out of 18 reported coefficients for paternal exposure, 15 are associated with a negative sign.

Table 6 and 7 show similar patterns for college attendance. A maternal fetal insult in the second trimester decreases the probability of attending college for women by about 3 to 3.6 percentage points, which corresponds to a decrease of about 10-12%. No effects for men are found. A paternal in utero shock in the second trimester decreases the probability of college attendance for male offspring by about 2.9-3.5 percentage points (11-13%). When parents conceived after the end of World War I are excluded, all magnitudes increase. As before, the effect of paternal exposure loses statistical signficance, which could be due to a loss in statistical precision. Now we furthermore find some evidence for positive effects of maternal exposure in the second trimester on male offspring, but the evidence is inconclusive here.

Long-run earnings: In tables 8 and 9, we present results for trend-adjusted long-run earnings (in absolute values, prices in 2010 SEK). As can be seen, we do not find any statistically significant effects.

NUMBER OF CHILDREN: Regression results for the number of children are reported in tables 10 and 11. In contrast to previous results where a maternal fetal insult during the second trimester affected educational outcomes of female offspring and a paternal fetal insult in the same trimester affected educational outcomes of male offspring, we find that paternal exposure in the first trimester lowers the number of children of female offsprings. Please note though that an interesting pattern regarding the signs emerge: for maternal exposure, almost all but three estimates are positive, and for paternal exposure, all estimates associated with female offspring are negative whereas all estimates associated with male offspring are positive. This indicates that there might be differential effects, but for reasons of statistical precision we are unable to detect these.[15]

Note that controlling for parental characteristics does not have a large impact on our estimated magnitudes, which suggests that our baseline estimates capture mechanisms not accounted for, e.g. direct biological effects. To investigate this further, we analyse the effect of intrauterine Spanish flu exposure 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.

[15]If a fetal insult does indeed not affect the number of children, i.e. if the true effects are zero, then we would expect the signs to follow a random pattern.

Table 4: Regression results for years of education

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.128	0.090	0.096	−0.031	−0.006	−0.033
	(0.098)	(0.086)	(0.078)	(0.098)	(0.096)	(0.087)
trimester 2	-0.211^{**}	-0.194^{**}	-0.173^{**}	0.057	0.009	0.019
	(0.065)	(0.061)	(0.056)	(0.071)	(0.071)	(0.062)
trimester 3	0.113	0.114	0.111	−0.102	−0.086	−0.082
	(0.072)	(0.074)	(0.070)	(0.066)	(0.058)	(0.056)
Adj. R^2	0.069	0.165	0.202	0.052	0.158	0.211
Num. obs.	30331	30331	30331	31730	31730	31730
Father:						
trimester 1	−0.030	−0.038	−0.062	0.076	0.020	0.011
	(0.082)	(0.078)	(0.077)	(0.090)	(0.083)	(0.084)
trimester 2	0.068	0.081	0.108	-0.247^{*}	-0.229^{*}	-0.202^{*}
	(0.077)	(0.073)	(0.067)	(0.102)	(0.094)	(0.094)
trimester 3	−0.003	−0.001	−0.007	0.166	0.096	0.115
	(0.077)	(0.067)	(0.069)	(0.089)	(0.078)	(0.075)
Adj. R^2	0.069	0.183	0.203	0.051	0.194	0.212
Num. obs.	30331	30331	30331	31730	31730	31730

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table 5: Regression results for years of education, exluding parents conceived after World War I.

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.149	0.146	0.190	−0.185	−0.056	−0.034
	(0.136)	(0.119)	(0.122)	(0.174)	(0.177)	(0.161)
trimester 2	−0.277**	−0.246**	−0.220**	0.096	0.057	0.074
	(0.085)	(0.080)	(0.067)	(0.094)	(0.095)	(0.083)
trimester 3	0.092	0.088	0.088	−0.225*	−0.170*	−0.139
	(0.089)	(0.082)	(0.084)	(0.088)	(0.080)	(0.075)
Adj. R^2	0.069	0.169	0.205	0.060	0.167	0.219
Num. obs.	16797	16797	16797	17591	17591	17591
Father:						
trimester 1	−0.188	−0.054	−0.139	−0.268	−0.064	−0.079
	(0.149)	(0.159)	(0.148)	(0.156)	(0.162)	(0.152)
trimester 2	−0.254	−0.136	−0.128	−0.383*	−0.300	−0.247
	(0.126)	(0.119)	(0.113)	(0.162)	(0.167)	(0.167)
trimester 3	−0.130	−0.099	−0.139	0.220	0.188	0.215
	(0.125)	(0.120)	(0.109)	(0.122)	(0.122)	(0.118)
Adj. R^2	0.070	0.186	0.207	0.058	0.202	0.220
Num. obs.	16797	16797	16797	17591	17591	17591

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table 6: Regression results for college attendance

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.016	0.009	0.010	−0.003	0.000	−0.004
	(0.016)	(0.015)	(0.015)	(0.016)	(0.016)	(0.014)
trimester 2	−0.036**	−0.033**	−0.030**	0.019	0.012	0.014
	(0.011)	(0.011)	(0.010)	(0.012)	(0.012)	(0.011)
trimester 3	0.013	0.013	0.012	−0.017	−0.015	−0.014
	(0.014)	(0.015)	(0.015)	(0.011)	(0.009)	(0.010)
Adj. R^2	0.029	0.113	0.140	0.022	0.105	0.145
Num. obs.	30331	30331	30331	31730	31730	31730
Father:						
trimester 1	0.011	0.011	0.006	0.029	0.020	0.019
	(0.015)	(0.014)	(0.014)	(0.015)	(0.016)	(0.017)
trimester 2	0.004	0.005	0.010	−0.035*	−0.033*	−0.029*
	(0.013)	(0.013)	(0.012)	(0.014)	(0.013)	(0.013)
trimester 3	0.004	0.005	0.004	0.026*	0.017	0.020
	(0.010)	(0.008)	(0.008)	(0.013)	(0.011)	(0.011)
Adj. R^2	0.029	0.122	0.141	0.020	0.131	0.146
Num. obs.	30331	30331	30331	31730	31730	31730

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table 7: Regression results for college attendance, exluding parents conceived after World War I.

