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MULTIGENERATIONAL EFFECTS OF THE 1918-19 INFLUENZA PANDEMIC IN SWEDEN

by

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Abstract

We use the 1918-19 influenza pandemic in Sweden as a natural experiment to estimate the effects of an in utero health shock on the children of fetally insulted parents. Potential exposure is constructed using historical records of regional influenza incidence matched to birth information available in Swedish register data. For female offspring, potential maternal in utero exposure to the Spanish flu lowers educational attainment by 2.4 months (1.8%) of schooling and decreases the probability of college attendance by 3.7 percentage points (12%). For male offspring, potential paternal in utero exposure in the second trimester decreases years of schooling by 2.9 months (2.1%) and decreases the probability of college attendance by 3.4 percentage points (12%). Maternal effects on female offspring are potentially dominated by direct biological effects, whereas paternal effects on male offspring can plausibly be explained by indirect mechanisms.

JEL classification: I12, J13, J24, N34

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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 adult life. Economists and epidemiologists have extensively tested this hypothesis, and favorable evidence exists both in correlational and quasi-experimental studies, the latter of which establishes causality by exploiting exogenous shocks to fetal health. Due to the wealth of evidence accumulated so far, 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 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 children of fetally insulted parents. 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, 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 behavioral, social and economic outcomes of the fetally insulted parents, which we refer to as indirect effects.

Following the literature on transgenerational epigenetic inheritance[1], 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.[2] Hence, fetal exposure to a health shock also exposes the germ cells that will eventually produce the children, which is why we need to consider the offspring of fetally insulted parents "treated" or "exposed", and thus

[1] While this literature typically documents effects over at least three generations, the current paper is only concerned with two generations: the fetally insulted parents and their children. A supplemental paper with a third generation is in progress.

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

direct effects being possible. Such direct effects have been widely documented in animal experiments for health or health-related outcomes, and some evidence in human populations exists as well. For related overviews, see Franklin and Mansuy (2010), Harper (2005), Jablonka and Raz (2009) and Lundborg and Stenberg (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 by 2.4 months (1.8%) of schooling and decreases the probability of college attendance by 3.7 percentage points (12%). For male offspring, potential paternal in utero exposure in the second trimester decreases years of schooling by 2.9 months (2.2%) and decreases the probability of college attendance by 3.4 percentage points (13%). These intent-to-treat estimates suggest potentially large effects on the infected (e.g. up to 10 months or 7% less schooling). Moreover, using regression controls and a comparison of effects on parents and children, we conclude that maternal effects on female offspring are potentially dominated by direct biological effects whereas paternal effects on male offspring can plausibly be explained by indirect mechanisms.

These results have important policy implications. While the fetal origins literature already points to intergenerational consequences of fetal health, e.g. Currie (2011), our results indicate that such consequences might be stronger than previously imagined, and that returns to investments in maternal health policies potentially span several generations.

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.

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 Ramadan exposure and find effects of these events on either early or later life outcomes.[3] Similar results are obtained when evidence for stress exposure is used, e.g. exposure to 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 e.g. 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 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 led 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

[3]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).

the ones reported by Almond (2006).

Other noteworthy studies are Kelly (2009), Parman (2012) and Karlsson et al. (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. Karlsson et al. (2012) uses Spanish flu mortality in Sweden as a labor supply shock to test empirical predictions of macroeconomic growth models.

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 noteworthy 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 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 as they might capture the importance of maternal health in general.

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).[4] In animal models, evidence for transgenerational effects

[4]Kaati et al. (2007) furthermore establish transgenerational (i.e. over three generations) responses to nutritional availability during the slow growth period, i.e. around the age of

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 Harper (2005) and 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 the peak occured during October to December. Over the course of the pandemic, at least 10% of the Swedish population had been infected.[5] 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 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 substantial threat (Åhman, 1990). See also 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. As in other countries, we see a

8-12. See also van den Berg and Pinger (2013). Moreover, Almond and Chay (2006) use improvements in infant health due to the civil rights era to track intergenerational effects.

[5] 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).

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.[6] We see two smaller waves of influenza infections, though. One shortly after the peak in 1918, and one in the beginning of 1920.[7]

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.[8] There is thus some concern that inference based on mortality data might capture the effect of living in a poor neigbourhood. This is less of a problem with morbidity.[9] Since influenza is an air-borne virus, the random component in infection rates is larger than for the case fatality rate.[10]

The Spanish flu is also particularly suited for testing the effects of a fetal 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 and 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]

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

[7] 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.

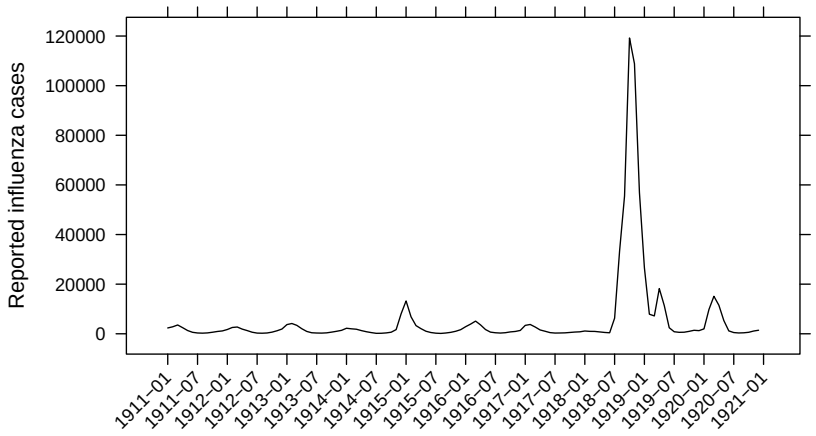
[8] Using apartment size as a measure for individual wealth in two selected parts of Oslo, Mamelund reports that individuals who were able to rent apartments with two, three or four rooms had 34, 41 or 56% lower mortality rates than individuals in one-room apartments, respectively. While these estimates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

[9] 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.

[10] There is some concern about an rural-urban divide, though. We adress this in a robustness check in section 7 by excluding the major cities in Sweden: Stockholm, Malmö and Gothenburg.

[11] See figure A.1. 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

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



Source: Medicinalstyrelsen 1911-1920

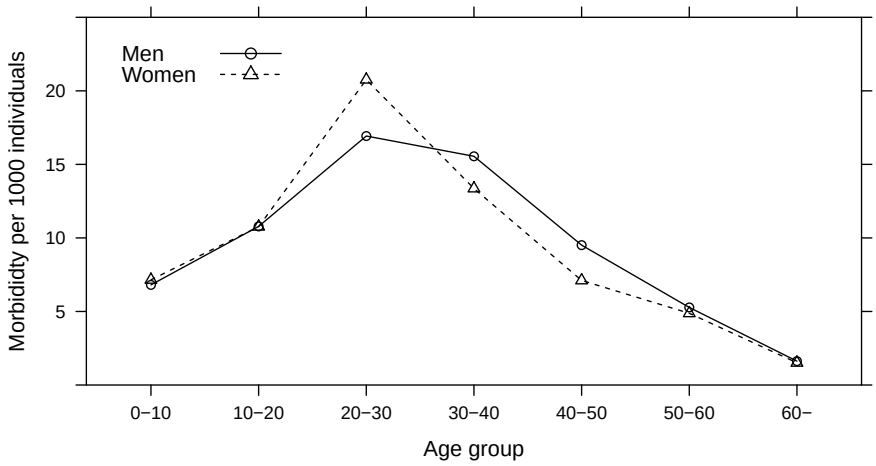
3.2 World War I and parental quality

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 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).

The immediate years after the armistice in November 1918 developed ambiguously, so that the economic and social environments are likely to be different compared to the war-time period. 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

is thus limited.

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



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. *Source:* Medicinalstyrelsen 1920.

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 peace-time production (Montgomery, 1955).

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 argument 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 important aspect to note 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.[12] Most importantly, individuals conceived in late November onwards experienced the Spanish flu as a fetal insult at an early stage during pregnancy, but are potentially born to parents of higher quality. This shift in parental quality might cushion the effect of the flu.

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. Therefore, the subsequent analysis will be carried out with and without excluding individuals conceived after the end of World War I.

4 Data

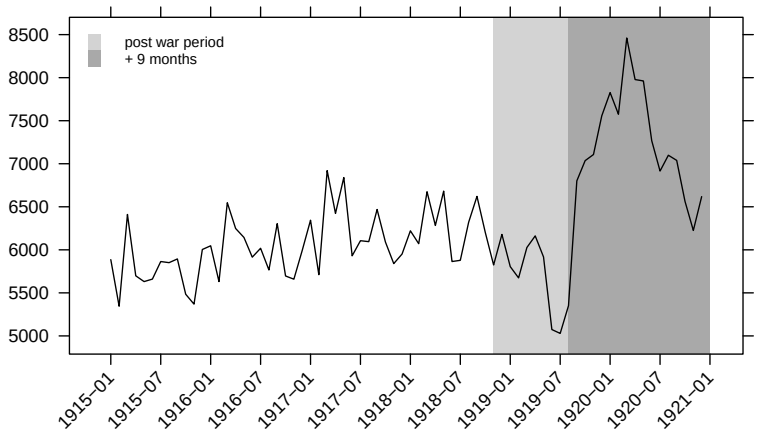
4.1 Measures and construction of influenza exposure

As discussed in section 3.1, we rely on influenza morbidity data, standardized by population as of 1915, for our analysis.[13] Morbidity data by month and

[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 A.4.

[13] 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,

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



Source: Multigeneration register, Statistics Sweden

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 1 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

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 1: 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.

infer the timing of the influenza. This approach also makes our estimates more comparable to the existing literature.[14]

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

[14]Results obtained using morbidity rates instead are included as a robustness check.

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.

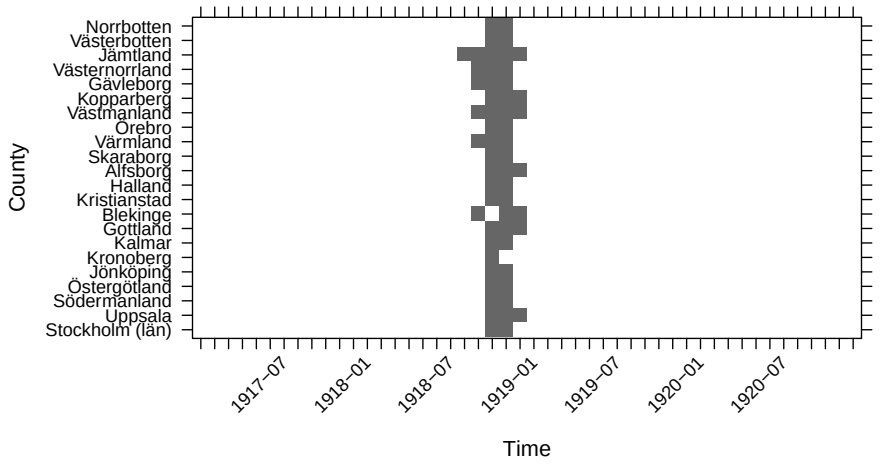
Third, there will be measurement errors due to misclassification. Since our main focus lies on fetal insults, we match individuals to regional influenza morbidity rates during their parents' in utero months. However, since we do not observe the date of conception, we need to infer conception by the time of birth. In the absence of information on 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. This uncertainty as to the timing of influenza exposure is aggravated by the fact that we only observe the birthmonth, and not the exact birthday.[15] 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 before 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.[16]

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 as this threshold captures the peak of the influenza wave fairly well. Figure 4 shows the exact exposure status for all counties and all months during the period considered.

[15] 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.

[16] If misclassification is a severe problem, we expect the exposure dummy for the second trimester to capture influenza exposure more generally.