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.009	0.008	0.014	−0.010	0.006	0.009
	(0.022)	(0.019)	(0.021)	(0.029)	(0.030)	(0.027)
trimester 2	−0.041*	−0.036*	−0.033*	0.033*	0.027	0.030*
	(0.017)	(0.016)	(0.014)	(0.013)	(0.014)	(0.013)
trimester 3	−0.001	−0.002	−0.002	−0.029	−0.022	−0.017
	(0.014)	(0.014)	(0.015)	(0.015)	(0.014)	(0.014)
Adj. R^2	0.031	0.117	0.143	0.025	0.108	0.147
Num. obs.	16797	16797	16797	17591	17591	17591
Father:						
trimester 1	−0.021	0.000	−0.015	−0.008	0.016	0.014
	(0.025)	(0.029)	(0.028)	(0.026)	(0.028)	(0.027)
trimester 2	−0.046	−0.029	−0.028	−0.058*	−0.047*	−0.040
	(0.023)	(0.023)	(0.022)	(0.022)	(0.022)	(0.023)
trimester 3	−0.022	−0.017	−0.023	0.036	0.032	0.036
	(0.021)	(0.020)	(0.018)	(0.017)	(0.018)	(0.018)
Adj. R^2	0.032	0.124	0.144	0.023	0.135	0.149
Num. obs.	16797	16797	16797	17591	17591	17591

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table 8: Regression results for long-run earnings

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	2776	2135	2264	886	1484	564
	(2451)	(2288)	(2182)	(3696)	(3549)	(3296)
trimester 2	−3139	−2928	−2566	−1739	−3026	−2719
	(2115)	(2166)	(2146)	(3333)	(3415)	(3172)
trimester 3	−1170	−1117	−1177	−845	−370	−283
	(1675)	(1615)	(1564)	(2874)	(2890)	(2797)
Adj. R^2	0.014	0.046	0.060	0.013	0.043	0.063
Num. obs.	30331	30331	30331	31730	31730	31730
Father:						
trimester 1	1168	1095	725	5605	4079	3869
	(2601)	(2619)	(2664)	(5015)	(4927)	(4772)
trimester 2	2958	3118	3560	−4701	−4172	−3625
	(2147)	(1996)	(1966)	(4606)	(4504)	(4377)
trimester 3	2836	2899	2727	3206	1381	1900
	(1944)	(1819)	(1926)	(3231)	(3296)	(3319)
Adj. R^2	0.013	0.053	0.059	0.011	0.057	0.062
Num. obs.	30331	30331	30331	31730	31730	31730

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table 9: Regression results for long-run earnings, exluding parents conceived after World War I.

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	1762	1723	2568	−5411	−2293	−1604
	(4131)	(4070)	(3972)	(6163)	(6016)	(5789)
trimester 2	−3945	−3499	−3030	3797	2838	3445
	(3012)	(3012)	(2914)	(4787)	(4647)	(4287)
trimester 3	−1969	−1966	−1894	−6604	−5109	−4054
	(2296)	(2091)	(2046)	(4421)	(4719)	(4585)
Adj. R^2	0.016	0.049	0.061	0.018	0.048	0.066
Num. obs.	16797	16797	16797	17591	17591	17591
Father:						
trimester 1	−6847	−4584	−5981	−2609	3264	2820
	(3926)	(4081)	(3962)	(7755)	(8770)	(8266)
trimester 2	−5283	−3351	−3161	−5455	−3139	−2233
	(4487)	(4410)	(4398)	(7347)	(7741)	(7678)
trimester 3	−298	181	−582	2412	1671	2258
	(3153)	(2965)	(2971)	(4885)	(4960)	(4929)
Adj. R^2	0.015	0.054	0.060	0.015	0.061	0.065
Num. obs.	16797	16797	16797	17591	17591	17591

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table 10: Regression results for number of children

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.023	0.024	0.023	0.000	0.001	−0.001
	(0.027)	(0.027)	(0.027)	(0.040)	(0.041)	(0.040)
trimester 2	−0.008	−0.008	−0.008	0.018	0.016	0.016
	(0.025)	(0.025)	(0.025)	(0.026)	(0.026)	(0.026)
trimester 3	0.041	0.040	0.040	−0.011	−0.011	−0.011
	(0.031)	(0.030)	(0.030)	(0.024)	(0.024)	(0.024)
Adj. R^2	0.005	0.005	0.005	0.006	0.007	0.007
Num. obs.	30331	30331	30331	31730	31730	31730
Father:						
trimester 1	0.000	0.001	0.001	0.028	0.025	0.024
	(0.041)	(0.041)	(0.040)	(0.036)	(0.036)	(0.036)
trimester 2	−0.044	−0.045	−0.047	−0.026	−0.025	−0.023
	(0.028)	(0.027)	(0.027)	(0.038)	(0.039)	(0.039)
trimester 3	−0.007	−0.006	−0.006	0.012	0.011	0.012
	(0.027)	(0.027)	(0.027)	(0.025)	(0.025)	(0.025)
Adj. R^2	0.005	0.005	0.006	0.007	0.008	0.008
Num. obs.	30331	30331	30331	31730	31730	31730

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table 11: Regression results for number of children, exluding parents conceived after World War I.

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.063	0.061	0.059	0.023	0.029	0.030
	(0.060)	(0.060)	(0.060)	(0.068)	(0.068)	(0.068)
trimester 2	0.003	0.002	0.001	0.064	0.062	0.063
	(0.043)	(0.044)	(0.044)	(0.046)	(0.046)	(0.047)
trimester 3	0.076	0.076	0.074	−0.023	−0.021	−0.019
	(0.037)	(0.037)	(0.037)	(0.031)	(0.031)	(0.032)
Adj. R^2	0.004	0.004	0.004	0.006	0.007	0.007
Num. obs.	16797	16797	16797	17591	17591	17591
Father:						
trimester 1	−0.209*	−0.213*	−0.213*	0.052	0.057	0.056
	(0.078)	(0.078)	(0.078)	(0.093)	(0.092)	(0.091)
trimester 2	−0.091	−0.093	−0.096	0.007	0.009	0.013
	(0.049)	(0.049)	(0.048)	(0.067)	(0.068)	(0.069)
trimester 3	−0.044	−0.044	−0.043	0.056	0.055	0.058
	(0.050)	(0.050)	(0.050)	(0.061)	(0.060)	(0.060)
Adj. R^2	0.006	0.006	0.006	0.007	0.007	0.008
Num. obs.	16797	16797	16797	17591	17591	17591

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

6.2 Parent generation

In table 12, we present estimation results for the parent generation. The upper panel shows the sample from 1915 to 1920, and the lower panel shows the results when individuals conceived after the end of World War I have been excluded. As discussed in section 3.1, we restrict our sample in the lower panel to cohorts conceived during the war to control for potential changes in parental quality.[16]

As can be seen in table 12, when the sample is not restricted and a change in parental quality is potentially allowed, we find no significant effects of flu exposure. When the sample is restricted to include only parents conceived during war time, we obtain significant negative effects of influenza exposure in the first trimester on human capital variables for women. In utero exposure in the first trimester leads to a decrease in about 1.4 months of schooling (1.5%), as well as a decrease in the probability of high school completion by 3 percentage points. We furthermore find that a fetal insult in the second trimester lowers the probability of high school completion of men by 2.4 percentage points (baseline: 19%).

These results are in the same ballpark as those obtained by Almond (2006), but they are probably lower bounds for the effect in the parent generation. This is because observing information on parents necessitates survival until childbearing age and actual childbearing. The most severely affected parents are likely to have died or to stay childless until the data was collected, which is why all estimates for the parent generation should be interpreted with caution. Nevertheless, it is interesting to note that the effects for women are found in the first trimester and not in the second. If our results of section 6.1 were driven by changes in the socioeconomic status of parents, then we would expect to find effects in the same trimester. Since this is not the case, we interpret these results as evidence for direct biological effects, rather than indirect effects, which will be discussed more thoroughly in section 8.

[16] Parental quality here refers to the parents of our parent generation, i.e. the grandparents of the index generation.

Table 12: Regression results for the parent generation

	Women			Men		
	Education	High School	Earnings	Education	High School	Earnings
Sample: 1915 to 1920						
trimester 1	0.004	−0.003	0.011	0.087	0.014	0.022
	(0.035)	(0.007)	(0.021)	(0.059)	(0.009)	(0.022)
trimester 2	0.028	0.007	0.019	−0.007	−0.009	−0.008
	(0.032)	(0.007)	(0.020)	(0.063)	(0.009)	(0.020)
trimester 3	0.028	0.004	0.001	0.086	0.018	0.014
	(0.045)	(0.009)	(0.022)	(0.065)	(0.012)	(0.023)
Adj. R^2	0.028	0.019	0.009	0.037	0.032	0.022
Num. obs.	54203	54203	51493	52963	52963	52058
Sample: 1915 to 1919-07						
trimester 1	−0.115*	−0.030*	−0.042	−0.124	−0.019	0.046
	(0.044)	(0.012)	(0.042)	(0.125)	(0.019)	(0.046)
trimester 2	−0.016	−0.005	−0.009	−0.115	−0.024*	0.026
	(0.041)	(0.009)	(0.019)	(0.082)	(0.012)	(0.025)
trimester 3	−0.001	−0.004	−0.035	−0.018	0.002	0.039
	(0.067)	(0.014)	(0.034)	(0.088)	(0.014)	(0.026)
Adj. R^2	0.027	0.020	0.009	0.037	0.033	0.022
Num. obs.	34266	34266	32145	44447	44447	43625

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

Notes: Standard errors in parentheses, clustered on the county level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, a quadratic county of birth-specific time trend, and fixed effects for county of birth, year of birth and calendar month fixed effect.

6.3 Discussion of results

Two statistical issues need to be taken into consideration for the interpretation of our results. First, 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 classified as exposed but who never contracted the disease do not show any departure from the cohort, we can approximate the effect of the Spanish flu on those who contracted the disease. This is done 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).