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



Source: Medicinalstyrelsen 1911-1920

4.2 Individual level data

The individual level data are based on Statistics Sweden's multigeneration 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.

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 individuals with both parents born between 1915 and 1920. As mentioned in section 3.2, all our estimations are carried out including and excluding parents conceived after the end of World War I. Descriptive statistics for the index generation by gender and exposure are shown in table 2. The first row for each variable in table 2 reports the mean and the second (in parentheses) the standard deviation.

The table shows that, unconditionally, mean and women with exposed parents have slightly higher educational attainment and are born later those with unexposed parents. Long-run earnings are higher for exposed women but lower for exposed men. 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 2: Descriptive statistics for the index generation

	Females			*Males*		
	Exposed Parent	*Unexposed Parent*	*All*	*Exposed Parent*	*Unexposed Parent*	*All*
Years of education	11.52 (7.28)	11.43 (7.37)	11.46 (7.35)	11.43 (8.36)	11.38 (8.32)	11.39 (8.33)
College	0.33 (0.22)	0.32 (0.22)	0.32 (0.22)	0.30 (0.21)	0.29 (0.20)	0.29 (0.21)
Long-run earnings[a]	179.22 (6314.22)	176.87 (6116.26)	177.51 (6171.11)	264.71 (19360.40)	266.06 (21550.02)	265.69 (20944.52)
Year of birth	1948 (27)	1947 (28)	1947 (28)	1947 (26)	1947 (28)	1947 (28)
Mother's education	8.13 (4.18)	8.04 (3.80)	8.06 (3.91)	8.12 (4.04)	8.05 (3.76)	8.07 (3.83)
Father's Education	8.87 (7.38)	8.77 (7.00)	8.79 (7.11)	8.94 (7.63)	8.74 (6.85)	8.80 (7.08)
Mother's high school	0.21 (0.16)	0.19 (0.15)	0.19 (0.16)	0.20 (0.16)	0.19 (0.15)	0.19 (0.16)
Fater's high school	0.34 (0.22)	0.32 (0.22)	0.33 (0.22)	0.35 (0.23)	0.32 (0.22)	0.33 (0.22)
Mother's age at birth	29.43 (28.24)	29.44 (29.22)	29.44 (28.95)	29.39 (27.67)	29.53 (28.63)	29.49 (28.37)
Fathers's age at birth	30.34 (27.85)	30.54 (28.50)	30.48 (28.33)	30.29 (27.66)	30.63 (28.32)	30.53 (28.16)
n	8726	23287	32013	9226	24150	33376

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 is mean long-run earnings (measured as 1000 SEK in 2010 prices) and not mean residual earnings that is the measure used in our estimations. *Source:* Multigeneration register, Statistics Sweden

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.[17] 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.[18] The main equation used in the analysis for the index generation is as follows:

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

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 $h(t_c^p)$ indicates a county-specific quadratic time trend in parental time of birth.

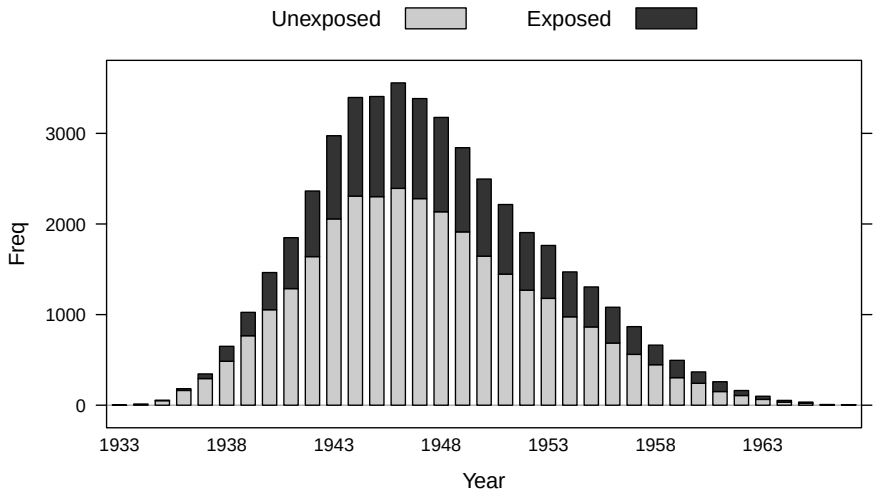
Our motivation for the inclusion of these control variables is as follows: We control for post-birth exposure to avoid confounding exposure in the third trimester with postnatal exposure.[19] The calender month fixed effects capture season of birth effects, which are known to affect outcomes. We include 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, differences in age at birth are proxied for.

[17] 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.

[18] The 1.5% threshold captures the peak of the influenza wave fairly well, which is why we have chosen this particular threshold in our baseline specification.

[19] Note that individuals can be exposed in two consecutive trimesters.

Figure 5: Distribution of birthyears of index generation



Source: Multigeneration register, Statistics Sweden

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:

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

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 and m_i are calender month fixed effects. $h(t_c)$ 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.[20]

[20]For binary dependent variables, the linear probability model has been used. Note that average marginal effects for probit estimates are very similar. Furthermore, following Horrace

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 to 4, we present our estimation results for years of schooling and college attendance, 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 robust to clustering on the county of birth 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 a regression control associated with a given mechanism to affect the estimates for our exposure dummies. We therefore add control variables in model (2) that reflect two indirect mechanisms: first, exposed parents might have lower education levels due to exposure, which could mechanically translate to lower education levels of their children. Second, 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. We therefore add a set of educational attainment dummies for both parents as control variables to model (1), thus flexibly accounting for both mechanisms.[21]

and Oaxaca (2006), note that predicted probabilities lie outside the unit interval for at most 0.3% of the regression-specific sample, and only for 0.01% in the baseline regressions. We are thus confident that the scope for bias due to non-linearity is limited.

[21] Complete information on parental education is missing for roughly 8.7% of the sample, which means they either died or emigrated before 1970 when this data was collected. Inclusion or exclusion of these individuals leaves our estimates virtually unchanged, but since parental exposure in the second trimester appears to increase the probability of missingness, we leave these parents in the sample and include an own category for this group.