Our baseline results regarding potential maternal exposure in the second trimester are -0.211 and -0.036 for years of education and college, respectively. For women's years of education, this translates into a decrease in schooling by roughly 10 months or 7%. For college attendance, we find a decrease in the probability of attending college for women by 14.4 percentage points (baseline: 30%). Note that while these estimates have to be taken with a grain of salt due to the associated uncertainty, this exercise shows that the potential effects of intrauterine health shocks on the offspring can potentially be quite large.

Second, despite the seemingly systematic nature of our results, they might be driven by random chance. This is because in each regression, we simultaneously

test three hypotheses, one for each trimester. Hence, the type-I error probability of the overall regression does not coincide with the corresponding probability of each individual test, making chance findings more likely by testing many hypotheses. In this context, it is useful to note that our results concerning maternal fetal insults during the second trimester are robust to multiplicity adjustments like the Bonferroni correction when applied to each regression independently.[17]

7 Robustness checks

We have conducted a series of robustness checks to test the sensitivity of our results. First, since morbidity data for Stockholm, Gothenburg and Malmö is only available for the poor, we have previously imputed the timing of the flu in these cities by assuming a similar timing as in the surrounding counties. In tables A.1 to A.6, we investigate the implications of this choice by excluding the cities of Stockholm, Gothenburg and Malmö in all regressions. Our main results of negative effects of maternal exposure in the second trimester on female offspring's educational attainment and paternal exposure in the second trimester on male offspring' educational attainment remains unchanged. For long run earnings, we find some evidence for positive effects of paternal exposure in the second trimester on female offspring's earnings, but this is not robust to the exclusion of parents conceived after the end of World War I. For this sample, there is also some indication that maternal exposure in the third trimester might increase in the number of female offspring's children.

Second, given that the flu occurred in a seasonally distinct time, there is some concern that our estimates might capture seasonal fluctuations despite the inclusion of calendar month fixed effects for both the fetally insulted parent and the offspring. To investigate this possibility, we consider a pseudo exposure by shifting and reshuffling the exposure indicators one year backward and one year forward in time. The results are reported in tables A.7 to A.12. We find only weak evidence for seasonal effects when forward shifted random exposure is considered. The seasonal patterns are rather unsystematic though and are found only in the first and third trimester, thus leaving our main results unaffected.

Third, we have experimented with different alternative thresholds, different sam-

[17] This implies multiplying each p-value with three, the number of hypotheses tested.

ple restrictions, and with using the morbidity level instead of the timing.[18] Given the patterns observed in our tables, it appears that our main results are robust against changes in the specifications. Additional results indicating predominantly positive effects of exposure in the first or third trimester (in addition to negative effects in the second trimester) appear for some specifications, but these findings do not appear robust.

8 Discussion and conclusion

Our results indicate that there are strong and potentially direct multigenerational effects of the Spanish flu in Sweden. Potential maternal in utero exposure in the second trimester leads to decreased educational attainment for female offspring (2-2.5 months). For paternal in utero exposure in the second trimester, we find indications of an analogous effect on male offspring, albeit issues of statistical precision prevent us from making definitive statements. We will therefore focus on maternal exposure in the second trimester.

The question arises how much of these results are mediated by effects on the socioeconomic status of exposed parents, i.e. indirect effects, and how much they represent direct, i.e. biological effects. We are not able to provide a definitive answer to this question, but we believe the latter to be the dominant effect for maternal exposure in the second trimester due to the following observations: First, controlling for socioeconomic status proxies of parents changes the estimated magnitudes by at most 20%, with the remaining effect still being statistically significant. If the estimated effects on the second generation capture effects on the first, we would expect to see large changes in the magnitude of these effects combined with a loss in statistical significance when controlling for parental socioeconomic status. This pattern is only observed for paternal exposure in the second trimester when parents conceived after the end of World War I are excluded, but not for maternal exposure in the second trimester.

Second, when effects on the parents are considered, exposure in the first trimester appears to have the largest impact on socioeconomic status, whereas no effects of exposure in the second trimester on women can be detected. Only for men we find that a fetal insult in the second trimester appears to lower the probability of

[18] In the interest of space, we do not report these tables, but they are available on request.

high school completion. If the effect of the Spanish flu was driven by exposure-induced changes in the socioeconomic status of parents, we would expect to find that both the parents and their children are affected by exposure in the same trimester. For women, we do not observe this in our data, which we interpret as evidence that the mediating effect of parental socioeconomic status is limited.

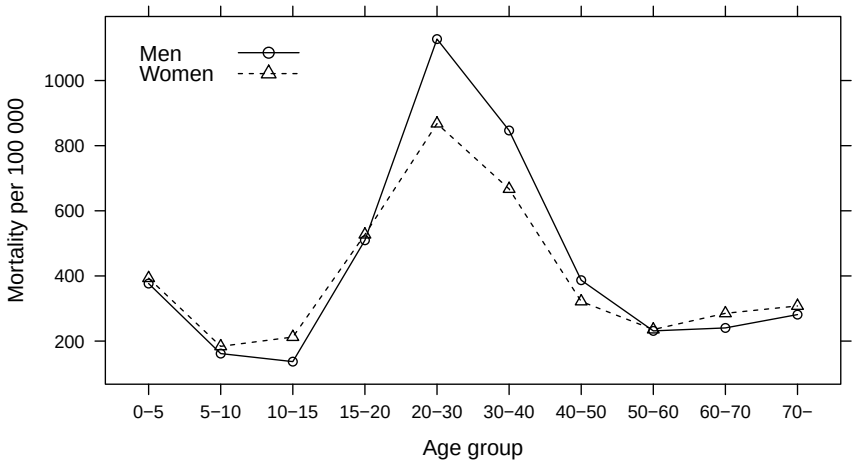
Third, since intergenerational correlations in education and earnings are typically less than one, indirect effects on the index generation are bounded from above by the effect on their parents. Since these correlations tend to be rather small in Sweden (Björklund and Salvanes, 2011; Niknami, 2010; Lindahl et al., 2013), we would expect effects on the second generation to be much smaller unless they represent direct biological effects. The most comparable measure across our two generations is years of schooling, and we previously found that when exlcuding parents conceived after the end of World War I, exposure in the first trimester reduces schooling of women by 1.4 months or 1.5%. The same sample restriction for their children gives a reduction in schooling by 2.6-3.3 months, or 1.9 to 2.4%. We see that the effect on the second generation is larger than the effect on their parents. While this discrepancy could be explained by differential measurement errors, it is consistent with direct effects.