EDUCATIONAL ATTAINMENT: As can be seen in table 3, a maternal fetal insult in the second trimester has detrimental effects on educational attainment of female offspring. Exposure lowers years of schooling by 2.4 months (1.8%). This estimate is attenuated to 2.1 months (1.5%) when parental education levels are controlled for, which corresponds to a 13% reduction in the magnitude of the effect. When parents conceived after the end of World War I are excluded, we see an increase in magnitudes. For instance, a maternal fetal insult in the second trimester is now estimated to decrease female offspring's schooling by about 2.6 to 3.1 months (1.9-2.3%). 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 38%) and a loss of statistical significance when controlling for both parental education levels.

For paternal exposure, we find negative effects on male offspring's educational attainment of 2.9 months (2.1%), which is attenuated by 17% to 2.4 months (1.8%) in model (2). In the restricted sample, we find the expected increase in the point estimates and an additional negative effect of exposure in the first trimester. These effects are potentially driven by indirect effects though, since controlling for parental education levels diminishes statistical significance of the estimates.

Table 4 shows similar patterns for college attendance. A maternal fetal insult in the second trimester decreases the probability of attending college for women by about 3.2 to 3.7 percentage points (11-12%), which increases to 3.4 to 4.3 percentage points in the restricted sample. The results on men are somewhat less conclusive. There is some evidence for a positive effect of a maternal insult in the second trimester, but a negative effect for the third trimester.[22] A paternal in utero shock in the second trimester decreases the probability of college attendance for male offspring by about 2.8-3.4 percentage points (10-13%). When parents conceived after the end of World War I are excluded, all magnitudes increase. As before, the effect of paternal exposure decreases by about 30% and loses statistical signficance, so that indirect mechanisms could be driving these results.

[22]These results appear sensitive to the specification and the sample used, though.

Note that for both maternal and paternal effects, the reduced form estimates of model (1) and the attenuated estimates of model (2) are well within the range of one standard error for each estimate. Furthermore, maternal effects appear to be stronger as they are estimated with a much higher precision than the paternal effects.

Long-run earnings: For measures of long-run earnings[23], no robust pattern emerges across samples and specifications, despite the relatively robust patterns in educational outcomes. We speculate that this might either be due to the compressed earnings distribution in Sweden or to larger measurement errors in earnings measures. We thus conclude that there was no discernible effect on earnings. For the sake of brevity, we do not report these tables, but they are available on request.

[23] Apart from our preferred measure, i.e. mean trend-adjusted life time earnings, we also tested (log) mean trend-adjusted earnings at the age 36-40, which did not give different results.

Table 3: Regression results for years of education

	Women				Men			
	Full sample		*WWI*		*Full sample*		*WWI*	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Maternal exposure in:								
trimester 1	0.121	0.090	0.104	0.154	−0.055	−0.062	−0.218	−0.076
	(0.095)	(0.075)	(0.127)	(0.114)	(0.088)	(0.076)	(0.161)	(0.146)
trimester 2	−0.204**	−0.177**	−0.261**	−0.214**	0.071	0.032	0.102	0.083
	(0.065)	(0.055)	(0.089)	(0.070)	(0.071)	(0.063)	(0.086)	(0.075)
trimester 3	0.119	0.119	0.084	0.084	−0.103	−0.082	−0.229*	−0.143
	(0.073)	(0.071)	(0.089)	(0.081)	(0.063)	(0.056)	(0.089)	(0.074)
Adj. R^2	0.070	0.199	0.070	0.202	0.052	0.208	0.060	0.215
Paternal exposure in:								
trimester 1	−0.031	−0.066	−0.157	−0.134	0.029	−0.033	−0.348*	−0.164
	(0.082)	(0.078)	(0.130)	(0.121)	(0.091)	(0.082)	(0.137)	(0.134)
trimester 2	0.092	0.121	−0.200	−0.096	−0.242*	−0.201*	−0.346*	−0.223
	(0.077)	(0.065)	(0.122)	(0.109)	(0.099)	(0.091)	(0.141)	(0.147)
trimester 3	−0.030	−0.022	−0.172	−0.175	0.150	0.097	0.189	0.177
	(0.080)	(0.068)	(0.109)	(0.094)	(0.087)	(0.071)	(0.119)	(0.114)
Adj. R^2	0.069	0.199	0.070	0.203	0.051	0.208	0.058	0.216
Parental SES:	no	yes	no	yes	no	yes	no	yes
Num. obs.	32013	32013	17749	17749	33376	33376	18513	18513

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

(1) refers to baseline model, (2) controls for education levels of both parents. 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 4: Regression results for college attendance

	Women				Men			
	Full sample		*WWI*		*Full sample*		*WWI*	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Maternal exposure in:								
trimester 1	0.015	0.008	0.004	0.008	−0.005	−0.006	−0.019	−0.002
	(0.016)	(0.014)	(0.022)	(0.020)	(0.014)	(0.012)	(0.027)	(0.025)
trimester 2	−0.037**	−0.032**	−0.043*	−0.034*	0.021	0.015	0.030*	0.028*
	(0.011)	(0.009)	(0.017)	(0.014)	(0.012)	(0.011)	(0.014)	(0.013)
trimester 3	0.015	0.015	0.000	0.000	−0.014	−0.012	−0.029*	−0.018
	(0.014)	(0.015)	(0.013)	(0.014)	(0.011)	(0.010)	(0.013)	(0.013)
Adj. R^2	0.030	0.137	0.033	0.138	0.022	0.141	0.025	0.143
Paternal exposure in:								
trimester 1	0.009	0.003	−0.020	−0.020	0.021	0.012	−0.017	0.007
	(0.015)	(0.014)	(0.020)	(0.022)	(0.015)	(0.016)	(0.021)	(0.023)
trimester 2	0.008	0.012	−0.034	−0.019	−0.034*	−0.028*	−0.051*	−0.036
	(0.013)	(0.011)	(0.022)	(0.021)	(0.014)	(0.013)	(0.021)	(0.021)
trimester 3	0.004	0.006	−0.020	−0.021	0.023	0.017	0.033	0.033
	(0.011)	(0.009)	(0.020)	(0.018)	(0.012)	(0.010)	(0.016)	(0.016)
Adj. R^2	0.030	0.137	0.032	0.139	0.020	0.142	0.023	0.145
Parental SES:	no	yes	no	yes	no	yes	no	yes
Num. obs.	32790	32790	18202	18202	33927	33927	18831	18831

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

(1) refers to baseline model, (2) controls for education levels of both parents. 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.

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.

6.2 Parent generation

In table 5, 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.[24]

As can be seen in table 5, 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 (1.4%), 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 effects 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. We will discuss this issue more thoroughly in the following section.

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

Table 5: Regression results for the parent generation

	Women			Men		
	Education	High school	Earnings	Education	High school	Earnings
Full sample:						
trimester 1	0.004	−0.003	732	0.087	0.014	2972
	(0.035)	(0.007)	(1874)	(0.059)	(0.009)	(3291)
trimester 2	0.028	0.007	581	−0.007	−0.009	2467
	(0.032)	(0.007)	(1466)	(0.063)	(0.009)	(4210)
trimester 3	0.028	0.004	561	0.086	0.018	5353
	(0.045)	(0.009)	(1277)	(0.065)	(0.012)	(3958)
Adj. R^2	0.028	0.019	0.019	0.037	0.032	0.029
Num. obs.	54203	54203	54195	52963	52963	52963
Only parents conceived during WWI:						
trimester 1	-0.115^{*}	-0.030^{*}	−1730	−0.124	−0.019	−951
	(0.044)	(0.012)	(2475)	(0.125)	(0.019)	(6240)
trimester 2	−0.016	−0.005	−1507	−0.115	-0.024^{*}	1129
	(0.041)	(0.009)	(1485)	(0.082)	(0.012)	(5231)
trimester 3	−0.001	−0.004	−1484	−0.018	0.002	3988
	(0.067)	(0.014)	(2063)	(0.088)	(0.014)	(4270)
Adj. R^2	0.027	0.020	0.017	0.037	0.033	0.029
Num. obs.	34266	34266	34261	44447	44447	44447

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

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 ster after birth, a quadratic county of birth-specific time trend, and fixed effects for county of birth and calendar month.

6.3 Direct versus indirect effects?

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 propose that if the results were driven by indirect mechanisms, then we would expect the following: first, 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. Second, we expect to see that effects of exposure in a certain trimester for the children is associated with effects for the same trimester for the parents. That is, if the effects of exposure in trimester 2 on children of fetally insulted parents are driven by indirect mechanisms, then exposure in trimester 2 should be associated with effects on the fetally insulted parents. 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.

For women, we find none of these implications in our results. First, controlling for socioeconomic status proxies of parents changes the estimated magnitudes by at most 20% when maternal exposure is considered, with the remaining effect still being statistically significant. Second, maternal exposure in the first trimester appears to have the largest impact on maternal socioeconomic status, whereas no effects of exposure in the second trimester on maternal outcomes can be detected. This is in stark contrast to our findings that maternal exposure in the second trimester affects female offspring. Third, a comparison of effects of fetal insults on women and female offspring seems to imply that the latter is larger. 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 2.6-3.1 months, or 1.9 to 2.3%. 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.

For men, the picture is reversed. When parents conceived after the end of World War I are excluded, we find that controlling for parental education levels tends to substantially attenuate the reduced form effects, and that significance is typically diminished. Furthermore, paternal exposure in the second trimester is associated with a lower probability of high school completion. Given that we find paternal exposure in the second trimester to decrease male offspring's educational attainment, a pathway via indirect mechanisms is credible in this case. Moreover, a paternal insult in the second trimester leads to a decrease in the probability of high school completion that is roughly in the same ballpark of the effect on male offspring (12.6% versus 10-13%). Hence, while the estimated effects could still represent direct mechanisms, we cannot rule out that they represent indirect mechanisms.[25]

6.4 Understanding Effect Sizes

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

[25] Given that germ cells already fully develop at the fetal stage in female fetuses whereas they develop both at the fetal stage and continue development around the time of puberty in men, a gender difference in the effects is maybe not surprising.

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 for potential maternal exposure in the second trimester are -0.204 and -0.037 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 14.8 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.

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.2, 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.

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 placebo exposure by shifting and reshuffling the exposure indicators one year backward in time. The results are reported in tables A.3 to A.4. We find only weak evidence for seasonal effects. In both tables, only two of the reported coefficients are statistically significant, both of which are associated with a positive effect. We interpret this as evidence that seasonal fluctuations do not drive our results.

Third, we investigate if similar patterns emerge when morbidity levels are used

instead of timing indicators. The results are reported in tables A.5 to A.6. Overall, the effects of maternal fetal insults in the second trimester on women are robust, whereas the results of paternal fetal insults in the second trimester essentially disappear. Having said that, the associated signs are still negative.

Fourth, we have experimented with different alternative thresholds and different sample restrictions.[26] Given the patterns observed in our tables, our main results about maternal in utero exposure to the Spanish flu and female offspring's educational attainment are robust against changes in the specifications. Our results about paternal exposure and male offspring's education levels are somewhat less robust, which might be due to a larger estimation uncertainty. 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 are not systematic and potentially due to chance.

8 Discussion and conclusion

We use the 1918-19 influenza pandemic in Sweden as a natural experiment to estimate the effects on the children of fetally insulted parents. Using historical records of regional influenza incidence matched to birth information available in Swedish register data, we are able to construct potential exposure indicators. 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 (2.1-2.4 months). For paternal in utero exposure in the second trimester, we find indications of an analogous effect on male offspring, albeit the results are somewhat less robust. These intent-to-treat estimates indicate potentially large effects on the treated. Furthermore, a comparison of critical periods, the magnitude and the behavior of estimates when control variables are added suggests that the effects of maternal exposure on female offspring are dominated by direct biological effects, whereas effects of paternal exposure on male offspring can plausibly be explained by indirect mechanisms. We generally do not find effects on earnings, though, which might be due to the compressed earnings distribution in Sweden.