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. It could however be the case that our controls for socioeconomic status of parents are not comprehensive enough, and that therefore residual family factors drive our results. Unfortunately, data limitations do not allow us to explore this possibility in much greater detail, but for the reasons discussed previously, this does not appear to be a likely explanation. A further 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 5, the birthyears of our index generation are spread out fairly evenly over time, which makes alternative historical events affecting the offspring of the fetally insulted less likely.

We therefore conclude that maternal exposure to the Spanish flu had multigenerational effects on female offspring, and that these effects appear to be driven predominantly by direct biological mechanisms.

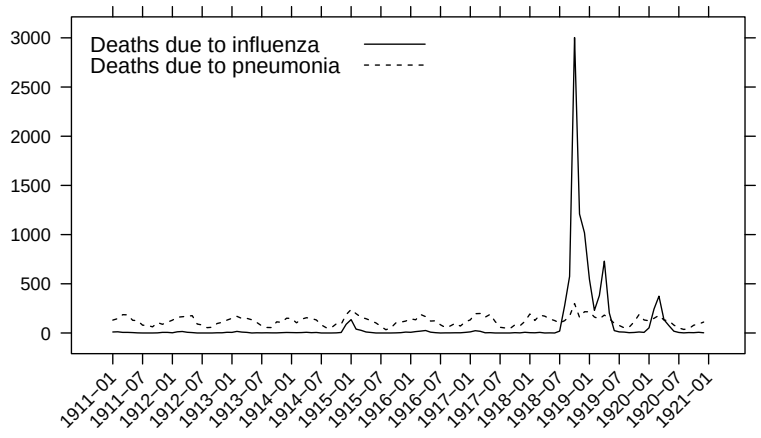
A Appendix

Figure A.1: Age and gender profile of influenza mortality in Sweden.



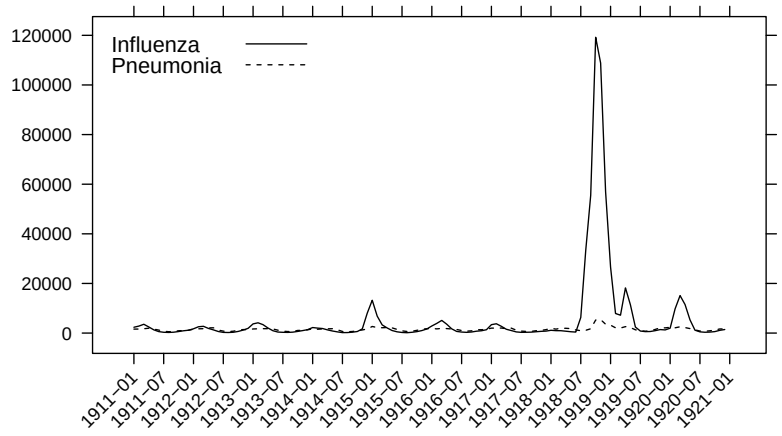
Source: Medicinalstyrelsen 1920

Figure A.2: A comparison of deaths due to influenza and pneumonia in Sweden.



Source: Medicinalstyrelsen 1911-1920

Figure A.3: A comparison of influenza and pneumonia incidence in Sweden.



Source: Medicinalstyrelsen 1911-1920

Table A.1: Results without Stockholm, Gothenburg and Malmö: years of education

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.093	0.069	0.078	−0.074	−0.057	−0.084
	(0.110)	(0.103)	(0.096)	(0.097)	(0.093)	(0.090)
trimester 2	−0.213**	−0.190*	−0.168*	0.025	0.004	0.019
	(0.070)	(0.069)	(0.063)	(0.076)	(0.076)	(0.074)
trimester 3	0.117	0.097	0.091	−0.102	−0.084	−0.090
	(0.076)	(0.072)	(0.067)	(0.077)	(0.070)	(0.067)
Adj. R^2	0.069	0.151	0.186	0.046	0.137	0.188
Num. obs.	24790	24790	24790	25758	25758	25758
Father:						
trimester 1	−0.043	−0.079	−0.100	0.110	0.064	0.047
	(0.096)	(0.090)	(0.089)	(0.087)	(0.080)	(0.081)
trimester 2	0.077	0.110	0.139	−0.288*	−0.287**	−0.266**
	(0.089)	(0.089)	(0.081)	(0.108)	(0.095)	(0.095)
trimester 3	0.021	0.001	−0.008	0.157	0.097	0.121
	(0.069)	(0.066)	(0.064)	(0.083)	(0.080)	(0.079)
Adj. R^2	0.068	0.168	0.186	0.047	0.172	0.188
Num. obs.	24790	24790	24790	25758	25758	25758

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.2: Results without Stockholm, Gothenburg and Malmö: years of education, excluding parents parents conceived after World War I.