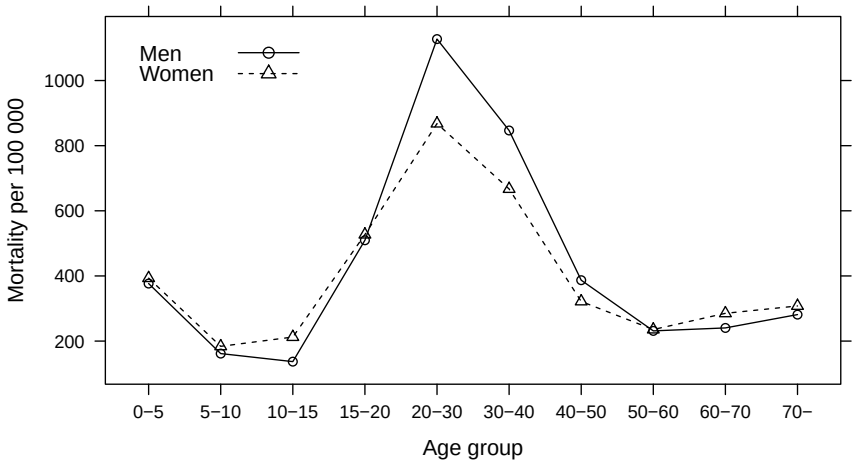
[26] In the interest of space, we do not report these tables, but they are available on request.

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 the children of the fetally insulted parents 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 the Spanish flu had gender-specific multigenerational effects. Maternal in utero exposure to the flu decreased female offspring's educational attainment, and these effects appear to be driven predominantly by direct biological mechanisms. For men, there is some evidence for analogous effects, but these are less robust and plausibly driven by indirect mechanisms.

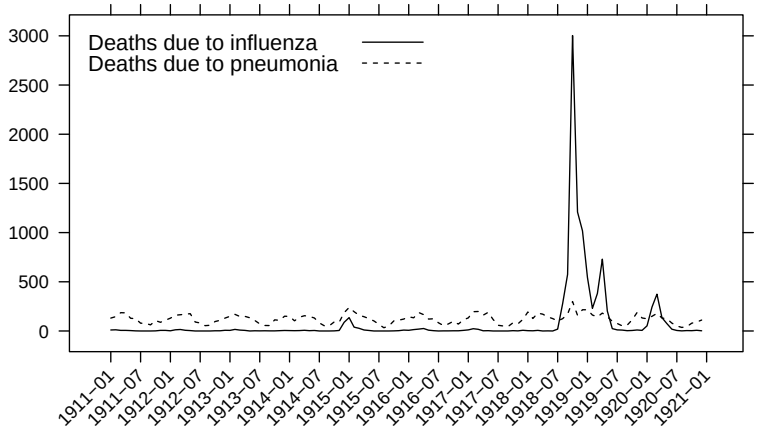
A Appendix

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



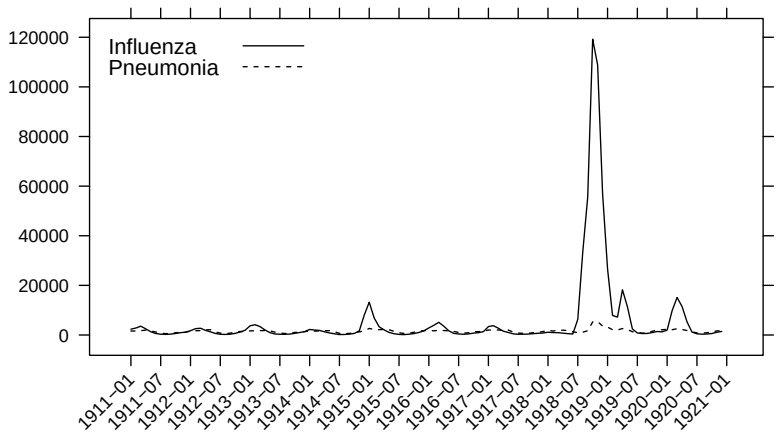
Source: Medicinalstyrelsen 1920

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



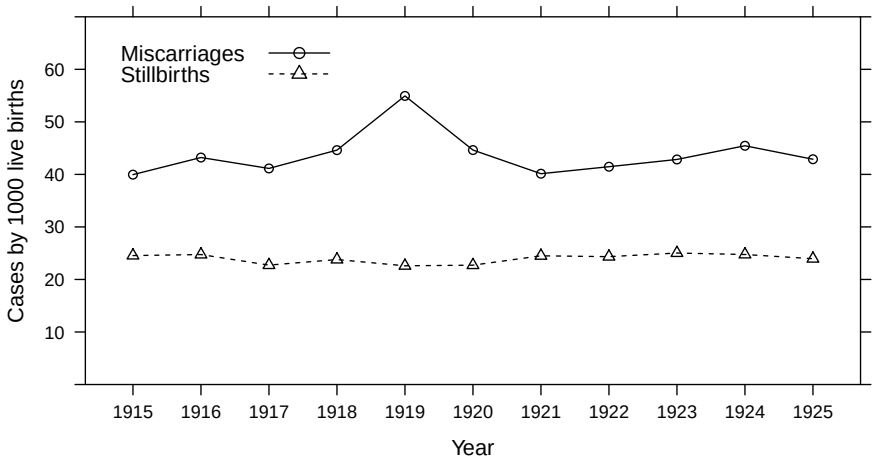
Source: Medicinalstyrelsen 1911-1920

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



Source: Medicinalstyrelsen 1911-1920

Figure A.4: Stillbirths and miscarriages in Sweden, 1915-1925



Source: Medicinalstyrelsen and SCB Befolkningsrörelsen 1915-1925.