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.091	0.124	0.171	−0.139	−0.031	−0.024
	(0.139)	(0.126)	(0.130)	(0.164)	(0.170)	(0.157)
trimester 2	−0.285**	−0.226*	−0.207*	0.121	0.106	0.113
	(0.100)	(0.096)	(0.079)	(0.080)	(0.081)	(0.079)
trimester 3	0.091	0.072	0.062	−0.146	−0.113	−0.091
	(0.085)	(0.082)	(0.079)	(0.102)	(0.095)	(0.091)
Adj. R^2	0.067	0.154	0.190	0.053	0.146	0.193
Num. obs.	13659	13659	13659	14247	14247	14247
Father:						
trimester 1	−0.323	−0.214	−0.264	−0.308	−0.059	−0.070
	(0.184)	(0.183)	(0.172)	(0.161)	(0.165)	(0.149)
trimester 2	−0.219	−0.119	−0.101	−0.419*	−0.348*	−0.293
	(0.163)	(0.146)	(0.142)	(0.172)	(0.166)	(0.173)
trimester 3	−0.130	−0.124	−0.155	0.238	0.206	0.257
	(0.119)	(0.111)	(0.102)	(0.124)	(0.133)	(0.129)
Adj. R^2	0.068	0.174	0.191	0.054	0.177	0.194
Num. obs.	13659	13659	13659	14247	14247	14247

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.3: Results without Stockholm, Gothenburg and Malmö: college attendance

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.010	0.006	0.007	−0.009	−0.007	−0.012
	(0.018)	(0.017)	(0.016)	(0.015)	(0.015)	(0.015)
trimester 2	−0.039**	−0.036**	−0.033**	0.015	0.012	0.014
	(0.013)	(0.012)	(0.011)	(0.014)	(0.014)	(0.014)
trimester 3	0.013	0.009	0.008	−0.018	−0.015	−0.017
	(0.015)	(0.015)	(0.014)	(0.011)	(0.010)	(0.010)
Adj. R^2	0.027	0.102	0.127	0.016	0.089	0.126
Num. obs.	24790	24790	24790	25758	25758	25758
Father:						
trimester 1	0.011	0.005	0.001	0.029	0.022	0.020
	(0.015)	(0.014)	(0.014)	(0.016)	(0.017)	(0.017)
trimester 2	0.005	0.010	0.015	−0.039*	−0.039**	−0.036**
	(0.014)	(0.014)	(0.013)	(0.015)	(0.012)	(0.012)
trimester 3	0.007	0.004	0.003	0.022	0.014	0.018
	(0.012)	(0.010)	(0.010)	(0.012)	(0.012)	(0.012)
Adj. R^2	0.028	0.109	0.128	0.016	0.113	0.126
Num. obs.	24790	24790	24790	25758	25758	25758

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.4: Results without Stockholm, Gothenburg and Malmö: college attendance, excluding parents parents conceived after World War I.

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.002	0.006	0.013	0.004	0.017	0.018
	(0.022)	(0.020)	(0.022)	(0.029)	(0.030)	(0.029)
trimester 2	-0.048^{*}	-0.039^{*}	-0.036^{*}	0.039^{**}	0.037^{**}	0.039^{**}
	(0.019)	(0.018)	(0.016)	(0.012)	(0.013)	(0.013)
trimester 3	-0.004	-0.008	-0.009	-0.016	-0.012	-0.010
	(0.014)	(0.014)	(0.014)	(0.017)	(0.017)	(0.017)
Adj. R^2	0.028	0.104	0.128	0.018	0.093	0.126
Num. obs.	13659	13659	13659	14247	14247	14247
Father:						
trimester 1	-0.062	-0.045	-0.054	-0.026	0.005	0.004
	(0.031)	(0.034)	(0.033)	(0.029)	(0.030)	(0.028)
trimester 2	-0.043	-0.028	-0.025	-0.059^{*}	-0.050^{*}	-0.042
	(0.026)	(0.025)	(0.025)	(0.025)	(0.023)	(0.024)
trimester 3	-0.028	-0.026	-0.032	0.031	0.028	0.034
	(0.023)	(0.022)	(0.020)	(0.019)	(0.021)	(0.021)
Adj. R^2	0.029	0.112	0.130	0.019	0.113	0.127
Num. obs.	13659	13659	13659	14247	14247	14247

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.5: Results without Stockholm, Gothenburg and Malmö: number of children

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.041	0.042	0.041	−0.020	−0.020	−0.021
	(0.029)	(0.029)	(0.029)	(0.043)	(0.043)	(0.044)
trimester 2	−0.010	−0.011	−0.011	0.032	0.032	0.032
	(0.028)	(0.029)	(0.029)	(0.032)	(0.032)	(0.032)
trimester 3	0.050	0.050	0.050	−0.029	−0.029	−0.028
	(0.034)	(0.034)	(0.034)	(0.030)	(0.030)	(0.030)
Adj. R^2	0.004	0.005	0.005	0.006	0.007	0.007
Num. obs.	24790	24790	24790	25758	25758	25758
Father:						
trimester 1	0.013	0.015	0.014	0.058	0.056	0.056
	(0.039)	(0.039)	(0.039)	(0.043)	(0.043)	(0.043)
trimester 2	−0.052	−0.053	−0.055	−0.027	−0.026	−0.025
	(0.033)	(0.033)	(0.033)	(0.041)	(0.041)	(0.041)
trimester 3	−0.007	−0.006	−0.005	0.008	0.007	0.008
	(0.033)	(0.033)	(0.032)	(0.029)	(0.030)	(0.030)
Adj. R^2	0.005	0.005	0.005	0.007	0.007	0.007
Num. obs.	24790	24790	24790	25758	25758	25758

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.6: Results without Stockholm, Gothenburg and Malmö: number of children, excluding parents parents conceived after World War I.

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.127	0.125	0.122	0.011	0.014	0.014
	(0.062)	(0.062)	(0.062)	(0.066)	(0.066)	(0.066)
trimester 2	0.000	−0.001	−0.002	0.095	0.094	0.095
	(0.048)	(0.049)	(0.049)	(0.053)	(0.053)	(0.053)
trimester 3	0.104*	0.104*	0.103*	−0.047	−0.046	−0.045
	(0.041)	(0.041)	(0.041)	(0.042)	(0.043)	(0.043)
Adj. R^2	0.004	0.005	0.004	0.007	0.007	0.007
Num. obs.	13659	13659	13659	14247	14247	14247
Father:						
trimester 1	−0.140	−0.142	−0.146	0.011	0.015	0.015
	(0.097)	(0.096)	(0.096)	(0.101)	(0.102)	(0.102)
trimester 2	−0.081	−0.084	−0.087	−0.023	−0.022	−0.018
	(0.058)	(0.058)	(0.058)	(0.072)	(0.073)	(0.074)
trimester 3	−0.041	−0.040	−0.041	0.021	0.020	0.022
	(0.057)	(0.057)	(0.056)	(0.060)	(0.060)	(0.060)
Adj. R^2	0.005	0.005	0.005	0.006	0.006	0.006
Num. obs.	13659	13659	13659	14247	14247	14247

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

Notes: (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.7: Placebo regression for years of education

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	−0.032	−0.022	−0.041	0.078	0.061	0.051
	(0.082)	(0.068)	(0.065)	(0.106)	(0.105)	(0.100)
trimester 2	0.147	0.147	0.115	−0.089	−0.090	−0.090
	(0.090)	(0.093)	(0.093)	(0.073)	(0.066)	(0.059)
trimester 3	0.082	0.068	0.062	0.087	0.134	0.096
	(0.086)	(0.075)	(0.074)	(0.078)	(0.071)	(0.076)
Adj. R^2	0.069	0.165	0.202	0.052	0.158	0.211
Num. obs.	30331	30331	30331	31730	31730	31730
Father:						
trimester 1	0.099	0.043	0.037	0.051	−0.031	−0.036
	(0.067)	(0.062)	(0.064)	(0.091)	(0.083)	(0.079)
trimester 2	−0.021	−0.028	−0.018	0.117	0.110	0.092
	(0.079)	(0.083)	(0.080)	(0.096)	(0.086)	(0.089)
trimester 3	0.021	0.037	0.027	−0.057	−0.034	−0.018
	(0.053)	(0.056)	(0.060)	(0.058)	(0.056)	(0.054)
Adj. R^2	0.069	0.183	0.203	0.050	0.193	0.212
Num. obs.	30331	30331	30331	31730	31730	31730

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

Notes: Exposure indicators shifted one year back in time. (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.8: Placebo regression for college attendance

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	−0.008	−0.006	−0.009	0.002	0.000	−0.002
	(0.014)	(0.012)	(0.012)	(0.014)	(0.014)	(0.014)
trimester 2	0.011	0.011	0.006	−0.012	−0.012	−0.012
	(0.017)	(0.017)	(0.017)	(0.010)	(0.010)	(0.009)
trimester 3	0.017	0.015	0.014	0.004	0.010	0.005
	(0.015)	(0.012)	(0.013)	(0.011)	(0.011)	(0.011)
Adj. R^2	0.029	0.112	0.140	0.022	0.105	0.145
Num. obs.	30331	30331	30331	31730	31730	31730
Father:						
trimester 1	0.015	0.006	0.005	0.010	0.000	−0.001
	(0.007)	(0.008)	(0.009)	(0.016)	(0.015)	(0.015)
trimester 2	0.005	0.004	0.005	0.018	0.017	0.015
	(0.014)	(0.014)	(0.014)	(0.015)	(0.013)	(0.014)
trimester 3	0.012	0.014	0.013	−0.002	0.001	0.003
	(0.010)	(0.010)	(0.011)	(0.013)	(0.012)	(0.012)
Adj. R^2	0.029	0.122	0.141	0.020	0.131	0.146
Num. obs.	30331	30331	30331	31730	31730	31730

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

Notes: Exposure indicators shifted one year back in time. (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.9: Placebo regression for number of children

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.033	0.033	0.034	0.044	0.044	0.045
	(0.025)	(0.025)	(0.025)	(0.029)	(0.029)	(0.029)
trimester 2	−0.005	−0.006	−0.006	−0.020	−0.020	−0.020
	(0.033)	(0.034)	(0.034)	(0.028)	(0.028)	(0.028)
trimester 3	−0.070*	−0.070*	−0.069	−0.010	−0.008	−0.008
	(0.034)	(0.034)	(0.033)	(0.042)	(0.042)	(0.042)
Adj. R^2	0.005	0.005	0.005	0.006	0.007	0.007
Num. obs.	30331	30331	30331	31730	31730	31730
Father:						
trimester 1	0.035	0.036	0.037	−0.013	−0.014	−0.015
	(0.027)	(0.027)	(0.027)	(0.038)	(0.038)	(0.038)
trimester 2	0.007	0.007	0.007	0.018	0.016	0.015
	(0.035)	(0.035)	(0.035)	(0.047)	(0.047)	(0.047)
trimester 3	0.052*	0.051*	0.052*	−0.010	−0.009	−0.008
	(0.023)	(0.023)	(0.023)	(0.030)	(0.030)	(0.030)
Adj. R^2	0.005	0.005	0.006	0.007	0.008	0.008
Num. obs.	30331	30331	30331	31730	31730	31730