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

	Women				Men			
	Full sample		*WWI*		*Full sample*		*WWI*	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Maternal exposure in:								
trimester 1	0.077	0.066	0.041	0.137	−0.088	−0.099	−0.153	−0.044
	(0.108)	(0.092)	(0.132)	(0.120)	(0.092)	(0.086)	(0.167)	(0.160)
trimester 2	−0.226**	−0.189**	−0.286*	−0.212*	0.033	0.030	0.107	0.105
	(0.065)	(0.058)	(0.103)	(0.081)	(0.079)	(0.078)	(0.078)	(0.080)
trimester 3	0.140	0.115	0.109	0.083	−0.095	−0.084	−0.147	−0.093
	(0.074)	(0.064)	(0.080)	(0.071)	(0.078)	(0.069)	(0.100)	(0.090)
Adj. R^2	0.069	0.184	0.068	0.188	0.046	0.185	0.053	0.189
Paternal exposure in:								
trimester 1	−0.061	−0.108	−0.315	−0.271	0.065	0.006	−0.399**	−0.154
	(0.096)	(0.087)	(0.161)	(0.146)	(0.085)	(0.079)	(0.141)	(0.135)
trimester 2	0.095	0.145	−0.197	−0.093	−0.280*	−0.257**	−0.379*	−0.259
	(0.087)	(0.078)	(0.160)	(0.138)	(0.104)	(0.091)	(0.154)	(0.155)
trimester 3	−0.007	−0.024	−0.185	−0.206*	0.136	0.097	0.190	0.204
	(0.072)	(0.062)	(0.104)	(0.087)	(0.077)	(0.072)	(0.127)	(0.133)
Adj. R^2	0.068	0.184	0.069	0.188	0.047	0.185	0.053	0.190
Parental SES:	no	yes	no	yes	no	yes	no	yes
Num. obs.	26129	26129	14396	14396	27018	27018	14943	14943

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

(1) refers to baseline model, (2) controls for education levels of both parents. 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: Regression results for college attendance

	Women				Men			
	Full sample		*WWI*		*Full sample*		*WWI*	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Maternal exposure in:								
trimester 1	0.010	0.008	0.001	0.014	−0.010	−0.012	−0.001	0.012
	(0.018)	(0.016)	(0.022)	(0.021)	(0.015)	(0.014)	(0.030)	(0.030)
trimester 2	−0.044**	−0.038**	−0.052*	−0.041*	0.016	0.016	0.035*	0.035*
	(0.012)	(0.011)	(0.020)	(0.016)	(0.015)	(0.014)	(0.013)	(0.014)
trimester 3	0.018	0.014	0.004	−0.001	−0.015	−0.014	−0.015	−0.008
	(0.015)	(0.014)	(0.014)	(0.013)	(0.012)	(0.011)	(0.017)	(0.017)
Adj. R^2	0.027	0.125	0.028	0.125	0.016	0.123	0.018	0.123
Paternal exposure in:								
trimester 1	0.010	0.001	−0.053	−0.048	0.021	0.013	−0.035	−0.004
	(0.016)	(0.015)	(0.027)	(0.029)	(0.016)	(0.017)	(0.025)	(0.024)
trimester 2	0.007	0.015	−0.042	−0.027	−0.036*	−0.034**	−0.050*	−0.034
	(0.013)	(0.012)	(0.025)	(0.024)	(0.014)	(0.011)	(0.023)	(0.022)
trimester 3	0.007	0.004	−0.029	−0.033	0.018	0.014	0.026	0.029
	(0.012)	(0.010)	(0.022)	(0.018)	(0.011)	(0.010)	(0.018)	(0.020)
Adj. R^2	0.028	0.125	0.029	0.127	0.016	0.123	0.018	0.124
Parental SES:	no	yes	no	yes	no	yes	no	yes
Num. obs.	26129	26129	14396	14396	27018	27018	14943	14943

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

(1) refers to baseline model, (2) controls for education levels of both parents. 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: Placebo regression (-1 year): Results for years of education

	Women				Men			
	Full sample		*WWI*		*Full sample*		*WWI*	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Maternal exposure in:								
trimester 1	−0.029	−0.038	−0.009	−0.039	0.101	0.068	0.132	0.068
	(0.079)	(0.062)	(0.075)	(0.060)	(0.099)	(0.095)	(0.108)	(0.110)
trimester 2	0.141	0.105	0.182*	0.140	−0.096	−0.097	−0.072	−0.127
	(0.085)	(0.089)	(0.087)	(0.090)	(0.073)	(0.061)	(0.083)	(0.071)
trimester 3	0.084	0.075	0.060	0.027	0.056	0.071	0.048	0.061
	(0.087)	(0.074)	(0.092)	(0.080)	(0.072)	(0.069)	(0.083)	(0.078)
Adj. R^2	0.069	0.199	0.070	0.201	0.052	0.208	0.060	0.215
Paternal exposure in:								
trimester 1	0.130	0.069	0.126	0.033	0.059	−0.024	0.005	−0.052
	(0.068)	(0.065)	(0.073)	(0.068)	(0.087)	(0.073)	(0.108)	(0.088)
trimester 2	−0.001	0.006	0.035	0.035	0.117	0.093	0.181	0.120
	(0.075)	(0.077)	(0.085)	(0.082)	(0.096)	(0.086)	(0.124)	(0.103)
trimester 3	0.017	0.026	0.085	0.085	−0.066	−0.030	−0.070	−0.060
	(0.054)	(0.060)	(0.068)	(0.074)	(0.058)	(0.053)	(0.070)	(0.071)
Adj. R^2	0.069	0.199	0.070	0.203	0.051	0.208	0.057	0.216
Parental SES:	no	yes	no	yes	no	yes	no	yes
Num. obs.	32013	32013	17749	17749	33376	33376	18513	18513

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

(1) refers to baseline model, (2) controls for education levels of both parents. 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: Regression results for college attendance