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

Notes: Exposure indicators shifted one year back in time. (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.10: Placebo regression for years of education

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.075	0.102*	0.090*	−0.032	−0.003	0.004
	(0.048)	(0.042)	(0.042)	(0.063)	(0.056)	(0.054)
trimester 2	0.004	−0.008	−0.002	0.031	0.038	0.030
	(0.052)	(0.043)	(0.042)	(0.039)	(0.040)	(0.038)
trimester 3	−0.102*	−0.098*	−0.096*	−0.005	−0.008	−0.001
	(0.047)	(0.045)	(0.043)	(0.047)	(0.040)	(0.041)
Adj. R^2	0.070	0.159	0.195	0.052	0.153	0.205
Num. obs.	67328	67328	67328	69865	69865	69865
Father:						
trimester 1	−0.105	−0.048	−0.050	−0.076*	−0.047	−0.049
	(0.053)	(0.046)	(0.049)	(0.035)	(0.032)	(0.029)
trimester 2	−0.036	−0.050	−0.064	0.042	0.039	0.032
	(0.047)	(0.043)	(0.040)	(0.072)	(0.058)	(0.052)
trimester 3	−0.024	−0.035	−0.033	0.037	0.019	0.014
	(0.045)	(0.047)	(0.044)	(0.057)	(0.058)	(0.059)
Adj. R^2	0.069	0.175	0.194	0.051	0.187	0.205
Num. obs.	67328	67328	67328	69865	69865	69865

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

Notes: Exposure indicators shifted one year forward in time. (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.11: Placebo regression for college attendance

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	0.019*	0.023*	0.021*	−0.004	0.000	0.001
	(0.009)	(0.008)	(0.008)	(0.007)	(0.006)	(0.006)
trimester 2	0.006	0.005	0.006	0.008	0.009	0.008
	(0.010)	(0.009)	(0.008)	(0.006)	(0.006)	(0.006)
trimester 3	−0.018*	−0.017*	−0.017*	0.000	−0.001	0.001
	(0.008)	(0.007)	(0.007)	(0.008)	(0.007)	(0.007)
Adj. R^2	0.029	0.106	0.133	0.020	0.101	0.142
Num. obs.	67328	67328	67328	69865	69865	69865
Father:						
trimester 1	−0.022*	−0.013	−0.013	−0.003	0.001	0.001
	(0.010)	(0.009)	(0.009)	(0.009)	(0.008)	(0.008)
trimester 2	−0.013	−0.015	−0.018	0.000	−0.001	−0.001
	(0.009)	(0.009)	(0.009)	(0.013)	(0.010)	(0.010)
trimester 3	−0.003	−0.005	−0.004	0.000	−0.002	−0.003
	(0.010)	(0.010)	(0.009)	(0.008)	(0.008)	(0.009)
Adj. R^2	0.028	0.115	0.133	0.019	0.127	0.142
Num. obs.	67328	67328	67328	69865	69865	69865

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

Notes: Exposure indicators shifted one year forward in time. (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

Table A.12: Placebo regression for number of children

	Women			Men		
	(1)	(2)	(3)	(1)	(2)	(3)
Mother:						
trimester 1	−0.010	−0.011	−0.011	0.006	0.007	0.007
	(0.022)	(0.022)	(0.022)	(0.025)	(0.025)	(0.025)
trimester 2	−0.003	−0.002	−0.002	0.048	0.048	0.048
	(0.024)	(0.024)	(0.024)	(0.030)	(0.029)	(0.029)
trimester 3	0.001	0.001	0.001	0.000	0.000	0.000
	(0.021)	(0.021)	(0.021)	(0.023)	(0.024)	(0.024)
Adj. R^2	0.005	0.005	0.006	0.007	0.008	0.008
Num. obs.	67328	67328	67328	69865	69865	69865
Father:						
trimester 1	0.009	0.007	0.007	−0.016	−0.015	−0.015
	(0.035)	(0.035)	(0.035)	(0.026)	(0.026)	(0.026)
trimester 2	−0.020	−0.019	−0.018	0.052	0.052	0.052
	(0.023)	(0.023)	(0.023)	(0.033)	(0.033)	(0.033)
trimester 3	−0.010	−0.010	−0.010	0.017	0.016	0.016
	(0.025)	(0.025)	(0.025)	(0.029)	(0.029)	(0.029)
Adj. R^2	0.005	0.005	0.005	0.007	0.007	0.007
Num. obs.	67328	67328	67328	69865	69865	69865

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

Notes: Exposure indicators shifted one year forward in time. (1) refers to baseline model, (2) controls for education level of fetally insulted parent, (3) controls for both parents education. Standard errors reported in parentheses, clustered on the county-level. All exposure indicators are based on a 1.5% threshold. Control variables include an indicator for postnatal flu exposure during the first trimester after birth, parental county of birth fixed effects, calendar month of birth fixed effects for the fetally insulted parent and the index person, birthyear fixed effects and a quadratic (parental) county of birth specific time trend.

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