	Women				Men			
	Full sample		*WWI*		*Full sample*		*WWI*	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Maternal exposure in:								
trimester 1	−0.009	−0.010	0.000	−0.004	0.002	−0.002	0.001	−0.007
	(0.012)	(0.010)	(0.013)	(0.012)	(0.014)	(0.014)	(0.016)	(0.016)
trimester 2	0.011	0.005	0.024	0.019	−0.014	−0.014	−0.007	−0.014
	(0.016)	(0.017)	(0.015)	(0.016)	(0.011)	(0.010)	(0.012)	(0.011)
trimester 3	0.016	0.015	0.011	0.006	0.002	0.005	0.002	0.004
	(0.014)	(0.012)	(0.015)	(0.014)	(0.010)	(0.010)	(0.011)	(0.011)
Adj. R^2	0.029	0.137	0.031	0.139	0.021	0.142	0.025	0.144
Paternal exposure in:								
trimester 1	0.019*	0.009	0.012	−0.002	0.013	0.003	0.003	−0.003
	(0.008)	(0.009)	(0.010)	(0.010)	(0.016)	(0.014)	(0.019)	(0.017)
trimester 2	0.006	0.007	0.023	0.024	0.016	0.014	0.024	0.016
	(0.013)	(0.013)	(0.018)	(0.017)	(0.015)	(0.014)	(0.023)	(0.020)
trimester 3	0.012	0.014	0.019	0.019	−0.004	0.000	−0.008	−0.007
	(0.010)	(0.011)	(0.012)	(0.013)	(0.012)	(0.011)	(0.013)	(0.013)
Adj. R^2	0.029	0.138	0.031	0.140	0.020	0.143	0.022	0.146
Parental SES:	no	yes	no	yes	no	yes	no	yes
Num. obs.	32013	32013	17749	17749	33376	33376	18513	18513

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

(1) refers to baseline model, (2) controls for education levels of both parents. 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: Using morbidity levels: Results for years of education

	Women				Men			
	Full sample		*WWI*		*Full sample*		*WWI*	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Maternal exposure in:								
trimester 1	0.040	0.035	−0.077	−0.012	−0.070	−0.059	−0.128	−0.016
	(0.046)	(0.038)	(0.096)	(0.084)	(0.048)	(0.039)	(0.118)	(0.109)
trimester 2	−0.116**	−0.097**	−0.190**	−0.142*	0.040	0.007	0.035	0.043
	(0.038)	(0.032)	(0.063)	(0.055)	(0.038)	(0.031)	(0.060)	(0.044)
trimester 3	0.108*	0.092	0.025	0.031	−0.063	−0.045	−0.124	−0.043
	(0.047)	(0.046)	(0.075)	(0.065)	(0.039)	(0.036)	(0.080)	(0.073)
Adj. R^2	0.070	0.199	0.070	0.202	0.052	0.208	0.060	0.215
Paternal exposure in:								
trimester 1	0.029	0.009	−0.032	−0.035	0.019	0.001	−0.125	−0.020
	(0.042)	(0.037)	(0.073)	(0.070)	(0.056)	(0.041)	(0.116)	(0.080)
trimester 2	0.022	0.046	−0.151*	−0.094	−0.093	−0.092	−0.110	−0.059
	(0.044)	(0.039)	(0.071)	(0.064)	(0.055)	(0.046)	(0.097)	(0.087)
trimester 3	0.036	0.023	−0.039	−0.075	0.076	0.050	0.134	0.139
	(0.056)	(0.052)	(0.069)	(0.067)	(0.063)	(0.056)	(0.082)	(0.073)
Adj. R^2	0.069	0.199	0.070	0.203	0.051	0.208	0.057	0.216
Parental SES:	no	yes	no	yes	no	yes	no	yes
Num. obs.	32013	32013	17749	17749	33376	33376	18513	18513

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

(1) refers to baseline model, (2) controls for education levels of both parents. 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: Using morbidity levels: Results for college attendance

	Women				Men			
	Full sample		*WWI*		*Full sample*		*WWI*	
	(1)	(2)	(1)	(2)	(1)	(2)	(1)	(2)
Maternal exposure in:								
trimester 1	0.005	0.004	−0.018	−0.010	−0.004	−0.003	−0.002	0.012
	(0.009)	(0.008)	(0.017)	(0.015)	(0.008)	(0.006)	(0.018)	(0.017)
trimester 2	−0.019*	−0.016*	−0.027*	−0.020	0.007	0.003	0.012	0.014
	(0.007)	(0.006)	(0.011)	(0.010)	(0.006)	(0.005)	(0.011)	(0.009)
trimester 3	0.014	0.012	−0.006	−0.006	−0.004	−0.002	−0.007	0.004
	(0.010)	(0.010)	(0.013)	(0.012)	(0.006)	(0.006)	(0.011)	(0.010)
Adj. R^2	0.029	0.137	0.031	0.139	0.021	0.142	0.025	0.144
Paternal exposure in:								
trimester 1	0.009	0.006	−0.014	−0.014	0.012	0.009	−0.010	0.002
	(0.008)	(0.008)	(0.012)	(0.013)	(0.008)	(0.007)	(0.015)	(0.012)
trimester 2	0.004	0.007	−0.034**	−0.025*	−0.014	−0.014*	−0.017	−0.010
	(0.006)	(0.005)	(0.011)	(0.010)	(0.008)	(0.006)	(0.015)	(0.014)
trimester 3	0.004	0.002	−0.014	−0.019	0.010	0.007	0.015	0.016
	(0.007)	(0.006)	(0.013)	(0.012)	(0.008)	(0.007)	(0.010)	(0.010)
Adj. R^2	0.029	0.138	0.031	0.140	0.020	0.143	0.023	0.146
Parental SES:	no	yes	no	yes	no	yes	no	yes
Num. obs.	32013	32013	17749	17749	33376	33376	18513	18513

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

(1) refers to baseline model, (2) controls for education levels of both parents. 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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